

IS METAMEMORY IMPLICIT?
EVIDENCE OF PRIMING AND ANTIPRIMING IN INDIVIDUALS
WITH AND WITHOUT TRAUMATIC BRAIN INJURY

A DISSERTATION
SUBMITTED TO THE FACULTY OF THE GRADUATE SCHOOL
OF THE UNIVERSITY OF MINNESOTA
BY

Pradeep Ramanathan

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

Mary R. T. Kennedy, Adviser

July, 2009

© Pradeep Ramanathan 2009

Acknowledgements

To adapt a contemporary American saying, it takes a village to train a doctoral student. First among the individuals in my village has been my doctoral adviser, Dr. Mary Kennedy. In India, we say that one can never repay the debt of gratitude we owe our parents or our mentor (guru). The unfailing and constant mentorship Mary has provided over the years (including sacrificing many hours on nights and weekends), and her ever-present kindness and generosity are a source of inspiration. As a mentor, she has helped to shape and hone my thinking and writing. The scholar I am today is in large part due to her. And then there has been the almost maternal care she has shown, whether knitting scarves to keep her doctoral students warm in the bitter cold January days of Minnesota, or bringing us chocolates. I hope that I can model for the doctoral students I one day mentor the example Mary provided to me.

The words above apply almost verbatim to Dr. Chad Marsolek, who has been my co-advisor through the last four years. His warmth and good will, his humor and humility are as much an inspiration to me as his scholarship. Chad was the one who lit the fire of my interest in cognitive neuropsychology, and who patiently gave me the opportunity and guidance in exploring with unfettered abandon the world of implicit memory and artificial neural networks. And his Amazon.com gift cards each Christmas played no small part in paying for my textbook purchases each year!

I would also like to thank my two other committee members, Drs. Peter Watson and Benjamin Munson. Both Peter and Ben have gone well above and beyond the call of duty, contributing much time, energy, and mentorship to my growth and maturity as

a scholar. Peter taught me how to read journal articles critically – a skill for which I will be ever grateful to him. Ben taught me to think about my own research from fresh perspectives by temporarily wearing the hats of other disciplines (though he does not realize he had this effect). Both have also been so very patient and giving of their time. Dr. Edward Carney also deserves special mention, providing so much mentorship and assistance that he might as well have been a fifth committee member. With patience, humor, and much humanity, he taught me all about PERL scripts, the idiosyncrasies of E-Prime, and countless other details of the daily work of a scientist.

I would also like to thank my first doctoral adviser, Dr. Nancy Pearl Solomon. She was as much a friend and coach to me, during my clinical master's degree, as she was an adviser. I have missed her cheerful smile and personal caring. Drs. Deirdre D. (D.D) Michael, and George (Shep) Goding Jr. at the Lions Voice Clinic in the department of Otolaryngology, made the last seven years of my time at the University of Minnesota both a financial possibility (nothing beats a steady job to support a Ph.D.) as well as the necessary clinical escape from my doctoral rigors to help me keep my sanity. Their friendship, kindness, and camaraderie are sorely missed.

I would like to thank my doctoral siblings and cousins, Hsinhuei Chiou, Rebecca Deason, Lane Beckes, Mike Blank, and Vaughn Steele. Their companionship and example along the path have been the reassurance I've needed to keep moving forward. I am indebted to Krystle Baumgarten for her help in running subjects through standardized tests, and to Katie Huwe for administrative help. Mr. Eric Lindell of the Minnesota Historical Society is owed a huge thanks for fashioning a beautiful custom-made chin rest. I am deeply grateful to Ardis Sandstrom and to the Brain Injury

Association of Minnesota for their tremendous help in the recruitment of research participants. I am also indebted to my colleagues at North Memorial Medical Center, Park Nicollet, and Hennepin County Medical Center for their assistance with recruitment of participants. And of course, I am most deeply indebted to, and appreciative of, all of those individuals who kindly gave their time and support by participating as research subjects in this dissertation.

Perhaps most importantly, I owe James Mislá a tremendous debt of gratitude. As my best friend since the seventh grade, Jim has been my friend, psychotherapist, and cheerleader as I struggled through the darkest hours of despair that this dissertation would be my own personal “Groundhog Day”.

Finally, I am profoundly grateful to the University of Minnesota for its generous funding of my doctoral dissertation, through the Doctoral Dissertation Fellowship, several block grants, a scholarship from the College of Liberal Arts, and the Bryng Bryngelson fund of the Department of Speech-Language-Hearing Sciences. The Minnesota Speech-Language-Hearing Association also contributed to the funding of this research through its scholarship.

Thank you to the village that trained this doctoral student.

Dedication

This dissertation is dedicated to those who have sustained a traumatic brain injury. May the implications of this work and its offshoots ultimately contribute to the advancement of rehabilitative praxis, that TBI survivors may heal faster and enjoy a better quality of life.

Abstract

Implicit memory refers to the phenomenon of prior exposure to a stimulus influencing cognitive processing on subsequent exposure to that stimulus, irrespective of one's awareness of such influence. Metamemory refers to the presumably explicit act of making judgments about one's memory. Theories vary regarding possible relationships between implicit memory and metamemory, with mixed findings of previous research. Some researchers argue that there is no relationship, that metamemory does not rely on input from implicit memory only on explicit memory (Hart, 1965; Koriat, 1995), while others argue that metamemory is driven only by implicit processes (Reder & Schunn, 1996). Still others argue that the metamemory system relies on input from both implicit and explicit memory (Vernon & Usher, 2003). To date, no study has examined whether subliminal masked priming and antipriming can influence individuals' metamemory judgments of learning (JOLs).

The present study independently varies subliminal masked priming conditions (using baseline, prime, and antiprime conditions) and observes the influence on participants' immediate and delayed JOLs and relative predictive accuracy, in a paired associate learning task. This study also examines long term repetition priming and antipriming (measured in a visual object identification task) to determine whether the magnitudes of these effects correlate with those found in the paired associate learning task. Both neurologically normal individuals and those with traumatic brain injury (TBI) participated in this study. Metamemory deficits are common after TBI. Thus, inclusion of this clinical population in the present study is critical in evaluating whether or not such individuals' metamemory judgments can be influenced by, or correlated

with, priming or antipriming. Furthermore, to date, no study has examined antipriming among TBI survivors; therefore their participation in the visual object identification task will shed light on antipriming in that population, and will provide a perceptual implicit memory benchmark against which to compare any findings of implicit metamemory.

Results demonstrate no main effect of subliminal masked priming and antipriming on participants' metamemory judgments for JOL ratings, predictive accuracy (gamma correlation), or response times for judgments of the likelihood of future recall of target words. However, there was a trend towards an interaction effect of priming and JOL timing (i.e.: immediate vs. delayed), such that JOLs made immediately after study were higher for the primed items than for the baseline or antiprimed items. In contrast, antipriming did significantly influence explicit recall, with slowing of explicit recall response times for antiprimed target words, relative to word-pairs in the baseline condition. Difference scores between JOL ratings and recall accuracy reflected overconfidence among TBI survivors, and good calibration among controls. There was a significant decrease in the overconfidence of TBI survivors for antiprimed items. However, much of this lowering was caused by an increase in recall accuracy for antiprime items. In the visual object identification task, antipriming was robustly observed for both participant groups; there was significant slowing observed for responses to antiprimed items. Finally, a mild positive correlation between the degree of priming for visual object identification and the degree of priming of relative predictive accuracy in the paired associate learning task was found across participants.

The present research finds weak evidence for the role of implicit memory, in the form of masked priming and antipriming, in JOLs and relative predictive accuracy in

paired associate learning. Future research providing masked priming stimuli immediately prior to the solicitation of JOLs may address possible attenuation of such effects which may have occurred in the present study. Additionally, using numbers from the JOL rating scale as subliminal stimuli may shed additional light on the type of representation taken as input by the metamemory system. Finally, exploration of long term priming and antipriming of metamemory in the TBI population should be undertaken to determine whether or not there is a priming benefit to metamemory judgments through supraliminal implicit memory.

Table of Contents

List of Tables.....	x
List of Figures.....	xii
Introduction.....	1
Implicit Memory, Priming, and Implicit Learning.....	9
Metamemory.....	13
Neural Substrates Of Implicit Memory And Metamemory.....	18
Implicit Memory and Priming.	19
Metamemory	25
Measures of Metamemory	32
Judgments of Learning in the TBI Population.....	36
Is Metamemory Implicit?	39
Purpose of the Present Study.....	51
Research Questions.....	52
Significance of the Research.....	56
Method.....	62
Summary	62
Participants.....	64
Experimental Design.....	71
Implicit metamemory task.....	71
Task summary.....	71
Materials.....	74
Hardware.....	77
Procedures.....	78
Variables.....	82
Visual antipriming task.....	83
Task summary.....	83
Materials.....	84
Hardware.....	85
Procedures.....	85
Variables	88

Results.....	89
Overview.....	89
Validation task.....	89
Implicit metamemory task.....	95
Visual antipriming task.....	103
Additional analyses.....	106
Discussion.....	110
References.....	150
Tables.....	168
Figures.....	194
Footnotes.....	208
Appendix.....	209
Appendix A:	209
Appendix B:	213
Appendix C:	217
Appendix D:	218
Appendix E:	221
Appendix F:	226
Appendix G:.....	228

List of Tables

Table 1.	Demographic Characteristics of Healthy Controls (n=14) and Adults with Brain Injury (n=17)	168
Table 2.	Detailed Demographic information for ABI survivors.....	169
Table 3.	Detailed Demographic information for Control participants.....	170
Table 4.	Descriptive Brain Injury Information.....	171
Table 5.	Means, <i>SDs</i> , and statistical comparisons of neurocognitive performance for adults with ABI (n = 17) and healthy controls (n = 14)	174
Table 6.	Means and <i>SDs</i> for word lists used in implicit metamemory task.....	176
Table 7.	Measures of Normality for the Validation Task, by participant group..	177
Table 8.	Means and Standard Deviations for Percent Old and Response Times, for each participant group, in the Validation Task.....	178
Table 9.	Repeated Measures Analysis of Variance for Percent Old and Response Time in the Validation Task.....	179
Table 10.	Pairwise Comparison for the Effects of Item Type on Percent Old and Response Times, in the Validation Task.....	180
Table 11.	Measures of Normality for each Cell of the Implicit Metamemory Task, by Participant Group.....	181
Table 12.	Mauchly's Test of Sphericity Results for All Dependent Measures in Implicit Metamemory task, for the Main Effect of Prime and the Prime-by-JOL timing Interaction.....	183

Table 13. Descriptive Statistics for the Implicit Metamemory Task: Means and Standard Deviations for JOL, Recall Accuracy, Gamma Correlation, JOL RT, and Recall RT, for each participant group. ...	185
Table 14. Descriptive Statistics for the Implicit Metamemory Task: Means and Standard Deviations for JOL RT, and Recall RT, for each participant group.....	186
Table 15. Repeated Measures Analysis of Variance for JOL, Recall Accuracy, Gamma Correlation, JOL RT, and Recall RT in the Implicit Metamemory Task.....	187
Table 16. Measures of Normality for the Visual Antipriming Task, by participant group.....	190
Table 17. Means and Standard Deviations for Recall Accuracy and Response Times, for each participant group, in the Visual Antipriming task.....	191
Table 18. Repeated Measures Analysis of Variance for Accuracy and Response Time in the Visual Antipriming Task.....	192
Table 19. Repeated Measures Analysis of Variance for Difference Scores in the Implicit metamemory Task.....	193

List of Figures

Figure 1. Nelson and Narens' (1990) model of metacognitive monitoring and control.....	194
Figure 2. Nelson and Narens' (1994) model of metamemory monitoring and control.....	195
Figure 3. Recursive hierarchical model of frontal lobe function, Stuss (1991)	196
Figure 4. Sequence of task steps for the implicit metamemory task.....	197
Figure 5. Sequence of task steps for the visual antipriming task (Marsolek et al., 2006)	198
Figure 6. Examples of the grey scale visual objects used in the visual antipriming experiment.	199
Figure 7. Results of the validation task immediately following the last trial block of the implicit metamemory task.	200
Figure 8. Subliminal masked priming and antipriming of target words does not affect JOL ratings (collapsed across JOL timing condition) for either participant group.....	201
Figure 9. Collapsed across JOL timing condition, the TBI survivor group shows a significant reduction in confidence (difference score between JOL rating and recall accuracy) for antiprimed items. They are also generally overconfident, while the control group is well calibrated.....	202

Figure 10. Long term repetition priming in visual object identification task results in significantly increased identification accuracy for both participant groups. Antipriming has non-significantly decreased accuracy. All responses were self-paced..... 203

Figure 11. When responses in the visual object identification task were rejected if they took longer than two seconds, the decrease in object identification accuracy for antiprimed items becomes statistically significant for both participant groups..... 204

Figure 12. Weak correlation [$r = 0.334$ ($p = .089$)] between the relative benefit of priming for gamma correlation to the priming benefit to object identification accuracy (where priming benefit is defined as the ratio of primed values to baseline values). 205

Figure 13. Each data point represents average JOL vs. average recall accuracy for all items for one individual in the delayed JOL timing condition. Relative to baseline, antipriming decreases JOL for controls with poor recall, and increases it for those with high recall..... 206

Figure 14. Each data point represents average JOL vs. average recall accuracy for all items for one individual in the delayed JOL timing condition. Relative to the baseline trend, antipriming produces an overall reduction in JOL for TBI survivors across the range of recall accuracy, relative to baseline..... 207

INTRODUCTION

Over a century of research has distinguished a number of types of memory representations and processes (Lashley, 1929; Squire, 1987; Gabrieli, 1998). One such distinction is between implicit and explicit memory. Implicit memory refers to changes in the speed or accuracy of processing a stimulus attributable to prior processing of that (or a similar) stimulus, irrespective of awareness of such changes (Schacter, Chiu, & Ochsner, 1993). An example of this is repetition priming: viewing an image or reading a word improves the accuracy and response time for subsequently identifying that item for some period of time thereafter, whether or not the individual is aware of this effect. This priming effect of improved performance may last anywhere from several seconds to several weeks, depending on numerous factors (Squire, 1994). In contrast, explicit memory refers to the conscious recollection or recognition of items from memory. Explicit memory includes recall or recognition of facts and events, such as general knowledge, episodic memories, autobiographical information, etc. (Schacter, 1987).

Over the last several decades the study of metamemory (awareness, monitoring, and control of one's memory processes) has been a growing field of research. Initially, metamemory was presumed to be an explicit, declarative, process in which agents are aware of their monitoring and control of memory (Tulving, 1985). In the last two decades, researchers have begun to examine relationships between metamemory and implicit memory. Some researchers claim that implicit memory (in the form of pre-retrieval information and processes) does not affect metamemory judgments, and that metamemory is based on post-retrieval processes (Jameson, Narens, Goldfarb, &

Nelson, 1990; Koriat, 1993; Kinoshita, 1997). Others claim that implicit memory does influence metamemory decisions (Rajaram, 1993; Reder & Schunn, 1996). In fact, Reder and Schunn (1996) state that metacognitive control is based *primarily* on implicit learning and memory. Vernon and Usher (2003), however, claim that both implicit and explicit processes are involved with metamemory judgments, but that implicit effects dominate very rapid (e.g.: < 850 ms) metamemory judgments, while explicit memory dominates slow metamemory judgments.

The current research has a two-fold purpose. The first is to test the hypothesis that metamemory is influenced by implicit processes in a masked priming paradigm. To do so, the study will make use of JOLs, as well as priming and the recently discovered phenomenon of antipriming (Marsolek, Schnyer, Deason, Ritchey, & Verfaellie, 2006). The second purpose has the clinical research goal of examining deficits to metamemory and implicit memory as sequelae of TBI. The inclusion of both neurologically normal and traumatically brain injured participants serves both purposes. Towards testing the hypothesis, it is expected to shed light on whether or not individuals living with TBI are able to make use of implicit memory to facilitate metamemory function – a skill which is typically an area of some deficit in this population. Furthermore, given the variability of degree of deficit to metamemory in the TBI population, their inclusion will provide a wider range of implicit memory and metamemory abilities over which to determine whether the two forms of memory are correlated.

IMPLICIT MEMORY, PRIMING, AND IMPLICIT LEARNING

Schacter (1987) defines implicit memory as the non-conscious influence of past experiences on future behavior. He includes priming, skill learning, and habit formation as examples of implicit memory. Squire (1994) adds simple classical conditioning to this list. Forster, Mohan, and Hector (2003) differentiate three main types of priming: identity or repetition priming, form priming, and semantic or associative priming. Repetition priming is the strongest type of priming, and can be defined as the increase in accuracy, or decrease in response time, for identification of a stimulus when that same item has been recently processed (e.g.: valley – VALLEY, where the prime and target represent the same item, albeit with a change of case between the initial prime presentation and the subsequent target presentation that occurs shortly after presentation of the prime). Form priming involves a slight change of form between the prime and the target, either by a single letter or by letter transposition (e.g.: elophant – ELEPHANT, and elehphant – ELEPHANT respectively). Semantic priming occurs when the prime and target bear some relationship; this is further subdivided into three general types: morphological priming, involving a morphological difference between prime and target (e.g.: give – GAVE), semantic association priming (dog – CAT), and translation priming wherein the prime and target represent the same word in two different languages (hombre – MAN).

Relevant to the present research, conceptual priming should be added to the above classification scheme; it arguably differs from semantic priming in both the degree of abstraction or semantic relatedness of the prime and target words, and on the

task methodology. As an example of the former, a picture of a length of rope may prime the concept of exercise for a fitness enthusiast, whereas for a construction workman it may prime work-related activities. The relationships between the image of a rope and the concepts of exercise and work are not universal and immediate semantic associations, and indeed they are not quite so direct as doctor-NURSE, or dog-CAT. Shah and Kruglanski (2003) further elucidate conceptual priming by pointing to the functional rather than purely semantic nature of the relationship between the prime and target. As such, conceptual priming may be viewed as priming at a more abstract and fluid level of association than semantic priming. The two primary types of tasks used to measure conceptual priming are category generation (producing exemplars given a category name), and answering general knowledge questions (Vaidya et al., 1997). However, additional tasks have also included category verification, word-cued association, and making abstract/concrete or living/non-living judgments of the target tokens (Vakil & Sigal, 1996; Bishop & Curran, 1998; Mulligan, Guyer, & Beland, 1999; Friedman et al., 2003). Taken together, these studies on conceptual priming provide evidence that cognitive processes which occur at a fairly high level (e.g.: at an abstract/conceptual level) can be influenced by priming. This is particularly relevant to the present research in that it is at an abstract level (metacognition) that the present research attempts to find evidence of the effects of priming; this will be further addressed below.

Apart from such a taxonomy of priming, another distinction has been in terms of the methods of priming. Two of the main methodologies are long-term priming and subliminal masked priming (Forster & Davis, 1984). Whereas the present research will

employ both long term repetition priming and subliminal masked priming to examine the effects of priming on metamemory, a brief description of each is germane. Long term priming is so called due to the long time interval between presentation of the priming stimulus and the target stimulus to which the subject must respond; this time interval may be on the order of several minutes, with one or more intervening stimuli, or even brief tasks. Furthermore, the prime stimulus is presented long enough to be clearly and consciously perceived by the subject; in fact, they are often asked to make like/dislike, or other judgments, to ensure elaborative encoding of the prime stimulus (Forster & Davis, 1984; Schacter, 1987). Subliminal masked priming, conversely, is so called because the priming stimulus is very briefly presented (e.g.: < 60 ms), and is preceded by a forward mask (a masking row of nonsense characters, such as #####, or #&#&#&#&#&#, etc.) for about 500 ms. Sometimes the prime stimulus is additionally succeeded by a backward mask – another 500 ms presentation of the nonsense character string. As a result of these manipulations, the prime stimulus is rendered subliminal and the research participant is not consciously aware of the presence of the prime (Forster & Davis, 1984).

To this last point, much early research concerned the debate as to whether priming involved conscious or unconscious perception, particularly with regard to subliminal masked priming (Marcel, 1983). However, a great deal of research exploring many aspects of priming has shown a strong priming effect when the priming stimuli are subliminally masked and under circumstances which cannot be attributed to conscious awareness (Evetts & Humphreys, 1981; Forster & Davis, 1984; Cheesman, & Merikle, 1984; Greenwald, Klinger, & Liu, 1989; Greenwald, Klinger, & Schuh, 1995;

Kihlstrom, 1999). Of particular note, Dehaene, Naccache, Cohen, Le Bihan, Mangin, Poline et al. (2001) used fMRI and ERP to study which cortical areas were activated while participants attempted to read aloud masked and unmasked words flashed on a screen for 43 – 71 ms. If they could not read the word, but thought they had seen a word flash by, participants were to indicate that they had detected a word.

Behaviorally, unmasked words were read or detected 90.3% of the time, while masked words were detected only 0.7% of the time and were only read aloud once by one participant. Unmasked words activated a broad network of cortical areas, through the left occipito-temporal, parietal, frontal and prefrontal areas. Conversely, masked words did not activate prefrontal or parietal areas. Rather, only a much reduced portion of the left fusiform gyrus and a small region within the left precentral sulcus were activated. The authors contend that the absence of prefrontal activation for masked tokens, correlating with the 0.7% behavioral detection rate in that condition, provides a neural basis explaining the absence of conscious awareness or conscious perception of masked words. Since the present study attempts to examine whether individuals' metamemory judgments may be primed without their conscious awareness, this distinction that subliminal masked priming is indeed a process not available to conscious awareness is an important one.

In contrast to masked priming, stimuli in long term priming tasks appear for many hundreds, and even thousands, of milliseconds, with research participants making judgments or ratings of the priming stimuli. Therefore, long term priming constitutes a supraliminal task, and the stimuli are perceptible by conscious awareness (Forster, Mohan, & Hector, 2003).

Although the issue of conscious awareness has largely subsided, another question is still being quite actively researched: what is the level, or depth, to which the effects of long term and masked priming extend? When a subliminal masked prime is presented for 50 ms after a forward mask, does the effect of the prime extend only to the perceptual, pre-lexical, level of cognitive processing? Does it influence lexical and semantic processing? Is it possible that the effects of a subliminal masked prime influence abstract conceptual, or even metacognitive, processes?

Some researchers have argued that priming affects only pre-lexical processes, pointing to orthographical and phonological influences on priming strength (Humphreys, Evett, Quinlan, & Besner, 1987; Bodner & Masson, 1997). However, a large body of research provides strong evidence that long term priming also extends into the lexical and semantic levels (e.g., Meyer & Schvaneveldt, 1971; Neely, 1976; Koriat, 1981; Neely, Keefe, & Ross, 1989; Becker et al. 1997; Thompson-Schill & Gabrieli, 1999; Bodner & Masson, 2003). For example, priming is observed even when there can be no pre-lexical contribution at all. Jiang (1999), and Jiang and Forster (2001) have shown cross language priming between Chinese and English in bilingual speakers, using a word in one language as the prime and the translated word in the other language as the target word. The prime and target stimuli have unrelated orthographical and phonological representations in the two languages. Gollan, Forster, and Frost (1997) showed similar findings for Hebrew and English word equivalents.

There is also evidence using long term priming methodology to support conceptual (or complex semantic) priming as a separate phenomenon from perceptual implicit memory (Vaidya, et al., 1997; Mulligan, Guyer, & Beland, 1999). For

example, Bishop and Curran (1998) used scopolamine and lorazepam (both of which impair explicit memory, but only the latter of which impairs implicit memory), to examine perceptual implicit memory (pattern completion and pattern recognition) and conceptual implicit memory (category generation and general knowledge questions). They found that lorazepam impaired perceptual implicit memory, but not conceptual implicit memory.

Taken together, the above studies provide evidence of conceptual priming in a long term priming paradigm. More recently, evidence has begun to mount that even subliminal masked priming may feed into post-perceptual representations, such as at the level of lexical and semantic representations (e.g., Abrams & Greenwald, 2000; Stenberg, Lindgren, Johansson, Olsson, & Rosen, 2000; Damian, 2001). A particularly compelling series of studies by Chris Davis and his colleagues (Davis & Kim, 2000; Davis, Kim, & Sanchez-Casas, 2003; Kim & Davis, 2003) investigated lexical and semantic priming across languages (Spanish-English, and Korean-English) using subliminal masked priming. Robust cross-language priming occurred even for prime-target equivalents that were not cognates between the languages. Conversely, no priming occurred for semantically unrelated homophones.

A very recent development in the field of implicit memory research has been the discovery of “antipriming” by Marsolek and colleagues (Marsolek, Schnyer, Deason, Ritchey, & Verfaellie, 2006; Marsolek, et al., 2007; Deason, 2008). Antipriming may be defined as a significant *reduction* in accuracy, or increase in response time, for stimuli that are *different* from those recently processed (Marsolek et al., 2006). The thesis in these studies is that neural representations of objects are distributed and superimposed,

so that viewing an object strengthens those neural connections that aid its identification, and weaken those connections that aid the representation of other objects that weren't viewed recently, but which share some representational overlap. Thus, when objects are viewed during a priming phase and are viewed again at test, the identification at test is more rapid and accurate due to the strengthened connections. Conversely, objects viewed at test which differ from the primed objects will have weakened connections, and therefore will be identified more slowly or less accurately than had they not been "antiprimed" by the encoded objects. Given the recency of the discovery of antipriming, no published study has examined antipriming in a subliminal masked priming paradigm. However, a recently completed doctoral dissertation has explored this question (Deason, 2008), finding clear behavioral and event related potential (ERP) effects of subliminal masked antipriming.

Thus far, the discussion has focused on implicit memory and priming. However, a great deal of research has examined the closely intertwined phenomenon of implicit learning, both in terms of basic science understanding of such processes, as well as leveraging such abilities to rehabilitate populations with cognitive impairment. Seger (1994) provides a very nice overview of implicit learning research over the 1970s and 1980s, from a cognitive neuropsychological perspective. She defines implicit learning in terms of four general criteria; 1) individuals cannot provide a complete, declarative, account for what they have learned, 2) information must be learned at a more complex level than simple association, 3) implicit learning does not involve conscious hypothesis testing, and 4) it is preserved in individuals with amnesia. Seger draws several distinctions between implicit learning and implicit memory. Whereas

implicit memory involves information produced or processed verbatim, implicit learning involves acquiring a new pattern or rule. Whereas stimuli in implicit memory tasks are usually verbal, those of implicit learning tasks are usually visual or visuo-spatial. Whereas awareness is not required for implicit memory, it is possibly required for implicit learning. Finally, whereas attention is minimally necessary for implicit memory, it is important for implicit learning. Tasks which Seger identifies as implicit learning include learning of artificial grammars, puzzle learning, motor learning, contingent response tasks, and serial reaction time tasks.

Turning to more clinically relevant implicit learning tasks, Schacter, Rich, and Stamp (1985) describe remediation of memory dysfunction using spaced retrieval therapy. Spaced retrieval refers to the retrieval of information after longer and longer time intervals. Schacter, Rich, and Stamp presented pictures of faces to four participants with memory dysfunction. They were later given recall tests, during which characteristics associated with the faces were evaluated. Training on spaced retrieval was provided in several ways. All participants demonstrated significant improvement to recall. Spaced retrieval has been used in other clinical populations with memory disorders. Hopper, Mahendra, Kim, Azuma, Bayles, et al. (2005) reviewed 15 studies which provided class II and class III scientific evidence for the use of spaced retrieval therapy for individuals with dementia. Across the studies reviewed, implicit learning tasks included cue-behavior association (e.g.: using verbal cues to stimulate use of an external memory aid), or face/object-name associations (e.g.: associating objects and their names with where these objects are likely to be located). The studies provided

evidence for information acquisition, retention of skills from several days to several months, and generalization of skills to specific contexts or situations.

Spaced retrieval is one of several implicit learning approaches. Kessels and de Haan (2003) conducted a meta-analysis of evidence for the rehabilitative value of the method of vanishing cues and errorless learning, compared against a control intervention, for individuals with amnesia. The method of vanishing cues involves progressively eliminating cues during successive learning trials, while maintaining a high level of recall. Thus, for example, a list of new words may be learned, and then during recall testing the individual may be presented with a definition of one of the words recently learned as well as the first several letters of that target word. Over successive recall trials, the number of letters presented as a cue are reduced, until the participant can recall the target word on the basis of the definition alone, with no cue letters. Errorless learning may employ the vanishing of cues, however the emphasis is on eliciting the correct response from the participant in every trial, while successively removing cues or other assistance. Errors are prevented by providing the correct answer immediately after the question is asked. With successive trials, cues are still presented in order to avoid errorful responses. Across the 11 studies that were included in the Kessels and de Haan review, a large and statistically significant effect size was found for errorless learning, but not for the method of vanishing cues.

Errorless learning and spaced retrieval can be used in conjunction, so that cues continue to be presented at the spaced retrieval attempts, to ensure errorless recall. Bourgeois, Lenius, Turkstra, and Camp (2007) evaluated this kind of errorless spaced retrieval therapy in comparison to a control treatment (“didactic strategy instruction”) in

an over-the-telephone intervention paradigm for individuals in the chronic phase of recovery from traumatic brain injury. Results of their randomized, controlled, clinical trial indicated that there was a significant increase in treatment goal attainment and strategy use for spaced retrieval therapy, both immediately after training and at one-month post treatment, but not for the didactic strategy instruction.

The importance of evidence for implicit learning in the TBI population is underscored by Schmitter-Edgcombe (2006), who points out that even “persons with severe TBI can acquire and use automatic processes in complex, cognitive task performance” and that these skills are retained as long as controls” (p. 136). She further states, “once a skill is automatized, participants with TBI appear as adept as controls at retaining the information over long retention intervals without practice” (p. 136). In terms of the present research, the literature on implicit learning motivates the hypothesis that individuals with TBI should be able to demonstrate priming (and antipriming) effects similar to controls.

At this point, some key aspects of the above review of implicit memory, priming, and implicit learning bear summarizing, as they are relevant to the present research. Firstly, implicit memory refers to the influence of prior stimulus exposure on subsequent processing of that or a similar stimulus. Secondly, priming has historically been the main way by which implicit memory has been studied, with antipriming being a recent discovery in the field. Thirdly, there is robust evidence that masking and very briefly presenting a priming stimulus (e.g.: < 60 ms or so) renders the prime undetectable by conscious awareness. Fourthly, there is strong evidence that the long term priming paradigm can prime high level conceptual mental representations, and

early emerging evidence that subliminal masked presentation of stimuli may prime mental representations beyond the perceptual level, into the semantic domain and possibly beyond. Finally, there is a great deal of research that shows that such implicit manipulations as spaced retrieval, errorless learning, etc., do affect learning among individuals with brain injury. Given that these manipulations are supraliminal and occur over long time intervals, they may share some similar properties with long term priming. However, unsurprisingly, none of the implicit learning techniques used by rehabilitation professionals employs any kind of subliminal masked priming methodology.

These key aspects of the literature lead to the question as to whether or not masked priming can influence the highest levels of cognition, namely metacognition.

METAMEMORY

The study of metacognition has been a rapidly developing field of research over the last several decades. Metacognition has been described as “cognition about one’s own cognition” (Nelson, 1992; p. 1). Flavell (1979) provided an early definition, proposing four classes of metacognition: metacognitive knowledge, experiences, goals, and strategies. In his taxonomy, these respectively comprised knowledge and beliefs about one’s and others’ cognitive processes, specific experiences in which metacognitive knowledge or beliefs are generated, goals of metacognitive activity, and strategies to achieve those goals. This can be applied to all aspects of cognitive

processing, so that one can discuss meta-language, meta-attention, meta-memory, etc. in these terms. For our purposes, we will focus on metamemory.

Johnson-Laird (1983) offered a computational analysis as an early attempt to place constraints on models of consciousness. He proposed four fundamental principles that an adequate model must incorporate: 1) there must be a distinction between conscious and unconscious processes, 2) while some cognitive processes can be intentionally controlled, others cannot, 3) the model must have self-awareness, and 4) the model must be able to exhibit intentionality. He suggests that to achieve these, a complete theory of consciousness must incorporate hierarchical parallel processing, recursively embedded models, and a high-level model of itself.

Nelson and Narens (1990, 1994) revolutionized the field of metacognition research by proposing a unifying framework for metacognitive theory and measurement. Prior to their seminal work, metacognition research proceeded in a fragmented and isolated fashion (Dunlosky & Metcalfe, 2009). Their model describes the flow of information between a so-called “meta-level,” and an “object-level” (wherein the meta-level contains a model of the object level). Information received by the meta-level from the object level reflects self-monitoring of memory, while information flowing from the meta-level to the object level represents self-control processes (see Figure 1). Apropos Johnson-Laird’s (1983) computational analysis, Nelson and Narens (1990) generally satisfy all three constraints; there is hierarchical parallel processing in that the object-level is subordinate to the meta-level and both function in parallel, the meta-level contains a high-level model of the object-level

(though not of itself), and there are recursively embedded models, although with only two levels this is not particularly sophisticated.

Nelson and Narens (1994) further specify their model in terms of the main processes associated with memory, namely: acquisition, retention, and retrieval of information (Figure 2). They associate monitoring during acquisition of information with such metamemory judgments as Ease-Of-Learning (EOL), Feeling of Knowing (FOK), and Judgments of Learning (JOLs). EOL-s are predictions of the ease with which information is to be learned, made during or prior to acquisition.

FOKs are judgments of whether or not information that one failed to recall is likely to be known at all, and if known then how likely it will later be recalled or recognized; these judgments can be made during acquisition or retrieval. JOLs are predictions of how likely one is to recall information at a later time, made either during acquisition or shortly thereafter. The fourth type of metamemory monitoring that Nelson and Narens include in their model is the confidence judgment, also called Retrospective Confidence Judgment (RCJ). RCJs are ratings of how confident one is about a response one has given during recall. Thus, Nelson and Narens' model organizes theory and measurement into a coherent overall paradigm.

Whereas Nelson and Narens (1990, 1994) approach metacognition from the perspective of cognitive psychology, others have attempted to integrate neuroscientific evidence into their models. In this regard, Stuss (1991) locates metacognition within a broader model of frontal lobe functioning, and in the process provides a model that better satisfies the constraints indicated by Johnson-Laird (1983). Stuss posits three

hierarchical levels of function for the frontal lobes, with recursive modules providing feedback and feedforward loops to the adjacent frontal modules (see Figure 3).

Monitoring and control of information occur within each level. The lowest, “sensory-perceptual,” module maintains and organizes information from the posterior, perceptual, regions of the brain, and initiates and drives behavior. Stuss locates this function to the posterior dorsolateral, and medial prefrontal, regions. This module not only interacts with the posterior brain regions, it also feeds information forward to the next, higher, level of frontal function – executive function. The executive module in turn judges and organizes information from the sensory-perceptual level, providing conscious direction (intentionality) to the sensory-perceptual module for the achievement of goals in non-routine contexts. The executive also sends information up to the next, and highest, level. Stuss locates executive function to the prefrontal cortex. The highest level of frontal function involves self-awareness and self-reflectiveness and is mediated by the prefrontal cortex as well. The output of this metacognitive module feeds back down to the executive module. Thus, the self-monitoring and self-control, which together constitute metacognition as defined by Nelson and Narens, would involve aspects of the second and third of Stuss’ three levels of frontal lobe function. Note that Stuss’ (1991) model satisfies Johnson-Laird’s (1983) requirements of hierarchical parallel processing, recursively embedded models, and that the metacognitive model must contain a high-level model of itself.

Interestingly, 14 years later, Busch McBride, Curtiss, and Vanderploeg (2005) performed a principle components factor analysis on a corpus of neuropsychological

data from 104 individuals with a history of TBI, and generated three principle factors that bear some similarity to Stuss' (1991) model. One factor determined by Busch et al. (2005) involves mental control of ongoing working memory. This may be analogous to Stuss' "sensation and basic knowledge" level; he describes the function of that level as to "maintain and organize information in meaningful sequences," which would certainly involve control of ongoing working memory. The second factor Busch et al. discovered was of self-generative behavior and cognitive flexibility/set shifting. This is similar to the second level of Stuss' model, executive function, which he describes as involving conscious direction of posterior cortical regions towards a goal in novel situations (i.e.: cognitive flexibility in self-generative goal directed behavior). Finally, Busch et al.'s third factor involved "failure to inhibit reporting of inaccurate information." Stuss' highest level of cognitive function involves awareness of oneself and one's relation to the environment. Certainly, "failure to inhibit reporting inaccurate information" would constitute a lack of self-awareness. Busch et al.'s (2005) sophisticated analysis is a powerful endorsement of Stuss' earlier ideas.

The brief summary of metacognition research above does not fully capture the rich body of literature on metacognitive theory. Furthermore, developing a comprehensive model of metacognition, or even of metamemory, based on the extant literature is a task that is well beyond the scope and purpose of the present research. However, the above models of metacognition are sufficient to illustrate certain commonalities to the various theories of metacognition, and it is within the context of these common features that the present research is conducted. Firstly, all of the theories recognize that models of metacognition must include both self-monitoring and self-

regulation; the present research will constrain its focus to the self-monitoring of memory. Secondly, cognition involves both unconscious and conscious (read implicit and explicit) processes; although I will not advance a new model for metacognition, in the section below titled “Is Metamemory Implicit?” I will suggest a theoretical rationale for a relationship between implicit and explicit processes in metamemory. Thirdly, and finally, the brain regions which give rise to metacognition primarily involve the frontal lobes. Although this issue will be revisited in more detail below in the following section on the neural substrates of implicit memory and metamemory, the number of brain injured participants in the present study does not permit analysis of the data in terms of frontal vs. posterior locus of injury, and so deeper examination of this last issue is also outside the scope of the present research.

NEURAL SUBSTRATES OF IMPLICIT MEMORY AND METAMEMORY

To this point, the discussion has covered theories of implicit memory and metamemory. A very brief overview is in order regarding the neural substrates of these processes, and to some extent, of executive function (due to its close relationship to metacognition), as this provides evidence to help shape and constrain theories on their relationships. We will begin with the neural substrates of implicit memory, particularly in terms of priming, in the neurologically normal population, and then examine studies of individuals with TBI and what light this sheds on implicit memory. We will then

repeat this approach for metamemory, beginning with non-clinical studies and then those including brain injured participants.

Implicit Memory and Priming

Early evidence from functional brain imaging and lesion studies tended to support the idea that perceptual priming mainly reflected posterior perceptual processes (Tulving & Schacter, 1990; Squire et al., 1992; Schacter, Church, & Treadwell, 1994). In a comprehensive review article of imaging and lesion studies through the 1980-s and 1990-s, Schacter (1997) argued that priming is a posterior phenomenon which does not involve the hippocampus or medial temporal lobes, or require the frontal lobes. However, he argued that conscious retrieval attempts and other forms of explicit memory do involve the frontal lobes, the medial temporal lobe and hippocampal formation, and may also include posterior brain regions. He claimed that frontal and prefrontal activation during tests of implicit memory (e.g.: word stem completion) reflected effortful attempts at recall (left hemisphere) or attempts to recreate contextual cues to guide explicit episodic retrieval attempts (right hemisphere).

Since the late 1990-s, however, a great deal of research has uncovered significant frontal lobe involvement in priming which cannot simply be attributed to effortful recall attempts. Gabrieli's (1998) literature review, for example, provides greater detail than Schacter (1997), and examines evidence for possible frontal lobe involvement in priming. He argues that while the perceptual priming reviewed by Schacter occurs in modality-specific primary (posterior) neocortical areas, conceptual priming occurs in association neocortical areas, including frontal, temporal and parietal

areas. So, for example, repetition priming with visual objects, or written words, would be mediated by visual cortex. Similarly, repetition priming with auditory stimuli would be mediated by auditory cortex. However, conceptual priming involves association cortices in the left frontal areas.

To this point, Wagner, Maril, and Schacter (2000) examined fMRI patterns of activation in the left inferior prefrontal cortex (LIPC) during a semantic decision task (abstract/concrete decisions) for words that had either been previously encoded semantically (abstract/concrete decisions) or perceptually (uppercase/lowercase decisions). Compared against reference activation patterns for novel words, the anterior portion of the LIPC showed lower fMRI signals during the semantic decision task for the semantically encoded words only. The posterior LIPC, however, showed lower signals in the semantic decision task for both items that had previously been semantically and perceptually processed, but with a significantly lower magnitude signal (i.e. : greater priming effect) for semantically encoded words. Thus, the left inferior prefrontal cortex appears to show evidence of neural perceptual and semantic priming, with topographic differentiation of these types of priming. Indeed as Schacter, Wig, and Stevens (2007) state in their review of the imaging literature on priming, “correlations between behavioral priming and activity reductions are robust across a range of tasks and procedures in prefrontal regions but not in posterior regions” (p.171).

However, as is oft stated, correlation does not prove causation. Stronger evidence is needed to attribute causation of conceptual priming to left frontal areas. In this regard, a recent study provides strong, direct support for the causal role of left frontal areas in conceptual priming. Wig, Grafton, Demos, and Kelley (2005) mapped

cortical activation during a semantic classification (e.g.: living/non-living) task with picture stimuli, using fMRI. They administered transcranial magnetic stimulation (TMS) to the left inferior frontal gyrus (L-IFG) during a subsequent session, while participants performed the same task on different items. Fifteen minutes after that second session, participants were again scanned with event-related fMRI while performing the task a third time, with both primed and non-primed images. TMS disrupted the neural priming (i.e.: eliminated the typical decrease in neural activation for repeated items) in the L-IFG for primed items, but did not disrupt neural priming in the middle occipital gyrus (Brodmann's areas 18 and 19) which was used as a control site. The behavioral data for this third session reflected TMS disruption of priming; those tokens encoded while TMS was delivered in the second session did not have improved response times, as compared to non-primed items in the third session. Wig et al. (2005), thus, demonstrate that selective disruption of L-IFG eliminates behavioral and neural priming effects. More to the point, it provides strong evidence that left frontal areas play a causal role in the mechanism by which the conceptual prime is neurally encoded and behavioral priming is generated.

