August 2018

Neural Correlates of Memory Decisions Made in the Face of Conflict

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by

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A Dissertation Submitted in

Partial Fulfillment of the

Requirements for the Degree of

Doctor of Philosophy

in Psychology

at

The University of Wisconsin – Milwaukee

August 2018

ABSTRACT

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The University of Wisconsin – Milwaukee, 2018
Under the Supervision of Professor Deborah Hannula

We've all experienced moments where, for some reason or another, we don't want to reveal to others what we truly know. The current experiment investigated questions about the behavioral and neural correlates of these types of memory decisions made in the face of a conflicting goal. Participants in this experiment studied several scene-face pairs and were tested with three-face displays preceded by studied scene cues. They were instructed to indicate whether the three-face display contained the matching associate or not. Critically, half of the participants were instructed to simulate feigned memory impairment (i.e. simulators), while the remainder were instructed to perform optimally (i.e. controls). Eye movements and neural activity were recorded throughout this test. Consistent with the instructional manipulation, simulators performed worse than controls with their explicit responding. However, both groups showed comparable early viewing of the associate after the three-face display was presented. Analyses were conducted to identify the memory, attention, and cognitive control processes that contributed to these memory decisions while feigning memory impairment. Hippocampal activity during the scene cue predicted early viewing effects for simulators, even when they made incorrect responses. During the three-face display, hippocampal activity reflected memory accuracy among controls, but the opposite pattern was evident in simulator data. This pattern, with greater activity for incorrect than correct trials, was also seen for simulators in the anterior

cingulate cortex, an outcome that likely reflects conflict between memory retrieval and decision making. Finally, group differences in parietal regions likely indicate greater reorienting of attention among simulators than controls. Together, these results suggest the recruitment of memory retrieval, attentional allocation, and cognitive control regions as individuals work to succeed at simulating memory impairment.

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ACKNOWLEDGEMENTS

First and foremost, I must thank my advisor, Dr. Debbie Hannula, for all of her guidance and mentorship throughout graduate school. With her assistance I have grown leaps and bounds as a scientist over the past five years. I would also like to thank my committee members, Drs Chris Larson, Ira Driscoll, Fred Helmstetter, and Adam Greenberg, for their unique insights into and perspectives on the current work. Furthermore, this work wouldn't have been possible without my lab members, especially Allison Nickel, for the hours spent talking through ideas and analyses, as well as venting about the inevitable roadblocks. Along those lines, I am grateful for everyone who logged hours with me in the imaging suite, offering advice, answers to questions, and most of all, support. Everlasting thanks to the scan techs at the Medical College, especially Tina, who struggled with me through the Herculean task of getting two technological systems to both work at the same time. And last but certainly not least, I must thank the two women, Tara and Laura, who have gone through this journey with me and kept me (mostly) sane along the way. This work was supported by a National Science Foundation CAREER award 1349664 to DEH. Any opinions, findings, and conclusions or recommendations expressed in this material are those of the authors and do not necessarily reflect the views of the National Science Foundation.

Much of the time, we desire accurate memory retrieval in order to inform behavior. On the other hand, we have all likely experienced situations in which, despite accurate memory, we aim to hide our knowledge. This deception is usually fairly innocuous, done to spare another's feelings or maintain one's own public image. However, there are times at which deception about what one remembers can be very costly to society. For example, individuals may feign memory impairment for financial gain or to gain access to resources, and in doing so make it more difficult for individuals who truly require help. The primary objective of this investigation was to examine the cognitive and neural correlates of memory-based decision making in the face of conflict. To this end, half of the participants in this between-groups study were instructed to simulate or feign memory impairment on a recognition test, while others were instructed to perform their best. I posit that memory, attention, and cognitive control all contribute to performance, likely interacting in order to guide behavior. Specifically, using a combined eye tracking and functional magnetic resonance imaging (fMRI) approach, I investigated whether information retrieved from long-term memory is subject to attentional prioritization despite participants' attempt to feign impairment, whether and how attention is redirected away from known materials, and how cognitive control processes may be recruited to convincingly feign impairment.

In the field of memory research, the structures of the medial temporal lobe (MTL) have been demonstrated to be critical for the encoding and retrieval of episodic memories (Graf & Schacter, 1985; Squire & Zola, 1997). According to one proposal, the hippocampus itself is critically involved in the processing and representation of relationships among items, including the relationships between items and their contexts (Eichenbaum, Otto, & Cohen, 1992; Eichenbaum, Yonelinas, & Ranganath, 2007). The neuroanatomical structure of the MTL is the

basis for this proposal. The MTL consists of the hippocampal complex, as well as the perirhinal, entorhinal, and parahippocampal cortices (Squire, Stark, & Clark, 2004). The MTL receives highly-processed multimodal input, including visual information conveyed by two visual processing streams: a ventral visual stream that represents object information (i.e. the "what" information) and a dorsal visual stream that represents information about the location and orientation of the object (i.e. the "where" information; Goodale & Milner, 1992). Where these processing streams project provides information about what types of representations the various regions of the MTL may support. The ventral visual stream projects primarily to the perirhinal cortex (Suzuki & Amaral, 1994) and so it has been proposed that this region is capable of supporting object-level representations for memory and other cognitive processes (e.g., perception; Murray & Richmond, 2001; Buckley & Gaffan, 2006). In contrast, the dorsal visual stream projects to the parahippocampal cortex (Suzuki & Amaral, 1994), which is proposed to support representations of contexts (Eichenbaum & Lipton, 2008). Following these initial projections into the MTL, the perirhinal and parahippocampal cortices project to the lateral and medial entorhinal cortex, respectively (Insausti, Amaral, & Cowan, 1987). Finally, these two processing streams converge in the hippocampus. This positioning at the apex of the hierarchy within the MTL supports the proposal that the hippocampus is well suited to bind item and context information. These relational representations are then able to support the rich, episodic memories that are formed as we interact with our environment (Cohen & Eichenbaum, 1993; Eichenbaum, Yonelinas, & Ranganath, 2007; Eichenbaum & Cohen, 2014; Montaldi & Mayes, 2010; Diana, Yonelinas, & Ranganath, 2007; Davachi, 2006).

This proposal that the hippocampus supports relational binding has been supported by a number of neuroimaging investigations with healthy individuals (e.g. Sullivan, Giovanello,

Schnyer, & Verfaellie, 2004; Prince, Daselaar, & Cabeza, 2005; Hannula & Ranganath, 2008; Hannula, Libby, Yonelinas, & Ranganath, 2013; see also Cohen, Ryan, Hunt, Romine, Wszalek, & Nash, 1999). In these fMRI investigations, activity differences in the hippocampus were greater for tasks that required the retrieval of relational, as compared to item, memories (e.g., Sullivan et al, 2004). It has also been demonstrated that hippocampal activity is greater when a person is successful in their relational memory retrieval, compared to when retrieval fails (e.g., Prince et al., 2005; Hannula et al., 2013). Additional investigations have been conducted with patients with MTL damage (e.g. Crane & Milner, 2005; Holdstock, Mayes, Gong, Roberts, & Kapur, 2005; Hannula, Tranel, & Cohen, 2006; Hannula, Ryan, Tranel, & Cohen, 2007; Konkel, Warren, Duff, Tranel, & Cohen, 2008). One such investigation tested healthy participants, as well as amnesic patients whose lesions were either restricted to the hippocampus or extended beyond the hippocampus to adjacent MTL regions (Konkel et al., 2008). In the experiment, they tested three different types of relational memory (spatial, associative, and sequential) along with item memory. Participants in this experiment first studied a number of sets of 3 novel visual objects presented sequentially, each in a unique location on the screen. Following the encoding block, participants' memory was tested for either their knowledge of the individual items presented in the encoding block (i.e. item memory), of the locations in which items in a set were presented (i.e. spatial relational memory), of the groupings of objects in the sets within the block (i.e. associative relational memory), or of the order in which items in a set were presented (i.e. sequential relational memory). They found that all of the patients were impaired on the three relational memory tasks as compared to healthy controls. However, the patients with lesions limited to the hippocampus showed relatively spared item memory as compared to their relational memory performance. From this, they concluded that all three types of relational

memory rely disproportionately on the hippocampus, in comparison to item memory which can be supported, in part, by other MTL structures. These findings therefore support the relational memory theory described above.

Consistent with the basic tenets of relational memory theory, Moscovitch (2008) proposed that the hippocampus supports rapid, obligatory retrieval of associated details in the presence of memory cues as the first step of a two-stage process leading to conscious recollection. According to this two-stage model, when a memory cue is present, an involuntary, unconscious retrieval of the associated details occurs and this is hippocampus-dependent. This first stage is consistent with research with both human and non-human animals that indicates that the hippocampus is involved in rapid pattern completion processes (Marr, 1971; Mizumori, McNaughton, Barnes, & Fox, 1989; Bakker, Kirwan, Miller, & Stark, 2008; see Rolls, 2013 for review). In the model, following this obligatory memory retrieval, a slower, more effortful stage of retrieval occurs with conscious awareness, which allows for the explicit recall of the memory. This second stage also requires the hippocampus, but additionally relies on its connections with prefrontal and parietal regions.

Consistent with the proposed two-stage model, it has been reported that hippocampal activity during a memory cue predicts eye-movement-based expression of relational memory and that this effect is present even when explicit recollection fails (Hannula & Ranganath, 2009). In this investigation, a scene-face paradigm was used, one that was adapted and used in the current investigation. In this task, participants first learned a series of scene-face pairs. Following this encoding phase, participants were cued with one of the studied scenes after which three studied faces were superimposed on top of the scene. While the behavioral response required by the participant to this display differs across investigations, analysis of viewing patterns has reliably

shown that individuals tend to view the associate of the scene disproportionately, an effect that is evident early in the trial (i.e. within 500-750ms of display onset), despite the fact that all three faces were presented equally often during the encoding phase (See Figure 1A; Hannula, Ryan, Tranel, & Cohen, 2007; Hannula & Ranganath, 2009; Williams et al., 2010). This early viewing effect therefore, reflects memory for the learned scene-face relationships (see Hannula, Althoff, Warren, Riggs, Cohen, & Ryan, 2010 for review). As mentioned previously, a neuroimaging investigation demonstrated that hippocampal activity during the scene cue predicted these early eye-movement-based relational memory effects (Hannula & Ranganath, 2009). The observation that these hippocampal effects predicted viewing even when participants misidentified the associate is consistent with the first stage of the two-stage model where obligatory hippocampal retrieval in the presence of memory cues is not sufficient for conscious recollection. In the

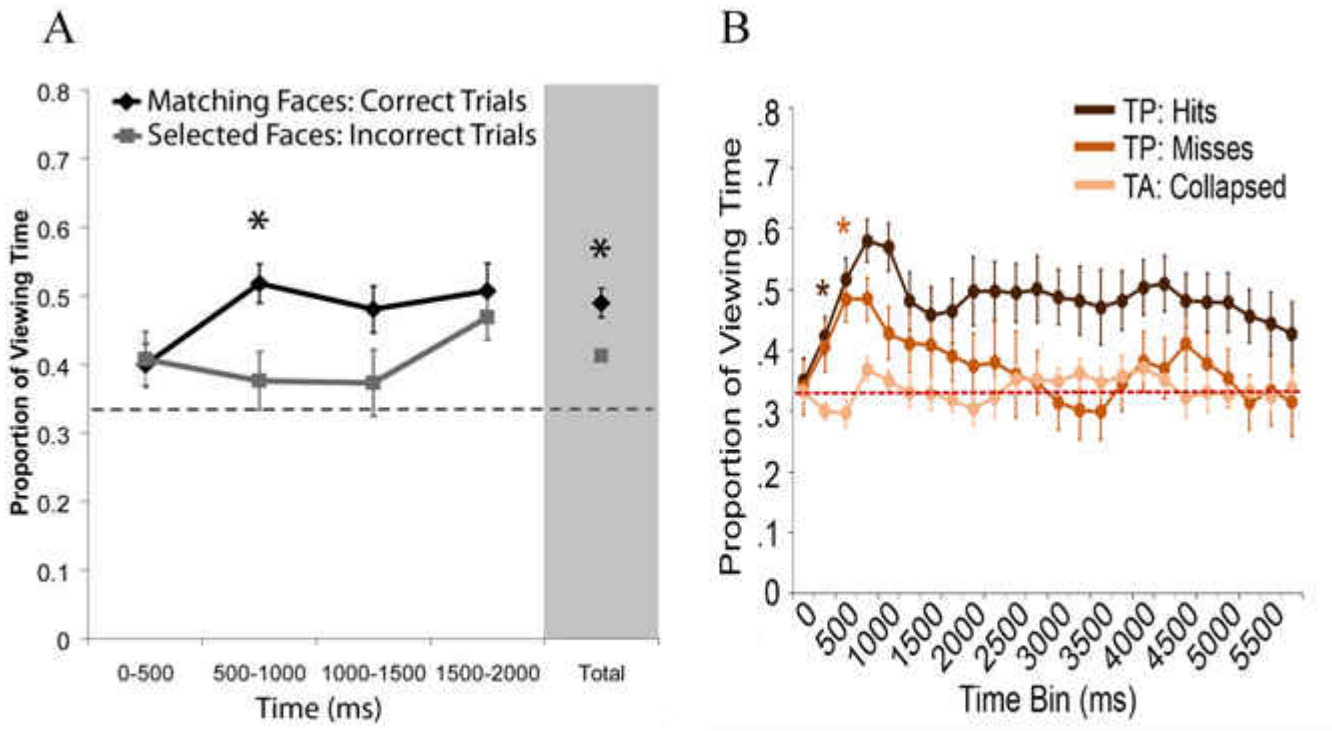

Figure 1. Early eye-movement based memory effects. A) From Hannula et al. (2009): Early disproportionate viewing is seen between 500-1000ms for individuals performing optimally. B) From Mahoney et al. (in prep): Viewing patterns of participants feigning memory impairments. Early disproportionate viewing of associates occurred for both correct and incorrect trials.

current investigation, I examined whether or not hippocampal activity would predict viewing on incorrect trials when participants feign memory impairment. Given the proposal that hippocampal retrieval is an obligatory process in response to a memory cue, effects similar to those reported previously (Hannula & Ranganath, 2009) were expected in simulator data.

Additional behavioral investigations using the scene-face task above have lent support to the idea that retrieval of associates occurs obligatorily as predicted by the two-stage model. Specifically, it has been shown that these eye-movement-based relational memory effects occur extremely quickly after stimulus presentation (i.e. within 500-750ms following stimulus onset), are present before explicit responses are made, and are resistant to change in the face of a variety of task instructions (Hannula et al., 2007). Indeed, results from a recent study indicate that these eye movement effects are present even when individuals attempt to feign memory impairment and whether, in this context, explicit recognition responses are correct or not (Mahoney, Osmon, Kapur, & Hannula, in prep). Taken together, these characteristics suggest that the eye-movement-based relational memory effect may be an obligatory expression of hippocampus-supported retrieval that occurs when a memory cue is presented. As such, we might expect to see comparable effects here in viewing patterns of simulators who are attempting to feign memory impairment, and control participants who are performing optimally. These eye-movement-based memory effects are expected to be seen even on trials where simulators successfully conceal their memory by making incorrect explicit responses.

As the above work investigating relational memory employed eye movement methodology, it can also be used to make inferences about attentional deployment of the participants. While attention can be deployed covertly, without a corresponding eye movement, where the eyes are directed usually provides some information about the focus of attention (e.g.,

Hoffman & Subramaniam, 1995). Based on the characteristics of the eye-movement-based memory effects described above, these studies suggest that these effects may represent an automatic or obligatory prioritization of attention towards content retrieved from long-term memory that is related to retrieval by the hippocampus in response to the memory cue. A primary aim of the current investigation was to investigate the neural bases of this potential interaction between memory retrieval and attentional prioritization of learned materials, indexed with eye movement behavior, while participants complete the scene-face task described above under the instruction to feign memory impairment for the associations.

As described above, one aim of the current investigation was to use early eye movement effects to determine whether attention is directed to learned information retrieved from memory, despite instructions to simulate memory impairment. In addition, this experiment addressed whether and how eye movements throughout the trial may change when individuals are trying to feign memory impairment as compared to performing optimally. More specifically, it was investigated whether attention was redirected from these learned materials, as seen in eye movement behavior, and how this reorientation of attention is supported in the brain.

Two ways in which attention can be deployed have been differentiated: bottom-up attention, which is generally thought of as a capture of attention by a stimulus, and top-down attention, which is attention deployed in accordance with some goal (Todd & Van Gelder, 1979; Posner, 1980; Serences & Yantis, 2007). Through neuroimaging investigations, bottom-up deployment of attention has been linked to a ventral pathway in the parietal and frontal lobes, including the temporoparietal junction (TPJ) and the ventral frontal cortex (Corbetta & Shulman, 2002; Vossel, Geng, & Fink, 2014). In contrast, top-down allocation of attention has been found to be supported by a dorsal frontoparietal network including regions such as the intraparietal

sulcus (IPS), the superior frontal cortex, and the frontal eye fields (Corbetta, Kincade, Ollinger, McAvoy, & Shulman, 2000; Corbetta & Shulman, 2002; Shulman, McAvoy, Cowan, Astafiev, Tansy, d'Avossa, & Corbetta, 2003).

There are several attention-related regions that are of interest for the current investigation. One is the TPJ which has been shown to be activated when target stimuli appeared in an uncued, unattended location, leading researchers to propose that this neural region is responsible for stimulus-driven reorienting of attention (Corbetta et al., 2000; Corbetta & Shulman, 2002). Activation of TPJ was also seen when distractors were present during a task, an effect that was disproportionately greater when the distractor shared a feature with the target stimulus (Serences, Shomstein, Leber, Golay, Egeth, & Yantis, 2005). Again this led researchers to conclude this region is activated when reorientation to a stimulus occurs, particularly when this stimulus has some sort of behavioral relevance. Here, the interest was whether attention that is reoriented *away* from the behaviorally relevant stimulus, in line with task instruction, might also be supported by this region.

In addition to the TPJ, regions involved in top-down deployment of attention are of interest. Uncapher, Boyd-Meredith, Chow, Rissman, and Wagner (2015) investigated the role of such resources when individuals attempted to make deceptive old/new responses to previously encoded faces. Here, when instructed to be deceptive, participants were encouraged to modulate memory responses to avoid detection by a neural classifier by engaging specific strategies when presented with each face. When they were presented with a previously encoded face, they were instructed to respond that it was new and then encouraged to shift their attention to features of the photograph, such as the lighting, that they had not previously attended. In contrast, when presented with novel faces, they were instructed to respond that it was old and then encouraged

to engage in memory-related processing, such as remembering a friend who looked similar. A contrast between activity during old faces, which required a shift of visual attention, and during new faces showed increased activation of the left IPS and superior parietal lobule. They concluded that participants were successful in employing the instructed strategies and that this activation represented goal-directed reorientation of attention to perceptual features of the photograph. In the current investigation, the act of redirecting attention away from learned materials when attempting to feign impairment, as seen in eye movement behavior, may be related to these top-down attentional mechanisms and/or bottom-up reorienting regions, such as the TPJ.

In addition to addressing questions about changes in how attention is directed to learned materials across groups, and the neural correlates of these effects, the current investigation aimed to identify additional cognitive and neural resources that are recruited when individuals attempt to feign memory impairment. Studies using neuropsychological performance validity tests have demonstrated that individuals malingering memory deficits tend to respond more slowly than individuals giving optimal effort (van Hooff, Sargeant, Foster, & Schmand, 2009; Vagnini, Berry, Clark, & Jiang, 2008). It should be noted that in these experiments participants are not given specific strategies, but rather, are instructed to convince the examiner that they have a memory problem. They do not, for example, respond incorrectly on every trial, which differs from standard concealed memory tests where participants are told specifically when to make incorrect responses and when to respond accurately. From the response-time findings in these types of investigation, I posit that in order to successfully simulate feigned memory impairment, additional cognitive control processes are required to update decision making based on past

responses, as well as to overcome conflict between memory retrieval and the instructed task objective.

Cognitive control has been described as a group of processes that coordinate other cognitive resources (including attention and memory) in order to flexibly adapt behavior to progress towards a current goal. (Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004). Some of the processes under the cognitive control label include the selection and ongoing maintenance of goals (Arana, Parkinson, Hinton, Holland, Owen & Roberts, 2003; Valentin, Dickinson, & O'Doherty, 2007), response inhibition (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Booth et al., 2003), working memory (D'Esposito, Detre, Alsop, Shin, Atlas, & Grossman, 1995; D'Esposito, 2007), and performance monitoring (Ridderinkhof et al., 2004; Ullsperger & Von Cramon, 2004). Upregulation of this system is thought to occur when additional control is required to keep behavior in line with current goals (Ridderinkhof, Ullsperger, Crone, & Nieuwnehuis, 2004). Increased engagement of cognitive control, therefore, should occur in situations where it may be more difficult to act effectively, such as when task demands increase or when items that must be ignored are present in the environment. In the current investigation, the engagement of cognitive control was expected to be greater for participants who were given the demanding task of feigning memory impairment in the presence of encoded information.

