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The Effects of Acute Exercise Intensity on Episodic Memory and False Memory

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THE EFFECTS OF ACUTE EXERCISE INTENSITY ON EPISODIC MEMORY AND FALSE MEMORY

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ABSTRACT

The Effects of Acute Exercise on Episodic Memory and False Memory

Previous experimental work demonstrates that acute exercise may enhance episodic memory performance. However, limited research has examined the extent to which acute exercise influences false memory production, and no studies, to date, have examined whether there is an intensity-specific effect of acute exercise and true and false memories. Thus, the present experiment evaluated the effects of acute exercise on episodic memory and false memory. A three-arm, parallel, between-group randomized controlled trial was employed, with participants (M_{age} = 20.8 years) randomized into a moderate-intensity exercise group (15-minute bout of treadmill exercise at 50% Heart Rate Reserve; N = 20), a high-intensity exercise group (15-minute bout of treadmill exercise at 80% Heart Rate Reserve; N = 20), or a control group (15 minutes of sitting; N = 20). Episodic and false memory were both assessed using the Deese-Roediger-McDermott (DRM) paradigm. For the number of words recalled across each of the 6 lists, there was a significant main effect for list (F(list)=10.2, P<.001, η^2_p=.15), marginally significant main effect for group F(group)=2.7, P=.07, η^2_p=.09), but no list by group interaction effect (F(list x group)=1.00, P=.44, η^2_p=.03). Those in the high-intensity exercise group recalled significantly (P<0.05) more words than the control group. For the false word recall, there was not a significant main effect for list (F(list)=2.15, P=.06, η^2_p=.04), group (F(group)=2.20, P=.12, η^2_p=.07) or list by group interaction (F(list x group)=1.27, P=.24, η^2_p=.04), but across various lists, high-intensity acute exercise was associated with a greater rate of false memories. For the memory recognition task, there was no main effect for word type (F=.85, P=0.46, η^2_p=.01), group (F=.85, P=.43, η^2_p=.03), word type by

group interaction (F=.97, P=.44, η^2_p=.03), recall by group interaction (F=1.03, P=.41, η^2_p=.04), or word type by recall by group interaction (F=1.13, P=.32, η^2_p=.04). However, there was a main effect for recall (F=64.3, P<.001, η^2_p=.54) and a word type by recall interaction (F=182.6, P<.001, η^2_p=.77). That is, participants, across all three experimental conditions, were more likely to perceive the studied and critical lure words as being "old" (i.e., that they previously were exposed to them during the study session). These findings suggest that high-intensity exercise may enhance true episodic memories, and, possibly, increase the rate of select false memories. We discuss these findings in the context of how different acute exercise intensities may have unique and differential effects on underlying mechanistic processes related to true and false episodic memory.

Keywords: fuzzy trace theory; hippocampus; prefrontal cortex; recollection

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BACKGROUND

Episodic memory, or the retrospective recall of an event, is characteristically conceptualized as population of communicating neurons, ultimately leading to the physical memory trace. Recent work suggests that acute exercise can help to facilitate episodic memory. Many of the potential mechanisms behind this include exercise-induced alterations in parameters that help develop and stabilize the memory trace. A memory trace forms through three phases: memory encoding (i.e., acquisition), consolidation (i.e., storage), and retrieval. Memory encoding can be instant (i.e., a flashbulb memory) or incremental, meaning that situations accumulate over several experiences to begin the encoding process. Memory performance is optimized when the same cue is present at encoding and retrieval, which is known as the 'encoding-specificity principle.' Further, encoding and retrieving in the same environment is also beneficial to memory performance. This context-dependent learning can be demonstrated during exercise (i.e., encode and retrieve at rest) and learning (i.e., encoding and retrieving in the same mood state). Additionally, research suggests an attention-encoding relationship, meaning that memory retrieval is enhanced when a higher degree of attention is put forth. The underlying mechanism for this is that cognitive attention may help to facilitate neuronal gamma synchronization, which may subserve synaptic potentiation. Enhanced attention may also facilitate memory encoding via dopamine modulation; however, dopamine's effect on memory will be discussed later. Further, emerging research suggests that exercise prior to encoding and during consolidation may help facilitate memory retrieval. The second phase of a memory trace is consolidation, where memories undergo consolidation to transform from short-term to long-term

memories. During this process, memories are strengthened and modified; however, some memories need to be reconsolidated due to reactivation. By improving the strength of functional connections, neurons are able to encode memories through communication, which involves electronic conduction, action potential, and synaptic transmission. Respectively, depolarization, repolarization, and summation must occur for these functional connections to be made. The strength of inputs decide whether or not potentiation is produced. For example, short-term studying represents a theoretical weak input that may not evoke potentiation; however, exercising prior to studying may strengthen this input, producing potentiation. Strengths of input and potentiation will also be discussed later in this section.

The hippocampus plays an important role in consolidation, specifically in the standard consolidation theory, in which it serves as a temporary repository for memories before they go to the neocortex as long-term memories. Adjustments to the memory can be made by replaying the hippocampal representation to the neocortex, which results in hippocampal representatives decaying. Multiple traces can be formed from a single, initial trace, known as the multiple trace theory. This occurs when a memory is reactivated, which causes a newly encoded hippocampal memory trace to be formed, which results in a new trace going into the neocortex. This means that, over time, memories that have been reactivated are moved from the cortical-hippocampal circuity to the semantic memories of the cortex, potentially removing contextual details. Moreover, the cortical version of the memory develops into a gist-like memory. The last phase of a memory trace is retrieval, the subsequent re-accessing of events through a stimulus-

induced or spontaneous activation of the internal representation. Specific retrieval cues are necessary to access a memory and can affect the speed and success of retrieval. This process has two critical components, the item-specific and item-invariant component. The item-specific component helps to recover the memory, while the item-invariant actually searches, controls, and verifies the process. The hemispheric encoding retrieval asymmetry model is very interesting in that it hypothesizes that the left and right prefrontal cortexes differ in the type of memory they retrieve, semantic and episodic, respectively. The process of retrieval provides us with the terms 'free-recall,' 'cued-recall,' 'intentional recall,' and 'incidental recall.' Similarly to the encoding phase, the retrieval phase may be optimized by similar encoding and retrieval phases that are context- and state-dependent. The main purpose of a cue is to retrieve a specific memory; however, cues can cause individuals to select, rearrange, and distort memories during the process. Overall, the process of forming a memory trace is critical to understanding memory, and more specifically, the effects of acute exercise on episodic and false memory.

