Alcohol and Its Effect on Gabba-Amino-Butyric Acid

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Abstract

Today, alcohol use is widely accepted, and sometimes even encouraged. Thus, the consequences of its intake, such as a change in the brain's idea of what "normal" functions and reception should look like, is not always considered. Through literature review via Web of Science, Academic Search Complete, and Google Scholar, this poster examines the impact alcohol has on gamma-amino-butyric acid (GABA), a neurotransmitter that contributes to crucial everyday functions in mammals, as well as its receptors, for both are heavily affected by alcohol use. Once the "normal" functions of GABA and its receptors are established, how alcohol disrupts this function is examined. Then, there is further assessment on how GABA levels and receptors evolve with prolonged alcohol use. Lastly, the reasoning behind the occurrence of tolerance and withdrawal symptoms are explored. The overall results demonstrate that GABA is an inhibitory neurotransmitter that can provide a sedative effect when in contact with a receptor. Likewise, alcohol can bind to these receptors and have a similar effect as GABA does, resulting in behaviors that people most commonly associate with alcohol use. With prolonged use, the brain must adjust to a continuous input of alcohol, so there is a decrease in the level of GABA and a change in receptor expression. This leads to a tolerance that not only requires more alcohol for the same effect, but that also forces the brain to reestablish homeostasis. Consequently, when someone whose brain has undergone this change quits drinking, withdrawal symptoms occur. All in all, understanding the consequences of increased and prolonged alcohol intake is essential in bringing awareness to the serious, and often overlooked, risks of alcohol use.

Introduction

GABA is an inhibitory neurotransmitter on which alcohol has a great effect. Responsible for sedative effects, GABA promotes hyperpolarization of the neurons via attachment to its receptor. Once hyperpolarization occurs, an inhibitory postsynaptic potential is created, resulting in sedation. A mimetic of GABA, alcohol can also bind to the GABA receptors and cause the same effect to occur. How both the receptors and the GABA levels are influenced by alcohol will be explored in order to discover how they and the user are affected by prolonged alcohol use.

Methods

In researching this topic, keywords such as "GABA," "alcohol," "alcohol abuse," and "GABA receptors" were used on the databases Academic Search Complete, Google Scholar, and Web of Science in order to find credible literature reviews and experimental research.

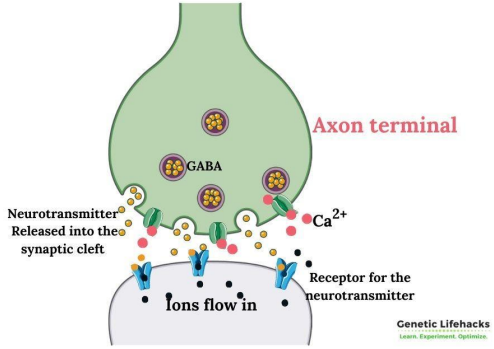

Figure 1. The image gives an overview of the mechanisms by which GABA and its receptors perform in order to act in an inhibitory manner.

Results

- When alcohol binds to type A receptors, a channel opens and allows chloride into the cell.
- The entrance of chloride into the cells results in inhibition.
- Studies show that GABA levels decrease in cases of abuse and that abusers tend to have a lower average GABA level.
- GABA receptors become less sensitive to GABA with prolonged alcohol use, and their numbers decrease.
- The change in the GABA receptors results in tolerance.
- When the brain becomes of deprived of alcohol after periods of abuse, it becomes overexcited due to the physiological changes that it made to maintain homeostasis during the abuse period.

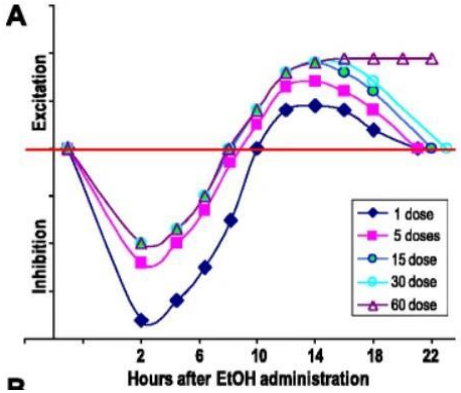

Figure 2. The graph above demonstrates the behavior of rats after being given certain doses of alcohol. With the red line representing equilibrium, it is seen that prior to the full absorption of alcohol, the rats are in a depressed state; however, the intensity of this depressed state varies depending on the rats' tolerance to alcohol. The amount of hyperexcitability and the duration of hyperexcitability increases with higher doses, representing withdrawal states.

Discussion/Future Work

Long-term testing should be done in individuals with alcohol use disorder to fully understand how GABA levels affect other neurotransmitters (ex. Glutamine) and the user.

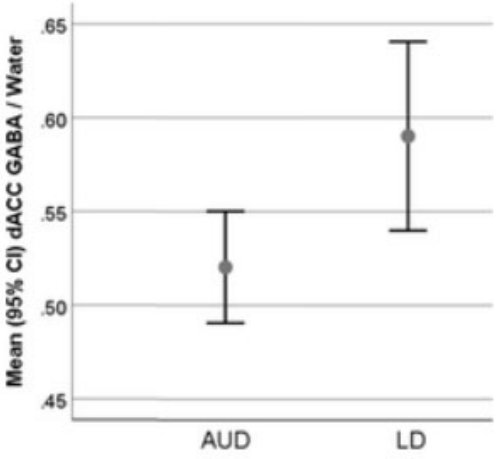

Figure 3. The following graph depicts both the mean GABA levels and the ranges of GABA in those with alcohol use disorder (AUD) and light drinkers (LD). The data supports that alcohol abuse results in an overall decrease in GABA levels.

Conclusion

Ultimately, the literature suggests that there are physiological changes in the brain that occur when alcohol use turns into abuse. These changes are the result of the brain attempting to maintain homeostasis, and include adjusting the amount of GABA levels and the number of receptors.

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