

# Can a Concussion History Affect the Susceptibility to the Misinformation Effect?

Geoffrey Genova<sup>1</sup>, Elle Gilbert<sup>1</sup>, Chris Churchill<sup>1</sup>, Amanda Acquaire<sup>1</sup>

<sup>1</sup>Middlebury College, Middlebury, Vermont 05753

Concussions have been shown to have damaging effects to multiple functions of the brain, including memory. One of the key components of memory is the ability to determine the original source of a memory, a concept called source monitoring. The current study was designed to explore how individuals with a history of concussions would compare with individuals without a concussion history when their source monitoring ability was tested. The researchers investigated the influence a concussion history would have on the susceptibility to the effects of misinformation. Twenty individuals with a concussion history and twenty-two individuals without a concussion history participated in the study. It was expected that individuals without a concussion history would perform better on tests evaluating their ability to correctly monitor the source of information. Statistical analysis revealed that both the concussed and non-concussed group performed similarly in their ability to source monitor. The contradictory results this study exhibits, when compared to previous research, suggests that further exploration into this particular portion of memory may be useful when understanding how concussions affect memory performance. The results of this study suggest that concussions may not always produce negative long-term memory related cognitive effects.

Abbreviations: TBI – Traumatic Brain Injury; LE – Low Expectancy; HE – High Expectancy

Keywords: Memory; Working Memory; Social Contagion

---

## Introduction

A concussion is a syndrome induced by some sort of force or impact to the head that results in neural dysfunction within the brain (Giza et al., 2014). Concussions have long been an issue in public health particularly in relation to athletics (Gavett et al., 2011). Recently, there has been significant discussion about the potential dangers they pose not just during the acute phase, where an individual is experiencing strong physical and mental symptoms, but also for post recovery phase mental health and functioning, when an individual is not actively experiencing symptoms. There is significant evidence in the psychological literature that concussions do have long-term effects on the functioning of the human brain in multiple areas of cognition and operation (Gavett, et al. 2011; Guskiewicz et al., 2005). Numerous studies

have been conducted on individuals with a history of concussions to assess specific brain abnormalities and performance deficits in comparison to individuals who have never been concussed (Broglia & Peutz, 2008; Schultz et al., 2012; DeKosky et al., 2010).

### *Long-Term Effects*

Investigations into the post recovery phase of concussed individuals have revealed numerous findings. Neuroimaging investigations (Tremblay et al., 2013), neuropsychological assessments (Killam et al., 2005) and other investigations into cognitive function of individuals who have suffered concussions in their past (Covassin et al., 2010; Moser and Schatz, 2002) have all yielded results pointing to the potentially harmful and enduring

effects that concussions can have on the brain long after the initial recovery period, including memory (Killam et al., 2005), concentration (Moser and Shatz, 2002), and processing speed (Covassin et al., 2010) impairments. These objective assessments of the long-term effects of concussions are not the only way in which this issue has been approached. More subjective assessment methods of interviewing select individuals (Caron et al., 2013, Mayers et al., 2013) and providing them with general health and memory questionnaires to relay their own subjective experience (Guskiewicz et al., 2005) have also yielded results that suggest concussions negatively affect cognitive functioning, specifically memory, following acute symptom recovery.

### *Hippocampus*

Concussions have been found to result in neuronal degeneration within hippocampal areas of the brain (Kiryaly et al., 2007). Previous research has indicated that the hippocampus is responsible in large part for performing two related but separate memory functions (Eldridge et al., 2005). Different sub-regions of the hippocampus are selectively responsible for episodic memory formation (encoding novel information) and retrieval of the episode (Eldridge et al., 2005). Episodic memory can be characterized as memory of autobiographical events (who, what, when, where, why knowledge) that can be explicitly stated, such as what an individual saw at a particular time. Damage to the hippocampus not only creates deficits in learning but also in retrieval, especially for memories created from the recent past (Manns et al., 2003). The formation of memory representations was found to be accompanied by specific increases in synaptic activity within particular regions of the hippocampus (Eldridge et al., 2005). Further indicating hippocampal involvement in memory function is the activation of glutamate receptors and increased activity of proteins in the hippocampus as part of a biochemical sequence of events that begins during the process of memory formation (Izquierdo et al., 1997).

While it has been well established that the hippocampus is responsible for long term memory processes (Lynch, 2004), research has

also concluded that hippocampal damage can impair performance in tasks that involve processes in the prefrontal cortex, suggesting that working memory may also be negatively affected by changes in the functioning of the hippocampus (Lipska et al., 2002).

