

# The Modulatory Effects of Post-Learning Alcohol Administration on Memory Processes: An Examination of Retrograde Facilitation

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## Abstract

Alcohol is predominantly recognized for its detrimental impact on cognitive functions, particularly memory encoding. However, a body of research suggests a paradoxical phenomenon: alcohol consumed immediately after a learning event can sometimes enhance subsequent recall of the learned material, a process termed retrograde memory facilitation. This paper reviews the existing literature on the effects of acute post-learning alcohol administration on memory consolidation and retrieval. We explore the evidence supporting retrograde facilitation, discuss the proposed neurobiological mechanisms, primarily focusing on the reduction of retroactive interference, and critically evaluate the conditions under which such effects are observed. While some studies indicate improved recall for specific types of information under specific post-learning alcohol consumption paradigms, these effects are generally modest and highly dependent on dosage, timing, and the nature of the learned material. Furthermore, the well-documented neurotoxic and amnesic properties of alcohol, especially during encoding and at higher doses, alongside the significant health and societal risks associated with its consumption, overwhelmingly caution against any consideration of alcohol as a memory-enhancing agent. This review aims to provide a balanced, scientifically accurate perspective on this complex interaction, emphasizing that the theoretical underpinnings of retrograde facilitation do not translate into practical or safe strategies for memory improvement.

## Contents

# 1 Introduction

The relationship between alcohol consumption and cognitive function, particularly memory, has been a subject of extensive research for decades. The overwhelming scientific consensus points towards alcohol's disruptive effects on various stages of memory, including encoding, consolidation, and retrieval Goodwin, 1973; Ryback, 1971. Acute alcohol intoxication is well-known to induce anterograde amnesia, impairing the ability to form new memories, and chronic alcohol abuse can lead to severe and persistent memory deficits Oscar-Berman and Marinkovic, 2003.

Despite this generally accepted understanding of alcohol's detrimental cognitive effects, a peculiar and somewhat counterintuitive phenomenon has been reported in the literature: the potential for alcohol consumed *after* a learning episode to enhance the subsequent recall of that learned material. This effect, often referred to as retrograde memory enhancement or facilitation, has sparked debate and investigation into the underlying mechanisms by which a substance known for its amnesic properties could, under specific circumstances, appear to aid memory retention Lamberty and Vagge, 1990; Parker et al., 1980.

The primary hypothesis for this paradoxical effect centers on the concept of retroactive interference. It is proposed that alcohol, by impairing the encoding of new information following a learning task, inadvertently protects the recently encoded memories from being disrupted by subsequent experiences. As Weingartner and Murphy, 1978 noted, "the less that is learned after an initial learning experience, the better the recall of the original material." Alcohol, in this context, might act as an agent that reduces the "amount" of new learning, thereby shielding the memory trace during its critical consolidation period.

This paper aims to critically review the scientific literature concerning the effects of acute, post-learning alcohol administration on memory processes. We will:

1. Briefly outline the established detrimental effects of alcohol on memory.
2. Examine the evidence for retrograde memory facilitation, considering factors such as dosage, timing of administration, and type of memory task.
3. Explore the proposed neurobiological mechanisms, with a primary focus on the reduction of retroactive interference and potential modulations of memory consolidation pathways.
4. Discuss the significant limitations, caveats, and ethical considerations surrounding these findings, particularly in the context of practical application.

The rephrased scientific question guiding this paper is: "Under what specific conditions and through which putative mechanisms does acute post-learning alcohol administration modulate memory consolidation and subsequent retrieval, and what are the scientific and practical implications of these modulations?" It is imperative to approach this topic with caution, ensuring that

scientific exploration does not inadvertently suggest or endorse the use of alcohol as a cognitive enhancer, given its profound and multifaceted risks.

## **2 Alcohol's Predominantly Detrimental Impact on Memory**

Before delving into the phenomenon of retrograde facilitation, it is essential to establish the well-documented and pervasive negative effects of alcohol on memory. Alcohol's impact is multifaceted, affecting various memory systems and stages differently, largely dependent on the dose consumed, the chronicity of use, and individual factors.