Loprinzi et al.[1,2] suggest that there are several mechanisms that demonstrate the effects of acute exercise on episodic memory function; therefore, a summary of these potential mechanisms will provide a better understanding of the present study. The main focus will be on the effects of acute exercise on the functional connectivity between neurons, known as long-term potentiation (LTP). The first hypothesis that this section shall discuss covers the pathways in which acute exercise induces neuronal excitability. The first pathway is of the muscle spindle, which explores how acute exercise enhances neuronal firing and

excitability. This pathway begins when skeletal muscles contract, activating the respective muscle spindles, which then generate action potentials that are eventually transmitted via peripheral nerves to the brain stem, where it activates the nucleus tractus solitaries. The nucleus tractus solitaries projects into the locus coeruleus and then projects into key related structures, such as the dentate gyrus, Cornu Ammonis (CA3 and CA1) – all housed in the hippocampus. When we exercise, it activates the muscle spindles within the skeletal muscle fiber and that sends a signal all the way up to the brain to increase neuronal firing within the hippocampus. The second pathway involves the vagus nerve, the longest cranial nerve in the body and made up of 80% afferent fibers and 20% efferent fibers. The vagus nerve can be activated either from tissues (e.g., lungs and heart) or increases in catecholamines induced by exercise. The afferent fibers transmit the information collected from the tissues and relay it to the nucleus tractus solitaries. As stated previously, the nucleus tractus solitaries projects into the locus coeruleus, which has connections with the CA1 hippocampal structure, a critical area of study under the context of hippocampal long-term potentiation. Through the vagus nerve pathway, the stimulation of the brain stem can increase the Cornu Ammonis region, which contains a myriad of memory-related neurotransmitters (i.e., norepinephrine, dopamine, serotonin, and acetylcholine). Similar to the muscle spindle pathway, the vagus nerve pathway is stimulated with exercise. When we exercise, our lungs expand and our heart beats faster. The afferent nerve fibers in these organs transmit a signal all the way up to the nucleus tractus solitaries and locus coeruleus, so it finally reaches the hippocampus. This pathway is another example acute exercise increasing neuronal excitability within hippocampal

structures, further supporting that anatomical connections may allow for functional connectivity to occur.

The third mechanism discusses the mechanistic actions of neurotransmitter mediation influencing the exercise-memory interaction. Both the muscle spindle and the vagus nerve activation induce hippocampal neuronal excitability via various neurotransmitters in the CA1 structure. There are several neurotransmitters that play an important role in the communication of pre- and post-synaptic neurons that include norepinephrine, dopamine, serotonin, and acetylcholine. Exercise has been shown to upregulate each of these different neurotransmitters, of which have different projections into the hippocampus. So acute exercise may increase communication across neurons by increasing synaptic plasticity as well as the production of these key neurotransmitters. Since memory is essentially a pattern of neuronal activity across a pool of different neurons, when you form a memory, a series of different neurons start to communicate with each other. Exercise aids in this by increasing long-term potentiation, or the functional connectivity of different neurons. This is useful because it allows for these different neurons to encode and retrieve particular memories. An example of this is exercise increasing the transcription of certain proteins, such as plasticity related proteins from CREB, which then travel to other synapses to help subserve long-term potentiation. Exercise tends to increase the strength of long-term potentiation by increasing the production of proteins that strengthen that synapse that allow for proper communication. If the synapse is not strong that may influence the degree to which the pre- and post-synaptic neurons can effectively communicate and send signals. Now, let's discuss how

each individual neurotransmitter facilitates long-term potentiation. Norepinephrine subserves long-term potentiation through β-receptors, one of three type of metaboreceptors to which norepinephrine binds. These β-receptors produce cyclic adenosine monophosphate (cAMP) via activated adenylate cyclase, which eventually leads to CREB protein activation. Therefore, norepinephrine helps facilitate long-term potentiation with increased protein kinase activation. Dopamine influences long-term potentiation several ways (e.g., mediating memory trace formation, metaplastic control), with the help of D1/D5 receptors that activate cAMP and PKA. Interestingly, dopamine may help stabilize the synaptic tag by initiating processes related to the synthesis of plasticity-related proteins. Overall, dopamine plays a vital role in synaptic connections and memory consolidation. Serotonin's role in hippocampal long-term potentiation is stress-specific, meaning that acute exercise triggers the release of more serotonin. The activation of the $G\alpha_s$-coupled receptor leads to the activation of PKA via adenylate cyclase stimulation and cAMP elevations. PKA activation activates CREB-modifying gene expression, which has been demonstrated to show that increased serotonin levels may mediate memory enhancement effects. Potentially, Acetylcholine also plays a role in mediating improvements in memory function. When bound to muscarinic cholinergic receptors, acetylcholine release increases, furthermore enhancing hippocampal CA1 long-term potentiation. This may be a result of K+ blockage mediating the conductances and enhancements of N-methyl-D-aspartate (NMDA) receptor currents.

The second hypothesis behind these mechanisms is that acute exercise may subserve episodic memory via synaptic tagging and associativity effects. Once again, long-term potentiation is involved, except this time it is serving as a trigger for short-term

memories, which, through synaptic tagging and stronger memory triggers, are converted to a stable long-term memory. The stronger memory triggers plasticity-related proteins, produced via CREB mechanisms, assists in retrieving the weaker memory and tags another synapse to help facilitate long-term potentiation at the memory synapse. Loprinzi et al. hypothesize that acute exercise serves as an initial priming event that induces changes in the neurons and synapses that regulate plasticity induced by the memory stimuli.

Another mechanism related to neuronal activity and acute exercise is the associativity effect. In this mechanism, when an acute bout of exercise occurs roughly around the same time as a memory stimulus, the exercise may help to stabilize the memory trace through long-term potentiation mechanisms. These two pathways have strong and weak inputs with exercise and the memory stimulus, respectively. When a high action potential is induced from the acute bout of exercise, the memory trace becomes activated. Then there is a rising accumulation of post-synaptic sodium and calcium ions, which may leak over and induce potentiation of the memory trace, and induce long-term potentiation of the pre- and post-synaptic memory stimulus neuron. The action potential and accumulation of ions may lead to synaptic depolarization of the memory stimulus, which eventually will conduct the priming of the NMDA mechanisms to facilitate long-term potentiation.