Research on the neural basis of cognitive control has consistently shown that these processes are largely supported by the prefrontal cortex (PFC; Miller, 2000; Koechlin, Ody, & Kouneiher, 2003; Ridderinkhof et al., 2004; Kouneiher, Charron, & Koechlin, 2009). Processes that are expected to be engaged as participants feign memory impairment in the current investigation include working memory, particularly the active maintenance of the instructed

goal, response inhibition, and performance and error monitoring. In fMRI investigations, working memory, and specifically goal maintenance, have been shown to be supported by the lateral PFC (D'Esposito, 2007; Paxton, Barch, Racine, & Braver, 2008). Both the dorsolateral and ventrolateral PFC have been related to response inhibition (Chikazoe, Konishi, Asari, Jimura, & Miyashita, 2007; Mostofsky et al., 2003). Finally, performance and error monitoring have been found to be related to activity in the medial PFC and the anterior cingulate cortex (ACC; Carter, Braver, Barch, Botvinick, Noll, & Cohen, 1998; Ridderinkhof et al., 2004).

If the cognitive control system is responsible for activating, coordinating, and monitoring other cognitive processes in the brain, the question remains as to what provides this kind of oversight for the cognitive control system. Botvinick, Braver, Barch, Carter, & Cohen (2001) proposed that this role was the job of the ACC. As mentioned above, the ACC has been linked to performance monitoring and error detection (Carter et al., 1998). In their hypothesis, Botvinick et al. (2001) describe the ACC as a conflict-monitoring region. They concluded this on the basis of several consistent findings about the region. The first was that the ACC is activated when a person needs to override a prepotent response, which they interpreted as activation due to the conflict between the prepotent response and the response that needs to be made to achieve the current goal. They also point to research that showed ACC activation during tasks that have multiple, equally correct responses and stated that the conflict between these possible choices is what recruits the ACC. Finally, they discussed research that showed that the ACC is more active when a person commits an error, especially on a speeded task. They construe this finding as the ACC reacting to the conflict between the given response and the correct answer. In addition to considering these research findings, they presented two computer simulations that show that the ACC as a conflict-monitoring unit, along with feedback from other cognitive control regions, can

account for a number of established behavioral findings in the literature. Therefore, they propose that the ACC is a continually active conflict monitoring processor that can alert other prefrontal regions to situations that may require greater cognitive control.

Consistent with this conflict-monitoring hypothesis, an fMRI study showed that during the Stroop task, the ACC showed conflict related activity when participants committed errors (i.e. read the word when the task was to name the ink color; Kerns, Cohen, MacDonald, Cho, Stenger, & Carter, 2004). Furthermore, this increased activity in the ACC was related to an increase in activity in the PFC, specifically the dorsolateral PFC, and to behavioral adjustments on trials following the error. Overall, this study provides support for the notion that the ACC is a region that monitors conflict in information processing, and once such conflict is detected, recruits the other cognitive control regions to bring behavior back in line with the current goal. More recent research has suggested that in addition to performance and conflict monitoring, the ACC is involved in evaluating outcomes of potential actions (Shenhav, Botvinick, & Cohen, 2013; Brown, 2013). With this added function of the region, it is posited that the ACC is able to integrate information about the benefit of additional cognitive control and effectively modulate cognitive control regions for optimal performance (Shenhav et al., 2013).

Few studies have been conducted to examine the neural correlates of malingered memory performance, but the results from these studies are consistent with the proposal that increased cognitive control is required for successful feigned memory impairment (Lee et al., 2002; Lee, Liu, Chan, Ng, Fox, & Gao, 2005; Browndyke et al., 2008). Again, participants in these experiments were instructed to convince the examiner that they had a memory deficit, but not provided with specific strategies on how to do so, giving the participant the freedom to make correct and incorrect responses as they wished. In one such experiment, a block design task was

employed where participants had to complete a digit memory task under different conditions. In this task, participants were first shown a three-digit number, followed by a short delay, and the presentation of another number. Participants were required to indicate whether the number matched the one seen earlier in the trial, either accurately or while trying to feign memory impairment. They found increased activation in the feigned impairment block in the frontopolar prefrontal regions, which was explained by the increased need to keep a primary goal in mind. The dorsolateral PFC also showed greater activation and they stated this could be related to a number of processes including performance anticipation, intentional retrieval, working memory, general cognitive control, and the selection of retrieval strategies. Increased cingulate activity was explained as a need for the inhibition of previously learned rules (i.e. responding honestly) and self-monitoring of errors. A later block design study by the same group showed that the prefrontal regions implicated in the original study, including the frontopolar regions, dorsolateral PFC, and anterior cingulate, are generalizeable across stimulus types, gender of participant, and native language (Lee et al., 2005). In addition, they identified consistent activation of dorsomedial PFC and orbitofrontal cortex related to feigned memory impairment compared to optimal performance. One limitation of these block-design investigations is the inability to evaluate trial-specific activity differences. The current investigation employed an event-related design where activity was examined during two trial components – a memory cue and a recognition test display.

One event-related fMRI study of memory malingering used a modified version of a neuropsychological performance validity test, where participants first learned a number of simple line drawings (Browndyke et al., 2008). They then completed a recognition phase in which items were presented one at a time and old/new judgments were required, again either under

instruction for optimal or feigned memory performance. Researchers investigated both types of malingered responses: malingered misses, which is saying a stimulus was new when it was really old, and malingered false alarms, which is saying a stimulus was old when it was really new. They found increased activity for malingered misses compared to hits made in the optimal performance condition in the left dorsomedial PFC, which they explained as an increase of inhibition and response conflict, and the inferior parietal lobule, which they concluded was due to performance and probability monitoring. They also found increased activity bilaterally in the ventrolateral PFC during malingered false alarms compared to correct rejections from the optimal performance condition, explained as an increased need for the inhibition of prepotent responses. These three studies employing neuroimaging methods and an instructional manipulation to encourage feigned memory impairment have provided early evidence for the role of cognitive control regions in the behavior of malingering. As in these studies, individuals feigning memory impairment in the current investigation were likely to engage in processes such as inhibition, performance monitoring and conflict detection. I expanded upon these prior studies in several ways. First, I investigated eye movement behavior as well as explicit responding to more fully understand the behavioral expression of the memory, attention, and cognitive control processes that contribute to feigned memory impairment. Furthermore, correlated activity differences between brain regions were evaluated to examine potential neural interactions that support performance.

While the neuroimaging studies focusing specifically on feigned memory impairment are few, additional evidence for the contribution of cognitive control processes to deceptive behavior has been reported using other tasks (Abe et al., 2008; Phan, Magalhaes, Ziemlewicz, Fitzgerald, Green, & Smith, 2005). Many of these investigations instructed participants to respond

incorrectly throughout an entire block or on a particular type of trial, thereby not giving them the same control over their correct and incorrect responses as is seen in investigations of memory malingering. In one such investigation, participants were given two playing cards and were asked to tell the truth about possessing one and lie about possessing the other, while undergoing fMRI scanning. From this task, researchers were able to identify neural regions that were activated by the act of making deceptive responses. Specifically, these deceptive responses were associated with greater activity in the ventrolateral, dorsolateral, and dorsomedial PFC (Phan et al., 2005). Across these studies, it appears that prefrontal regions, such as the dorsolateral and ventrolateral PFC are activated in both simulated memory impairment and directed concealed memory tasks, while the ACC is differentially activated by feigned memory impairment.

As mentioned previously, activation of cognitive control regions was expected in the current investigation as individuals attempt to simulate memory impairment. We expected that participants concealing their memory would show greater recruitment of prefrontal regions as they attempted to maintain and meet the goal of concealing their memory, as well as deal with the conflict between accurate memory retrieval and explicit responding in accordance with the goal. Specifically, the lateral PFC and ACC were two regions of particular interest due to their established roles in response inhibition, goal maintenance, performance monitoring, and conflict detection, all of which seem critical for successful concealment of memory. As such, differences in PFC activity between participants feigning memory impairment and those performing optimally were investigated here. Furthermore, the ACC was interrogated specifically when conflict was expected to be the highest among simulators; namely, when these participants made incorrect recognition responses.

The current work is based on a previous study that we had conducted (Mahoney et al., in prep). This strictly behavioral experiment employed the scene-face task described previously and an instructional manipulation that encouraged half of the participants to feign memory impairment. As a reminder, in this task participants first encode a series of scene-face pairs. In a subsequent memory test, participants are cued with one of the studied scenes and are then shown a display with three studied faces superimposed on top of the scene. In this version of the task, participants were instructed to indicate whether the display contained the associate of the scene (i.e. target-present trials) or not (i.e. target-absent trials), while either trying to perform optimally (i.e. controls) or to feign memory impairment (i.e. simulators). Eye movements were recorded throughout the experiment. Results from this investigation showed that simulator participants were able to conceal memory in explicit responses with performance at chance. Furthermore, they responded more slowly than controls, consistent with the idea that malingering requires more cognitive effort. Despite differences in behavioral performance, early eye movement patterns showed comparable disproportionate viewing of matching associates for both control and simulator participants. Following this early eye-movement-based memory effect, viewing of associates declined rapidly for simulators, suggesting a redirection of attention away from learned materials consistent with instructions to simulate memory impairment. This decline was greater when simulators made incorrect responses and may therefore represent increased engagement of voluntary attentional control mechanisms (see Figure 1B). Finally, in a post-test where memory was tested again but where all participants were now instructed to perform optimally, simulators still showed poorer memory than controls. Exploratory post-hoc analyses suggested that this difference might be a consequence of poor encoding by a subset of simulator participants. Because it was important in the current experiment that materials were well-

encoded by both groups of participants, task instructions were modified. Here, participants were told prior to testing that they should attempt to commit the materials to memory regardless of group assignment and test instructions because a post-test would be administered at the end of the experiment and everyone would be required to do their best. In short, this was meant to ensure that any effects of memory, attention, and cognitive control associated with simulated memory impairment at test could be evaluated under conditions of comparable memory encoding between groups.

The primary question that we aimed to investigate in the current experiment was whether and how information retrieved from long-term memory is prioritized by attention, even when it is counterproductive to the goal of memory concealment. The findings from Mahoney et al. (in prep), which showed early eye movement based memory effects despite feigned memory impairment, indicate that prioritization of retrieved information is likely to occur shortly after test displays are presented. We expected to replicate this eye movement finding and to see that these eye movement effects are predicted by hippocampal activity during the scene cue for both the control and the simulator group. In addition, activity differences in the hippocampus during presentation of the scene cue were expected to predict preferential viewing even when simulators made incorrect recognition responses. These outcomes would reflect an extension of past work using a similar task (Hannula et al., 2009). Again, this experiment showed early eye-movement-based memory effects that were predicted by hippocampal activity during a memory cue in individuals performing the task optimally, even when they made errors, but in that case errors were made in the context of standard recognition memory task instructions.

In addition, based on work that has demonstrated that the hippocampus is activated by successful relational memory retrieval (Prince et al., 2005; Hannula et al., 2013), we expected to

find that activity in the region during presentation of the three-face display would be sensitive to memory success. Specifically, during the test phase, we expected to find greater hippocampal activity for correct over incorrect trials for controls, but not for simulators, as their explicit responding would not consistently represent what was retrieved from memory. For both groups, hippocampal activation at test was hypothesized to predict recognition accuracy on the post-test, where both groups were instructed to perform optimally.

While the above effects investigated hippocampal activity differences based on explicit behavior and eye movements within the simulator and control groups individually, a final hippocampal analysis aimed to see if differences in the region existed between groups. It might be expected that hippocampal activity would be suppressed in data obtained from simulator participants as they attempted to comply with the instruction of feigned memory impairment. Studies of directed memory retrieval suppression have demonstrated reduced hippocampal activation as compared to trials where retrieval is encouraged (Anderson et al., 2004; Depue, Curran, & Banich, 2007; see Anderson & Levy, 2009 for review). Therefore, if simulator participants engage in suppression, despite the absence of any specific instruction to do so, they would be expected to exhibit lower hippocampal activation than controls when three-face displays were in view and memory decisions were being made.

In previous work (Mahoney et al., in prep) redirection of attention away from known materials was demonstrated as a decrease in proportion of viewing of the matching face as the trial progressed, especially on trials where simulator participants incorrectly stated that the matching face was not present. This led to differences in global viewing of the matching associate across the entire trial between controls and simulator participants, and between the correct and incorrect trials of simulators. A similar reduction in global viewing was expected in

the current investigation. Attentional resources that are related to this redirection of attention away from learned materials were investigated with whole-brain analyses conducted on target-present trials, in which simulators might redirect attention away from the associate. It was expected that simulator participants would demonstrate greater recruitment of regions related the voluntary deployment of top-down attention, such as has been found in previous work with the IPS and SPL (Uncapher et al., 2015), and/or to involuntary reorientation, such as TPJ, compared to controls once the test display was presented.

In addition to the memory and attention processes described above, the act of feigning memory impairment was expected to require additional cognitive control as compared to completing the memory test optimally. We expected to see greater activity in the lateral PFC for our simulator group as compared to the control group once the three-face display is presented. At this time, the simulator participants must maintain the goal of concealing their memory and inhibit correct responses, processes that have been linked to lateral PFC in prior work (D'Esposito, 2007; Paxton, Barch, Racine, & Braver, 2008; Chikazoe, Konishi, Asari, Jimura, & Miyashita, 2007; Mostofsky et al., 2003). In addition to greater activity in the lateral PFC for the simulator participants, we also expected to see greater activity in the ACC, especially when simulators respond incorrectly. As described above, this region has been conceptualized as a conflict-monitoring region (Botvinick et al., 2001). The simulator participants should be experiencing conflict between their knowledge and their goal to respond counter to this knowledge on incorrectly answered trials.

Finally, exploratory analyses examined whether or not any correlated activity patterns among brain regions active in this task contributed to simulated memory impairment. This was addressed using psychophysiological interaction (PPI) connectivity analyses. One possibility is

that increased recruitment of the hippocampus, associated with successful memory retrieval would result in increased cognitive control among simulators. To address this possibility connectivity analyses were conducted with four hippocampal seeds (left and right, anterior and posterior) to identify regions where coactivation with these seeds was greater during the three-face display for incorrect than correct trials among simulators. Next, with an aim to identify if ACC activity, reflecting the degree of conflict experienced, led to the recruitment of other prefrontal control regions, as has been suggested in previous work (Kerns et al., 2004), a connectivity analysis with an ACC seed was conducted. Again, this analysis aimed to identify regions where coactivation with the ACC seed was greater for incorrect than correct trials among simulators. Finally if, as predicted, activity differences were reduced among simulators in the hippocampus during presentation of the three-face display, then correlated activity patterns between prefrontal and hippocampal regions would be investigated. Any evidence for lateral PFC-hippocampal connectivity would be consistent with similar interactions observed in studies of directed retrieval suppression (Anderson et al., 2004; Depue et al., 2007), and might be expected to drive post-test performance down among simulators.

Method

Participants

Forty participants were recruited from the University of Wisconsin-Milwaukee and the general Milwaukee area. Four additional participants were scanned, but were removed from all analyses due to insufficient eye movement data. One participant lacked data due to experimenter error, while for the other three, reliable eye movement data could not be obtained. All participants were over the age of 18, right-handed, native English speakers, had normal or

corrected-to-normal vision, and were screened to ensure that they had no MR contraindications. Participants were compensated monetarily or with course credit. Half of these participants were randomly assigned to the simulator group, while the remainder was in the control group. Pre-screening and practice procedures took place at the University of Wisconsin – Milwaukee; fMRI scanning was conducted at the Medical College of Wisconsin Center for Imaging Research. Approval for this investigation was granted by the Institutional Review Boards of both the University of Wisconsin – Milwaukee and the Medical College of Wisconsin.

Materials

Materials for this investigation included 344 images of scenes (172 indoor and 172 outdoor) and 344 faces (172 male and 172 female) selected from a pre-existing stimulus set (cf. Hannula et al., 2007). Scenes were sized to 800x600 pixels, while faces were sized to 280x280 pixels and were superimposed on a 300x300 pixel grey background. From the above set of materials, 28 scenes and 28 faces were used in a practice phase and 100 scenes and 100 faces were used as materials for a functional localizer task. The remaining 216 scenes and 216 faces were used in the experiment proper.

Procedure and Design

Participants for this experiment completed two sessions. In the first session, informed consent was obtained and participants were screened for exclusion criteria and MR contraindications. Following this, task instructions were reviewed for the encoding and test phases regardless of group assignment (see below for more detail) and participants completed a short set of practice trials. During this session, it was determined whether reliable eye tracking data could be obtained from the participant. If participants were deemed eligible to continue in the experiment, a second session was scheduled for MRI scanning. Eight participants were

screened but not scheduled for a second session; four had MR contraindications and another four had unreliable eye movement acquisition.

During the second session, consent, safety issues, and general task instructions were reviewed. Participants had either been assigned to the control group or the simulator group during session 1, and instructions corresponding to group membership were provided at the beginning of the second session. Both groups of participants were instructed to imagine that they had been in a car accident at the fault of another driver that had resulted in a concussion. Participants assigned to the control group were told that they needed to perform their best in order to be cleared to return to work. Participants assigned to the simulator group were told to attempt to conceal memories (i.e. feign memory impairment) in order to obtain a larger judicial settlement. This instructional manipulation was similar to what has been used in prior work (Mahoney et al., in prep; Suhr & Boyer, 1999) and can be found in full in Appendix A. Specific strategies indicating how exactly to simulate memory impairment were not provided – for example, participants were not told to respond incorrectly to a particular percentage of items nor were they given any instruction about how to direct their gaze when test displays were presented. They were, however, encouraged not to engage in behavior that would make their intentions obvious (e.g. making an incorrect response on every single trial, not responding to trials). All participants were also instructed that they would have to complete a post-test optimally without an additional opportunity to study the materials. This instruction was included to encourage comparable initial encoding between groups, as poorer encoding may have contributed to the performances of a subset of simulator participants in previous work (Mahoney et al., in prep).

During fMRI scanning, participants completed 6 sets of interleaved encoding and test blocks with new materials used in each block. Eye movements were recorded throughout. Prior

to the first block, an eye tracking calibration procedure was conducted using a 9-point calibration screen. Thirty-six scene-face pairs were presented in each encoding block and participants were instructed to commit these pairs to memory. For each trial, a scene was presented for 500ms; a face was then superimposed on top of the scene and the pair remained in view for 1 second. A jittered inter-trial interval consisting of a blank grey screen was presented for 500, 2500, or 4500ms. Individual pairs were presented two times, each in a different random order prior to administration of the corresponding test block.

Test blocks consisted of 12 trials and began with the presentation of one of the encoded scenes for 1 second. Participants were instructed to use this scene as a cue to recall the associated face. A jittered delay of 1000, 3000, or 5000ms followed, where participants were instructed to maintain a picture of the matching face in mind. Then, three studied faces (matched for gender)

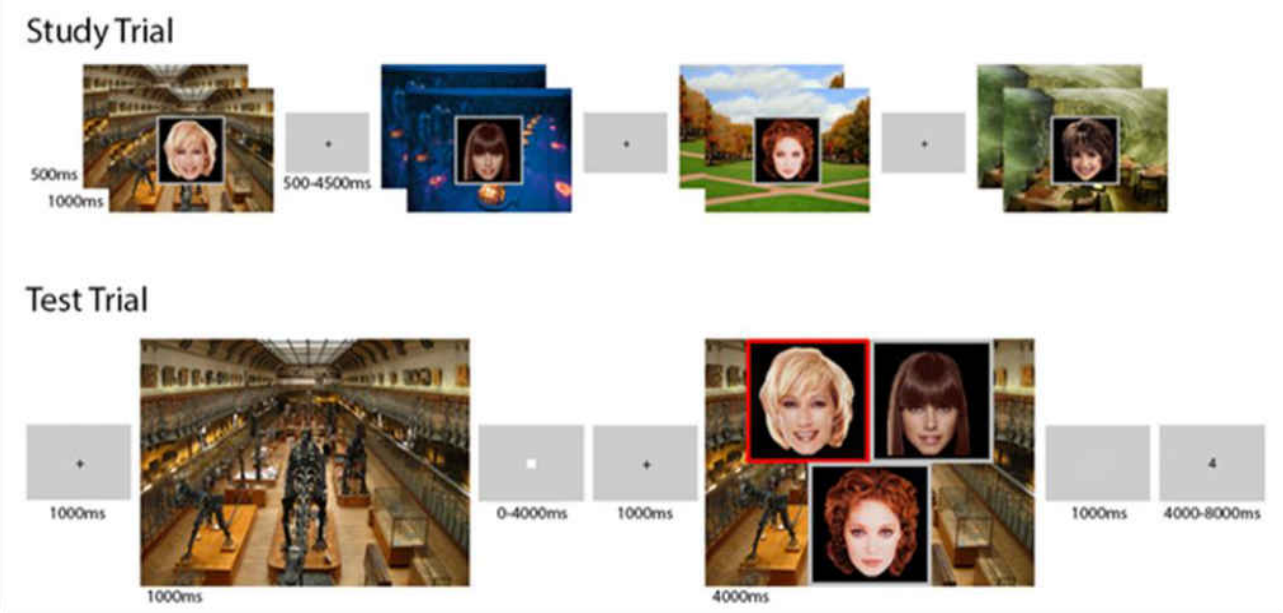

Figure 2. Example study and test trials. During encoding, participants study a series of scene face pairs. At test, participants are cued with a scene, which is then superimposed with three studied faces. Participants are instructed to indicate if the matching associate is present or absent. The same test display is seen by 3 participants in each group, for two of whom it is target-present (as seen here) and for one it is target-absent. Proportion of viewing directed to the same face, depicted here with a red square for illustration purposed, was used in all eye movement analyses.

were superimposed on top of the scene, and this three-face display remained in view for 4 seconds. Eight of the test displays included the face that had been paired with the scene during encoding (i.e. target-present trials), while the remaining four did not (i.e. target-absent trials; see Figure 2).