We see from the above studies evidence for both anterior and posterior involvement of brain areas in priming, with the posterior areas primarily involved with lower level (e.g.: perceptual) priming, while anterior areas are implicated in more abstract forms of priming, such as conceptual priming. The question then arises as to how diffuse and focal injury to these areas of the brain affects priming. To this point, Haut, Petros, Frank, and Haut (1991) conducted a semantic categorization task, in which control and participants with TBI decided whether, given a category name, the

subsequent word was an exemplar of the category or not. By varying stimulus onset asynchrony (SOA) between presentation of the category name and potential exemplar they were able to produce three levels of priming (SOA of 0, 400, and 800 ms); they then measured response times. Although the TBI group demonstrated significantly longer response times overall, they did show the same degree of priming as the control group.

Vakil, Jaffe, Eluze, Groswasser and Aberbuch (1996) examined whether long term repetition priming was affected by TBI in a list learning task. In a task that is reminiscent of the California Verbal Learning Test (Delis, Kramer, Kaplan, & Ober, 2000), TBI survivors and healthy controls were presented a list of 15 typed words on a sheet and asked to read them aloud as quickly as possible, and then to recall as many words from the list as possible. This procedure was then repeated with the same list four more times in rapid succession. It was repeated a sixth time after a 20 minute delay, and then a final time after a one hour delay. Reading speed and number of items recalled were measured. The authors found that the TBI survivor group recalled significantly fewer words than controls, and learned these with a slower rate of acquisition. However, although the raw scores of reading speed were slower in the TBI group, using reading speed index scores (ratio of reading time difference to reading time sum between successive readings), the authors found that the overall pattern of priming benefit (decreased reading speed from one list-reading trial to the next) was the same for both groups.

While the above studies explored priming in individuals with TBI, Marsolek et al. (2006) used visual object identification to measure both priming and antipriming in

individuals with amnesia from bilateral medial temporal lobe damage (due to anoxia, encephalitis, or stroke). They found that participants with medial temporal lobe damage did show significant priming and antipriming effects, although the priming effect was not as strong as for non-brain injured individuals ($p < .01$). Those with medial temporal lobe injury also showed overall slower reaction times ($p < .05$). In a follow up study, Marsolek et al. (2009) provided imaging evidence in neurologically normal individuals that neural activity associated with priming and antipriming in the visual object identification task occurred in posterior neocortical regions, particularly along the ventral visual stream; they did not examine frontal cortical regions. While Marsolek et al. (2006) studied individuals with medial temporal lobe injury, to date no one has examined whether antipriming is measurable in individuals with TBI. Given the prevalence of frontal lobe and diffuse axonal injuries, with attendant impairments to explicit memory and metamemory, further exploration of priming and antipriming in this population may shed light on frontal lobe and white matter involvement in visual perceptual priming and antipriming.

With findings that depart from the above studies, Eskes, Szostak and Stuss (2003), examined a range of tasks which might be considered perceptual/constructional (picture fragment completion), lexical (word-fragment completion), and semantic (category exemplar generation), in individuals with focal lesions: either in the left or right dorsolateral prefrontal cortex (DLPFC), or with medial frontal lesions. Cued recall was used to test explicit memory. Interestingly, individuals with L-DLPFC lesions showed impaired word fragment completion. However, priming was not affected by lesions in other locations. Explicit memory was impaired for individuals

with L-DLPFC lesions as well as individuals with medial lesions. The finding of significant implicit memory impairment in individuals with L-DLPFC lesions is unique.

Whereas a vast body of research has shown that injury to medial temporal lobe structures can result in profound explicit memory impairment (see for just a few examples, Scoville & Milner, 1957; Zola-Morgan, Squire, & Amaral, 1986; Parkin & Leng, 1993), with little exception, the literature generally shows a pattern of intact, or only mildly impaired, priming in individuals with TBI. Nevertheless, there are still some areas of research that require additional exploration. Surprisingly, Medline and PsycINFO searches of the research literature reveal that while there is a large body of research on subliminal masked priming in individuals with injury primarily or solely to medial lobe structures, to date no study appears to have examined subliminal masked priming in individuals with TBI.

As for antipriming, as it has only recently been discovered, it is unsurprising that no study has yet questioned whether it can be observed in individuals with TBI. However, this is particularly important because Marsolek et al. (2006) contend that visual antipriming is the result of accumulated synaptic modifications, over many exposures to different but overlapping visual object representations in posterior neocortex. Because TBI produces pervasive dorsal and ventral frontal lesions, as well as frequently causing diffuse axonal injury (e.g.: Meythaler, Peduzzi, Eleftheriou, & Novack, 2001), antipriming theory would suggest that since synaptic connections in the posterior ventral stream are likely to be relatively spared in the TBI population, antipriming in the visual object identification task should still be observable. Findings to the contrary may present some challenges to the theory.

Metamemory

We turn now to a summary of the neural substrates of metamemory. As with the neural bases for priming, since there is a large body of research in this area, this too will simply be a brief outline of the literature. In their review of the research in cognitive neuroscience and cognitive psychology, Fernandez-Duque, Baird, and Posner (2000) point out the overlap between metacognition (particularly metacognitive control) and executive function; indeed they repeatedly use the phrase “metacognitive/executive control”. They then constrain their review to what they believe are some of the more elementary functions of which metacognitive/executive control are comprised, namely: conflict resolution, inhibitory control, error detection, and emotion regulation.

Fernandez-Duque et al. (2000) conclude that the anterior cingulate cortex (ACC) is the frontal region involved in all of these functions, although for regulatory aspects of these functions, the dorsolateral prefrontal cortex (DLPFC) is also implicated. Commenting on Fernandez-Duque et al. (2000), Shimamura (2000) argues that metacognitive and executive function essentially are comprised of four primary functions: selecting (selective attention), maintaining information (in short term memory), updating (which requires monitoring information and updating it as needed), and rerouting (e.g.: set/task shifting). He assigns the first function (selective attention) to the anterior cingulate cortex, but the rest to the prefrontal cortex.

With regard to the dorsolateral prefrontal cortex, Stuss and Levine (2002) attribute to it such executive and metacognitive functions as spatial and conceptual reasoning, memory monitoring (particularly in the right DLPFC), and higher level

language (again in the right DLPFC) such as elaboration of details, topic maintenance, and inhibiting expression of irrelevant details.

In their discussion of frontal lobe function, the papers cited above have generally treated metacognition and executive function together since the relationships and distinctions between the two are still very much unclear, and there is some difference of opinion on the underlying processes and brain structures which mediate the processes. As Shimamura (2000) points out, “both metacognitive control and executive control share the primary feature of enabling top-down modulation of cognitive processes....the relationship between metacognition and executive control has not been fully appreciated” (p. 315).

Recently, however, some interesting imaging studies have begun to explore what would generally be considered to be purely metacognitive functions in neurologically normal individuals. In a study by Johnson et al. (2002), participants made yes/no responses to questions requiring self-reflection (e.g.: “I have a quick temper”), as compared to yes/no control questions (“ten seconds is more than a minute”). On analysis of the fMRI BOLD subtraction signal, anterior medial prefrontal and posterior cingulate areas were consistently active across all participants. As with the neuropsychological literature, medial prefrontal and cingulate regions were again implicated.

While the types of questions asked of participants by Johnson et al. (2002) are certainly metacognitive in nature, they are not particularly similar to the type of metacognitive judgments common to metamemory research. In contrast, Kao, Davis, and Gabrieli (2005) conducted an fMRI study to examine JOLs in neurologically

normal individuals. Participants viewed 350 photographs of scenes (e.g.: an outdoor vista, a bedroom, etc.), and made binary “will remember” or “will forget” JOLs as to whether they would remember the scene for a recognition test following the scan. At test, they made old/new judgments of 700 pictures, half of which were presented during the scan, and also made binary high/low confidence ratings about their old/new judgments. The authors found that, as expected, medial temporal lobe activation was strongly correlated with successful encoding of scenes. “Will remember” JOLs were strongly correlated with activation in a bilateral network of dorsal and ventral regions of medial prefrontal cortex. Furthermore, greater activity was observed in ventro-medial prefrontal cortex (VMPFC) for participants with particularly high predictive accuracy; that is, individual differences among participants in relative predictive accuracy were correlated with respective VMPFC activity.

Thus, throughout the various studies that have attempted to assign brain regions responsible for metacognition and executive function, the dorsal and ventral medial prefrontal cortices appear to play a significant and consistent role. Further research is needed to unpack these large regions into a more fine-grained understanding of component functions and interactions.

Nevertheless, given the consistent findings that medial prefrontal cortex is implicated in metacognitive function, the question arises as to metacognitive and metamemory dysfunction when these regions are injured. As Courville (1937) has shown, abrasions are common to the ventral surface of the frontal lobe and anterior surface of the temporal lobe during TBI. It is therefore to be expected that encoding and retrieval of memory, mediated by temporal lobe structures, are particularly

vulnerable. Yet, although the relatively insulated placement of the medial prefrontal cortex protects it to some degree from the aforementioned direct abrasion against bone, it does contact the falx cerebri and portions of the ethmoid bone. Furthermore, the medial prefrontal regions have pervasive white matter connections to the ventral fronto-limbic system (Malykhin, Concha, Seres, Beaulieu, & Coupland, 2008). Kennedy et al. (2008) have shown robust evidence of decreased white matter integrity in the frontal lobes, rostral to the genu of the corpus callosum (as well as in the in the centrum semiovale) in survivors of severe traumatic brain injury. Given these findings, metacognitive deficit secondary to TBI is also to be expected.

In an fMRI study similar to Johnson et al. (2002), Schmitz, Rowley, Kawahara, and Johnson (2006) examined the accuracy of self-reflective statements in individuals with TBI and matched controls. Participants made yes/no judgments in terms of whether or not they thought adjectives (e.g.: “daring”, “shy”, etc.) applied to them. On analysis of the fMRI BOLD subtraction signal, both participant groups demonstrated activation of dorsal and ventral regions of the medial prefrontal cortex, as well as retrosplenial cortices. Additionally, the TBI survivor group demonstrated increased activation, as compared with the control group, in the right temporal pole, anterior cingulate gyrus, and several sub-regions of the retrosplenial cortex. Further analysis of the TBI data revealed that, across participants, greater activation in the right DLPFC correlated with greater accuracy of self appraisal of post injury abilities as measured by the Patient Competency Rating Scale. These findings not only echo the involvement of, particularly, the medial prefrontal cortex in metacognition, but the converse – that damage to these areas results in decreased metacognitive skills such as self-appraisal.

Apart from imaging research, several neuropsychological studies have also examined deficits to metamemory as a result of brain injury. Janowsky, Shimamura, and Squire (1989) compared both memory and metamemory performance in individuals with and without brain injury. The brain injured groups included individuals with frontal lobe lesions as well as individuals with amnesia from temporal lobe lesions due to a variety of causes, but generally from Korsakoff's syndrome. Participants studied sentences and after a five-minute delay were presented the sentences again, but with one word missing. They were asked to recall the missing word from each sentence. FOKs were solicited for non-recalled items, followed by a seven-alternative forced choice recognition test. Participants with amnesia demonstrated significantly decreased recall and recognition compared against controls or individuals with frontal lobe lesions, with no difference between the latter two groups. Interestingly, the frontal lobe lesion and control groups both demonstrated similar accuracy of FOKs made five minutes after the recognition test (as measured by gamma correlation of FOKs with recognition accuracy). However, when retested one to three days later, gamma correlation for the frontal lobe group dropped to chance levels, whereas they remained at nearly the same level for controls. Individuals with amnesia demonstrated near chance levels of gamma correlation at five minutes after the recognition test, and they were not retested days later.

In order to examine potential metamemory differences between individuals with documented frontal lobe injury vs. those with diffuse brain injury, Kennedy and Yorkston (2004) employed the Nelson and Dunlosky (1991) paired associate learning task (to be described in greater detail in the Measures of Metamemory section below),

with immediate and delayed JOLs during the study phase, followed by a cued recall test. They found that individuals with frontal lobe injury had significantly greater recall than those with diffuse injury, but they had both decreased confidence in their predictions as well as decreased predictive accuracy compared with the diffuse brain injury group. These findings confirm the privileged role of the frontal lobes in such metacognitive phenomena as metamemory judgments and confidence in one's abilities.

Finally, Pinon, Allaina, Kefia, Dubasa, and Le Galla (2005) conducted a similar experiment, comparing individuals with frontal lobe injury with neurologically normal controls, but also including the FOK judgment, in addition to JOLs. The FOKs were solicited during recall testing, on non-recalled items. The JOLs were obtained immediately after study, with no delay. The authors found that the individuals with brain injury had lower recall than controls. Although relative predictive accuracy of JOLs (as measured by Goodman-Kruskal gamma correlation between JOLs and recall accuracy) was higher for controls, this difference did not reach statistical significance ($p = 0.14$); this may be due to the fact that JOLs were solicited immediately after study, and such JOLs are poorer predictors of recall than delayed JOLs (Nelson & Dunlosky, 1991) for both brain injured and neurologically normal individuals (Kennedy & Yorkston, 2000). Finally, brain injured participants had significantly poorer FOK accuracy than controls.

Taken together the above studies from this section show several important findings. Firstly, whereas priming was historically thought to reflect posterior processes, more recent research has revealed frontal lobe involvement in priming,

particularly when it involves post-perceptual priming, such as conceptual priming. Secondly, the frontal lobes are strongly implicated in metacognitive processes. Thirdly, while medial temporal lobe injury tends to result in explicit memory deficit, implicit memory tends to remain relatively intact in such injury. This minimal to mild deficit to implicit memory after brain injury, even in the context of profound explicit memory impairment, also seems to be the case for diffuse and frontal injury, not just injury to medial temporal lobe. Finally, frontal lobe injury differentially impairs metacognitive self-monitoring.

These findings lead to important research questions. If the frontal lobes are involved in conceptual priming, and if frontal lobe injury results in metacognitive deficits but not implicit memory deficits, is it possible that priming can leverage the relatively intact implicit memory skills of the frontal lobes to improve metacognitive self-monitoring? This chain of reasoning is contingent on several important issues which bear further examination. Firstly, although there is evidence that priming can influence such higher cognitive functions as complex semantic processing, general knowledge, and abstract/concrete judgments (Vaidya, et al., 1997; Mulligan, Guyer, & Beland, 1999; Bishop & Curran, 1998), few studies have attempted to measure whether or not *metacognitive* processes can be influenced by subliminal masked priming rather than supraliminal long term priming (Nelson, Gerler, & Narens, 1984; Jameson, Narens, Goldfarb, & Nelson, 1990); and none of these studies has examined whether prospective memory judgments (e.g.: JOLs) can be subliminally primed. Thirdly, will any such findings still obtain for individuals with TBI, which frequently results in frontal lobe damage? Finally, will TBI survivors demonstrate visual antipriming, given

the relative sparing of posterior occipito-temporal neocortex, compared to the prevalence of frontal and diffuse axonal injury in that clinical population? These and other questions will be explored in the present research.

MEASURES OF METAMEMORY

As mentioned previously, Nelson and Narens (1990) described four different measures of metamemory: Ease Of Learning (EOL) judgments, Judgments Of Learning (JOL-s), Feeling Of Knowing (FOK) judgments, and Confidence Judgments (CJ-s). While these are the most common measures of metamemory, some have also described Feeling of Familiarity (Kinoshita, 2002), Feeling Of Warmth (Metcalf, 1986), and Source Monitoring Judgments (Dunlosky & Metcalfe, 2009). Whereas FOKs and JOLs are by far the most widely used measures of metamemory, this review will constrain its focus to these two, and build a rationale for the use of the latter in studying the performance of individuals with TBI.

Hart (1965) was the first to define and begin systematically studying FOK judgments, building on related research by others into the “tip-of-the-tongue” phenomenon (James, 1950; Woodworth & Schlosberg, 1964). Hart describes this effect by writing, “a person will feel that an elusive memory is close, very close – right on the tip of his tongue” (p. 208). Hart operationalized the definition of FOK by asking participants the question, “Even though I don't remember the answer now, do I know

the answer to the extent that I could pick the correct answer from among several wrong answers?” Binary yes/no responses were solicited.

Researchers have varied in how they operationally defined FOK judgments. Nelson, Narens, and Gerler (1984) defined FOKs in terms of the rank order of “most likely to recognize” to “least likely to recognize”, out of each set of three consecutive questions whose answers the participant failed to provide. Koriat (1993) used a 0 – 100 scale of the chance the participant felt they had of identifying the target in a recognition test, and later (Koriat, 1995) modified this to a percent likely to recognize from a field of four choices (so that participants were asked to provide an FOK of 25%, 50%, 75%, or 100%). All of these variations are widely used in FOK research.

As with the FOK judgment, the Judgment of Learning (JOL) is a measure of metamemory monitoring. However, whereas FOKs purportedly evaluate how well one knows or might be able to recall information previously learned, JOLs attempt to evaluate how well information currently being (or soon to be) learned is likely to be recalled at a later time; this is typically done using Likert scale ratings of the likelihood of future recall. In this regard, FOKs constitute a retrospective metamemory judgment, while JOLs are prospective in nature. Another difference is that FOK judgments are traditionally made on items which the individual has failed to recall (since items one *can* recall would be given a 100% FOK judgment, and are therefore not particularly informative of the degree to which one feels he or she knows something). Conversely, since JOLs are prospective, they reflect metamemory processes operating on all items, whether these items later result in failed or successful recall.

Arbuckle and Cuddy (1969) were the first researchers to empirically measure prospective metamemory judgments, in a paired associate learning task. They performed two paired associate learning experiments, with simple binary yes/no prospective metamemory judgments in the first experiment, and a 60 mm analog scale anchored at “very likely” (to recall) at the left, and “very unlikely” at the right for the second experiment. All judgments were made immediately on viewing each word-pair; there were no delayed judgments. Data from both experiments demonstrated better than chance relationship between recall accuracy and JOL.

Although Arbuckle and Cuddy (1969) demonstrated better than chance predictive accuracy of metamemory judgments, much other early research showed that JOLs were notoriously inaccurate (e.g.: Vesonder & Voss, 1985), with Goodman-Kruskal gamma correlations ranging from +0.09 to + 0.48 (where $\gamma = 0.50$ represents pure chance and $\gamma = 1.0$ represents perfect predictive accuracy). Against this background, Nelson and Dunlosky (1991) discovered circumstances in which JOL predictive accuracy could be dramatically improved: if they were made after a delay following the encoding or learning of the items. This is now called the “delayed JOL effect.” Although JOLs made immediately after learning do not well correlate with actual subsequent recall performance, delayed JOLs are highly accurate predictors of recall, with gamma values averaging +0.90 (Nelson & Dunlosky, 1991). A more detailed description of the experiment by Nelson and Dunlosky will be provided in the methods section below, as the present research attempts to follow their procedure, but modified with the introduction of subliminal masked priming and antipriming of target items.

Nelson and Dunlosky (1991) hypothesized that the metamemory system monitors information from both short term memory (STM) and long term memory (LTM), and that STM provides very noisy and dominant input immediately after study. According to this “Monitoring Dual Memories” hypothesis, once at least 30 seconds or so have passed and STM interference has attenuated, the metamemory process can then rely primarily on LTM. Since LTM is the same source to be later accessed during recall, it is hypothesized to provide better input to the metamemory system. This explanation for the delayed JOL effect has been challenged, and other explanations advanced (Spellman & Bjork, 1992; Kimball & Metcalfe, 2003), with Nelson and colleagues responding and further illuminating the nature of delayed JOLs (Nelson & Dunlosky, 1992; Dunlosky & Nelson, 1997; Nelson, Narens, & Dunlosky, 2004; Overschelde & Nelson, 2006). However, since the delayed JOL effect is well documented and robustly replicated, it is left to the interested reader to see the above, and other, studies (e.g.: Schwartz, 1994) for details of the various attempts to explain its underlying cause. For the purposes of the present research, details of the various hypotheses for the mechanism of the delayed JOL effect are not relevant; rather, the conditions under which the delayed JOL effect obtains, and the magnitude of the effect, are most germane. Again, the effect occurs when JOLs are delayed by at least 30 seconds after learning (i.e.: long enough to clear short term memory), and the gamma value reported by Nelson and Dunlosky (1991) increases from approximately +0.33 in the immediate JOL timing condition to approximately +0.90 in the delayed JOL timing condition.

Before proceeding with a review of the JOLs in the TBI population, it is worth noting that several studies have compared FOKs and JOLs (Leonesio & Nelson, 1990; Schraw, 1995; Maki, 1999; Souchay, Isingrini, Clarys, Tacconnat, & Eustache, 2004). Two consistent findings emerge from this literature: firstly, FOKs and JOLs are approximately equally good predictors of recall accuracy, and secondly, they are not well correlated with each other. The latter finding implies that FOKs and JOLs reflect different underlying metacognitive mechanisms. This being the case, and again given that the present research is concerned with examining the prospective metamemory of individuals with TBI, the focus of the present research will be on JOLs.

JUDGMENTS OF LEARNING IN THE TBI POPULATION

Several studies have examined JOLs in the TBI population, primarily by Kennedy and colleagues (Kennedy, Yorkston, & Rogers 1995; Kennedy & Yorkston, 2000; Kennedy, Carney, & Peters, 2003; Kennedy & Nawrocki, 2003; Kennedy, 2004). Kennedy, Yorkston, and Rogers (1995) was the first study to compare online self monitoring of memory between individuals with and without brain injury. Following the Nelson and Dunlosky (1991) paradigm, they confirmed the delayed JOL effect in controls, but not for the TBI survivors. This may have been due to the very small sample size (two participants per group) and the variability of performance in individuals with TBI. Kennedy and Yorkston (2000) expanded the study and modified the Nelson and Dunlosky protocol to assess the effect of making overt retrieval attempts

immediately prior to making a JOL. Both groups demonstrated the delayed JOL effect. Importantly, in the delayed JOL timing condition TBI survivors had predictive accuracy as high as controls. However, TBI survivors tended to overestimate their recall (and controls underestimate their recall), when they made predictions immediately after studying (i.e.: in the immediate condition). Verbal retrieval attempts neither improved nor worsened recall or predictive accuracy for either group.

In order to examine relationships between predictions of recall and study strategy decisions, Kennedy, Carney, and Peters (2003) allowed participants to select items to restudy prior to the recall test. They were later presented items to restudy either based on their own prior selection or computer-selected; items selected by the computer were items which participants rated as unlikely to recall. The TBI survivors demonstrated better recall for those items they selected to restudy after making a delayed JOL, as compared to their immediate JOL items or items selected by the computer. The authors concluded that individuals with TBI should make study strategy decisions based on delayed predictions rather than immediately after the event, and that they should choose to restudy those items they think they will have trouble recalling.

In order to examine predictive accuracy in narrative recall, Kennedy and Nawrocki (2003) presented 15 brain injured participants and 15 neurologically normal matched controls with narratives from the Discourse Comprehension Test (Brookshire & Nicholas, 1993), and solicited JOLs for all four DCT categories (main ideas vs. details, and implied information vs. stated information). For both participant groups, predictive accuracy of JOLs for stated information was fairly strong ($\gamma \geq 0.70$),

regardless of whether occurring in main ideas or details. Predictive accuracy was poor ($\gamma < 0.28$) for implied information for both groups, irrespective of salience.

Finally, in order to assess accuracy and generalizability of prediction skills between the paired associate learning and narrative recall tasks, Kennedy (2004) re-analyzed the data from Kennedy, Carney, and Peters (2003) and Kennedy and Nawrocki (2003). She found no relationship between the predictive accuracies of one task vs. the other; that is, individuals with high or low predictive accuracy in one task did not necessarily demonstrate similar predictive accuracy in the other task.

Overall, the corpus of research generated by Kennedy and colleagues has revealed several important findings about metamemory in the TBI population that bear summarizing. In particular, for paired associate learning, while TBI survivors have poorer recall accuracy in general than neurologically normal controls, the accuracy of their prospective metamemory judgments (as measured by JOLs) tends to equal that of controls when made after a delay ($\gamma > 0.90$). For immediate JOLs, both groups show poor predictive accuracy (with a non-significant group difference), thus confirming the delayed JOL effect for both groups.

Two other studies of note generally found results quite similar to the work of Kennedy and colleagues. Prior to administering the California Verbal Learning Test (CVLT), and several subtests of the Wechsler Memory Scales-Revised (WMS-R), Schmitter-Edgecombe and Woo (2004) provided participants with a description of each test. They then asked participants to predict their immediate and delayed recall of test information, and to rate their confidence in their predictions (ranging from 0% = no confidence, to 100% = complete confidence). The TBI survivor group performed as

well as controls in predictive accuracy, with both groups providing lower prediction scores in the delayed condition than the immediate condition. Thus, despite the use of different memory tasks and measures of metamemory performance, Schmitter-Edgecombe and Woo found results quite similar to those of Kennedy and colleagues.

Hanten, Dennis, Zhang, Barnes et al. (2004), examined metamemory performance in children with TBI. Participants were given a list of words and were asked to make Ease of Learning (EOL) ratings for each word, followed by a study phase and recall testing. After all study-recall trials, participants were asked to make item-by-item delayed JOL predictions as to the likelihood of recall in two hours. Then, during recall testing, participants were asked to make retrospective confidence judgments. While children with TBI had impaired performance relative to controls on the Ease of Learning judgments, neither their predictive accuracy in delayed JOL-s, nor the accuracy of their retrospective confidence judgments, was significantly different from controls. These findings are quite in line with the findings for brain injured adults in the work of Kennedy and colleagues.

IS METAMEMORY IMPLICIT?

As we review the history of research into metamemory, a dialectic begins to emerge regarding a number of interrelated and highly overlapping issues. The first such issue concerns the nature of the representations on which the metamemory process operates, with some researchers arguing that the metamemory system relies solely on

access to some or all of the target memory trace (Hart, 1965; Koriat 1993, 1995; Kinoshita, 1997), while others argue that it relies only on familiarity with non-target cues, such as the context of the target, features of the question, etc. (Metcalf et al., 1993; Reder & Schunn, 1996). These divergent perspectives give rise to the second issue: the process by which metamemory operates. Researchers who favor the “trace-access” model of metamemory tend to view metamemory as a process that is either unitary with the actual retrieval process, or inextricably symbiotic with it (Hart, 1965; Koriat 1993, 1995; Kinoshita, 1997). Furthermore, this view requires that the metamemory process occur simultaneous to, or subsequent to, retrieval – that is, as a post-retrieval process (in that it relies on some or all of the products of retrieval). Conversely, those researchers who adhere to the “cue-familiarity” hypothesis view the metamemory process as distinct and dissociable from the retrieval process (Reder, 1987; Jameson et al., 1990; Rajaram, 1993; Metcalfe et al., 1993). Here the metamemory process is often viewed as occurring prior to retrieval, as a cognitive control mechanism whose purpose is to determine whether a search and retrieval attempt would even be worthwhile. In this view, therefore, metamemory is seen as a pre-retrieval process. Finally, the trace access model is generally seen as an explicit process since it relies on the products, or partial products, of an explicit recall process. Conversely, some assert that the cue-familiarity model is entirely implicit (Reder & Schunn, 1996), while others are non-committal as to whether the process is implicit or explicit (Metcalf, 1993).

With regard to this last issue of whether metamemory involves implicit or explicit processes, we turn now to a brief overview of that subset of research which

bears directly on the present research questions and task methodologies. It should be noted that the vast majority of research on metamemory judgments have focused on FOK, however a few studies have examined JOLs. Given that FOKs and JOLs have been shown to be uncorrelated measures of metamemory (Leonesio & Nelson, 1990; Schraw, 1995; Maki, 1999; Souchay, Isingrini, Clarys, Taconnat, 2003) the relative paucity of research using JOLs to examine the implicit/explicit question underscores the importance of doing so moving forward.

Hart (1965) was the first researcher to examine metamemory judgments empirically. Based on his research, he argued that FOK judgments are products of a memory monitoring system, operating on the contents of stored memory. Hart (1967) accounted for the FOK judgment with what Nelson, Gerler, and Narens (1984) refer to as the “trace-access” mechanism, wherein individuals partially access aspects of the memory trace they have failed to recall, but feel they know. That he viewed this process as explicit may be inferred from such statements as, “When subjects make judgments, they ... monitor or check what they do remember to arrive at a decision about what they might remember” (p.215). In particular, here he views the metamemory process as being comprised of a slow *decision* process that contemplates the results of a recall attempt before arriving at a conclusion.

Nelson, Gerler, and Narens (1984) contrast this to “inferential” mechanisms, wherein the metamemory system monitors information not directly part of the memory trace, but which could form the basis of a FOK judgment. An example of this might include related episodic information, such as the context in which the memory trace occurs. To test this distinction, Nelson, Gerler, and Narens asked participants general

knowledge questions (e.g.: “what is the capital of Australia?”), requiring FOK judgments on the non-recalled items. They then tachistoscopically presented participants with answers to the non-recalled items, beginning with subliminal exposure durations and increasing exposure duration for each subsequent presentation until participants could answer the question. They found that the higher an FOK rating for a particular item, the fewer the number of exposures to the correct answer before the participant was able to identify the word. The explanation of these results that is consistent with the trace-access model is that the metamemory system takes as input accessibility of the memory trace, so that brief flashes of the target word act to prime the memory trace; items that had been given higher FOK ratings had had stronger memory traces and therefore required less priming to reach threshold activation for identification. Yet, the results give rise to the question of whether or not the subliminal, or near threshold, prime could not only prime the memory trace, but could actually prime the metamemory system directly rather than indirectly by strengthening the memory trace.

Jameson, Narens, Goldfarb, and Nelson (1990) tested this latter hypothesis by presenting participants with a general knowledge question, followed by subliminal presentation of either a prime or nonsense word stimulus, a backward mask of 150ms, and then the general knowledge question again. Participants then spoke the answer to the question and made FOKs on non-recalled items. The researchers found that FOKs for the nonsense word condition were significantly higher than FOKs for the correct answer prime condition. This was taken as evidence that the metamemory process is not as sensitive to perceptual information about the target as is the retrieval process, and

therefore that the metamemory system likely relies primarily on access to the memory trace. Jameson et al. (1990) did not account for the finding that nonsense words had increased FOK ratings. Furthermore, since they did not use unrelated real words as foils or use a true baseline condition (such as nonsense character strings), they did not generate data that might shed light on any effect of antipriming. That the nonsense word condition had significantly higher FOK ratings bears further exploration, particularly in terms of examining antipriming effects since this might argue against the researchers' conclusion.

Koriat (1993, 1995) supported Jameson et al.'s (1990) interpretation, and refers to this as the "accessibility" model. He asserts that the metamemory process relies on information associated with the target. However, he did depart from Hart (1965) in one regard; Koriat (1993) argued that while the metamemory process was not entirely independent of the retrieval process, it was also not unitary with it. Rather, he argued that metamemory involved an inferential process operating on target-related cues. He states,

Thus, FOK judgments must be computed on-line on the basis of clues accumulated during the initial stages of search and retrieval: The abortive attempt to retrieve the target leaves behind scattered debris that feed into a memory-monitoring process, which assesses the likelihood that the target will eventually be located. This process, then, is not independent of the retrieval process; if the latter goes astray, so will the former. (p. 612)

Reder and Ritter (1992) were among the first to produce findings to challenge the trace-access model. They presented participants with two-digit by two-digit arithmetic problems (e.g.: “17 x 49”), and asked them to make a rapid (< 850 ms) decision as to whether they could retrieve the answer from memory, or had to calculate it. The individual numbers as well as the arithmetic operators were independently varied over trials and trial blocks to control the frequency of exposure to the individual numbers or operators, and to each problem as a whole. Reder and Ritter found that the FOK judgment was most significantly correlated with the frequency of occurrence of the parts of the problem (e.g.: “17 x ”) than with knowledge of the answer. Thus, for example, participants who had previously seen the question “17 x 49” were subsequently more likely to select “retrieve” for the question “17 x 32”, than they were for the question “14 x 56”, irrespective of the fact that they had not seen either of these latter two questions before. Reder and Ritter concluded that feelings of knowing were not based on retrieval of the target (the answer to the question), but rather on features of the question itself.

Metcalfe and her colleagues (Metcalfe, 1993; Metcalfe, Schwartz, & Joaquim, 1993; Metcalfe & Finn, 2008) then further specified the argument to what they refer to as the “cue-familiarity” hypothesis. The idea, here, is that the degree of familiarity with aspects of the *cues* to the memory trace, rather than aspects of the memory trace itself, form the basis for the FOK judgment. Again, as with the broader “inferential” model, here the FOK is not based on partial retrieval of aspects of the actual memory.

While Reder and colleagues (Reder & Ritter, 1992; Reder & Schunn, 1996) had simply tested the cue-familiarity hypothesis, Metcalfe, Schwartz, and Joaquim (1993)

conducted experiments to contrast the trace access and cue-familiarity hypotheses. In a series of four experiments utilizing paired associate learning of cue-target word-pairs, the familiarity and retrievability of target words were systematically varied to dissociate the effects of each on the FOK judgments. Participants' cued-recall was then tested, and for incorrect responses, they were asked to make an FOK rating. Finally, a forced choice recognition task was given. The authors found that FOKs were correlated with the number of times the cue word was repeated, and not with recognition of the target. However, an analysis which included intrusion errors among items retrieved from memory at the cued-recall test showed that recall of *all* items from memory (whether correct targets or intrusions) was also strongly correlated with the number of times the cue word was repeated. This latter finding presents some evidence in support of Koriat's (1993) trace-access model; the "scattered debris" of the retrieval process may contribute to the metamemory judgment. Nevertheless, Metcalfe, Schwartz, and Joaquim did provide useful evidence that cues to the target, and not just information intrinsic to the target, played a role in FOKs. It should be noted, however, that they do not distinguish whether such cues are implicit or explicit.

Reder and colleagues (Reder, 1987; Reder, 1988; Reder & Ritter, 1992; Miner & Reder, 1994; Reder & Schunn, 1996) took this line of thinking one step further and argued that implicit memory, in the form of such pre-retrieval information as features contained within the question or problem presented to participants (which she defines as "intrinsic" variables) as well as task instructions, knowledge of successful strategies, etc. (defined as "extrinsic" variables) determine FOK judgments. Thus, the agent may not even be aware of what is influencing their FOK judgments. In fact, Reder and

Schunn (1996) go so far as to state, “our claim [is] that this rapid feeling of knowing is actually an implicit process rather than an explicit process” (p. 50).

While the above studies have generated much data on the possible contributions of implicit and explicit processes to the formation of Feelings of Knowing, it is important to note that since FOKs and JOLs are uncorrelated, research relying solely on FOKs cannot be used to construct a complete model of the metamemory system in general. Models of metamemory (and not just of a particular metamemory measure) must necessarily be informed by the findings from research using JOLs and other metamemory measures as well.

To that end, we now review three studies which employ JOLs, and which shed additional light on the metamemory system. In particular, Jang and Nelson (2005) conducted a standard paired-associate learning task with immediate and delayed JOLs and a subsequent recall test, to study the effects of such implicit factors as item difficulty, relatedness, number of study presentations, and study duration, on the JOLs. They hypothesized that if JOLs depend solely on information related to the cue-target word-pair (e.g.: item difficulty and relatedness), then manipulations of such “extrinsic factors” as number of study presentations and study durations should not affect the relationship between JOL ratings and recall accuracy. Thus, for example, by increasing study duration, recall accuracy will increase and JOLs will increase in turn (due to their correlation to recall accuracy). Conversely, if JOLs can be independently influenced by the extrinsic factors without affecting recall, then the different JOL vs. recall accuracy curves corresponding to different number of presentations or study durations should

reflect functionally different relationships. The authors found the curves shared the same functional relationship, irrespective of extrinsic cue manipulation.

Jang and Nelson's (2005) findings are interesting because they differ markedly from the metamemory findings of Reder and Ritter (1992), and particularly those of Metcalfe, Schwartz, and Joaquim (1993). While Reder and Ritter, and Metcalfe, Schwartz, and Joaquim showed effects of stimulus presentation frequency on FOKs, dissociated from recall accuracy, Jang and Nelson did not. One possibility is that these sets of findings simply reflect differences in the metamemory processes by which FOKs and JOLs are produced. However, given the differences in task methodology (e.g.: Metcalfe, Schwartz, & Joaquim only provided multiple presentations of the cue word, while Jang & Nelson gave multiple presentations of the cue-target word-pair as a whole), the different findings may also reflect differences at the level of representation – what representations are used as input to the metamemory system.

Even within a particular metamemory measure, the issue of differences in task methodology can produce remarkably different outcomes. Finn (2008) used Nelson and Dunlosky's (1991) standard paired associate learning task with immediate and delayed JOLs, but with a twist: half of the participants made JOLs based on the likelihood that they would *remember* the target word at recall testing (the standard procedure), while the other half made JOLs based on the likelihood that they would *forget* the target word by the time of recall testing. This simple change to the task methodology produced interesting results. For immediate JOLs the “forget” group gave significantly lower JOL ratings than the “remember” group, while no difference was found for delayed JOLs. For immediate JOLs, confidence scores (mean JOL minus mean accuracy, for

each condition) were higher for the group using the standard JOL wording of likelihood of future recall than for the group given the altered JOL wording (however, both groups were still overconfident). This slight change in wording changes the emphasis from one's judgment of the likelihood of future remembering to future forgetting. It is likely that this reflects two related, but distinct, metamemory processes, perhaps taking input from different, or overlapping, sets of mental representations. With so few studies examining metamemory in terms of JOLs, these findings underscore the importance of additional research using measures other than FOKs to construct a comprehensive model of metamemory processes and representations.

Exploring yet another aspect of metamemory processing, and based on the work of Koriat and Levy-Sadot (2001), Vernon and Usher (2003) used JOLs to test the idea that there might be a temporal course of involvement of pre- and post- retrieval mechanisms for metamemory judgments. In an initial encoding phase participants studied 60 words. Then, for the first experiment, participants made JOLs while looking at word triples, such as, "salt, deep, foam". The JOLs were made at two, four, eight, and 12 seconds after stimulus onset and required that participants rate the likelihood of future recognition of the correct associate to the triple (e.g.: "sea" in response to the triple "salt, deep, foam"). Word triples remained on the screen until the fourth, final, rating was done. The authors found that JOLs increased over time, and began to plateau toward the eight and twelve second times. Goodman-Kruskal gamma correlation of the JOL ratings with recognition accuracy, increased from 0.24 at two seconds to 0.47 at four seconds. In the second experiment, participants were shown riddles whose question included a single letter cue (e.g.: "what is an insect that stings and starts with the letter

‘b’?’ Answer = bee). Again, JOLs were solicited at the same time intervals as the previous experiment. During the test phase, the authors included same and transformed conditions (wherein the “same” condition was a repeat of a riddle presented earlier for JOLs, and the “transformed” condition involved a change of only the final letter in the riddle, e.g.: “what is an insect that stings and starts with the letter ‘s’?” Answer = scorpion). The authors hypothesized that when JOLs were made, since the transformed riddles would have high familiarity, even though they had not been seen before, they would have high initial ratings, but then as time goes on, there should be dropping JOLs. Although there was a large initial drop for transformed tokens, contrary to expectations there was a subsequent rise in JOLs. The authors point to the initial drop as supportive of the hypothesis of combined processes unfolding, or trading primacy, over time. However, an alternative hypothesis, which may better explain the unexpected subsequent rise in JOLs for transformed tokens is that as time proceeds participants are not actually recalling more information about the target, but are simply deducing logically the relationship among triple components; perhaps the increase of the JOLs over time reflects a combination of remembering and verbal reasoning, or perhaps only verbal reasoning. Since there is a logical relationship between the elements of the triple and the correct answer, it is possible that what was being measured was not a pure metamemory process, but one contaminated by an inferential heuristic.

What becomes apparent from the foregoing review of the literature is the lack of a clear and coherent model of metamemory to account for the findings, which itself is due to the disparate findings. In part, this is due to differences in the metamemory

measures employed (e.g.: FOKs vs. JOLs, or other measures), task type (e.g.: paired associate learning, general knowledge questions, arithmetic problems, etc.), and the type of manipulation (e.g.: number and duration of presentations, subliminal vs. supraliminal presentation, etc.). Clearly, there is a need for additional studies to further flesh out some of the issues that have been presented above, thereby providing a larger set of findings to better constrain metamemory theories. It is within this context that the present research is conducted. The significance of this research, and its placement within the extant literature will be addressed below.

Finally, the suggestion that metamemory may be driven by implicit mechanisms may find theoretical support from a connectionist perspective. From such a framework, cognitive systems (including sensory/perceptual systems) may be viewed as networks of processing units (e.g.: neurons or collections of neurons) representing information in a distributed and superimposed pattern of activation, and with weight changes taking place on connections between units to enable learning (McClelland, 1995). Marsolek, et al. (2006) have argued that such implicit processes as priming and antipriming are a direct result of ongoing adjustments of overlapping mental representations. Marsolek et al. (2007) also presented fMRI data on human subjects showing that the same cortical areas are activated by different visual objects being viewed, and there are changes in these activations due to previous object processing. This provides support for the idea that a single network maintains distributed, superimposed representations of multiple items that undergo continual adjustments. Haxby, Gobbini, Furey, Ishai, et al. (2001) conducted an fMRI study, investigating cortical representation of faces and objects

(e.g.: man-made objects, and pictures with no identifiable image). They found distributed and overlapping representations of all items in ventral temporal cortex.