The last mechanism relating to acute exercise and episodic memory is the cognitive attention mechanism, which involves arousal/attentive effects. Before explaining this mechanism, it is important to understand the three pertinent systems of psychological attention: alerting (readiness to receive the stimulus), orienting (directing attention to

target stimulus), and executive control (management of mental resources to focus and inhibit conflicting stimuli). Attentional processing can occur in two forms, which include bottom-up attention (involuntary) and top-down attention (voluntary). Bottom-up processing involves the mesencephalic reticular formation, thalamus, and limbic system. This process begins when (sensitive) neurons within a group of neuronal memory traces become self-activated after a sensory stimulus. If other sources stimulate these neurons through any sort arousal-induced (mesencephalic reticular formation and thalamus) or affectively reinforced (limbic system) excitatory input, then they will continue to fire. Notable evidence suggest that acute exercise may induce neuronal excitation in the mesencephalic reticular formation, thalamus, and limbic structures; thus, acute exercise may help to facilitate bottom-up attentional processing. Top-down processing is a higher-order process that involves the frontal and parietal structures, along with the neuronal ensemble of the bottom-up structures. This process integrates feedback from bottom-up structures and other dynamic brain structures. By increasing neuronal activity in both the frontal and parietal structures, acute exercise may subserve top-down processing.

Overall, various exercise-induced mechanisms, such as neurogenesis, gliogenesis, angiogenesis, cerebral circulation, and growth factor production, may help explain why acute bouts of exercise over a long period of time subserve cognition and memory. The potential mechanisms summarized above include the vagus nerve and muscle spindle pathways, alterations in neurotransmitters, synaptic tagging/capturing, associativity, and psychological attention. Future research should look into the effects of intensity-specific exercise on cognition and memory performance.

Limited research has examined the extent to which acute exercise influences false memory production. False memories, or confabulations occur when there is a perception of a recalled past event or episode that never actually occurred. Several potential mechanisms have been suggested in emerging research. False memories may arise as a result of encoding or retrieving a semantically related memory. Green et al. hypothesized that exposure to semantically related words (during the use of the DRM paradigm) may have caused an activation of the related lure words, making participants think that the words were previously stated during the encoding task. Another potential mechanism includes source monitoring, which includes attributions about the origin of activation information. The binding of features (i.e., semantic content, spatial location) of memories that occurs during source monitoring can be disrupted by stress or brain damage.[3]

Plancher et al.[4] examined the underlying mechanisms of false memory production in young adults, healthy adults, and Alzheimer's Disease (AD) patients. The three potential mechanisms suggested were changes in executive functioning and both semantic and episodic memory. To account for the increase in false memory production in older, healthy adults, Plancher et al.[4] confirmed that it may result from the loss of verbatim information associated with episodic memory but a preservation of the gist information. Additionally, research suggests that the quality of episodic memory may have a greater role in modulating false memories.[4] Indeed, research indicates that a decrease in verbatim memory might make a greater reliance on the gist trace, which suggests that the number of correct memories will decrease and false memory production will increase.[4] Plancher et al.[4] concludes by hypothesizing (cautiously) that false memory mechanisms might depend on changes in executive functions and both semantic and episodic memory.

Further, this hypothesis is supported by Plancher et al.[4] with two findings: 1) healthy older adults were unable to suppress the influences of semantic ideas during memory tasks (i.e., recall and recognition tasks), which is supported by research suggesting that memories are stored in the brain and tagged for future sorting and organized retrieval by the prefrontal cortex;[5] therefore, damage to the prefrontal cortex would provide evidence that memories reconstructed afterwards are false memories and are reassembled. Future research should explore inhibition versus updating during the encoding/retrieval process and determine how this might influence false memory production.

Additionally, executive functioning has shown to decrease with age and AD. AD patients are more inclined to produce cross-list intrusions, indicating damage to executive functioning structures (i.e., prefrontal cortical). According to a regression analysis, executive functioning served as a mediator for semantically-related false memories.[4]

In summary, the mechanisms discussed underlie acute exercise and help facilitate episodic memory (e.g., long-term potentiation) and false memory (e.g., executive function). Such mechanisms that influence long-term potentiation include, e.g., muscle spindle and vagus nerve pathways, neurotransmitter mediation, synaptic tagging and capturing, associativity, and cognition attention. Other mechanisms that influence executive function include, e.g., fuzzy trace theorem, changes in semantic and episodic memory, and synaptic tagging and capturing. These hypothesized mechanisms and models assume that the bout of acute exercise occurs before the episodic and false memory stimuli; therefore, changes in temporality will also change the mechanistic

relationships. Future research should continue to examine the potential intensity-specific effect of acute exercise on episodic and false memories.

INTRODUCTION

Emerging work demonstrates that acute exercise can help to facilitate episodic retrospective memory,[6] but to date, no studies have examined the effects of exercise on false memory function. Previous work has discussed potential mechanisms of false memory.[4,7-9] In brief, false memories, or memory distortions, may arise from culturally determined expectations, labeling of the memory/event, and imperfect reality monitoring processes, such as source monitoring, which includes attributions about the origin of activation information. The source monitoring framework[10] is perhaps one of the more extensive theoretical accounts of false memories, which highlights several key aspects of false memories.

These key aspects indicate that memory attributions arise from 1) various qualitative characteristics of the mental experience (e.g., perceptual, spatial, temporal, or emotional details), 2) the embeddedness of the mental experience (e.g., availability of supporting memories), and 3) goals, beliefs, motivation, and social factors. Per this model, false memories occur because mental experiences arising from different events have overlapping characteristics that are imperfectly differentiated. Additional work also indicates that episodic memory and executive function performance predicts false memory function.[4] Both of these cognitive functions have been shown to be influenced by acute exercise,[2,11] providing plausibility for a potential relationship between acute exercise and reducing false memory performance. Further, acute exercise may subserve the encoding of contextually specific information (reactivate verbatim memory traces and attenuate the reactivation of gist traces[12]), and in turn, minimize false memory recall.

Lastly, as we have recently discussed,[13] few studies have examined the potential intensity-specific effects of acute exercise on episodic memory, let alone false memory function. To address these two gaps in the literature, the purpose of this experiment was to evaluate the effects of acute exercise, across varying intensities, on episodic memory and false memory performance.

METHODS

Study Design

The present study was a three-arm parallel between-group randomized controlled trial, consisting of two exercise experimental groups and a control group. The exercise groups engaged in an acute 15-minute bout of moderate-intensity exercise or high-intensity exercise. The control group completed a time-matched seated task (on-line game). Both groups completed one laboratory visit.