Participants were instructed to view the display and to indicate via button press whether the associated face was present or absent. As described above, control participants had been instructed to perform their best, while simulators were instructed to simulate memory impairment. A jittered inter-trial interval of 6000ms, 8000ms, or 10,000ms followed each trial, during which participants were instructed to respond to a series of numbers, each presented for 2000ms, as to whether they are less than or greater than five. This procedure represented an active baseline meant to disrupt any residual memory effects or mind wandering between trials that might be expected to involve MTL, prefrontal, or parietal lobe structures and make retrieval-based activity more difficult to detect (Stark & Squire, 2001; Stark & Okado, 2003). Examples of study and test trials are illustrated in Figure 2. This encoding-test process was repeated 5 more times with new materials each block. Collapsed across blocks, data were obtained from 72 test trials (48 target-present trials and 24 target absent trials).

Following all 6 study-test block sequences, participants completed a functional localizer task. This task consisted of 10 blocks of 10 faces alternated with 10 blocks of 10 scenes. Each face and scene was presented for 1500ms separated by a 500ms blank screen. Participants were asked to perform a 1-back task, and indicated for each stimulus whether or not it was an exact match of the face (or scene) presented on the previous trial by making button press. Data from the functional localizer are not reported here as they are not central to predicted activity differences.

A post-test was administered approximately 20 minutes after the functional localizer, outside of the scanning environment. During the post-test, participants were presented with all 72 three-face displays (48 target-present, 24 target-absent) that had been seen in the experiment proper, in a new random order. Event timing was contingent on participant's button presses. During the post-test, participants were told to identify the face that had been paired with the scene cue during encoding, selecting a face and making a response even if they felt that the associate was not present. Following their button press, the three-face display was removed from view and the participants were instructed to make another button press indicating whether they felt the associate was in the display or not (i.e. a present/absent judgment). By requiring a selection of the associate along with the present/absent judgment, we were able to identify scene-face pairs that were successfully learned, using a strict criterion of both correct selection of the associate and appropriate identification of the trial as target-present. Critically, and in contrast to what was described above, all of the participants, irrespective of group assignment, were instructed to perform optimally on the post-test (i.e. simulators were no longer encouraged to conceal memories). Post-test administration allowed us to evaluate whether encoding of materials was comparable between groups and permitted evaluation of neural activity that was associated with successful memory outcomes for both groups.

Finally, following completion of the post-test, all of the participants were asked to fill out a questionnaire, which assessed their understanding of the group assignment instructions, along with their effort, confidence, and motivation to comply with those instructions. Compliance ratings were made using a scale from 0 ("no effort/motivation/confidence") to 5 ("great effort/motivation/confidence"). Participants assigned to the simulator group were then also asked to report any strategies they had used to accomplish the task, selecting from several provided

strategies as well as having the opportunity to write in strategies not listed. Following completion of the post-test questionnaire, all participants were compensated and fully debriefed.

Counterbalancing

For counterbalancing purposes, individual scenes were randomly assigned to one of 9 lists (18 scenes per list), each with equal numbers of indoor and outdoor exemplars. Faces were also randomly assigned to one of 9 lists (18 faces per list), each with an equal number of male and female exemplars. Across participants, lists of faces and scenes rotated over experimental blocks and conditions, and respective lists of faces and scenes were paired equally often.

Individual participants within a group were yoked so that each three-face display was seen by three participants. Differences in encoding history meant that the same display was target-present (i.e. contained the studied associate) for two participants and target-absent (i.e. did not contain the studied associate) for another. Use of this yoking procedure means that the same face, presented in the context of the same test display, served as the critical face for purposes of viewing time analyses for both target-present and target-absent trials. Participants were also yoked across groups so that corresponding controls and simulators experienced the exact same encoding and test events. Finally, counterbalancing also ensured that across test trials, the critical face appeared equally often in all three spatial locations for each experimental condition (i.e. target-present, target-absent).

Image Acquisition and Preprocessing

Functional MRI data were collected using a GE Healthcare Discovery MR750 3T MRI scanner at the Medical College of Wisconsin Center for Imaging Research. Stimuli were presented onto a screen from a rear projector and viewed by the participant through a mirror attached to the 32-channel head coil. Whole brain functional images were collected using a

gradient echoplanar imaging (EPI) pulse sequence (TR: 2s; TE: 25ms, FOV: 22cm; 64 x 64 matrix). Each volume consisted of 36 axial slices with a slice thickness of 3.4mm, resulting in a voxel size of 3.44 x 3.44 x 3.4mm. In addition to the EPI scans, a high-resolution T1-weighted spoiled gradient recalled (SPGR) anatomical image (TR: 8.1s; TE: 3.2ms; FOV: 25.6cm; 256 x 256 matrix) was collected from each participant consisting of 208 axial slices with a slice thickness of 1.2mm, resulting in a voxel size of 1.00 x 1.00 x 1.20mm.

Preprocessing of MRI data was performed using Statistical Parametric Mapping (SPM8) software including slice-time correction, realignment, normalization, and spatial smoothing. The T1-weighted anatomical image was segmented into grey matter, white matter, CSF, and non-brain tissue and then coregistered to a template in Montreal Neurological Institute (MNI) space. Functional EPI data was slice-time corrected using sinc interpolation, to account for differences in time of acquisition of different slices, and realigned to correct for motion using a six-parameter, rigid body transformation. EPI data were realigned to MNI space, resliced into 3mm isotropic voxels, and smoothed using a 6mm full-width at half-maximum Gaussian filter. The Artifact Detection Tools (ART; http://www.nitrc.org/projects/artifact_detect) SPM-based toolbox was used to identify time points that exhibited greater than 1mm or .1 radians of translational or rotational scan-to scan movement, respectively, or greater than a 2% global mean signal change (e.g., Wang, Ritchey, Libby, & Ranganath, 2016); as indicated below, each of these time points was entered as a covariate of no interest in the general linear model. No participants exhibited excess motion (>3mm) during the functional runs.

Data Analysis

Explicit response accuracy and response times. Participants made their explicit recognition responses using a button box. From these responses, each target-present trial was

classified as a hit if the participant indicated that the associate was present and a miss if they stated that it was not present. Each target-absent trial was classified as a correct rejection if the participant correctly indicated that the associate was not present and a false alarm if they stated that it was present. The percentage of hits, misses, correct rejections, and false alarms was used to compute d' and corrected recognition (Hits + Correct Rejections/2) scores for each participant. Similar indices of accuracy were computed for the post-test data. In addition, a strict measure of accuracy was computed for target-present trials from post-test data. In this case, a trial was designated correct if participants correctly identified the associate and then went on to indicate that the display was "target-present". All other combinations of trials were coded as incorrect.

In addition to gathering information about the accuracy of the participants' button presses, we also collected response time data. For each participant, average response times for target-present and target-absent trials were calculated separately for correct and incorrect responses. Response times were investigated separately based on accuracy of response because incorrect responses are typically made more slowly than correct responses.

Eye movement behavior. Eye position was monitored during fMRI scanning at a rate of 120 Hz using an MRI-compatible Applied Science Laboratories (ASL) long-range optics eye tracker. Eye-tracking analyses focused on eye movements that occurred during the 4 seconds that the three-face display was presented. Fixations made during this period were assigned to particular regions of interest (ROIs) within each three-face display (i.e. left face, right face, bottom face, background scene) and the proportion of total viewing time allocated to each ROI was calculated. Trials for which eye position was lost or unreliable (i.e. total viewing time less than 55% of the 4000ms trial) were dropped from all of the eye-movement-based contrasts reported below, as has been done in previous work using this task (Hannula & Ranganath, 2009).

On average, approximately 9 trials were removed per participant and there was no significant difference between the number of trials dropped for simulators [M=9.5, SD=9.5] and controls [M=7.8, SD=8.9; t(38)=.60, p=.55].

Eye movement analyses were based on viewing time directed to two faces of interest: 1) associates of scene cues from target-present trials (referred to subsequently as *targets*), and 2) *matched comparison faces* from target-absent trials. As indicated above, matched comparison faces served as targets for other participants in the counterbalanced design. Because matched comparison faces were not associates of corresponding scene cues, and cannot be differentiated from other encoded faces in the three-face test display as a function of past associative experience, viewing time directed to these faces was expected to be well-matched whether present/absent memory responses were correct or not. Analyses confirmed that there was no difference in viewing directed to comparison faces in target-absent displays that were correctly or incorrectly identified for simulators, with one simulator excluded from the analysis due to insufficient incorrect trials [t(18)=1.25, p=.23]. The same analysis was performed on the 11 control participants that had sufficient incorrect target-absent trials, and again no differences in viewing were found [t(10)=.40, p=.70]. Therefore, across all eye-tracking analyses correct and incorrect target-absent trials were collapsed. Consistent with previous work (e.g., Mahoney et al., in prep; Hannula et al., 2007), effects of memory on eye movement data were evaluated using: 1) global (or collapsed) indices of viewing time, and 2) time-course analyses.

Global/collapsed viewing analyses. The proportion of total viewing time directed to faces of interest (i.e. targets from target-present displays and matched comparison faces from target-absent displays) was calculated for every trial collapsed across the entire 4 second three-face display presentation period. Comparisons were then performed to determine whether or not there

were differences in the proportion of total viewing time directed to faces that had been paired with corresponding scene cues (i.e. targets) versus matched comparison faces that were not studied associates of the scene cues. An additional analysis was performed in order to determine whether the expected relative reduction in memory-based viewing by simulators was eliminated when error trials were removed.

Time-course analyses. Time-course analyses permit evaluation of viewing as it unfolds over the course of the test trial. For this purpose, data from individual trials were subdivided into consecutive 250ms time bins starting with three-face display onset (i.e. 0-250ms, 250-500ms … 3750-4000ms) and the proportion of viewing time directed to targets and comparison faces was calculated for each bin. Analyses were conducted across all sixteen time bins as well as within individual time bins to establish how early disproportionate viewing occurs. Difference scores were also used to index the magnitude of memory-based viewing (i.e. viewing directed to targets minus comparison faces) and contrasts were performed to determine whether early effects are well matched between groups.

Finally, a set of analyses was performed on simulator data only to identify whether memory-based viewing effects differ whether explicit recognition responses were correct or not. Similar to the analyses above, analyses were conducted across the sixteen times bins, within early time bins to establish when disproportionate viewing emerges, and using difference scores to assess the magnitude of the memory-based viewing. Here the analyses included a factor of accuracy or were conducted separately for correct and incorrect target-present trials.

Neuroimaging analyses. Event-related BOLD responses for each component of the test trials (i.e. scene cue, three-face display) were deconvolved using linear regression (Zarahn, Aguirre, & D'Esposito, 1997). These vectors of neural activity were then convolved with

canonical hemodynamic response functions (HRF) to create covariates of interest. Specific covariates of interest for particular trial components (scene cue and three-face display) were created based on behavioral and eye movement behavior as dictated by the objective of a particular contrast. Additional covariates of no interest modeled suspect time points as identified by ART, effectively removing these time points from the sequence, motion, scan-specific baseline shifts, and trials with insufficient eye-tracking data or no behavioral responses. Regression analyses were then performed on single-subject data using the general linear model with a high-pass filter of (1/128) Hz applied to eliminate low-frequency noise. These analyses resulted in a set of parameter estimates for each participant, for which the magnitude can be interpreted as an estimate of the BOLD response amplitude associated with a particular trial element. After single-subject analyses were completed, two types of group-level analyses were conducted. When anatomically-specific predictions had been made, parameter estimates were extracted from targeted regions of interest (ROIs) for trial components and conditions of interest for each subject using MarsBaR (http://marsbar.sourceforge.net/). These parameters were then evaluated using repeated measures ANOVAs and/or t-tests as dictated by the objective of each analysis. Exploratory, whole-brain analyses were corrected for multiple comparisons using non-parametric permutation testing implemented in FSL's Randomise function (http://fsl.fmrib.ox.ac.uk/fsl). For each contrast, the null distribution of the maximum cluster mass was obtained by randomly flipping the sign of individual statistical maps 5000 times and thresholded with a voxel-wise threshold of $p<.005$. Clusters were identified using these distributions that were significant at a family-wise error rate of $p<.05$.

In order to investigate interactions between memory, attention, and cognitive control regions, three psychophysiological interaction (PPI) functional connectivity analyses were

planned. SPM's PPI toolbox was used for this purpose. To start, for each participant, the time series of BOLD signal was extracted and deconvolved for a particular contrast from a seed region. An interaction term was then created by combining the deconvolved activity from the seed region and the experimental vector. This interaction term was convolved with a canonical HRF and entered as a regressor into a new first-level model, along with regressors for the seed activity and experimental contrast individually to account for main effects. Regions where this psychophysiological interaction regressor predicted activity were identified and corrections for multiple comparisons were conducted as above using FSL's Randomise function.

ROI definition. For each participant, four hippocampal ROIs were manually traced on their high-resolution anatomical image (left and right, anterior and posterior hippocampus). Anterior and posterior subdivisions of the hippocampus were identified according to previously published guidelines (Insausti et al., 1998; Franko, Insausti, Artacho-Perula, Insausti, & Chavoix, 2014). Specifically, the hippocampus was traced on each slice in the coronal plane. The most posterior slice of the anterior hippocampus was the last slice in which the gyrus intralimbicus was visible; the posterior hippocampus began on the very next slice. These four ROIs were then coregistered along with the high-resolution structural image to a participant's mean functional image and resliced (for a representative example, see Figure 6F). For hippocampal ROI analyses, functional images were slice-time corrected and realigned to account for motion. All parameters were therefore extracted from native-space, unsmoothed data. In addition, an ACC ROI was created for use in an ROI analysis from the Cingulate Cortex Anterior Division structure in the Harvard-Oxford probabilistic atlas, using a threshold of 25 percent (see Figure 7A), as has been done in previous work (Merkl et al., 2013).

Scene cue analysis. It was predicted that early disproportionate viewing of matching associates would be predicted by hippocampal activity during the scene cue, as has been demonstrated in previous work (Hannula & Ranganath, 2009). This effect was predicted for both groups, as well as specifically for trials in which simulator participants responded incorrectly. For this analysis, the proportion of time spent viewing the target face in the first 1000ms was calculated for each target-present trial. This time period was chosen based on prior work that demonstrates this is when early eye movements are comparable between controls and simulators (Mahoney et al., in prep). Trials were then divided for each subject into "high" and "low" viewing trials using a median split. Parameter estimates were extracted from the hippocampal ROIs for the scene cue for high and low viewing trials separately and then compared with repeated-measures ANOVAs for each group. This analysis was repeated for simulators using only incorrect target-present trials in order to investigate whether hippocampal activity predicted early disproportionate viewing despite successful concealment of memory. Efforts were made, in this experiment, to ensure that encoding was successful (e.g., two encoding exposures to each pair). This was done to ensure that participants assigned to the simulator group could simulate impairment despite having good memory for the pairs. As such, several participants assigned to the control group made few errors on test phase present/absent judgments and evaluation of viewing effects associated with incorrect trials in this group was not a priority, or a possibility.

Three-face display analyses. In addition to examining how hippocampal activity during the scene cue relates to later eye movement effects, several analyses examined differences in hippocampal activity once the three-face display was presented. The first analysis was conducted to investigate whether hippocampal activity at this time was related to accuracy in present/absent judgments during the test phase. For controls, test accuracy reflects memory encoding and

retrieval success, and thus it was expected that hippocampal activity would be greater for correct compared to incorrect trials. On the other hand, the explicit responding of simulators does not reflect memory success, and therefore this difference was not expected. To conduct this analysis, parameters were extracted from the hippocampal ROIs during the three-face display for correct and incorrect trials separately and were subjected to repeated measures ANOVAs for each group.

Accuracy on the post-test should reflect encoding success for both groups, as all participants were instructed to perform optimally. Therefore, similar analyses as above were performed to examine how activity during the three-face display at test predicted accuracy on the post-test. It was predicted that hippocampal activity would be greater for trials that were subsequently answered correctly for both groups. To conduct this analysis, parameter estimates were extracted from the hippocampal ROIs during the three-face display for trials that were subsequently responded to correctly and incorrectly on the post-test separately and were subjected to repeated measures ANOVAs for each group.

A final hippocampal ROI analysis was conducted to investigate group differences in activity in the region during the three-face display, regardless of explicit or eye movement behavior. It was expected that, due to attempts to feign memory impairment, hippocampal activity might be suppressed during the three-face display in simulators compared to controls. Here, parameter estimates were extracted from the three-face displays of all trials and subjected to a mixed model ANOVA.

The ACC was also specifically targeted due to a priori hypotheses of its involvement in feigned memory impairment based in its demonstrated role in conflict monitoring. Here differences in ACC activity were examined during the three-face display for correct compared to incorrect trials. For simulators, responding incorrectly was expected to generate more conflict

than responding correctly and so greater ACC activity was expected for these incorrect trials. This difference was not expected for control participants, who should not experience the same conflict when answering incorrectly. Parameter estimates were extracted from the ACC ROI described above for correct and incorrect trials for each subject and then compared in paired t-tests for each group.

Exploratory whole-brain analyses were performed to identify brain regions that were more active for simulators than for controls. In the first analysis, activation was examined during the three-face display of all trials. Simulator participants were expected to be engaging cognitive control processes, such as performance monitoring and response inhibition, during the three-face display on both target-present and target-absent trials. Therefore, it was predicted that there would be greater lateral PFC activation for simulators over controls in this analysis.

The next whole-brain analysis examined activation of brain regions during the three-face display, limited to target-present trials. In these trials, where the matching associate appeared in the display, it was expected that simulators might engage in greater redirection of attention away from the associate than controls. Therefore, it was expected that there would be greater activation for simulators than controls in regions implicated in top-down, voluntary deployment of attention, such as the IPS and SPL, as has been seen in previous work on deceptive responding (Uncapher et al., 2015) and in reorientation regions such as the TPJ.

Connectivity analyses. Two PPI connectivity analyses were conducted to test for relationships between memory, attention, and cognitive control regions during this task. In the first, which was conducted only with simulator participants, four hippocampal seeds were used. The time series was extracted from each of the four anatomically defined seeds as described above (left and right, anterior and posterior hippocampus) this time manually drawn on the MNI

single-subject template brain. For this analysis the contrast used was incorrect trials greater than correct trials during the three-face display, based on results from the univariate contrasts. It was expected that correlated activity differences would be observed between the hippocampus and cognitive control regions for incorrect compared to correct trials, as simulators might require recruitment of these processes to make incorrect responses in the face of strong memory retrieval.

In the next analysis, again performed within the simulator group, the ACC was used as a seed region. Again, the anatomically defined ACC ROI was used and the time series was extracted from the incorrect greater than correct trials contrast, based on findings from the univariate analysis. Here it was expected that greater connectivity between the ACC and other prefrontal cognitive control regions would be found for incorrect trials based on previous work demonstrating that ACC activity differences are correlated with these regions when representation of a goal needs to be reestablished in working memory (Kerns et al., 2004).

As results did not show evidence of hippocampal suppression among simulator participants, and there was no evidence for suppression-related impairment in post-test performance, the final proposed connectivity analysis with a prefrontal seed was not conducted.

Results

Recognition Accuracy

Test phase: Controls outperform simulators in accordance with instructional manipulation. During the test phase, between-groups comparisons of corrected recognition (i.e. Hits + Correct Recognitions/2) and d' scores confirmed that controls outperformed simulators [see Figure 3A; t's(38)≥6.03, p's<.001, Cohen's d≥1.96]. Furthermore, while control group performance was reliably greater than chance on both measures [t's(19)≥7.07, p's≤.001], the

same could not be said for simulators [t's(19)≤ .20, p's≥.84]. Collectively, these outcomes indicate that simulators complied with the instructional manipulation and successfully concealed memories in their behavioral responses.

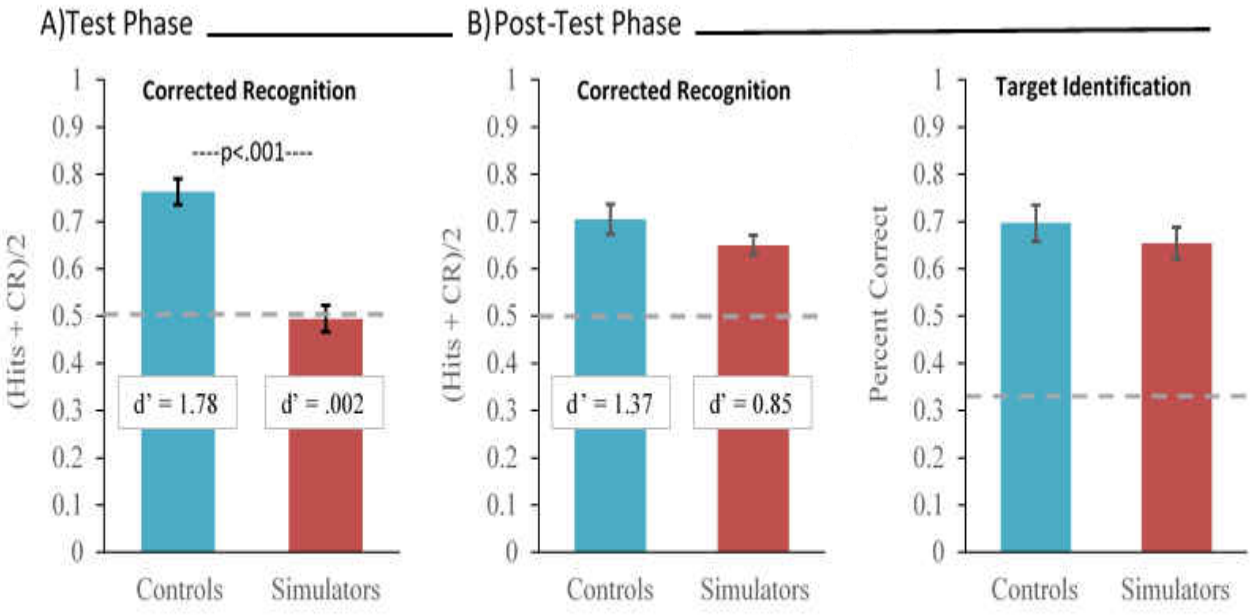

Figure 3. Explicit recognition performance. A) Corrected recognition and d' prime scores for present/absent decisions during the test phase; simulator participants were instructed to feign memory impairment. B) Corrected recognition and d' scores for present/absent decisions during the post-test phase (left) and percentage of successful target (i.e. associate) identification from target-present displays during the post-test (right). Both groups of participants were instructed to perform optimally prior to post-test administration. Standard error bars are plotted around the mean and dashed grey lines mark chance performance.

