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Phosphodiesterase inhibition rescues chronic cognitive deficits induced by traumatic brain injury

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Traumatic brain injury (TBI) alters several protein kinase signaling pathways in the hippocampus that are critical for hippocampal-dependent memory formation. Previous studies suggest that the cAMP–PKA signaling cascade is downregulated after TBI, and that treatment with a phosphodiesterase IV inhibitor rolipram rescues this decrease in cAMP levels. Thus, we hypothesized that preventing the degradation of cAMP with rolipram may rescue memory deficits induced by TBI. Accordingly, in the present study, we examined the effect of rolipram on TBI-induced cognitive impairments. At 2 weeks after moderate parasagittal fluid-percussion brain injury or sham surgery, adult male Sprague Dawley rats received vehicle (0.15% DMSO in saline) or rolipram (0.03mg/kg, i.p.) 30 minutes prior to water maze acquisition or contextual and cue fear conditioning. The TBI animals treated with rolipram showed a significant improvement in water maze acquisition and cue and contextual fear conditioning as compared to TBI animals treated with vehicle. Furthermore, hippocampal long-term potentiation (LTP) was investigated in area CA1 at 2 weeks after TBI or sham surgery. The expression of LTP in TBI animals was significantly impaired as compared to sham surgery animals and this impairment in TLP was rescued by rolipram treatment. Thus, these results indicate that administration of a rolipram rescues cognitive impairments after TBI by improving hippocampal synaptic plasticity.

Biography

Coleen Atkins, Ph.D. is currently an Assistant Professor at The Miami Project to Cure Paralysis in the Department of Neurological Surgery at the University of Miami Miller School of Medicine. She received her undergraduate degree from the University of Minnesota and her doctorate in Neuroscience at Baylor College of Medicine in Houston, Texas. Dr. Atkins' post-doctoral training was completed at Oregon Health & Sciences University. Dr. Atkins is funded by the NIH to understand how synaptic plasticity mechanisms are altered in the brain after trauma and is developing therapeutic strategies for learning and memory rehabilitation of chronic TBI survivors.

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