We have seen that there is much evidence for the phenomenon of conceptual priming. Thus, taking the above connectionist argument a step further, if ongoing adjustments to superimposed representations contribute to priming and antipriming of visual object identification, is it possible that such a model can be at play in conceptual priming? Extended to an even more abstract level, perhaps metamemory monitoring processes take as input distributed, superimposed representations. If so, the ongoing adjustments of connection weights that occur following stimulus presentation may prime and antiprime the metamemory system. Thus, a connectionist framework may in part provide a rationale to support Reder and Schunn's (1996) claim that metacognition is driven primarily by implicit processes.

PURPOSE OF THE PRESENT STUDY

The purpose of the present study is to examine implicit memory and metamemory in individuals with and without TBI. More specifically, we seek to determine whether or not independent manipulation of subliminal masked priming and antipriming conditions can cause changes in individuals' metamemory judgments, and whether or not TBI alters any such findings. Several studies have begun to explore the role of implicit memory in metamemory, but none has used a subliminal masked

priming paradigm with individuals with TBI, nor explored the effects of antipriming in this population.

In the present study, these purposes are achieved through the subliminal masked presentation of baseline, prime, and antiprime stimuli immediately prior to studying word-pairs and making item-by-item metamemory judgments of the likelihood of future recall. A recall test is subsequently administered so that recall accuracy and relative predictive accuracy of metamemory judgments can be obtained. Furthermore, a long term repetition priming and antipriming task was conducted with the same participants, to determine whether the perceptual antipriming effect discovered by Marsolek et al. (2006) is observable in the TBI population. If, for this population, the antipriming effect is not observed in the metamemory task but it is observed in the visual object identification task, it may indicate that the implicit memory deficits of the TBI population occur at the post perceptual level. Thus, it will have been important to establish whether or not the antipriming effect is even observable at the perceptual level in this clinical population.

RESEARCH QUESTIONS

Several research questions are asked in the present study. The most fundamental of these is whether manipulations of masked priming condition affect measures related to metamemory judgments (JOL ratings, relative predictive accuracy, or JOL response times). In contrast to the null hypothesis (that manipulation of the

prime condition will have no effect) it was expected that JOL ratings would be increased for primed items and decreased for antiprimed items, relative to baseline. Also, JOL response times will be faster for primed items and slower than antiprimed items, relative to the baseline condition. However, these effects were expected only for immediate JOLs, not significantly in the delayed condition; thus, there will be an interaction effect for prime condition and JOL timing condition. This last hypothesis is based on the observation that the effects of a subliminal masked prime generally attenuate within a few seconds of the presentation of the prime (Forster & Davis, 1984; Forster, Booker, Schacter & Davis, 1990). Thus, since delayed JOLs are made minutes after exposure to the subliminal prime, it is expected that they will not be affected.

A second research question is whether or not masked priming in the metamemory task would affect explicit recall. Recall that Jameson, et al. (1990) found that when participants were given masked primes of answers to non-recalled general knowledge questions, their recall accuracy improved. Similarly, Kinoshita (1997) found that recall for masked primed items was greater than non-primed items in a word-list memorization task which included FOK judgments on non-recalled items. Thus, the expectation was that recall accuracy would be improved for primed items relative to baseline items. Conversely, perhaps due to interference, antiprimed items were expected to demonstrate lower recall accuracy.

A third question was whether or not there would be any differences between TBI survivors and neurologically normal controls for any of the dependent measures? Given the consistent findings in the literature that individuals with TBI demonstrate poorer explicit recall (e.g.: Lezak, 1979) and slower response times (e.g.: Bashore &

Ridderinkhof, 2002), it was expected that these findings would be replicated. However, one caveat is that this hypothesis may be compromised by the fact that participants were only allowed to study each word-pair list once, as opposed to the procedure by Kennedy and Yorkston (2000). Thus, recall accuracy for both groups is expected to be lower. Otherwise, it was expected that the priming effects described above would obtain for both TBI survivors and control participants, however the magnitude will be smaller in the TBI survivor group.

The next research question was whether or not masked priming would differentially affect metamemory as a function of participants' explicit recall skills? That is, for example, would individuals with poor explicit recall skills demonstrated a greater or lesser effect of priming on metamemory measures than those with very good recall? If metamemory judgments are based on both implicit and explicit processes as, for example, suggested by Vernon and Usher (2003), then we expect that individuals with poor explicit memory may base their metamemory judgments primarily on input from implicit memory. Thus, priming effects should be stronger in those with poor recall, and weaker in those with very good recall.

Finally, with regard to the implicit metamemory task, on a practical note, the validity of the assumption that the masked priming paradigm was indeed implicit (that participants were not aware of the prime) must be demonstrated. Thus, a research question is whether or not antiprime stimuli in the implicit metamemory task would be judged as "new" approximately as often as items which actually were new (in the sense of not having been previously viewed in the experiment). The validation task was designed for this purpose, and the expectation was that antiprime stimuli and new items

would demonstrate approximately equal percent judged as old, and both types of stimuli would be judged as old significantly less often than prime stimuli.

Turning now to the visual object identification task, our first research question is whether or not TBI survivors would show both priming and antipriming effects, for object identification accuracy and response times. The expectation was that TBI survivor participants would show both priming and antipriming effects in both dependent measures. Overall, accuracy and response times for TBI survivors are expected to be worse than for controls. However, given that antipriming of visual object identification is hypothesized to be dependent on synaptic modifications in the ventral visual pathway (Marsolek et al., 2006), and given the relative sparing of this region as well as of synaptic transmission, in comparison to the prevalence of frontal and diffuse axonal injury in the TBI population (Inglese, et al., 2005), it may be that antipriming and priming will be preserved following TBI.

A related research question is, how does the magnitude of priming and antipriming in TBI survivors compare with those reported by Marsolek et al. (2006) for amnesic individuals? It is expected that the magnitude of priming and antipriming for TBI survivors will be smaller than matched controls, particularly for response times, but as stated above with the caveat of an even smaller magnitude for the antiprime condition. However, given the relatively mild cognitive impairment of the TBI survivor group, the magnitude of priming increase to accuracy, relative to baseline, is expected to be greater than for Marsolek et al.'s amnesic participants. As for antipriming, since the magnitude of antipriming decrement to accuracy is relatively small to begin with, and given the great deal of variability in the TBI population, it is expected that the

magnitude of antipriming decrement will not be significantly different between controls and the TBI survivor group. Furthermore, based on the review in the section above on the neural bases of implicit memory and metamemory, it is expected that individuals with TBI will demonstrate longer reaction times, but be more accurate compared to the data from amnesic participants in the Marsolek et al. study.

Finally, the last research question examines whether or not there is a relationship between the degree of priming and antipriming in the visual object recognition task, and the degree of priming and antipriming in the paired associate learning task. For example, is there a correlation between an individual's "primability" (as measured by their increase in object identification accuracy for primed items in the visual antipriming task over their baseline accuracy), and the increase in their relative predictive accuracy for primed word-pairs in the metamemory judgments? Such a finding would provide a weaker form of support for a relationship between implicit memory and metamemory. The expectation was that such a correlation would be found.

SIGNIFICANCE OF THE RESEARCH

The present study is significant both along theoretical as well as clinical lines. It seeks to add to the existing body of research in implicit metamemory in several ways in both of these dimensions. From a theoretical standpoint, one limitation to the literature examining implicit effects in metamemory is that many of the studies include tasks

which rely on cognitive processes beyond memory (e.g.: verbal reasoning, arithmetic problem solving, etc.). The paired associate learning paradigm has been selected for the present research; presentation of cue-target pairs for study, followed later by a cued recall test, avoids some of the concerns of involvement of extraneous cognitive processes.

Another very important theory-driven aspect of the present research is the type of manipulation of independent variables. In particular, to examine whether indeed metamemory is driven by non-conscious processes (as Reder & Schunn, 1996, imply), the use of subliminal masked priming vs. supraliminal manipulations (e.g.: presentation duration, # presentations, etc.) is an important choice to ensure that participants are truly unaware of the manipulation. This allows a purer exploration of the contribution of subliminal implicit memory processes to metamemory. Furthermore, while the subliminal masked priming paradigm has been used to explore FOKs in the paired associate learning task, it has not previously been used to explore JOLs in such a task. That said, we also include a supraliminal implicit memory task (long term repetition priming) to examine potential correlations between visual perceptual priming and metamemory priming.

A third important significance of the present research, is that although a number of studies have examined the effects of priming on metamemory judgments, no research has yet been conducted to determine the possible effects of the newly discovered phenomenon of antipriming. While such effects, if present, are expected to be quite subtle, positive findings may have profound implications for the nature of information representation and processes within and across cortical regions. Furthermore, as stated

above in the section on the neural substrates of implicit memory and metamemory, whereas antipriming is thought to be the result of accumulated weight changes on connections across a network of neurons, testing the antipriming paradigm in a population in which diffuse axonal injury is more common than in other types of brain injury, may shed further light on antipriming.

Fourthly, although much research has already been conducted using the paired associate learning task, little of it has used JOLs beyond the work of Kennedy and colleagues. Much of it has relied on FOKs, leaving a need for additional evidence regarding JOLs. Furthermore, FOKs are retrospective, in that they require rating the likelihood of successful retrieval of previously encoded information from memory. In contrast, we use JOLs, in which subjects rate the likelihood of future recall of information currently being encoded. This choice is based on several considerations. Firstly, whereas JOLs can be solicited for all items, FOKs are only obtained for items that one has failed to recall. JOLs are therefore able to provide information over a broader range of what is in memory. Secondly, since JOLs are prospective in nature, they can be used by TBI survivors to assess the likelihood of future recall failure and adjust their memory strategies accordingly, particularly if they rely on delayed JOLs (Kennedy, Carney, & Peters, 2003). Since TBI survivors' likelihood of using compensatory memory strategies depends on their predictions and beliefs of future recall failure, this was judged to be a more relevant metric of metamemory for this population. Given the clinical implications of the present study, this is an important consideration.

Therefore, with regard to this last point of the clinical aspect of metamemory research, another contribution of the present study is to examine JOLs in the TBI population as well as in neurologically normal matched controls. How an impaired memory and metamemory system behaves is expected to shed light on metamemory processes. The clinical relevance of this lies in potential findings regarding the relationship between implicit memory and metamemory; if the former plays a significant role in the latter (e.g.: as argued by Reder & Schunn, 1996), then perhaps intact implicit memory skills in this population can be leveraged to assist with rehabilitation of dysfunctional metamemory.

An unfortunate irony of TBI rehabilitation is that the very skills necessary to realize significant gains in treatment, and ultimately return to employment or even activities of daily living (i.e.: self-awareness, self-monitoring, and self-control) are among those which are compromised by the injury. Hart and Evans (2006) state,

Most of our everyday actions use a combination of implicit or unconscious processes and conscious decision making that occurs within, not before, the stream of action. Moreover, disabling conditions are apt to disrupt the fabric of everyday life that supplies us with goals in major areas of activity such as work, family responsibilities, and leisure time pursuits. At the same time there may be demands in unfamiliar goal areas, such as management of medical problems and new appointments. Added to the challenges that may be faced by anyone with a disability is the fact that for many people with TBI, the ability to set and achieve goals is compromised by cognitive impairment. Deficits in executive

function include problems with planning, self-monitoring, and behavioral control, the very abilities that are most needed for productive goal directed behavior (p.143).

Therefore, it is hoped that better understanding of the “combination of implicit ... processes and conscious decision making” may translate into direct clinical application.

To that end, the implications for learning are explained by Reder and Schunn (1996), who suggest that if the metacognitive skill of strategy selection is an implicit process, then during education or rehabilitation, “the implicit strategy selection processes should be left implicit, and attempts to tune them through explicit instructions will fail” (p. 71). They point to research that indicates that for non-brain injured individuals, learning new strategies is best done explicitly. So, the conclusion would be that non-brain injured individuals should explicitly learn new task strategies, but then once learned, they must allow implicit, subconscious processes to select which of those explicitly learned strategies to use for a particular task. Conversely, if individuals with TBI have difficulty with explicit learning, but demonstrate preserved implicit memory, then they should rely on implicit memory strategies and not explicit strategies. Thus, Reder and Schunn state that the goal for educators may be, “to determine whether poor performance on a task is due to incorrect strategy selection among an existing repertoire or lack of knowledge of the appropriate strategy. If the appropriate strategy is missing, then explicit teaching methods are likely to be best. However, if the appropriate strategy is known but simply not selected, then more implicit methods of teaching are required” (p. 72). It should be noted that Kennedy, Carney, and Peters (2003) rightly

caution against direct application of this kind of approach to the brain injured population. They state, “In complex, everyday situations, individuals with ABI may not be able to self-monitor and use self-assessments to guide strategy decisions given additional cognitive demands” (p. 1061).

In summary, the importance of the present proposal is to provide evidence for or against a relationship between metamemory and implicit memory in individuals with and without brain injury. Furthermore, this line of research may provide rehabilitation professionals with additional guidance in terms of assisting TBI survivors in the rehabilitation of metacognitive deficits.

METHOD

Summary of Experiments

The design included two experiments. The first consisted of a modification of the design of Kennedy and Yorkston (2004), itself based on Nelson and Dunlosky (1991). Its purpose was to study participants' metacognitive skills and how judgments-of-learning (JOLs) may be influenced by subliminal masked priming. The second experiment is based on experiment 4 of Marsolek et al. (2006). It was included to measure implicit memory, determine whether there is a correlation between magnitude of priming and metamemory predictive accuracy, and how these may differ between TBI and neurologically normal controls.

All of the research experimental procedures were approved by the University of Minnesota Institutional Review Board for human subjects prior to initiation of participant recruitment and experimentation. Basic demographic information (e.g.: age, sex, education, history of brain injury [for participants with brain injury], employment, etc.) was obtained during a phone interview (see Appendices A and B for the telephone interview forms), and was rechecked at the beginning of the first session. Prior to the start of the study, participants read informed consent and Health Information Portability and Accountability Act (HIPAA) forms, and were given the opportunity to ask questions about the study. They were then asked a series of questions to determine whether or not they had understood the contents of the forms (see Appendix C). Once all questions had been correctly answered, or explained by the experimenter if the

participant failed to answer any question correctly, then the participant was invited to sign the consent form. Individuals with acquired brain injury were further asked to sign HIPAA release forms for all health care providers from whom they had received services related to their brain injury, to facilitate obtaining medical records associated with their brain injury.

All individuals participated in two experimental sessions, and were given \$25 in compensation for each session completed, for a total of \$50 for the two sessions. During the first session, participants completed paperwork as described in the previous paragraph, and completed the two experimental tasks. Participants were given mandatory 15 minute breaks approximately every 20-30 minutes during this time. The average times, including rest breaks, to complete the metamemory experiment were 1 hr 56 min for ABI survivors, and 1 hr 35 min for controls. The average times to complete the antipriming experiment were respectively 38 min for ABI survivors and 30 min for controls. The total times, including paperwork, rest breaks, etc., for the entire first session averaged 3 hr 21 min for ABI survivors, and 2 hr 51 min for controls. During the second session participants completed all of the standardized tests. Total times, including rest breaks, averaged 3 hr 8 min for ABI survivors, and 2 hr 34 min for controls. The average number of days between sessions was 6.8 days (*SD* 3.8) for ABI survivors, and 9.4 days (*SD* 7.7) for controls. All participants completed the experimental session (including the paperwork and the two experiments) in the Neuro-Cognitive Communication Research laboratory at the University of Minnesota. Of the 17 ABI survivors, two completed the standardized testing in their residences, while the remaining 15 ABI survivors completed both sessions in the research laboratory. Of the

14 control participants, one completed the standardized testing in their residence, and the remaining 13 completed both sessions in the research laboratory.

PARTICIPANTS

Two groups of participants were recruited, consisting of 18 adults with a reported history of brain injury, and 14 healthy adult controls (without any history of neurological illness or injury). Brain injury survivors were recruited from local rehabilitation programs, hospitals, support groups, and through the online newsletter of the Brain Injury Association of Minnesota. One individual with a history of head injury was excluded from the study after participation, after review of his medical records; all neurological findings and reports indicated no evidence that the participant's head injury resulted in any cognitive impairment, despite his persisting complaints. Fourteen healthy control participants were included in this study, and were matched to the brain injury survivors on the basis of age, sex, and education. See Table 1 for summary demographic information, and Tables 2 and 3 for detailed participant demographic information.

Adults with brain injury averaged 47.42 years of age (*SD* 10.56), with 14.68 years of education (*SD* 1.94). Control participants were matched to brain injured participants on the basis of age (*M* 47.22, *SD* 10.17), and education (*M* 15.36, *SD* 1.66). Age at time of injury for brain injured participants averaged 34.83 years (*SD* 11.84). They were an average of 12.60 years (*SD* 11.18) post onset of injury at the time of participation in the experimental tasks. All group differences are non-significant at $p < .05$.

Etiologies of Acquired Brain Injury

Of the 17 participants with acquired brain injury, one of these was the result of a right frontal lobe glial tumor. The remaining 16 acquired brain injuries were of closed head traumatic origin. The medical records of three of the participants with closed head TBI confirm a diagnosis of brain injury, but do not provide any information from neurological imaging or other sources to indicate specific details of the brain regions injured. Glasgow Coma Scale scores at the time of injury were available for seven of the 16 TBI survivors. Length of coma could be approximately determined for only seven TBI survivors. Length of post traumatic amnesia could be determined by self-report for only three TBI survivors. Based on these data, and following Stein's (1996) classification of severity of head injury, initial severity of TBI was mild for two TBI survivor participants, severe for seven TBI survivors, and could not be determined for the remaining seven TBI survivors. Details of the specific etiologies, lesions, and other findings for the ABI participants may be found in Table 4.

Inclusion Criteria

All participants were native speakers of English, within the ages of 18-65 years old, and with at least a 10th grade education completed. Additionally, inclusion criteria for adults with brain injury included a diagnosis of brain injury acquired in adulthood. Medical records of adults with reported brain injury were reviewed (after informed consent, and authorization for release of information were obtained) to verify the brain injury.

Exclusion Criteria

TBI survivors and control participants with a history of prior neurological disease or stroke were excluded from the study. Participants with known learning disabilities, uncorrected auditory or visual impairments, or any reading impairment were excluded as well. Individuals with ambulatory or mobility problems that might prevent them from coming to the research facility or carrying out the tasks were excluded. Individuals with a history of alcohol or drug abuse requiring hospitalization or chemical detoxification, or hospitalization for a history of psychiatric problems, were excluded. Individuals who reported being classified as gifted in primary or secondary school were to be excluded as well, but no such individuals presented for participation in the experiment.

Acquired brain injury survivors were to be excluded if they had aphasia of even mild severity (i.e.: Western Aphasia Battery aphasia quotient ≤ 93.8 ; Kertesz, 1982). However, none of the brain injury survivors who passed the phone screening had aphasia (minimum WAB aphasia quotient of all participants = 95.2). Finally, those who were fewer than six months post injury were also excluded to minimize the impact of spontaneous neurological recovery.

Standardized tests

Several test batteries, and subtests of test batteries, were used to characterize the participant groups. Please see Table 4 for means, standard deviations, and statistical comparisons of all of the following neurocognitive measures. The Visual Reproduction I, Visual Reproduction II, and Digit Span subtests of the Wechsler Memory Scale – 3rd

Edition (Wechsler, 1987) were used to characterize learning, as well as visual, short term, and working, memory. Whereas the antipriming experiment of the present research involves a visual object recognition task in which visual objects are very briefly presented, the Visual Reproduction I & II subtests of the WMS-III were used to characterize explicit visual memory following long presentation.

In the Visual Reproduction I subtest participants are shown a series of designs for 10 seconds each. After each design is shown, it is then hidden from view and participants are required to draw it immediately from memory. The Visual Reproduction II subtest is administered after a 25 – 35 minute delay; participants are asked to draw from memory all of the designs shown during Visual Reproduction I. Following this, they are shown a number of designs, including some that were shown in Visual Reproduction I, and asked to indicate whether the design had been shown before or not. The digit span subtest of the WMS-III requires that participants immediately recite a string of single digits presented verbally, at one digit per second, by the tester. The number of digits increases systematically, until the participant is no longer able to recite the entire string correctly. The second portion of the subtest requires that participants recite the number string in reverse order from what was presented, again with increasing number of digits.

Brain injured participants demonstrated non-significantly lower scaled scores for immediate and delayed visual reproduction (9.53 and 11.24 respectively) compared to control participants (11.86 and 13.50 respectively). Visual delayed recognition memory was not significantly higher for the TBI survivor group compared to the control group (11.31 and 10.54 respectively). For the Digit Span subtest, total digit span scaled score

was 10.71 for TBI survivors, and 11.71 for controls; this difference was not statistically significant. It must be noted that the WMS-III was not normed on clinical populations. Various deficits that are not associated with memory may adversely impact participant scores. One brain injured participant in the present study demonstrated moderate incoordination of his dominant hand secondary to the brain injury; he had difficulty drawing the visual shapes with precision and this adversely affected his scores.

The Western Aphasia Battery (Kertesz, 1982) is a standardized test battery consisting of numerous subtests, which together assess verbal expression, auditory comprehension, reading comprehension, and written expression in individuals being evaluated for aphasia. The verbal expression, auditory comprehension, and reading comprehension portions of the WAB were used to determine whether participants met the exclusion criteria for aphasia. WAB aphasia quotient (WAB-AQ) and reading quotient (WAB-RQ) were respectively 98.7 and 99.5 for TBI survivors, and respectively 99.5 and 99.7 for controls. The lowest WAB AQ for ABI survivors was 95.2, and 95.6 for the lowest control. The group differences were not significantly different (Table 4).

The National Adult Reading Test, second edition (NART-R; Nelson & Willison, 1991), is a reliable metric of full scale intelligence quotient (IQ) in neurologically normal individuals, and pre-morbid full scale IQ in the brain injured population (Bright, Jaldow, & Kopelman, 2001). The NART-R was used in the present study to characterize participants' (premorbid) IQ and determine whether there were significant IQ differences between the participant groups. In this test, participants are presented with a set of written words one at a time and asked to read them aloud.

Pronunciation is recorded and scored. NART-R full scale IQ, verbal IQ, and performance IQ were respectively 106.94, 105.76, and 106.76 for TBI survivors, and 112.14, 110.43, and 111.43 for controls. There was no statistically significant difference between the groups.

The California Verbal Learning Test, second edition (Delis, Kramer, Kaplan, & Ober, 2000) is a standardized test battery used to characterize participants' verbal learning and memory. Whereas the implicit metamemory task involves verbal memory, this test was used to establish an objective standardized measure in participants. Scaled scores for free and cued verbal recall after short and long delays were obtained for both participant groups (Table 4), with no significant group differences observed.

The Delis-Kaplan Executive Function System (Delis, Kaplan, & Kramer, 2001) is a test battery designed to assess aspects of executive function. The Verbal Fluency, Tower, Design Fluency, and Trail Making subtests were used in the present study to characterize aspects of executive function in the participant population. The Verbal Fluency subtest requires participants to generate words rapidly, within specified semantic and phonemic categories, and to switch fluently between categories. The Tower subtest requires spatial planning, rule learning, inhibition, and establishing and maintaining a cognitive set. It requires participants to create towers of stacked disks of graduated size on a single peg, within a field of three pegs, to match a configuration depicted in the test booklet in front of them. Movement of the disks is subject to a number of rules (e.g.: only one disk at a time may be moved, larger disks may not be stacked onto smaller ones, disks may not be set aside, etc.), and goals (using the fewest possible moves, completing the task as quickly as possible and with the fewest errors).

The towers increase in difficulty as participants proceed. The Design Fluency subtest is a measure of cognitive flexibility which requires participants to connect dots in an array, using 4 straight lines, in as many different patterns as they can within a fixed time, without repeating patterns or violating certain rules. The Trail Making subtest is a visual-motor task which requires participants to draw a line connecting in ascending order numbers which are arranged in a random pattern across a large sheet of paper. This task is repeated with individual letters of the alphabet in place of numbers. The task is again repeated, with participants being required to alternate between letters and numbers.

There were few differences between the participant groups across these tests. Neurologically normal controls demonstrated significantly higher total verbal fluency for total correct category fluency ($p < .05$), and total correct category switching ($p < .01$). Verbal category switching approached significance ($p = .077$), with controls better able to switch between categories. Additionally, controls demonstrated significantly better motor speed scaled scores than TBI survivors ($p < .05$). Thus, the primary differences between the groups across these tests were that brain injury survivors demonstrating reduced verbal fluency and motor speed compared with matched controls. The groups were quite similar in overall estimated (pre-morbid) intelligence, immediate and delayed recall and recognition of visual designs, short term memory, free and cued verbal recall over both short and long delays, design fluency, and executive control (including spatial planning, rule learning, inhibition of impulsive responses, and establishing and maintaining a cognitive set).

EXPERIMENTAL DESIGN

Implicit Metamemory task

Task Summary

The implicit metamemory task was based on Kennedy and Yorkston (2004), but modified to include a standard three-field subliminal masked priming paradigm (Forster & Davis, 1984). In this task, subjects stared at a centrally located fixation point for 500 ms, followed by a 500 ms forward masking row of ampersands. Then an item was presented subliminally for 50 ms in the center of the screen in lower case. This item was either a row of x-s (baseline condition), the ensuing target word of the cue-target word-pair (prime condition), or a word unrelated either to the ensuing cue or target (antiprime condition). The subliminal item was immediately followed by the cue-target word-pair, in capital letters, for several seconds (5 seconds for controls, 9 for ABI survivors).

Forty-two word-pairs were presented, in two trial sets of 21 word-pairs per set. For a random half of the word-pairs, a JOL rating was made immediately after the studying of the word-pair. For the remaining half of the items, the JOL was delayed until the end of the trial set (2-3 minutes after studying). Thus, participants studied 21 word-pairs and made immediate judgments on 10 or 11 of them. At the end of that trial set, they made delayed judgments on the 11 or 10 items for which they had not already made immediate judgments. All JOL ratings were self-paced. Once these delayed

judgments were completed, participants repeated this procedure for the second trial set of 21 word-pairs in the 42 word-pair trial block. After completion of this second trial set, the study and judgment phase of the first 42 word-pair trial block was complete. If the elapsed time was less than 10 minutes, participants were engaged in a filler task of 2 minutes of conversation before proceeding to the cued-recall test. Otherwise, participants went directly into the cued-recall test.

During the cued-recall test, the cue words from the first trial set were randomly presented, followed by presentation of the cue words from the second trial set, again in random order. Participants spoke their self-paced response to each item into the microphone. The verbal response triggered a dialog box to appear on the screen, and the experimenter typed in the participant's verbal response. If the microphone was triggered either too soon or too late (e.g.: due to a noise prior to the spoken response, or because the spoken response was too quiet), then an "x" was entered into the dialog box followed by the participant's verbal response. Later, during data reduction, items marked with an "x" were removed from the analyses of response time, but were still included in the accuracy analysis.

Once all 42 items in the first trial block were completed, the participant was given a 15 minute break. This was followed by the second block of studying, making judgments, and taking the cued-recall test on the second block of 42 word-pairs. Again, following a 15 minute break, participants completed the third and final trial block, as above, for the last block of 42 word-pairs.

Validation that the masked prime was subliminal

Immediately upon completion of the implicit metamemory task (i.e.: after both the study and recall phases of the third block), participants were asked if they thought they had seen any words or symbols during the ampersand “flash” for any trial in any of the three trial blocks (during which the subliminal “primed,” “baseline,” or “antiprimed” items were presented). If the participant answered in the negative, no further questions were asked. If they answered in the affirmative, they were asked whether they thought that what they had seen was a word or a string of nonsense characters. If they stated that it was a string of nonsense characters, no further questions were asked. If they stated that it was a word, they were then asked what percentage of the time they believed words appeared during the “flash”, even if they could not read the word. They were then asked what percentage of the time they could actually read the word, even if they could no longer recall that word now. Of the 29 participants who completed the study, one claimed to have seen something during the subliminal presentation, and stated that this occurred one percent of the time, and that he could read the word one half of one percent of the time.

After participants answered these questions, they were given a validation task designed to determine whether or not the subliminal masked items were perceived in a manner that could be reflected in a recognition memory test rather than in the preceding recall memory test. For this task, single words were presented centrally one at a time. Participants were asked to make self-paced “old/new” judgments on the items. That is, if they thought the word being presented had appeared at any time in the experiment they were to press the button labeled “old”. If they thought the word had not appeared

in the experiment, they were to press the button labeled “new”. Forty two single words were presented for the self-paced “old/new” judgments. Of these, 14 words consisted of the 14 target words corresponding to the positive priming condition of the third, most recent, trial block of the implicit metamemory task. It should be noted that these words were not only seen supraliminally (for the 5 or 9 second study time), but were also presented subliminally (i.e.: primed) for 50 ms immediately prior to study of that item during the third block. Another 14 words were the antiprime words that were subliminally presented immediately prior to each of the word-pairs that were in the antiprime condition. It should be noted that these items were only presented subliminally, and were never presented as part of the cue-target stimuli in any block of the experiment. The last set of 14 words consisted of words that had not been used previously in the experiment at all. The order of presentation of the single words in this validation task was randomized.

Materials

List Construction

One hundred and thirty one unrelated English noun word-pairs (e.g.: LIME – COLLAR) were constructed, within constraints of imageability and concreteness (Kennedy & Yorkston, 2000). The MRC Psycholinguistic database (http://www.psy.uwa.edu.au/mrcdatabase/uwa_mrc.htm, January 7, 2008; Coltheart, 1981) at the University of Western Australia was used to generate a list of 367 words of three to seven characters in length, whose imageability values ranged between 510 – 690, and with concreteness ranging between 600 – 700. In the MRC Psycholinguistic

database, these variables range from 100 – 700 for words in the English language, with 700 being the maximum for both concreteness and imageability. The numerical ranges in the present study were targeted in order to match previous research (Kennedy & Yorkston, 2000), and to maintain the narrowest range of imageability and concreteness values while still producing a sufficient number of words to construct lists of the required size. The MRC Psycholinguistic Database derives imageability values from Paivio (1968), Toggia and Battig (1978) and Gilhooly and Logie (1980). Concreteness values were derived from a merging of the Paivio, Toggia-Battig, and Gilhooly-Logie norms.

Of these 367 words, those that were judged to be potentially disturbing either to normal controls, or to survivors of acquired brain injury (e.g.: blood, gun, etc.) were removed. Words that appear as items in the California Verbal Learning Test, National Adult Reading Test, Western Aphasia Battery, or among the items from the antipriming experiment were also removed from the list. Compound words were removed from the list. The remaining words were trimmed by random deletion to produce a final set of 318 words. Of these 318 words, 262 were selected at random. These were then randomly paired to create 131 word-pairs. Of the remaining 56 words, a random 42 were sequestered for use as subliminal antiprime items. The final 14 words were set aside for use in the validation task of this implicit metamemory experiment. Since these last 14 items were randomly selected from among the 318 original words, they reflect the same range of imageability and concreteness as the words used as prime or antiprime stimuli.

Using the “pairwise comparison” feature of the Latent Semantic Analysis (LSA) website at the University of Colorado at Boulder (<http://lsa.colorado.edu/>, January 7, 2008; Laham, 1998), the similarity rating for the two words in each of the 131 word-pairs was determined. LSA similarity ratings are determined by the cosine of the angle contained by the vectors of the two words in question; the vectors are calculated using factor analysis to condense a very large matrix of word-by-context data into a much smaller vector representation. Thus, a similarity rating of approximately 0.0 reflects no similarity at all between the words in the pair (e.g.: TOOTH – VALLEY), while a similarity rating of > 0.70 reflects a high degree of similarity (e.g.: BREATHE – INHALE). The web application is based on work by Dumais, Landauer and colleagues (Deerwester, Dumais, Furnas, Landauer & Harshman, 1990; Dumais, 1991, 1994; Landauer & Dumais, 1997). For the present study, word-pairs with a similarity rating greater than or equal to 0.15 (out of a maximum of 1.00) were randomly reshuffled until a total of 131 word-pairs were created, wherein no pairing resulted in similarity greater than 0.14. Examples of word-pairs with similarity of 0.14 (the maximum similarity of any word-pair used in this study) are: CLOWN-LUNCH, SISTER-PALM, ORANGE-FLOOD. The global mean value of similarity for the 131 word-pairs was 0.061 (*SD* 0.048). Of the 131 unrelated word-pairs thus produced, five word-pairs were selected at random and sequestered for use as training items.

Given that this experiment contains three priming conditions (baseline, prime, antiprime), two JOL timing conditions (immediate, delayed), and three trial blocks (first, second, third), there are a total of 18 possible combinations of conditions. Therefore, the remaining 126 word-pairs were then divided into 18 lists of seven words

each. These lists were created by successive random replacement of word-pairs within a list until the mean values for similarity of the word-pairs within the list, and the mean values for the concreteness and imageability for the set of words in the list, were within 0.5 standard deviations of the global mean, thus ensuring that T-Tests would fail to find significant between-list differences in similarity, concreteness, or imageability values. This procedure was repeated list by list, until a total of 18 lists of seven words each were created. See Table 6 for listwise mean and standard deviation values for word-pair similarity rating, imageability, concreteness, and word frequency.

These 18 lists were then permuted across participants, in an attempt to achieve full counterbalancing of all combinations of conditions across all participants. However, since there were fewer than 18 participants in each group (17 ABI, 14 control), full counterbalancing was not achieved. For each participant, the trial order was constructed using pseudo-randomization with replacement, such that from one trial to the next, no more than three successive occurrences of any one experimental condition appeared. Thus, for example, no more than three of the same priming condition (baseline, prime, antiprime), or three of the same JOL timing condition (immediate, delayed), would occur in immediate succession.

Hardware

A Dell Optiplex GX260 personal desktop computer, running Windows XP, interfaced with a five-button serial response box and running E-Prime™ software version 1.1 (Psychology Software Tools, Inc. 2001; Schneider, Eschman, & Zuccolotto, 2002) controlled stimulus delivery and data acquisition for this task. The computer

monitor was a Dell Ultrasharp 15" flat panel LCD monitor, with a refresh rate of 75 Hz, resulting in a refresh duty cycle of 13.3 ms. An Audio-Technica ATR-30 cardioid, low-impedance microphone was used to trigger participants' verbal responses. A custom-made, adjustable, non-restraining chin-rest was fashioned for this experiment, to ensure consistent vertical eye level, and eye-screen distance.

Procedures

General procedures

Upon participant arrival at the research laboratory, precautions were taken to ensure the environment was quiet and distraction free. After all forms were completed and informed consent was obtained, participants were given the opportunity of a restroom break prior to starting the first experiment. Participants were then seated in a comfortable office chair, with adjustable seat height and back support. The non-restraining chin rest was adjusted to provide a comfortable and consistent positioning of the participant's head for the duration of the task. The computer screen height was adjusted so that the center of the screen was at eye level. Eye to screen distance was adjusted to 86 +/- 1 cm. The positioning and height adjustments that were established for the chair, chin rest, and computer monitor prior to the first block were maintained through the entire experiment.

Implicit metamemory task procedure

A training and practice block was then initiated, wherein the computer provided written instructions detailing all phases of the experiment. Participants read the

instructions silently, one page at a time. When they completed reading each page, the experimenter verbally summarized the instructions of that page. When the participant indicated understanding, the experimenter cued the next page of instructions.

Participants were encouraged to ask questions to resolve any points of confusion or uncertainty. After the first portion of the instructions was completed, a practice phase was initiated; this was designed as a shorter version of the actual experiment, using the five word-pairs which had been sequestered for use in training. Thus, participants were shown five word-pairs for several seconds per word-pair (5 seconds for control participants, 9 seconds for TBI survivors). They used the five-button serial response box, with the buttons respectively labeled “0%”, “20%”, “40%”, “60%”, “80%”, and “100%”, to make immediate and delayed JOLs. Figure 4 depicts the task sequence.

During the study and judgment phase of the training block, the sequence of events was identical to that used in the actual experiment, with the exception that only the baseline condition (a row of x-s) was used for the 50 ms subliminal presentation. Participants were especially instructed on the importance of staring without blinking at the fixation point, and the ensuing row of ampersands, with special attention to the ampersand “flash” (which, in fact, was the subliminal masked stimulus which was presented for 50 ms, immediately after the row of ampersands and immediately before the cue-target word-pair). They were told that the purpose of the row of ampersands was to clear any retinal afterimage of the preceding word-pair, and that they should avoid blinking and should observe with vigilance the brief “flash”.

Once participants completed the training for the study and judgment phase, they were immediately given a practice cued-recall test on all of the training items, and

practice using the microphone. Practice with the microphone was particularly important in familiarizing participants with the appropriate level of loudness required to trigger the microphone, and to practice avoiding making extraneous noises (e.g.: lip smacking, speaking interjections such as “uh”, “um”, etc.) which might trigger the microphone too soon. Following this, participants returned to reading the instruction pages, where the sequence of events was again repeated, to ensure that participants understood the task. Once the training and practice block was completed, participants were again asked if they had any questions or points of confusion. If they did, these were answered prior to proceeding with the first trial block.

Immediately after completion of the training and practice block, participants began the first trial block. A mandatory 15 minute break was provided after each block. During this time, participants were allowed to either rest in the laboratory or leave the room as needed to attend to any personal needs. The procedures for the second and third trial blocks were an exact replication of the first trial block, but using respectively the second and third sets of 42 word-pairs. Please see Appendix D for details of the sequence of steps for the implicit metamemory task procedure.

As described in the task summary section above, immediately upon completion of both the study and recall phases of the third and final block of the implicit metamemory task, and prior to initiating the validation task, participants were asked if they thought they had seen any words or symbols during the ampersand “flash” for any trial of the implicit metamemory task. If they answered negatively, no further questions were asked. If they answered affirmatively, they were asked whether they thought that what they had seen was a word or a string of nonsense characters. If they stated that it

was a string of nonsense characters, no further questions were asked. If they stated that it was a word, they were then asked what percentage of the time they believed words appeared during the “flash”, even if they could not read the word. They were then asked what percentage of the time they could actually read the word, even if they could no longer recall that word now.

Validation task procedure

Participants were then instructed on the validation task. As with the instruction phase for the study, judgment, and recall portions of the implicit metamemory task, here participants were again presented with the instructions on the screen and asked to read them silently. Once participants indicated they had completed reading each page, the contents of the page were summarized and they were asked if they had any questions. No practice session was provided. Single words were then presented, centrally, one word at a time. If participants thought the word being presented had appeared in the experiment they pressed the button labeled “old”. If they thought the word had not appeared in the experiment, they pressed the button labeled “new”. Forty-two single words were presented. Of these, 14 words consisted of the 14 target words corresponding to the positive priming condition of the third, most recent, trial block; another 14 words were the antiprime words that were subliminally presented immediately prior to each of the word-pairs that were in the antiprime condition, and the last set of 14 words consisted of words that had not been used previously in the experiment at all. The order of presentation of the single words in this validation task

was randomized. Once participants had completed making the old/new judgments of the validation task, they were again given a mandatory 15 minute break.

Variables

Independent Variables

Independent variables included group, JOL timing condition, priming condition, and trial block. Groups included participants with ABI and neurologically normal control participants. JOL timing condition included immediate and delayed, wherein the delay in making JOL ratings was approximately two minutes after study of the word-pair. Priming condition had three levels: baseline (no prime – a row of x-s), prime (the ensuing target word), and antiprime (wherein a word unrelated to either the cue or target was presented subliminally). Trial block had three levels: first, second, and third.

Dependent Variables

Dependent measures included judgments-of-learning (JOL ratings), response time for JOLs, Goodman-Kruskal gamma correlation for predictive accuracy of JOLs, recall accuracy (proportion recalled correctly), and response time for recall (computed only for those items recalled correctly). Of these, the response times for recall were analyzed for correct answers only.

Visual Antipriming Task

Task Summary

The second experiment is based on experiment 4 of Marsolek et al. (2006). All of the materials, E-Prime programs, and procedures were obtained from Dr. Chad Marsolek's laboratory in the Department of Psychology, at the University of Minnesota. The experiment consists of four phases (see Figure 5). In the first phase, participants made ratings on a four-point scale of the degree to which they liked the meaning of auditory words they heard. The second phase required participants to name 100 visual objects that were presented sequentially, and very briefly, on a computer screen. These 100 items will be referred to as "baseline" items, since these words were not primed or antiprimed in the preceding phase. During the third phase of the task, participants made ratings using the same four-point scale as in the first phase. Ratings were made of 50 visual objects, presented for 3 seconds each; none of these objects was among those previously viewed in the second phase of the experiment. In the final phase of the experiment, participants again attempted to name 100 visual objects presented sequentially and very briefly. Of this second set of 100 objects, 50 were the same as were presented in the previous, third, phase of the experiment (and will be referred to as "primed" items), and the other 50 were new, in that they had not been previously viewed during the experiment (and will be referred to as "antiprimed" items). For the second and third phases of the experiment, accuracy, and response time measures (for correct responses), were acquired.

Although participants were offered a break between the second and third phases of this experiment, none took advantage of this, since the entire experiment was completed quickly for both groups (average time was 38 minutes for ABI survivors, and 30 minutes for controls).

Materials

Instructions

Unlike the implicit metamemory experiment, here the instructions for each phase of the task were printed on a standard 8.5" x 11" sheet of paper, in Times New Roman size 12 font.