### *Frontal Cortex*

In addition to the hippocampus, the frontal cortex is a brain region particularly susceptible to damage by concussions (Kiryaly et al., 2007). As the soft tissue of the brain is jostled within the brain case, the frontal areas have been shown to be vulnerable to injury against the sharp ridges inside the skull (Kiryaly et al., 2007). Research has shown that the prefrontal cortex is involved in long-term memory processes (Braver et al., 2001), particularly episodic long-term memory (Blumenfeld & Ranganath, 2007), but it is also regarded as the hub of the brain's working memory system (Markowitz et al., 2015). Working memory serves as a cognitive system that is responsible for holding, processing, and manipulating new information (Baddeley, 2007). Working memory is used as a temporary workspace for reasoning and guiding decision-making and behavior. Delayed-response tasks have repeatedly shown that on a cellular level, prefrontal neurons are activated in processing mnemonic events (Goldman-Rakic, 1995) and a strong linear relationship exists between neuronal activity in the prefrontal cortex and the working memory load (Braver et al., 1996).

Damage to the frontal cortex in the wake of a concussion has been well researched and studies document long-term neurophysiologic changes. During a spatial working memory task, patients who were in the post recovery phase of a concussion had a smaller increase in regional cerebral blood flow than control patients in their prefrontal cortex (Chen et al., 2003). Studies utilizing positron emission tomography (PET) have demonstrated frontal lobe hypometabolism (abnormally low metabolic rate) following a mild trauma to the brain both at rest and during working memory tasks (Umile et al., 2002). Several studies have shown that concussions can cause long-term motor system dysfunctions including abnormalities to intracortical inhibitory systems

and neurotransmission alterations (De Beaumont et al., 2007; De Beaumont et al., 2012; Henry et al., 2011). In more severe cases, in which concussions led to Traumatic Brain Injury (TBI), atrophy of the frontal lobe can occur (McKee and Daneshvar, 2015).

The frontal lobe damage associated with concussions has been hypothesized to impair episodic memory processing because of the role of the frontal lobe in mediating working memory processes (Owen et al., 1990). The frontal cortex is believed to contribute to the ability to organize multiple pieces of information in working memory, thereby enhancing memory for associations among items in long-term memory and integrating them into episodic memory (Eldridge et al., 2005). With an impaired working memory, one could expect general memory functioning and associated processes to decline. This connection between working memory and aspects of long-term memory such as episodic memory and memory monitoring is important when considering the extent of potential damage from concussions. Consistent with this view, long-term memory storage is impaired by concussions' long-term effects, shown in a study that highlighted how difficult it was for participants to retrieve previously stored information. (Gronwall & Wrightson, 1981). Another study focusing on the later life of American Football players found that even after multiple decades into the post-recovery phase, participants still reported episodic memory difficulties (Guskiewicz et al., 2005). These findings show the potential long-term detriments to frontal lobe function for previously concussed individuals. In addition, the prefrontal cortex may implement different control processes that support long-term memory formation and retrieval (Blumenfeld & Ranganath, 2007).

### *Source Monitoring*

A key retrieval-based process, source monitoring, is performed in the frontal cortex of the brain and contributes to accurate long-term memory. Source monitoring is the process of identifying the origin of recalled information (Niedźwieńska et al., 2002). This process is critical to several different cognitive tasks, such as the ability to exercise power over our own

beliefs, the experience of autobiographical recollection, and the capacity to differentiate between reality and expectation (Johnson et al., 1993; Marsh et al., 1997). Several studies have shown that the prefrontal cortex is activated when subjects are asked to complete source monitoring tasks (Cansino, Maquet, Dolan, & Rugg, 2002; Dobbins, Foley, Schacter, & Wagner, 2002; Mitchell, Johnson, Raye, & Greene, 2004; Turner et al., 2008). The present research looks to investigate if concussions, with their connection to frontal cortex impairment, impact source monitoring. Damage to the frontal cortex could imply that source monitoring is impaired following a concussion.

When trying to remember past events, problems in source monitoring can create incorrect memories. Individuals occasionally make errors when monitoring the source of information, in the form of remembering something that previously happened but incorrectly remembering how they experienced it. An example of this could be misremembering whether an individual saw an event or heard someone else's depiction of the event. A source monitoring error would be a person believing that they saw or experienced an event, when they only heard someone else describe it.

### *Misinformation Effect*

One form of a source monitoring error is known as the misinformation effect. The misinformation effect states that after experiencing an event, if presented with false information, people will misremember that false information as a part of the original event (Roediger et al., 1996; Loftus et al., 1989). Subjects will incorporate the given false information into their own personal account of the event (Roediger et al., 1996; Loftus, 1992). One way the misinformation effect has been investigated is to use a social contagion paradigm, in which participants are exposed to misinformation via collaborative recall. In a study designed by Roediger and Meade (2001), participants were instructed to observe several scenes and remember items observed in those scenes. The participants then collaborated with a confederate, who they believed to be another participant that had viewed the same scenes, to

recall items from each scene. The confederate recalled items that were not in the original scenes, in effect exposing the participant to misinformation about the contents of the pictures they viewed. Results from the study indicated that participants were affected by social contagion, as they recalled items in their final (individual) recall test that had only been suggested by the confederate during the collaborative recall portion of the study. Further, participants produced misinformation in their individual recall of the scenes at a significantly higher rate than participants in the control group, where no confederate collaboration occurred.