Participants

All three groups included 20 participants (N=60). This is based from a power analysis indicating a sample size of 20 would be needed for sufficient power (d, 0.90; two-tailed α error probability, 0.05; 1-β error probability, 0.80). This was informed from other related work demonstrating relatively large effect sizes (d, 1.04; η^2_p=0.23-0.29).[6,14] We recruited through classroom announcements and word-of-mouth. Participants included male and females between the ages of 18 to 35 yrs. Additionally, participants were excluded if they:

Self-reported as a daily smoker[15,16]

Self-reported being pregnant[17]

Exercised within 5 hours of testing[6]

Consumed caffeine within 3 hours of testing[18]

Took medications used to regulate emotion (e.g., SSRI's)[19]

Had a concussion or head trauma within the past 30 days[20]

Took marijuana or other illegal drugs within the past 30 days[21]

Were considered a daily alcohol user (>30/month for women; >60/month for men)[22]

Experimental Conditions

Similar to other related research,[23] the control condition played a medium-level, on-line administered, Sudoku puzzle. Participants in this control group completed this time-matched puzzle for 20-minutes prior to completing the memory task (described below). The website for this puzzle is located here: https://www.websudoku.com/

The two exercise conditions (moderate-intensity and vigorous-intensity) engaged in a 15-min bout of treadmill exercise, followed by a 5-min recovery period. The HRR equation used to evaluate exercise intensity is:

$$HRR = [(HR_{max} - HR_{rest}) * \% \ intensity] + HR_{rest}$$

To calculate HR_{rest}, at the beginning of the visit, participants sat quietly for 5 minutes, and HR was recorded from a Polar HR monitor. To estimate HR_{max}, we calculated the participants estimated HR_{max} from the formula 220-age. For the moderate-intensity and vigorous-intensity exercise, respectively, 50% and 80% will be entered into the above formula. These respective intensities represent moderate- and vigorous-intensity exercise.[24]

Memory Assessment

The procedures of this false memory task was modeled after Roediger and McDermott.[25] Participants listened (via headphones) to a recording of a list of 15 words; each word was read at a rate of 1 word per 1.5 seconds. They listened to six separate word lists. After each list, they wree asked to write down (on paper) all the words they could remember

from the list. As an example, each list was composed of associates (e.g., bed, rest, awake) of 1 non-presented word/lure (e.g., sleep). If, for example, they wrote down the word "sleep", then this was evidence of constructing a false memory.

After their recall of the 6th list, participants watched an on-line video (The Office Bloopers) for 10-minutes as a distractor task. After this, we assessed their false memory recognition by giving them a piece of paper that has 42 words on it. Of these 42 words, 12 words were words that they studied from one of the previous six lists. However, 30 were non-studied words. Among the 30 non-studied words, 6 were critical words/lures from which the lists were generated (e.g., sleep), 12 were unrelated to any of the items on the list, and 12 were related to the words on the lists (2 per list). The 42 items were subdivided into 6 blocks, with each block consisting of 7 items. Each block included 2 studied words, 2 related words, 2 unrelated words and the critical non-studied word/lure. For each of the 42 items, they were asked to rate the item on a 4-point scale, including the following response options: 4 for sure that they item was old (studied); 3 for probably old, 2 for probably new, and 1 for sure it was new.

Statistical Analysis

All statistical analyses were computed in JASP (v. 0.9.1). The proportion of items classified as Sure Old (a rating of 4), Probably Old (3), Probably New (2) and Sure New (1) were calculated. A 3 (group) x 4 (memory recognition categories) x 4 (word type; studied, unrelated lure, weakly related lure, or a critical lure) ANOVA was employed for the false memory assessment. For the number of words recalled, a 3 (group) by 6

(number of word lists) ANOVA was employed. Statistical significance was set at a nominal alpha of 0.05. Partial eta-squared (η^2_p) was calculated for effect size estimates.

RESULTS

Table 1 displays the demographic and behavioral characteristics of the sample. There were no significant differences in these parameters across the experimental groups.

Table 2 and Figures 1 and 2 display the physiological (heart rate) and psychological (RPE) responses to the experimental conditions. For both heart rate (F(time x group)=132.9, P<.001, η^2_p=.82) and RPE (F(time x group)=81.7, P<.001, η^2_p=.74) there was a significant time by group interaction. In the control group, heart rate remained in the upper 70's and low 80's bpm; in the moderate-intensity exercise group, heart rate increased from 81.7 bpm to 140 bpm; and in the high-intensity exercise group, heart rate increased from 77.6 bpm to 170.2 bpm.

Table 3 and Figure 3 displays the episodic memory recall scores across the experimental groups. For the number of words recalled across each list, there was a significant main effect for list (F(list)=10.2, P<.001, η^2_p=.15), marginally significant main effect for group F(group)=2.7, P=.07, η^2_p=.09), but no list by group interaction effect (F(list x group)=1.00, P=.44, η^2_p=.03). Across the 6 lists, those in the high-intensity exercise group recalled significantly (P<0.05) more words than the control group for List 1, List 2, and List 5. Similarly, the moderate-intensity exercise group recalled significantly more words than the control group for List 2.

The proportion of false word recall is shown in Table 3 and Figure 4. There was not a significant main effect for list (F(list)=2.15, P=.06, η^2_p=.04), group (F(group)=2.20, P=.12, η^2_p=.07) or list by group interaction (F(list x group)=1.27, P=.24, η^2_p=.04).

Table 4 and Figure 5 displays the memory recognition results. Word type refers to whether it was a studied, unrelated lure, weakly related lure, or a critical lure. Recall type refers to whether it was responded as sure old, probably old, probably new, or sure new. There was no main effect for word type (F=.85, P=0.46, η^2_p=.01), group (F=.85, P=.43, η^2_p=.03), word type by group interaction (F=.97, P=.44, η^2_p=.03), recall by group interaction (F=1.03, P=.41, η^2_p=.04), or word type by recall by group interaction (F=1.13, P=.32, η^2_p=.04). However, there was a main effect for recall (F=64.3, P<.001, η^2_p=.54) and a word type by recall interaction (F=182.6, P<.001, η^2_p=.77). That is, participants, across all three experimental conditions, were more likely to perceive the studied and critical lure words as being "old" (i.e., that they previously were exposed to them during the study session).