Stimuli

The stimuli consisted of 250 greyscale pictures and drawings of uniform height, each depicting a unique everyday object. See Figure 6 for examples of these visual objects. Two hundred and fifty auditory recordings of the respective object names, corresponding to the visual objects, comprised the auditory stimuli for the first part of the task. Additional pictures and drawings were used for practice. Similarity of objects to one another was visually inspected to ensure that none of the objects was highly similar to any other object in the set. Marsolek et al. (2006) do not report mean values for word frequency, concreteness, or imageability. However, all pictures are of common everyday objects and animals, and therefore likely have high degrees of both imageability and concreteness. Full counterbalancing to ensure that every visual object was represented in each particular combination of experimental conditions an equal

number of times across participants was attempted. However, this was not fully achieved since there were five lists, but the number of participants was not a multiple of five. Otherwise, list construction was left unchanged from Marsolek et al.

Hardware

The same computer hardware, software, E-Prime button box, chin rest, and microphone were used as for the implicit metamemory experiment. In addition, two Harman-Kardon HK 195 stereo speakers were placed on either side of the computer screen, to present auditory stimuli for phase one of this experiment.

Procedures

This experiment consisted of four phases. Prior to the start of each phase, participants were handed a sheet of instructions for that phase. See Appendix G for the instructions of all phases of this experiment. After reading the sheet, the experimenter summarized the instructions and asked if the participant had any questions. Any such questions were answered at that time. Participants were then once again seated at the computer station used in the implicit metamemory experiment; no changes were made to the positions of the seat height, back rest, chin rest, or computer screen.

In the first phase of the experiment, participants stared at a fixation point in the center of an otherwise blank computer screen. They listened to a list of 50 words presented one at a time via stereo speakers. For each word, participants pressed one of four buttons on a serial response box (labeled 1, 2, 3, 4) to indicate the degree to which

they liked what each word represents, considering only their meaning and not their sound: the higher the rating, the greater the degree of liking. Stimulus presentation did not proceed until a rating had been made. Once all 50 words had been heard and rated, participants were given a brief practice session for the task in phase two (the “baseline” phase) of the experiment; the practice session was a brief, truncated, version of the baseline phase, using stimuli sequestered for training.

In the baseline phase participants stared at a fixation point on the computer screen at the start of each trial. Following the procedure of Marsolek et al. (2006), a visual object was presented very briefly (15 ms) in the horizontal center of the screen, and either 4.3° above or below the center of the screen. Based on data from Marsolek et al., older matched controls and brain injured individuals with amnesia required longer exposure durations (33 ms) than the young adults (15 ms), for similar levels of correct object identification. Although the intention was to set the exposure duration for 33 ms, in the present study the 15 ms exposure time was set inadvertently and left in place for all participants. The off center presentation was to avoid a ceiling effect for centrally presented objects. Half of the objects were presented above and half below the center of the screen; the vertical displacement was pseudo-randomized in that no more than three successive items could be all above or all below the center of the screen.

The visual objects were greyscale images of everyday objects. Participants were required to visually identify and speak into the microphone the name of the object as rapidly and accurately as possible. As with the implicit metamemory experiment described in the previous section, the training phase immediately prior to the baseline phase provided an opportunity for participants to learn how to speak their responses

with sufficient loudness to trigger the microphone, but without extraneous noises that might trigger the microphone prior to the actual verbal response. If the microphone was triggered either too soon or too late (e.g.: due to a noise prior to the spoken response, or because the spoken response was too quiet), then an “x” was hand-entered onto a response sheet, followed by the participant’s verbal response. Later, during data reduction, items marked with an “x” were removed from the analyses of response time, but included in the accuracy analysis. The baseline phase contained 100 visual objects to name. As participants named each object, the experimenter hand recorded the response on the response sheet. As with the metamemory experiment, response times were recorded by the computer and E-Prime program, based on a voice activated key. Once the baseline phase was complete, participants were allowed to take a brief five to ten minute break if needed. None of the participants opted for this break.

The third phase of the experiment will be referred to as the “encoding” phase. During the encoding phase, participants again stared at a fixation point at the start of each trial. Fifty visual objects were presented one at a time in the center of the screen for three seconds each. After each object was presented, participants were required to use the same rating scale and serial response box as was used in the first, auditory, phase of the experiment, to rate the degree to which they liked the meaning of what the visual object represented. The 50 visual objects presented in this phase were different from those of the baseline phase, and the names of these objects were not among those used in the first, auditory, phase of the experiment.

The final, “test”, phase immediately followed the encoding phase. Here participants again stared at a fixation point in the center of the screen. As in the

baseline phase, a visual object was briefly presented (15 ms), 4.3° above or below the center of the screen, and participants were required to speak the object name into the microphone as quickly and accurately as possible. In this test phase, 50 of the 100 items were the same as the 50 presented during the encoding phase, and the remaining 50 were new items. The new items were new in the sense that although they represent everyday objects, none of them had been previously viewed in the experiment, nor were their names heard during the auditory phase of the experiment. The order of presentation of trials was pseudo-randomized (random, with replacement to ensure that no more than three items in a row were new or previously viewed). Response times for the spoken responses were recorded by the computer's voice activated key, as before. The spoken responses themselves were hand recorded by the experimenter, as in the baseline phase. Once all 100 items had been named, the task was complete.

Variables

Independent Variables

Independent variables included group (individuals with acquired brain injury vs. neurologically normal controls), and priming condition (baseline, prime, and antiprime).

Dependent Variables

Dependent measures consisted of object identification accuracy (% correct) and response time (ms). Response time was calculated only for correct responses.

RESULTS

Overview

Several repeated measures analyses of variance were conducted to answer the research questions. As described in the methodology section, the present study consisted of two primary tasks (the paired associate learning task with masked priming, and the visual object identification task). A third task was employed subsidiary to the paired associate learning task; this was a validation task designed to confirm that participants were in fact unaware of the masked priming. Each task involved a different experimental design. Therefore, the details of data reduction, statistical assumptions, analysis techniques, and results for each task will be presented separately in each respective subsection below.

Because the validation task was crucial to establish the validity of the paired associate learning (or “implicit metamemory”) task, it will be discussed first. Then, the implicit metamemory task itself will be addressed. This will be followed by discussion of the visual object identification, or “visual antipriming,” task. Finally, various other post hoc analyses will be presented to address additional questions.

Validation Task

The purpose of the validation task was to confirm that participants were unaware of the subliminal masked priming in the implicit metamemory task. Predictor variables included group (with two levels: TBI survivors, controls), and “item-type” (with three

levels: prime, antiprime, and new). The criterion variables were percent judged as old (“percent old”), and response time in milliseconds. If indeed participants are unaware of the antiprime stimuli, then the prediction is that there should be a low percentage of antiprime stimuli judged as “old”. In fact, the hypothesis is that the percent old judgment and response time measures for antiprime and new stimuli should not be different from one another, but both should be significantly different from the prime stimuli. For this task, two separate two-by-three between-within repeated measures ANOVAs were conducted, one for each dependent measure, with group as the between subjects factor and item-type as the within-subjects factor.

Data reduction

A PERL script was written to reduce the data output from the E-Prime program into a comma-delimited format that could be imported into Microsoft Excel and/or SPSS for further analysis. The PERL script was designed to eliminate all irrelevant information (e.g.: computer time seed values, etc.) from the E-Prime data file, and sort the remaining data to facilitate further analysis. The Excel template computed item-by-item accuracy, accuracy by priming condition, and mean response times by item-type.

Statistical Assumptions

The assumption of normality was assessed by examining kurtosis and skewness values. These values were variable across item-type (prime, antiprime, new) and for both dependent measures (percent old and response time), for each of the participant groups. Across item-type for the two dependent measures, the magnitude of z-scores

for kurtosis ranged from -1.55 to 2.01 for the control group, and -1.22 to 1.60 for the TBI survivor group. For skewness, these values ranged from -0.90 to 1.16 for control participants and -1.23 to 1.08 for participants with brain injury (Table 7). There was no consistent pattern in skewness or kurtosis between the participant groups.

Although skewness and kurtosis generally fell within the acceptable range of -1 to +1 for both dependent variables, as seen above there were several combinations of variables and conditions under which assumptions of normality were marginally violated. It should be noted that even with a large sample size, such mild violation of normality is still anticipated for this task; the primed items would still be expected to have a very high percentage identified as old, and both antiprimed and new items would have a very low percentage identified as old. Furthermore, several studies have shown that ANOVA and MANOVA are highly robust to violations of normality (Box & Anderson, 1955; Lindman, 1974; Levy, 1980). Given that the violations of normality are rather mild and the fact that ANOVA and MANOVA are robust to such violations, results for this task may be viewed as reliable.

The variance-covariance matrices respectively for the repeated measures of percent old and response time were equal across both participant groups, supporting the homogeneity assumption [for percent old, Box's $M = 4.724$, $F(6, 5456.191) = .697$, $p = .652$; for response time, Box's $M = 8.902$, $F(6, 5456.191) = 1.313$, $p = .247$]. This indicates that as the independent variables are changed, the changes in the dependent measures are similar for the different groups. Bartlett's test of sphericity was significant for percent old (Mauchly's $W = .703$, approximate Chi-squared = 9.851, $p = .007$) and borderline significant for response time (Mauchly's $W = .809$, approximate

Chi-squared = 5.937, $p = .051$). This result for percent old is both expected and desired, since it indicates that the various combinations of levels of the independent variables (i.e.: the repeated measures of the same dependent measure) are strongly correlated with one another (positively or negatively). Thus, for example, we expect that the higher the percent of prime items judged as old, the lower the percent of antiprime or new items judged as old. As for response time, since Mauchly's test was borderline significant, a Huynh-Feldt correction was applied to the degrees of freedom.

Summary Results

For a summary of mean and standard deviations for percent old and response times by item-type for the two groups, please see Table 8. Repeated measures ANOVA (Table 9) shows a highly significant main effect of the within-subjects variable (item-type) on both percent old ($p < .001$) and response time ($p < .001$). The main effect of group was non-significant for percent old ($p = 0.823$), but significant for response time ($p = .014$). The interaction of group and item-type was non-significant for both dependent measures ($p = .129$ for percent old; $p = .663$ for response time). The univariate within-subjects F-test, collapsed across groups, for the effect of the item-type was highly significant. Pairwise comparison for the effects of the different item-types on percent old judgment shows that all item-types had a statistically significant and different effect on percent old judgments (Table 10). Examination of descriptive statistics (Table 8) shows that for control participants, percent old judgments averaged 90.8 (SD 8.6) for primed targets, 13.3 (SD 10.8) for antiprime stimuli, and 25.5 (SD 20.9) for new items. For ABI survivors, these values were 86.1 (SD 11.1) for primed

targets, 19.8 (*SD* 13.3) for antiprime stimuli, and 26.1 (*SD* 18.4) for new items (see Figure 7).

Thus, the expected result that both antiprime and new items would be significantly less likely to be judged as old, compared to prime items, was certainly obtained for both groups. However, an interesting and unexpected finding was that the antiprime items were significantly much less likely to be judged as old when compared to the new items. Both groups judged antiprime items as old significantly *less* often than items they truly had not seen in the experiment (the new items). This finding provides evidence that the subliminal masked priming paradigm did successfully affect participants of both groups. Additional implications of this finding will be addressed in greater detail in the discussion chapter.

As reported in the summary section above, the univariate between-subjects F-test for the effect of group on percent old judgment was non-significant. That is, the participant groups did not differ in their judgments of items as old. Thus, all participants judged primed target items as “old” approximately 90% of the time (collapsed across groups); these items had been primed and studied by participants for 5s (TBI survivor), or 9s (control), as targets in the cue-target word-pairs, so this result was expected.

Results for Response Time

As reported in the summary section above, the univariate within-subjects F-test for the effect of the item-type on the response time was highly significant. As with the percent old judgments, pairwise comparisons for the effects of the different item-types

on response times show that all item-types had a statistically significant different effect (significance tests were conducted on the mean differences of each dependent variable; see Table 10). Thus, response times for antiprime and prime items were significantly faster than for new items, for both groups. However, an interesting and unexpected finding was that the response times for antiprime items were significantly faster than for new items; this pattern held for both participant groups. This finding provides evidence that the subliminal masked priming paradigm did successfully prime (and antiprime) participants of both groups. Additional implications of this finding will be addressed in greater detail in the discussion chapter.

The between-subjects F-test for the effect of the group variable on the response times was also quite significant, as reported in the summary section above. Thus, the participant groups differed significantly in their response times, and this is clearly seen in the descriptive statistics. On average, TBI survivors were 359.2 ms slower to respond than controls. However, the group by prime interaction effect was non-significant, [$F(2, 28) = .418, p = .663, \text{partial eta squared} = .029$], so that the two groups showed the same pattern of response times. That is, both groups responded significantly faster to prime items than to antiprime items, and significantly faster to antiprime items than to new items.

This last pattern, combined with the findings for percent old judgment, bears emphasis. The results show that both participant groups judged prime target items most rapidly, and rated them as “old” significantly more often than the antiprime or new items; this was the expected outcome. However, both groups made the old/new judgment for antiprime items the second fastest, and were significantly less likely to

rate them as “old” compared to the new items. That is, they decided that antiprime items were “new” more often than they did “new” items, and they came to this conclusion faster than they did for the new items themselves.

One final source of evidence that participants were unaware of the subliminal masked presentation derives from participant responses on questioning. As described in the methods chapter, at the conclusion of the validation task participants were asked if they thought they had seen any words or symbols during the ampersand “flash” of the implicit metamemory task. If they answered “yes”, they were then asked follow up questions designed to determine whether or not they really had some conscious awareness of the masked items (e.g.: whether or not what they saw were words or strings of nonsense characters). Of the 29 participants who completed the implicit metamemory task, only one claimed to have seen something during the subliminal presentation. When asked how often he thought a word was presented during the ampersand flash, that participant reported that this occurred only 1% of the time.

Implicit Metamemory Task

Overview

Separate two-by-three-by-two, within-within-between, repeated measures ANOVA analyses were conducted for each dependent measure, with JOL timing condition as the two-level within-subjects factor, prime condition at test as the three-level within-subjects factor, and group as the two-level between-subjects factor. For the first analysis, average JOL rating was the dependent measure; this same analytical

method was then repeated for average recall accuracy, gamma correlation, and for the response time measures for the judgments of learning and the spoken responses during recall testing. This methodology was applied in preference to a repeated measures MANOVA, in which several dependent variables are simultaneously analyzed.

Although a repeated measures MANOVA was conducted for the present research, the results could not be interpreted, and so multiple separate repeated measures ANOVAs were employed instead.¹ However, given that multiple comparisons were made, a Bonferroni correction to the degrees of freedom was made to control familywise Type I error. For all analyses, the alpha level was set at .05.

Data Reduction

A PERL script was written to reduce the data output from the E-Prime program into a comma-delimited format that could be imported into Microsoft Excel and/or SPSS for further analysis. The PERL script was designed to eliminate all irrelevant information (e.g.: computer time seed values, etc.) from the E-Prime data file, and sort the remaining data to facilitate further analysis. An Excel template filtered the response times to eliminate those corresponding to incorrect spoken responses, as well as those values that exceeded two standard deviations of the respective cell mean for that participant. The template also computed item-by-item accuracy, mean accuracies for each cell (minus primacy items), and mean filtered response times by cell. Additional details of data reduction, including definition of acceptable responses and determination of outliers may be found in Appendix E.

Statistical Assumptions

For the results of the statistical tests of the assumption of normality, the reader is referred to Table 11. After removal of outliers, for 17 out of the 18 cells (three dependent measures by 3 levels of priming by two levels of JOL timing), skewness values were generally within, or very close to the -1 to +1 range for control participants. The exception was the antiprime JOL timing condition combination for gamma correlation, which had a skewness value of 2.746. Similarly, for controls, the kurtosis values generally ranged between -1 and +1, or very close to that range, with the exception again being the antiprime JOL timing condition combination for gamma correlation, which had kurtosis of 7.974. This particular combination of conditions for this dependent measure demonstrates a mean value of 0.891 for controls, causing the distribution to be highly peaked and skewed to the high end of gamma correlation.

For TBI survivors, skewness and kurtosis values again generally varied from -1 to +1, or very near to this range. For this group, three of the 18 cells were mildly out of the desired range; gamma correlation for primed items for both the immediate and delayed JOL timing conditions had respective kurtosis values of 3.179 and 5.286. The kurtosis value for accuracy of primed items in the delayed JOL timing condition was 4.823. Skewness and kurtosis values for response time measures, for both JOL and recall accuracy, across all combinations of priming and JOL timing conditions, were well behaved. Skewness values ranged from -0.2 to 1.1, and kurtosis ranged from -1.8 to 1.4.

Thus, overall there are only mild violations to the assumption of normality for the data sets. Furthermore, as stated in the previous section, numerous studies have

shown that ANOVA is highly robust to violations of normality (see for example, Box & Anderson, 1955; Lindman, 1974; Levy, 1980).

For the analyses of JOL, accuracy, and gamma correlation, Box's Test was non-significant [for JOL, Box's $M = 29.529$; $F(21, 2650.820) = 1.023$, $p = .382$; for accuracy, Box's $M = 40.749$; $F(21, 2650.820) = 1.467$, $p = .078$; and for gamma correlation, Box's $M = 45.295$; $F(21, 1663.707) = 1.500$, $p = .068$]. Thus, the variance-covariance matrices for each of these dependent measures were generally equal across both participant groups, supporting the homogeneity assumption. For the response time data, Box's Test was highly significant for both JOL and recall response times [for JOL response time, Box's $M = 52.720$; $F(21, 2368.124) = 1.870$, $p = .010$; for recall response time, Box's $M = 78.005$; $F(21, 2001.100) = 2.682$, $p < .001$]. Thus, the variance-covariance matrices for each of these dependent measures were generally unequal across both participant groups, violating the homogeneity assumption for response time measures.

Bartlett's test of sphericity was highly significant [for JOL, the approximate Chi-squared = 288.725, $p < .001$; for accuracy, the approximate Chi-squared = 180.553, $p < .001$; for gamma the approximate Chi-squared = 63.310, $p < .001$]. This result is both expected and desired, since it indicates that the repeated measures of the same dependent measure (corresponding to the various combinations of levels of the independent variables) are strongly correlated with one another (positively or negatively). Thus, for example, we expect that even if there are differences among the average JOL ratings of one participant for the different priming conditions, on average

all of those values may be quite a bit higher than those for another participant whose JOL ratings may all be low.

Mauchly's test of sphericity is non-significant for accuracy, gamma correlation, and JOL response time (for both priming and the prime-by-JOL timing interaction), indicating that the data meet the assumption of sphericity and no corrections are needed. (Please see Table 12 for a summary of these sphericity statistics for this task.) For JOL, however, Mauchly's test of sphericity is significant for priming, but not the prime-by-JOL timing interaction [for priming, Mauchly's $W = .609$; approximate Chi-squared = 12.909, $p = .002$]. Therefore, a Huynh-Feldt correction was applied for the within-subjects effects of priming condition on average JOL. Also, for recall response time Mauchly's test of sphericity is significant for both priming and the prime-by-JOL timing interaction [for priming, Mauchly's $W = .676$; approximate Chi-squared = 8.993, $p < .001$; for the prime-by-JOL timing interaction, Mauchly's $W = .627$; approximate Chi-squared = 10.753, $p = .005$]. Again, a Huynh-Feldt correction was applied for the within-subjects effects of priming and the priming-by-JOL timing interaction on recall response time. Otherwise, for all other terms in the model, sphericity is assumed.

Results

Summary of the descriptive statistics may be found for JOL, recall accuracy, and gamma correlation in Table 13, and for JOL response time and recall response time in Table 14. For the control group there were 14 participants' data per cell for analysis of the average JOL ratings, average accuracy, and average gamma correlation. For the response time measures (for JOLs and for spoken recall responses), there were 12

participants' data per cell for the control group, due to missing cases. For the TBI survivor group, again due to missing cases there were 14 participants' data per cell for analysis of the average JOL ratings, average accuracy, and average gamma correlation, as well as for response times for both JOLs and spoken responses during recall. It should be noted that recall accuracy was calculated as mean proportion recalled, thus providing a range of values from 0.0 to 1.0. Finally, with regard to recall response time, since this dependent measure was calculated only for correct responses, and average accuracy was low, recall response times were often based on one or a few trials, resulting in unstable data. Therefore, the recall response time data should be viewed critically. Since JOL response time was computed for all responses, it does not suffer from the same difficulty.

Several repeated measures ANOVAs were conducted to examine the effects of priming, JOL timing, and participant group on metamemory judgments, cued recall, and predictive accuracy, as well as the response times for making judgments and for recall. Results for all main effects and interactions are summarized in Table 15.

The main effect of priming was not significant for JOL ($p = .424$), recall accuracy ($p = .188$), gamma correlation ($p = .511$), JOL response time ($p = .362$), or for spoken response times during recall testing ($p = .447$). See Figure 8.

The main effect of JOL timing condition (immediate vs. delayed) was significant for all dependent measures. For JOL, delayed JOLs were lower than immediate JOLs (mean difference = -4.248 , $SE = 1.964$, $p = .040$). For recall accuracy, items for which delayed JOLs were made were more accurately recalled than items for which immediate JOLs were made (mean difference = $.070$, $SE = .016$, $p < .001$).

Delayed JOLs had significantly greater gamma correlation than immediate JOLs (mean difference = .452, $SE = .057$, $p < .001$). Response times were longer for delayed JOLs than immediate JOLs (mean difference = 1548.9 ms, $SE = 300.5$ ms, $p < .001$).

Response times for recall were significantly longer for those items with immediate JOLs than for delayed JOLs (mean difference = 1146.916 ms, $SE = 185.468$ ms, $p < .001$).

The main effect of participant group was not significant for JOL ($p = .914$), or gamma correlation ($p = .257$). For recall accuracy, the main effect of group approached significance ($p = .067$), with controls being more accurate on average than TBI survivors (mean difference = .141, $SE = .074$). The main effect of group on response time for JOL was significant, with TBI survivors taking longer than controls (mean difference = 1614.9 ms, $SE = 464.2$ ms, $p = .002$). There was a significant effect of group on the response time for spoken responses during recall testing, with TBI survivors again taking longer than controls (mean difference = 1558.284 ms, $SE = 637.195$ ms, $p = .022$).

Interactions that were significant or approaching significance will be listed here; for a summary of these as well as the non-significant interaction effects, please see Table 15. For JOL, the prime by JOL timing interaction was borderline significant ($p = .060$). Collapsed across groups, exploration of simple effects shows no significant comparisons due to large standard errors relative to mean differences. For example, while the mean difference in average JOL between the baseline condition ($M 46.43 SE 4.26$) and the prime condition ($M 40.01 SE 3.77$) is the largest among the combinations of conditions, it is not significant [$t(55.3) = 1.164$, $p = .250$]. Also for JOL, the group

by JOL timing interaction approached significance ($p = .085$); control participants demonstrated virtually no difference in JOL timing condition, averaging 43.831 for immediate JOLs and 43.090 for delayed JOLs. TBI survivors, however, averaged 46.503 for immediate JOLs and 38.749 for delayed JOLs, and this was borderline significant [$t(85.651) = 1.911, p = .059$].

Recall accuracy demonstrated a significant prime by JOL timing interaction ($p = .025$). Collapsed across groups, in the baseline condition, for items that had received delayed JOLs ($M = .415, SE = .046$) recall was higher than immediate JOLs ($M = .300, SE = .036$); this difference approached significance [$t(51.965) = -1.860, p = 0.069$]. Comparing the baseline items in the immediate JOL timing condition with antiprime items in the delayed JOL timing condition demonstrates a very significant difference [$t(54.727) = -2.270, p = 0.027$].

Also for recall, the three way interaction of prime by JOL timing by group approached significance ($p = .081$); however none of the individual comparisons approached significance. For example, although baseline items for control participants demonstrated a seemingly large difference in recall accuracy between items with immediate JOLs ($M .345$) and items with delayed JOLs ($M .511$), this difference was not significant [$t(23.045) = -1.643, p = .114$]. Similarly, although antiprime items for TBI survivors showed an apparently large difference in recall between items with immediate JOLs ($M .292$) and delayed JOLs ($M .377$), this difference was also not significant [$t(24.944) = 1.313, p = .201$].

Gamma correlation did not exhibit any interaction effects that were either significant or approaching significance.

Response times for JOLs did demonstrate a borderline significant JOL timing by group interaction ($p = .067$). The control group's response times for JOLs increased by approximately one second for delayed JOLs ($M = 3155.1$ ms, $SE = 515.6$ ms) as compared to immediate JOLs ($M = 2180.0$ ms, $SE = 248.6$ ms), [$t(54.391) = -5.328$, $p < .001$]. However, the TBI survivor group's increase in response time for JOLs was even greater, by over two seconds for delayed JOLs ($M = 5343.8$ ms, $SE = 480.0$ ms) vs. immediate JOLs ($M = 3221.1$ ms, $SE = 231.5$ ms), [$t(62.805) = -5.335$, $p < .001$].

Response times for spoken responses during the recall test were not significant for any two or three way interactions.

Visual Antipriming Task

Data reduction

A PERL script was written to remove all irrelevant information from the E-Prime data file (e.g.: computer time seed values, etc.), sort the remaining data to facilitate further analysis, and generate a comma-delimited output file. That file was then imported into a Microsoft Excel template for further analysis. Spoken responses to each of the 100 baseline items and 100 test items for each participant were transferred from the response record sheets to the Excel template. The template computed item-by-item accuracy of recall by comparing the spoken response against the actual target response and returning a "1" for an exact match, and "0" otherwise. Since synonyms and close synonyms were not exact matches, the computer recorded these as errors. Those items were subsequently hand-corrected to "1". Additional details of data

reduction, including definition of acceptable responses and determination of outliers may be found in Appendix F.

Statistical Assumptions

For a summary of normality statistics, the reader is referred to Table 16. As with the implicit metamemory task, here the final values of skewness and kurtosis for accuracy and response time values across priming conditions generally fall within the accepted range, with a few mild violations. Levene's test of equality of error variances was non-significant for all priming conditions for each dependent measure, with the exception of response time in the antiprime condition. Therefore, the data were judged to generally show equal error variances of the dependent measures across groups. Mauchly's test of sphericity is non-significant for both accuracy and response time [for accuracy, Mauchly's $W = .857$; approximate Chi-squared = 3.860, $p = .145$; for response time, Mauchly's $W = .886$; approximate Chi-squared = 3.026, $p = .220$], indicating that the data meet the assumption of sphericity and no corrections are needed. Bartlett's test of sphericity is highly significant [for accuracy, the approximate Chi-squared = 38.855, $p < .001$; for response time, the approximate Chi-squared = 34.960, $p < .001$] but this is both expected and desired, since it indicates that the repeated dependent measures are strongly correlated with one another (positively or negatively); thus, for example, if an individual has a baseline accuracy rate that is higher than the group average for baseline accuracy, then his or her accuracy rates for the prime and antiprime conditions will also likely be higher than the respective group averages.

Statistical Methods

Whereas this is based on experiment four of Marsolek et al. (2006), with the only difference being the etiology of brain injury in the TBI survivor group, the statistical methods employed for this task follow those of Marsolek et al. Thus, a two-by-three, between-within repeated measures ANOVA were conducted for each dependent variable, with group as the between-subjects factor, and prime condition at test as the within-subjects factor. Accuracy, and response time (for correct responses) were the dependent measures. For all analyses, the alpha level was set at .05, however, given that multiple comparisons were made, a Bonferroni correction was made to control familywise Type I error.

Results

For a summary of the following descriptive statistics, please refer to Table 17. For a summary of the following statistics, the reader is referred to Table 18. The main effect of test presentation condition (priming) was significant for both accuracy ($p < .001$), and response time ($p = .005$). Primed objects were significantly more accurately identified (86.9%) than baseline objects [72.6%; $F(1,26) = 100.023$, $p < .001$, partial eta squared = .794] or antiprimed objects [71.3%; $F(1,26) = 59.898$, $p < .001$, partial eta squared = .697]. See Figure 10 for a depiction of the results for accuracy. Antiprimed objects (71.3%) did not differ from baseline objects (72.6%) [$F(1,26) = .546$, $p = .467$, partial eta squared = .021]. Response times were significantly longer for antiprimed objects (1200.7 ms) than for baseline objects [1096.3 ms; $F(1,26) = 5.397$, $p = .028$, partial eta squared = .172] or primed objects [1069.7 ms; $F(1,26) = 9.511$, $p = .005$,

partial eta squared = .268]. Response times for primed objects (1069.7 ms) were not significantly shorter than for baseline objects [1096.3 ms; $F(1,26) = .646$, $p = .429$, partial eta squared = .024].

There was a significant main effect of group for response time ($p = .005$), but not accuracy ($p = .139$). Collapsed across priming condition, TBI survivors were significantly slower (M 1267.2 ms, SD 57.0 ms) than control participants (M 1029.8 ms, SD 61.2 ms) to respond [$F(1, 26) = 8.054$, $p = .009$, partial eta squared = .236].

The prime by group interaction was not significant for either accuracy ($p = .935$), or response time ($p = .191$).

Additional Analyses

The primary research question of the present study concerned whether or not masked priming and antipriming could affect metamemory judgments. More specifically, it asked, “does masked priming prior to learning cue-target word-pairs cause changes in metamemory judgments of the likelihood of future recall of the target word?” Thus, insofar as causality can be evaluated, the experiment was designed to evaluate the causal relationship between priming/antipriming, and metamemory judgments. We have seen that in the present study no such significant causal effect was found.

A weaker form of support for a relationship between implicit memory and metamemory would be to explore correlation between the two. For example, is there a

correlation between an individual's "primability" (as measured by their increase in object identification accuracy in the visual antipriming task over their baseline accuracy), and the increase in their relative predictive accuracy for primed word-pairs in the metamemory judgments? To examine this question, a Pearson product moment correlation was performed to compare the ratio of object identification accuracy for primed items over baseline items in the visual antipriming task against the ratio of Goodman-Kruskal gamma correlation for delayed JOLs of primed items over baseline items in the implicit metamemory task. The delayed items were chosen because of the greater stability of gamma correlations in that condition; gamma correlation values for immediate JOLs are extremely variable both within and across participants. Average object identification accuracy in the visual antipriming task for primed items was 85.2% (*SD* 11.8%), vs. 71.7% (*SD* 12.5%) for baseline items. Average gamma correlation for delayed JOLs following presentation of subliminal masked positive prime stimuli was 0.866, vs. 0.795 following baseline (non-primed) stimuli. The Pearson product moment correlation between the ratio of object identification accuracy in the primed condition over baseline condition in the visual antipriming task, and the ratio of gamma correlation for positively primed JOLs over baseline JOLs is 0.334 ($p = .089$). Figure 12 depicts this weak correlation. Since the correlation approaches significance at $p = .089$, it therefore bears further investigation in follow up studies.

Another question of interest is whether or not masked priming differentially affects metamemory as a function of participants' explicit recall skills. That is, are those with poor explicit recall more likely to have their metamemory judgments

improved by priming (or worsened by antipriming), as compared to individuals with high explicit recall? And does any such relationship differ for the two participant groups? This analysis was again conducted for the delayed condition, given its relative stability.

As expected, average JOL ratings of participants are well predicted by their respective average recall accuracies, for all three priming conditions (see Figures 13 and 14). In particular, the standardized regression coefficients respectively for the baseline, prime and antiprime conditions for control participants are 0.702 ($t = 5.033$, sig. 0.000), 0.689 ($t = 4.847$, sig. 0.000), and 0.631 ($t = 4.147$, sig. 0.000). For TBI survivors, the standardized regression coefficients respectively for the baseline, prime and antiprime conditions are 0.371 ($t = 2.114$, sig. 0.044), 0.449 ($t = 2.660$, sig. 0.013), and 0.512 ($t = 3.156$, sig. 0.004). Comparing the regression coefficients across priming conditions within group, the null hypothesis that the regression lines are not different for controls is rejected [$F(2, 78) = 36.632$, $p < .001$, r^2 change = .263] and for TBI survivors as well [$F(2, 84) = 62.109$, $p < .001$, r^2 change = .478].

Thus, priming condition appears to differentially affect JOL ratings across the range of recall accuracy. Antipriming, in particular, appears to increase JOLs for control participants with high recall accuracy, and lower them for those with poor recall, while having an overall suppressive effect across recall accuracy for TBI survivors. One potential implication of this finding is that antipriming improves calibration of JOLs across control participants (even if it does not do so within participants). However, to confirm that this is the case, it is necessary to repeat the

above analysis, but by examining the within-group effect, across the range of accuracy, of priming and antipriming on gamma correlation, particularly with regard to controls.

For control participants, the standardized regression coefficients of gamma correlation vs. recall accuracy, respectively for the baseline, prime and antiprime conditions are .028 ($t = .142$, sig. 0.888), -.009 ($t = -.045$, sig. 0.965), and 0.192 ($t = 0.976$, sig. 0.338). For TBI survivors, the overall conclusion is the same. The standardized regression coefficients of gamma correlation vs. recall accuracy, respectively for the baseline, prime and antiprime conditions are -.014 ($t = -.071$, sig. 0.944), -.088 ($t = -.468$, sig. 0.644), and 0.185 ($t = 0.997$, sig. 0.327). As shown, these values all demonstrate highly insignificant results for both participant groups. Thus, unlike JOL ratings, gamma correlation does not appear to be well predicted by recall accuracy across individuals. This is consistent with previous research (Nelson & Dunlosky, 1991; Kennedy & Yorkston, 2000).

DISCUSSION

At the outset of this dissertation a number of theoretical issues in the extant literature were described. These included issues of representation and process, such as whether the metamemory system takes as input representations of the memory itself (i.e.: the memory trace) vs. associated context, etc., and whether the metamemory process was dissociable from recall. In particular, several researchers have argued that it is access to all or portions of the memory trace upon which metamemory judgments are made (Hart, 1965; Koriat, 1993, 1997; Jameson, Narens, Goldfarb, & Nelson, 1990; Kinoshita, 1997). Others have argued that implicit factors, such as context, degree of familiarity with cues, etc. form the necessary input to the metamemory system (Rajaram, 1993; Reder & Schunn, 1996). Although the present research was not designed with the intention of addressing these issues directly, it is worthwhile to examine whether or not the findings herein may shed some light on these.

With regard to the issue of representation, in this study the paired associate learning task provided little opportunity for participants to rely on any other information to retrieve the target word than the cue word. Furthermore, the degree of cue familiarity was never varied throughout the experiment. Similarly, context was likely to be of little help, since this remained the same throughout. Nevertheless, participants did make metamemory judgments, and insofar as the delayed JOLs were concerned, they demonstrated a high degree of relative predictive accuracy. These findings demonstrate that degree of familiarity with cues and context is not necessary either to form metamemory judgments, or even to make relatively accurate judgments. Moreover, the

distinction between the relatively accurate delayed JOLs and the fairly inaccurate immediate JOLs was not the degree of familiarity with the cues and context, but rather, the degree of recall of the target. For immediate JOLs, target information was still in short term memory and so participants could not assess the degree to which the memory would decay over the ensuing minutes. Inability to assess the degree of target decay resulted in chance level predictive accuracy. Conversely, for delayed JOLs target words had undergone some degree of consolidation or decay and the metamemory system was able to take evaluate this, either in terms of the degree of decay/consolidation, or in terms of the amount or strength of trace information, in order to form the metamemory judgment. Perhaps, then, the input to the metamemory process is the rate or degree of decay of the target trace.

It would seem, then, that this should support the trace access hypothesis. Reder and Schunn (1996) only advanced the argument that the aspects of cognition *which affect strategy choice* are implicit. Since the present results do not address the issue of metacognitive control (strategy choice), the findings here do not contradict Reder and Schunn's claim. This distinction between monitoring and control of memory is important in that it is quite reasonable to suspect that metamemory monitoring may take as input part or all of the memory trace, while metamemory control or strategy choice may have little or nothing to do with the memory trace.

Another aspect of this, however, is that much of the extant literature examining metamemory judgments, has relied on FOKs. As has been pointed out in the introductory chapter, FOKs and JOLs are not well correlated with one another (Leonesio & Nelson, 1990; Schraw, 1995; Maki, 1999; Souchay, Isingrini, Clarys,

Tacconat, 2003), and as such likely reflect differing underlying metamemory processes. It is possible, then, that one reason why priming and antipriming did not affect the metamemory judgments is that the particular metamemory judgment selected for this study (JOL) is different from that upon which much of the extant metamemory research is based (FOK). If such a seemingly small change in metamemory judgment as asking participants to rate the likelihood of future forgetting, rather than future recall, as Finn (2008) made, can so dramatically affect metamemory judgments, then it would seem that metamemory judgments are quite sensitive to task manipulations, and the choice of metamemory measure alone may be responsible for the different findings.

Another challenge to the trace access hypothesis, within the present study itself, is that priming and antipriming did not affect the metamemory judgment or response time to make that judgment. If the metamemory system relies on access to all or part of the memory trace, why did priming and antipriming have no effect on the JOLs? One possibility is that the effect of the subliminal masked stimulus had attenuated over the five or nine seconds (for immediate JOLs) or few minutes (for delayed JOLs) intervening between study of the word-pair and making the metamemory judgment. Yet, the fact that the subliminally presented antiprime stimuli from the final trial block of the paired associate learning task influenced the old/new judgment of the validation task undermines this suggestion. Clearly, the metamemory system is not so simple as only involving consulting the degree of target decay (i.e.: decreases in trace strength) in order to produce a judgment. Further research will need to investigate this.

The other issue related to representation is process. A particularly important question was whether or not the metamemory process relied on, or was dissociable

from, explicit recall. The present study found that mean JOL ratings were well correlated to recall accuracy between subjects, so that higher explicit recall skill predict higher JOL ratings. These findings are more in line with Jang and Nelson's (1995) study, showing that JOLs demonstrated the same functional relationship to explicit recall irrespective of implicit manipulations.

Turning to the most central research question of the present study, we consider whether or not subliminal masked priming and antipriming of cue-target word-pairs would influence participants' metamemory judgments of the word-pairs. The clinical purpose underlying this question is to determine whether or not individuals living with TBI are able to make use of priming to facilitate metamemory – an area commonly of deficit in this population. As we review the findings that shed light on these questions, we will also examine related questions, such as whether or not masked priming in the metamemory task will affect explicit recall.

The main purpose of the visual object identification task was to provide an implicit memory benchmark of priming and antipriming against which to compare any findings of implicit metamemory; that is, we can examine whether or not there is a relationship between the degree of priming and antipriming in the visual object identification task, and the degree of priming and antipriming in measures of metamemory in the paired associate learning task. Additionally, examination of visual antipriming in the TBI population may shed light on fundamental aspects of perceptual implicit memory, perhaps helping to differentiate between the hypotheses of cognitive slowing and explicit memory contamination to explain the visual object priming

differences found between controls and individuals with amnesia by Marsolek et al. (2006).

Finally, there are a number of research questions that span the two experimental tasks. An important question is whether masked priming differentially affects metamemory as a function of participants' explicit recall skills? For example, will any priming effects on metamemory be stronger in those with poor recall, and weaker in those with very good recall? In any such relationships, are there differences between TBI survivors and neurologically normal controls? These and other issues will be discussed in the following sections.

Findings and interpretations

Validating the assumptions

Before the research questions associated with the implicit metamemory task can be examined, we must first be assured that the important assumptions underlying the task are valid. In particular, one assumption was that the subliminal priming was indeed subliminal. That is, were participants aware of the briefly presented and masked stimuli? The purpose of the validation task was to examine this question. As hypothesized and as reported in the results chapter, antiprime stimuli and new items were both judged as "old" significantly less often than target study items, with no group differences in this pattern for percent old. Collapsed across the groups, primed target stimuli were judged "old" 88.5% of the time, new items 25.8% of the time, and antiprime items 16.5% of the time. As for response time, there was a main effect of

group, with the TBI group being significantly slower (359.2 ms) than controls in the old/new judgment task. With antiprime items judged as “old” less often than items that truly were new, there is evidence that the subliminal masked items were indeed subliminal. Furthermore, all but one participant denied seeing words during the masked presentation, and the one participant who claimed to have seen words stated that this occurred less than one percent of the time.

One very interesting and unexpected result was that antiprime stimuli had significantly lower percent old judgments, and faster response times, than new items. There are several possible explanations for this finding. One possibility is that this reflects negative priming, which has traditionally been viewed as an attentional inhibitory process (Tipper, 2001). The idea here is that when a competing stimulus distracts attention from the stimulus to be attended, the neural representation of the distractor must be inhibited in order to succeed at the desired task. Later, if that inhibited item becomes the actual target of the task, then the persisting effect of the former inhibition causes reduced performance for that item. As an example, if the task is to name black ink line drawings of objects, in the presence of distracting overlaid line drawings in grey ink, then the object names for the items in grey ink must be inhibited in order to name the drawings in black. Later, if an item that had formerly appeared as a distractor in grey ink is presented in black ink as a test item, the naming accuracy and/or response time will be adversely affected by the previous inhibition of that object name. One problem for such a hypothesis in the present task is that even though antiprime items had decreased percent old judgments compared to new items (which would support the idea that they had been inhibited), the response times for these

antiprime items were *faster*, not slower than the new items. This should not be the case if an inhibitory process were suppressing their representation.

The antipriming hypothesis also has difficulties explaining the present findings. Here the argument would be that the antiprime stimuli have been weakened and therefore antiprime items were identified as “old” less often than the new items. The antipriming hypothesis might account for the faster response time by positing that the old/new judgment process takes as input some aspect of the representation, such as its strength, or the amount of processing needed to reconstitute the item for the decision task, etc. If a representation is particularly weak, a search and retrieval process may quickly come up negative and feed that information to the old/new judgment process, resulting in a more rapid response time for antiprimed items.