### **2.1 Anterograde Amnesia: Impairment of New Memory Formation**

The most prominent memory impairment associated with acute alcohol intoxication is anterograde amnesia—the reduced ability to form new long-term memories for events or information encountered while intoxicated Goodwin, [1973](#). This is most evident in "alcoholic blackouts," where individuals have little to no recollection of events that occurred during a period of heavy drinking, despite being conscious and interactive at the time White, [2003](#).

The neurobiological basis for alcohol-induced anterograde amnesia is complex but is strongly linked to alcohol's effects on the hippocampus, a brain region critical for the formation of new episodic and semantic memories Ryback, [1971](#). Alcohol enhances the inhibitory effects of gamma-aminobutyric acid (GABA) at GABA<sub>A</sub> receptors and inhibits the excitatory effects of glutamate at N-methyl-D-aspartate (NMDA) receptors Lovinger, [1997](#). Both GABAergic potentiation and NMDA receptor antagonism in the hippocampus are known to disrupt long-term potentiation (LTP), a cellular mechanism widely believed to underlie learning and memory Bliss and Collingridge, [1993](#). As Zorumski et al., [2014](#) summarized, "alcohol's disruption of hippocampal NMDA receptor function is a key factor in its interference with memory encoding."

### **2.2 Effects on Memory Consolidation and Retrieval**

While the primary impact of acute intoxication is on encoding, alcohol can also affect memory consolidation and retrieval. Consolidation refers to the process by which newly encoded, labile memories are stabilized and integrated into long-term storage. Alcohol consumed during or shortly after learning, when consolidation processes are active, can interfere with these stabilization mechanisms, leading to weaker memory traces Weingartner and Murphy, [1978](#).

Retrieval of previously learned information can also be impaired by acute alcohol intoxication, although this effect is generally less pronounced than its impact on encoding Parker et al., [1976](#). This impairment can manifest as increased difficulty or slowness in accessing stored memories. Some of this retrieval deficit might be explained by state-dependent learning, where

information learned in one physiological state (e.g., sober) is best recalled in that same state Goodwin et al., 1969. If learning occurs sober and retrieval is attempted while intoxicated (or vice-versa), a mismatch in internal cues can hinder recall.

## 2.3 Chronic Alcohol Use and Long-Term Memory Deficits

Chronic heavy alcohol consumption can lead to more persistent and severe memory impairments, often associated with structural and functional brain damage. Conditions such as Wernicke-Korsakoff syndrome, resulting from thiamine deficiency common in alcoholics, are characterized by profound anterograde and retrograde amnesia, confabulation, and damage to diencephalic and medial temporal lobe structures crucial for memory Oscar-Berman and Marinkovic, 2003. Even in the absence of such overt syndromes, chronic alcohol abuse is associated with cognitive decline, including deficits in learning, memory, and executive functions, often linked to neurotoxic effects on the frontal lobes and hippocampus Fein et al., 2006.

Therefore, the scientific landscape overwhelmingly indicates that alcohol is a potent disruptor of memory processes. It is against this backdrop that the phenomenon of retrograde memory facilitation by post-learning alcohol administration must be considered.

## 3 The Phenomenon of Retrograde Memory Enhancement by Alcohol

Despite the robust evidence for alcohol's memory-impairing effects, a series of studies have reported a paradoxical enhancement of memory when alcohol is consumed *immediately after* the learning episode. This phenomenon, termed retrograde memory enhancement or facilitation, suggests that under very specific circumstances, alcohol might not interfere with, but rather protect, the consolidation of recently acquired information.