To summarize, while there has been significant research done on both the long-term effects of concussions and the misinformation effect separately, little has been studied on the intersection between the two. Existing research shows that concussions have long-term negative effects on the frontal lobe. Findings also show that source monitoring occurs in the frontal lobe (Turner et al., 2008). Thus, it is possible that people who have suffered concussions may experience difficulties with source monitoring.

### *Current Study*

Our study aimed to determine if people who had suffered from concussions in the past were more prone to the misinformation effect than those who had never experienced a concussion, as source monitoring difficulties should result in greater susceptibility to the misinformation effect. The literature suggests that people with a concussion history will have a harder time monitoring their memory, and thus will be more susceptible to the misinformation effect. In the current study, we examined participants' susceptibility to the misinformation effect using Roediger and Meade's (2001) social contagion paradigm. The social contagion paradigm of memory involves the implanting of false memories into an individual's mind through social influence. The paradigm showcases that we are susceptible to changing our memory based on another individual's account of the same event. Our style of social influence comes in the form of written lists of items that subjects believed were written by previous test participants. These lists were

comprised of items that were in the viewed scenes, as well as items that were not in the scenes, and were given to the participants after they had created their own original list of items they remembered from the scene. Participants were asked to evaluate whether they or the participant who produced the written list of words better recalled the contents of the scenes they studied. Thus, by reading the written list containing items that were not shown in the scenes during the study, we exposed participants to misinformation about the contents of the scenes they studied. Our study tested how concussions affected the validity of memory recall, which was vital information when considering the accuracy of memory produced by someone who had been previously concussed. We hypothesized that the performance of participants with a concussion history would show a larger misinformation effect in the form of accepting and endorsing misinformation items at a higher rate than people without a concussion history.

## **Material and Methods**

### *Participants*

This research was retroactively approved after the original experiment was executed as a class project, and publication was pursued after data collection. The proposal (15713, Examining the relationship between concussion history and memory) was reviewed by the Middlebury Institutional Review Board (IRB) and it was found to be exempt from further review under Federal Guideline 46.101(b)(2). All participants provided their written informed consent. Forty-two students (aged 18-22) from Middlebury College's undergraduate student body were recruited for this study. Participants were volunteers contacted via e-mail correspondence with sport team coaches. As a result of this recruiting method, all of the participants included in the study were members of a collegiate sports team. Twenty of our participants had previously suffered concussions and twenty-two of them had never suffered a concussion, as determined by the sports medicine department. Of the

twenty individuals with a concussion history, 30% had become unconscious as a result of a concussion. No individuals currently concussed were eligible to participate in the study.

### *Study Materials*

Participants were shown a series of six images of common household scenes: a desk, a kitchen, a bathroom, a toolbox, a bedroom, and a closet. These images were taken from a previous study examining social contagion of memory (Meade & Roediger, 2002). The scenes contained an average of 24 objects that were a mixture of high and low expectancy items. The expectancy ratings used were gathered from the same Meade and Roediger (2002) study in which thirteen Washington University in St. Louis undergraduates were asked to imagine each of the six scenes from a simple verbal description (“a typical kitchen scene”) and list at least 10 items they might expect to be in the scene. The items listed by five people were considered high-expectancy items, and items listed by one person were considered low-expectancy items. The range of the number of people listing each item was one to twelve.

A general information sheet was used to collect information about the participants that could be useful in later analysis. They were asked to report the number of concussions they had experienced, the age of their first concussion, the date of their most recent concussion, and if they have ever become unconscious from a concussion.

Each participant was given a packet upon his or her arrival at the experiment. The first six sheets of paper were blank sheets of printer paper designed for the free recall task after the presentation of the six scenes. The next six sheets were the social contagion lists designed for each scene. The social contagion lists for all six scenes were created the same way, following the design of Meade and Roediger’s (2002) Experiment 3. Each list was designed to look like a free recall list that could have been created by a previous participant in the study. It was created by randomly selecting various items from the scene and then inserting two items that were not present in the scene. These items were the misinformation items: one was considered a high expectancy item (likely to

be found in the scene) and one was considered a low expectancy item (unlikely to be found in the scene). The high expectancy item was placed third on the list and the low expectancy item was placed seventh on the list.

The final six pieces of paper were the recognition lists corresponding to each scene. The following description details how a recognition list was designed for a single scene. The lists for all six scenes were created the same way. The recognition list contained all of the items actually present in the scene. This number varied by the scene. The high expectancy item that was presented in the social contagion list was placed third in the list. The low expectancy item was placed seventh. There were also two additional control items included in this list to ensure that people were not simply likely to circle items because they were on the list. Control items had not been presented anywhere previously in the experiment and were placed in positions eleven and fifteen in each test list.

For the social contagion lists and the corresponding recognition lists for all six scenes, there were two groups: group A and group B. This was done to eliminate the possibility for any biases towards particular words. There were two groups of high expectancy/low expectancy items for each scene, and two groups of different corresponding control words for each scene. Half of the previously concussed participants received the lists from group A (10 participants) and the other half received the lists from group B (10 participants). The same was done with our never-concussed participants, half (11 participants) received the lists from group A and the other half received the lists from group B (11 participants).