However, one problem with the concept of weakened representations for antiprime stimuli is that the subliminal stimuli were presented *prior* to the word-pair, not after. Therefore, in order to argue that the antiprime stimuli suffered representational weakening by the ensuing word-pair, one must claim that processing of subliminal stimuli continues during study of the subsequent word-pair, and that studying the word-pair interferes with the normal resolution of processing of the (preceding) antiprime stimuli. Such an argument is speculative. However, even if there were retroactive weakening of the subliminally presented antiprime items by the word-pairs, the antiprime items had still been processed more recently than the new items (which had not been previously used in the experiment at all), and should therefore suffer less weakening than the new items by the word-pairs that were studied.

Finally, antipriming theory posits a specific neural mechanism; for antipriming to be observed there must be some degree of representational overlap between the antiprime stimulus and the target word in the word-pair. However, in this experiment, there was no attempt to control the degree of similarity between the antiprime items and target items. Furthermore, the antiprime and new items were randomly selected from the larger corpus of words during list construction. Therefore, mean similarities between the antiprime items and the cue-target words-pairs, and between the similarity of the new items in the validation task to the cue-target word-pairs, are likely to average out.

Although the present findings of significant differences in both old/new judgments and response times for antiprime vs. new items in the validation task are difficult to explain, they are indeed intriguing and bear further experimentation to explore and specify their underlying mechanisms.

Returning to the assumptions of the implicit metamemory task, another assumption was that the paired associate learning task must remain valid despite introduction of masked priming. Whereas the implicit metamemory task involved the insertion of a standard three-field, forward masking paradigm (Foster, 1984) into the traditional judgment of learning task as implemented by Nelson and Dunlosky (1991), we must first determine whether or not the introduction of masked priming compromised the well established findings from previous metamemory research utilizing this task and metamemory measure.

In particular, there are several findings from previous research that were replicated. The first is the so-called “delayed JOL effect”, discovered by Nelson and

Dunlosky (1991). Nelson and Dunlosky found that individuals' relative predictive accuracy, as measured by the Goodman-Kruskal gamma correlation between recall accuracy and judgment of learning, is poor for those JOLs made immediately after studying the respective word-pair (e.g.: 0.38), but extremely high when made after a delay of approximately a minute from the studying of the cue-target word-pair for which the judgment is made (e.g.: 0.90). Secondly, Kennedy and Yorkston (2000) later replicated this in neurologically normal controls and, importantly, observed it in individuals with TBI, even in TBI survivors with documented frontal lobe injury (Kennedy & Yorkston, 2004). Thirdly, Kennedy and Yorkston (2000) also found that participants with TBI demonstrated weaker cued recall skills in the JOL paired associate learning task, as compared with neurologically normal control participants.

As shown in the results chapter, the delayed JOL effect was robustly replicated in the present study for both participant groups ($p < .001$). Gamma correlation for delayed JOLs (collapsed across groups) averaged 0.84 ($SD .03$), whereas gamma correlation averaged 0.39 ($SD .06$) for immediate JOLs. As for cued recall accuracy, results in the present study approached significance for the main effect of participant group ($p = .067$), with brain injured participants having mean proportion recalled of .30 ($SD .05$) and controls having mean proportion recalled of .44 ($SD .05$). The primary difference between the implementation of the present research, and Kennedy and Yorkston (2000) was that here participants were not allowed to pre-study each list prior to the study and learning trials. As a result, the participant groups in the present study were given significantly reduced exposure to the word-pairs and this is reflected in the reduction in recall accuracy for both groups as compared to Kennedy and Yorkston

[wherein mean proportion recalled for brain injured participants was .43 (*SD* .29), and .58 (*SD* .22) for controls]. If Kennedy and Yorkston's greater amount of study is the reason they obtained a significant between-groups difference in recall, controls would have to have benefitted more from restudy than TBI survivors. Thus, according to that hypothesis, compared to having only one study opportunity (as in the present study), having a chance to study and then restudy would increase both groups' recall, but more so for controls. This hypothesis bears future analysis.

Finally, another finding from previous research that is replicated here is that of cognitive slowing in the brain injured population (see, for example, the meta-analyses of Ferraro, 1996, and Bashore & Ridderinkhof, 2002). Thus, we expect that the response times will be slower for the TBI survivor group than for controls. Indeed, there was a significant main effect of group for both of the response time measures in the implicit metamemory task; TBI survivors took approximately 1.6 seconds longer than controls to make JOLs ($p = .002$), and approximately 1.6 seconds longer to make recall responses ($p = .022$). Additionally, there was a borderline significant difference in JOL response times for the group by JOL timing interaction for JOLs ($p = .067$). As reported above, the control group's response times for JOLs increased by approximately one second for delayed JOLs vs. immediate JOLs, but the TBI survivor group's response times for JOLs increased by more than two seconds for delayed JOLs vs. immediate JOLs. These findings provide much evidence for slowed response times in the brain injured participants. Furthermore, all of the above findings, particularly with regard to replicating the delayed JOL effect, support that the introduction of the masked priming paradigm did not compromise the paired associate learning task.

Principle findings

The following discussion will be organized around the research questions posed in the first chapter, and following the same order.

Do manipulations of masked priming condition affect measures related to metamemory judgments (JOL ratings, relative predictive accuracy, or JOL response times)?

It was expected that JOL ratings would be increased for primed items and decreased for antiprimed items, relative to baseline, and that JOL response times would be faster for primed items and slower for antiprimed items, relative to the baseline condition. However, these effects were expected only for the immediate JOL timing condition, not significantly in the delayed condition; thus, an interaction effect was expected for prime condition and JOL timing condition. Again, this expectation of interaction of priming and JOL timing was based on historical data showing that the effect of subliminal masked priming attenuates within seconds of stimulus presentation (Forster & Davis, 1984; Forster, Booker, Schacter & Davis, 1990; Squire, 1994).

From the results, we see that there was no main effect of priming on JOL ratings, relative predictive accuracy (as measured by Goodman-Kruskal gamma correlation), or response times for making JOLs. However, there was a borderline significant interaction effect that supported the hypothesis; for JOL ratings the prime by JOL timing interaction approached significance ($p = .060$). Immediate JOLs were higher for the primed items ($M 46.432$) than for the baseline ($M 44.390$) or antiprimed ($M 44.679$) items. Given the variability and relatively small sample size of this study, these results are intriguing as they are in line with the hypothesis. If this finding

becomes significant with expansion of the sample size, it may provide evidence for the influence of subliminal priming on metamemory judgments. However, the absence of significant findings for delayed JOLs implies that any such effects of subliminal priming would be fairly short lived.

Based on the above findings, there does not appear to be sufficient evidence at the present time to conclude that there were significant effects of subliminal masked priming on measures associated with metamemory judgments. However, additional studies, or perhaps simply expanding the number of participants in the present study, will have to be undertaken to explore the borderline significant findings.

Before leaving this research question, however, there was another finding of interest that may provide another window into implicit metamemory. In order to address one of the findings associated with a research question below, difference scores were computed between JOLs and recall accuracy in this task. The difference score is an index of absolute confidence (Kennedy, 2001; Kennedy & Yorkston, 2004). For example, if a participant's prediction of the likelihood of future recall averages 63.4% for a particular combination of conditions (e.g.: for all immediate JOLs which were antiprimed), but that participant's actual recall accuracy for that combination of conditions averaged 27.9%, then they would have a difference score of +35.5%, and would be considered overconfident. Negative difference scores, conversely, reflect underconfidence. Absolute confidence is distinguished from relative confidence, of which the Goodman-Kruskal gamma correlation would be an example. For simplicity, hereafter absolute confidence will simply be denoted as confidence.

Repeated measures ANOVA of difference scores (Table 19) shows significant main effects of priming ($p = .007$), JOL timing ($p < .001$), and group ($p < .041$). The latter two findings will be discussed below under the research question regarding group differences. None of the interactions was significant. Collapsed across groups and JOL timing condition, participants were most overconfident in the baseline condition ($M 7.864, SD 3.347$), followed by the prime condition ($M 6.739, SD 3.328$) and least overconfident for the antiprimed condition ($M 3.088, SD 3.070$). Pairwise comparison shows that at the $\alpha = .05$ level, antipriming significantly decreased confidence relative to baseline (mean difference = 4.776. $SE = 1.465$, sig. = .009), but priming was not significantly lower than baseline (mean difference = 1.125. $SE = 2.065$, sig. = 1.000). Thus, antipriming significantly reduces participants' overconfidence (see Figure 9). Although the prime by group interaction was not significant ($p = .224$), antipriming reduced controls' difference score from 0.535 ($SE 4.814$) to -1.725 ($SE 4.416$), while it reduced TBI survivors' difference score from 15.193 ($SE 4.651$) to 7.901 ($SE 4.266$). Thus, the antipriming reduction in overconfidence was quite large in magnitude for the TBI survivor group.

Confidence, as operationally defined by difference scores, is a difficult to interpret measure of metamemory. Since it is a derived measure, care must be taken to ensure that apparent changes in confidence do not simply reflect changes in recall accuracy alone. In fact, examining the present results for confidence, it appears that for TBI survivors, the significant reduction in overconfidence in the antiprime condition is largely due to an increase in recall accuracy for that condition. More specifically, the increase in recall accuracy in the antiprime condition accounts for approximately two-

thirds of the difference score, while decreased JOL accounts for the remaining one-third. Together, these conspire to create a highly significant drop in overconfidence in the TBI survivor group, for antiprimed items.

Another difficulty in interpreting the confidence finding as a reflection of metamemory is that participants were not asked to rate the degree of over- or underconfidence they may be displaying in their predictions of future recall. Rather, their confidence was epiphenomenal, and they may or may not have been aware of whether they were overconfident, underconfident, or realistic. In this regard, like long term priming (the evidence of which occurs irrespective of participants' conscious awareness) the phenomenon of confidence may be implicit in nature. As such, it may be argued that confidence in one's abilities may be an example of implicit metamemory: reflecting an aspect of a participant's belief in their abilities with or without that person consciously or intentionally focusing on constructing those beliefs. If confidence is an example of implicit metamemory, then the findings that antipriming significantly reduces participants' (particularly TBI survivors') overconfidence provide evidence that subliminal masked antipriming can indeed affect a type of metamemory process. However, this requires further investigation, particularly in terms of dealing with the potential confound that confidence may reflect disproportionate changes in recall.

Will masked priming in the metamemory task affect explicit recall?

The hypothesis was that recall accuracy would be improved for primed items relative to baseline items, and worsened for antiprimed items. As seen from the

analyses in the results chapter, there was no main effect of priming on recall accuracy. However, for the interaction of priming and JOL timing, collapsed across groups, recall accuracy was significantly higher ($p = .025$) for baseline and antiprimed items that had received delayed JOLs (baseline: $M = .415$, $SE = .046$; antiprime: $M = .427$, $SE = .042$) than immediate JOLs (baseline: $M = .300$, $SE = .036$; antiprime: $M = .358$, $SE = .040$); this was not the case for items that had been primed. Put another way, we may say that relative to baseline (and antiprime) items, recall accuracy was decreased for primed items for which delayed JOLs were made. The implication here is that a masked repetition prime of target items, combined with a lack of immediate JOL (and any additional processing of the word-pair that this may cause) results in decreased recall accuracy at test. One explanation might be that the baseline and antiprime items are more salient, due to their difference from the target. This increased salience may cause increased attention to the ensuing target, resulting in greater encoding. One could then argue that this effect was not observed for those items with immediate JOLs because the requirement of making a JOL immediately after encoding the word-pair interfered with the consolidation process. However, such an argument is weak since the differences in recall accuracy among the priming conditions favors both prime and antiprime items over baseline, though this is a non-significant difference.

Overall, while there may be some theoretical rationale for why *all* items with delayed JOLs should have higher recall accuracy (see below), why this does not occur for primed items is unclear. Nevertheless, in terms of the research question as to whether or not priming affects explicit recall, the answer appears to be “yes,” for recall accuracy in the interaction of priming with JOL timing.

For recall accuracy, the three way interaction of prime by JOL timing by group approached significance ($p = .081$). For the control group, recall accuracy was greater for primed and antiprimed items than baseline items, but only for items that received immediate JOLs (baseline: $M = .345$, $SE = .052$; prime: $M = .443$, $SE = .053$; antiprime: $M = .423$, $SE = .057$). Many participants did informally report that they often applied the time for making an immediate JOL toward coming up with a better word association or mnemonic, rather than actually deliberating on the JOL. If this is the case, then perhaps increased accuracy for items that received immediate JOLs simply reflects the extra time spent encoding. Furthermore, this may differentially affect controls because they may be better able to make better use of that “borrowed” time. Finally, perhaps an argument similar to the one above, that salience (in this case that a real word appears, rather than a row of uninteresting x-s) flags the attentional system and causes improved encoding. All of this is, however, speculation regarding a borderline significant finding. Further research will need to examine whether this finding becomes significant, and if so, what the underlying mechanism is.

Turning to recall response times, no hypotheses were advanced regarding this dependent measure because it was believed that accuracy rates would be so low that there would be too few correct responses upon which to compute stable mean recall response times for all combinations of conditions. The low overall recall for the two groups did result in several missing cases, and many of the combinations of conditions that were not missing for a particular participant were based on only one or a few recall responses. Thus, this measure is not deemed particularly stable and the relevant results

should be viewed critically. Nevertheless, it should be noted that there were neither main effects nor interaction effects involving priming on recall response times.

Finally, there is another finding of interest in the present study, but which is unrelated to the question of priming effects. This will be discussed here given its relevance to recall in the implicit metamemory task. As mentioned above, for both participant groups, items for which delayed JOLs were made were more accurately and rapidly recalled than items for which immediate JOLs were made (accuracy mean difference = .070, $SE = .016$, $p < .001$; recall response time mean difference = 1146.9 ms, $SE = 185.468$ ms, $p < .001$). The recall accuracy finding is in contrast to the findings of Nelson and Dunlosky (1991), Kennedy and Yorkston (2000, 2004), or Kennedy, Carney and Peters (2003) who found no difference in recall accuracy between items for which immediate vs. delayed JOLs were solicited.

However, commenting on Nelson and Dunlosky's (1991) study, Spellman and Bjork (1992) argued that "one strategy for making a delayed JOL is to use the presented stimulus as a cue to try to recall the response item... successful covert recall during the JOL task will in turn increase the likelihood that the subject will successfully recall that item on the later overt recall test" (p. 315). Thus, for immediate JOLs, the target word is still in short term memory since the word-pair had been on the computer screen immediately prior to the JOL request; therefore, it can be argued that there was no re-encoding or re-instantiating of the memory for the target word. However, delayed JOLs occur long enough after presentation of the word-pair that memory of the target will have decayed to some degree. When the delayed JOL is then solicited, a covert recall process may be made, as suggested by Spellman and Bjork. If this hypothesis is true,

we would expect to see longer JOL response times for delayed JOLs. Indeed, in the present study, JOL response times for immediate JOLs took an average of 2.70 s, while response times for delayed JOLs took an average of 4.25 s. During this additional 1.55s, participants may be able to strengthen the long term memory representation of the cue-target words pairs that were successfully retrieved, making them less susceptible to memory loss by the time of the recall test and thus resulting in higher recall accuracy for those items for which delayed JOLs were made.

However, this hypothesis does not explain why Nelson and Dunlosky, Kennedy and Yorkston, and Kennedy, Carney, and Peters did not obtain the same findings. One possibility is that the difference in recall accuracy between JOL timing conditions found in the present study is muted by the study-restudy paradigm. It may be that the second opportunity to study the word-pairs increases recall accuracy overall, to the point where the difference between JOL timing conditions that would otherwise have been observed is washed out.

Will there be any differences between TBI survivors and neurologically normal controls for any of these dependent measures?

Kennedy and Yorkston (2000) found that despite TBI survivors being given more study time than controls (9 s and 5 s respectively), there was still a main effect of group, with TBI survivors demonstrating lower recall accuracy. Based on those data, it was expected that TBI survivors would exhibit poorer explicit recall here as well, even though in contrast to Kennedy and Yorkston there was no restudy phase in the present

research. Also, due to the cognitive slowing commonly observed in TBI survivors, slower response times were expected for the TBI survivor group than for controls.

For recall accuracy, the main effect of group approached significance ($p = .067$), with controls being more accurate on average than TBI survivors. Additionally, for spoken responses during recall testing, TBI survivors took approximately five seconds longer than controls ($p = .043$). While the trend toward reduced accuracy among TBI survivors may simply reflect that their explicit recall is only mildly impaired, there was a very significant slowing of their recall responses. It is possible that the TBI survivors were taking more time to respond during recall testing, and as a result were able to recall more items correctly than they would have otherwise, thus bringing their recall accuracy closer to that of controls. If this was the case, then the TBI survivors were sacrificing speed for accuracy and this would reflect a “speed-accuracy trade off”.

As for JOL response time, TBI survivors took significantly longer than controls ($p = .002$). The group difference is further illuminated by examining the interaction effects with JOL timing condition; the control group’s response times for JOLs increased by approximately one second for delayed JOLs as compared to immediate JOLs, however, the TBI survivor group’s response times for JOLs increased by over two seconds for delayed vs. immediate JOLs. Both participant groups took longer to make delayed JOLs, and this may reflect the additional time involved in making covert retrieval attempts since the target words are no longer in short term memory.

Furthermore, that the TBI survivor group was slower than controls is not surprising, given their well-documented overall cognitive slowing. Together, these factors likely

account for the significant group by JOL timing interaction in JOL response time and therefore the result is not surprising.

Another finding of interest is that for JOL ratings, the group by JOL timing interaction approached, but did not achieve, significance ($p = .085$). Control participants demonstrated virtually no difference in JOL timing condition, averaging 43.8 for immediate JOLs and 43.1 for delayed JOLs. TBI survivors, however, averaged 46.5 for immediate JOLs and 38.8 for delayed JOLs. This may be due to TBI survivors overestimating their recall when they made JOLs immediately after studying the word-pairs. To better understand the significance of these findings, it is helpful to examine difference scores between prediction of recall and actual recall.

As discussed above, difference scores were computed comparing JOLs to recall accuracy for each condition combination. There was a significant main effect of group. Controls were neither very overconfident nor underconfident, with a mean difference score of -0.718 ($SE 4.434$), while TBI survivors were significantly overconfident ($M 12.513$, $SE 4.283$). Since these numbers are the result of collapsing across priming conditions, it should be noted that in the absence of priming or antipriming (i.e.: for the baseline condition), TBI survivors were even more overconfident ($M 15.193$, $SE 4.651$), while controls were still relatively realistic ($M .535$, $SE 4.814$). These results are somewhat similar to those of Kennedy and Yorkston (2004), who examined differences between TBI groups with and without documented frontal lobe injury. They found that TBI survivors without documented frontal lobe injury were significantly overconfident ($M 19.05$), as well as significantly more confident than TBI survivors with documented frontal lobe injury ($M -8.99$). Of the present TBI survivor group, five out of the 15

individuals included in the analysis had documented frontal lobe injury. Thus, the overconfidence of the present TBI group, with its preponderance of individuals without documented frontal brain injury, is consistent with the finding of overconfidence of the non-frontal brain injury group of Kennedy and Yorkston.

Will masked priming differentially affect metamemory as a function of participants' explicit recall skills?

The hypothesis was that priming effects should be stronger in those with poor recall, and weaker in those with very good recall. As shown in the results chapter, priming appears to differentially affect JOL ratings across the range of recall accuracy (see Figures 13 and 14). Antipriming across the range of recall accuracy appears to increase JOLs for control participants with high recall accuracy, and lower them for those with poor recall, while having an overall uniformly depressive effect across recall accuracy for TBI survivors.

To check whether or not the finding that antipriming improves calibration of JOLs across control participants (even if it does not do so within participants), a within-group analysis of regression lines for average JOL scores across the range of recall accuracy was conducted. While Kennedy and Yorkston (2000) have shown that JOL ratings and recall accuracies are not correlated either in controls or TBI survivors, theirs was a within-subjects analysis rather than an examination of gamma correlation trend across the range of mean recall accuracies of all participants. The results of the present study show that unlike JOL ratings, gamma correlation does not appear to be well predicted by recall accuracy across controls. So, even though JOL ratings were

increased in those with poor recall, and decreased in those with good recall, this did not translate into improved calibration for across the populations. Also, this finding may reflect what Kennedy and Yorkston (2004) describe in the context of TBI survivors, referring to previous research in health controls, as the “fan effect”. Here, the idea is that there is an inverse relationship between working memory and JOL ratings, whereby the more items that one can hold in working memory, the lower the per item probability of future recall (e.g.: if one can hold four items in memory, one may predict a 25% chance of future recall of any one item, whereas if one can hold only two items in memory, one may predict a 50% chance of future recall of one of those items). Thus, those with better recall may actually discount, and thus underestimate, their recall – resulting in decreased JOL ratings for those with high recall.

Additionally, there may also be floor and ceiling effects. That is, if someone has very high recall accuracy (e.g.: > 90%), there may be a tendency to make ratings lower than 90% simply because there is a larger range of values below their performance level than above thus effectively skewing their JOL ratings downward. Conversely, someone with poor recall may experience the mirror image effects, skewing their JOL ratings upward.

Will TBI survivors show both priming and antipriming effects, for visual object identification accuracy and response times?

The hypothesis was that TBI survivors would show both priming and antipriming effects in both dependent measures, and that, overall, accuracy and response times for TBI survivors were expected to be worse than for controls.

Although controls and TBI survivors did demonstrate both priming and antipriming effects, both effects were not present for both dependent measures.

As with Marsolek et al. (2006), a robust priming effect for object identification accuracy was observed for both participant groups ($p < .001$). However, in the present study, the decreased identification accuracy for antiprimed items was not statistically significant for either group ($p = .467$), although significant antipriming was observed in response times (see below). Interestingly, both participant groups in the present study showed overall higher accuracy rates (baseline objects: 72.6%; primed objects: 86.9%; antiprimed objects: 71.3%) than was obtained by Marsolek et al. for their older adults and amnesic participants (baseline objects: 40.2%; primed objects: 53.6%; antiprimed objects: 33.5%).

There were two main differences between the implementation of the task in the present study and experiment four of Marsolek et al.'s (2006) procedure. Firstly, here participants were given decreased exposure time for visual object presentation (15 ms, vs. 33 ms used by Marsolek et al.). Nevertheless, it should have resulted in *decreased* accuracy rates for the present study relative to Marsolek et al. However, the second procedural difference of the present study was that participant responses were self-paced, whereas Marsolek et al. required participants to respond within two seconds for object identification, after which the next item was presented (R. G. Deason, personal communication, October 29, 2007). It may be that Marsolek et al.'s participants were unable to identify many of the items within that time window and that this resulted in overall decreased accuracy across priming condition for both of their groups.

Furthermore, given the cognitive slowing demonstrated by the individuals with

amnesia, their accuracy rates may have been differentially affected by the two second time limit more so than for controls. It should be noted that in the present study, this deviation from Marsolek et al.'s procedure was taken precisely out of concern for the anticipated cognitive slowing of the TBI survivors in this study and whether or not some of them may demonstrate floor effects.

One way to shed some light on the effect of this procedural deviation in the present study is simply to reanalyze the data by not counting as correct any response provided after two seconds. This was done, and the object identification accuracies for baseline, prime and antiprime for the control group were found to be respectively 71.4%, 67.9%, and 84.3%. For TBI survivors, the corresponding accuracies were 61.8%, 56.8%, and 74.0%. See Figure 11 for a graphical depiction of these results. Clearly, recall accuracies for both groups have dropped substantially, although not down to the level of the Marsolek et al. (2006) study. Interestingly, using this truncated data set, repeated measures ANOVA shows that antipriming becomes significant. Collapsed across groups, antiprime items had 62.3% accuracy as compared to the baseline accuracy of 66.6% [$F(1, 28) = 4.879$, $p = .036$, partial eta squared = 0.148]. The prime by group interaction for accuracy was still not significant ($p = 0.791$). This procedural deviation from Marsolek et al. may thus be the primary reason no antipriming effect in object identification accuracy was found in the present study. Had participants been given a two second limit in the present study, it is possible that they may have adapted to some degree and increased the speed of their responses with possible consequences to their accuracy. Therefore, it cannot be assumed that the above

reanalysis accurately reflects what would have happened had the two second limit been imposed. Nevertheless, the above reanalysis is intriguing.

In terms of the basic research question as to whether or not antipriming is seen in the present study, as noted above, no significant effect was observed for accuracy (without removal of items with longer than two second response times). However, there was a significant effect of antipriming observed for response time. Response times were longer for antiprimed objects (1200.65 ms) than for baseline objects (1096.34 ms) by a difference of 103.4 ms ($p = .028$), and longer than primed objects (1069.67 ms) by a difference of 131.0 ms ($p = .005$). Interestingly, response times for primed objects were not significantly shorter than for baseline objects, with a difference of only 27.7 ms ($p = .429$). Thus, we see that antipriming is observed in the present study. Furthermore, this effect is quite large (over 100 ms), whereas there is no significant response time benefit to priming. As a comparison, it should be noted that Marsolek et al. (2006) found that the main effect of test presentation condition on response times for older controls and individuals with amnesia was not significant. However, for younger adults, test presentation was borderline significant ($p < 0.06$), with primed items correctly identified 32 ms faster than baseline items, and antiprimed items correctly identified 28 ms slower than baseline items. Marsolek et al. (2009) did observe significantly faster response times for primed items vs. baseline (26 ms), and slower response times for antiprimed vs. baseline items (17 ms). Although these values were significant, they are generally on the same order as the degree of priming benefit in the present study. However, given the much greater variability of response times of the TBI survivor population in the present study, here the results for priming did not

reach significance. The main points, however, are that there was approximately the same degree of speed improvement in priming as with the studies of Marsolek and colleagues, and that the degree of slowing due to antipriming was substantially larger. However, with a larger sample size for participant groups, both in the present research and with Marsolek et al. (2006), it is likely that clearer and more consistent results would be obtained.

Given the above findings, TBI survivors do indeed show both priming and antipriming effects. However, as with controls in the present study, without filtering responses for those provided within two seconds of stimulus presentation, the priming was measured only as increased accuracy rates relative to baseline (with no difference in response times), and the antipriming effects were only measured as slowing of response times (with no difference in accuracy rates). In contrast, Marsolek et al. (2006) found both priming and antipriming effects in accuracy, and no priming or antipriming effects in response time. Therefore, the magnitudes of the antipriming findings in the present study cannot be compared to those of Marsolek et al. (2006). As for the magnitude of priming, in the present study, controls experienced a 13.7% increase in accuracy for primed items over baseline, while TBI survivors experienced a 14.9% increase in accuracy for primed items over baseline items. This is in contrast to Marsolek et al.'s findings of an 18.5% increase in accuracy of primed vs. baseline items for controls, and only a 7.8% increase in accuracy for amnesic participants. This difference between the findings for TBI survivors vs. individuals with amnesia will be discussed in greater detail below.

How do the magnitudes of priming and antipriming in TBI survivors compare with those reported by Marsolek et al. (2006) for amnesic individuals?

Given the relatively mild degree of impairment for the present TBI survivor group, it was hypothesized that individuals with TBI in the present study would demonstrate much smaller impairment to overall object identification accuracy and response times (compared to the control group) than was demonstrated by the amnesic participants of Marsolek et al. (2006). Furthermore, the magnitude of priming increase, relative to baseline, was expected to be greater than experienced by the amnesic participants, but less than controls. Marsolek et al.'s amnesic participants showed a significant decrease in accuracy for antiprimed items. However, given the smaller magnitude of antipriming than for priming, for Marsolek et al.'s control group, and the expected heterogeneity and variability of TBI survivors, it was hypothesized that in the present study the reduction in magnitude of accuracy for antiprimed items would not achieve significance.

Marsolek et al. (2006) found a group difference in both identification accuracy and response time between the control and amnesic participants, with amnesic participants significantly slower (1133 ms) and less accurate (33.7%) in identifying objects than controls (response time = 998 ms, accuracy = 50.2%). In the present study, as reported above, the group difference in accuracy for the TBI survivor group relative to the control group did not reach significance ($p = .139$). Thus, as expected, TBI survivors did demonstrate lower accuracy than controls and greater accuracy than Marsolek et al.'s amnesic participants. However, the decreased accuracy of TBI survivors compared with controls did not reach significance in the present study. As for

response time, as with Marsolek et al., in the present study there was a significant group difference ($p = .005$), with TBI survivors averaging 1267.2 ms to identify objects, as compared with controls who averaged 1029.8 ms. As reported in the results chapter, and similar to the Marsolek et al.'s results for control vs. amnesic participants, the prime by group interaction for response time was not significant ($p = 0.191$).

To explain why control participants experienced an 18.5% accuracy benefit to priming, while amnesic participants only saw a 7.8% gain, Marsolek et al. (2006) advanced two possible hypotheses. The first hypothesis was of cognitive slowing. Marsolek et al. suggested that priming is the result of one trial's worth of synaptic modifications, while antipriming for a particular object is the cumulative result of many trials worth of synaptic modifications due to the fact that different visual object representations are only partially overlapping. They argued that when cognitive systems are slowed, they tend to retain general information (which Marsolek et al. associated with the many trials' worth of weight changes in antipriming) better than relatively specific information (i.e.: the single trial's weight changes associated with priming). Thus, since individuals with amnesia demonstrate significant slowing of response times compared with controls, the cognitive slowing hypothesis would predict that they would demonstrate a lower priming benefit than controls, but very little difference in antipriming.

However, a second hypothesis can also explain the difference in magnitude of priming between Marsolek et al.'s participant groups, and that is explicit memory contamination of priming in the control group. Since the visual object identification task is supraliminal, participants are explicitly aware of seeing the primed items. Thus,

if some of the priming benefit is simply due to participants having explicit recall of those items, then amnesic individuals should demonstrate less of a priming benefit, since their explicit recall is impaired. This would not occur for antipriming, since the antiprimed items were not previously viewed in the experiment and so could not manifest explicit memory contamination.

Marsolek et al. (2006) were unable to resolve the competing explanations for the decreased priming accuracy in their TBI survivor group. However, in the present study, the TBI population exhibits cognitive slowing, but does not have severely impaired explicit memory compared to the control group as did Marsolek et al.'s amnesic group. As described in the participants section of the methods chapter, standard scores for cued recall in the California Verbal Learning Test – II were not significantly different for the TBI survivor and control groups. Thus, for the TBI group, the cognitive slowing hypothesis would predict that the magnitude of priming would be reduced compared to controls. However, the explicit memory contamination hypothesis would predict that the TBI survivors' magnitude of priming would be similar to, or non-significantly different from, that of controls. Indeed, with a 13.7% accuracy increase of primed items over baseline for controls, and 14.9% for TBI survivors, there is no significant difference in priming benefit between the two groups. This lends some evidence in support of the explicit memory contamination hypothesis.

Is there a relationship between the degree of priming and antipriming in the visual object recognition task, and the degree of priming and antipriming in the paired associate learning task?

The hypothesis was that a correlation would be found. To examine this question the ratio of object identification accuracy in the primed condition to accuracy in the baseline condition in the visual object identification task was compared to the ratio of gamma correlation for positively primed items over baseline items in the paired associate learning task. Figure 12 depicts this relationship. Visually, there appears to be a trend, and the Pearson product moment correlation between the two ratios ($r = 0.334$) confirms that while the correlation does not reach significance ($p = .089$), it does demonstrate a trend. Thus, it is possible that those participants who demonstrate stronger long term priming benefit in the visual object identification task will also show a stronger subliminal masked priming benefit in relative predictive accuracy in the metamemory task.

While this trend may seem at odds with the fact that there was no evidence for such a relationship *within* subjects, it is possible that collapsing data across participants with different “primability” hides the possibility that individuals who respond differently to priming may have fundamental differences in their neural responses. Priming in the visual object identification task is believed to be caused by single trial synaptic changes that strengthen representations. Perhaps for those individuals who demonstrate particularly large increases in accuracy for primed items, their neural systems produce particularly robust synaptic modifications from single trials. Conversely, perhaps there are weaker synaptic changes in individuals with smaller magnitudes of accuracy increase for primed items. If this is the case, it is possible that the more robust synaptic modification seen in more “primable” individuals may not be restricted to the ventral visual stream. Perhaps the stronger synaptic modifications are a

property of their neural systems throughout the neocortex, and this manifests as increased primability of their relative predictive accuracy.

It should also be noted that the two methods of priming in the tasks are different, with long term repetition priming in the visual object identification task and subliminal masked priming in the paired associate learning task. It would be interesting to examine whether a stronger relationship between “primability” in one task vs. the other would be observable if both tasks employed the same method of priming.

Summary of findings

In summary, in terms of the principle research question, priming was not observed in this study for JOL ratings, relative predictive accuracy, or JOL response times. Weak evidence that immediate JOLs were higher for the primed items than baseline or antiprimed items was noted. Regarding difference scores, for delayed JOLs controls were neither overconfident nor underconfident, but TBI survivors were significantly overconfident; this overconfidence in TBI survivors was strongly reduced by delaying JOL responses, and also by antipriming; however, this finding must be further examined to neutralize the effects of increased recall for antiprimed items.

Priming and antipriming also appear to differentially affect JOL ratings across the range of recall accuracy. Relative to baseline, antipriming decreases JOLs for controls with poor recall, and increases it for those with high recall, while priming has the opposite effect. For TBI survivors, antipriming depresses JOL ratings across the range of recall accuracy, while priming has no effect for those with poor recall but progressively depresses JOL ratings over increasing recall accuracy across participants.

As for the effects of masked priming on individuals' recall, recall accuracy was substantially lower for primed items that had received delayed JOLs than immediate JOLs or baseline and antiprimed items in the delayed JOL condition.

In terms of differences between participant groups in the implicit metamemory task, for recall accuracy controls tended to be more accurate on average than TBI survivors. Secondly, TBI survivors took quite a bit longer than controls overall to make JOL ratings. Additionally, response times for control participants increased by about one second from the immediate JOL timing condition to the delayed condition, while for TBI survivors the increase was over two seconds.

Robust priming for object identification accuracy was observed for both participant groups in the visual antipriming task. However, decreased identification accuracy for antiprimed items was not observed for either group. Yet, if we ignore responses made after two seconds, then priming and antipriming were observed for object identification accuracy, and these followed the pattern shown by Marsolek et al. (2006). As for response times, these were quite a bit longer for antiprimed objects than for baseline objects or primed objects; however, response times for primed objects were not notably faster than for baseline objects.

When comparing the magnitude of priming effect in the visual antipriming task to any effect in the implicit metamemory task, it was found that there was a trend, whereby those who demonstrated stronger priming benefit in the visual object identification task also showed somewhat stronger priming benefit in relative predictive accuracy in the metamemory task.

Finally, one last finding of some interest was that in the validation task, antiprime stimuli had remarkably lower percent old judgments, and faster response times, than new items.

Interpretations and implications

So, what does all this mean? Within the context of the present study, one of the principle findings is that there is no significant evidence for any direct effect of subliminal masked priming or antipriming on measures of metamemory judgment, such as JOL ratings or response times. However, there is some evidence for the effects of these on implicit aspects of metamemory, and other decisional processes.

What is meant by “implicit aspects of metamemory” should be further clarified. In the introductory chapter, the question was posed, “Is metamemory implicit?” By this was meant, “could explicit metamemory judgments be influenced by implicit processes?” Tacit throughout this investigation has been the idea that the metamemory judgment itself was explicit. Indeed, participants were explicitly trained to make explicit metamemory judgments. However, arguably some types of metamemory judgments may manifest without conscious awareness, much the same way that the effects of long term repetition priming manifest irrespective of individuals’ awareness. In particular, confidence, as measured by difference scores between JOL ratings and actual recall, certainly reflects one’s appraisal of one’s abilities (e.g.: overconfidence reflecting a persistent over-appraisal of one’s ability). Yet participants’ degree of confidence in making JOLs was never explicitly addressed by the experimenter or participants. Participants’ confidence manifested itself irrespective of their conscious

awareness of it; they may or may not have been aware of the degree of over- or under-confidence they displayed in their responses. Thus, just as with object identification accuracy, in which the results of one's performance are available to conscious awareness, so also the degree of confidence one has in one's responses is also available to conscious awareness, but may be made without it.

The important point here is that just as conscious awareness is not necessary for the effects of the priming and antipriming to be observed in long term repetition priming, so also is the case with confidence. In the present study, participants' confidence was significantly affected by subliminal masked antipriming, yet they were never asked to think about their confidence, either prospectively or retrospectively, and were likely too busy during the task to give this any explicit thought. In other words, not only was the metamemory phenomenon of confidence occurring irrespective of awareness, so were the changes in confidence as a function of implicit manipulation. This is a finding that bears further investigation.

Apart from implicit aspects of metamemory, the present research also found an effect of masked priming on another decisional process. Subliminal masked antiprime stimuli significantly lowered both participant groups' judgments of items as old in the validation task. While the mechanism underlying this effect (whether due to negative priming, antipriming, interference, or some other process) is unclear, what is clear is that the antiprime items influenced individuals' old/new judgments. The judgment cannot be considered a true metamemory judgment in the sense that it does not require participants to cogitate on their own memory skills or abilities – there is no “thinking about one's thinking” required. Nevertheless, the old/new judgment does invoke some

kind of slow decisional process, reminiscent of more pure metamemory decisions in that it requires a judgment on the contents of one's memory. Additionally, the old/new judgment certainly is a cognitive process that occurs at a fairly high, conceptual level. Thus, the present research adds to the growing body of research showing that subliminal masked presentation of stimuli can influence levels of cognition substantially higher than perceptual, or even lexical and semantic, processing. In fact, the present research is the first study to show that subliminal masked antipriming can influence higher level cognition. Finally, it bears consideration that the old/new judgment was conducted many minutes after the subliminal presentation. Thus, the effects of the subliminal antiprime either persisted for many minutes, or had some kind of cascade effect wherein the subliminal antiprime influenced the representational strength of the item or engaged an inhibitory process for that item, which in turn decreased the "old" judgment, and increased the speed of that decision.

Finally, and perhaps most importantly, is the question of clinical implications for the present research. The primary purpose of including a TBI population in the present study was to determine whether or not TBI survivors could make use of masked priming to improve metamemory judgments. Neither participant group showed influences of priming on JOLs or their response times. However, TBI survivors did experience a substantial decrease in their overconfidence. This is an intriguing finding and should be further explored for more direct clinical applications. Secondly, it was hypothesized that given the prevalence of frontal and diffuse axonal injury in the TBI population, and the relative sparing of the ventral visual pathway stream in that population, antipriming and priming of visual object identification would be preserved

following TBI. Indeed there were no significant group differences in priming or antipriming between the two participant groups.

Thus, the present research demonstrates that TBI survivors do demonstrate implicit memory that is not qualitatively different from controls; their priming and antipriming profiles across tasks and dependent measures are to much degree the same in both a subliminal masked priming paradigm as well as in long term repetition priming. The latter of these may be more informative in terms of clinical applications, since there is little clinical utility in, or ability to conduct, masked priming in such a setting. Furthermore, such implicit learning approaches as spaced retrieval, errorless learning, method of vanishing cues, etc., bear more similarity to long term repetition priming and so the intactness of the latter may provide a window into the function and utility of such approaches.

Limitations

There were numerous limitations to the present research, much of which has been discussed above. However, these limitations do bear summary. The first of these was that the implicit metamemory task required so many trials, due to the multiplicity of condition combinations and therefore lists, that the task was extremely long. As a result, participants could not be given a pre-study phase such as was used by Kennedy and Yorkston (2000), which would only have lengthened the task even more. This resulted in low recall accuracy for both participant groups, but particularly for the TBI survivor group. That, in turn, made recall response times for correct items fairly unstable, and with several missing cases.

Related to the issue of task length was the fatigue. Although participants were given frequent breaks, nevertheless, they reported feeling quite fatigued by the end of the approximately three hours of the experimentation. (Fortunately, perhaps because of the frequent breaks, repeated measures MANOVA did not find any trends across blocks, and therefore the data could be collapsed across blocks.) Furthermore, the session length, combined with the fact that participation required attendance of *two* three-hour sessions (the second used for the various standardized tests), made it difficult to recruit matched controls. Given the demographic, most control participants were fully employed, middle-aged individuals, with spouses and children; it was difficult for them to take that much time off of work or away from their families to participate, and that too for so little compensation. Thus, this resulted in a limited sample size of participants. Several findings were of borderline significance and with increased power, these findings may become significant.

Another limitation was soliciting the metamemory judgment so long after the masked stimulus was presented. As has been pointed out several times, the JOL was solicited at best five seconds after stimulus presentation (for immediate JOLs among controls) and as long as two to three minutes after stimulus presentation (for delayed JOLs). Effects which might otherwise have been observed in the JOLs may have largely attenuated by the time the JOLs were solicited. Alternative approaches will be discussed below.

The visual antipriming task also contained a limitation, one introduced as the result of a deviation from the protocol of Marsolek et al.'s (2006) experiment number four: making the responses self paced. Whereas Marsolek et al. limited response time

to two seconds, the change to self-paced response likely washed out the antipriming effect in object identification accuracy.

Finally, there was also the limitation that even if masked priming had shown significant effects on metamemory judgments, it is not a technique that can be easily translated into clinical utility. This issue will be addressed below.

