Neuroglial Alterations in Rats Submitted to the Okadaic Acid-induced Model of Dementia

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Dementia is characterized as a progressive decline in cognitive functions and severe memory loss. Alzheimer’s disease (AD) is the most common form of dementia and is an age-related neurodegenerative disorder. Several types of animal models have been developed to investigate AD. Okadaic acid (OA), a potent inhibitor of protein phosphatases 1 and 2A, induces characteristics that resemble AD-like pathology. Rats were submitted to bilateral intrahippocampal okadaic acid-injection (100 ng), 12 days after the surgery, biochemical tests were performed. Using this model, we evaluated the metabolism of glutamate and oxidative parameters in hippocampal slices of sham and OA-infused rats. The reduced glutathione content and protein carbonyls were used as parameters to evaluate possible oxidative stress caused by the OA-induced dementia model. The results showed that the infusion of OA did not affect the glutamate uptake (p=0.069). However, there was a significant decrease in the level of EAAT2/GLT-1 (p=0.042) and glutamine synthetase activity (p=0.034) in the rats submitted to OA infusion as compared to the sham group. Moreover, reduced glutathione content was lower in OA infused rats (p=0.039), and a significant increase in the protein carbonyls was observed in the OA group (p=0.027). In conclusion, the OA-induced model of dementia caused oxidative stress and specific astroglial alterations in the hippocampus. Therefore, our findings using the model of OA administration contribute to understanding of the molecular elements altered in neurodegenerative diseases accompanied by cognitive deficits and the neural damage.

Word Keys: Dementia, Animal models, Okadaic acid and Hippocampus.
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