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Commentary

Activating Effects of Alcohol on Brain Mitochondria and Synaptosomes: Apparent Alcoholic Apathy

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DESCRIPTION

Exalted effects of alcohol have been demonstrated in emanating the perplexed responsiveness seen in the society during the modern days which lead to entrapped morphological, behavioural and biochemical changes significantly noted in the article in a precise and systematic manner [1,2]. The profound effects of alcohol impart various anomalies like muscular dystrophy and cerebellar degeneration. Also, behavioural functions like cognitive decline and memory-based learning responses have been evaluated with significant changes after chronic alcohol exposure. Under the experimental laboratory conditions wild type strain of zebrafish of mixed gender (4 months old) has been chosen as an experimental animal due to an ease in handling and percent genome similarity with that of humans [3]. To induce alcohol generated harmful accumulation in the brain, two consecutive treatments viz; 0.20% and 0.40% for 22 days as an intermittent exposure depicted the significant reduction in ATPase activity that led to disturbances in energy metabolism of brain mitochondria, oxidative stress in terms of Lipid Peroxidation (LPO), Protein Carbonylation (PC) and differential modulation in antioxidants like Glutathione (GSH), Superoxide Dismutase (SOD) and Catalase (CAT) has been the marker for oxidative stress-induced degeneration in the mitochondria and synaptosomes.

Swimming pattern and anxiety analysis

The swimming behavior pattern and way of swimming entails motor characteristics of the zebrafish in the behavioural apparatus [4]. Alterations in the swimming pattern and deviation from the control pattern reveal alcohol-induced anxiety as noted by measuring the nearest neighbour distance and the total area occupied in all the experimental groups. In light of the fact that the brain is far more susceptible to ethanol than other organs, the behavioural reactions that we observed are very certainly attributable to alcohol's influence on the brain.

In another behavioural test, the level of anxiety was noted after the two consecutive doses of alcohol. The novel tank diving test (geotaxis test) has been carried out in a trapezoidal tank that confirmed the stress among the zebrafish under treatment conditions as they spent maximum time at the bottom of the tank which was plausible and natural to anticipate that persistent ethanol therapy may have a neurotoxic effect.

Mitochondrial and synaptosomal impairment

Mitochondria play an essential role in ATP formation and different metabolic processes occurring in the cell like redox homeostasis. Dysfunction of mitochondria results in decreased ATP production and increased imbalance in the equilibrium of reactive Oxygen Species (ROS) and Reactive Nitrogen Species (RNS) leading to degenerative changes in the brain [5]. Apart from mitochondria, synaptosomes play a crucial role in the translocation of ions, maintenance of membrane potential and release of various neurotransmitters in a Ca^{2+} dependent manner. These are functionally active distinct structures between the neurons that participate in cognitive-related functions of the brain [6]. The effects of lipid peroxidation on membrane lipids, membrane receptors and membrane-bound enzymes can affect membrane function, shape and fluidity, as well as perturbations in ion flux. The augmentation of the toxicological damage and a cascade reaction by the ethanol treatment elevates the level of lipid peroxidation and protein carbonylation in zebrafish model. Reactive Oxygen Species (ROS) can be generated by a variety of xenobiotics or toxins, both naturally occurring and manmade in origin. Excessive ROS generation is often a result of oxidative stress, which disrupts the organism's natural redox balance. These reactive species, such as superoxide, nitric oxide and lipid peroxyl radicals are structurally unstable and highly reactive due to one or more unpaired electrons. ROS mediated damage to the mitochondria and synaptosomes leads to the disruption in membrane and produce peroxides of the lipids causing damage to the functioning of biomolecules. Cellular based efficient antioxidants like superoxide dismutase, catalase and GSH were used during the investigation as a biomarker for indicating antioxidant balance in the cells after chronic alcohol-induced cellular degeneration in mitochondria and synaptosomal fractions of the brain [7]. Furthermore, ATPase activity revealed

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notable differences in chronic toxicity that acts as an indicator for deregulated energy metabolism in brain mitochondrial and synaptosomal fractions.

CONCLUSION

This study may assist in a better understanding of the process of synaptosome and mitochondrial damage in the brain of zebrafish at the transcriptional and translational level. In addition, impaired mitochondrial function and cell redox imbalance in the central nervous system may play a vital role in neuronal damage caused by the addictive substance alcohol. Despite the need for more research, our findings helped to elucidate the core mechanism of persistent alcoholism and its possible implications on behavioural activities. Thus, our findings bring together the body of research and the impacts of alcohol, highlight a critical aspect that should be studied in detail, and should demonstrate the applicability of zebrafish as a translational model for the study of complex human disorders.

CONFLICTS OF INTEREST

None

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