**Figure 1.** Desk scene used in current study and Roediger and Meade's (2001) study.

### *Procedure*

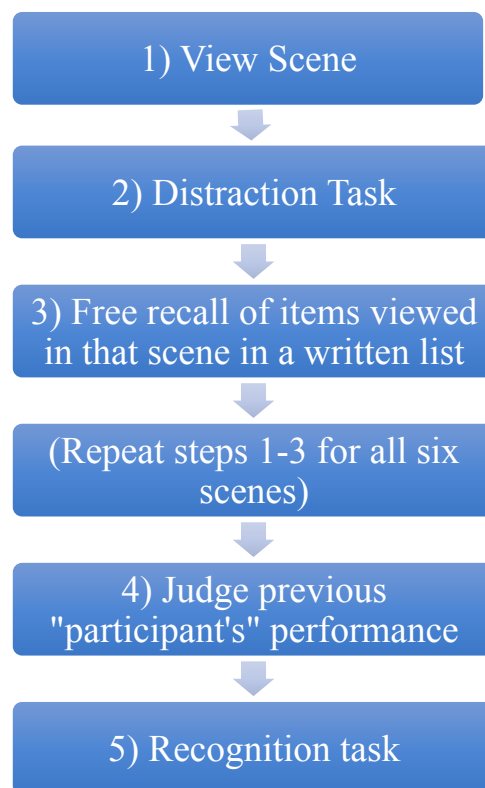
Participants entered the classroom and were seated with at least one desk in between them. In front of them on the desk was a consent form explaining the general procedures of the study that they were required to sign before the experiment began. They then filled out the general information sheet. Next, the researchers distributed a packet of papers to each participant that they were to leave untouched on the desk in front of them.

The experiment began by telling the participants that they were about to view six household scenes. These scenes were projected from a computer onto a screen at the front of the room so the participants could view them clearly and easily. They were shown each scene for one minute, and their job was to examine each scene closely because they would later be asked to perform tasks related to the content of the scenes. After the first scene was shown for one minute, the participants were instructed to complete a series of multiplication problems for one minute. They were then told that they had one minute to recall all of the items they could from the first scene. The same procedure occurred for the next five scenes. All six recall sheets were then collected.

After the free recall portion of the experiment was over, the participants were told that the next six sheets of paper in the stack contained the free recall responses of participants in a previous session of the study. These were the social contagion lists described above. At their own pace, they were to closely examine the items recorded by this other

participant and indicate on the paper whether they more accurately recalled the items in the scene or if this other participant did better at the task. The lists of items were presented in the same order as the scenes were previously shown.

After all of the participants in a session had finished reviewing the social contagion lists that introduced the misinformation, they were asked to turn their attention to the final stage of the experiment: the recognition task. They were told that the next six sheets of paper contained lists of items from each scene respectively. The tests for scenes were presented in the same order as study scenes were during encoding. At their own pace, their task was to go through the list from each scene and circle all of the items that they remembered from the scene, whether or not they had included the items in their initial recall of scenes. The sequence of a participant's actions can be seen in Figure 2. When everyone was finished with the final task, they were given a debriefing of the study and thank for their time.

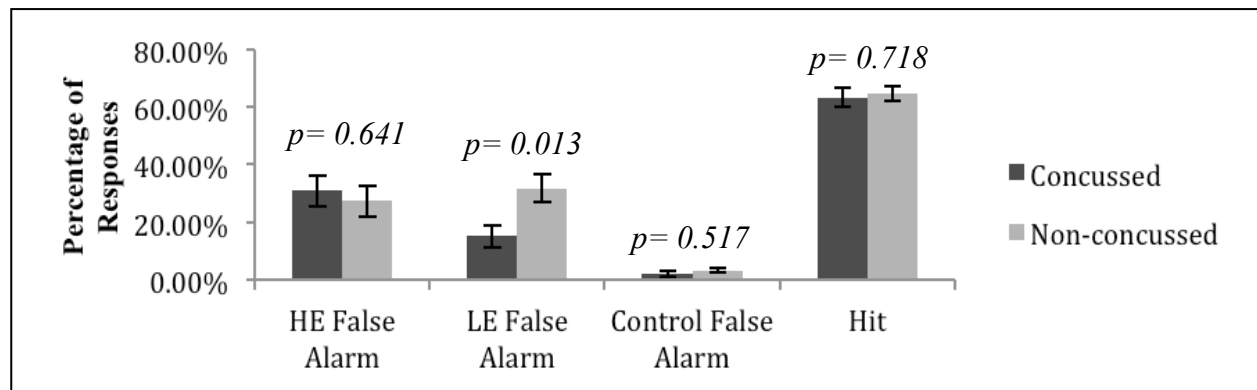


**Figure 2.** Experimental Design

For each study participant, we tallied the proportion of studied items correctly recognized (hits), the proportion of control items mistakenly recognized (control false alarms), the proportion of high expectancy items mistakenly recognized (HE false alarms), and the proportion of low expectancy items mistakenly recognized (LE false alarms). These data, displayed in Figure 3, were analyzed with a 4 (Item Type: hit, false alarm, HE false alarm, LE false alarm) x 2 (Group: Concussion vs. No Concussion) Analysis of Variance (ANOVA), with  $\alpha = .05$ . This was used to evaluate the main effects of Item Type and Group and the interaction effect between Item Type and Group.