Post-test phase: Comparable performance between controls and simulators.

Participants assigned to both groups were instructed to perform optimally on the post-test. In line with these instructions, there were no significant differences between control and simulator performance on the post-test. This was true when statistics were performed using corrected recognition scores [see Figure 3B; t(38)=1.53, p=.13, Cohen's d=.50], d' scores [t(38)=1.82, p=.08, Cohen's d=.62], or accuracy in identifying the associate on target-present trials [see Figure 3B; t(38)=.88, p=.38, Cohen's d=.28]. In addition, a strict, collapsed measure of post-test

performance defined previously was calculated for target-present trials and again with this measure of accuracy, no significant differences were seen in post-test accuracy between controls and simulators [t(38)=1.23, p=.28, Cohen's d=.40]. These results indicate that encoding of the scene-face pairs was comparable across groups, and that the act of feigning memory difficulties did not impair later memory retrieval for simulators.

Responses made on the post-test questionnaire confirmed that participants understood task instructions and had attempted to do their best to comply with what they had been told to do. While between group differences were found for self-reported effort [controls: M=4.8, SD=.52; simulators: M=4.3, SD=.66; t(38)=2.66, p=.01, Cohen's d=.85] and motivation [controls: M=4.75, SD=.55; simulators: M=4.3, SD=.80; t(38)=2.07, p=.05, Cohen's d=.67], ratings were fairly high for both groups and comparable to what has been seen in previous work (Mahoney et al., in prep). Furthermore, there was no difference in self-reported confidence that they had accomplished the instructed objective across groups [controls: M=3.65, SD= .81; simulators: M=3.3, SD=.73; t(38)=1.43, p=.16]. The most commonly reported strategies among simulators were "answering most/all items incorrectly" (n=10), "responding randomly" (n=9), "taking longer than necessary to make responses" (n=7), and "attempting to get a certain percentage correct" (n=7). See Table 1 for full information on the variety of combinations of strategies endorsed by simulator participants.

Table 1
Strategies Endorsed by Simulators on Post-Test Questionnaire

ID	Answered All/Most Incorrectly	Answered in a Pattern	Answered Randomly	Looked Away	Blurred Vision	Percentage	Did Not Respond	Took Longer
101	x		x			x		x
302	x	x		x		x		
103	x							
304						x		
105	x	x	x					
106				x				x
107	x					x		
108			x				x	
109						x	x	
110	x						x	
111	x		x					x
112				x			x	x
113	x							
114			x					
115		x				x		x
116		x						
317		x	x					
118	x		x					
119		x	x			x	x	x
120	x		x	x				x
Total	**10**	**6**	**9**	**4**	**0**	**7**	**5**	**7**

Response Times

To evaluate the response times for the test phase, a mixed model ANOVA with the factors Group (control, simulator), Trial Type (target-present, target-absent), and Accuracy (correct, incorrect) was conducted. Two control participants were excluded due to no incorrect trials for one of the trial types. Results showed faster responses for target-present than target-absent trials [$F(1,36)=3.96$, $p=.05$, $\eta^2=.10$], faster responses for correct over incorrect responses [$F(1,36)=21.29$, $p<.001$, $\eta^2=.37$], but no significant difference in response time across groups

[F(1,36)=.02, p=.91, η^2<.001]. However, there was a significant three-way interaction between trial type, accuracy, and group [F(1,36)=14.39, p=.001, η^2=.29].

In order to investigate the three-way interaction, mixed model ANOVAs were conducted for target-present and target-absent trials separately with the factors Group (control, simulator) and Accuracy (correct, incorrect) – see Figure 4. For target-absent trials, there were no effects of accuracy or group, and no significant interaction [F(1,36)<2.08, p's<.16, η^2's<.06]. In contrast, for target-present trials, while there was no significant difference in response times for controls and simulators [F(1,36)=.12, p=.73, η^2=.003], correct responses were faster than incorrect responses [F(1,36)=32.60, p<.001, η^2=.48], and there was a significant interaction between accuracy and group [F(1,36)=46.36, p<.001, η^2=.56]. Bonferroni-corrected follow-up tests demonstrated that for correctly-identified target-present trials controls made faster responses than

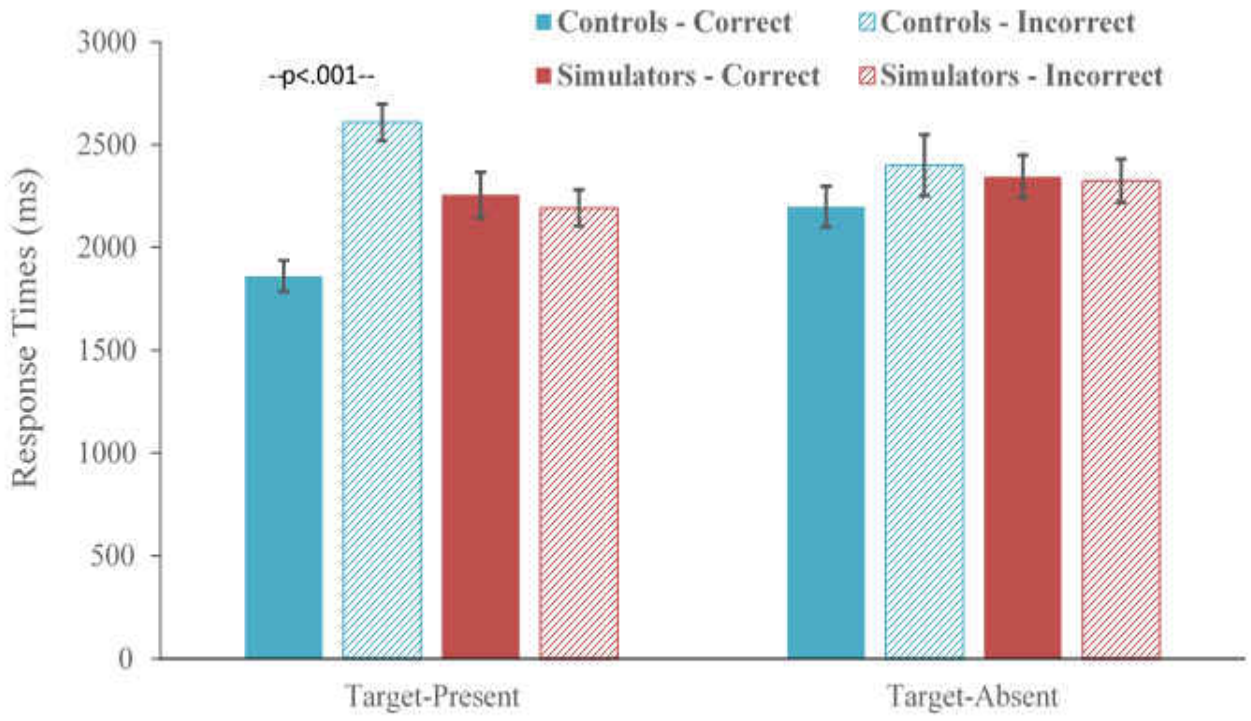

Figure 4. Test phase response times. Response time (ms) for target-present and target-absent trials for controls and simulators, separated by accuracy. Controls showed standard accuracy effects on target present trials, with slower responding for incorrect than correct trials, while simulators showed no difference. No differences in response time occurred on target-absent trials.

simulators [t(38)=2.95, p=.01, Cohen's d=.95], while when responses were incorrect, simulators made significantly faster responses than controls [t(38)=3.33, p=.008, Cohen's d=1.08]. This pattern of between-groups differences is explained by controls demonstrating the standard accuracy effect [i.e. faster responses for correct than incorrect trials; t(17)=8.64, p<.001, Cohen's d=2.08], while there was no significant difference in response times for correct and incorrect trials among simulators [t(19)=.80, p=.44, Cohen's d=.19].

Eye Movement Behavior

Global viewing: Eye-movement-based memory effects are reduced but not absent, among simulators. The first viewing analysis was conducted on the proportion of viewing directed to critical faces collapsed across the full four seconds that the three-face display was presented. Results from a mixed model ANOVA with the factors Face Type (targets, comparison faces) and Group (control, simulator) demonstrated a marginal interaction between face type and group [see Figure 5A; F(1,38)=3.81, p=.058, η^2=.09]. Bonferroni corrected t-tests confirmed that simulators spent reliably less time than controls looking at targets (i.e. the studied associates of scene cues) [t(38)=2.46, p=.02, Cohen's d=.83], but as expected in the absence of a matching associate, there was no between-group differences in proportion of total viewing time directed to comparison faces in target-absent displays [t(38)=.68, p=.50, Cohen's d=.21]. Despite this reduction in target viewing by simulator participants, both groups spent significantly more time viewing targets than comparison faces [controls: t(19)=6.50, p<.001, Cohen's d=1.58; simulators: t(19)=8.52, p<.001, Cohen's d=1.95], indicating that memory for studied relationships influenced eye movement behavior for both groups.

To determine whether the relative reduction in memory-based viewing would be eliminated when error trials were removed from analyses, follow-up comparisons were

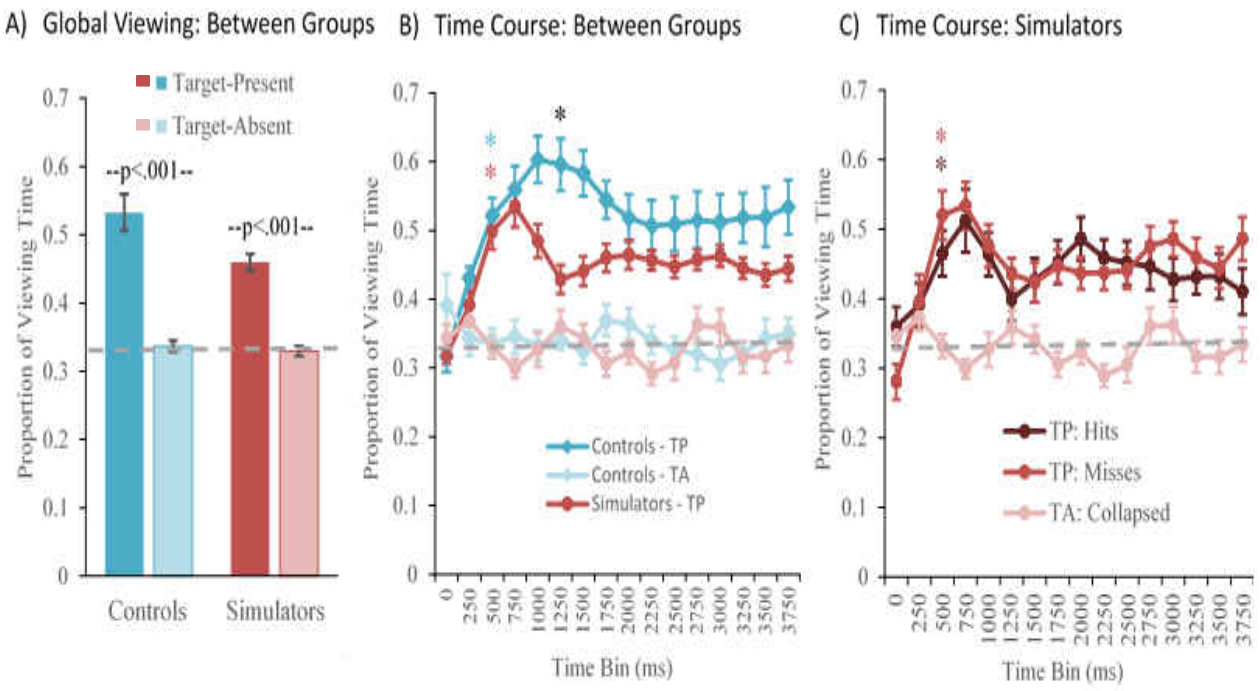

Figure 5. Test phase eye tracking data, controls and simulators. A) Proportion of total viewing time directed to target faces (target-present displays – dark teal and dark red) and comparison faces (target-absent displays – light teal and light red) for controls and simulators. Memory-based viewing was evident for both groups, but was more robust in the control group data. B) Time course of viewing, starting with three-face display onset, subdivided into consecutive 250ms time bins. For each bin, the proportion of total viewing time directed to targets (target-present display – dark teal and dark red) and comparison faces (target-absent displays – light teal and light red) was calculated. Significant disproportionate viewing of targets was evident for both groups within 500-750ms of viewing; significant differences in the magnitude of the effect between groups occurred in the 1250-1500ms time bin. C) Time course of simulator viewing subdivided into 250ms time bins beginning with three-face display onset. Proportion of total viewing time was calculated separately for each time bin with target-present trials subdivided as a function of recognition performance (i.e. hits = target-present displays called "target-present"; misses = target-present displays called "target-absent"). Disproportionate viewing of target faces relative to comparison faces was significant within 500-750ms for both TP-hits and TP-misses. The magnitude of viewing was comparable between TP-hits and TP-misses for the full four seconds.

performed selectively for correct target-present trials. In this analysis, the face type by group interaction remained statistically significant [$F(1,38)=10.26$, $p=.003$, $\eta^2=.21$]. As above, eye-movement-based prioritization of targets was reduced among simulators relative to control group participants [$t(38)=3.75$, Bonferroni corrected $p=.001$, Cohen's $d=1.24$], but effects of memory on eye movement behavior remained statistically significant regardless of group assignment [controls: $t(19)=7.11$, $p<.001$, Cohen's $d = 1.75$; simulators: $t(19)=6.04$, $p<.001$, Cohen's d

=1.42]. In short, despite behavioral performance that was no different from chance for simulators, associates of studied scene cues were still subject to attentional prioritization in this group, although the effect was reduced compared to controls.

Time-course analyses: Instructions to conceal memory do not disrupt early eye-movement-based prioritization of targets. The remainder of viewing results employed time-course analyses to investigate how viewing unfolded across the trial. A mixed model ANOVA with the factors Group (control, simulator), Face Type (targets, comparison faces), and Time Bin (0-250, 250-500 … 3750-4000ms) was calculated using proportion of viewing time as the dependent measure. Most important for our purposes, there was a significant 3-way interaction of these factors [see Figure 5B; $F(6.65, 252.86)=2.19$, $p=.04$, $\eta^2=.06$, G-G$\varepsilon=.44$]. There were also significant main effects of group, face type, and time bin [$F's \geq 4.68$, $p's < .006$, $\eta^2 \geq .11$], which indicate that, overall, controls viewed critical faces more than simulators, targets were viewed more than comparison faces, and viewing changes across the time bins that made up the four seconds of the trial. In an effort to unpack the 3-way interaction, Bonferroni corrected comparisons, limited to the first four time bins following display onset, were performed separately for each group to determine when in time preferential viewing of targets (relative to matched comparison faces) was significant. These comparisons indicated that both groups looked disproportionately at targets by 500-750ms of three-face display onset [controls: $t(19)=5.21$, $p<.001$, Cohen's $d=1.17$; simulators: $t(19)=6.19$, $p<.001$, Cohen's $d=1.44$].

Next, difference scores were calculated and used to index the *magnitude* of memory-based viewing (i.e. viewing directed to targets minus comparison faces) and contrasts were performed to determine whether early effects were well-matched between groups, and whether and when between-groups differences in eye movement-based relational memory effects

emerged. These analyses indicated that the magnitude of memory-based viewing was well-matched between groups within 0-250, 250-500, 500-750, 750-1000, and 1000-1250ms of display onset [t's(38)≤2.29, Bonferroni corrected p's≥.16, Cohen's d≤.73]. Magnitude differences between groups were evident in the next time bin [i.e. 1250-1500ms; t(38)=3.66, Bonferroni corrected p=.004, Cohen's d=1.16]. At this point, the magnitude of memory-based viewing was reduced among simulators, an outcome that is consistent with instructions to simulate memory impairment (see Figure 5B). These results indicate that early memory-based viewing effects were significant in simulator data and were equally robust among simulators and controls shortly after three-face display onset, but declined subsequently as might be expected given the instructional manipulation.

Time-course analyses: Early target viewing is well-matched in simulator data whether explicit recognition responses are correct or incorrect. Time-course contrasts were also performed on simulator data to address whether memory-based viewing effects were well-matched shortly after display onset whether explicit recognition responses were correct or not. A repeated measures ANOVA with the factors Face Type (target-hit, target-miss, comparison faces) and Time Bin (0-250, 250-500 ... 3750-4000ms) was calculated. Results indicated that there were viewing time differences as a function of face type [$F(2,38)=27.17$, $p<.001$, $\eta^2=.59$, G-Gε=.98], that there was a significant change in patterns of viewing with time [$F(4.93,93.60)=3.22$, $p=.01$, $\eta^2=.15$, G-Gε=.33], and that there was a significant interaction between face type and time bin [$F(9.05, 172.00)=2.43$, $p=.01$, $\eta^2=.11$, G-Gε=.30]. Comparisons were performed to determine when memory-based viewing effects emerged, and to determine whether and when the magnitude of memory-based viewing was well-matched or distinct as a function of recognition accuracy. Results indicated that eye-movement-based relational memory

effects were evident for both correct and incorrect trials by 500-750ms of display onset [correct: $t(19)=4.47$, Bonferroni corrected $p<.001$, Cohen's $d=1.12$; incorrect: $t(19)=5.30$, Bonferroni corrected $p<.001$, Cohen's $d=1.29$]. The magnitude of these memory-based viewing effects (calculated separately for correct and incorrect trials) was well-matched across the entire 4s test trial [see Figure 5C; t's$(19)<1.69$, p's$>.11$, Cohen's d's$<.38$]. Considered together, these outcomes indicate that eye movements were sensitive to memory for studied relationships early in viewing whether simulators made correct recognition responses or not.

Neuroimaging Analyses

Hippocampal activity during scene cue predicts early high viewing of matching associates for simulators, but not controls. In the first ROI analysis, the relationship between hippocampal activity during the scene cue and early viewing of the associate was investigated. As no main effects or interactions with viewing were found relating to hemispheric differences [$F(1,19)<2.41$, p's$>.14$, $\eta^2<.11$], analyses were collapsed across left and right ROIs. Thus, for this analysis, parameter estimates for scene

cue activity were extracted from anterior and posterior hippocampal ROIs separately for trials in which targets received "high" and "low" viewing, as defined by a median split of trials based on the proportion of viewing directed towards the target in the first 1000ms. These estimates from each group were subject to a repeated measures ANOVA with factors Viewing (high, low) and ROI (anterior, posterior). For controls, there were no significant differences in hippocampal activity between high and low viewing trials [see Figure 6A; $F(1,19)=.15$, $p=.70$, $\eta^2=.01$], between anterior and posterior hippocampal ROIs [$F(1,19)=.87$, $p=.36$, $\eta^2=.04$], and no significant interaction between viewing and ROI [$F(1,19)=1.47$, $p=.24$, $\eta^2=.07$]. In contrast, for simulators hippocampal activity during the scene cue was significantly greater on trials where

high compared to low viewing of targets subsequently occurred [F(1,19)=6.85, p=.02, η^2=.27]. Hippocampal activity was also greater in the anterior ROI than the posterior ROI [F(1,19)=5.80,

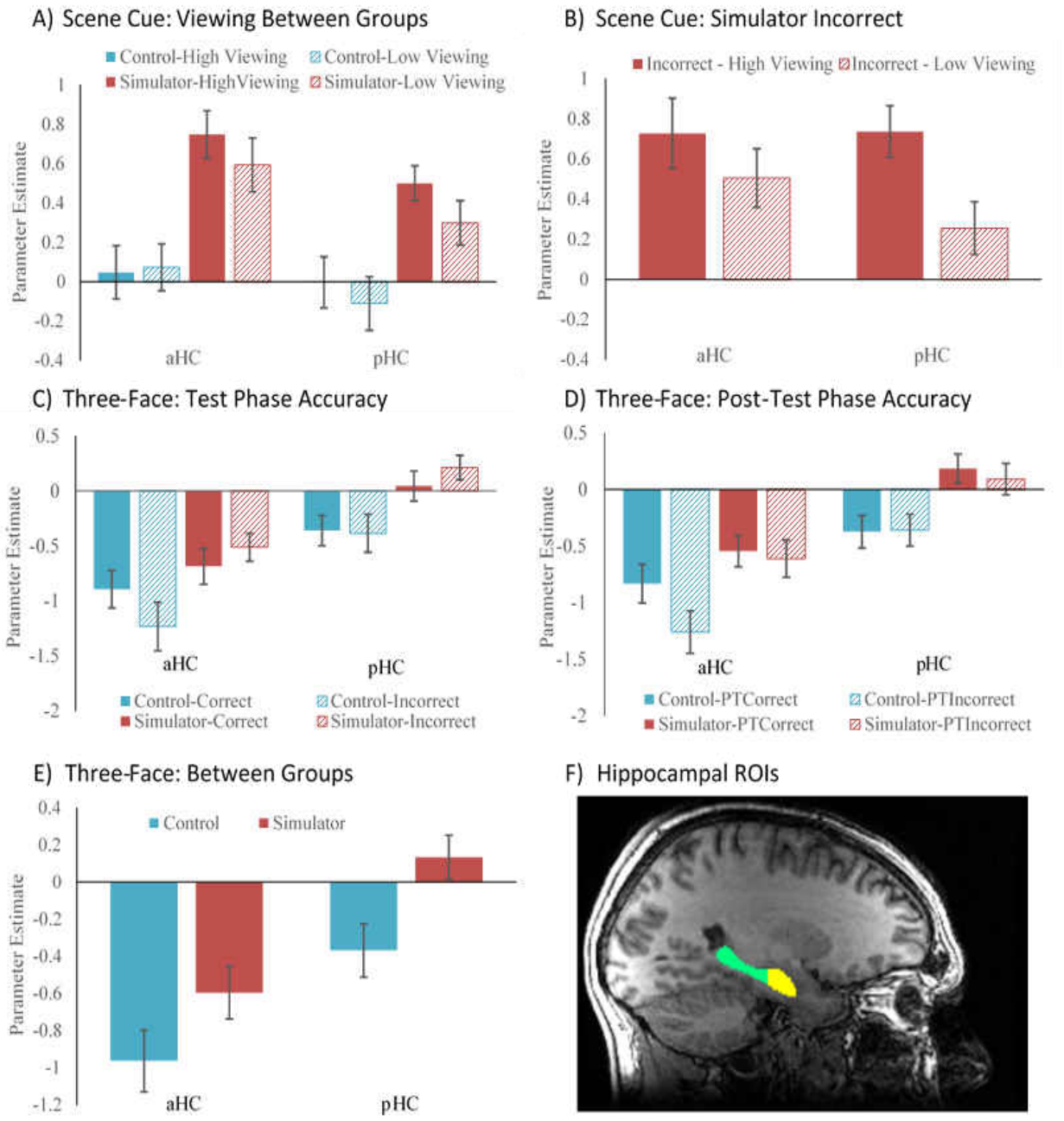

Figure 6. Hippocampal ROI analyses. A) Parameter estimates during the scene cue for trials where high and low viewing of targets subsequently occurred. B) Parameter estimates during the scene cue for incorrect trials made by simulators where high and low viewing of targets subsequently occurred. C) Parameter estimates during the three-face display for correct and incorrect test-phase trials. D) Parameter estimates during the three-face display of the test phase for trials that were subsequently correct and incorrect on the post-test. E) Parameter estimates during the three-face display across all trials. F) An example set of anterior and posterior hippocampal ROIs from one subject overlaid on that subject's high-resolution anatomical scan.

p=.03, η^2=.23], but there was no significant interaction between viewing and ROI [F(1,19)=.19, p=.67, η^2=.01].

