Alcohol Effects on Memory (non-blackout)

Alcohol is a central nervous system depressant. From the very first drink it can create confusion, memory loss, impaired judgement, behavioral changes, cognitive impairment and reduced inhibitions. Alcohol has serious effects with regard to a number of societal ills, including domestic violence, car accidents, and risky behaviors. For this paper, our focus is on its potential effects for sexual offense and true or false allegations. Approximately half of sexual assault allegations are associated with alcohol use by the accused, victim, or both. (Abbey, et al., 2001) and up to 75% of acquaintance rape allegations involve alcohol (Crowell & Burgess, 1996). Appendix A provides a number of research abstracts about alcohol and sexual disinhibition.

Notice that the negative effects of alcohol can occur without either party entering blackout. Before we take a deeper dive into blackouts, let examine how alcohol in general can compromise memory for events, crimes, and estimation of consumption.

Compromise of General Memory:

"Alcohol primarily interferes with the ability to form new long-term memories, leaving intact preciously established long-term memories and the ability to keep new information active in memory for brief periods. As the amount of alcohol consumed increases, so does the magnitude of the memory impairments." (White, 2003, p. 186)

Alcohol, and the effects of alcohol on memory and general cognitive functioning, has been the subject of much research over the years. Alcohol acts as a central nervous system depressant, but it affects some areas of the brain more than others. Specifically, it leads to distraction and inattention and significantly inhibits neuronal activity in the hippocampus, which impairs memory encoding since the hippocampus plays an important role in the formation of new declarative memories. Because procedural memory functions more or less automatically, retrieval of procedural memory ("remembering how") is not as severely impaired by alcohol as retrieval of declarative memory ("remembering what").

Alcohol particularly impairs the encoding of episodic memory (that part of declarative memory that relates to our personal experiences and specific events in time), especially for certain types of recall, such as cued recall, the recognition of completed word fragments and free recall. A "blackout" is an example of a difficulty in the encoding of episodic memories due to high doses of alcohol. It is caused by a rapid increase in blood alcohol concentration, which in turn distorts the activity of neurons in the hippocampus, thus impairing person's ability to form new episodic memories. Alcohol also severely disrupts the encoding and storage process of new semantic memories (our memory of facts, meanings and acquired knowledge about the external world), although apparently not that of previously learned information.

Alcohol also impairs short-term (working) memory, although mainly by affecting certain mnemonic strategies and executive processes rather than by shrinking the basic holding capacity of working memory or by physically altering the structure of those parts of the brain which are critical for working memory function. Although light-to-moderate drinking does not appear to impair long-term cognitive functioning significantly (and according to some studies, may actually decrease the risk of cognitive decline), heavy drinking and chronic alcoholism is associated with long-term impairment in sustained attention and working memory function, especially visual working memory.

Serious over-consumption of alcohol, especially in comparison with the intake of other foods, can cause a thiamine deficiency, leading to a much more serious form of amnesia known as Korsakoff's syndrome. The Human Memory website: http://www.human-memory.net/disorders_alcohol.html

How Alcohol Impacts the Brain

"If recreational drugs were tools, alcohol would be a sledgehammer. Few cognitive functions or behaviors escape the impact of alcohol, a fact that has been recognized in the literature. As Fleming stated nearly 70 years ago, 'the striking and inescapable impression one gets from a review of acute alcoholic intoxication is of the almost infinite diversity of symptoms that may ensue from the action of this single toxic agent' (1935, pp. 94-95). In addition to impairing balance, motor coordination, decision making, and a litany of other functions, alcohol produces detectable memory impairment beginning after just one or two drinks. As the dose increases, so does the magnitude of the memory impairments." (White, 2003, p. 186)