120) = 1.249,  $MSe = 0.051$ ,  $p = 0.270$ . This finding implies that the concussed and non-concussed groups judged a similar proportion of items found in the scenes to have been studied.

Finally, there was a significant interaction between Item Type and Group, ( $F(3, 120) = 3.432$ ,  $MSe = 0.024$ ,  $p = 0.019$ ). From these findings, we first investigated the basis for the Item Type main effect, regardless of group distinction, with a series of paired samples Bonferroni-adjusted t-tests. With four comparisons, our adjusted alpha level was 0.0125.



**Figure 3.** Graph of Response Percentage by Item Type. *P* values included showing significant difference between concussed and non-concussed groups on LE False Alarm response rates.

## Results

The purpose of this analysis was to determine if there was any misinformation effect present and also to examine if the group distinction (concussion vs. no concussion) impacted how the participants responded to the four different item types.

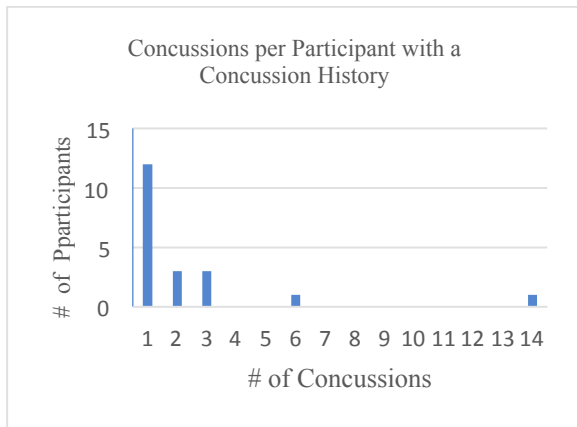
The ANOVA revealed a significant main effect of Item Type, ( $F(3,120) = 115.401$ ,  $MSe = 0.024$ ,  $p < 0.01$ ). This significant main effect demonstrates that there was a significant difference between the proportion of items that were judged to have been studied across the item types (HE, LE, Control, and Hit). There was no significant main effect found between the concussed and non-concussed groups, ( $F(3,$

The t-tests showed that the hit rate ( $M = 0.641$ ,  $SE = 0.021$ ) was significantly greater than any of the false alarm item rates, with the smallest significance coming from the comparison of hit rate to high expectancy false alarms ( $mean\ difference = 0.290$ ,  $SE = 0.037$ ;  $t(41) = -8.922$ ,  $p < 0.001$ ).

The paired samples t-tests revealed a significant difference between the rate of control false alarms ( $M = 0.025$ ,  $SE = 0.007$ ) and HE false alarms ( $M = 0.290$ ,  $SE = 0.037$ );  $t(41) = -7.007$ ,  $p < 0.001$ . There was also a significant difference found between the rate of control false alarms ( $M = 0.025$ ,  $SE = 0.007$ ) and LE false alarms ( $M = 0.238$ ,  $SE = 0.034$ );  $t(41) = -$

6.274,  $p < 0.001$ . These two tests establish statistical evidence for the presence of a misinformation effect as both high and low expectancy items were chosen at a rate significantly greater than the control items.

There was no significant difference between the rate of HE false alarms ( $M = 0.290$ ,  $SE = 0.037$ ) and LE false alarms ( $M = 0.238$ ,  $SE = 0.034$ );  $t(41) = 1.465$ ,  $p = 0.151$ .



**Figure 4.** Graph of Concussions per Participant with a Concussion History.

In order to understand the statistically significant interaction between group and item type, we compared the concussed and non-concussed groups' memory for the four item types with independent samples t-tests. There was no significant difference in the rate of hits ( $M = 0.633$ ,  $SE = 0.034$ ;  $M = 0.648$ ,  $SE = 0.025$ ;  $t(40) = -0.364$ ,  $p = 0.718$ ), high expectancy false alarms ( $M = 0.309$ ,  $SE = 0.052$ ;  $M = 0.273$ ,  $SE = 0.053$ ;  $t(40) = 0.470$ ,  $p = 0.641$ ), or control false alarms ( $M = 0.021$ ,  $SE = 0.010$ ); ( $M = 0.029$ ,  $SE = 0.008$ );  $t(40) = -0.654$ ,  $p = 0.517$  between the concussed and non-concussed groups respectively. However, there was a difference trending toward significance revealed when examining the low expectancy false alarms between groups. Individuals in the concussion group ( $M = 0.151$ ,  $SE = 0.038$ ) were found to have a lower rate of LE false alarms than individuals without concussions ( $M = 0.317$ ,  $SE = 0.050$ );  $t(40) = -2.613$ ,  $p = 0.013$ . This finding shows that individuals with a concussion history were potentially less likely to be susceptible to the misinformation effect when

exposed to low expectancy items on a recognition task.

