Alcohol and the Brain (non-blackout)

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. 1)

- 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.

Research Abstracts on Alcohol's Impact on the Brain

Altered anterior cingulate neurochemistry in emerging adult binge drinkers with a history of alcohol-induced blackouts.

Silveri, Marisa M., et al.

Alcoholism: Clinical and Experimental Research, Vol 38(4), Apr, 2014. pp. 969-979.

Abstract:

Background: Binge alcohol consumption is associated with multiple neurobiological consequences, including altered neurophysiology, brain structure, and functional activation. Magnetic resonance spectroscopy (MRS) studies have demonstrated neurochemical alterations in the frontal lobe of alcohol users, although most studies focused on older, alcohol-dependent subjects. Methods: In this study, neurochemical data were acquired using MRS at 4.0 Tesla from emerging adults (18 to 24 years old) who were binge alcohol drinkers (BD, n = 23) or light drinkers (LD, n = 31). Since binge drinking is also associated with increased prevalence of experiencing an alcohol-induced blackout, BD were stratified into alcohol-induced blackout (BDBO) and non-blackout (BDN) groups. Results: Overall, BD had significantly lower gamma amino-butyric acid (GABA) and N-acetyl-aspartate (NAA) in the anterior cingulate cortex (ACC) than LD. When stratified by blackout history, BDBO also had lower ACC glutamate (Glu) than LD. No group differences in MRS metabolites were observed in the parietal-occipital cortex. Lower ACC GABA and Glu remained significant after accounting for lower gray matter content in BD, however, NAA differences were no longer evident. In addition, low ACC GABA levels were associated with greater alcohol use consequences, and worse response inhibition and attention/mental flexibility in BD. Conclusions: These data indicate that binge drinking affects frontal lobe neurochemistry, more so in those who had experienced an alcohol-induced blackout. Characterization of the neurochemical profiles associated with binge alcohol consumption and blackout history may help identify unique risk factors for the later manifestation of alcohol abuse and dependence, in young individuals who are heavy, frequent drinkers, but who do not meet the criteria for alcohol abuse disorders.

Alcohol use disorders and the brain.

Rao, Rahul

Addiction, Vol 115(8), Aug, 2020. pp. 1580-1589.

Abstract:

A diagnosis of alcohol use disorder is associated with a higher risk of dementia, but a dose–response relationship between alcohol intake consumption and cognitive impairment remains unclear. Alcohol is associated with a range of effects on the central nervous system at different doses and acts on a number of receptors. Acute disorders include Wernicke's encephalopathy (WE), traumatic brain injury, blackouts, seizures, stroke and hepatic encephalopathy. The most common manifestations of chronic alcohol consumption are Korsakoff's syndrome (KS) and alcohol-related dementia (ARD). There is limited evidence for benefit from memantine in the treatment of ARD, but stronger evidence for the use of high-dose parenteral thiamine in the progression of neuropsychiatric symptoms for WE. Accumulating evidence exists for pharmacological treatment in the prevention of hepatic encephalopathy. Rehabilitation of people with ARD may take several years, and requires an approach that addresses physical and psychosocial factors.