- Hippocampus
  - Important for the formation of long-term memories by transferring sensory data into short- and long-term memory. (Atkinson and Shiffrin, 1968, Modal Model of Memory, White 2003, p.9)
  - Alcohol disrupts the function of the hippocampus. (Ryback 1971, Parker 1976; White 2003, p. 10-15)
  - The recorder is turned off.
- Proteins and Receptors
  - *"Substantial evidence now indicates that alcohol selectively alters the activity of specific complexes of proteins embedded in the membranes of cells (i.e., receptors) that bind neurotransmitters such as gamma–aminobutyric acid (GABA), glutamate, serotonin, acetylcholine, and glycine (for a review, see Little 1999). In some cases, only a few amino acids appear to distinguish receptors that are sensitive to alcohol from those that are not (Peoples and Stewart 2000). It remains unclear exactly how alcohol interacts with receptors to alter their activity."* (White, 2003, p.10)
- Medial Septum
  - *One brain region that is central to hippocampal functioning is a small structure in the fore brain known as the medial septum (Givens et al. 2000). The medial septum sends rhythmic excitatory and inhibitory signals to the hippocampus, causing rhythmic changes in the activity of hippocampal pyramidal cells. . . Given the powerful influence that the medial septum has on information processing in the hippocampus, the impact of alcohol on cellular activity in the medial septum is likely to play an important role in the effects of alcohol on memory. Indeed, in rats, putting alcohol directly into the medial septum alone produces memory impairments* (White, 2003, p. 14)
- Prefrontal Cortex
  - First glass of wine disrupts the prefrontal cortex.
  - Important for executive functioning – reasoning, planning, decision-making, assessment and interpretation of events.
  - Disinhibits primitive brain impulse.
  - "*The Horse and Rider*" analogy
- Amygdala
  - Attaches emotional status to a stimulus.
  - Reduces fear and anxiety.
  - Alcohol calms the chatter from the prefrontal cortex to the amygdala.
- "*Reward Center*" (Incentive Salience Circuitry)
  - Alcohol activates the "*Reward Center*"
  - Primarily dopamine, which creates a feeling of euphoria
- Some drink to reduce stress, others drink for pleasure. Quick drinkers are looking for the reward.

EFFECTS OF ALCOHOL ON MEMORY (White, 2003)

To evaluate the effects of alcohol, or any other drug, on memory, one must first identify a model of memory formation and storage to use as a reference. One classic, often–cited model, initially proposed by Atkinson and Shiffrin (1968), posits that memory formation and storage take place in several stages, proceeding from sensory memory (which lasts up to a few seconds) to short–term memory (which lasts from seconds to minutes depending upon whether the information is rehearsed) to long–term storage. This model often is referred to as the modal model of memory, as it captures key elements of several other major models. Indeed, elements of this model still can be seen in virtually all models of memory formation.

In the modal model of memory, when one attends to sensory information, it is transferred from a sensory memory store to short–term memory. The likelihood that information will be transferred from short–term to long–term storage, or be encoded into long–term memory, was once thought to depend primarily on how long the person keeps the information active in short–term memory via rehearsal. Although rehearsal clearly influences the transfer of information into long–term storage, it is important to note that other factors, such as the depth of processing (i.e., the level of true understanding and manipulation of the information), attention, motivation, and arousal also play important roles (Craik and Lockhart 1972; Otten et al. 2001; Eichenbaum 2002).1 (1 It is well beyond the scope of this review to assess the impact of alcohol on memory utilizing multiple perspectives on information processing and storage. For simplicity, this review will characterize the effects of alcohol on memory using a three–stage process of memory formation akin to the modal model. The interpretation of the effects of alcohol on memory likely would vary somewhat depending on the memory model one uses.)

Variability in the use of terms, particularly in operational definitions of short–term memory, makes it difficult to formulate a simple synopsis of the literature on alcohol–induced memory impairments. As Mello (1973) stated three decades ago with regard to the memory literature in general, “The inconsistent use of descriptive terms has been a recurrent source of confusion in the ‘short–term’ memory literature and ‘short–term’ memory has been variously defined as 5 seconds, 5 minutes, and 30 minutes” (p. 333). In spite of this inconsistency, several conclusions can be drawn from research on alcohol–induced memory impairments. One conclusion is that the impact of alcohol on the formation of new long–term “explicit” memories—that is, memories of facts (e.g., names and phone numbers) and events—is far greater than the drug’s impact on the ability to recall previously established memories or to hold new information in short–term memory (Lister et al. 1991). (See figure 1 for a diagram depicting the stages of memory and where alcohol interferes with memory.) Intoxicated subjects are typically able to repeat new information immediately after its presentation and often can keep it active in short–term storage for up to a few minutes if they are not distracted (for an early review, see Ryback 1971), though this is not always the case (Nordby et al. 1999). Similarly, subjects normally are capable of retrieving information placed in long–term storage prior to acute intoxication. In contrast, alcohol impairs the ability to store information across delays longer than a few seconds if subjects are distracted between the time they are given the new information and the time they are tested. In a classic study, Parker and colleagues (1976) reported that when intoxicated subjects were presented with “paired associates”—for example, the letter “B” paired with the month

“January”—they were impaired when asked to recall the items after delays of a minute or more. However, subjects could recall paired associates that they had learned before becoming intoxicated. More recently, Acheson and colleagues (1998) observed that intoxicated subjects

could recall items on word lists immediately after the lists were presented but were impaired when asked to recall the items 20 minutes later.