## Discussion

The findings from our study did not support our original hypothesis that previously concussed individuals would be more susceptible to the misinformation effect. The group of individuals in the concussed group, who had experienced at least one concussive episode, did not perform worse on any of our memory measures, which suggests their cognitive ability was similar to the non-concussed group, or those individuals who had never experienced a concussive episode. Unexpectedly, the concussion group's score on the low-expectation contagion items was close to significantly better than the non-concussed group's score. Thus, the concussed group was potentially better at avoiding the misinformation effect for contagion items that were unusual, but plausible to the scene, than the non-concussed group. Overall, these results suggest that concussed individuals do not experience a deficit in source monitoring after they have recovered from being concussed.

Concussions have become an issue of increasing concern recently because of the studies that show the detrimental effects and the prevalence of concussions in the sports realm. Most studies display negative performance outcomes when examining the effects of concussions (Gronwall & Wrightson, 1981; Guskiewicz et al., 2005; McKee & Daneshvar, 2015), but our results were able to present a positive outlook. Because our results show no significant difference between concussed and non-concussed individuals for the misinformation effect, it is possible that in the long term, concussions may not be as detrimental to our memory as previously thought. Previously concussed participants may be able to recover from the trauma and return to a level of cognitive function similar to that of before the concussive episode. While there is relatively little research showing the lack of long-term consequences, there are a few studies that support our results.



In Iverson and Brooks et al. (2006), 867 high school and collegiate athletes were given tests that examined their verbal memory, visual memory, reaction time, and processing speed. The participants were separated into three groups: individuals who had never suffered any concussions, individuals who had suffered one concussion, and individuals who had suffered two concussions. There were no measurable cumulative effects of the concussions for those athletes (Iverson et al., 2004). An additional study found that concussions caused significant impairments in reaction time, processing speed, and working memory capacity. After ten days post injury, however, memory impairments along with other deficiencies from concussions were shown to resolve (Sim et al., 2008).

Terry et al. (2012) provides further support for our results. In this study, individuals performing different tasks were monitored through neuropsychological assessment and fMRI scanning. Individuals who had suffered concussions were found to have slightly different patterns of brain activation than their non-concussed counterparts. However, this had no significant effect on tests of reaction time and Stroop tests which evaluate the effect of interference on reaction time (Terry et al., 2012). This led researchers to conclude that the slight differences in brain activation patterns found between the concussed and non-concussed groups meant that those who were concussed still possessed the ability to recover their cognitive function, even with a different brain activation pattern (Terry et al., 2012). Our study, in combination with these studies, supports the idea that concussions may not have substantial negative long-term effects on cognition.

Other studies have highlighted the potential for lingering effects from a history of concussions. Findings from Iverson et al. (2004) revealed that young athletes who sustained multiple concussions reported significantly more symptoms and demonstrated a trend toward lower memory scores at a baseline test. This was suggestive of a cumulative, lingering effect of multiple concussions.

Tremblay et al. (2013) examined the long-term effects of concussions on otherwise

healthy athletes, 30 years after their last concussive episode. Using neuroimaging, a correlation was established between the cognitive deficits measured and the neuroimaging findings in concussed participants. This research revealed an episodic memory decline in former athletes with concussions, highlighting patterns often associated with abnormal aging.

A meta-analysis performed by Belanger et al. (2010) examined the differences in delayed memory and executive functioning performance between individuals with a single concussion and individuals with multiple concussions. Multiple concussions were found to be associated with poorer performance on both measures, suggesting that the number of concussions an individual sustained has a significant impact on their level of cognitive impairment.

It appears that there is a general pattern in previous literature that suggests multiple concussions may cause more serious lingering cognitive deficits. It is possible that because our study utilized a majority of participants who had only experienced a single concussion (60%, refer to Figure 4 for further information), our results failed to show that concussion history produced performance deficits. In regards to studies considering the long-term effects of concussions on aging (Gronwall & Wrightson, 1981; Guskiewicz et al., 2005), our study was limited to individuals who were in the 18-21 age range so any direct comparison does not appear appropriate.

Our results go beyond the findings of previous studies by exhibiting potentially superior performance on a memory distortion task by concussed individuals when compared to non-concussed individuals. The studies above display an equal cognitive performance by both concussed and non-concussed individuals, but our findings show a difference that favorably trends in the direction of the concussed individuals. No previous studies have displayed a result similar to this. It is difficult to explain this phenomenon, but an answer could possibly be found in the differences our study possesses when compared to previous research.

One possible explanation of our results was the subject group. All of our subjects came

from Middlebury College. Several studies have shown that exposure to higher education, or specialized education beyond the level of high school, alters the brain (Yen et al., 2004; Tun and Lachman, 2008; Lachman et al., 2010). It has been found that individuals who received a higher education displayed more synaptic connectivity in areas involved with memory, language and neurogenesis (Kim et al., 2015). In our study, the exposure to higher education for the participants may have negated the possible long-term effects of the concussions and allowed them to do just as well, and in some cases better, than the subjects who had never been concussed before.

