Nicotine attenuates spatial learning deficits induced by sodium metavanadate.

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Source

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Abstract

Learning can be severely impaired as a consequence of exposure to environmental pollutants. Vanadium (V), a metalloid which is widely distributed in the environment, has been shown to exert toxic effects on a variety of biological systems including the nervous system. However, studies exploring the impact of vanadium on learning are limited. Herein, we investigated the effects of oral administration of sodium metavanadate (SMV) (١٥, ٢٠ and ٢٥mg/kg/day for ٢weeks) on spatial learning using Morris water maze (MWM). Our results showed that pre-training administration of sodium metavanadate impaired learning in Morris water maze. Analyzing the role of cholinergic system in SMV-induced learning deficit, we found that bilateral intra-hippocampal infusion of nicotine (١μg/side) during training could significantly diminish the SMV-induced learning impairment. We next examined the expression of choline acetyltransferase (ChAT) and vesicular acetylcholine transporter (VAChT) as cholinergic markers in CA١ region of hippocampus as well as in medial septal area (MSA). Our molecular analyses showed that vanadium administration decreased ChAT and VAChT protein expression, an effect that was attenuated by nicotine. Altogether, our results confirmed the toxic effects of SMV on spatial acquisition, while also pointing to the neuroprotective effects of nicotine on SMV-induced impairments in learning capabilities. These findings might open a new avenue for the prevention of vanadium adverse effects on spatial learning and memory through activation of cholinergic signaling pathway.

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