DISCUSSION

This study evaluated the effects of acute exercise on episodic memory and false memory. In alignment with previous studies,[26,27] with regard to acute exercise and episodic memory, the present experiment demonstrates that acute exercise improves episodic memory. Specifically, the high-intensity exercise group recalled significantly more words than the control group for three out of the six lists (e.g., Lists 1, 2, and 5). Similarly, the moderate-intensity exercise group recalled significantly more words than the control group for one out of the six lists (e.g., List 2). These results display that acute exercise is optimal for enhancing episodic memory, and may occur in an intensity-specific fashion. This aligns with our other recent work suggesting that, for episodic memory, high-intensity exercise may be more beneficial than lower-intensity exercise.[13] Regarding the false word recall, there was not a significant main effect for list, group, or list by group interaction, suggesting that exercise may have a less pronounced effect on false memory recall, when compared to true episodic memory. The memory recognition results showed a significant main effect for recall and a word type by recall interaction. Participants from all three experimental conditions were more likely to perceive the studied and critical lure words as being "old" (i.e., that they previously were exposed to them during the study session).

Similar to the present experiment, other work by Green et al.[27] and Siddiqui et al.[26] also modeled their false memory task after Roediger and McDermott. The high false memory rate was explained by exposure to the semantically related words. Green et al.[27] hypothesized that exposure to the semantically related words may have caused activation

of the related lure words, making participants think that the words were previously stated during the encoding task. This explanation could be applied to the results that were found in the present study, as our current experiment also demonstrated a relatively high false memory rate. Taken together, our employed false memory paradigm was robust in inducing false memories.

Limited research has examined the effects of exercise on false memory recall. However, several of our past experimental studies have provided insight into the potential effects of acute exercise on false memory. Siddiqui et al. investigated the time course effects of acute exercise on false episodic memory. They demonstrated that acute exercise prior to the memory task may be optimal in enhancing episodic memory, which is the procedure that the present study utilized. Even though Siddiqui et al.[26] found no statistically significant results, their study presented evidence to suggest that acute exercise may reduce false memory production. That is, both exercise conditions (before or during encoding) had lower false memory scores when compared to the control condition. Siddiqui et al.[26] expounded their results with two suggestions. Firstly, they suggest that exercise prior to memory encoding may have a priming effect on the neurons, helping prepare them for integration into the memory trace, and overall, creating an optimal environment for true episodic memories. Secondly, they suggest that moderate-intensity exercise both before and during memory encoding may positively affect executive functioning, an important factor for reducing false memories. Follow-up work by Green et al.[27] explored the effects of acute exercise on prospective memory and false memory. In the context of their false memory recall results, there was some suggestive evidence

that acute exercise reduced the production of false memories. Although the present experiment did not provide strong evidence of a consistent relationship between exercise intensity and false memory recall, our findings provide some suggestive evidence that higher-intensity exercise may actually increase the likelihood of false memories. This is in contrast to the findings of Green et al.[27] and Siddiqui et al.[26] that employed a moderate-intensity exercise protocols. These potential intensity-dependent effects may be a result of the effect that acute exercise intensity has on true episodic memory. Higher-intensity acute exercise is more effective in enhancing true episodic memory, and given that our employed false memory paradigm (Roediger and McDermott) involves a critical lure word that is highly semantically related to the studied words, it is plausible that higher-intensity exercise may actually increase the likelihood of false memories, when compared to lower-intensity exercise. Per the fuzzy trace theory,[28] when a memory is encoded, two memory traces are formed, including a verbatim trace and a gist trace, with the latter more likely to decay over time. Speculatively, higher-intensity exercise may be more effective in stabilizing both traces, given the role of higher-intensity exercise on synaptic plasticity. Further, given the role of the prefrontal cortex in inhibiting false memories,[5] it is possible that moderate-intensity exercise, which activates the prefrontal cortex,[29] may help reduce false memories, whereas high-intensity exercise, which reduces prefrontal cortex activity,[30] may accentuate false memories. These assertions align with the accumulating body of research on this topic, including the past two studies by Green et al. and Siddiqui et al., along with the present study's findings.

In addition to false memory, future work should also continue to evaluate whether there is an intensity-specific effect of acute exercise on true episodic memory. As stated, our present findings suggest that higher-intensity exercise is more beneficial for enhancing true episodic memories. This findings aligns with the conclusions of our recent systematic review on this topic.[13] However, as noted in our systematic review, very few studies have directly compared different exercise intensities within the same study. The present experiment bridges this gap in the literature by directly comparing control, moderate-intensity and high-intensity exercise protocols. We have discussed these intensity-specific mechanisms in detail elsewhere,[2,31] which, in brief, including intensity-specific effects on long-term potentiation. Future work should continue to evaluate whether there is a potential intensity-specific effect of acute exercise on false memories.

In conclusion, this study examined the effects of acute exercise on episodic memory and false memory. We did not observe a strong, consistent association between acute exercise and false word recall; however, we did observe evidence to suggest that acute exercise, particularly high-intensity exercise, can improve true episodic memory. Future work on this novel line of inquiry should aim to overcome the limitations of our present experiment. For example, such work should employ a more heterogeneous, representative sample, as well as utilize a within-subject design.

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Table 1. Demographic and behavioral characteristics of the sample.

Variable	Control (N=20)	Moderate-Intensity (N=20)	High-Intensity (N=20)	P-Value
Age, mean years	20.5 (1.1)	20.8 (1.1)	21.1 (0.9)	0.31
Gender, % Female	80.0	95.0	95.0	0.19
Race-Ethnicity, % White	70.0	70.0	80.0	0.88
BMI, mean kg/m^2	27.5 (6.2)	25.0 (5.8)	25.7 (6.2)	0.42
MVPA, mean min/week	131.9 (116.1)	103.3 (91.1)	153.4 (106.5)	0.33
Affect, mean				
Positive	28.2 (7.0)	29.0 (7.2)	27.5 (7.9)	0.80
Negative	12.3 (2.4)	13.4 (3.6)	13.0 (2.7)	0.50

BMI, Body mass index
MVPA, Moderate-to-vigorous physical activity
Values in parentheses are standard deviations
P-value is calculated from a one-way ANOVA (continuous variables) or chi-square analysis (categorical variables)

Table 2. Physiological (heart rate) and psychological (perceived exertion) responses.