Another possibility for the results found between concussed and non-concussed individuals in the low expectancy contagion items could be attributed to the frequency of testing that concussed individuals have previously experienced. It is conceivable that the non-concussed participants have never participated in more than the baseline memory test administered to all athletes in order to gauge their normal levels of performance in the event of a concussion, while it is probable that concussed athletes have had to go through a series of memory tests to prove that their concussive symptoms have subsided before returning to their respective fields of play. If that were the case, concussed athletes would have more experience with memory tests. Through this additional experience they could have had an advantage in terms of the development of strategies that enabled them to better monitor their memories or distinguish between studied and plausible, but non-studied, items. Because of their familiarity with this style of testing, this could be a possible explanation of our statistically significant result of concussed participants outperforming the non-concussed participants.

Our results suggest that concussed and non-concussed individuals do not always differ in their susceptibility to the misinformation effect. Although this is not the result we expected, its implications are positive. It is possible that concussed individuals are actually less susceptible to the misinformation effect, particularly when exposed to low-expectancy contagion items. This suggests that individuals

who have previously been concussed do not necessarily suffer long-term consequences in source monitoring tasks as well as potentially different memory tasks.

## Acknowledgements

We would like to thank Professor Jason Arndt of the Middlebury College Psychology department for his guidance and encouragement throughout the entire process. We would also like to thank Professor Kim Cronise for her encouragement to pursue publication.

## Corresponding Author

Geoffrey Genova  
Middlebury College  
11 Crescent Road  
Lexington, MA 02421  
geoffreygenova@gmail.com

## References

- Baddeley, A (2007). *Working memory, thought, and action* (Vol. 45). OUP Oxford.
- Belanger HG, Spiegel E, & Vanderploeg RD (2010). Neuropsychological performance following a history of multiple self-reported concussions: a meta-analysis. *J Int Neuropsychol Soc*, 16(02), 262-267.
- Blumenfeld RS, & Ranganath C (2007). Prefrontal cortex and long-term memory encoding: an integrative review of findings from neuropsychology and neuroimaging. *Neuroscientist*, 13(3), 280-291.
- Braver TS, Barch DM, Kelley WM, Buckner RL, Cohen NJ, Miezin FM, ... & Petersen SE (2001). Direct comparison of prefrontal cortex regions engaged by working and long-term memory tasks. *Neuroimage*, 14(1), 48-59.
- Broglio SP, & Puetz, TW (2008). The effect of sport concussion on neurocognitive function, self-report symptoms and postural control. *Sports Med*, 38(1), 53-67.

- Caron JG, Bloom GA, Johnston KM, & Sabiston CM (2013). Effects of multiple concussions on retired national hockey league players. *J Sport Exerc Psychol*, 35(2), 168-179.
- Chen SHA, Kareken DA, Fastenau PS, Trexler LE, & Hutchins GD (2003). A study of persistent post-concussion symptoms in mild head trauma using positron emission tomography. *J Neurol Neurosurg Psychiatry*, 74(3), 326-332.
- Covassin T, Elbin R, Kontos A, & Larson E (2010). Investigating baseline neurocognitive performance between male and female athletes with a history of multiple concussion. *J Neurol Neurosurg Psychiatry*, 597-601.
- DeKosky ST, Ikonovic MD, & Gandy S (2010). Traumatic brain injury—football, warfare, and long-term effects. *N Engl J Med*, 363(14), 1293-1296.
- Eldridge LL, Engel SA, Zeineh MM, Bookheimer SY, & Knowlton BJ (2005). A dissociation of encoding and retrieval processes in the human hippocampus. *J Neurosci*, 25(13), 3280-3286.
- Gavett BE, Stern RA, & McKee AC (2011). Chronic traumatic encephalopathy: a potential late effect of sport-related concussive and subconcussive head trauma. *Clin Sports Med*, 30(1), 179-188.
- Giza CC, & Kutcher JS (2014). An introduction to sports concussions. *Continuum (Minneapolis)*, 20(6), 1545-1551.
- Gronwall, D., & Wrightson, P (1981). Memory and information processing capacity after closed head injury. *J Neurol Neurosurg Psychiatry*, 44(10), 889-895.
- Guskiewicz KM, Marshall SW, Bailes J, McCrea M, Cantu RC, Randolph C, & Jordan BD (2005). Association between recurrent concussion and late-life cognitive impairment in retired professional football players. *Neurosurg*, 57(4), 719-726.
- Henry LC, Tremblay J, Tremblay S, Lee A, Brun C, Lepore N, ... & Lassonde M (2011). Acute and chronic changes in diffusivity measures after sports concussion. *J Neurotrauma*, 28(10), 2049-2059.
- Iverson GL, Brooks BL, Lovell MR, & Collins MW (2006). No cumulative effects for one or two previous concussions. *Br J Sports Med*, 40(1), 72-75.
- Iverson GL, Gaetz M, Lovell MR, & Collins MW (2004). Cumulative effects of concussion in amateur athletes. *Brain Inj*, 18(5), 433-443.
- Killam C, Cautin R, & Santucci A (2005). Assessing the enduring residual neuropsychological effects of head trauma in college athletes who participate in contact sports. *Arch Clin Neuropsychol*, 20(5), 599-611.
- Kiraly MA, & Kiraly SJ (2007). Traumatic Brain Injury and Delayed Sequelae: A Review - Traumatic Brain Injury and Mild Traumatic Brain Injury (Concussion) are Precursors to Later-Onset Brain Disorders, Including Early-Onset Dementia. *ScientificWorldJournal*, 7, 1768-1776.
- Lachman ME, Agrigoroaei S, Murphy C, & Tun PA (2010). Frequent Cognitive Activity Compensates for Education Differences in Episodic Memory. *Am J Geriatr Psychiatry*, 18(1), 4-10.
- Lipska BK, Aultman JM, Verma A, Weinberger, DR, & Moghaddam B (2002). Neonatal damage of the ventral hippocampus impairs working memory in the rat. *Neuropsychopharmacol*, 27(1), 47-54.
- Loftus EF (1992). When a lie becomes memory's truth: Memory distortion after exposure to misinformation. *Curr Dir Psychol Sci*, 1(4), 121-123.
- Loftus EF, & Hoffman, HG (1989). Misinformation and memory: The creation of new memories. *J Exp Psychol Gen*, 118(1), 100-104.
- Lynch M (2004). Long-Term Potentiation and Memory. *Physiol Rev*, 84(1), 87-136.
- Manns JR, Hopkins RO, & Squire LR (2003). Semantic memory and the human hippocampus. *Neuron*, 38(1), 127-133.
- Markowitz DA, Curtis CE, & Pesaran B (2015). Multiple component networks support working memory in prefrontal cortex. *Proc Natl Acad Sci USA*, 112(35)
- Marsh RL, Landau JD, & Hicks JL (1997). Contributions of inadequate source monitoring to unconscious plagiarism during idea generation. *J Exp Psychol Learn Mem Cogn*, 23(4), 886.