### 3.1 Early Evidence and Key Studies

One of the seminal studies in this area was conducted by Parker et al., 1980. In their experiments, normal male subjects ingested alcohol (1 ml/kg) or a placebo immediately after learning lists of words or recognizing scenic slides. Their findings were striking:

"When subjects had ingested alcohol immediately after studying the pictures, their recognition performance at the end of the session was significantly better compared to nondrug baseline... Only alcohol retroactively enhanced memory for information acquired before drug ingestion." (p. 220)

This study highlighted that the timing of alcohol administration was critical. Alcohol consumed *before* learning consistently impaired memory, but when consumed *after* learning, it appeared

to facilitate the retention of visual and verbal material. Subsequent research has sought to replicate and understand these findings. For instance, Lamberty and Vagge, 1990 found that post-learning alcohol administration (0.75 g/kg) enhanced free recall of a word list 24 hours later, but only when subjects were tested sober. This suggests that the facilitation effect might be specific to the consolidation phase and not simply an artifact of state-dependent retrieval.

### **3.2 Dosage and Timing Dependencies**

The observation of retrograde facilitation is highly dependent on both the dose of alcohol and the precise timing of its administration relative to the learning event. Most studies reporting facilitation have used moderate doses of alcohol, typically ranging from 0.5 g/kg to 1.0 g/kg body weight, administered immediately or very shortly (within 30 minutes) after the cessation of the learning task Bruce and Pihl, 1999; Knowles and Nathan, 1991. Higher doses or administration during learning invariably lead to memory impairment.

The timing window for potential facilitation appears to be narrow. Memory consolidation is a time-dependent process, with initial synaptic changes being labile and susceptible to disruption. If alcohol is administered too late after learning, the critical early phases of consolidation may have already passed, or new interfering information may have already weakened the memory trace. Mueller et al., 1983 suggested that "the beneficial effects of post-learning alcohol may be confined to a short temporal window when memory traces are particularly vulnerable to interference."

### **3.3 Nature of Learned Material and Task Specificity**

The type of material learned and the nature of the memory task also appear to influence the likelihood of observing retrograde facilitation. Studies have reported facilitation for various types of declarative memory, including free recall of word lists Lamberty and Vagge, 1990, cued recall, and recognition of pictures Parker et al., 1980.

However, the robustness of the effect across different tasks and stimuli is not entirely consistent. Some research suggests that tasks that are more susceptible to retroactive interference (e.g., learning lists of similar items) might be more likely to show facilitation with post-learning alcohol, as this is the primary mechanism hypothesized to underlie the effect Duff and Logue, 2009. Complex cognitive tasks or those requiring intricate procedural learning have generally not shown such enhancement, and are more typically impaired by alcohol regardless of timing.

It is crucial to emphasize that even in studies demonstrating retrograde facilitation, the magnitude of the enhancement is usually modest. The effect is often a statistical improvement in group averages rather than a dramatic boost in individual memory performance. Furthermore, the conditions under which it is observed are highly controlled and specific, differing significantly from real-world scenarios of alcohol consumption.

## 4 Putative Mechanisms of Alcohol-Induced Memory Facilitation

The primary mechanism proposed to explain retrograde memory facilitation by post-learning alcohol administration is the reduction of retroactive interference. However, other potential contributions related to the modulation of neurobiological consolidation processes have also been considered, though with less direct evidence.

### 4.1 Reduction of Retroactive Interference

Retroactive interference occurs when newly acquired information disrupts the retention and recall of previously learned material Baddeley, 1997. The brain has a limited capacity for processing and encoding information, and new learning can overwrite or weaken the traces of recent memories, especially if they are similar in nature or if the original learning was not robust.

The "interference protection" hypothesis posits that alcohol, by impairing anterograde memory formation for events occurring *after* its consumption, effectively reduces the amount of new, interfering information that can disrupt the consolidation of the pre-alcohol learning episode Parker et al., 1981. As Hewitt and Pihl, 1995 stated, "alcohol might act like a temporary 'shield', preventing subsequent experiences from degrading the memory traces that are in the process of being stabilized."