Future directions

The present research found weak evidence for the role of masked priming and antipriming in metamemory judgments. Regarding solicitation of a JOL so long after presentation of the masked stimulus, another possibility to explore is to present the subliminal prime immediately prior to making the JOL. This was considered during the development of this task. However, due to the asymmetry of immediate vs. delayed JOLs, one possibility was that participants would not be shown the masking row of ampersands and subliminal items immediately after each word-pair was studied. Rather, some word-pairs would immediately be followed by another word-pair (under the delayed JOL timing condition), while other word-pairs would be followed by the forward mask and subliminal stimulus. (Later, for the delayed JOLs, all such trials would be preceded by the forward mask and subliminal stimulus presentation, so that does not present a problem.) This may raise the concern of covertly cueing participants that something significant was occurring for some of the items during the study phase. Future research may attempt to address the asymmetry simply by presenting a forward mask and a subliminal presentation of the baseline row of x-s immediately following study of word-pairs which are to be given delayed JOLs. That way, all trials would be

succeeded by the same forward mask and subliminal presentation, however, only the immediate JOLs would contain the randomization of subliminal baseline, prime, or antiprime items immediately after study of the word-pairs. Such a manipulation would result in all JOLs being immediately preceded by a subliminal baseline, prime, or antiprime presentation, with virtually no time for decay of the subliminal item.

Another possibility for future research would be to present not a word as a prime/antiprime stimulus, but rather the JOL rating itself. So, for example, the briefly presented subliminal item may simply be a visual reproduction of the JOL rating scale with one item highlighted. For example, the 50 ms subliminal presentation following the forward mask may appear like:

x x x x 80 x

Finally, taking the implicit metamemory line of research to a new level, exploration of long term priming and antipriming of metamemory in the TBI population should be undertaken to determine whether or not there is a priming benefit to metamemory judgments through supraliminal implicit memory approaches, that might translate better such as the aforementioned clinical approaches as spaced retrieval, errorless learning, etc. For example, what can priming and antipriming tell us about which participants will respond better to implicit learning techniques? Is there a correlation between magnitude of long term repetition priming and degree of retention during spaced retrieval? Should antiprime-like foils be used during tasks, while

maintaining errorless learning, to promote the interleaved learning necessary to successfully reinstantiate items in memory?

Conclusion

In conclusion, the current research provides interesting evidence that builds on the extant literature exploring the relationship between implicit memory and metamemory. It has shown that subliminal masked priming and antipriming do not influence individuals' judgments of learning for paired associate learning. It has also produced some evidence for more subtle manifestations of implicit memory in metamemory, such as the trend of interaction effects of prime condition and JOL timing condition on metamemory, as well as the effects on confidence and old/new judgments. Regarding this last item, the present research is the first to produce evidence that subliminal masked antipriming can influence the relatively high level of cognitive decisions. Thus, it has contributed to the early and growing literature on the newly discovered phenomenon of antipriming. Future research will examine relationships between long term repetition priming and antipriming, and clinically relevant forms of implicit learning.

REFERENCES

- Abrams, R.L., & Greenwald, A.G. (2000). Parts outweigh the whole (word) in unconscious analysis of meaning. *Psychological Science, 11*(2), 118-124.
- Arbuckle, T.Y., & Cuddy, L.A. (1969). Discrimination of item strength at time of presentation. *Journal of Experimental Psychology, 81*, 126-131.
- Bashore, T.R. and Ridderinkhof, K.R. (2002). *Psychological Bulletin, 128* (1), 151–198.
- Becker, S., Moscovitch, M., Behrmann, M., & Joordens, S. (1997). Long-term semantic priming: A computational account and empirical evidence. *Journal of Experimental Psychology: Learning, Memory, & Cognition, 23*, 1059–1082.
- Bishop, K.I., & Curran, H.V. (1998). An investigation of the effects of benzodiazepine receptor ligands and of scopolamine on conceptual priming. *Psychopharmacology, 140*, 345 - 353.
- Bodner, G.E., & Masson, M.E.J. (2003). Beyond spreading activation: An influence of relatedness proportion on masked semantic priming. *Psychonomic Bulletin & Review, 10*(3), 645-652.
- Bourgeois, M.S., Lenius, K., Turkstra, L., & Camp, C. (2007). The effects of cognitive teletherapy on reported everyday memory behaviours of persons with chronic traumatic brain injury. *Brain Injury, 21*(12), 1245–1257.
- Box, G. E. P., & Anderson, S. L. (1955). Permutation theory in the derivation of robust criteria and the study of departures from assumptions. *Journal of the Royal Statistical Society, 17*, 1-34.

Bright, P., Jaldow, E., & Kopelman, M.D. (2001). The National Adult Reading Test as a measure of premorbid intelligence: A comparison with estimates derived from demographic variables. *Journal of the International Neuropsychological Society*, 8, 847–854.

Brookshire, R. H. & Nicholas, L. E. (1993). *The Discourse Comprehension Test*. Tucson, AZ: Communication Skill Builders.

Busch, R. M., McBride, A., Curtiss, G., & Vanderploeg, R. D. (2005). The Components of Executive Functioning in Traumatic Brain Injury. *Journal of Clinical and Experimental Neuropsychology*, 27(8), 1022 - 1032 .

Cheesman, J. & Merikle, P.M. (1984). Priming with and without awareness. *Perceptual Psychophysics*. 36, 387–395.

Coltheart, M. (1981). MRC Psycholinguistic Database, version 2.00 [Web database and application]. Retrieved from http://www.psy.uwa.edu.au/MRCDataBase/uwa_mrc.htm on January 7, 2008.

Courville, C.B. (1937). *Pathology of the Central Nervous System*. Pacific Press Publishing Association, Mountain View, CA.

Damian, M.F. (2001). Congruity effects evoked by subliminally presented primes: Automaticity rather than semantic processing. *Journal of Experimental Psychology: Human Perception and Performance*, 27(1), 154-165.

Davis, C., & Kim, J. (2000). Masked priming by translation and phonological primes in Korean and English. Paper presented at the XXVII International Conference of Psychology, Stockholm, Sweden.

Davis, C., Kim, J., & Sanchez-Casas, R. (2003). Masked priming across languages: An insight into bilingual lexical processing. In S. Kinoshita & S. Lupker (Eds.), *Masked Priming: The State of the Art*, 309-322. New York, NY: Psychology Press.

Deason, R. G. (2008). *Masked antipriming: A behavioral and event-related potential examination of short-term antipriming effects*. Unpublished doctoral dissertation, University of Minnesota, Minneapolis.

Deason, R. G., & Marsolek, C. J. (2005, April). Word-to-object antipriming: Are visual word and visual object representations functionally superimposed? Poster presented at the Cognitive Neuroscience Society Annual Meeting, New York, NY.

Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (2000). *California Verbal Learning Test—Second Edition, Adult Version*. San Antonio, TX: Psychological Corporation.

Delis, D. C., Kaplan, E., & Kramer, J.H. (2001). *Delis-Kaplan Executive Function System*. San Antonio, TX: Psychological Corporation.

Deerwester, S., Dumais, S.T., Furnas, G.W., Landauer, T.K., & Harshman, R. (1990). Indexing by latent semantic analysis. *Journal of the American Society for Information Science*, 41(6), 391 – 407.

Dehaene S., Naccache, L., Cohen, L., Le Bihan, D., Mangin, J., Poline, J., & Riviere, D. (2001). Cerebral mechanisms of word masking and unconscious repetition priming. *Nature Neuroscience*, 4, 1-7.

Dumais, S.T. (1991). Improving the retrieval of information from external sources. *Behavior Research Methods, Instruments, & Computers*, 23 (2), 229-236.

Dumais, S. T. (1994). Latent semantic indexing (LSI) and TREC-2. In D. Harman (Ed.), *The Second Text REtrieval Conference (TREC2)*, National Institute of Standards and Technology Special Publication 500-215 , pp. 105-116.

Dunlosky, J., and Metcalfe, J. (2009). *Metacognition*. Sage Publications: Thousand Oaks, CA.

Eskes, G.A., Szostak, C., & Stuss, D.T. (2003). Role of the frontal lobes in implicit and explicit retrieval tasks. *Cortex*, 39(4-5), 847-869.

Evett, L.J., & Humphreys, G.W. (1981). The use of abstract graphemic information in lexical access. *Quarterly Journal of Experimental Psychology* 33(A), 325–350.

Fernandez-Duque, D. Baird, J.A., & Posner, M.I. (2000). Executive Attention and Metacognitive Regulation. *Consciousness and Cognition*, 9, 288–307.

Flavell, J. H. (1979). Metacognition and cognitive monitoring: A new area of cognitive-developmental inquiry. *American Psychologist*, 34, 906-911.

Forster, K.I., Booker, J., Schacter, D.L., & Davis, C. (1990). Masked repetition priming: Lexical activation or novel memory trace? *Bulletin of the Psychonomic Society*, 28, 341-345.

Forster, K.I., and Davis, C. (1984). Repetition priming and frequency attenuation in lexical access. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 10, 680-698.

Forster, K.I., Mohan, K., & Hector, J. (2003). The mechanics of masked priming. In S. Kinoshita, & S.J. Lupker (Eds.), *Masked priming: The state of the art* (pp. 3 – 37). New York, NY: Psychology Press.

Friedman, D. Cycowicza, Y.M., & Dziobek, I. (2003). Cross-form conceptual relations between sounds and words: effects on the novelty P3. *Cognitive Brain Research, 18*, 58–64.

Gabrieli, J.D. (1998). Cognitive neuroscience of human memory. *Annual Review of Psychology, 49*, 87-115.

Gilhooly, K.J. & Logie, R.H. (1980). Age of acquisition, imagery, concreteness, familiarity and ambiguity measures for 1944 words. *Behavior Research Methods and Instrumentation, 12*, 395-427.

Gollan, T.H., Forster, K.I., & Frost, R. (1997). Translation priming with different scripts: Masked priming with cognates and non-cognates in Hebrew-English bilinguals. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 23*(5), 1122-1139.

Greenwald, A.G., Klinger, M.R., & Liu, T.J. (1989). Unconscious perception of dichoptically masked words. *Memory & Cognition, 17*, 35-47.

Greenwald, A.G., Klinger, M.R., & Schuh, E.S. (1995). Activation by marginally perceptible (“subliminal”) stimuli: Dissociation of unconscious from conscious cognition. *Journal of Experimental Psychology: General, 124*, 22-42.

Hanten, G., Dennis, M., Zhang, L., Barnes, M.A., Roberson, G., Archibald, J., Song, J.X., & Levin, H.S. Childhood head injury and metacognition processes in language and memory. *Developmental Neuropsychology, 25*, 86-106.

Hart, J.T. (1965). Memory and the feeling-of-knowing experience. *Journal of Experimental Psychology, 56*(4), 208 – 216.

- Hart, J.T. (1967). Memory and the memory monitoring process. *Journal of Verbal Learning and Verbal Behavior*, 6, 685-691.
- Hart, T., & Evans, J. (2006). Self-regulation and goal theories in brain injury rehabilitation. *Journal of Head Trauma Rehabilitation*, 21(2), 142-155.
- Haut, M.W., Petros, T.V., Frank, R.G., & Haut, J.S. (1991). Speed of processing within semantic memory following severe closed head injury. *Brain & Cognition*, 17(1), 31-41.
- Haxby, J.V., Gobbini, M.I., Furey, M.L., Ishai, A., Schouten, J.L., & Pietrini, P. (2001). Distributed and Overlapping Representations of Faces and Objects in Ventral Temporal Cortex. *Science*, 293, 2425- 2430.
- Hopper, T., Mahendra, N., Kim, E., Azuma, T., Bayles, K.A., Cleary, S.J., & Tomoeda, C.K. (2005). Evidence Based Practice Recommendations for working with individuals with dementia: Spaced-Retrieval Training. *Journal of Medical Speech-Language Pathology*, 13 (4), xxvii-xxxiv.
- Humphreys, G.W., Evett, L.J., Quinlan, P.T., & Besner, D. (1987). Orthographic priming: Qualitative differences between priming from identified and unidentified primes. In M. Coltheart (Ed.), *Attention and Performance XII* (pp. 201-219) Hillsdale, NJ: Erlbaum.
- Inglese, M., Makani, S., Johnson, G., Cohen, B.A., Silver, J.A., Gonen, O., & Grossman, R.I. (2005). Diffuse axonal injury in mild traumatic brain injury: a diffusion tensor imaging study. *Journal of Neurosurgery* 103, 298–303.
- James, W. (1950). *The Principles of Psychology*, New York, NY: Dover.

Jameson, K.A., Narens, L., Goldfarb, K., & Nelson, T.O. (1990). The influence of near-threshold priming on metamemory and recall. *Acta Psychologica* 73, 55-68.

Janowsky, J.S., Shimamura, A.P., & Squire, L.R. (1989). Memory and metamemory: Comparisons between patients with frontal lobe lesions and amnesic patients. *Psychobiology*, 17(1), 3-11.

Jiang, N. (1999). Testing processing explanations for the asymmetry in masked cross-language priming. *Bilingualism: Language & Cognition*, 2(1), 59-75.

Jiang, N., & Forster, K.I. (2001). Cross-language priming asymmetries in lexical decision and episodic recognition. *Journal of Memory & Language*, 44(1), 32-51.

Johnson, S.C., Baxter, L.C., Wilder, L.S., Pipe, J.G., Heiserman, J.E., & Prigatano, G.P. (2002). Neural correlates of self-reflection. *Brain*, 125, 1808-1814.

Johnson-Laird, P.N. (1983). A computational analysis of consciousness. *Cognition and Brain Theory*, 6, 499-508.

Kennedy, M.R. (2001). Retrospective confidence judgments made by adults with traumatic brain injury: relative and absolute accuracy. *Brain Injury*, 15(6), 469-487.

Kennedy, M.R., Carney, E., & Peters, S.M. (2003). Predictions of recall and study strategy decisions after diffuse brain injury. *Brain Injury*, 17(12), 1043-1064.

Kennedy, M.R. & Nawrocki, M. (2003). Delayed predictive accuracy of narrative recall after traumatic brain injury: Salience and explicitness. *Journal of Speech, Language, and Hearing Research*, 46(1), 98-112.

Kennedy, M.R., Yorkston, K.M., Rogers, M. (1995). Self-monitoring abilities of two adults with traumatic brain injury during verbal learning. *American Journal of Speech-Language Pathology*, (4), 159-163.

Kennedy, M.R., & Yorkston, K.M. (2000). Accuracy of metamemory after traumatic brain injury: predictions during verbal learning. *Journal of Speech Language Hearing Research*, 43(5), 1072-1086.

Kennedy, M.R., & Yorkston, K.M. (2004). The effects of frontal injury on self-monitoring during verbal learning by adults with diffuse brain injury. *Neuropsychological Rehabilitation*, 14, 449-465.

Kertesz, A. (1982). *Western Aphasia Battery*. New York, NY: The Psychological Corporation.

Kessels, R.C.P., & de Haan, E.H.F. (2003). Implicit learning in memory rehabilitation: A meta-analysis of errorless learning and vanishing cues methods. *Journal of Clinical and Experimental Neuropsychology*, 25(6), 805-814.

Kihlstrom, J.F. (1999). Conscious vs. unconscious cognition. In R.J. Sternberg (Ed.), *The nature of cognition* (pp. 173-203). Cambridge, MA: MIT Press.

Kinoshita, S. (1997). Masked target priming effects on feeling-of-knowing and feeling-of-familiarity judgments. *Acta Psychologica* 97, 183-199.

Kinoshita, S. (2002). Feeling of familiarity. In P. Chambres, I. Marie, & P. Marescaux, (Eds.), *Metacognition: Process, function and use* (pp. 79-90). Dordrecht, Netherlands: Kluwer Academic Publishers.

Koriat, A. (1981). Semantic facilitation in lexical decision as a function of prime-target association. *Memory & Cognition*, 9, 587-598.

Koriat, A. (1993). How do we know that we know? The accessibility model of the feeling of knowing. *Psychological Review*, *100* (4), 609–639.

Koriat, A. (1995). Dissociating knowing and the feeling of knowing: Further evidence for the accessibility model. *Journal of Experimental Psychology: General*, *124* (3), 311–333.

Koriat, A., & Levy-Sadot, R. (2001). The combined contributions of the cue-familiarity and accessibility heuristics to feelings of knowing. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *27*(1), 34-53.

Laham, D. (1998). Latent Semantic Analysis, pairwise comparison feature [Web application]. Retrieved from <http://lsa.colorado.edu/> on January 7, 2008.

Landauer, T.K. & Dumais, S.T. (1997). A solution to Plato's problem: The latent semantic analysis theory of acquisition, induction and representation of knowledge. *Psychological Review*, *104*(2), 211-240.

Lashley K.S. (1929). *Brain Mechanisms and Intelligence*. Chicago: Univ. Chicago Press

Leonesio, R.J., & Nelson, T.O. (1990). Do different metamemory judgments tap the same underlying aspects of memory? *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *16*(3), 464-470.

Levy, K.J. (1980). A Monte Carlo study of analysis of covariance under violations of the assumptions of normality and equal regression slopes. *Educational and Psychological Measurement*, *40*(4), 835-840.

Lezak, M.D. (1979). Recovery of memory and learning functions following traumatic brain injury. *Cortex*, *15*, 63-72.

Lindman, H. R. (1974). *Analysis of variance in complex experimental designs*.

San Francisco, CA: W. H. Freeman & Co.

Maki, R.H. (1999). The roles of competition, target accessibility, and cue familiarity in metamemory for word pairs. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 25, 1011-1023.

Malykhin, N., Concha, L., Seres, P., Beaulieu, C., & Coupland, N. (2008). Diffusion tensor imaging tractography and reliability analysis for limbic and paralimbic white matter tracts. *Neuroimaging*, 164(2), 132-142.

Marcel, A. (1983). Conscious and unconscious perception: Experiments on visual masking and word recognition. *Cognitive Psychology*, 15, 197-237.

Marsolek, C. J., Ketz, N.A., Ramanathan, P., Deason, R.G., Davis, S., Verfaellie, M., & Schnyer, D.M. (2007, June). *Neural priming effects in fMRI: Integrated fMRI and neurocomputational evidence for what may be reflected*. Poster session presented at the annual meeting of the Organization for Human Brain Mapping, Chicago, IL.

Marsolek, C.J., Schnyer, D.M., Deason, R.G., Ritchey, M., & Verfaellie, M. (2006). Visual antipriming: Evidence for ongoing adjustments of superimposed visual object representations. *Cognitive, Affective, & Behavioral Neuroscience*, 6 (3), 163 – 174.

Marsolek, C.J., Deason, R.G, Ketz, N.A., Ramanathan, P., Bernat, E.M., Steele, V.R., Patrick, C.J., Verfaellie, M., & Schnyer, D.M. (2009). *Identifying objects impairs knowledge of other objects: A relearning explanation for the neural repetition effect*.

Manuscript submitted for publication.

McClelland, J. (1996). Neural mechanisms for the control and monitoring of memory: A parallel distributed processing perspective. In L. Reder (Ed.), *Implicit Memory and Metacognition* (pp 275-286). Mahwah, New Jersey: Lawrence Erlbaum Associates.

Metcalfe, J. (1986). Premonitions of insight predict impending error. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 12(4), 623-634.

Metcalfe, J., Schwartz, B.L., & Joaquim, S.G. (1993). The cue-familiarity heuristic in metacognition. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 19(4), 851-861.

Metcalfe, J., & Finn, B. (2008). Familiarity and retrieval processes in delayed judgments of learning. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 34(5), 1084-1097.

Meyer, D., & Schvaneveldt, R. (1971). Facilitation in recognizing pairs of words: Evidence of a dependence between retrieval operations. *Journal of Experimental Psychology*, 90, 227-234.

Meythaler, J.M., Peduzzi, J.D., Eleftheriou, E., & Novack, T.A. (2001). Current concepts: diffuse axonal injury-associated traumatic brain injury. *Archives of Physical Medicine & Rehabilitation*, 82(10),1461-1471.

Miner, A., & Reder, L.M. (1994). A new look at feeling of knowing: Its metacognitive role in regulating question answering. In J. Metcalfe and A.P. Shimamura (Eds.), *Metacognition: Knowing about knowing* (pp. 47 – 70). Cambridge, MA: Bradford press.

Mulligan, N.W, Guyer, P.S., & Beland, A. (1999). The effects of levels-of-processing and organization on conceptual implicit memory in the category exemplar production test. *Memory & Cognition*, 27(4), 633-647.

Neely, J. H. (1976). Semantic priming and retrieval from lexical memory: Evidence for facilitatory and inhibitory processes. *Memory & Cognition*, 4, 648-654.

Neely, J.H., Keefe, D.E., & Ross, K.L. (1989). Semantic priming in the lexical decision task: Roles of prospective prime-generated expectancies and retrospective semantic matching. *Journal of Experimental Psychology: Learning, Memory, & Cognition*, 15(6), 1003-1019.

Nelson, H.E. & Willison, J.R. (1991). *The Revised National Adult Reading Test—Test manual*. Windsor, UK: NFER-Nelson.

Nelson, T. O. (1992). *Metacognition: Core readings*. Boston, MA: Allyn & Bacon.

Nelson, T.O., & Dunlosky, J. (1991). When people's judgments of learning (JoLs) are extremely accurate at predicting subsequent recall: The "Delayed-JOL Effect." *Psychological Science* 2(4), 267-270.

Nelson, T.O., & Dunlosky, J. (1992). How shall we explain the delayed-judgment-of-learning effect? *Psychological Science*, 3(5), 317-318.

Nelson, T. O., & Narens, L. (1990). Metamemory: A theoretical framework and new findings. In G. H. Bower (Ed.), *The psychology of learning and motivation* (Vol. 26, pp. 125-173). New York: Academic Press.

Nelson, Narens, and Dunlosky (2004). A revised methodology for research on metamemory: Pre-judgment recall and monitoring (PRAM). *Psychological Methods*, 9 (1), 53–69.

Paivio, A., Yuille, J.C. & Madigan, S.A. (1968). Concreteness, imagery and meaningfulness values for 925 words. *Journal of Experimental Psychology Monograph Supplement*, 76 (3, part 2), 1 – 25.

Parkin, A.J. & Leng, R.C. (1993). *Neuropsychology of the Amnesic Syndrome*. Erlbaum: Hillsdale, New Jersey.

Pinon, K. Allaina, P., Kefia, M.Z., Dubasa, F., & Le Galla, D. (2005). Monitoring processes and metamemory experience in patients with dysexecutive syndrome. *Brain and Cognition* 57, 185–188.

Rajaram, S. (1993). Remembering and knowing: Two means of access to the personal past. *Memory and Cognition*, 21, 89-102.

Reder, L.M. (1987). Strategy selection in question answering. *Cognitive Psychology*, 19, 90 – 137.

Reder, L.M. (1988). Strategic control of retrieval strategies. In G.H. Bower (Ed.), *The psychology of learning and motivation* (Vol. 22, pp. 227 – 259). San Diego, CA: Academic Press.

Reder, L.M., & Ritter, F.E. (1992). What determines initial feeling of knowing? Familiarity with question terms, not the answer. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 18, 435-451.

Reder, L.M., & Schunn, C.D. (1996). Metacognition does not imply awareness: Strategy choice is governed by implicit learning and memory. In L. Reder (Ed.),

Implicit Memory and Metacognition (pp. 45-77). Mahwah, New Jersey: Lawrence Erlbaum Associates.

Schacter, D.L. (1987). Implicit memory: History and current status. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 13, 501-518.

Schacter D.L. (1997). The cognitive neuroscience of memory: perspectives from neuroimaging research. *Philosophical Transactions of the Royal Society of London - Series B: Biological Sciences*, 352(1362), 1689-1695.

Schacter, D.L., Chiu, C-Y, & Ochsner, K.N. (1993). Implicit memory: A selective review. *Annual Reviews Neuroscience*, 16, 159-182

Schacter, D.L., Church, B., & Treadwell, J. (1994). Implicit memory in amnesic patients: Evidence for spared auditory priming, *Psychological Science*, 5, 20–25.

Schacter, D. L., Rich, S.A., & Stampff, M. S. (1985). Remediation of memory disorders: Experimental evaluation of the spaced-retrieval technique. *Journal of Clinical and Experimental Neuropsychology*, 7(1), 79-96.

Schacter, D.L., Wig, G.S., & Stevens, W.D. (2007). Reductions in cortical activity during priming. *Current Opinion in Neurobiology*, 17.

Schmitz, T.W., Rowley, H.A., Kawahara, T.N., & Johnson, S.C. (2006). Neural correlates of self-evaluative accuracy after traumatic brain injury. *Neuropsychologia*, 44, 762–773.

Schmitter-Edgecombe, M. (2006). Implications of basic science research for brain injury rehabilitation: A focus on intact learning mechanisms. *Journal of Head Trauma Rehabilitation*, 21(2), 131-141.

Schmitter-Edgecombe, M., & Woo, E. (2004). Memory self-awareness and memory self-monitoring following severe closed-head injury. *Brain Injury, 18* (10), 997-1016.

Schneider, W., Eschman, A., & Zuccolotto, A. (2002). *E-Prime User's Guide*. Pittsburgh: Psychology Software Tools Inc.

Schraw, G. (1995). Measures of feeling of knowing accuracy: A new look at an old problem. *Applied Cognitive Psychology, 9*, 321–332.

Schwartz, B.L. (1994). Sources of information in metamemory: Judgments of learning and feeling of knowing. *Psychonomic Bulletin & Review, 1*, 357-375.

Scoville, W.B. & Milner, B.J. (1957). Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery, & Psychiatry, 20*, 11–21.

Seger, C.A. (1994). Implicit Learning. *Psychological Bulletin, 115* (2), 163-196.

Shah, J.Y., & Kruglanski, A.W. (2003). When opportunity knocks: bottom-up priming of goals by means and its effects on self-regulation. *Journal of Personality & Social Psychology, 84*(6), 1109-1122.

Shimamura, A.P. (2000). Toward a cognitive neuroscience of metacognition. *Consciousness and Cognition, 9*, 313 – 323.

Souchay, C. Isingrini, M., Clarys, D., Taconnat, L., & Eustache, F. (2004). Executive Functioning and Judgment-of-Learning versus Feeling-of-Knowing in Older Adults. *Experimental Aging Research, 30*(1), 47-62.

Spellman B.A. and Bjork R.A. (1992). When predictions create reality: Judgments of learning may alter what they are intended to assess. *Psychological Science, 3*(5), 315-316.

Squire L.R. (1987). *Memory and Brain*. New York, NY: Oxford University Press

Squire, L.R. (1994). Declarative and non-declarative memory: Multiple brain systems supporting learning and memory. In D.L. Schacter & E. Tulving (Eds.), *Memory Systems 1994* (pp. 203-231). Cambridge, MA: MIT Press.

Squire, L.R., Ojemann, J.G., Miezin, F.M., Petersen, S.E., Videen, T.O., & Raichle, M.E. (1992). Activation of the hippocampus in normal humans: A functional anatomical study of memory, *Proceedings of the National Academy of Sciences USA*, 89, 1837–1841.

Stein, S.C. (1996). Classification of head injury. In: R.K. Narayan, J.T. Povlishock, & J.E. Wilberger, Jr. (Eds.), *Neurotrauma* (pp. 31-42). New York, NY: McGraw-Hill.

Stenberg, G., Lindgren, M., Johansson, M., Olsson, A., & Rosen, I. (2000). Semantic processing without conscious identification: Evidence from evoked response potentials. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 26, 973-1004.

Stuss, D.T. (1991). Self, awareness, and the frontal lobes: A neuropsychological perspective. In J. Strauss, & G.R. Goethals (Eds.), *The self: Interdisciplinary approaches* (pp. 255-278). New York, NY: Springer-Verlag.

Stuss, D.T., & Levine, B. (2002). Adult clinical neuropsychology: Lessons from Studies of the Frontal Lobes. *Annual Reviews of Psychology*, 53, 401–433.

Thompson-Schill, S.L., & Gabrieli, J.D. (1999). Priming of visual and functional knowledge on a semantic classification task. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 25(1), 41-53.

Tipper, S.P. (1985). The negative priming effect: Inhibitory priming with to be ignored objects. *The Quarterly Journal of Experimental Psychology*, 37A, 571-590.

Toglia, M. P., & Battig, W. F. (1978). *Handbook of semantic word norms*. Hillsdale, NJ: Erlbaum.

Tulving, E. (1985). Memory and consciousness. *Canadian Psychologist*, 26, 1-12.

Tulving, E., & Schacter, D.L. (1990). Priming and human memory systems. *Science*, 247, 301–306.

Vaidya, C. J., Gabrieli, J.D., Keane, M.M., Monti, L.A., Gutierrez-Rivas, H., & Zarella, M.M. (1997). Evidence for multiple mechanisms of conceptual priming on implicit memory tests. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 23 (6), 1324-1343.

Vakil, E., Jaffe, R., Eluze, S., Groswasser, Z., & Aberbuch, S. (1996). Word recall versus reading speed: evidence of preserved priming in head-injured patients. *Brain & Cognition*, 31(1), 75-89.

Vakil, E., & Sigal, J. (1997). The effect of level of processing on perceptual and conceptual priming: Control versus closed-head-injured patients. *Journal of the International Neuropsychological Society*, 3(4), 327-336.

Vernon, D., & Usher, M. (2003). Dynamics of metacognitive judgments: Pre- and post retrieval mechanisms. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 29(3), 339-346.

Vesonder, G.T., & Voss, J.F (1985). On the ability to predict one's own responses while learning. *Journal of Memory and Language*, 24, 363-376,

Wagner, A.D., Maril, A., & Schacter, D.L. (2000). Interactions between forms of memory: When priming hinders new episodic learning. *Journal of Cognitive Neuroscience*, 12, (Supplement 2), 52 – 60.

Wechsler, D. (1987). *Wechsler Memory Scale-Revised*. San Antonio, TX: Psychological Corporation.

Wig, G.S., Grafton, S.T., Demos, K.E., & Kelley, W.M. (2005). Reductions in neural activity underlie behavioral components of repetition priming. *Nature Neuroscience*, 8(9), 1228 – 1233.

Woodworth, R.S., & Schlosberg, H. (1964). *Experimental Psychology, (revised edition)*. New York, NY: Holt.

Zola-Morgan, S., Squire, L.R., & Amaral, D.G. (1986). Human amnesia and the medial temporal region: enduring memory impairment following a bilateral lesion limited to field CA1 of the hippocampus. *Journal of Neuroscience*, 6, 2950–2967.

Table 1.

Demographic Characteristics of Healthy Controls (n=14) and Adults with Brain Injury (n=17)

	Control	Brain Injured
Age at Experiment (yrs) ^a	47.22 +/- 10.17	47.42 +/- 10.56
Sex (male/female)	7/7	10/7
Years of Education (yrs) ^b	15.36 +/- 1.66	14.68 +/- 1.94
Age at Injury (yrs)	NA	34.83 +/- 11.84
Years Post Onset (yrs)	NA	12.60 +/- 11.18

Note: Group differences are non-significant at $p < .05$.

^a $t(28.242) = 0.054$, sig. = .957; ^b $t(28.94) = -1.054$, sig. = .300

Table 2.

Detailed Demographic information for ABI participants

Ss	Sex	Age at Experiment (yrs)	Education (yrs)	Current/former occupation
1	M	37.40	16.0	Electronics Technician
2	M	44.92	11.0	Factory Worker
3	M	54.94	14.0	Artist/Flooring contractor
4	F	50.23	12.5	Medical secretary
5	F	55.26	16.0	Free lance writer
6	F	20.80	14.0	College junior (student)
7	F	56.38	13.5	Clerical (data entry)
8	F	56.78	15.0	Part time interior decorator
9	F	56.64	16.0	Hardware store office mgr.
10	F	45.65	12.0	unemployed (nanny/waitress)
11	M	54.96	18.0	Small business owner; musician (violinist)
12	M	36.26	16.0	unemployed; volunteer
13	M	29.55	16.0	unemployed
14	M	49.68	12.5	retired; was a mechanic
15	M	54.22	17.0	unemployed; was electrical engineer
16	M	49.64	14.0	Owns/runs caulking company
17	M	52.93	16.0	unemployed; was owner and CEO of window/door mfg.
M		47.43	14.68	
SD		10.56	1.94	

Table 3.

Detailed Demographic information for Control participants

Ss	Sex	Age at Experiment (yrs)	Education (yrs)	Current/former occupation
1	M	39.08	15.0	Civil engineering intern
2	M	51.31	13.0	Shipping manager
3	M	56.69	15.5	Artist/carpenter
4	F	52.33	13.0	Health unit coordinator
5	F	51.91	16.5	ESL teacher
6	F	21.41	15.0	College junior (student)
7	F	56.80	16.0	Custodian
8	F	43.51	13.0	Stage hand/truck driver
9	M	53.00	16.0	Mfg. production planner
10	F	43.54	14.0	Massage therapist, data entry
11	F	52.43	18.0	Artistic director/musician
12	M	32.87	16.0	Realtor (B.S. Biochemistry)
13	M	51.89	18.0	Systems analyst
14	M	54.35	16.0	Journalist/theater critic
M		47.22	15.36	
SD		10.17	1.66	

Table 4.

Descriptive Brain Injury Information

Ss	GCS	Length of coma (days)	Length of PTA†	Initial Severity	T.P.O (years)	Neurological Findings
1	7	1-14	NA	Severe	5.53	L. temporal hematoma, subarachnoid hemorrhage, possible anterior peduncular fossa shear injury along R. tentorium, L. cerebral peduncle shear injury
2	6	~ 8	NA	Severe	18.99	Diffuse edema with increased ICP, possible subarachnoid hemorrhage, small punctate hemorrhages in L. posterior thalamus
3	NA	NA	NA	NA	6.44	Right frontal glioma
4	NA	NA	NA	NA	6.55	Pneumocephalus, small acute sub-dural hematoma at R. temporal tip without mass effect
5	NA	NA	6 weeks	NA	35.09	TBI Dx (8/5/74). No other information available.
6	3T	~ 2	4 weeks	Severe	1.53	Severe edema, diffuse subarachnoid hemorrhage, shear injuries, possible brain stem injury, small hemorrhagic contusion in high L. frontal deep white matter.
7	NA	NA	NA	NA	37.96	Small contusion anterior temporal lobe, lesion to R. subinsular region, R. basal ganglia and anterior limb of internal capsule
8	15	NA	NA	Mild	15.46	Only findings were an old lacunar infarct in R. medial temporal lobe

9	NA	NA	Several minutes	NA	13.21	Neuropsychology, SLP, OT reports all refer to TBI and describe deficits; no documentation of neurologic findings.
10	NA	17	NA	Severe	6.02	Diagnosed 4/27/02: "severe closed head injury with cerebral contusion"; brainstem/axonal shear injuries
11	7	1-10	NA	Severe	0.51	Acute R. intraventricular hemorrhage, w/ bilateral parenchymal hemorrhages, minimal subarachnoid blood, blood in R. ventricle.
12	5	1 - 12	NA	Severe	14.11	Small L. frontal subdural hematoma, with punctate intraparenchymal hemorrhages in multiple sites; multiple bilateral shear bleeds; frontal and temporal hemorrhages. Small posterior subarachnoid hemorrhage. Mild R. shift of septum pellucidum.
13	NA	NA	NA	NA	0.63	Epidural hematoma, encephalomalacia involving the anterior-inferior L. frontal lobe, inferomedial R. frontal lobe, and L. temporal lobe.
14	NA	NA	NA	NA	2.87	Neurologist gave Dx of TBI. Only other information available refers to "basilar/basal skull fracture".
15	3	NA	NA	Severe	22.95	R. frontal epidural hematoma, contiguous with a R. frontal temporo-parietal subdural hematoma. Small L. sub-occipital epidural hematoma.
16	NA	NA	NA	NA	16.14	Small L. superior temporal and mid-parietal subdural hematoma, L. posterior frontal & anterior parietal hemorrhagic contusion

17	NA	No LOC	NA	Mild	10.16	Primary care M.D. refers to status post TBI, and seen at brain injury clinic.
M	*	*	*		12.60	
SD	*	*	*		11.18	

Note: NA indicates that the relevant information was Not Available

† Length of post traumatic amnesia is based on patient self report of first memory after injury.

* Not computed due to insufficient data

Table 5.

Means, *SDs*, and statistical comparisons of neurocognitive performance for adults with TBI (n = 17) and healthy controls (n = 14)

Neurocognitive Measures	TBI (mean +/- <i>SD</i>)	Control (mean +/- <i>SD</i>)	F	η_p^2
Aphasia quotient (WAB)	98.65 ± 1.21	99.51 ± 1.17	4.080	.123
Estimated IQ (NART-R)				
Full scale	106.94 ± 8.33	112.14 ± 8.96	2.796	.088
Verbal	105.76 ± 7.74	110.43 ± 8.40	2.581	.082
Performance	106.76 ± 7.37	111.43 ± 8.00	2.848	.089
Visual memory (WMS-III)				
Immediate recall (VR-I)	9.53 ± 3.68	11.86 ± 2.71	2.437	.083
Delayed recall (VR-II)	11.24 ± 2.82	13.50 ± 2.68	3.331	.110
Delayed recognition (VR-II)	11.31 ± 3.16	10.54 ± 2.07	0.578	.021
Short term memory (WMS-III)				
Digit span – total	10.71 ± 3.46	11.71 ± 3.77	0.602	.020
Short delay verbal recall (CVLT-II)				
Free	-0.09 ± 1.00	0.36 ± 0.93	1.616	.053
Cued	-0.15 ± 0.98	0.21 ± 1.01	1.012	.034
Long delay verbal recall (CVLT-II)				
Free	-0.18 ± 0.77	0.36 ± .089	3.221	.100
Cued	-0.18 ± 0.90	0.21 ± 0.78	1.632	.053
Executive functions: verbal fluency (D-KEFS)				
Letter fluency, total correct	9.76 ± 2.91	12.92 ± 4.35	5.684*	.169
Category fluency, total correct	10.88 ± 2.78	13.69 ± 2.29	8.73**	.238

Category switching, total correct	10.88 ± 3.46	15.46 ± 3.04	14.3**	.338
<hr/>				
Executive functions: design fluency (D-KEFS)				
Total correct (filled + empty + switching)	6.65 ± 2.85	8.62 ± 3.84	2.603	.085
Composite (filled + empty)	6.71 ± 2.31	7.62 ± 2.84	.934	.032
Contrast (total – [filled + empty])	10.35 ± 2.32	12.15 ± 2.38	4.356*	.135
<hr/>				
Executive functions: tower test (D-KEFS)				
Total achievement	11.65 ± 3.26	11.46 ± 2.85	0.027	.001
<hr/>				
Executive functions: trails (D-KEFS)				
Number-letter switching	10.41 ± 3.26	12.15 ± 1.91	2.928	.095
Number-letter switching vs. motor speed	10.65 ± 2.52	10.54 ± 2.03	.016	.001
<hr/>				
Speed: simple trails (D-KEFS)				
Number sequencing	9.47 ± 3.79	11.69 ± 1.65	3.871	.121
Letter sequencing	9.94 ± 3.73	12.23 ± 1.74	4.171	.130
Motor speed	9.76 ± 2.36	11.69 ± 1.11	7.382*	.209

Note.

CVLT-II, California Verbal Learning Test – II; D-KEFS, Delis-Kaplan Executive Function System; NART-R, National Adult Reading Test – Revised; WAB, Western Aphasia Battery; WMS-III, Wechsler Memory Scales – III. Comparison of participant groups on neurocognitive performance was conducted with ANOVA for WAB, WMS-III Digit Span, and D-KEFS Tower. For all other comparisons, MANOVA was used. MANOVA omnibus F test is non-significant for group difference [$F(4, 24) = 1.824, p = .298, \text{partial eta squared} = .916$].

Reported scores are all scaled scores.

* $p < .05$

** $p < .01$

Table 6.

Means and *SDs* for word lists used in implicit metamemory task

List	Similarity	Concreteness	Imageability	Frequency (K-F)	Frequency (T-L)
1	0.07 ± 0.02	597.8 ± 14.9	596.1 ± 10.9	24.3 ± 17.0	208.6 ± 118.1
2	0.06 ± 0.05	595.5 ± 11.7	588.4 ± 19.8	26.1 ± 14.7	191.5 ± 40.4
3	0.09 ± 0.06	597.5 ± 14.2	590.1 ± 8.1	18.9 ± 9.1	197.4 ± 168.1
4	0.05 ± 0.03	596.7 ± 7.7	599.1 ± 13.6	30.0 ± 20.3	283.0 ± 195.8
5	0.06 ± 0.06	594.1 ± 12.2	591.2 ± 9.6	31.7 ± 16.2	245.5 ± 120.9
6	0.07 ± 0.05	596.1 ± 11.1	598.4 ± 16.2	16.1 ± 16.8	176.9 ± 136.5
7	0.06 ± 0.04	599.1 ± 10.2	598.2 ± 20.9	25.8 ± 20.4	214.4 ± 166.9
8	0.08 ± 0.05	589.8 ± 15.9	592.2 ± 14.3	30.4 ± 24.9	198.4 ± 123.4
9	0.07 ± 0.05	598.0 ± 21.1	589.9 ± 15.8	15.6 ± 6.4	156.3 ± 90.8
10	0.04 ± 0.05	590.6 ± 9.8	594.1 ± 10.1	40.2 ± 30.5	183.1 ± 143.0
11	0.04 ± 0.05	596.1 ± 12.0	598.3 ± 13.5	24.4 ± 18.3	190.6 ± 111.2
12	0.08 ± 0.05	596.5 ± 17.6	597.6 ± 18.6	19.2 ± 17.8	209.4 ± 151.8
13	0.02 ± 0.02	595.8 ± 23.8	592.7 ± 16.3	34.5 ± 19.9	276.5 ± 159.9
14	0.03 ± 0.07	597.3 ± 12.6	596.4 ± 21.2	40.6 ± 47.6	209.4 ± 199.4
15	0.05 ± 0.05	595.3 ± 16.6	593.1 ± 11.9	28.0 ± 27.5	197.6 ± 153.0
16	0.07 ± 0.05	595.7 ± 16.9	591.7 ± 8.2	35.8 ± 43.5	278.1 ± 185.0
17	0.07 ± 0.05	590.1 ± 24.8	597.4 ± 14.2	43.1 ± 46.0	220.8 ± 143.6
18	0.07 ± 0.04	597.5 ± 23.4	588.3 ± 19.9	26.6 ± 8.9	264.9 ± 243.9
M	0.06	595.5	594.1	28.4	216.8
SD	0.02	2.7	3.7	8.2	37.4

Note. K-F, Kucera-Francis; T-L, Thorndike-Lorge. The latent semantic analysis similarity rating ranges from 0.0 to 1.0, with 0.0 indicating no similarity between the members of a word-pair, and 1.0 for identical words. Concreteness and imageability values range 100 – 700, with higher scores respectively indicating greater concreteness and imageability.