Variable	**Control**	**Moderate-Intensity**	**High-Intensity**	**P-Value**
Heart Rate, mean bpm				
Rest	79.9 (12.6)	81.7 (11.7)	77.6 (18.3)	F(3,171; time)=466.9, P<.001, η^2_p=.89 F(2,57; group)=81.6, P<.001, η^2_p=.74 F(6,171; time x group)=132.9, P<.001, η^2_p=.82
Midpoint	80.8 (11.7)	138.8 (12.7)	149.9 (19.6)	
Endpoint	79.8 (11.2)	140.1 (7.2)	170.2 (13.1)	
5-Minutes Post	82.2 (10.9)	91.2 (13.8)	97.0 (14.3)	
RPE, mean				
Rest	6.3 (0.9)	6.0 (0.0)	6.0 (0.0)	F(3,171; time)=320.9, P<.001, η^2_p=.85 F(2,57; group)=35.4, P<.001, η^2_p=.55 F(6,171; time x group)=81.7, P<.001, η^2_p=.74
Midpoint	6.6 (1.1)	11.1 (1.4)	11.4 (1.7)	
Endpoint	6.9 (2.4)	11.4 (1.3)	14.3 (1.6)	
5-Minutes Post	6.9 (2.2)	6.5 (0.8)	7.1 (1.1)	

Table 3. Episodic memory recall scores.

Variable	Control	Moderate-Intensity	High-Intensity	P-Value
True Word Recall				F(5,285; list)=10.2, P<.001, η^2_p=.15 F(2,57; group)=2.7, P=.07, η^2_p=.09 F(10,285; list x group)=1.00, P=.44, η^2_p=.03
List 1, mean # words	6.4 (1.2)	6.8 (1.4)	7.4 (1.7) *	
List 2, mean # words	6.7 (1.2)	6.9 (1.7) †	8.2 (1.6) **	
List 3, mean # words	8.0 (1.9)	8.2 (2.7)	8.5 (1.6)	
List 4, mean # words	6.4 (2.0)	7.3 (2.1)	6.9 (1.4)	
List 5, mean # words	7.0 (1.5)	7.8 (2.2)	8.6 (1.7) **	
List 6, mean # words	7.8 (2.0)	7.9 (1.7)	8.5 (2.2)	
False Word Recall				F(5,285; list)=2.15, P=.06, η^2_p=.04 F(2,57; group)=2.20, P=.12, η^2_p=.07 F(10,285; list x group)=1.27, P=.24, η^2_p=.04
List 1, % false recall	35.0 (48.9)	30.0 (47.0)	25.0 (44.4)	
List 2, % false recall	50.0 (51.3)	55.0 (51.0)	60.0 (50.3)	
List 3, % false recall	30.0 (47.0)	50.0 (51.3)	50.0 (51.3)	
List 4, % false recall	35.0 (48.9)	45.0 (51.0)	70.0 (47.0) *	
List 5, % false recall	45.0 (51.0)	45.0 (51.0)	50.0 (51.3)	
List 6, % false recall	20.0 (41.0)	60.0 (50.3) †	65.0 (48.9) **	

* High-intensity different (P<0.05) than control
** High-intensity different (P<0.01) than control

† Moderate-intensity different (P<0.05) than control

Table 4. Memory recognition scores.

Word Type	Recall	Group	Mean	SD
Studied	Sure Old	Control	69.456	15.403
		Moderate-Intensity	77.920	14.113
		High-Intensity	76.700	12.267
	Probably Old	Control	12.956	11.163
		Moderate-Intensity	12.915	11.930
		High-Intensity	10.820	9.405
	Probably New	Control	9.261	12.086
		Moderate-Intensity	5.835	7.215
		High-Intensity	6.735	5.027
	Sure New	Control	8.328	7.571
		Moderate-Intensity	3.330	5.673
		High-Intensity	4.995	8.284
Unrelated Lure	Sure Old	Control	3.239	5.819
		Moderate-Intensity	2.495	4.757
		High-Intensity	1.665	3.417
	Probably Old	Control	5.089	6.485
		Moderate-Intensity	7.920	10.289
		High-Intensity	8.750	9.928
	Probably New	Control	34.722	26.546
		Moderate-Intensity	35.410	17.502
		High-Intensity	37.085	26.423
	Sure New	Control	56.944	26.555
		Moderate-Intensity	54.170	21.552
		High-Intensity	52.500	29.878
Weakly Related Lure	Sure Old	Control	19.917	10.366
		Moderate-Intensity	17.905	10.905
		High-Intensity	17.075	9.922
	Probably Old	Control	14.806	13.266
		Moderate-Intensity	20.430	8.334
		High-Intensity	23.330	14.700
	Probably New	Control	26.394	21.252
		Moderate-Intensity	38.715	18.635
		High-Intensity	30.420	16.933
	Sure New	Control	38.883	27.421
		Moderate-Intensity	22.920	18.109
		High-Intensity	29.170	22.541
Critical Lure	Sure Old	Control	70.361	25.276
		Moderate-Intensity	81.650	19.426
		High-Intensity	74.985	19.870
	Probably Old	Control	14.778	17.957

Table 4. Memory recognition scores.

Word Type	Recall	Group	Mean	SD
		Moderate-Intensity	10.835	15.550
		High-Intensity	12.510	16.110
	Probably New	Control	5.556	11.428
		Moderate-Intensity	6.675	9.977
		High-Intensity	5.835	11.177
	Sure New	Control	9.267	14.262
		Moderate-Intensity	0.835	3.734
		High-Intensity	6.670	11.341