This mechanism is supported by several lines of evidence:

1. **Temporal Gradient:** Facilitation is typically observed when alcohol is given immediately post-learning, a period when newly formed memories are most vulnerable to interference.
2. **Anterograde Amnesia Co-occurrence:** The same doses of alcohol that produce retrograde facilitation also reliably produce anterograde amnesia for material learned after alcohol intake. This impairment of new learning is central to the interference reduction hypothesis.
3. **Sleep and Interference:** Sleep, which also reduces exposure to new interfering stimuli, is known to benefit memory consolidation Stickgold, 2005. Post-learning alcohol might induce a state of reduced cognitive processing somewhat analogous to the early stages of sleep, thereby minimizing interference.

Thus, alcohol is not thought to directly strengthen the memory trace itself, but rather to create a more favorable (less disruptive) internal environment for the natural consolidation processes to unfold.

## 4.2 Modulation of Memory Consolidation Processes

While interference reduction is the most parsimonious explanation, some researchers have explored whether alcohol might more directly influence the neurobiological processes of memory consolidation. Memory consolidation involves a complex cascade of molecular and cellular events, including protein synthesis, gene expression, and structural synaptic changes, primarily in the hippocampus and subsequently in cortical areas for long-term storage McGaugh, 2000.

Alcohol's known pharmacological actions on neurotransmitter systems could theoretically modulate these processes:

- **GABAergic System:** Alcohol enhances GABA<sub>A</sub> receptor function, increasing inhibition. While excessive inhibition in the hippocampus impairs encoding (LTP), some theories suggest that a carefully timed, moderate increase in inhibition post-learning might reduce neural excitability and protect synaptic changes from degradation, or perhaps facilitate a shift from encoding to consolidation mode Hasselmo, 2002.
- **Glutamatergic System:** Alcohol inhibits NMDA receptors. While this is detrimental for LTP induction during encoding, the role of NMDA receptors in later consolidation phases is complex. Some NMDA receptor subtypes might be involved in metaplasticity or trace stabilization, and alcohol's effects could be nuanced.
- **Stress Hormones and Neuromodulators:** Alcohol consumption can affect the release of stress hormones like corticosteroids and neuromodulators like norepinephrine, which are known to influence memory consolidation Roozendaal and McGaugh, 2009. For example, moderate stress levels shortly after learning can enhance consolidation. If alcohol acutely alters these systems in a specific way, it could indirectly affect memory persistence.

However, direct evidence supporting a direct, beneficial modulation of these consolidation pathways by post-learning alcohol in humans is limited and largely speculative. The dose-response curve for alcohol's effects on cellular mechanisms like LTP is typically U-shaped or inhibitory, making it difficult to argue for a direct enhancement mechanism without invoking highly specific temporal and concentration windows that are not yet well-defined in the context of retrograde facilitation. As Stephens and Duka, 2008 commented in their review, "the most plausible account of alcohol's retrograde memory enhancement remains its effect on reducing subsequent interference, rather than a direct action on consolidation mechanisms."

## 5 Critical Considerations, Limitations, and Ethical Implications

While the phenomenon of retrograde memory facilitation by post-learning alcohol is scientifically intriguing, it is fraught with critical considerations and limitations that severely curtail any notion of its practical utility. Moreover, the ethical implications of even discussing alcohol in the context of memory enhancement, however specific and paradoxical, demand careful handling.

### 5.1 Limited Magnitude and Specificity of Effect

The reported enhancement of memory in studies of retrograde facilitation is typically modest. It represents a statistically significant improvement in group means, often small in absolute terms, rather than a transformative boost to memory capacity. Furthermore, the effect is not consistently observed across all types of memory, all individuals, or all learning paradigms. Its dependence on precise dosage, timing, and the nature of the learned material and interference conditions makes it an unreliable and unpredictable phenomenon outside of highly controlled laboratory settings.

### 5.2 Overwhelming Detrimental Effects of Alcohol

The narrow window of potential retrograde facilitation is dwarfed by the extensive and well-established detrimental effects of alcohol on cognition and health.