Table 7.

Measures of Normality for the Validation Task, by participant group

Dependent Variable	Item Type	Control (N = 14)		TBI survivors (N = 17)	
		Skewness	Kurtosis	Skewness	Kurtosis
Percent Old	Prime	-0.899	0.501	-1.229	1.596
	New	1.160	2.007	0.045	-1.220
	Antiprime	-0.032	-1.553	0.522	-0.812
Response Time	Prime	0.644	-0.673	0.367	-0.458
	New	1.057	1.007	1.084	0.712
	Antiprime	0.749	1.444	0.992	1.366

Note: Scores within the range of -1 to +1 indicate normality

Table 8.

Means and Standard Deviations for Percent Old and Response Times, for each participant group, in the Validation Task

Dependent Variable	Item Type	Control (n = 14)	TBI survivors (n = 17)
Percent Old (%)	Prime	90.82 +/- 8.60	86.13 +/- 11.14
	New	25.51 +/- 20.91	26.05 +/- 18.37
	Antiprime	13.27 +/- 10.80	19.75 +/- 13.25
Response Time (ms)	Prime	1227.29 +/- 275.28	1631.35 +/- 416.69
	New	1706.69 +/- 555.44	2096.69 +/- 590.92
	Antiprime	1582.51 +/- 432.80	1866.13 +/- 502.15

Table 9.

Repeated Measures Analysis of Variance for Percent Old and Response Time in the Validation Task

Analysis and Source	<i>Huynh-Feldt df</i>	<i>F</i>	<i>p</i>	η^2
<u>Percent Old</u>				
Group x Item Type ANOVA				
Between Subjects				
Group	1	.051	0.823	.002
Error	29			
Within Subjects				
Item type	2	264.150	< .001	.901
Group x Item Type	28	2.202	.129	.136
Error	58			
<u>Response Time</u>				
Group x Item Type ANOVA				
Between Subjects				
Group	1	6.810	.014	.190
Error	29			
Within Subjects				
Item type	1.832	14.137	< .001	.328
Group x Item Type	28	.418	.663	.029
Error	53.130			

Table 10.

Pairwise Comparison for the Effects of Item Type on Percent Old and Response Times, in the Validation Task

Dependent Variable	(I) Item Type	(J) Item Type	Mean Diff. (I-J)	Standard Error	Sig. *	95% Confidence Interval for Difference ^a	
						Lower Bound	Upper Bound
Percent							
Old (%)	AP	PR	-71.969*	3.132	.000	-79.927	-64.010
		New	-9.274*	2.693	.005	-16.117	-2.430
	PR	AP	71.969*	3.132	.000	64.010	79.927
		New	62.695*	4.213	.000	51.990	73.400
	New	AP	9.274*	2.693	.005	2.430	16.117
		PR	-62.69*	4.213	.000	-73.400	-51.990
RT (ms)							
RT (ms)	AP	PR	294.998*	91.216	.009	63.226	526.771
		New	-177.4*	69.869	.050	-354.91	.156
	PR	AP	-294.9**	91.216	.009	-526.77	-63.226
		New	-472.37*	104.704	.000	-738.42	-206.329
	New	AP	177.375*	69.869	.050	-.156	354.906
		PR	472.374*	104.704	.000	206.329	738.418

Note: AP = antiprime stimuli; PR = prime targets; New = not previously used in the experiment

* The mean difference is significant at the .05 level.

a Adjustment for multiple comparisons: Bonferroni.

Table 11.

Measures of Normality for each Cell of the Implicit Metamemory Task, by Participant Group

Dependent Variable	JOL timing condition	Priming Condition	Control (N = 13)		TBI survivors (N= 15)	
			Skewness	Kurtosis	Skewness	Kurtosis
JOL	Immediate	Baseline	.663	-.015	.197	-.372
		Prime	.316	-.874	.353	-.757
		Antiprime	.312	-.489	.211	-.373
	Delayed	Baseline	-.008	-1.251	.241	.881
		Prime	-.254	-1.018	.708	.501
		Antiprime	.033	-1.591	.573	.928
Recall Acc.	Immediate	Baseline	-.021	-1.078	1.218	.764
		Prime	-.227	-1.569	.464	-.562
		Antiprime	-.039	-1.158	-.001	-.698
	Delayed	Baseline	-.285	-1.600	.592	1.468
		Prime	-.366	-1.228	1.674	4.823
		Antiprime	-.616	-1.282	.462	-.110
G-K Gamma	Immediate	Baseline	.213	-1.239	-.585	-.510
		Prime	-.523	-.629	-1.519	3.179
		Antiprime	-.293	-1.207	-1.098	.511
	Delayed	Baseline	-.949	-.737	-1.153	1.462
		Prime	-1.084	.234	-2.184	5.286
		Antiprime	-2.746	7.974	-.494	-.998
JOL RT	Immediate	Baseline	.000	-.859	.721	-.467
		Prime	.326	-.281	.678	-.383
		Antiprime	-.006	-1.698	.667	-.374
	Delayed	Baseline	.885	.336	.791	-.808

		Prime	.164	1.402	.874	-.635
		Antiprime	-.172	-.128	.915	-.497
Recall RT	Immediate	Baseline	.121	-.672	.820	-.342
		Prime	.714	-.063	.838	-.640
		Antiprime	-.130	-1.596	1.113	-.093
	Delayed	Baseline	.330	-1.364	.826	-.951
		Prime	-.048	-1.833	.785	-.462
		Antiprime	.654	-.894	.514	-.956

Note: Scores within the range of -1 to +1 indicate normality

Table 12.

Mauchly's Test of Sphericity Results for All Dependent Measures in Implicit Metamemory task, for the Main Effect of Prime and the Prime-by-JOL timing Interaction.

Analysis and Source	<i>Huynh-Feldt df</i>	<i>W</i>	<i>p</i>	<i>Approx. χ^2</i>
<u>JOL</u>				
Group x Prime x JOL timing ANOVA				
Within Subjects				
Prime	2	.609	.002	12.909
Prime-by-JOL timing	2	.979	.754	.564
Error (prime)	54.000			
Error (prime x JOL timing)	54.000			
<u>Recall Accuracy</u>				
Group x Prime x JOL timing ANOVA				
Within Subjects				
Prime	2	.934	.413	1.767
Prime-by-JOL timing	2	.955	.548	1.202
Error (prime)	54.000			
Error (prime x JOL timing)	54.000			
<u>Gamma Correlation</u>				
Group x Prime x JOL timing ANOVA				
Within Subjects				
Prime	2	.887	.285	2.511
Prime-by-JOL timing	2	.957	.632	.917

Error (prime)	44.000			
---------------	--------	--	--	--

Error (prime x JOL timing)	44.000			
----------------------------	--------	--	--	--

JOL RT

Group x Prime x JOL timing
ANOVA

Within Subjects

Prime	2	.850	.132	4.048
-------	---	------	------	-------

Prime-by-JOL timing	2	.998	.970	.060
---------------------	---	------	------	------

Error (prime)	50.067			
---------------	--------	--	--	--

Error (prime x JOL timing)	52.000			
----------------------------	--------	--	--	--

Recall RT

Group x Prime x JOL timing
ANOVA

Within Subjects

Prime	2	.412	<.001	22.171
-------	---	------	-------	--------

Prime-by-JOL timing	2	.718	.016	8.279
---------------------	---	------	------	-------

Error (prime)	34.957			
---------------	--------	--	--	--

Error (prime x JOL timing)	44.346			
----------------------------	--------	--	--	--

Table 13.

Descriptive Statistics for the Implicit Metamemory Task: Means and Standard Deviations for JOL, Recall Accuracy, Gamma Correlation, JOL RT, and Recall RT, for each participant group.

Dependent Variable	JOL timing condition	Priming Condition	Control (n = 14)	TBI survivors (n = 15)
JOL	Immediate	Baseline	41.71 +/- 23.40	47.07 +/- 21.98
		Prime	45.50 +/- 24.23	47.36 +/- 21.62
		Antiprime	44.28 +/- 22.86	45.08 +/- 20.09
	Delayed	Baseline	44.90 +/- 25.50	40.69 +/- 17.65
		Prime	42.18 +/- 22.76	37.85 +/- 17.73
		Antiprime	42.19 +/- 24.75	37.71 +/- 18.42
Recall Accuracy	Immediate	Baseline	.345 +/- .214	.255 +/- .173
		Prime	.443 +/- .246	.261 +/- .135
		Antiprime	.423 +/- .269	.292 +/- .145
	Delayed	Baseline	.511 +/- .311	.318 +/- .164
		Prime	.454 +/- .260	.302 +/- .174
		Antiprime	.477 +/- .246	.377 +/- .207
G-K Gamma	Immediate	Baseline	.342 +/- .315	.414 +/- .438
		Prime	.310 +/- .419	.471 +/- .491
		Antiprime	.272 +/- .423	.532 +/- .365
	Delayed	Baseline	.761 +/- .283	.810 +/- .160
		Prime	.844 +/- .155	.900 +/- .162
		Antiprime	.891 +/- .192	.847 +/- .132

Table 14.

Descriptive Statistics for the Implicit Metamemory Task: Means and Standard Deviations for JOL RT, and Recall RT, for each participant group.

Dependent Variable	JOL timing condition	Priming Condition	Control (n = 13)	TBI survivors (n = 15)
JOL RT	Immediate	Baseline	2152.6 +/- 518.1	3251.9 +/- 1219.3
		Prime	2190.0 +/- 409.7	3243.3 +/- 1192.6
		Antiprime	2197.4 +/- 414.3	3168.1 +/- 1091.4
	Delayed	Baseline	3094.2 +/- 739.4	5439.2 +/- 2460.1
		Prime	3203.0 +/- 898.1	5460.0 +/- 2554.6
		Antiprime	3167.9 +/- 923.4	5132.2 +/- 2374.2
Recall RT	Immediate	Baseline	5655.7 +/- 2254.4	10396.7 +/- 6530.8
		Prime	5695.9 +/- 2627.0	11402.6 +/- 7404.6
		Antiprime	5551.7 +/- 2354.6	10998.0 +/- 7771.0
	Delayed	Baseline	3945.8 +/- 1385.5	9776.3 +/- 7166.9
		Prime	4766.9 +/- 1657.0	8900.7 +/- 5643.3
		Antiprime	10843.6 +/- 7075.0	8906.8 +/- 5318.3

Table 15.

Repeated Measures Analysis of Variance for JOL, Recall Accuracy, Gamma Correlation, JOL RT, and Recall RT in the Implicit Metamemory Task

Analysis and Source	<i>Hypothesis df*</i>	<i>Error df*</i>	<i>F</i>	<i>p</i>	η^2
<u>JOL</u>					
Prime x JOL timing x Group ANOVA					
Within Subjects					
Prime	1.553*	41.918*	.809	.424	.029
JOL timing	1	27	4.679	.040	.148
Prime x JOL timing	2	26	3.140	.060	.195
Prime x Group	1	27	.012	.914	.000
JOL timing x Group	1	27	3.189	.085	.106
Prime x JOL timing x Group	2	54	.613	.545	.022
Between Subjects					
Group	1	27	.012	.914	.000
 <u>Recall Accuracy</u>					
Prime x JOL timing x Group ANOVA					
Within Subjects					
Prime	2	54	1.724	.188	.060
JOL timing	1	27	20.198	< .001	.428
Prime x JOL timing	2	26	4.258	.025	.247
Prime x Group	2	52	8.577	.001	.248
JOL timing x Group	1	26	11.365	.002	.304
Prime x JOL timing x Group	2	26	2.776	.081	.176

Between Subjects					
Group	1	2	3.644	.067	.119
<u>Gamma Correlation</u>					
Prime x JOL timing x Group ANOVA					
Within Subjects					
Prime	2	44	.682	.511	.030
JOL timing	1	22	63.055	< .001	.741
Prime x JOL timing	2	44	.241	.787	.011
Prime x Group	2	21	.145	.865	.007
JOL timing x Group	1	22	1.591	.220	.067
Prime x JOL timing x Group	2	21	.810	.451	.036
Between Subjects					
Group	1	22	1.355	.257	.058
<u>JOL RT</u>					
Prime x JOL timing x Group ANOVA					
Within Subjects					
Prime	1.926*	50.067*	1.029	.362	.038
JOL timing	1	26	26.559	< .001	.505
Prime x JOL timing	2	52	.725	.489	.027
Prime x Group	2	25	1.511	.230	.055
JOL timing x Group	1	26	3.645	.067	.123
Prime x JOL timing x Group	2	25	.587	.559	.022
Between Subjects					
Group	1	26	12.105	.002	.318
<u>Recall RT</u>					
Prime x JOL timing x Group ANOVA					

Within Subjects					
Prime	1.344*	34.957*	7.033	.077	.213
JOL timing	1	26	1.204	.283	.044
Prime x JOL timing	1.706*	44.346*	7.041	<.001	.287
Prime x Group	2	25	4.893	.016	.281
JOL timing x Group	1	26	11.365	.002	.304
Prime x JOL timing x	2	25	6.992	.004	.359
Group					
Between Subjects					
Group	1	26	4.508	.043	.148

* Huynh-Feldt corrected degrees of freedom are used for variables with significant Mauchly's test of sphericity.

Table 16.

Measures of Normality for the Visual Antipriming Task, by participant group

Dependent Variable	Prime Condition	Control (N = 13)		TBI (N = 15) survivors	
		Skewness	Kurtosis	Skewness	Kurtosis
Accuracy	Baseline	-.405	-1.267	-.469	-.895
	Prime	-1.503	3.940	-.477	.378
	Antiprime	-1.097	.825	-.922	2.062
Response Time	Baseline	2.707	8.510	-.061	-1.491
	Prime	.832	.375	1.514	3.977
	Antiprime	.765	-.237	1.385	1.676

Note: Scores within the range of -1 to +1 indicate normality

Table 17.

Means and Standard Deviations for Recall Accuracy and Response Times, for each participant group, in the Visual Antipriming task

Dependent Variable	Item Type	Control (n = 13)	TBI survivors (n = 15)
Accuracy (%)	Baseline	75.69 +/- 9.96	69.53 +/- 12.51
	Prime	89.38 +/- 7.97	84.40 +/- 10.99
	Antiprime	74.31 +/- 10.19	68.27 +/- 14.24
Response Time (ms)	Baseline	1012.59 +/- 191.45	1180.09 +/- 195.78
	Prime	974.45 +/- 151.50	1164.89 +/- 197.22
	Antiprime	1047.04 +/- 119.75	1354.26 +/- 388.68

Table 18.

Repeated Measures Analysis of Variance for Accuracy and Response Time in the Visual Antipriming Task

Analysis and Source	<i>Hypothesis df*</i>	<i>Error df*</i>	<i>F</i>	<i>p</i>	η^2
<u>Accuracy</u>					
Group x Prime ANOVA					
Between Subjects					
Group	1	26	2.325	.139	.082
Within Subjects					
Prime	2	52	48.175	<.001	.649
Group x Prime	2	25	.067	.935	.003
<u>Response Time</u>					
Group x Prime ANOVA					
Between Subjects					
Group	1	26	9.222	.005	.262
Within Subjects					
Prime	2	52	5.843	.005	.183
Group x Prime	2	52	17.12	.191	.062

* Huynh-Feldt corrected degrees of freedom are used for variables with significant Mauchly's test of sphericity.

Table 19.

Repeated Measures Analysis of Variance for Difference Scores in the Implicit
Metamemory Task

Analysis and Source	<i>Hypothesis df</i>	<i>Error df</i>	<i>F</i>	<i>p</i>	η^2
<u>Difference Score</u>					
Prime x JOL timing x Group ANOVA					
Within Subjects					
Prime	2	26	6.053	.007	.318
JOL timing	1	27	28.758	< .001	.516
Prime x JOL timing	2	26	.845	.441	.061
Prime x Group	2	26	2.219	.129	.146
JOL timing x Group	1	27	1.791	.192	.062
Prime x JOL timing x Group	2	26	1.422	.259	.099
Between Subjects					
Group	1	27	4.607	.041	.146

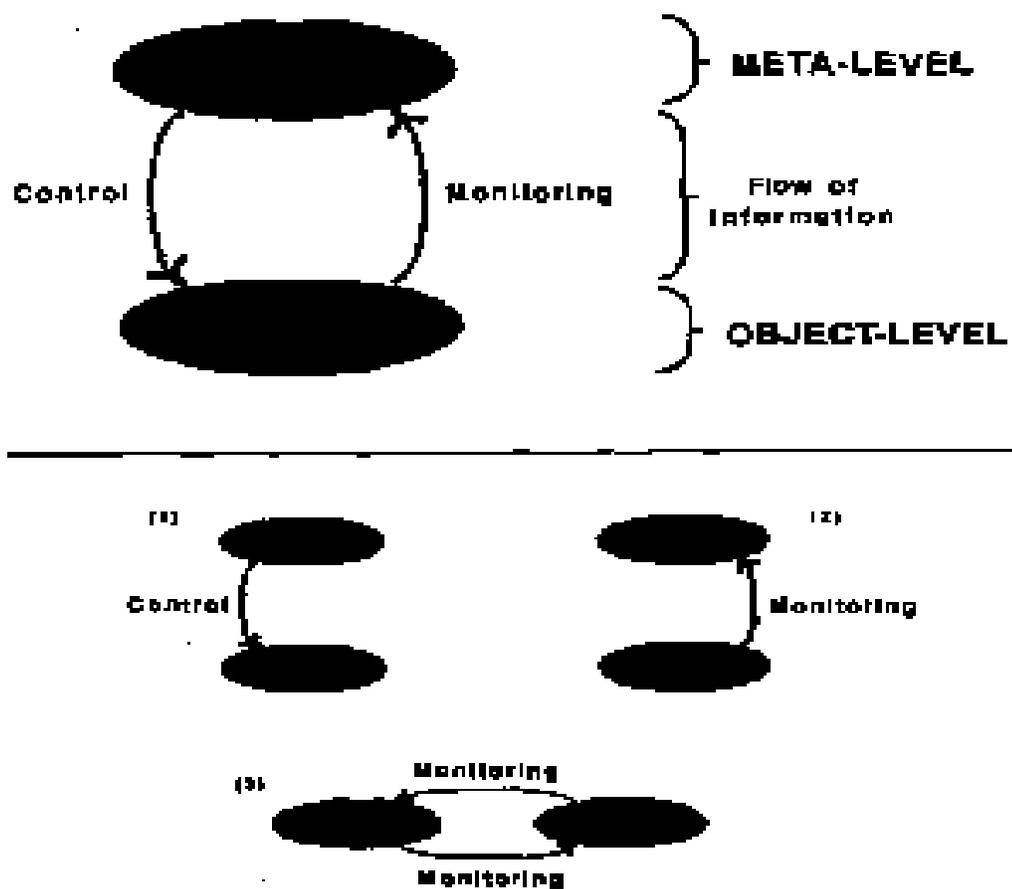


Figure 1. Nelson and Narens' (1990) model of metacognitive monitoring and control.

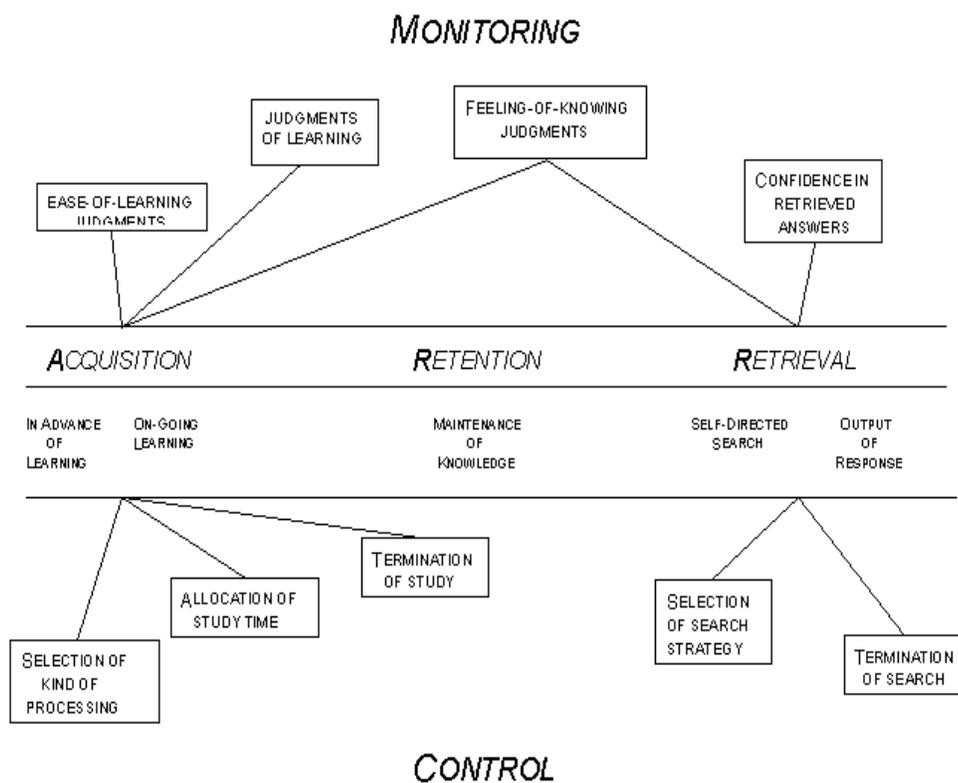


Figure 2. Nelson and Narens' (1994) model of metamemory monitoring and control.

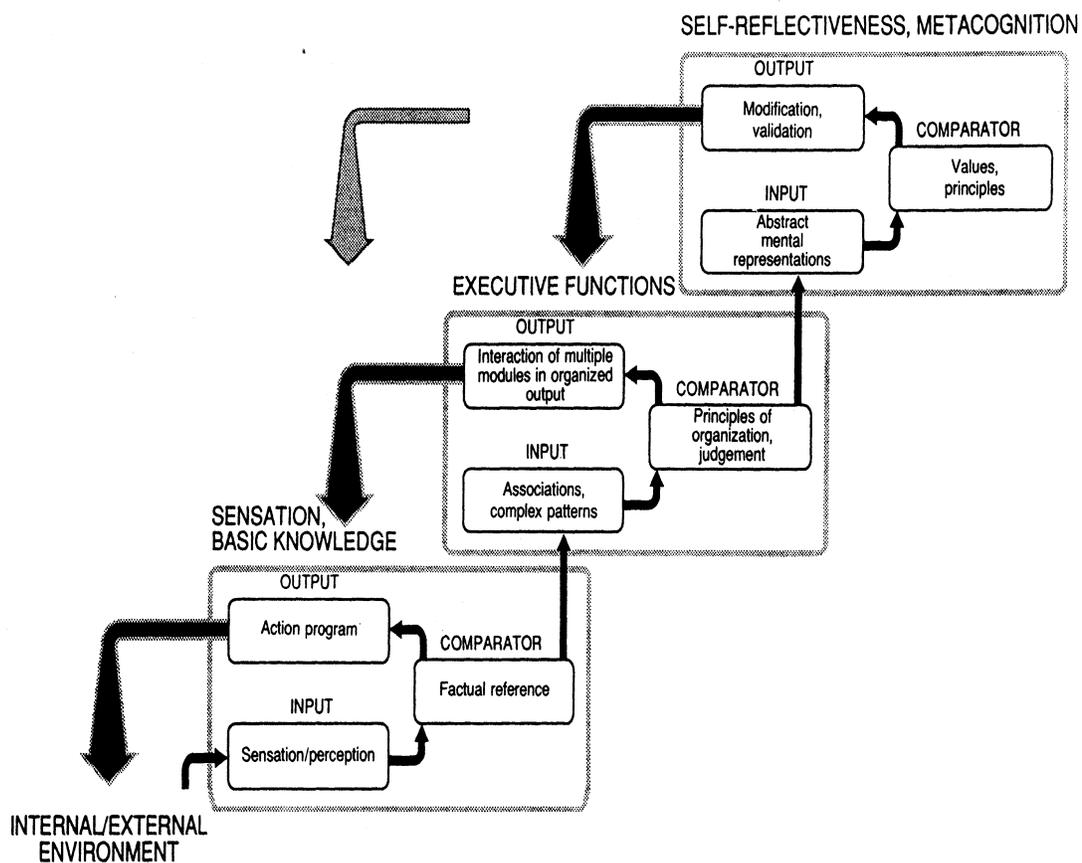


Figure 3. Recursive hierarchical model of frontal lobe function, Stuss (1991).

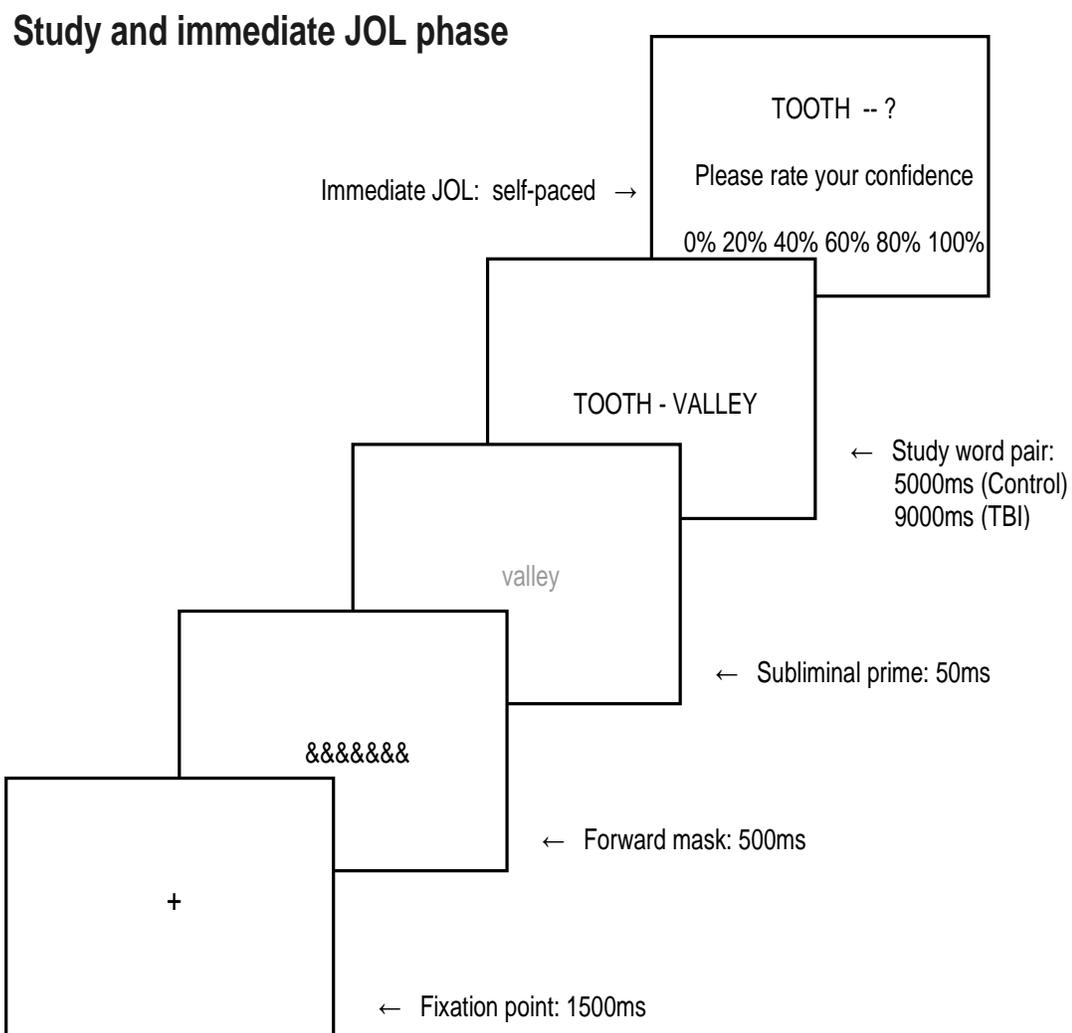


Figure 4. Sequence of task steps for the paired associate learning/implicit metamemory task.

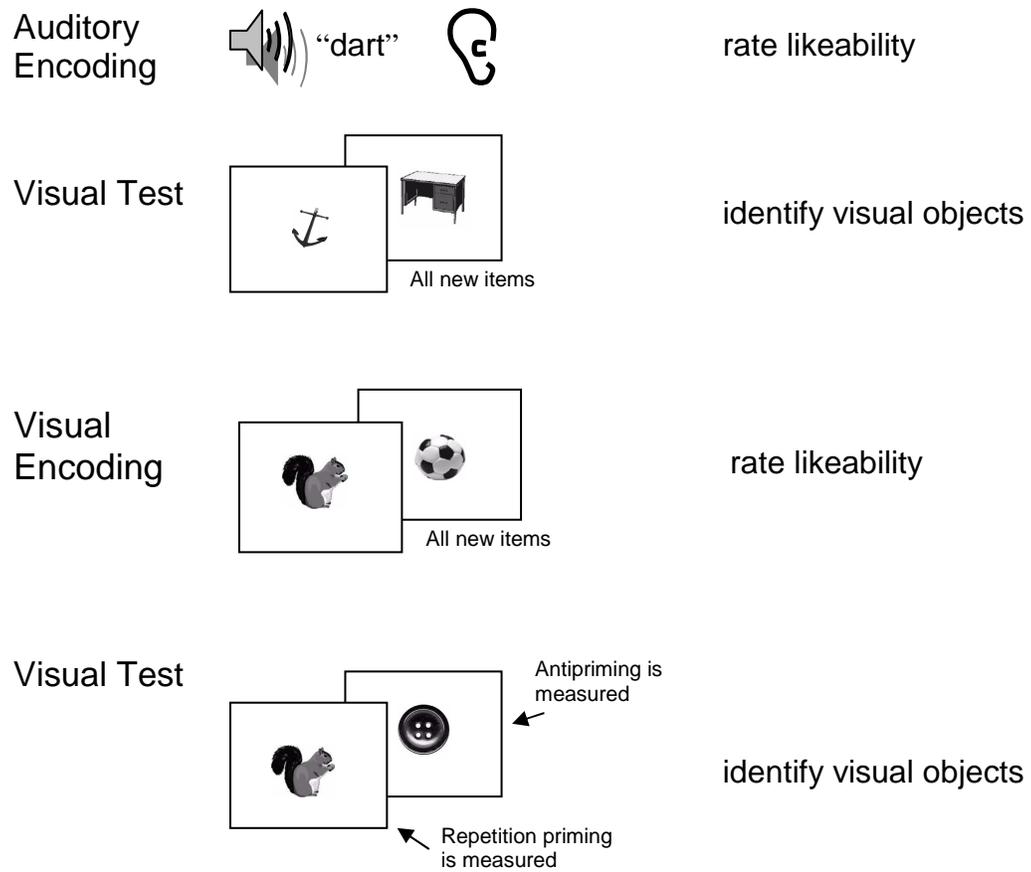
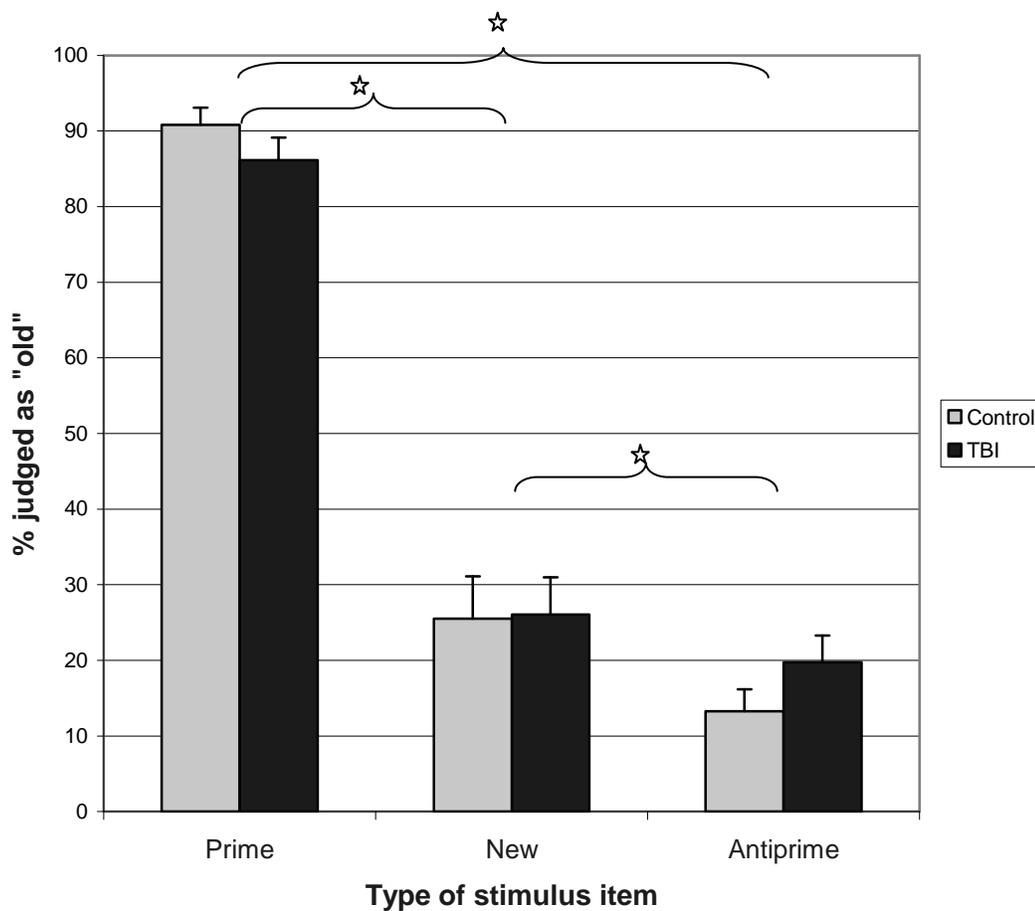


Figure 5. Sequence of task steps for the visual antipriming task (experiment number four of Marsolek, 2006).



Figure 6. Examples of the greyscale visual objects used in the visual antipriming experiment.



☆ Differences are statistically significant

Figure 7. Results of the validation task immediately following the last trial block of the implicit metamemory task. Both participant groups judged both new and subliminally presented antiprime items (“antiprime”) as “old” significantly less often than those items which had been studied and subliminally primed (“prime”). Also, antiprime items were judged “old” significantly less often than “new” items.

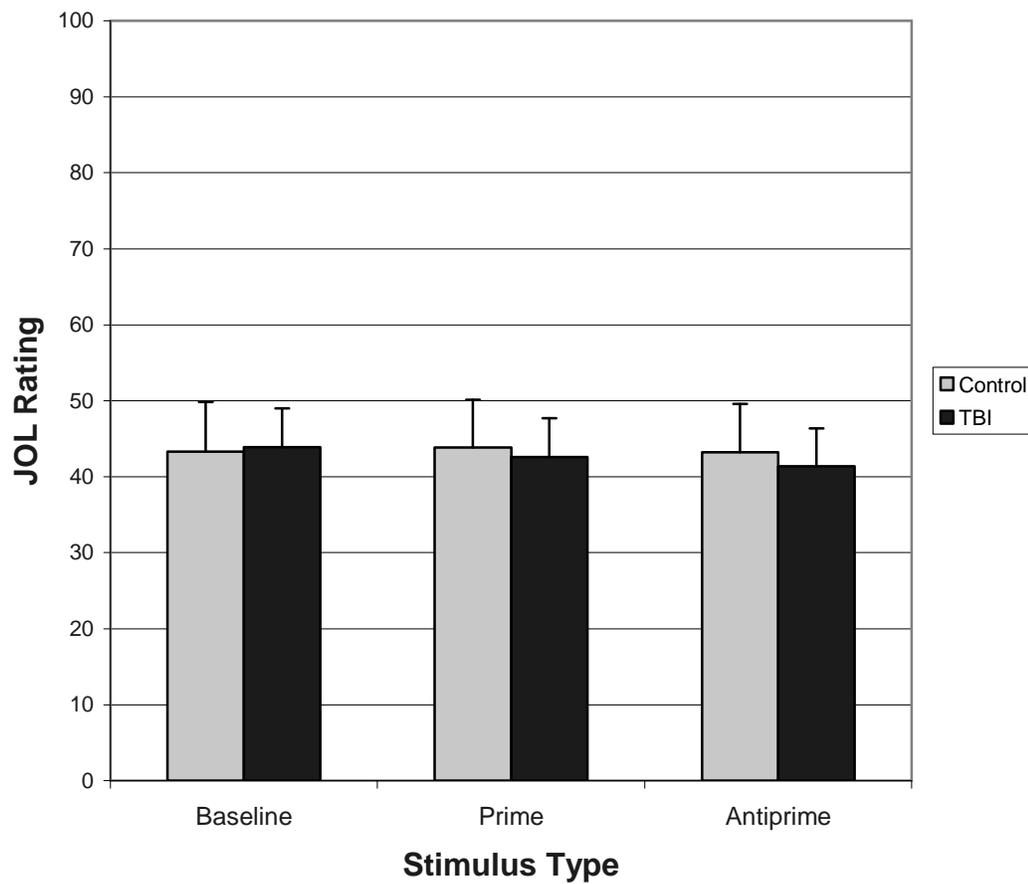


Figure 8. Subliminal masked priming and antipriming of target words does not affect JOL ratings (collapsed across JOL timing condition) for either participant group.