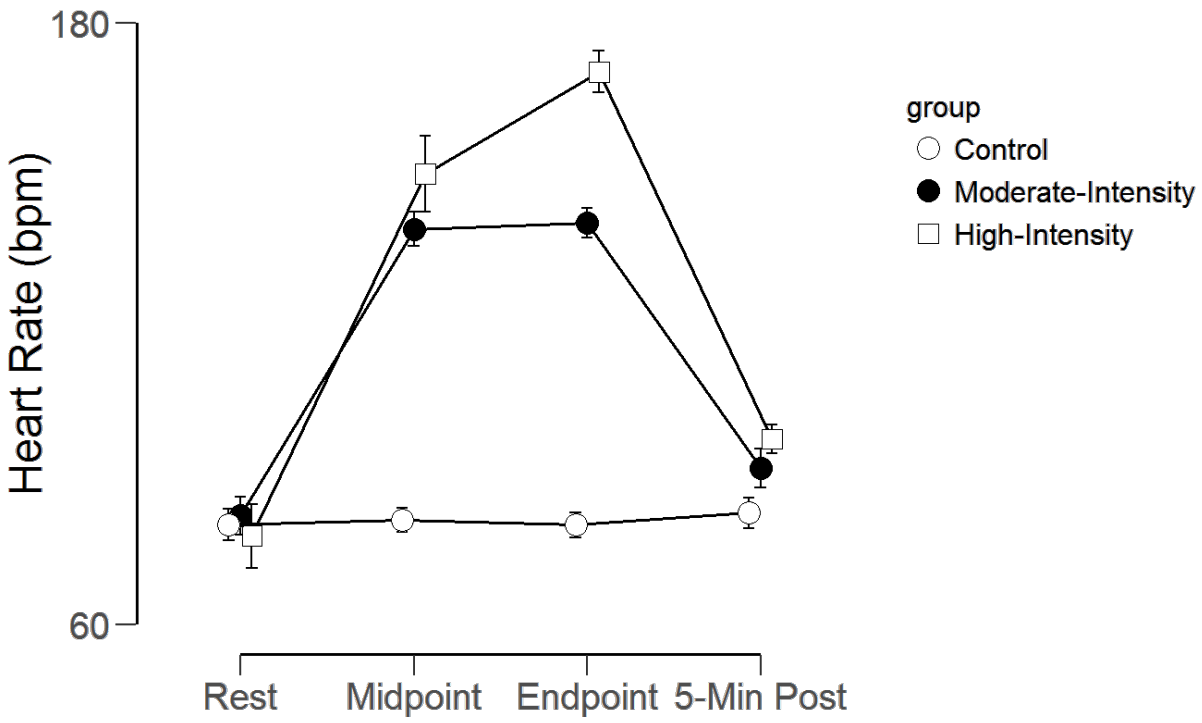


Figure 1. Heart rate responses across the four time points.

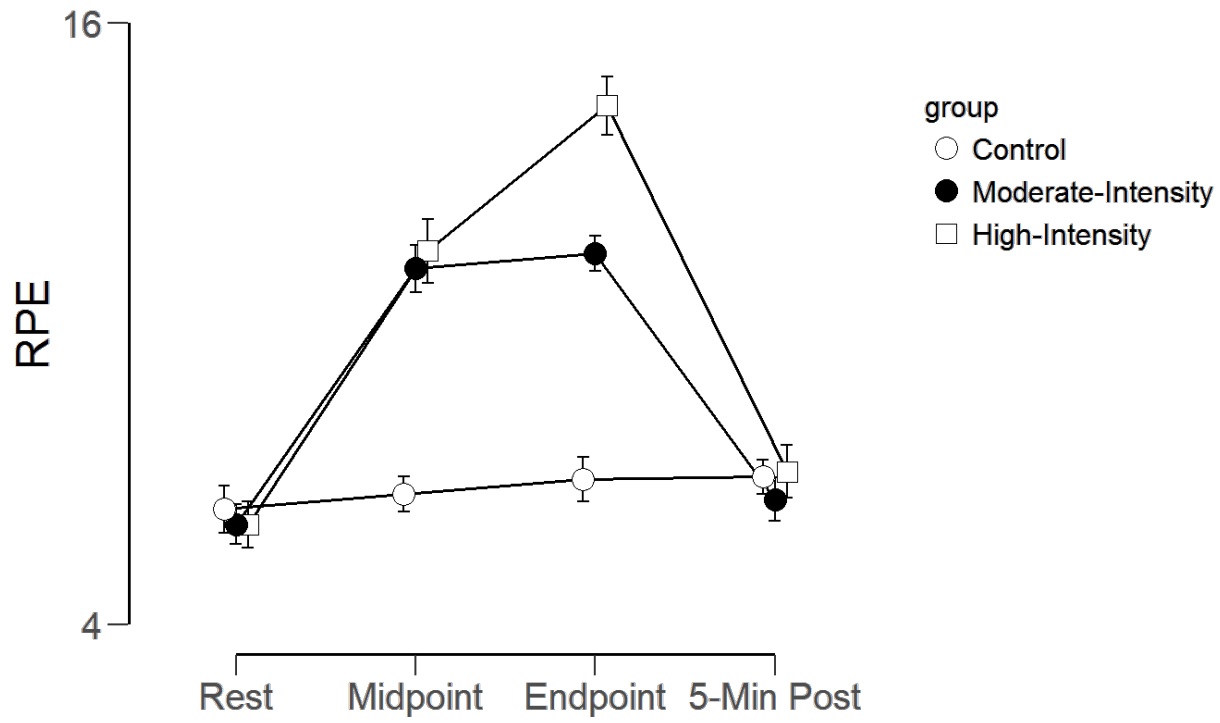


Figure 2. RPE responses across the four time points.

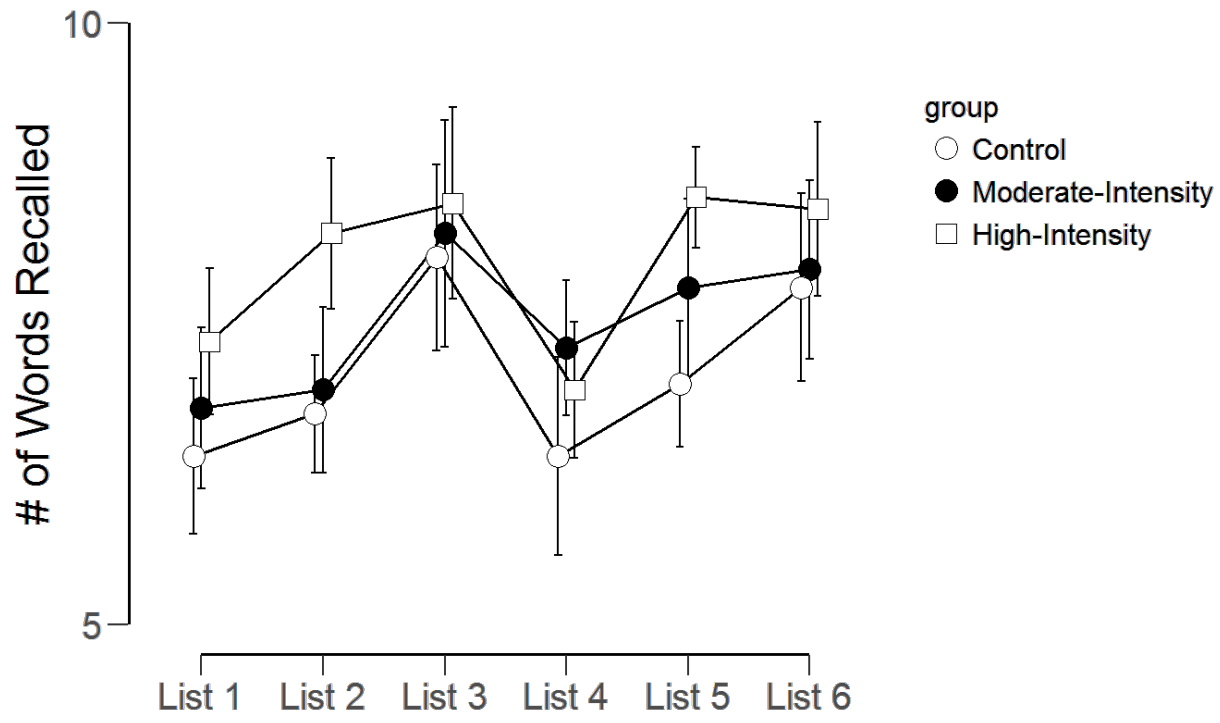


Figure 3. Number of words recalled across the 6 memory lists.

