



## PEER-REVIEW REPORT

**Name of journal:** *World Journal of Experimental Medicine*

**Manuscript NO:** 100402

**Title:** Alcohol and Alcoholism associated Neurological disorders: Current updates in a global perspective and recent recommendations

**Provenance and peer review:** Invited Manuscript; Externally peer reviewed

**Peer-review model:** Single blind

**Reviewer's code:** 08354567

**Position:** Peer Reviewer

**Academic degree:** MD

**Professional title:** Doctor

**Reviewer's Country/Territory:** China

**Author's Country/Territory:** India

**Manuscript submission date:** 2024-08-15

**Reviewer chosen by:** Jia-Lin Zhang

**Reviewer accepted review:** 2024-10-29 09:16

**Reviewer performed review:** 2024-11-07 12:46

**Review time:** 9 Days and 3 Hours

<b>Scientific quality</b>	<input type="checkbox"/> Grade A: Excellent <input checked="" type="checkbox"/> Grade B: Very good <input type="checkbox"/> Grade C: Good <input type="checkbox"/> Grade D: Fair <input type="checkbox"/> Grade E: Do not publish
<b>Novelty of this manuscript</b>	<input type="checkbox"/> Grade A: Excellent <input checked="" type="checkbox"/> Grade B: Good <input type="checkbox"/> Grade C: Fair <input type="checkbox"/> Grade D: No novelty
<b>Creativity or innovation of this manuscript</b>	<input type="checkbox"/> Grade A: Excellent <input checked="" type="checkbox"/> Grade B: Good <input type="checkbox"/> Grade C: Fair <input type="checkbox"/> Grade D: No creativity or innovation



<b>Scientific significance of the conclusion in this manuscript</b>	<input type="checkbox"/> Grade A: Excellent <input checked="" type="checkbox"/> Grade B: Good <input type="checkbox"/> Grade C: Fair <input type="checkbox"/> Grade D: No scientific significance
<b>Language quality</b>	<input type="checkbox"/> Grade A: Priority publishing <input checked="" type="checkbox"/> Grade B: Minor language polishing <input type="checkbox"/> Grade C: A great deal of language polishing <input type="checkbox"/> Grade D: Rejection
<b>Conclusion</b>	<input type="checkbox"/> Accept (High priority) <input checked="" type="checkbox"/> Accept (General priority) <input type="checkbox"/> Minor revision <input type="checkbox"/> Major revision <input type="checkbox"/> Rejection
<b>Re-review</b>	<input checked="" type="checkbox"/> Yes <input type="checkbox"/> No
<b>Peer-reviewer statements</b>	Peer-Review: <input type="checkbox"/> Anonymous <input checked="" type="checkbox"/> Onymous
	Conflicts-of-Interest: <input type="checkbox"/> Yes <input checked="" type="checkbox"/> No

**SPECIFIC COMMENTS TO AUTHORS**

The paper provided a detailed overview of the neurobiology of alcohol addiction, followed by recent studies published in the genetics of alcohol addiction, molecular mechanism and detailed information on the various acute and chronic neurological manifestations of alcoholism for the Future research. The article is well written. I have some suggestions for this paper. 1) Alcoholism appears to be a complex genetic disorder, with variations in many genes influencing risk. Some of these genes have been identified, including two alcohol metabolism genes, ADH1B and ALDH2, which have the most potent known effects on the risk of alcoholism. The author should provide more references to reveal the related mechanisms. 2) Signs that might be battling with alcohol dependence include: Unit of alcohol, Hazardous drinking, Alcohol dependence, Alcohol Tolerance, Reverse tolerance to alcohol, Alcohol WithdrawalAlcohol abuse, Alcohol Addiction. The author should provide a brief description of these concepts. 3) Brain plasticity events contribute to the development of AUD and result in cravings and habitual alcohol-seeking behavior. Furthermore, chronic or high-dose alcohol intake causes adverse or adaptive reactions in the central nervous system (CNS) as well as in



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nearly every organ system. The author should focus on the relationship between alcoholism and the nervous system, provide more information. 4 ) Serotonin is an inhibitory neurotransmitter produced by neurons in the raphe nuclei. It is also known as 5-hydroxytryptamine or 5-HT. Reduced serotonin neurotransmission has been linked to higher alcohol use and susceptibility to alcoholism. There is an increase in extracellular 5-HT levels after acute alcohol intake. Chronic alcohol consumption, on the other hand, causes a general decrease in 5-HT neurotransmission, as demonstrated by reduced levels of 5-hydroxyindoleacetic acid (5-HIAA), the major metabolite of 5-HT, in heavy drinkers' cerebrospinal fluid. This decrease in extracellular 5-HT in the context of chronic alcohol exposure could be attributed to either increased reuptake of 5-HT from the extracellular space via the serotonin transporter (5-HTT) or defective 5-HT release in the raphe nuclei. The author should provide more references to reveal the related mechanisms.