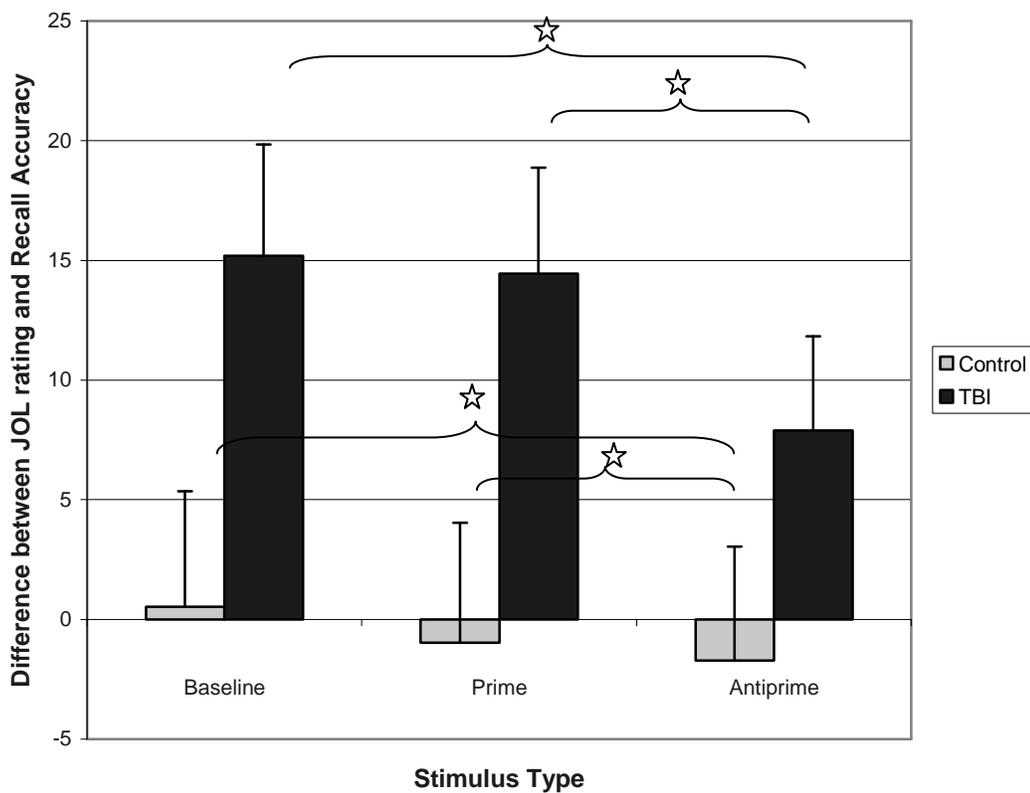
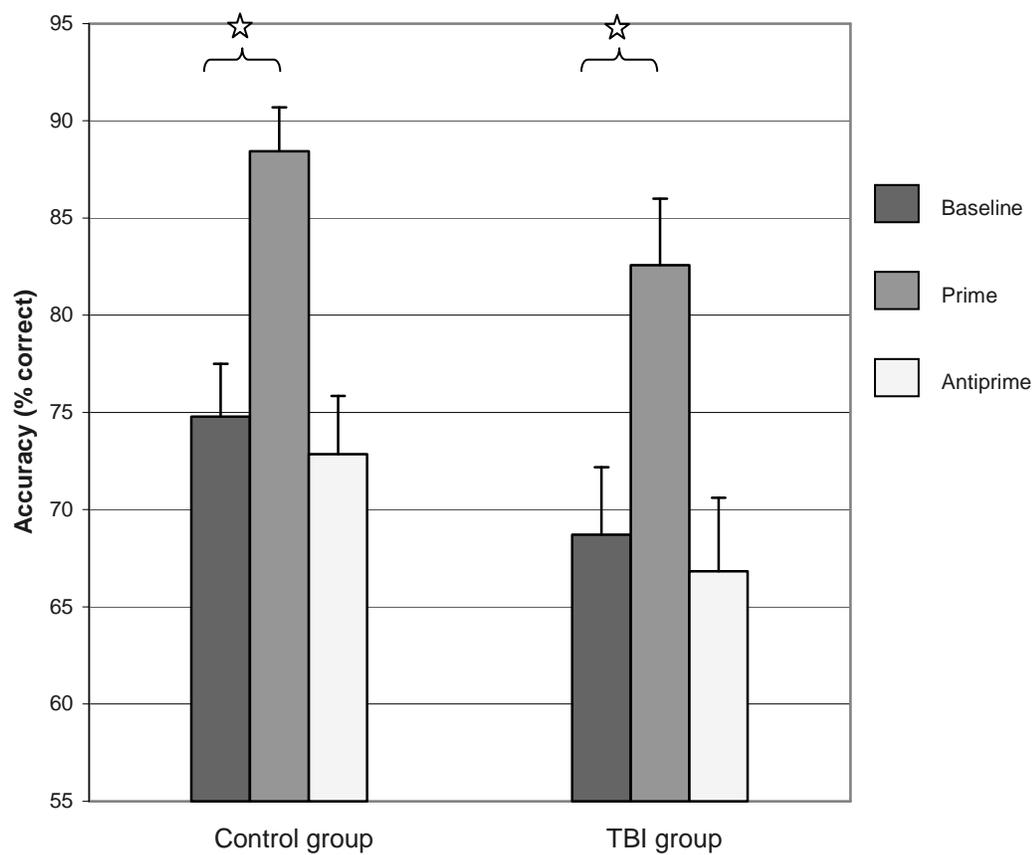
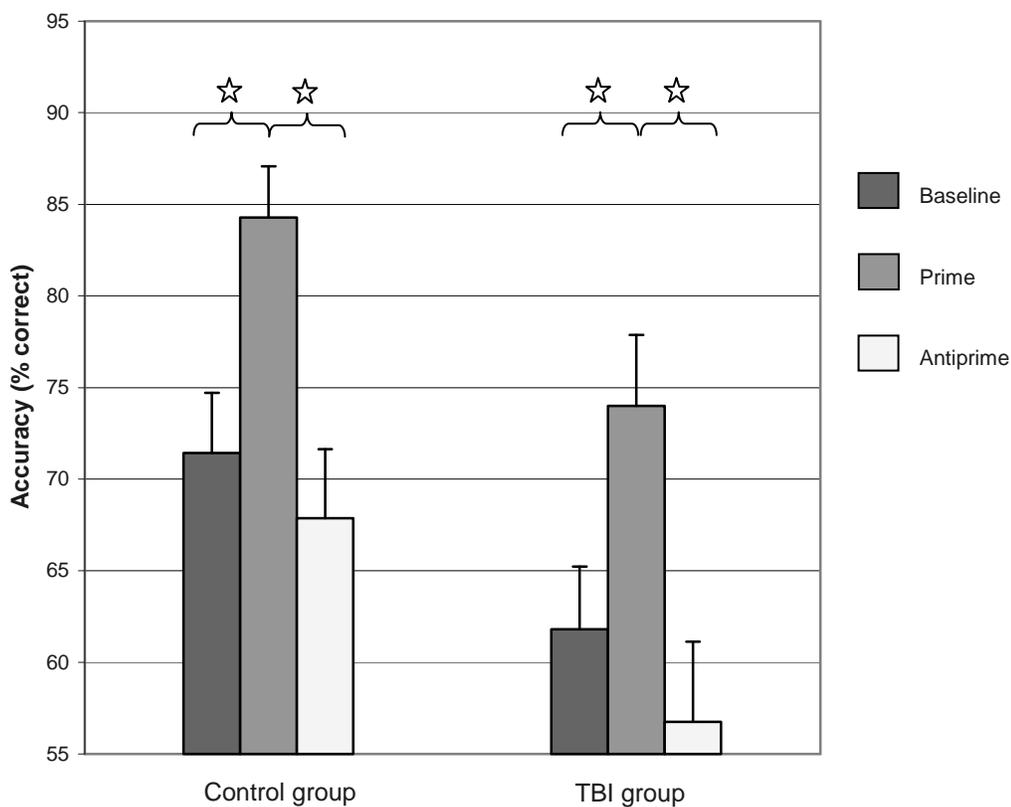


Figure 9. Collapsed across JOL timing condition, the TBI survivor group shows a significant reduction in confidence (difference score between JOL rating and recall accuracy) for antiprimed items. They are also generally overconfident, while the control group is well calibrated.



☆ Differences are statistically significant

Figure 10. Long term repetition priming in visual object identification task results in significantly increased identification accuracy for both participant groups. Antipriming has non-significantly decreased accuracy. All responses were self-paced.



☆ Differences are statistically significant

Figure 11. When responses in the visual object identification task were rejected if they took longer than two seconds, the decrease in object identification accuracy for antiprimed items becomes statistically significant for both participant groups.

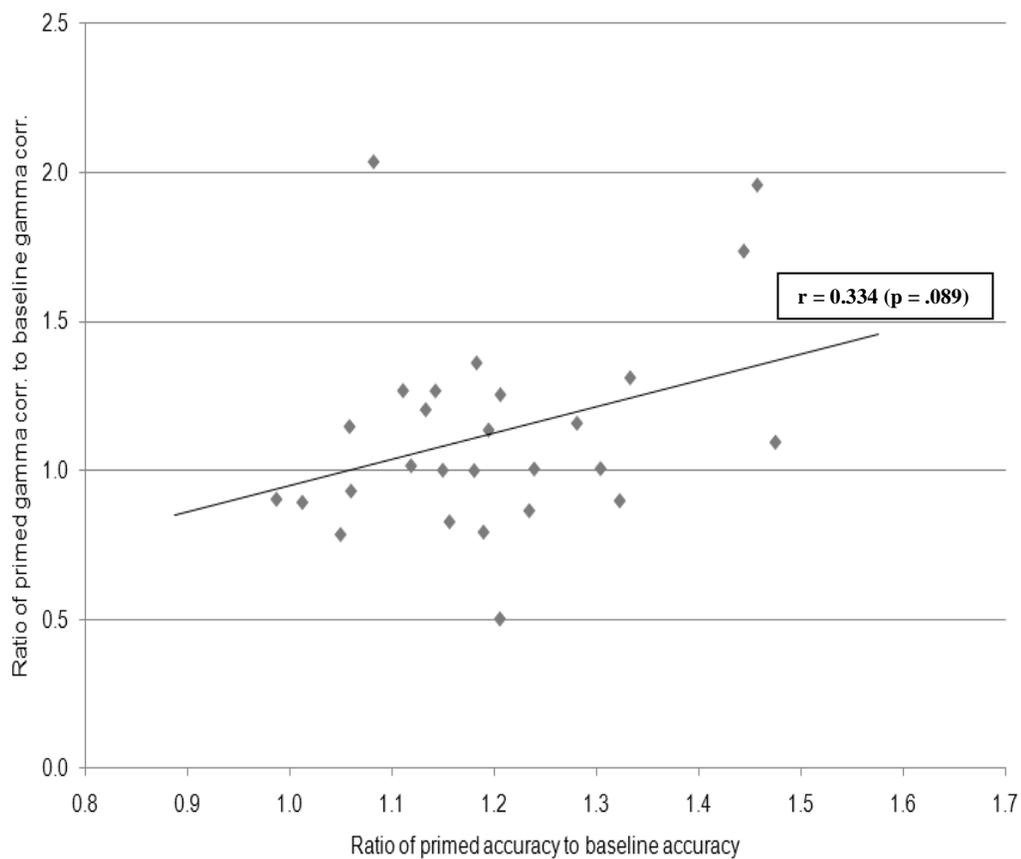


Figure 12. Weak correlation [$r = 0.334$ ($p = .089$)] between the relative benefit of priming for gamma correlation to the priming benefit to object identification accuracy (where priming benefit is defined as the ratio of primed values to baseline values).

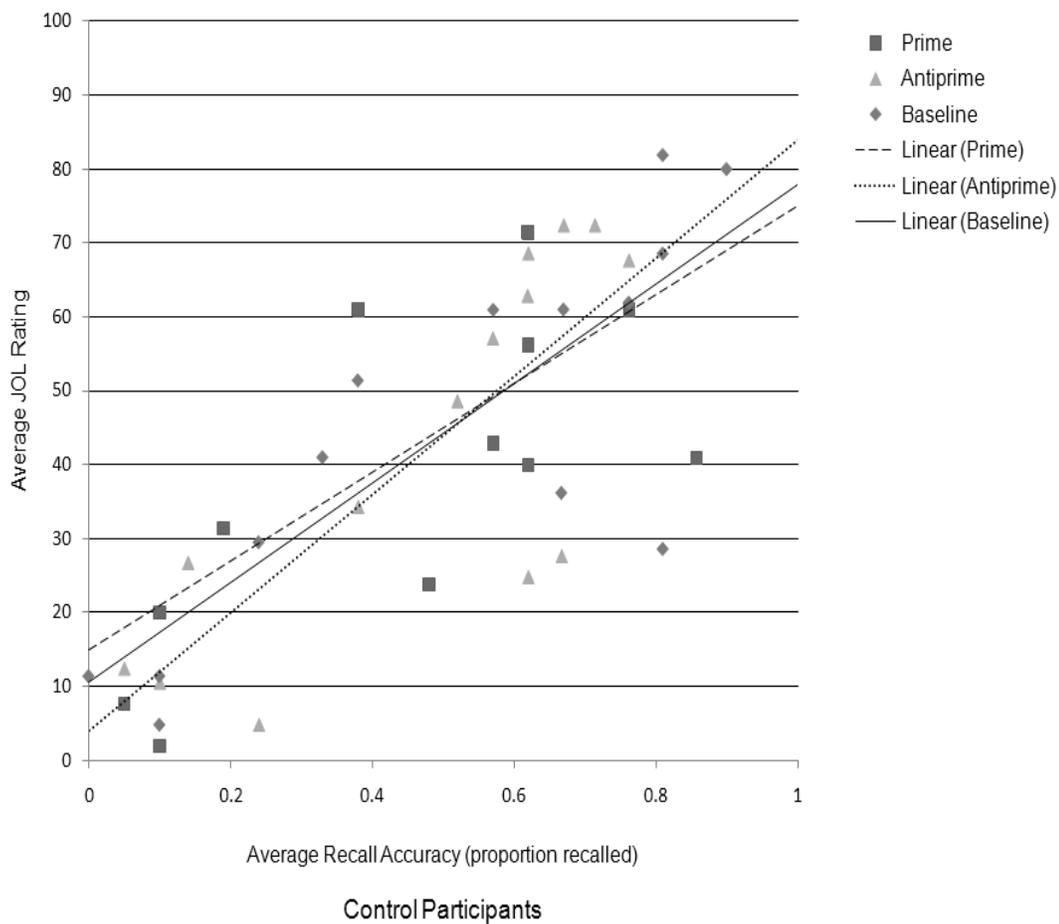


Figure 13. Each data point represents average JOL vs. average recall accuracy for all items for one individual in the delayed JOL timing condition. Relative to baseline, antipriming decreases JOL for controls with poor recall, and increases it for those with high recall.

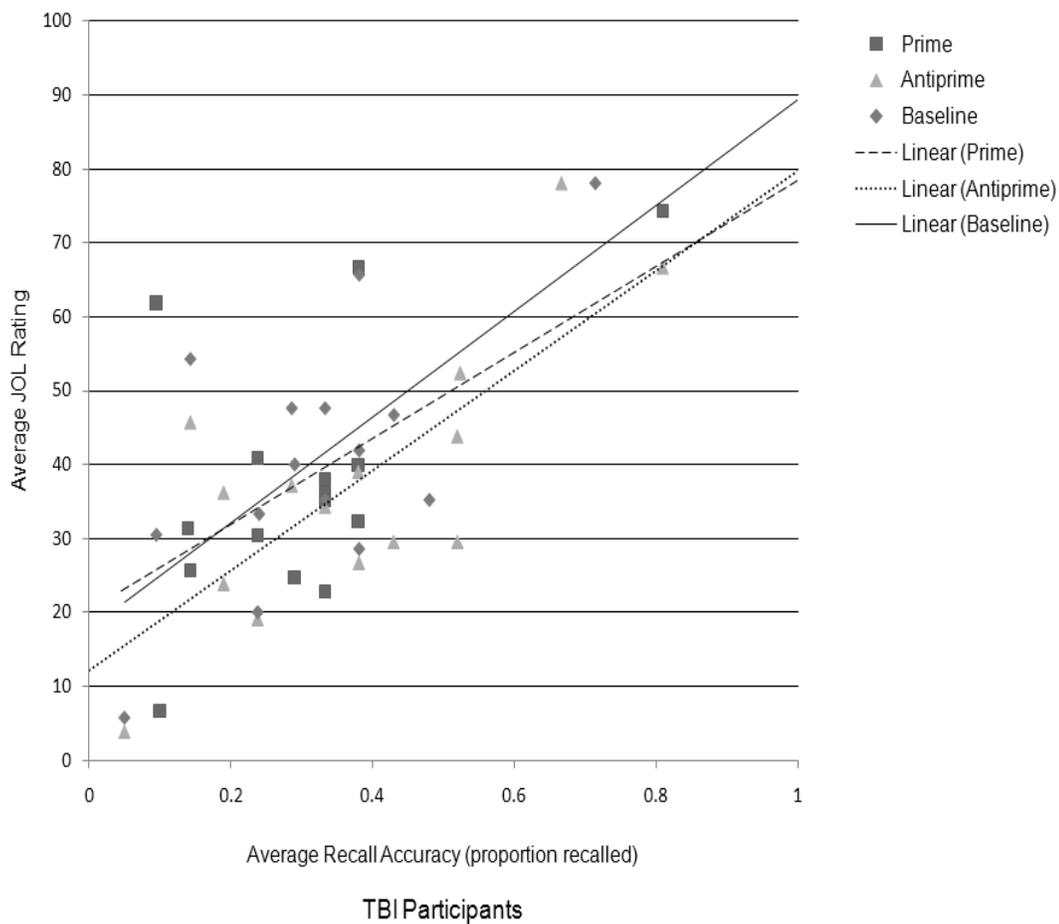


Figure 14. Each data point represents average JOL vs. average recall accuracy for all items for one individual in the delayed JOL timing condition. Relative to the baseline trend, antipriming produces an overall reduction in JOL for TBI survivors across the range of recall accuracy, relative to baseline.

Footnotes

¹ I consulted with Dr. Ming Chen, Ph.D., of the department of Statistics at the University of Connecticut regarding the appropriateness of multiple separate ANOVAs vs. a single MANOVA. Upon detailed review of the data and analyses, as well as conducting his own analyses using the present data, he concluded that although MANOVA would seem to be the method of choice on theoretical grounds, the results could not be interpreted without a detailed analysis including Bayesian modeling and Monte Carlo simulations (M. Chen, personal communication, May 4, 2009). Whereas these are beyond the scope of the present research, he advised using the analytical methods I have currently employed.

APPENDIX A

Telephone Interview form for control participants

Subject Code# _____

Phone contact date: _____

Study of Learning and Memory**Initial Telephone Contact Protocol - CONTROL Subjects**

After the investigator(s) has (have) received a postcard, or telephone call from a prospective subject indicating interest and willingness to discuss possible participation in this study, the following is the initial telephone contact protocol that will be used during this first contact.

Greetings and Introductions

"Hi, I'm Pradeep Ramanathan, from the Department of Speech-Language-Hearing Science, at the University of Minnesota. I received a postcard/phone message recently, indicating that you would be willing to discuss your possible participation in our study on learning and memory. I would like to discuss that with you now. Are you still interested in hearing more about this study?

Is this a good time to talk? Or should I call you back at a later time?"

Brief Description of the Study

Assuming they indicate willingness to continue, a description of the study would follow.

"I'd like to tell you a bit more about this study. This study is designed to determine how people who have survived a traumatic brain injury are able to remember new information after their injury. You would be participating in this study as a control subject. Control subjects are those individuals who are similar to the other subjects, except that in this study, the control subjects have not had a brain injury.

"We are interested in studying learning and memory after a brain injury, because there are still many aspects of this that remain a mystery to therapists and researchers. In this study, you will be first asked to take some tests in which you do some listening, speaking, naming pictures, and so on, just to make sure that you will be able to do the rest of the activities in the study. If you are able to do all these things, you will participate in a second session. At that time you will be asked to name pictures presented by a computer.

You will also be asked to read words presented on a computer screen. After reading the words, you will be asked to try to recall some of the words. If you should have difficulty with the first part of the study, that is with the listening, speaking, naming, etc., then you will likely not participate in the rest of the study.

You will receive approximately \$8.00 per hour for your participation. The entire study will take two sessions of approximately 3 hours each, for a total of about 6 hours. You will receive \$50.00 if you complete the whole study.

“If you are still interested, I'd like to spend about 5 minutes asking you some questions. You are free to decline to answer any of the questions that you do not wish to answer. Some questions have to do with your prior experiences in school, but others are more personal, like 'When you were in school, did you ever have difficulty learning how to read or write?' and 'Have you ever experienced an extended period of alcohol abuse?' and so on. Shall I proceed?”

If the individual indicates the investigator may continue, the following questions will be asked:

Controls - Phone Screening Questions:

1. Are you a high school graduate? **Yes** or **No**
2. What is the last grade you completed? _____
3. How old are you? _____ What is your birthday? _____
4. Is English your first language? _____
 - If NO - when did you learn English? _____

NOTE: if “after age 5”, candidate cannot participate; politely end telephone screening
5. When you were in school, did you have any difficulty learning to read or write? **Yes** or **No**
 - If yes, please explain: _____

6. Did you ever receive speech therapy, or remedial help for anything in school? **Yes** or **No**
 - If yes, please explain: _____

7. Were you ever told that you had a learning disability?

8. Did you ever participate in classes for gifted students, or did you ever skip a grade?

Yes or **No**

9. To the best of your knowledge, are your hearing AND vision adequate? **Yes** or

No

- If no, please explain:

10. Have you ever experienced any of the following?

- | | |
|---|-------------------------|
| <input type="radio"/> Stroke | Yes or No |
| <input type="radio"/> Neurological Disease | Yes or No |
| <input type="radio"/> Extended alcohol abuse | Yes or No |
| <input type="radio"/> Drug abuse | Yes or No |
| <input type="radio"/> Hospitalized for psychological difficulty | Yes or No |
| <input type="radio"/> Periods of unconsciousness | Yes or No |
| <input type="radio"/> Previous head or brain injury | Yes or No |

- If yes to any of these, please

explain:_____

11. Do you have any problems walking or moving, that would keep you from coming to the University to participate in this study? **Yes** or **No**

- If yes, please explain:_____

12. What type of work do you do? _____

13. Do you sign legal documents yourself, or do you have a co-signer? If you have a co-signer, who is that person? _____

14. Do you require any assistance in any of your activities of daily living? **Yes** or

No.

- If yes, please explain _____

For subjects who are screened out:

Should any subjects not meet the selection criteria or be excluded based on identified exclusion criteria, indicated by their answers to the above questions, they will be informed of this over the phone in the following manner. (This will vary slightly, depending on

which selection criteria they do not meet.) Examples of how this would be communicated are provided below:

- "You indicated that the primary language you speak is _____ (e.g. Spanish). However, **in this particular study** the activity involves reading and remembering words, as well as naming pictures in English, as quickly as possible. So, I appreciate your willingness and I want to thank you for your interest, for this study we need participants whom use English as their primary language.
- "This particular study needs control subjects who have negative medical histories for many types of illnesses, including encephalitis. However, you indicated that you had encephalitis when you were 11 years old. So, I appreciate your willingness and I want to thank you for your interest."

For subjects who pass the initial screening and express interest in participating:

"When would you be able to participate in the study? We should schedule two different visits and allow approximately 3 hours per visit".

(Date/time/place) _____

(Date/time/place) _____

"Do you have any questions you would like to ask? If you should have any questions before we meet, feel free to call me (the investigator) at (612) 626-9756.

Name:

Address:

Home Phone:

Cell Phone:

e-mail:

Closing Remarks

"At your first visit we will again discuss the study with you, and then we will ask you to sign your consent to participate in the study. Even after consenting to participate, you are free to withdraw from the study at any time without affecting your relationship with the University of Minnesota."

"Do you have any questions you would like to ask? [Pause for response. Answer questions as needed.] If you should have any questions before we meet, feel free to call me (the investigator) at (612) 626-9756. This is the Dept. of Speech-Language-Hearing Sciences at the University of Minnesota, where I work. You may have to leave a message on Voice Mail, and I will return your call as soon as I get it. Also, I will send you a letter confirming the date, time and location for our appointment. You should get the letter within a few days. We want to thank you for your time, and I look forward to our meeting. Good Bye."

APPENDIX B

Telephone Interview form for ABI participants

Subject Code# _____

Phone contact date: _____

Study of Learning and Memory**Initial Telephone Contact Protocol - TBI SURVIVOR
Subjects**

After the investigator(s) has (have) received a postcard, or telephone call from a prospective participant indicating interest and willingness to discuss possible participation in this study, the following is the initial telephone contact protocol that will be used during this first contact.

Greetings and Introductions

*"Hi, I'm Pradeep Ramanathan, from the Department of Speech-Language-Hearing Science, at the University of Minnesota. I received a postcard/phone message recently, indicating that you would be willing to discuss your possible participation in our study on learning and memory. I would like to discuss that with you now. Are you still interested in hearing more about this study?
Is this a good time to talk? Or should I call you back at a later time?"*

Brief Description of the Study

Assuming they indicate willingness to continue, a description of the study would follow.

"I'd like to tell you a bit more about this study. This study is designed to determine how people who have survived a traumatic brain injury are able to remember new information after their injury. You would be participating in this study as an TBI survivor subject. TBI survivor subjects are those individuals who have had a brain injury.

"We are interested in studying learning and memory after a brain injury, because there are still many aspects of this that remain a mystery to therapists and researchers. In this study, you will be first asked to take some tests in which you do some listening, speaking, naming pictures, and so on, just to make sure that you will be able to do the rest of the activities in the study. If you are able to do all these things, you will participate in a second session. At that time you will be asked to name pictures presented by a computer.

You will also be asked to read words presented on a computer screen. After reading the words, you will be asked to try to recall some of the words. If you should have difficulty with the first part of the study, that is with the listening, speaking, naming, etc., then you will likely not participate in the rest of the study.

You will receive approximately \$8.00 per hour for your participation. The entire study will take two sessions of approximately 3 hours each, for a total of about 6 hours. You will receive \$50.00 if you complete the whole study.

“If you are still interested, I'd like to spend about 5 minutes asking you some questions. You are free to decline to answer any of the questions that you do not wish to answer. Some questions have to do with your prior experiences in school, but others are more personal, like 'When you were in school, did you ever have difficulty learning how to read or write?' and 'Have you ever experienced an extended period of alcohol abuse?' and so on. Shall I proceed?”

If the individual indicates the investigator may continue, the following questions will be asked:

TBI - Phone Screening Questions:

15. Are you a high school graduate? **Yes** or **No**. Last grade completed?

16. How old are you? _____ What is your birthday? _____

17. Is English your first language? _____

- If NO - when did you learn English? _____

NOTE: if “after age 5”, candidate cannot participate; politely end telephone screening

18. When you were in school, did you have any difficulty learning to read or write? **Yes**
or **No**

- If yes, please
explain: _____

19. Did you ever receive speech therapy, or remedial help for anything in school? **Yes**
or **No**

- If yes, please explain: _____

20. Were you ever told that you had a learning disability?

21. Did you ever participate in classes for gifted students, or did you ever skip a grade?

Yes or **No**

22. To the best of your knowledge, are your hearing AND vision adequate? **Yes** or

No

- If no, please explain:

23. When did you have your brain injury ? _____ OPEN/CLOSED
head injury?

- Did you have any college classes after your brain injury; describe:

24. Did you receive any therapy after your injury? **Yes** or **No**

If yes, what type? _____

NOTE: if it has been < 6 months, subject must be excluded. Politely end phone screen.

25. Have you ever experienced any of the following?

- | | |
|---|-------------------------|
| <input type="radio"/> Stroke | Yes or No |
| <input type="radio"/> Neurological Disease | Yes or No |
| <input type="radio"/> Extended alcohol abuse | Yes or No |
| <input type="radio"/> Drug abuse | Yes or No |
| <input type="radio"/> Hospitalized for psychological difficulty | Yes or No |
| <input type="radio"/> Periods of unconsciousness | Yes or No |
| <input type="radio"/> Previous head or brain injury | Yes or No |

- If yes to any of these, please explain: _____

26. Do you have any problems walking or moving, that would keep you from coming to the University to participate in this study? **Yes** or **No**

- If yes, please explain: _____

27. Do you require any assistance in any of your activities of daily living? **Yes** or **No**.

- If yes, please explain _____

28. What type of work do you do? _____

29. Do you sign legal documents yourself, or do you have a co-signer? If you have a co-signer, who is that person? _____

For subjects who are screened out:

Should any subjects not meet the selection criteria or be excluded based on identified exclusion criteria, indicated by their answers to the above questions, they will be informed of this over the phone in the following manner. (This will vary slightly, depending on which selection criteria they do not meet.) Examples of how this would be communicated are provided below:

- "You indicated that the primary language you speak is _____ (e.g. Spanish). However, **in this particular study** the activity involves reading and remembering words, as well as naming pictures in English, as quickly as possible. So, I appreciate your willingness and I want to thank you for your interest, for this study we need participants whom use English as their primary language.
- "This particular study needs TBI survivor subjects who have negative medical histories for many types of illnesses, including encephalitis. However, you indicated that you had encephalitis when you were 11 years old. So, I appreciate your willingness and I want to thank you for your interest."

For subjects who pass the initial screening and express interest in participating:

"When would you be able to participate in the study? We should schedule two different visits and allow approximately 3 hours per visit".

(Date/time/place) _____

(Date/time/place) _____

"Do you have any questions you would like to ask? If you should have any questions before we meet, feel free to call me (the investigator) at (612) 626-9756.

Name:

Address:

Phone

Home:

Cell:

e-mail:

Closing Remarks

"At your first visit we will again discuss the study with you, and then we will ask you to sign your consent to participate in the study. Even after consenting to participate, you are free to withdraw from the study at any time without affecting your relationship with the Univ. of Minn." "Do you have any questions you would like to ask? [Pause for response. Answer questions as needed.] If you should have any questions before we meet, feel free to call me (the investigator) at (612) 626-9756. This is the Dept. of Speech-Language-Hearing Sciences at the University of Minnesota, where I work. You may have to leave a message on Voice Mail, and I will return your call as soon as I get it. Also, I will send you a letter confirming the date, time and location for our appointment. You should get the letter within a few days. We want to thank you for your time, and I look forward to our meeting. Good Bye."

Appendix C

Questions to evaluate participant comprehension of informed consent forms

1. About how long will each session take? [ANSWER: About 3 hours]
2. Describe three different tasks you will be doing in these sessions. [ANSWER: Reading words, remembering words, answering questions about words, following instructions, viewing and naming objects, etc.]
3. How long will the data from this study be kept in the locked cabinet? [ANSWER: Up to 10 years]
4. Does anyone other than the investigators, have access to your test results, personal information, or data? [ANSWER: No, only the investigators have access to my data, unless I have signed the agreement that they can release this information to professionals I have identified.]
5. Strictly in terms of the statements in the informed consent form, what are the benefits to you personally, from participating in this study? [ANSWER: There are no personal benefits to me.]
6. What are the risks to you personally from participating in this study? [ANSWER: I may become bored, tired, or frustrated. Otherwise, there are no risks.]

Appendix D

Summary of implicit metamemory task sequence

1. Fixation point is presented at center of computer screen (500ms).
2. Row of ampersands is presented at center of computer screen (500ms).
3. Subliminal token is presented in lower case at center of computer screen (50ms).
4. Cue-Target word-pair is presented in capital letters at center of computer screen (5s for control participants, 9s for participants with ABI), as below:

LIME – COLLAR

5. For word-pairs assigned to the immediate JOL timing condition, participants are immediately asked to make a JOL as below (self-paced):

LIME – ?

Please Make Your Rating Now

0% 20% 40% 60% 80% 100%

6. Steps 1 – 5 are repeated for the remainder of the first 21 word-pairs.
7. Delayed JOLs are made on word-pairs for which JOLs were not already made (self-paced).
8. Steps 1 – 7 are then repeated for the second set of 21 word-pairs in the trial block.

9. If steps 1 – 8 are completed in fewer than 10 minutes, two additional minutes were spent in conversation. Otherwise, participants proceeded to step 10 below.
10. The first word of a word-pair, randomly selected from the first 21 word-pairs studied, is centrally presented on the computer screen, for the recall test as below (self-paced):

LIME – ?

Please Speak Your Answer Now

11. Step 10 is repeated for the second set of 21 word-pairs of the trial block.
12. The participant is given a mandatory break (15 minutes).
13. Steps 1 – 12 are repeated for the second trial block.
14. Steps 1 – 11 are repeated for the third trial block.
15. Immediately on completion of the third trial block, instructions for the validation task appear (self paced).
16. A single word (either the primed target, antiprime stimulus, or not previously used word) is presented in capital letters at center of computer screen, with the query as below (self-paced):

COLLAR

Please Select

Old

New

17. Step 16 is repeated for all 42 words of the validation task.

18. The participant is then given a 15 minute break before the Priming/Antipriming experiment.

APPENDIX E

Implicit Metamemory task

Data reduction.

A PERL script was written to reduce the data output from the E-Prime program into a comma-delimited format that could be imported into Microsoft Excel and/or SPSS for further analysis. The PERL script was designed to eliminate the four primacy items from each trial block, as well as all other irrelevant information (e.g.: computer time seed values, etc.) from the E-Prime data file, and sort the remaining data to facilitate further analysis. It should be noted that recency effects were dealt with by administering the recall test for the first set of 21 word-pairs of a trial block after study of the second set of 21 word-pairs of that trial block, and administering the recall test for the second set of 21 word-pairs of the trial block after recall was tested on the first 21 word-pairs studied for that block. Thus, several minutes always elapsed prior to recall testing of any particular item. Therefore, it was deemed unnecessary to eliminate recent items during data reduction. Eight of the 31 resulting participant data files (25.8%) were then randomly selected and reviewed, line by line, to ensure that the PERL script did not introduce any errors. No discrepancies were found in any of the files, resulting in 100% reliability of the PERL script.

The comma delimited files were then imported into an MS Excel template which computed accuracy of recall by comparing the spoken response against the actual target response. The spreadsheet also averaged the seven values (of each dependent measure)

within each list, to produce average values for each cell of the combination of priming and JOL timing conditions for each block, for each participant. It should be noted that singular/plural (e.g.: “ribs” instead of “rib”) or expansion/contraction (e.g.: “telephone” instead of “phone”) were the only response variations that were accepted; even closely related words (e.g.: “dog” instead of “puppy”) were coded as errors. Self-corrected responses were accepted, however response time measures for such trials were flagged for later removal from analysis of response time data. All 31 participant files were then reviewed item-by-item to correct any errors in the MS Excel accuracy calculation that were due to its rejection of non-identical response variations. Over the 126 responses per participant, and 31 participants (i.e.: out of 3,906 total responses), only four items had to be corrected for singular/plural or expansion/contraction errors introduced by the MS Excel formula.

Given that there were only seven measurements per cell, gamma correlation calculation resulted in a large number of empty cells, making analysis of predictive accuracy impossible. Without collapsing the data across trial blocks, there would only be 7 cases (or measurements) per cell, prior to elimination of primacy items. Therefore, it was desirable to collapse the data across the three trial blocks, thus providing 21 measurements per cell; it should be noted that such collapsing of data across trial blocks is typical in studies following the Nelson and Dunlosky (1991) paradigm. In order to ensure that collapsing across blocks did not mask any block effects, repeated measures MANOVA was conducted to determine whether either JOL rating or recall accuracy demonstrated a trend or pattern across blocks for each of the participant groups. Results

indicated that there was no effect of the block variable on either dependent measure [$F(4,26) = 1.176, p = .344, \text{partial eta squared} = .153$].

Two participants (both from the TBI survivor group) were then removed as outliers from the analysis. Several criteria were used to determine whether a participant should be designated as an outlier. The first criterion was that no participant could have more than a total of three missing or out of range cells across the 18 cells that represent the three primary dependent measures (average JOL rating, average accuracy, and average gamma correlation) by six combinations of the levels of the independent variables of priming and JOL timing. Out of range was defined as a difference of more than two standard deviations of the mean value for that condition combination for that participant group. Thus, for example, if the recall accuracy for a particular participant (e.g.: among controls) was 84.5% for the antiprime/immediate condition combination, and the mean for that combination of conditions for the entire control group was 30.2% ($SD 22.9\%$), then the data from that cell would be judged as non-representative of the data set.

The second criterion was that no more than one cell of data, out of the two cells of data that represent a particular priming condition for a particular dependent variable, could contain a missing or out of range value. This criterion ensured that every participant had at least one cell of acceptable data, out of the two cells of data, representing each priming condition for each dependent measure.

Finally, the third criterion was that no more than two cells, out of the three cells of data that represent a particular JOL timing condition for a particular dependent variable, could contain a missing or out of range value. Thus, for example, if a

participant had out of range or missing data for all three cells representing the immediate JOL timing condition for the JOL rating, then that participant was designated an outlier. This criterion ensured that every participant had at least one cell of acceptable data, out of the three cells of data, representing each JOL timing condition for each dependent measure.

Based on these criteria, two TBI survivors were removed from the analysis of this task. The first of these two participants had 0% accuracy for all 126 trials, and therefore all six cells of the gamma correlation were missing data. That participant was also the only ABI survivor whose acquired brain injury was not the result of closed head trauma; it was the result of a brain tumor. Therefore, it was deemed appropriate to remove those data from analysis. The second TBI survivor consistently demonstrated extremely high recall accuracy (M 79.9%) as compared to the average for all TBI survivors (M 31.3%, SD 21.9%). As a result, seven out of 18 cells for that participant contained either missing or out of range data. Therefore, it was deemed appropriate to remove those data from analysis. On questioning after completion of the task, that participant admitted having learned, during outpatient rehabilitation, special techniques for achieving very high levels of recall of word lists through elaborative visual association.

For the analysis of response time data, an additional participant was removed. That control participant had extremely long response times for making judgments of learning, with five out of six of the JOL response times being greater than two standard deviations above the group means. Since this participant's JOL, accuracy, and gamma correlation data were well within range, that person was not excluded from the analysis

of those measures. This was deemed reasonable, since much previous research has not included response time measures, and therefore the remaining dependent measures of this study can be compared against previous results (Kennedy & Yorkston, 2000; Kennedy, 2004; Kennedy, Carney & Peters, 2003; Schmitter-Edgecombe & Woo, 2004).

APPENDIX F

Visual Antipriming task

Data reduction.

Since the stimuli in this task consisted of visual objects, it was possible that the spoken response might be correct, but not an exact match to the target object name. Since the stimuli in this task were visual objects rather than written words, the criteria for acceptable spoken responses were more relaxed for this task than for the implicit metamemory task. In addition to accepting singular/plural and expansion/contraction differences, close synonyms were also accepted (e.g.: “hatchet” for “axe”, “barbell” for “weight”, etc.). Therefore, all 31 participant files were visually inspected line-by-line, comparing the spoken responses to the target responses. Items which the computer automatically scored as inaccurate, but which were judged to be accurate were flagged as discrepant and were scored according to the above rules. A complete list of all such discrepancies across all participants was then submitted to Rebecca Deason (a co-author of the Marsolek et al., 2006, study) for reliability. Five hundred and forty-five out of the 587 discrepancies (92.84%) had been scored identically as acceptable/unacceptable by both raters. The inconsistently scored items were then corrected to match Ms. Deason’s scoring, in order to remain consistent with the methodology of Marsolek et al. This resulted in a final reliability of 100%.

In addition to item-by-item accuracy, the MS Excel template for each participant also automatically computed the means and standard deviations for accuracy and

response time by priming condition (baseline, prime, antiprime). Response times were computed only for correct responses. The template for each participant further filtered out response time outliers by excluding any response time that was more than two standard deviations from the mean of all items in the same priming condition. The resulting final values for mean accuracy and response time, by priming condition, for each participant were transferred to an SPSS data file for analysis.

Three participants (two from the TBI survivor group and one from the control group) were then removed as outliers from the analysis, on the basis that their mean accuracies and/or response times, averaged across all conditions, were more than two standard deviations from the respective group mean. Removal of outliers resulted in a reduction of the magnitudes of skewness and kurtosis. Prior to removal of outliers, the strongest skewness value among the three priming conditions was -1.424 , which became -1.092 after outlier removal. Maximum kurtosis of accuracy prior to removal was 2.743 , and this dropped to 1.969 after outlier removal. Similarly, for response times among the three priming conditions, prior to outlier removal, skewness had a maximum value of 2.832 and after removal this became 2.000 . Maximum kurtosis of response times prior to outlier removal was 8.687 , and this became 4.387 after outlier removal.

APPENDIX G

Visual Antipriming Instruction – part 1

You will be presented with a list of words, one at a time. Recordings of the names of objects will be played on the computer's speaker.

After a stimulus is presented, please decide how much you like that thing. Please use a 1 to 4 scale. If you like it very much, choose 4; if you dislike it very much, choose 1; if your feelings about it are anywhere between these extremes choose an intermediate number. When making your decision, please consider the meanings associated with the name of an object, as opposed to how they sound, or what they look like, etc. It is very important that you judge your true feelings about the stimulus; it is important that you choose a number that truly shows how much you like or dislike it. Also, please distribute your judgments across the whole 1-4 scale over the course of the many judgments you will be making.

Your instructions for each trial are as follows: A dot will appear in the center of the screen for one half of a second to signal that a stimulus will be presented next. Please focus your eyes on the dot. A recorded name of an object will be played and your task is to decide how much you like or dislike the stimulus. Please press the number key on the keyboard corresponding to your rating.

Before you begin, let's recap the procedure:

Before a trial, please look at the computer screen. Please keep your eyes on the fixation point when it appears. After the recorded name has been played, please report how much you like or dislike the stimulus.

If you have any questions at all, please ask the researcher now.

Visual Antipriming Instructions – parts 2 & 4

Next, you will participate in a study of how people identify familiar objects. We are investigating how quickly people can name common objects that appear on the computer screen.

Your instructions for each trial are as follows: A dot will appear in the center of the screen for one half of a second. Please focus your eyes on the dot the entire time it is on the screen--please don't let your eyes drift to one side or the other in anticipation of the coming object. As soon as the dot disappears, a line drawing of an object will appear in a position slightly above or slightly below the center of the screen. Your task is to speak aloud into the microphone the name of the object that is shown on the computer screen as quickly and accurately as you can. Please do not say, “um”, “uh”, or “I don't know”. If you are not sure what object was shown, please guess. The clock measuring your response time starts when the object appears on the screen, therefore you should try to respond just as soon as you realize the identity of the object. You need to speak somewhat loudly to stop the clock, so please hold the microphone close to your mouth and don't be afraid to speak up. The experiment will then proceed to the next trial. Each object will appear very briefly--these exposures may seem too brief to see anything, but we've found that with practice people can recognize objects that go by that quickly.

The researcher will first show you several practice trials to get you accustomed to the short viewing times used in this part of the study and to get you accustomed to viewing objects that are presented slightly above or below the center of the screen. This will also give you an idea of how loudly you must speak to trigger the microphone during the experiment.

Before we begin the practice trials, let's recap the procedure:

Before a trial, please look at the computer screen. Please keep your eyes on the fixation point the entire time it appears on the screen. When the object is flashed, please name it *as quickly and accurately as you can* (using a somewhat loud voice).

If you have any questions at all, please ask the researcher now.

Visual Antipriming Instruction – part 3

You will be presented with pictures, one at a time. Each object will be displayed visually on the computer screen.

After a stimulus is presented, the screen will go blank. Please decide how much you like that thing you just saw. Please use a 1 to 4 scale. If you like it very much, choose 4; if you dislike it very much, choose 1; if your feelings about it are anywhere between these extremes choose an intermediate number. When making your decision, please consider the meanings associated with the object, as opposed to how they sound, or what they look like, etc. It is very important that you judge your true feelings about the stimulus; it is important that you choose a number that truly shows how much you like or dislike it. Also, please distribute your judgments across the whole 1-4 scale over the course of the many judgments you will be making.

Your instructions for each trial are as follows: A dot will appear in the center of the screen for one half of a second to signal that a stimulus will be presented next. Please focus your eyes on the dot. An object will be shown for three seconds. Your task is to decide how much you like or dislike the stimulus. When the screen goes blank please press the number key on the keyboard corresponding to your rating.

Before you begin, let's recap the procedure:

Before a trial, please look at the computer screen. Please keep your eyes on the fixation point when it appears. After the object has been shown, and the screen goes blank, please make a 1 – 4 rating of how much you like or dislike the stimulus.

If you have any questions at all, please ask the researcher now.