The next analysis aimed to determine whether this prediction of viewing by scene cue hippocampal activity was present when simulators concealed their memory by making incorrect responses. Again, no hemispheric effects or interactions were found [F(1,19)<1.81, p's>.19, η^2<.09] so parameters were extracted from left and right ROIs combined. Here, parameter estimates for scene cue activity were extracted from anterior and posterior ROIs for trials in which simulators answered incorrectly, divided into high and low viewing trials with a median split based on the first 1000ms, and were subjected to a repeated measures ANOVA with factors Viewing (high, low) and ROI (anterior, posterior). As seen above, hippocampal activity was greater for trials in which targets received high compared to low viewing [see Figure 6B; F(1,19)=8.33, p=.009, η^2=.31]. There were no significant differences in activity between anterior and posterior hippocampus [F(1,19)=.81, p=.38, η^2=.04], and no significant interaction between viewing and ROI [F(1,19)=2.26, p=.15, η^2=.11]. Together, these analyses have shown that hippocampal activity during the scene cue predicts early disproportionate viewing of targets for simulator subjects, even when they successfully conceal their memory through their button press responses.

Hippocampal activity during three-face display is greater for correct compared to incorrect trials for controls, while the opposite pattern exists for simulators. In the next analysis, how hippocampal activity during the three-face display differentiated between correct and incorrect trials was investigated. One control participant who had perfect accuracy could not be included in this analysis. As above, no hemispheric effects or interactions with accuracy were found [F(1,18)<1.57, p's>.23, η^2<.08] and left and right ROIs were combined. Parameter

estimates for each group from anterior and posterior hippocampal ROIs were entered into repeated measures ANOVAs with factors of Accuracy (correct, incorrect) and ROI (anterior, posterior). For controls, posterior hippocampal activity was reliably greater than anterior [see Figure 6C; $F(1,18)=18.03$, $p<.001$, $\eta^2=.50$], but there was not a significant main effect of accuracy [$F(1,18)=2.00$, $p=.17$, $\eta^2= .10$]. However, there was a significant interaction between accuracy and ROI [$F(1,18)=9.41$, $p=.007$, $\eta^2=.34$]. Bonferroni-corrected follow-up t-tests indicated marginally greater activity for correct compared to incorrect trials in the anterior hippocampus [$t(18)=2.29$, $p=.07$, Cohen's $d=.57$], but no activity differences based on accuracy in the posterior hippocampus [$t(18)=.16$, $p=.88$, Cohen's $d=.04$]. In the ANOVA conducted on simulator data, posterior hippocampal activity was also reliably greater than anterior [$F(1,19)=10.61$, $p<.001$, $\eta^2=.56$] and incorrect trials elicited significantly greater hippocampal activity than correct trials [$F(1,19)=8.83$, $p=.008$, $\eta^2=.32$]. There was no significant interaction between accuracy and ROI [$F(1,19)=.004$, $p=.95$, $\eta^2<.001$]. The results presented here indicate both groups show hippocampal accuracy effects but while controls showed greater hippocampal activity, limited to the anterior hippocampus, for correct over incorrect trials, simulators displayed the opposite pattern, with greater activity for incorrect over correct trials.

Hippocampal activity during three-face display at test predicts post-test accuracy for controls, but not simulators. While the above analysis demonstrates differences in hippocampal activity as a function of test accuracy for the two groups, the next analysis examined whether hippocampal during the three-face display predicted accuracy on the post-test, where all participants were instructed to perform optimally and recognition memory performance was matched across groups. Post-test accuracy was determined by the strict criteria defined previously for target-present trials and present/absent judgments for target-absent trials. No

hemispheric effects or interactions with post-test accuracy were found [$F(1,19)<1.30$, p's>.27, $\eta^2<.06$], so left and right ROIs were combined. Parameter estimates from the three-face display during the test phase for each group from anterior and posterior hippocampal ROIs were entered into repeated measures ANOVAs with factors of Post-Test Accuracy (correct, incorrect) and ROI (anterior, posterior). For controls, most importantly there was a significant interaction between post-test accuracy and ROI [see Figure 6D; $F(1,19)=17.22$, p=.001, $\eta^2=.48$]. There were also significant main effects of ROI with greater posterior compared to anterior hippocampal activity [$F(1,19)=18.64$, p<.001, $\eta^2=.50$] and of post-test accuracy with greater activity during test on trials that were subsequently answered correctly [$F(1,19)=5.00$, p=.04, $\eta^2=.21$]. To address the interaction, Bonferroni-corrected follow-up t-tests indicated significantly greater test phase activity for trials that were subsequently answered correctly on the post-test (vs. those that were not) in the anterior hippocampus [$t(19)=3.56$, p=.004, Cohen's d=.80], but no activity differences based on accuracy in the posterior hippocampus [$t(19)=.15$, p=.89, Cohen's d=.03]. For simulators, posterior hippocampal activity was also reliably greater than anterior [$F(1,19)=10.22$, p<.001, $\eta^2=.53$], but there was no significant difference between activity during test for correct and incorrect post-test trials [$F(1,19)=.86$, p=.37, $\eta^2=.04$] and there was no significant interaction between accuracy and ROI [$F(1,19)=.10$, p=.75, $\eta^2=.005$]. For controls, the pattern here matches what was seen in the previous analysis during the test phase – anterior hippocampal activity during presentation of the three-face display was greater when memory was more accurate. This pattern was not observed for simulator participants.

Simulators display greater hippocampal activity than controls during three-face display. The final hippocampal ROI analysis aimed to identify whether any between-group differences existed in hippocampal activity during the three-face display, perhaps reflecting

suppression of retrieval among simulators. As before, no hemispheric effects or interactions with group were found [$F(1,38)<1.70$, p's$>.20$, $\eta^2<.04$] so left and right ROIs were combined. Parameter estimates were extracted from all trials, regardless of accuracy or viewing patterns, from anterior and posterior ROIs. The estimates were entered into a mixed model ANOVA with factors Group (control, simulator) and ROI (anterior, posterior). In this analysis, simulators showed greater hippocampal activity compared to controls [see Figure 6E; $F(1,38)=6.18$, $p=.02$,

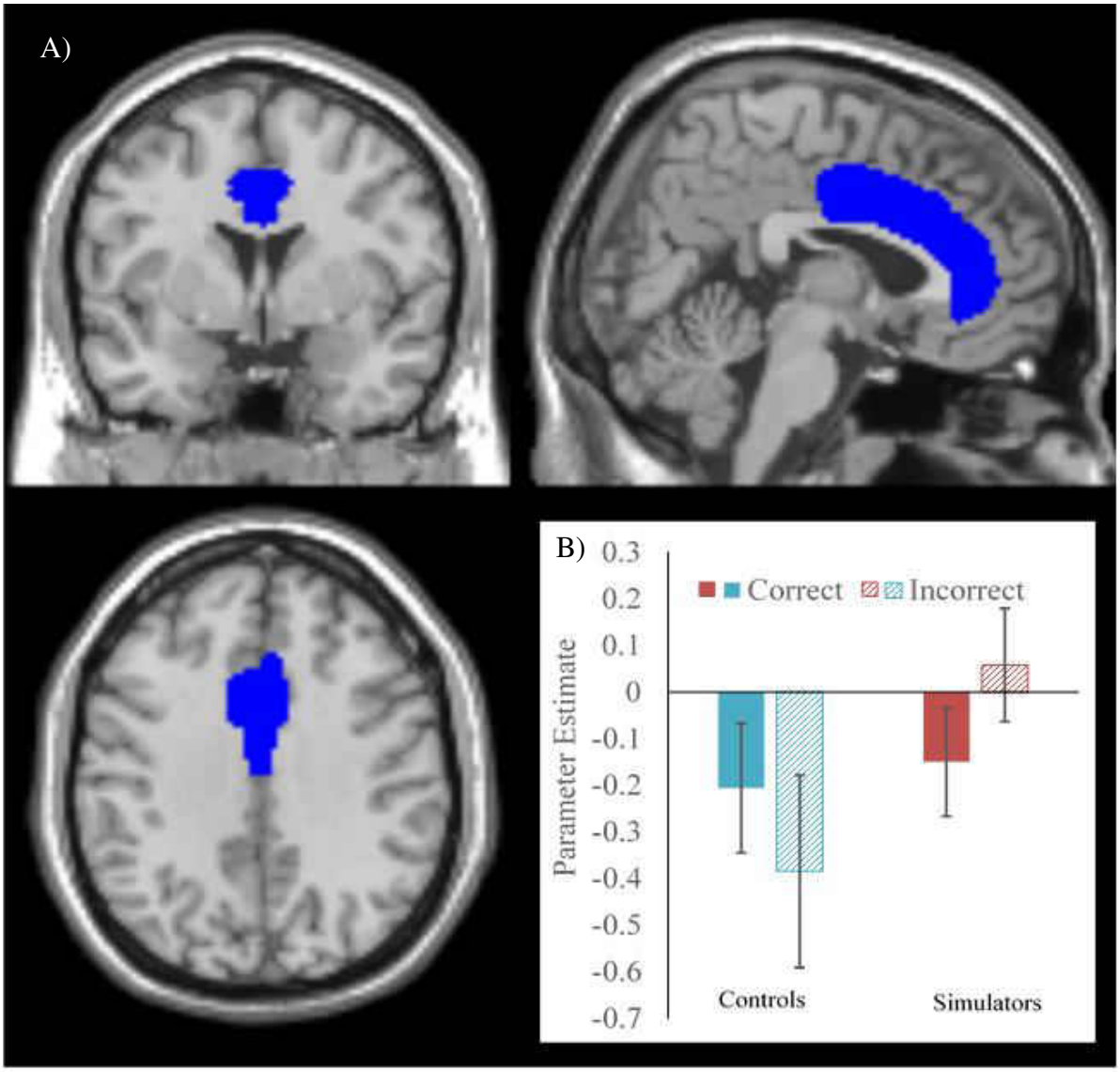

Figure 7. ACC ROI analysis. A) The anatomically-defined ACC ROI displayed on an MNI single subject template. B) Parameter estimates extracted from the ACC ROI during the three-face display for controls and simulators for correct and incorrect trials.

η^2=.14]. In addition, posterior hippocampus was more active than anterior hippocampus [F(1,38)=41.29, p<.001, η^2=.52] and there was no significant interaction between group and ROI [F(1,38)=.42, p=.52, η^2=.01].

Simulators show greater ACC activity during three-face displays for incorrect trials than correct trials. In addition to specifically interrogating the hippocampus, ROI analyses were conducted focusing on the ACC. Specifically, activity differences during the three-face display were investigated as a function of accuracy of explicit responses. For this analysis, parameter estimates were extracted from a bilateral ACC ROI and subjected to paired t-tests comparing correct and incorrect trials. For controls, there was no significant difference in ACC activity between correct and incorrect trials [t(18)=.58, p=.57, Cohen's d=.13]. In contrast, for simulator participants there was reliably greater ACC activity for incorrect compared to correct trials for simulator participants [see Figure 7; t(19)=2.50, p=.02, Cohen's d=.56]. These results suggest that for simulators, but not controls, responding incorrectly recruits greater cognitive control resources than responding correctly.

Simulators show greater right parietal activation than controls during the three-face display of target-present trials. Along with ROI analyses, two exploratory whole-brain analyses were conducted. In the first, activity during the three-face display was compared for simulators and controls across all trials, regardless of accuracy or viewing patterns, aimed to identify cognitive control regions that simulators might recruit for all trial types. Using nonparametric permutation testing with a FWE rate of p<.05, no suprathreshold clusters were identified where activation was greater for either group over the other.

In the second whole-brain analysis, activity during the three-face display was compared for simulators and controls for all target-present trials, regardless of accuracy or viewing

patterns. Limiting to target-present trials here was related to the aim of identifying regions that may be related to reallocation of attention by simulators away from the associate, which did not appear in target-absent trials. In this analysis, following non-parametric simulations to correct for multiple comparisons, one suprathreshold cluster was identified with a peak in the right angular gyrus [x=42, y=-52, z=28, t(38)=3.83] where activity was greater for simulators than controls

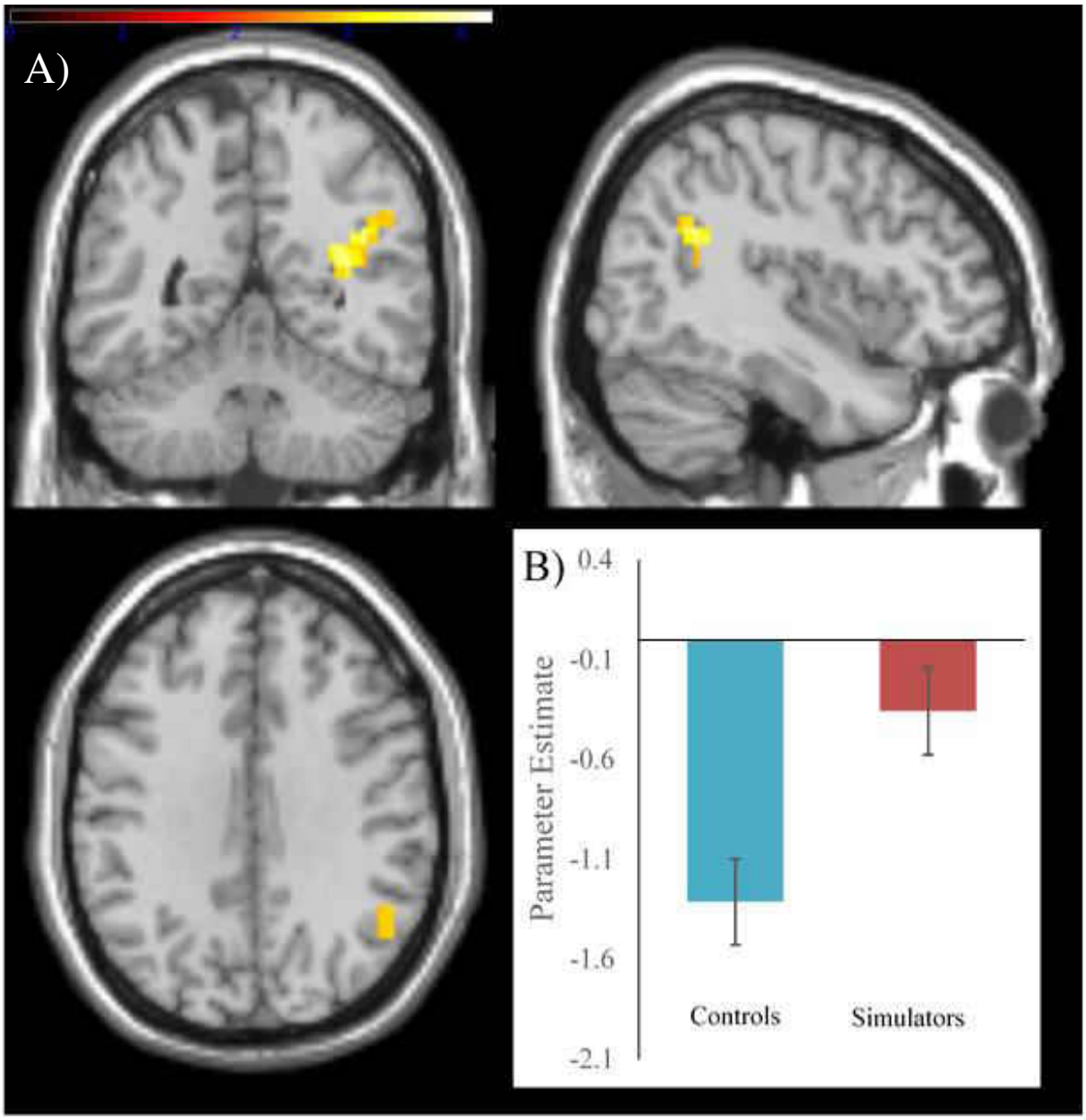

Figure 8. Right angular gyrus cluster. A) Significant cluster in the angular gyrus identified where activation is greater for simulators than controls during the three-face display of target-present trials. B) Parameter estimates from an 8mm sphere centered on the peak voxel from the cluster in the angular gyrus.

(see Figure 8). No suprathreshold clusters were identified in this contrast where control participants showed greater activity than simulators.

No differences in connectivity with the hippocampus or ACC during three-face display between correct and incorrect trials in simulator participants. Two PPI connectivity analyses were conducted to identify regions which showed greater coactivation with memory retrieval and cognitive control regions for incorrect compared to correct trials. First, PPI analyses were conducted using four anatomically defined hippocampal seed regions (left and right, anterior and posterior hippocampus). In another connectivity analysis, an anatomically defined ACC seed was employed. Using whole-brain analyses, regions in which the activity was explained by the interaction between activity in the seed and the incorrect greater than correct experimental contrast were identified. Using non-parametric permutation tests, no suprathreshold clusters were identified as showing greater connectivity with any of the four hippocampal seeds, or the ACC seed, during incorrect over correct trials.

Discussion

The goal of this investigation was to address questions about how memory, attention, and cognitive control are recruited and interact when individuals engage in the complex task of feigning memory impairment. Specifically, I aimed to address whether associations retrieved from LTM are prioritized by attention despite feigned impairment, how attention is reallocated away from learned material, and whether additional cognitive control resources are recruited as participants completed a memory task with the goal to feign memory impairment.

It must first be acknowledged that through explicit responding, the simulator group was successful in concealing their knowledge of the learned associations. Their performance was poorer than that of the control group and not reliably different from chance. This chance-level

performance is comparable to how true patients with hippocampal amnesia performed on a similar task (Hannula et al., 2007). However, these patients did not show any memory-based eye movement effects in patterns of viewing, thus distinguishing them from simulators. Regarding the first aim of this investigation, despite this difference in behavioral performance, there was comparable disproportionate viewing of learned materials in the early eye movement patterns of both groups. Within 500-750ms of the onset of the three-face display, both controls and simulators spent more time viewing target faces than matched comparison faces and the magnitude of this effect was well-matched between groups. This disproportionate viewing occurred even on trials where simulator participants successfully concealed their memory by designating trials that contained the target as "target-absent". These eye movement findings replicate what was seen in a recent experiment (Mahoney et al., in prep). Expanding upon previous work, the neural basis of these eye-movement-based memory effects was examined. ROI analyses demonstrated that hippocampal activity during the scene cue predicted early viewing of targets for the simulator group, with greater activity on trials where high compared to low viewing of targets subsequently occurred. This finding is consistent with previous work (Hannula & Ranganath, 2009), which showed the same pattern of hippocampal activity on a slightly different version of the scene-face task. The work here expands upon these findings by showing that that memory-based prioritization occurs, and is predicted by hippocampal activity, despite individuals' successful feigning of memory impairment.