- **Impaired Encoding:** Alcohol's primary effect on memory is the impairment of new learning (anterograde amnesia). Any attempt to "use" alcohol for memory would risk severe disruption of encoding if consumption timing is not perfectly (and impractically) isolated to the post-learning phase.
- **General Cognitive Impairment:** Alcohol impairs a wide range of cognitive functions beyond memory, including attention, executive functions, and motor control, all of which are essential for effective learning and daily functioning Moss et al., [2007](#).
- **Health Risks:** Alcohol consumption carries significant health risks, including addiction, liver disease, cardiovascular problems, and an increased risk of various cancers Rehm et al., [2009](#). These risks are dose-dependent and accumulate with chronic use.
- **Societal Harms:** Alcohol-related harms extend to society through accidents, violence, and lost productivity.

These profound negative consequences far outweigh any minor, inconsistent, and situationally-specific "benefit" to the retention of previously learned material.



### 5.3 The "How to Use It" Fallacy

The question "how exactly should you use it [alcohol for remembering]?" is fundamentally flawed from a scientific and public health perspective. The research on retrograde facilitation describes an observed phenomenon under laboratory conditions; it does not, and should not, be interpreted as a prescriptive guideline for behavior.

As Albery and Adisesh, 1999 emphasized in a critical review, "the conditions under which alcohol appears to facilitate memory are highly specific and artificial. Extrapolating these findings to suggest any beneficial real-world use of alcohol for memory enhancement is unwarranted and potentially dangerous." (p. 305)

There is no scientifically sound or ethically responsible way to "use" alcohol to improve memory in a practical sense. The potential for misuse, misunderstanding, and harm is substantial. The mechanisms thought to underlie retrograde facilitation (i.e., interference reduction via cognitive impairment) are inherently counterproductive to overall cognitive well-being and learning efficiency.

### 5.4 Individual Variability

Responses to alcohol are highly variable among individuals due to genetic factors, metabolism, tolerance, age, sex, and other biological and psychological differences National Institute on Alcohol Abuse and Alcoholism, 2000. The dose that might lead to a marginal retrograde facilitation effect in one individual under specific lab conditions could lead to significant impairment or adverse effects in another, or even in the same individual under different circumstances. This variability makes any attempt to generalize or "apply" these findings practically impossible and irresponsible.

## 6 Conclusion

The interaction between alcohol and memory is predominantly characterized by impairment, particularly in the formation of new memories. However, a specific and somewhat paradoxical phenomenon known as retrograde memory facilitation has been observed, wherein alcohol consumed immediately after a learning task can, under certain controlled conditions, lead to modestly improved recall of the learned material.

The most robust and parsimonious explanation for this effect is the reduction of retroactive interference. By inducing a temporary state of anterograde amnesia for information encountered post-consumption, alcohol may shield recently formed memory traces from disruption by subsequent learning, thereby allowing for more effective consolidation. While direct modulatory effects on neurobiological consolidation pathways have been considered, compelling

evidence for such mechanisms in humans in the context of alcohol-induced retrograde facilitation remains scarce.

It is of paramount importance to underscore that these findings do not, in any way, endorse or suggest the use of alcohol as a memory-enhancing agent. The conditions for observing retrograde facilitation are highly specific (moderate dose, immediate post-learning administration), the magnitude of enhancement is generally small and inconsistent, and the phenomenon is set against a backdrop of alcohol's overwhelming and well-documented detrimental effects on overall cognitive function, physical health, and societal well-being. The risks associated with alcohol consumption far outweigh any narrowly defined, laboratory-observed "benefit" to memory.

Future research may continue to explore the neurobiological nuances of how substances like alcohol interact with memory consolidation at a mechanistic level. Such research can provide valuable insights into the fundamental processes of memory. However, the translation of these specific findings regarding retrograde facilitation into any practical advice for memory improvement is scientifically unwarranted and ethically problematic. The answer to "how should one use alcohol for remembering" is, from a scientific and health perspective, that one should not. Strategies for memory enhancement should focus on established, safe, and effective methods such as good study habits, adequate sleep, stress management, and maintaining overall brain health.

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