Use of Ultra-High Field Magnetic Resonance Imaging to Evaluate the Effects of Zinc and Ethanol in Traumatic Brain Injury

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Introduction

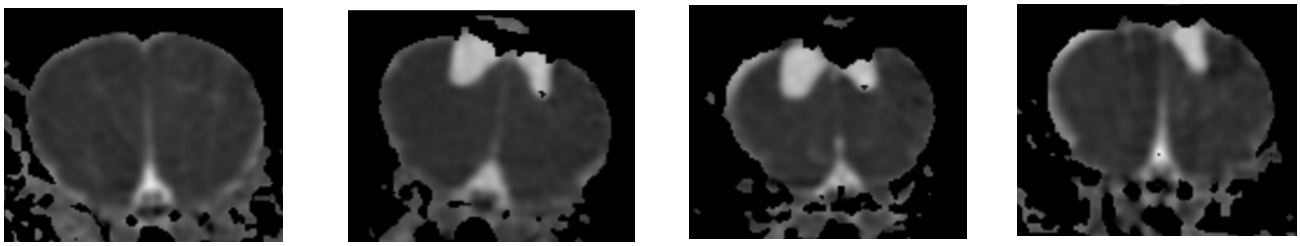
In addition to the high prevalence of traumatic brain injury (TBI), among National Guard and Reserve troops deployed with combat exposure, there is an increased risk for new-onset heavy weekly drinking, binge drinking, and alcohol-related problems[1]. Chronic alcoholism has also been linked to deficiencies of zinc[2] and there is clear evidence that TBI puts patients at significant risk for zinc deficiency[3]. Recently published data from our lab using an animal model of moderate TBI has shown that the essential trace element zinc can be used to reduce many of the poor outcomes associated with brain injury including learning and memory deficits and depression[4]. The goal of this work was to understand the impact of ethanol in the injured brain and to determine if zinc supplementation can be used as a nutritional therapy to combat the combined effects of TBI and ethanol.

Experimental

Young adult male rats were fed a zinc adequate (30 ppm) or a zinc supplemented (180 ppm) diet for 16-18 days followed by a moderate TBI using the standard controlled cortical impact method routinely employed in our lab. After injury, rats were given 3 g/kg dose of ethanol daily for 7 days via gavage. Control groups included zinc adequate fed animals that were given the injury only and sham (uninjured) animals. Animals were scanned with the 21.1 T MRI scanner using an NHMFL-designed in vivo MRI proton probe.

Results and Discussion

Fig. 1 is a map of water diffusion in which the light areas of the brain correspond to brain swelling from water accumulation (edema). An uninjured animal is shown in Fig. 1A. After a cortical brain injury, edema develops as seen in Fig. 1B. Fig. 1C shows that ethanol intake does not appear to significantly exacerbate cortical edema, However, Fig. 1D shows that when rats are supplemented with zinc, there appears to be a decrease in edema.



(A) Sham (B) TBI (C) TBI + Ethanol (D) ZS TBI + Ethanol

Fig. 1. *In vivo* MRI of water diffusion in the frontal cortex in rats on day 8 post-TBI. (A) Sham (uninjured) control (B) TBI untreated (C) TBI and 7 day ethanol exposure (D) Zinc supplemented animals given both a TBI and exposed to 7 days of ethanol.

Conclusions

Zinc supplementation before brain injury appears to reduce the edema associated with trauma. However, ethanol does not significantly exacerbate the amount of edema at the site of injury. This on-going work is also examining TBI-induced deficits in learning and memory, the development of depression, and anxiety that will enable us to determine the correlation between edema and behavioral outcomes.

Acknowledgements

This work is funded by the U.S. Army MRMC and NIH grant GM081382.

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