The finding above that hippocampal activity differences predicted high viewing of associates shortly after test display onset, and did so even when simulators made incorrect responses, also adds to a line of research regarding the automaticity of hippocampal retrieval and the relationship of this process to eye movements. This finding suggests that during the

presentation of the scene cue, pattern completion processes supported by the hippocampus may have led to the retrieval of the associate. Because the representation of this associate was active when the three-face display appeared, early viewing was directed towards the target, even when simulators intended to make an incorrect response. However, given the instructional manipulation used in this experiment, where participants were not informed that eye movements could be used to reveal their knowledge, it could be stated that the disproportionate viewing effects observed here simply represent search for the associate in order to successfully feign impairment, rather than a response to automatic hippocampal pattern completion processes. In this way, it could be that pattern completion in this case is not automatic, but effortful (based on instruction to retrieve the associate) and that viewing effects are purposeful rather than automatic. Nevertheless, there are several reasons to believe that this effect represents an obligatory reaction in response to retrieval of the associate. First is the observation that the effect occurs so rapidly after display onset, within 500-750ms. In addition, several of the simulator participants indicated employing strategies that would not require identification of the associate, such as responding randomly or in some sort of pattern. Unfortunately given the format of the post-test questionnaire, where participants were permitted to select as many strategies as they wished, it is difficult to isolate and compare those who employed strategies that required identification of the associate and those that did not, or to identify when each strategy was employed for those that indicated using both types. Despite this early evidence, it is evident that future work needs to be completed to test the obligatory nature of hippocampal retrieval and its relationship to early eye movement behavior. The results here demonstrate that even if retrieval is effortful, there is a relationship between hippocampal activity during a memory cue and early eye movement behavior.

Surprisingly, the same hippocampal effect predicting early disproportionate viewing was not observed among the control participants, as would be expected given the results of prior work with individuals completing a similar task optimally (Hannula & Ranganath, 2009). However, certain differences do exist between the task that was used in prior work and that in the current investigation. First, the task in the prior experiment was purposely difficult because a key objective concerned evaluation of activity differences in the hippocampus when participants made incorrect recognition responses. In contrast, here, efforts were made to ensure that materials were well encoded so that simulators could make principled decisions (based on memory) about how to modify performance to comply with task demands. Specific differences included a reduction in the number of pairs that were encoded per block as well as an increase in the number of study exposures. While participants in the current experiment were required to encode 36 scene-face pairs at a time, with two exposures per pair, there were 54 encoding trials per block previously and participants only had one opportunity to study each pair. Furthermore, close evaluation of behavioral data from the control group in this experiment revealed that there was considerable variability in performance with some participants performing at ceiling and others at or near chance. Both extremely good and extremely poor participants may have obscured the group-level findings, as there would likely be less of a memory-based difference between relatively high viewing trials and relatively low viewing trials following the median split in these groups. For example, memory-based viewing might be *relatively* high for trials categorized as high viewing *or* low viewing among participants performing at ceiling. In order to test this idea, the control group was split into two subgroups based on their memory strength as measured by the strict post-test measure of target-present accuracy: "extreme" performers, the top five and bottom five on this measure of accuracy, and "average" performers, the middle ten.

Parameter estimates from anterior and posterior hippocampal ROIs for high and low viewing trials from a median split based on proportion of viewing the target in the first 1000ms, as above, were entered into a mixed-model ANOVA with factors of Group (average, extreme), Viewing (high, low), and ROI (anterior, posterior). Results from this ANOVA revealed a significant interaction between viewing and group [see Figure 9 in Appendix A; $F(1,18)=12.73$, $p=.002$, $\eta^2=.41$]. To follow-up on this interaction, repeated measures ANOVAs were conducted separately for average and extreme control participants. For the extreme control participants, no significant effects were found [$F(1,9)<3.79$, $p's>.08$, $\eta^2=.30$], but for average control participants, there was significantly greater activity for trials in which there was high, as compared to low, viewing of the associate [$F(1,9)=10.94$, $p=.009$, $\eta^2=.55$]. Therefore, it appears that the lack of an overall viewing effect for control participants is due to participants within this group who had either very strong or very weak memory for the pairs, leaving little room to document a memory-based viewing effect in the hippocampus.

This investigation also demonstrated memory-based hippocampal effects during the three-face display. Here it was found that hippocampal activity during the three-face display was greater for correct compared to incorrect trials for control participants, reflecting successful recognition. This finding is consistent with previous research that has shown hippocampal activity to be reflective of accurate recognition memory for relational information (Yonelinas, Hopfinger, Buonocore, Kroll, & Baynes, 2001; Prince et al., 2005; Hannula et al., 2013). The same pattern was not hypothesized for simulator participants, as their explicit responding was not expected to reflect their memory success. Simulators actually exhibited the opposite pattern from controls with reliably greater hippocampal activity for incorrect as compared to correct trials. This seems to indicate that simulator participants were more likely to select an incorrect response

when their memory for the pair was strongest. However, when test data was back-sorted as a function of post-test accuracy (using the strict criterion for target-present trials, presence/absence judgment for target-absent trials), test accuracy for trials subsequently answered correctly (indicating strong memory for the pair) was comparable to test accuracy for trials subsequently answered incorrectly [$t(19)=1.59$, $p=.13$, Cohen's $d=.39$], although this difference was in the expected direction numerically, with slightly worse test performance for trials that were subsequently answered correctly on the post-test. Still, this proposal is consistent with the strategy reported on the post-test questionnaire by half of the simulator participants to intentionally answer all or most of the trials incorrectly, and for this subset of simulator participants, test accuracy for trials subsequently answered correctly on the post-test (i.e. for which memory was strong) was significantly worse than for trials that were subsequently answered incorrectly [$t(9)=2.70$, $p=.02$, Cohen's $d=1.06$]. Surprisingly, these participants showed comparable hippocampal accuracy effects (incorrect>correct) as the other ten, who did not report employing this strategy and who showed no difference in their test accuracy as a function of strength of memory, as assessed by post-test accuracy, for both the anterior [$t(18)=.21$, $p=.84$, Cohen's $d=.09$] and posterior [$t(18)=.80$, $p=.43$, Cohen's $d=.36$] ROIs. Based on these findings, it is still unclear why the simulator participants showed a pattern of accuracy-related hippocampal activity that is opposite of that of controls. Future work examining the neural basis of memory malingering or concealment should investigate whether this effect is consistent, and if so, what the basis of it might be.

How hippocampal activity during the three-face display at test predicted accuracy on the post-test was also examined. As with test accuracy, anterior hippocampal activity was greater for trials that were subsequently answered correctly compared to incorrectly among control

participants, again reflecting of strength of memory for the pairs. It was surprising therefore that no reliable difference in activity during test was seen between correct and incorrect post-test trials for the simulator group, who were also giving optimal performance on the post-test and whose post-test performance was matched to controls. However, for this contrast I was still examining activity during the test. The simulator participants were engaging in more than just memory-related processes at this time. It is possible that the additional processing required to comply with the goal of feigned memory impairment influenced hippocampal activity in a way that eliminated differences in activity based on memory strength for simulators. Instead of evaluating hippocampal activity during the test, where there are between-groups differences in the engagement of strategic processes, it may be more fruitful to examine how activity during the encoding phase predicts post-test memory accuracy. This is a possibility that will be evaluated in future analyses.

As eye movement analyses were conducted in the current investigation, and eye movements typically provide information about allocation of attention, the findings here can also inform how attentional processes were affected by the instruction to feign memory impairment. The results from this investigation first contribute to a line of evidence that suggests that information retrieved from long-term memory is prioritized by attention (Hannula et al., 2007; Hannula & Ranganath, 2009), as seen in the rapid eye-movements made to the associate of the scene cue after the three-face display was presented. This prioritization occurred for both groups, and even when simulator participants incorrectly stated that the associate was not present. However, as mentioned above, whether this prioritization is consistent with the goal of feigned memory impairment (i.e. identification of the associate in order to feign successfully) or a disruption of goal-directed behavior cannot be fully disentangled based on the current

experiment. Future work is needed that more definitively pits this early attentional prioritization against the goals of the individual. One way to do this would be to inform individuals of the possibility of memory detection through early eye movement effects and see if simulator participants were able to inhibit the effect.

While simulators and controls showed comparable early disproportionate viewing of targets over matched comparison faces, viewing of targets across the whole trial was reduced for simulators. This suggests that these participants were more likely that controls to reorient their attention away from the associate after the early eye-movement-based memory effects. Whole-brain analyses showed greater activity in the right angular gyrus when three-face display were presented on target-present trials for simulators than for controls. Similarly to the right TPJ discussed earlier, this region has been implicated in the automatic reorienting of attention towards behaviorally relevant objects in the environment (Corbetta et al., 2000; Corbetta & Shulman, 2002). In these fMRI studies, the angular gyrus was activated by unattended, task-relevant objects even if they had low perceptual salience (e.g. Indovina & Macaluso, 2007). This is an interesting finding in the context of the current experiment as shifting attention away from the associate is behaviorally relevant to simulate memory impairment, which may have encouraged reorienting to distractors (i.e. the other faces in the display). This effect is different from the standard effect in the attention literature where this region is usually associated with reorientation *to* a behaviorally relevant stimulus, rather than *away* from a target due to the current behavioral objective held by simulator participants. The lack of significant differences found in regions that have been shown to relate to voluntary reorientation of attention in a goal-directed manner (i.e. IPS and SPL) was unexpected given that the reorientation of attention away from associates would seem to be in pursuit of the goal of feigning memory impairment. This

may indicate that the reorientation of attention, as indexed here in eye movement behavior, is a reflexive act rather than a conscious, deliberate attempt to conceal memory. Recruitment of these voluntary reorientation regions might be greater with slight modifications to the instructional manipulation for simulators. For example, if simulators were informed that eye movements could reveal their knowledge and were instructed to redirect viewing away from associates, greater activity in these dorsal regions might be expected among simulators compared to controls.

In addition to the investigation memory and attention processes described above, this investigation also examined activity differences that are evident in the face of conflict and increased need for cognitive control. First, a between-groups analysis examined whether hippocampal suppression occurred in simulator participants as they attempted to feign memory impairment, presumably by cognitive control regions as has been seen in studies of directed suppression (Anderson et al., 2004; Depue et al., 2007). In these investigations of retrieval suppression, memory is poorer for associations whose retrieval was suppressed during an earlier test phase, compared both to information that was intentionally retrieved in the first test phase as well as information that was not seen between encoding and the second test phase. This difference in performance is associated with reductions in hippocampal activity during the first test, where participants are intentionally suppressing retrieval. As such, it seems that hippocampal suppression may lead to weakening of the memory trace that makes it more difficult to successfully retrieve the association at a later time. Here, however, there were no differences in group performance on the post-test and hippocampal activity during the three-face display of the test phase was found to be greater for simulators than controls. As simulators were informed of the post-test before the experiment and not directly instructed to suppress retrieval at any point during the test phase, it is not altogether surprising that no suppression-related effects

were found. This is not to say that suppression cannot be employed during feigned memory impairment, simply that this does not seem to be a process that occurred with this group of simulator participants.

The ACC was specifically investigated due to its proposed role as a conflict monitoring region (Botvinick et al., 2001) and the idea that feigning memory impairment would lead to conflict between accurate memory retrieval and this goal. This conflict was expected to be particularly great when simulator participants responded incorrectly. On such trials, simulator participants were responding in a manner which directly contrasted with their knowledge of the associate. On correct trials, there may be some conflict between accurate memory retrieval and the goal of feigned memory impairment that simulators are maintaining, but their explicit responding is congruent with their knowledge of the scene-face pair. In line with this prediction, the simulators showed greater ACC activity on trials where they responded incorrectly as compared to correctly. The same effect was not seen in control participants, therefore supporting the notion that the conflict between accurate memory, the goal of feigned memory impairment, and intentional incorrect responding leads to the recruitment of this region. This finding is also consistent with prior block-design fMRI investigations of memory malingering which identified the ACC as more active when individuals feigned memory impairment compared to when they performed optimally (Lee et al., 2002; Lee et al., 2005). The results here expand upon this finding by demonstrating greater activation of this region during malingering when incorrect responses were made.

The current investigation is not without limitations. The naturalistic instruction of memory malingering that was provided to simulator participants may be seen as a strength, as it increases the ecological validity of the investigation. However, the open-ended instruction also

allowed each participant to adopt whatever strategy they thought best in order to accomplish the task. This is seen especially in responses on the post-test questionnaire, where among the 20 participants all but one option (blurring one's vision) were selected at least once, and no option was selected by more than half of the participants. As each simulator was performing the task in an individual manner, presumably engaging different cognitive control processes and therefore different neural substrates, it is perhaps not surprising that group differences in cognitive control regions implicated in earlier fMRI investigations of memory malingering were not found here at the group level. Future investigations into memory retrieval under instruction of feigned memory impairment should attempt to explore specific strategies for accomplishing such tasks, and what neural resources are recruited in each case. For example, strategies that employ suppression of retrieval might look very different than those that use calculated monitoring to perform at chance-levels, which could look different from those than involve pre-selecting responses before the display is shown. However, despite these potential differences that may have occurred in the current group of simulators, these participants collectively displayed greater ACC activity for incorrect than correct trials, which may represent the conflict that necessarily exists for these memory decisions made under feigned memory instruction, regardless of the strategy an individual chooses to employ.

In addition to examining the roles of memory, attention, and cognitive control resources individually while participants completed the simulated memory impairment task, the current investigation conducted a number of exploratory connectivity analyses. These aimed to identify how connections between these different regions differed between trials in which simulators responded correctly or incorrectly. Through these connectivity analyses, I was not able to determine any reliable regions which showed differential connectivity with the hippocampus or

ACC between correct and incorrect trials for simulators. However, this does not mean that these regions are working in isolation as individuals attempt to feign memory impairment. The PPI analysis necessitates that for a region to be identified, coactivation between that region and the seed must occur for one condition of the experimental contrast and not the other (O'Reilly, Woolrich, Behrens, Smith, & Johansen-Berg, 2012). Therefore, if activity in a region was correlated with the hippocampus or the ACC across all trials, correct and incorrect, we would not expect to find a significant interaction. Future analyses will examine this possibility.

As described above, the current work has theoretical implications for our understanding the automaticity of hippocampal retrieval and how attentional prioritization of learned materials can occur, despite the goals of an individual. This investigation also has practical implications. The act of memory malingering is thought to be quite prevalent (Binder & Willis, 1991; Mittenberg et al., 2002; Slick et al., 2004) and therefore costly to society. The detection of individuals who are engaging in such behavior is therefore of great interest. The most commonly used way of detecting malingering in clinical settings is through the use of performance validity measures (Bush, 2012). These neuropsychological measures are tests of memory that are simple enough that even those with significant deficits can do fairly well. Therefore, if an individual does not succeed on these tasks it can be inferred that they are not giving optimal effort. However, just by being aware of the presence of these types of measures, individuals have been shown to become more sophisticated at malingering and can evade detection. Therefore, other methods for the detection of this type of deception have been investigated, including the use of fMRI, as was seen in the studies described previously (Lee et al., 2002; Lee et al., 2005; Browndyke et al., 2008). The current investigation expanded upon this work by collecting explicit behavioral responses, viewing patterns, and neural activity, as well as by examining

processes related to memory retrieval, attentional allocation, and cognitive control rather than just one process in isolation. Based on the findings here, there are several results that could represent good targets for detection of feigned memory impairment that should be investigated in future work.

First, has been shown in a previous study (Mahoney et al., in prep), eye movements might be useful in detecting those feigning impairment. As discussed earlier, patients with true relational memory deficits show no early disproportionate viewing of associates in this type of task. The early memory-based effects therefore could be used to distinguish between those trying their best, but failing due to real impairment, and those who are feigning such impairment. Furthermore, the current investigation has shown that the relationship between activity in the hippocampus and explicit accuracy may help differentiate between those feigning impairment and those trying their best, as these two groups showed opposite patterns of activation. Greater hippocampal activity during incorrect trials, therefore, may be a sign of feigned memory impairment. Finally, the recruitment of regions associated with reorientation of attention and cognitive control, such as the right parietal cortex and ACC, might be indicative of feigned memory impairment. The ACC might be a particularly good candidate, as neither individuals putting forth optimal effort and succeeding or those with real memory deficits should be experiencing the same level of conflict as those feigning impairment. However, this region has been activated in a number of studies, reflecting a number of processes besides feigned memory impairment. Therefore, additional work is needed to identify if this effect is specific enough. In all likelihood, given the complexity of the behavior of feigned memory impairment, detection will require the combination of multiple effects. Future work should take this into account and, as was done here, examine multiple processes through multiple methods. As mentioned above,

work is also needed to investigate how specific strategies to accomplish the goal impact these different effects.

In sum, the current work has provided an expansive investigation into the behavioral and neural correlates of memory decisions made in the face of conflict, specifically while individuals attempt to feign memory impairment. Despite successful feigned memory impairment through explicit responding, simulators showed comparable attentional prioritization of learned materials as those performing optimally. This effect was predicted by hippocampal activity during a memory cue for simulators, while attentional reorientation away from learned materials may have been supported by the right angular gyrus. I have also demonstrated that the recruitment ACC occurs during feigned impairment, particularly when incorrect responses are made, suggesting an increased role for cognitive control. These findings provide evidence for the contributions of memory, attention, and cognitive control while individuals engage in the complex task of feigned memory impairment. Results here provide evidence for new theoretical understanding of how memory, attention, and cognitive control can interact, as well as practical relevance for the detection of memory malingering.

References

Abe, N., Okuda, J., Suzuki, M., Sasaki, H., Matsuda, T., Mori, E., ... & Fujii, T. (2008). Neural correlates of true memory, false memory, and deception. *Cerebral Cortex*, *18*(12), 2811-2819.

Anderson, M. C., & Levy, B. J. (2009). Suppressing unwanted memories. *Current Directions in Psychological Science*, *18*(4), 189-194.

Anderson, M. C., Ochsner, K. N., Kuhl, B., Cooper, J., Robertson, E., Gabrieli, S. W., ... & Gabrieli, J. D. (2004). Neural systems underlying the suppression of unwanted memories. *Science*, *303*(5655), 232-235.

Arana, F. S., Parkinson, J. A., Hinton, E., Holland, A. J., Owen, A. M., & Roberts, A. C. (2003). Dissociable contributions of the human amygdala and orbitofrontal cortex to incentive motivation and goal selection. *The Journal of Neuroscience*, *23*(29), 9632-9638.

Bakker, A., Kirwan, C. B., Miller, M., & Stark, C. E. (2008). Pattern separation in the human hippocampal CA3 and dentate gyrus. *Science*, *319*(5870), 1640-1642.

Binder, L. M., & Willis, S. C. (1991). Assessment of motivation after financially compensable minor head trauma. *Journal of Consulting and Clinical Psychology*, *3*(2), 175-181.

Booth, J. R., Burman, D. D., Meyer, J. R., Lei, Z., Trommer, B. L., Davenport, N. D., ... & Mesulam, M. M. (2003). Neural development of selective attention and response inhibition. *Neuroimage*, *20*(2), 737-751.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, *108*(3), 624-652.

Browndyke, J. N., Paskavitz, J., Sweet, L. H., Cohen, R. A., Tucker, K. A., Welsh-Bohmer, K. A., ... & Schmechel, D. E. (2008). Neuroanatomical correlates of malingered memory

impairment: event-related fMRI of deception on a recognition memory task. *Brain Injury*, *22*(6), 481-489.

Buckley, M. J., & Gaffan, D. (2006). Perirhinal cortical contributions to object perception. *Trends in Cognitive Sciences*, *10*(3), 100-107.

Bush, S. S. (2012). *Mild traumatic brain injury: Symptom validity assessment and malingering*. D. A. Carone (Ed.). Springer Publishing Company.

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*, *280*(5364), 747-749.

Chikazoe, J., Konishi, S., Asari, T., Jimura, K., & Miyashita, Y. (2007). Activation of right inferior frontal gyrus during response inhibition across response modalities. J*ournal of Cognitive Neuroscience*, *19*(1), 69-80.

Cohen, N. J., Eichenbaum, H., & Memory, A. (1993). Memory, amnesia, and the hippocampal system. Cambridge, MA: MIT Press.

Cohen, N. J., Ryan, J., Hunt, C., Romine, L., Wszalek, T., & Nash, C. (1999). Hippocampal system and declarative (relational) memory: summarizing the data from functional neuroimaging studies. *Hippocampus*, *9*(1), 83-98.

Corbetta, M., Kincade, J. M., Ollinger, J. M., McAvoy, M. P., & Shulman, G. L. (2000). Voluntary orienting is dissociated from target detection in human posterior parietal cortex. *Nature Neuroscience*, *3*(3), 292-297.

Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience*, *3*(3), 201-215.

Crane, J., & Milner, B. (2005). What went where? Impaired object-location learning in patients with right hippocampal lesions. *Hippocampus*, *15*(2), 216-231.

Davachi, L. (2006). Item, context and relational episodic encoding in humans. *Current Opinion in Neurobiology*, *16*(6), 693-700.

Depue, B. E., Curran, T., & Banich, M. T. (2007). Prefrontal regions orchestrate suppression of emotional memories via a two-phase process. *Science*, *317*(5835), 215-219.

D'Esposito, M. (2007). From cognitive to neural models of working memory. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, *362*(1481), 761-772.

D'Esposito, M., Detre, J. A., Alsop, D. C., Shin, R. K., Atlas, S., & Grossman, M. (1995). The neural basis of the central executive system of working memory. *Nature*, *378*(6554), 279-281.

Diana, R. A., Yonelinas, A. P., & Ranganath, C. (2007). Imaging recollection and familiarity in the medial temporal lobe: a three-component model. *Trends in Cognitive Sciences*, *11*(9), 379-386.

