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TAIL SUSPENSION DISRUPTS COGNITION FUNCTION AND DOWN-REGULATES LEARNING-RELATED PROTEIN EXPRESSION IN RAT HIPPOCAMPUS

Abstract

Purpose: As a challenge to astronauts' health, microgravity plays an important role in the etiology of a series of neurological disturbances such as space motion sickness, space adaptation syndrome (SAS) and cognitive disorders. Yet the details of these physiological effects remain unclear. In addition to neuron death, incellular proteins appear to play a role in these processes. For instance, NMDA receptor (NR) has been shown to affect neuronal structure and synaptic plasticity. The present study is aimed at the effects of simulated microgravity on cognition function and learning-related protein expression. Methodology: We employed 7 days tail-suspension to simulate microgravity and investigated the spatial memory capability with Morris water maze test as well as the learning-related protein expression with Western Blotting test. Results: Behavioral analysis of Sprague-Dawley (SD) rats revealed that spatial learning was impaired. Tail-suspended rats exhibited decrease in the expression NR1/2B and phosphorylation of CaMKII and CREB1, which were activated by NR. Additionally, mechano-growth factor (MGF) was downregulated with its downstream Nrf2 mediated neuroprotection of heme oxygenase-1 (HO-1). Consistent with these findings, NR1/2B and MGF pathway in hippocampus displayed significant deficits under simulated weightlessness. Conclusions: Together, these data support a role of NR and MGF as regulator in cognitive change induced by simulated-microgravity and raise the possibility the dysregulation of memory-related protein may contribute to an array of cognitive disorders under simulated-microgravity.
Keywords: Microgravity, Spatial learning, NMDA receptor, Mechano-growth factor Acknowledgments

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