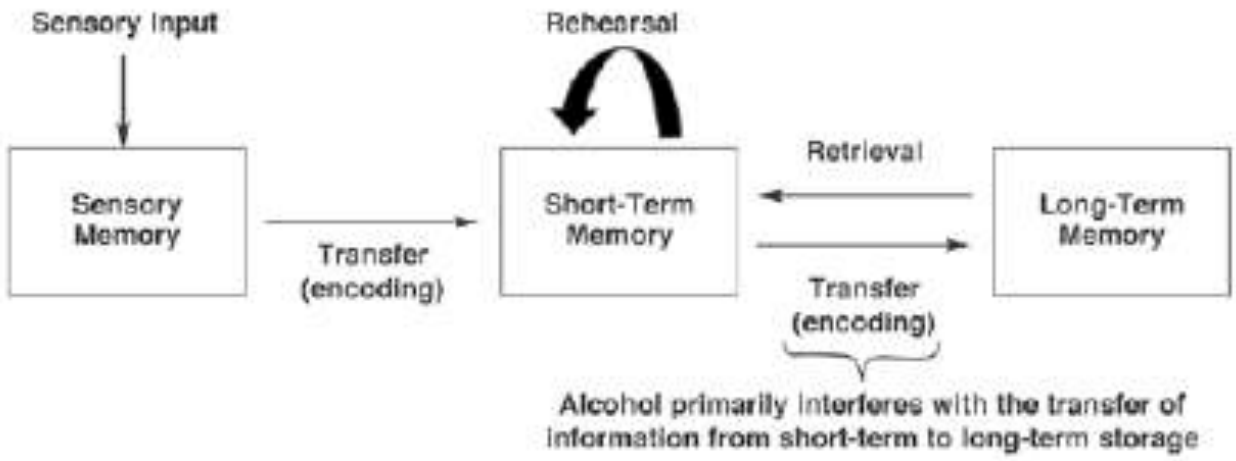


Figure 1 A general model of memory formation, storage, and retrieval based on the modal model of memory originally proposed by Atkinson and Shiffrin (1968). Alcohol seems to influence most stages of the process to some degree, but its primary effect appears to be on the transfer of information from short–term to long–term storage. Intoxicated subjects are typically able to recall information immediately after it is presented and even keep it active in short–term memory for 1 minute or more if they are not distracted. Subjects also are normally able to recall long–term memories formed before they became intoxicated; however, beginning with just one or two drinks, subjects begin to show impairments in the ability to transfer information into long–term storage. Under some circumstances, alcohol can impact this process so severely that, once sober again, subjects are unable to recall critical elements of events, or even entire events, that occurred while they were intoxicated. These impairments are known as blackouts.

Ryback (1971) characterized the impact of alcohol on memory formation as a dose–related continuum, with minor impairments at one end and large impairments at the other, all impairments representing the same fundamental deficit in the ability to transfer new information from short–term to long–term storage. When doses of alcohol are small to moderate (producing blood alcohol concentrations [BACs] below 0.15 percent), memory impairments tend to be small to moderate as well. At these levels, alcohol produces what Ryback (1971) referred to as cocktail party memory deficits, lapses in memory that people might experience after having a few drinks at a cocktail party, often manifested as problems remembering what another person said or where they were in conversation. Several studies have revealed that alcohol at such levels causes difficulty forming memories for items on word lists or learning to recognize new faces (Westrick et al. 1988; Mintzer and Griffiths 2002). As the dose increases, the resulting memory impairments can become much more profound, sometimes culminating in blackouts—periods for which a person is unable to remember critical elements of events, or even entire events, that occurred while he or she was intoxicated.

Aaron M. White, *What Happened? Alcohol, Memory Blackouts, and the Brain* (2003).
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