Eichenbaum, H., & Cohen, N. J. (2014). Can we reconcile the declarative memory and spatial navigation views on hippocampal function?. *Neuron*, *83*(4), 764-770.

Eichenbaum, H., & Lipton, P. A. (2008). Towards a functional organization of the medial temporal lobe memory system: role of the parahippocampal and medial entorhinal cortical areas. *Hippocampus*, *18*(12), 1314-1324.

Eichenbaum, H., Otto, T., & Cohen, N. J. (1992). The hippocampus—what does it do?. *Behavioral and Neural Biology*, *57*(1), 2-36.

Eichenbaum, H., Yonelinas, A. R., & Ranganath, C. (2007). The medial temporal lobe and recognition memory. *Annual Review of Neuroscience*, *30*, 123-152.

Frankó, E., Insausti, A. M., Artacho-Pérula, E., Insausti, R., & Chavoix, C. (2014). Identification of the human medial temporal lobe regions on magnetic resonance images. *Human Brain Mapping*, *35*(1), 248-256.

Goodale, M. A., & Milner, A. D. (1992). Separate visual pathways for perception and action. *Trends in Neurosciences*, *15*(1), 20-25.

Graf, P., & Schacter, D. L. (1985). Implicit and explicit memory for new associations in normal and amnesic subjects. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *11*(3), 501-518.

Hannula, D. E., Althoff, R. R., Warren, D. E., Riggs, L., Cohen, N. J., & Ryan, J. D. (2010). Worth a glance: using eye movements to investigate the cognitive neuroscience of memory. *Frontiers in Human Neuroscience*, *4*(166), 1-16.

Hannula, D. E., Libby, L. A., Yonelinas, A. P., & Ranganath, C. (2013). Medial temporal lobe contributions to cued retrieval of items and contexts. *Neuropsychologia*, *51*(12), 2322-2332.

Hannula, D. E., & Ranganath, C. (2008). Medial temporal lobe activity predicts successful relational memory binding. *The Journal of Neuroscience*, *28*(1), 116-124.

Hannula, D. E., & Ranganath, C. (2009). The eyes have it: hippocampal activity predicts expression of memory in eye movements. *Neuron*, *63*(5), 592-599.

Hannula, D. E., Ryan, J. D., Tranel, D., & Cohen, N. J. (2007). Rapid onset relational memory effects are evident in eye movement behavior, but not in hippocampal amnesia. *Journal of Cognitive Neuroscience, 19*(10), 1690-1705.

Hannula, D. E., Tranel, D., & Cohen, N. J. (2006). The long and the short of it: relational memory impairments in amnesia, even at short lags. *The Journal of Neuroscience*, *26*(32), 8352-8359.

Hoffman, J. E., & Subramaniam, B. (1995). The role of visual attention in saccadic eye movements. *Perception & Psychophysics*, *57*(6), 787-795.

Holdstock, J. S., Mayes, A. R., Gong, Q. Y., Roberts, N., & Kapur, N. (2005). Item recognition is less impaired than recall and associative recognition in a patient with selective hippocampal damage. *Hippocampus*, *15*(2), 203-215.

Indovina, I., & Macaluso, E. (2007). Dissociation of stimulus relevance and saliency factors during shifts of visuospatial attention. *Cerebral Cortex*, *17*(7), 1701-1711.

Insausti, R., Amaral, D. G., & Cowan, W. M. (1987). The entorhinal cortex of the monkey: II. Cortical afferents. *Journal of Comparative Neurology*, *264*(3), 356-395.

Insausti, R., Juottonen, K., Soininen, H., Insausti, A. M., Partanen, K., Vainio, P., ... & Pitkänen, A. (1998). MR volumetric analysis of the human entorhinal, perirhinal, and temporopolar cortices. *American Journal of Neuroradiology*, *19*(4), 659-671.

Kerns, J. G., Cohen, J. D., MacDonald, A. W., Cho, R. Y., Stenger, V. A., & Carter, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science*, *303*(5660), 1023-1026.

Koechlin, E., Ody, C., & Kouneiher, F. (2003). The architecture of cognitive control in the human prefrontal cortex. *Science*, *302*(5648), 1181-1185.

Konkel, A., Warren, D. E., Duff, M. C., Tranel, D., & Cohen, N. J. (2008). Hippocampal amnesia impairs all manner of relational memory. *Frontiers in Human Neuroscience*, *2*,(15), 1-15.

Kouneiher, F., Charron, S., & Koechlin, E. (2009). Motivation and cognitive control in the human prefrontal cortex. *Nature Neuroscience*, *12*(7), 939-945.

Lee, T. M., Liu, H. L., Chan, C. C., Ng, Y. B., Fox, P. T., & Gao, J. H. (2005). Neural correlates of feigned memory impairment. *Neuroimage*, *28*(2), 305-313.

Lee, T., Liu, H. L., Tan, L. H., Chan, C. C., Mahankali, S., Feng, C. M., ... & Gao, J. H. (2002). Lie detection by functional magnetic resonance imaging. *Human Brain Mapping*, *15*(3), 157-164.

Mahoney, E.J., Osmon, D.C., Kapur, N., & Hannula, D.E. (in prep). Relational memories are expressed obligatorily in eye movement behavior: Eye movements unmask feigned memory impairment.

Marr, D., Willshaw, D., & McNaughton, B. (1991). Simple memory: a theory for archicortex. In *From the Retina to the Neocortex* (pp. 59-128). Birkhäuser Boston.

Merkl, A., Schneider, G. H., Schönecker, T., Aust, S., Kühl, K. P., Kupsch, A., ... & Bajbouj, M. (2013). Antidepressant effects after short-term and chronic stimulation of the subgenual cingulate gyrus in treatment-resistant depression. *Experimental Neurology*, *249*, 160-168.

Miller, E. K. (2000). The prefontral cortex and cognitive control. *Nature Reviews Neuroscience*, *1*(1), 59-65.

Mittenberg, W., Patton, C., Canyock, E. M., & Condit, D. C. (2002). Base rates of malingering and symptom exeggeration. *Journal of Clinical and Experimental Neuropsychology*, *24*(8), 1094-1102.

Mizumori, S. J., McNaughton, B. L., Barnes, C. A., & Fox, K. B. (1989). Preserved spatial coding in hippocampal CA1 pyramidal cells during reversible suppression of CA3c

output: evidence for pattern completion in hippocampus. *Journal of Neuroscience*, *9*(11), 3915-3928.

Montaldi, D., & Mayes, A. R. (2010). The role of recollection and familiarity in the functional differentiation of the medial temporal lobes. *Hippocampus*, *20*(11), 1291-1314.

Moscovitch, M. (2008). The hippocampus as a" stupid," domain-specific module: Implications for theories of recent and remote memory, and of imagination. *Canadian Journal of Experimental Psychology*, *62*(1), 62-79.

Mostofsky, S. H., Schafer, J. G., Abrams, M. T., Goldberg, M. C., Flower, A. A., Boyce, A., ... & Pekar, J. J. (2003). fMRI evidence that the neural basis of response inhibition is task-dependent. *Cognitive Brain Research*, *17*(2), 419-430.

Murray, E. A., & Richmond, B. J. (2001). Role of perirhinal cortex in object perception, memory, and associations. *Current Opinion in Neurobiology*, *11*(2), 188-193.

O'Reilly, J. X., Woolrich, M. W., Behrens, T. E., Smith, S. M., & Johansen-Berg, H. (2012). Tools of the trade: psychophysiological interactions and functional connectivity. *Social Cognitive and Affective Neuroscience*, *7*(5), 604-609.

Paxton, J. L., Barch, D. M., Racine, C. A., & Braver, T. S. (2008). Cognitive control, goal maintenance, and prefrontal function in healthy aging. *Cerebral Cortex*, *18*(5), 1010-1028.

Phan, K. L., Magalhaes, A., Ziemlewicz, T. J., Fitzgerald, D. A., Green, C., & Smith, W. (2005). Neural correlates of telling lies: A functional magnetic resonance imaging study at 4 Tesla1. *Academic Radiology*, *12*(2), 164-172.

Posner, M. I. (1980). Orienting of attention. *Quarterly Journal of Experimental Psychology*, *32*(1), 3-25.

Prince, S. E., Daselaar, S. M., & Cabeza, R. (2005). Neural correlates of relational memory: successful encoding and retrieval of semantic and perceptual associations. *Journal of Neuroscience*, *25*(5), 1203-1210.

Ridderinkhof, K. R., Ullsperger, M., Crone, E. A., & Nieuwenhuis, S. (2004). The role of the medial frontal cortex in cognitive control. *Science*, *306*(5695), 443-447.

Ridderinkhof, K. R., Van Den Wildenberg, W. P., Segalowitz, S. J., & Carter, C. S. (2004). Neurocognitive mechanisms of cognitive control: the role of prefrontal cortex in action selection, response inhibition, performance monitoring, and reward-based learning. *Brain and Cognition*, *56*(2), 129-140.

Rolls, E. (2013). The mechanisms for pattern completion and pattern separation in the hippocampus. *Frontiers in Systems Neuroscience*, *7*, 74.

Serences, J. T., Shomstein, S., Leber, A. B., Golay, X., Egeth, H. E., & Yantis, S. (2005). Coordination of voluntary and stimulus-driven attentional control in human cortex. *Psychological Science*, *16*(2), 114-122.

Serences, J. T., & Yantis, S. (2007). Spatially selective representations of voluntary and stimulus-driven attentional priority in human occipital, parietal, and frontal cortex. *Cerebral Cortex*, *17*(2), 284-293.

Shenhav, A., Botvinick, M. M., & Cohen, J. D. (2013). The expected value of control: an integrative theory of anterior cingulate cortex function. *Neuron*, *79*(2), 217-240.

Shulman, G. L., McAvoy, M. P., Cowan, M. C., Astafiev, S. V., Tansy, A. P., d'Avossa, G., & Corbetta, M. (2003). Quantitative analysis of attention and detection signals during visual search. *Journal of Neurophysiology*, *90*(5), 3384-3397.

Slick, D. J., Tan, J. E., Strauss, E. H., & Hultsch, D. F. (2004). Detecting malingering: A survey of experts' practices. *Archives of Clinical Neuropsychology*, *19*(4), 465-473.

Squire, L. R., Stark, C. E., & Clark, R. E. (2004). The medial temporal lobe. *Annual Review Neuroscience*, *27*, 279-306.

Squire, L. R., & Zola, S. M. (1997). Amnesia, memory and brain systems. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, *352*(1362), 1663-1673.

Stark, C. E., & Okado, Y. (2003). Making memories without trying: medial temporal lobe activity associated with incidental memory formation during recognition. *Journal of Neuroscience*, *23*(17), 6748-6753.

Stark, C. E., & Squire, L. R. (2001). When zero is not zero: the problem of ambiguous baseline conditions in fMRI. *Proceedings of the National Academy of Sciences*, *98*(22), 12760-12766.

Suhr, J. A., & Boyer, D. (1999). Use of the Wisconsin Card Sorting Test in the detection of malingering in student simulator and patient samples. *Journal of Clinical and Experimental Neuropsychology*, *21*(5), 701-708.

Sullivan Giovanello, K., Schnyer, D. M., & Verfaellie, M. (2004). A critical role for the anterior hippocampus in relational memory: evidence from an fMRI study comparing associative and item recognition. *Hippocampus*, *14*(1), 5-8.

Suzuki, W. L., & Amaral, D. G. (1994). Perirhinal and parahippocampal cortices of the macaque monkey: cortical afferents. *Journal of Comparative Neurology*, *350*(4), 497-533.

Todd, J. T., & Van Gelder, P. (1979). Implications of a transient–sustained dichotomy for the measurement of human performance. *Journal of Experimental Psychology: Human Perception and Performance*, *5*(4), 625-638.

Ullsperger, M., & Von Cramon, D. Y. (2004). Neuroimaging of performance monitoring: error detection and beyond. *Cortex*, *40*(4), 593-604.

Uncapher, M. R., Boyd-Meredith, J. T., Chow, T. E., Rissman, J., & Wagner, A. D. (2015). Goal-directed modulation of neural memory patterns: Implications for fMRI-based memory detection. *Journal of Neuroscience*, *35*(22), 8531-8545.

Vagnini, V. L., Berry, D. T., Clark, J. A., & Jiang, Y. (2008). New measures to detect malingered neurocognitive deficit: Applying reaction time and event-related potentials. *Journal of Clinical and Experimental Neuropsychology*, *30*(7), 766-776.

Valentin, V. V., Dickinson, A., & O'Doherty, J. P. (2007). Determining the neural substrates of goal-directed learning in the human brain. *The Journal of Neuroscience*, *27*(15), 4019-4026.

van Hooff, J. C., Sargeant, E., Foster, J. K., & Schmand, B. A. (2009). Identifying deliberate attempts to fake memory impairment through the combined use of reaction time and event-related potential measures. *International Journal of Psychophysiology*, *73*(3), 246-256.

Vossel, S., Geng, J. J., & Fink, G. R. (2014). Dorsal and ventral attention systems distinct neural circuits but collaborative roles. *The Neuroscientist*, *20*(2), 150-159.

Wang, S. F., Ritchey, M., Libby, L. A., & Ranganath, C. (2016). Functional connectivity based parcellation of the human medial temporal lobe. *Neurobiology of Learning and Memory*, *134*, 123-134.

Williams, L. E., Must, A., Avery, S., Woolard, A., Woodward, N. D., Cohen, N. J., & Heckers, S. (2010). Eye-movement behavior reveals relational memory impairment in schizophrenia. *Biological Psychiatry*, *68*(7), 617-624.

Yonelinas, A. P., Hopfinger, J. B., Buonocore, M. H., Kroll, N. E. A., & Baynes, K. (2001). Hippocampal, parahippocampal and occipital-temporal contributions to associative and item recognition memory: an fMRI study. *Neuroreport*, *12*(2), 359-363.

Appendix A

Group Level Instructions

Control Instructions:

For this experiment I would like for you to imagine that you've been in a car accident and that another driver was responsible for the collision. You were unconscious following the accident and woke up in the hospital where you were kept overnight for observation. You were told that you had suffered from a severe concussion. Now, try to imagine that it's one year later, and that you're attempting to return to work. To do so you must convince an examiner that the accident has not caused any long-term cognitive deficits. As part of this process, you will take a memory test and it is important that you perform your best. When we begin, you will be asked to learn several scene-face pairs. Each pair will be presented twice and you should use the second study exposure to make sure that the pair is well-learned. Following this study phase, a recognition memory test will be administered – at this point your memory for the preceding pairs will be evaluated. Over the course of the experiment, this study-test process will be repeated several times with new materials. In each case, you should do your best to learn the pairs and to recognize them later. Finally, at the end of the experiment, a post-test will be administered and your memory for all of the pairs will be tested again. You will not have an opportunity to restudy the scene-face pairs before the post-test, so it is important that you attempt to learn the pairs as well as possible during the initial study exposures. As I have already emphasized, it is important that you try your best to remember the pairs that were studied so that you can convince the examiner that the accident did not cause any memory impairment.

Malingering Instructions:

For this experiment I would like for you to imagine that you've been in a car accident and that another driver was responsible for the collision. You were unconscious following the accident and woke up in the hospital where you were kept overnight for observation. You were told that you had suffered from a severe concussion. Now, try to imagine that it's one year later, and that you're involved in a lawsuit against the other driver. If it's determined that you have experienced significant injuries as a result of the accident, you're likely to receive a bigger settlement. To improve the liklihood of financial gain, you have decided to pretend that you are suffering from memory impairment as a result of the accident.

It is required by the court that you take a test, which will be used to determine whether you do indeed have a memory problem. You will take this test today, and must convince the examiner that this is the case. When we begin, you will be asked to learn several scene-face pairs. Each pair will be presented twice and you should use the second study exposure to make sure that the pair is well-learned. Following this study phase, a recognition memory test will be administered and your memory for the preceding pairs will be evaluated. At this point, if you can successfully convince the examiner that you have a memory deficit, then you are likely to be awarded a more substantial settlement. However, it is important that your performance does not tip the examiner off – you must convince the examiner that your memory impairment is real, and ensure that it's not obvious that you're faking. Some strategies would be too obvious and would alert the examiner. For instance, if you simply answer every test question incorrectly or fail to respond on a subset of the trials, this would tip the examiner off. Over the course of the experiment, this study-test process will be repeated several times with new materials.

Finally, at the end of the experiment, a post-test will be administered and your memory for all of the pairs will be tested again. At this point, we want you to try to do your best to recognize the pairs that were presented during the earlier part of the experiment when you were faking memory impairment. The examiner will alert you prior to administration of the post-test so that you can change strategies and try to perform your best. Until you have been told that you should change strategies, you should attempt to convince the examiner that you do have a memory impairment. You will not have an opportunity to restudy the scene-face pairs before the post-test, so it is important that you attempt to learn the pairs as well as possible during the initial study exposures – this will ensure the best possible performance on the post-test.

This experiment has several different parts and that can be hard to keep track of. Do you have any questions about what you are expected to do?

Appendix B

Control Viewing Effects by Memory Strength

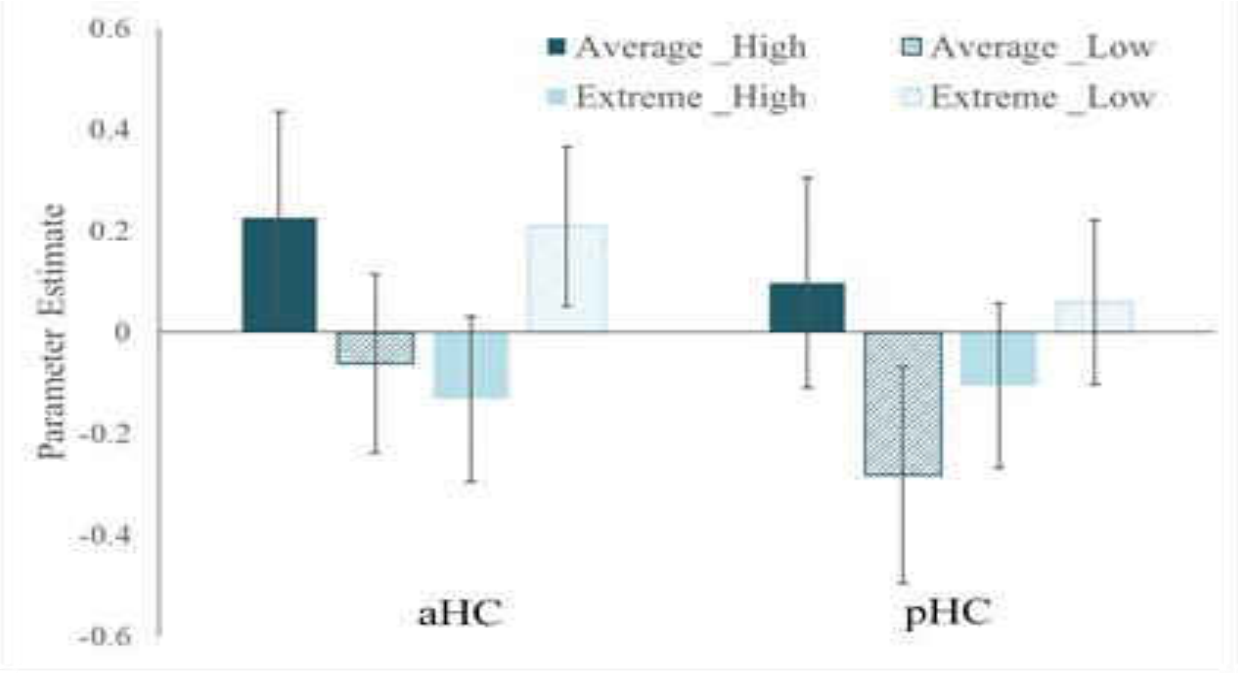

Figure 9. Controls Viewing Effects Split by Memory Strength. Parameter estimates extracted from anterior and posterior hippocampal ROIs during the scene cue for high and low viewing trials. Control participants are divided into "extreme" participants (i.e. those who performed in the top or bottom 5 on the post-test) and "average" participants (i.e. the middle 10 control participants on the post-test).

Elaine Mahoney, M.S.

Curriculum Vitae

EDUCATION

James A. Haley Veterans Hospital, Tampa, FL 2017-Present
Clinical Psychology Predoctoral Internship (APA-accredited)
Neuropsychology Track, Anticipated Completion July 2018

Anticipated Ph.D., University of Wisconsin – Milwaukee, Milwaukee, WI 2015-Present
Clinical Psychology (APA-accredited), Anticipated August 2018, GPA: 4.0
Minor in Quantitative Methods
Dissertation title: *Neural correlates of memory decisions made in the face of conflict.*
Defended: May 2017
Advisor: Deborah Hannula, Ph.D.

M.S., University of Wisconsin – Milwaukee, Milwaukee, WI 2012-2014
Clinical Psychology, Awarded December 2014, GPA: 4.0
Master's thesis: *Eye-movement-based detection of relational memory despite attempts to simulate memory impairment.*
Advisor: Deborah Hannula, Ph.D.

B.A., Rice University, Houston, TX 2008-2012
Psychology/Cognitive Sciences, Awarded May 2012
Graduated Summa cum Laude with Honors in Psychology (GPA: 4.11)
Honors thesis: *Influencing how people remember faces: A training study.*
Advisor: Jessica Logan, Ph.D.