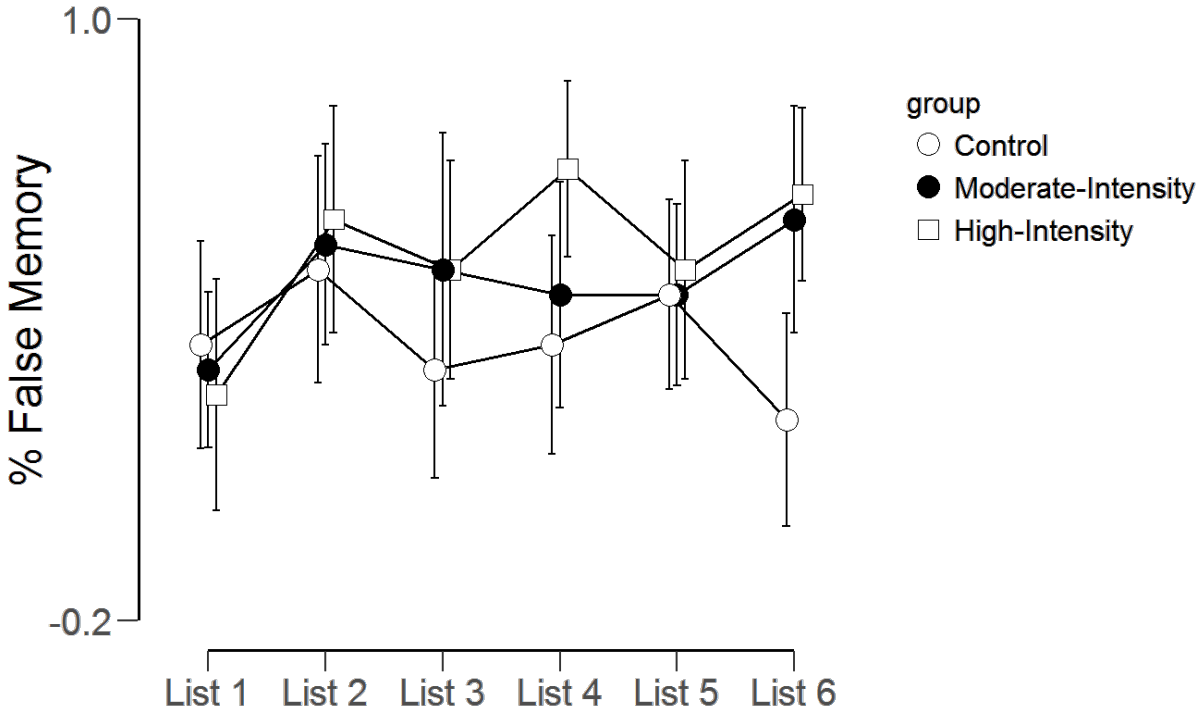
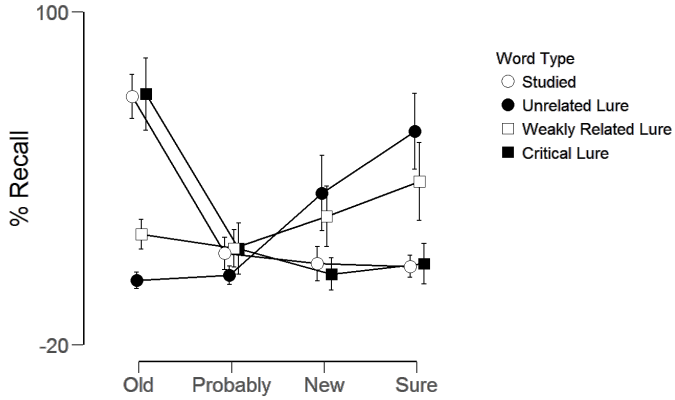
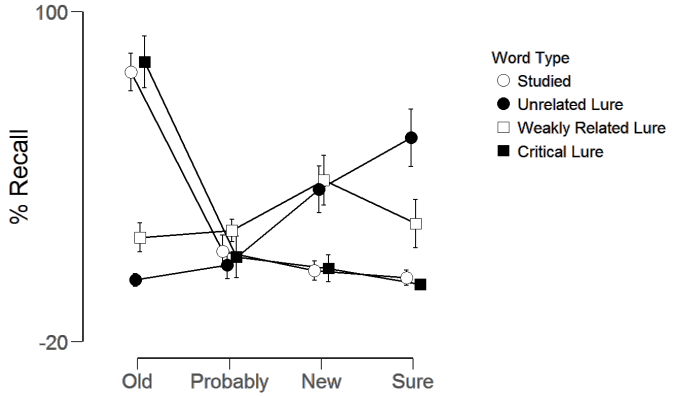


Figure 4. Proportion of false memories across the 6 lists.

group: Control



group: Moderate-Intensity



group: High-Intensity

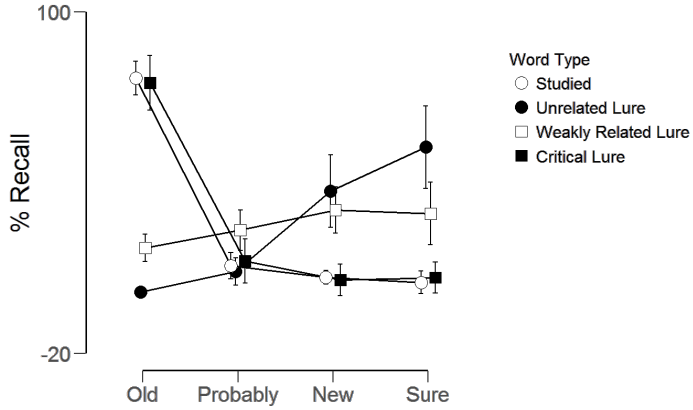


Figure 5. Percent recall across word type and for each experimental condition.

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