- Mayers L (2013). Outcomes of Sport-Related Concussion Among College Athletes. *J Neuropsychiatr*, 25(2), 115-115.
- McKee AC, & Daneshvar DH (2015). The neuropathology of traumatic brain injury. *Handb Clin Neurol* (Vol. 127). Elsevier.
- Meade ML, & Roediger HL (2002). Explorations in the social contagion of memory. *Mem Cognit*, 30(7), 995-1009.
- Moser RS, & Schatz P (2002). Enduring effects of concussion in youth athletes. *Arch Clin Neuropsychol*, 17(1), 91-100.
- Owen AM, Downes JJ, Sahakian BJ, Polkey CE, & Robbins TW (1990). Planning and spatial working memory following frontal lobe lesions in man. *Neuropsychologia*, 28(10), 1021-1034.
- Roediger III HL, Jacoby JD, & McDermott KB (1996). Misinformation effects in recall: Creating false memories through repeated retrieval. *J Mem Lang*, 35(2), 300-318.
- Roediger III HL, Meade ML, & Bergman ET (2001). Social contagion of memory. *Psychon Bull Rev*, 8(2), 365-371.
- Schultz SR, Bao F, Omana V, Chiu C, Brown A, & Cain DP (2012). Repeated mild lateral fluid percussion brain injury in the rat causes cumulative long-term behavioral impairments, neuroinflammation, and cortical loss in an animal model of repeated concussion. *J Neurotrauma*, 29(2), 281-294.
- Sim A, Terryberry-Spohr L, & Wilson KR (2008). Prolonged recovery of memory functioning after mild traumatic brain injury in adolescent athletes. *J Neurosurg*, 116(6), 511-516.
- Terry DP, Faraco CC, Smith D, Diddams MJ, Puente AN, & Miller LS (2012). Lack of long-term fMRI differences after multiple sports-related concussions. *Brain Inj*, 26(13-14), 1684-1696.
- Tremblay S, De Beaumont L, Henry LC, Boulanger Y, Evans AC, Bourgouin P, . . . Lassonde M (2013). Sports concussions and aging: A neuroimaging investigation. *Cereb Cortex*, 23(5), 1159-1166.
- Tun PA, & Lachman ME (2008). Age Differences in Reaction time and Attention in a National Telephone Sample of Adults: Education, Sex, and Task Complexity Matter. *Dev Psychol*, 44(5), 1421-1429.
- Umile EM, Sandel ME, Alavi A, Terry CM, & Plotkin RC (2002). Dynamic imaging in mild traumatic brain injury: support for the theory of medial temporal vulnerability. *Arch Phys Med Rehabil*, 83(11), 1506-1513.
- Yen YC, Yang MJ, Shih CH, & Lung FW (2004). Cognitive impairment and associated risk factors among aged community members. *Int J Geriatr Psychiatry*, 19(6), 564-569.