FELLOWSHIPS AND AWARDS

Association of Graduate Students in Neuropsychology Travel Award 2016
University of Wisconsin – Milwaukee, Milwaukee, WI
Distinguished Graduate Student Fellowship 2014-2015
University of Wisconsin – Milwaukee, Milwaukee, WI
Department of Psychology Summer Graduate Research Fellowship 2014
University of Wisconsin – Milwaukee, Milwaukee, WI
Graduate Student Travel Award 2013
University of Wisconsin – Milwaukee, Milwaukee, WI
Chancellor's Graduate Student Award 2012-2014
University of Wisconsin – Milwaukee, Milwaukee, WI
William C. Howell Award for Excellence in Undergraduate Research and Scholarship 2012
Rice University, Houston, TX
Rice Undergraduate Research Symposium Poster Award 2012
Rice University, Houston, TX
Social Sciences Undergraduate Research Enterprise (SSURE) Grant 2011
Rice University, Houston, TX

President's Honor Roll 2008-2012
Rice University, Houston TX

RESEARCH EXPERIENCE

James A. Haley Veterans Hospital, Tampa, FL 08/2017-Present
Research Advisors: Rodney Vanderploeg, Ph.D., ABPP-CN; Rachel Keelan, Ph.D.

- Conduct analyses with a TBI Model Systems database with neuropsychological data and subjective functional ratings to characterize the neuropsychological profile of individuals in a state of post-traumatic amnesia; this research question aims to elucidate whether this period represents a primary deficit in memory or a more global cognitive impairment.

Memory and Brain Lab, University of Wisconsin - Milwaukee 08/2012-Present
Research advisor: Deborah Hannula, Ph.D.

- Used combined eye-tracking/fMRI methodology to address questions about interactions between memory, attention and cognitive control both at a behavioral and neural while individuals simulated malingered amnesia.
- Conducted studies demonstrating that eye movement patterns reveal knowledge of learned associations despite feigned memory impairment.
- Investigated the impact of emotional contexts on memory and attention processes and how these effects relate to individual differences in state and trait anxiety.

Froedtert & The Medical College of Wisconsin (MCW), Milwaukee, WI 06/2015-05/2017
Research advisors: Sara Swanson, Ph.D., ABPP-CN; David Sabsevitz, Ph.D., ABPP-CN; Laura Umfleet, Psy.D.

- Conducted analyses with neuroimaging and neuropsychological data collected from patients with temporal lobe epilepsy pre- and post-anterior temporal lobectomy to address questions about cortical differences, cognitive decline, and quality of life in these patients.
- Participated in the development of a database to aggregate information from patients presenting to the clinic with memory complaints.

TIRR Memorial Hermann Brain Injury Research Center, Houston, TX 09/2011-03/2012
Research advisors: Mark Sherer, Ph.D., ABPP-CN; Angelle Sander, Ph.D.

- Interviewed and conducted neuropsychological assessments with patients after brain injury in order to develop a symptom-based classification system of TBI.

Memory and Cognition Lab, Rice University, Houston, TX 08/2009-05/2012
Research advisor: Jessica Logan, Ph.D.

- Developed an independent project investigating whether holistic processing training can influence how people process and remember faces.
- Assisted graduate students with investigations of memory in younger and older adults.

Research Experience for Undergraduates in Cognitive and Behavioral Sciences, University of Minnesota, Minneapolis, MN 06/2011-08/2011
Research advisor: Albert Yonas, Ph.D.

- Initiated a project investigating the eye gaze patterns of children with developmental prosopagnosia when viewing faces. Tasks included literature review, creation of an eye-tracking test, testing subjects, and analyzing and presenting preliminary results.

Moss Rehabilitation Research Institute (MRRI), Elkins Park, PA 05/2010-08/2010
Research advisors: Tessa Hart, Ph.D.; John Whyte, M.D., Ph.D.

- Assisted in the research institute in projects for the TBI Model System, primarily working on a project investigating differences in outcome after brain injury as a function of the intensity and duration of rehabilitation services.

Language and Memory Lab, Rice University 01/2009-05/2009
Research advisor: Randi Martin, Ph.D.

- Created stimuli and ran young adults in an experiment to study the connection between working memory capacity and comprehension of garden-path sentences.

PUBLISHED REVIEWS AND BOOK CHAPTERS

Mahoney, E.J.*, Nickel, A.E.*, & Hannula, D.E. (2015). Recognition. In James D. Wright (Ed.), *The International Encyclopedia of Social and Behavioral Science* (2nd ed., Vol. 20, pp. 37-43), Oxford: Elsevier.

Mahoney, E. J., & Hannula, D. E. (2014). Fractionation of Memory in Patient Populations: A Memory Systems Perspective. *SIG 2 Perspectives on Neurophysiology and Neurogenic Speech and Language Disorders*, *24*(2), 50-63.

EMPIRICAL RESEARCH PUBLICATIONS (UNDER REVIEW)

Mahoney, E. J., Kapur, N., Osmon, D., & Hannula, D.E. (revise and resubmit). Eye movements are drawn to remembered content despite instructions to simulate memory impairment.

EMPIRICAL RESEARCH PUBLICATIONS (IN PREPARATION)

Keelan, R.E, **Mahoney, E.J.**, Nakase-Richardson, R., & Vanderploeg, R.D. (in preparation). The underlying cognitive profile of posttraumatic amnesia in moderate to severe traumatic brain injury.

Kazakov, D., **Mahoney, E.J.,** Osmon, D.C., Kapur, N., & Hannula, D.E. (in preparation). Eye movements index object recognition despite efforts to simulate memory impairment.

PAPER PRESENTATIONS/INVITED TALKS

Mahoney, E.J., Nickel, A.E., & Hannula, D.E. (2018). Identifying new targets for detecting memory malingering: Insights from a combined fMRI and eye tracking investigation. Paper presentation at the 46th annual meeting of the International Neuropsychological Society, Washington, D.C.

Mahoney, E.J. (2016). "Effects of the presence of neutral associates of negatively-valenced scenes on memory and attention in individuals with high and low self-reported anxiety". 18th Annual Association of Graduate Students in Psychology Research Symposium, Milwaukee, WI.

POSTER PRESENTATIONS

Mahoney, E.J. & Hannula, D.E. (2017). Simulated memory impairment: Neural correlates of memory decisions made in the face of conflict. Poster presentation at the 47th annual meeting of the Society for Neuroscience, Washington D.C.

Korthauer, L.E., Sabsevitz, D.S., **Mahoney, E.J.,** Bauer, P., Quasney, E.E., Umfleet Glass, L., Swanson, S.J., & Binder, J.R. (2017). Regional subcortical volumes are associated with pre-operative memory performance in left anterior temporal lobectomy (ATL) patients. Poster presentation at the 45th annual meeting of the International Neuropsychological Society, New Orleans, LA.

Mahoney, E.J. & Hannula, D.E. (2016). Encoded materials draw attention, even when memories are successfully concealed in explicit recognition responses. Poster presentation at the 57th Annual Meeting of the Psychonomic Society, Boston, MA.

Mahoney, E.J., Sabsevitz, D.S., Glass Umfleet, L., Quasney, E.E., Binder, J.R., Mueller, W.M., & Swanson, S.J. (2016). The effects of objective cognitive decline on quality of life in patients following left anterior temporal lobectomy. Poster presentation at the 14th Annual American Academy of Clinical Neuropsychology Conference, Chicago, IL.

Mahoney, E.J., Fleuchaus, E.J., & Hannula, D.E. (2016). Neutral associates of negatively-valenced scenes are subject to attentional prioritization, but only in self-reported high worriers. Poster presentation at the 28th Annual Association for Psychological Science Convention, Chicago, IL.

Mahoney, E.J., Korthauer, L.E., Quasney, E.E., Sabsevitz, D., Binder, J.R., Glass Umfleet, L., & Swanson, S.J. (2016). The relationship between perceived and objective cognitive change following temporal lobectomy. Poster presentation at the Annual Meeting of the International Neuropsychological Society, Boston, MA.

Mahoney, E.J., Osmon, D.C., Kapur, N., & Hannula, D.E. (2013). Relational memories are expressed obligatorily in eye movement behavior: Eye movements unmask feigned memory impairment. Poster presentation at the Society for Neuroscience Annual Meeting, San Diego, CA.

Mahoney, E.J., Osmon, D.C., Kapur, N., & Hannula, D.E. (2013). Eye-movements reveal relational memory despite instructions to feign amnesia. Poster presentation at the Milwaukee Area Society for Neuroscience Annual Meeting, Milwaukee, WI.

Mahoney, E.J.. (2012). A new view on face recognition training. Poster presentation at the Rice Undergraduate Research Symposium, Houston, TX.

Corrow, S., **Mahoney, E.J.**, Mathison, J., Greiter, E., Platt, M., & Yonas, A. (2011). Screening for Developmental Prosopagnosia (face blindness): developing interventions. Poster presentation for the Entertainment Software and Cognitive Neurotherapeutics Society, San Francisco, CA.

Mahoney, E.J. (2011). Face recognition deficits and atypical gaze patterns in Developmental Prosopagnosia and Autism Spectrum Disorders. Poster presentation at the University of Minnesota's Undergraduate Summer Research Symposium, Minneapolis, MN.

SPECIALIZED TRAINING EXPERIENCES

Fundamentals of Neuropsychology Seminar 07/2017-Present
James A. Haley Veterans Hospital, Tampa, FL

Neuropsychology Post-Doctoral Seminar 09/2017-Present
James A. Haley Veterans Hospital, Tampa, FL

Cognitive Behavioral Therapy for Chronic Pain Training 09/2017
James A. Haley Veterans Hospital, Tampa, FL

Neuropsychology Case Conference 08/2016-05/2017
Clement J. Zablocki Veterans Affairs Medical Center, Milwaukee, WI

Neuropsychology Journal Club 06/2015-09/2016
Medical College of Wisconsin, Milwaukee, WI

Neuropsychology Seminar 06/2015-09/2016
Medical College of Wisconsin, Milwaukee, WI

Neuroimaging Journal Club 08/2014-05/2017
University of Wisconsin-Milwaukee, Milwaukee, WI

Analysis of Functional NeuroImages (AFNI) Bootcamp 09/2014
National Institutes of Health, Bethesda, MD

Introduction to Pattern Classification Analyses with FMRIB Software Library 07/2013
University of Wisconsin - Milwaukee, Milwaukee, WI

LEADERSHIP AND SERVICE

Future Success Program 2015-2017
Graduate Student Volunteer

- Participated in a program designed to encourage high school students from disadvantaged backgrounds to pursue science-based careers, including leading small groups in collection, analysis, and presentation of data from basic psychology experiments.

Association of Graduate Students in Neuropsychology 2014-2017
Association of Neuropsychology Students in Training (ANST) Interest Group, University of Wisconsin-Milwaukee
Vice President

- Wrote and defended grants to build a library of neuropsychology-related texts for graduate students in the department and to provide funding for travel to neuropsychology conferences.

Upward Bound Math and Science Program 2014-2017
Graduate Student Volunteer

- Participated in a program designed to encourage high school students from disadvantaged backgrounds to pursue science-based careers, including leading small groups in collection, analysis, and presentation of data from basic psychology experiments.

TEACHING EXPERIENCE

Department of Psychology, University of Wisconsin-Milwaukee
Teaching Assistantships
Psychological Statistics (PSYC 210) 08/2013-06/2014
Primary Instructors: Pamela Schaefer, Ph.D., Kamran Diba, Ph.D.

- Led weekly laboratory sections of an introductory statistics course for undergraduate psychology students with instruction on basic descriptive and inferential statistics, as well as the use of SPSS in conducting statistical analyses.

Research Methods in Psychology (PSYC 325) 08/2012-06/2013
Primary Instructors: Susan Lima, Ph.D., Marcellus Merritt, Ph.D.

- Led weekly laboratory sections of research methods course for undergraduate psychology students with instruction on APA format, conducting literature reviews, and preparing lab reports based on in-class experiments.

CLINICAL EXPERIENCE

James A. Haley Veterans Hospital, Tampa, FL 07/2017-Present
Psychology Pre-Doctoral Intern

Rotations:

Memory Disorders Clinic/General Outpatient Neuropsychology 10/2017-Present
Supervisors: Jessica Vasallo, Ph.D., ABPP-CN; Eric Spiegel, Ph.D.

- Conduct comprehensive neuropsychological evaluations using a flexible fixed-core battery approach in an outpatient setting with veterans suffering from a variety of neurodegenerative and neurological conditions including Alzheimer's disease, vascular dementia, dementia with Lewy bodies, Parkinson's disease, and multiple sclerosis, often with comorbid medical and psychiatric conditions.
- Conceptualize cases based on medical records review, interview, and results from neuropsychological measures to provide feedback and recommendations to veterans and their families as well as to produce written reports (approximately 3 reports/week).
- Participate in a weekly journal club reviewing literature that is pertinent to neuropsychological syndromes.

Inpatient Traumatic Brain Injury/Rehabilitation Neuropsychology 07/2017-10/2017
Supervisors: Rodney Vanderploeg, Ph.D., ABPP-CN; Tracy Kretzmer, Ph.D.

- Conducted bedside neuropsychological evaluations on an acute, inpatient rehabilitation unit with active duty and veteran service members who suffered brain injuries of varying types (e.g., TBI, CVA, anoxic/hypoxic events) and severities.
- Provided written and verbal feedback about cognitive functioning and recommendations to an interdisciplinary rehabilitation team through written reports and participation in team meetings.
- Provided psychoeducation, feedback about cognitive functioning, and recommendations to patients and their family members.

Primary Care Clinic - Behavioral Health (Anticipated Rotation) 01/2018-04/2018
Supervisors: Katherine Leventhal, Ph.D.; Benjamin Lord, Ph.D., Dawn Johnson, Ph.D.

- Will conduct brief, yet thorough, chart reviews and interviews to develop treatment plans for individuals raising psychological or behavior concerns during visits to their primary care physicians.
- Will select and implement brief, problem-focused interventions including brief CBT, MI, ACT, and PST.
- Will communicate with members of an interdisciplinary team through verbal and written formats.

Polytrauma/TBI Transitional Rehab (PTRP; Anticipated Rotation) 04/2018-07/2018
Supervisor: Jennifer Duchnick, Ph.D., ABPP-RP

- Will provide psychotherapeutic interventions to patients and their families that address cognitive, physical, emotional, and social sequelae following polytrauma with TBI.
- Will work within an interdisciplinary team to provide a holistic approach to recovery following polytrauma with TBI, including providing recommendations to team members in verbal and written formats.

Core Psychotherapy Experience, PTSD Clinical Team 08/2017-Present
Supervisor: Dr. Jessica Gallinati-Wilson

- Provide year-long outpatient psychotherapy, primarily Cognitive Behavioral Therapy for Depression (CBT-D), to veterans who have concurrent diagnoses of depression and PTSD, but whose functional impairment is primarily due to their depressive symptomology.

Clement J. Zablocki Veterans Affairs Medical Center, Milwaukee, WI 07/2016-05/2017
Neuropsychology Department, Mental Health Division
Neuropsychology Practicum Student
Supervisors: Kathleen Patterson, Ph.D., ABPP-CN; Eric Larson, Ph.D., ABPP-CN; Angela Gleason, Ph.D., ABPP-CN; Melissa Lancaster, Ph.D.

Rotations: General Adult Neuropsychology Clinic, Memory Disorders Clinic

- Completed comprehensive neuropsychological assessments with a veteran patient population referred for a number of neurological conditions, including dementia, traumatic brain injuries, attention difficulties, and learning disabilities, often with comorbid psychiatric symptoms.

- Observed and participated in psychodiagnostic intake interviews of patients.
- Aided in the selection of neuropsychological measures based on presenting symptoms.
- Administered, scored, and interpreted neuropsychological measures.
- Conceptualized cases based on information from medical records, interview, and results of neuropsychological testing, and integrated these into written reports (approximately 1 report/week)
- Participated in weekly neuropsychology case conference.

University of Wisconsin-Milwaukee (UWM), Milwaukee, WI 07/2016-05/2017
Psychology Clinic
Tiered Supervision Experience
Supervisors: Kristin Smith, Ph.D.; Han-Joo Lee, Ph.D.

- Conducted live, individual supervision of junior graduate students as they completed psychoeducational assessments of adult and child clients with referral questions relating to learning disabilities, attention deficits, psychiatric symptoms, and cognitive difficulties.
- Provided feedback on psychodiagnostic interviews, administration of assessment measures, and client feedback sessions.
- Co-led group supervision meetings covering assessment-related topics.

Froedtert & The Medical College of Wisconsin (MCW), Milwaukee, WI 06/2015-09/2016
Department of Neurosurgery and Neurology, Division of Neuropsychology
Neuropsychology Practicum Student
Supervisors: Sara Swanson, Ph.D., ABPP-CN; Michael McCrea, Ph.D., ABPP-CN; Julie Bobholz, Ph.D., ABPP-CN; David Sabsevitz, Ph.D., ABPP-CN; Laura Umfleet, Psy.D.

Rotations: General Adult Neuropsychology Clinic, Mild Traumatic Brain Injury Clinic, Neuro-Oncology Clinic

- Conducted neuropsychological assessments with adult patients presenting with cognitive difficulties relating to traumatic brain injuries, epilepsy (pre and post surgical), brain tumors, memory disorders, movement disorders (including deep brain stimulation candidates), normal pressure hydrocephalus, multiple sclerosis, attention deficits, learning disabilities, and other neurological conditions.
- Participated as a member of interdisciplinary teams assessing functioning in patients with mild traumatic brain injuries and brain tumors.
- Observed and participated in psychodiagnostic intake interviews of patients.
- Aided in the selection of neuropsychological measures based on presenting symptoms.
- Administered, scored, and interpreted neuropsychological measures.

- Conceptualized cases based on information from medical records, interview, and results of neuropsychological testing, and integrated these into written reports (approximately 1 report/week)
- Participated in didactics including neuropsychology seminar, neuropsychology journal club, neuropsychology case conference, brain cuttings, and WADA procedures.

University of Wisconsin-Milwaukee (UWM), Milwaukee, WI 05/2014-05/2016
Psychology Clinic
Psychotherapy Practicum
Supervisors: Bonita Klein-Tasman, Ph.D.; Shawn Cahill, Ph.D.

Rotations: Adult OCD and Anxiety Disorders Team, Child Anxiety Disorders Team

- Provided therapy services for adults with a variety of psychiatric conditions including obsessive-compulsive disorder, panic disorder with agoraphobia, generalized anxiety disorder, depression, and trichotillomania.
- Provided therapy services for children with a variety of anxiety disorders including generalized anxiety disorder, obsessive-compulsive disorder, and specific phobia, as well as with social skills and attention deficits.
- Received supervised training in evidence-based interventions including exposure therapy, exposure and response prevention, behavioral activation for depression, mindfulness, and a manualized skills-based treatment for childhood anxiety disorders (Kendall's Coping Cat).
- Maintained written records of case conceptualizations, treatment plans, and progress notes.
- Received weekly group and individual supervision.

University of Wisconsin-Milwaukee (UWM), Milwaukee, WI 06/2013-05/2014
Psychology Clinic
Assessment Practicum
Supervisors: Bonita Klein-Tasman, Ph.D.; Han-Joo Lee, Ph.D.

- Completed psychodiagnostic assessments with adults and children, including completion of structured and semi-structured interviews, administration of intellectual, academic, and cognitive assessment measures, and provision of in-person feedback and recommendations.
- Conceptualized cases based on information gathered from interview, collateral reports of symptoms, and results of psychodiagnostic testing, and integrated these into written reports.

Central City Cyberschool, Milwaukee, WI 10/2013
Psychoeducational Assessment
Supervisor: Bonita Klein-Tasman, Ph.D.

- Conducted psychoeducational assessments, including assessment of cognitive abilities, academic skills, and emotional functioning, for children attending a charter school in the inner city of Milwaukee.
- Conceptualized cases based on interview, collateral reports of symptoms, classroom observation, and results of psychodiagnostic testing and integrated these into written reports.
- Presented case conceptualization and recommendations to an interdisciplinary team in individualized education plan (IEP) meetings.

University of Wisconsin-Milwaukee (UWM), Milwaukee, WI 08/2012-05/2014
Psychology Clinic
Vertical Team Member
Supervisors: Douglas Woods, Ph.D.; Shawn Cahill, Ph.D.; Robyn Ridley, Ph.D.

Rotations: Behavioral Intervention for Tic Disorders and Body-Focused Repetitive Behaviors Team, Adult OCD and Anxiety Disorders Team; Cognitive-Behavioral Therapy Team

- Attended weekly group supervision meetings involving video review of therapy sessions and discussion of empirically supported interventions.
- Participated in didactic review and live observation of behavioral interventions for individuals with tic disorders and body-focused repetitive behaviors.
- Completed intake interviews and assessments of therapy clients with anxiety and mood disorders using self-report inventories and structured and semi-structured interviews.

Moss Rehabilitation Hospital, Einstein Healthcare System, Elkins Park, PA 5/2010-08/2010
Drucker Brain Injury Center
Undergraduate Intern
Supervisor: Eileen Fitzpatrick, Ph.D.

- Assisted neuropsychologists, physiatrists, and physical, occupational, and speech therapists in the assessment and treatment of patients in the post-acute stage following moderate to severe acquired brain injuries.
- Participated in interdisciplinary team rounds to discuss progress and treatment plans for patients.

PROFESSIONAL AFFILIATIONS

International Neuropsychological Society (INS), Student Associate
American Academy of Clinical Neuropsychology (AACN), Student Member
Society for Neuroscience (SfN), Student Member
Psychonomic Society, Student Affiliate
American Psychological Society (APS), Graduate Student Affiliate
Sigma Xi, Student Affiliate