New Findings on Cytokines from Yunnan University Summarized [Cordycepin (3 '-deoxyadenosine) Promotes Remyelination Via Suppression of Neuroinflammation In a Cuprizone-induced Mouse Model of Demyelination].

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Full Text:
2019 NOV 3 (VerticalNews) -- By a News Reporter-Staff News Editor at News of Science -- New research on Intercellular Signaling Peptides and Proteins - Cytokines is the subject of a report. According to news originating from Yunnan, People's Republic of China, by VerticalNews correspondents, research stated, "Multiple sclerosis (MS) is an inflammatory demyelination disease characterized by autoimmune damage to the central nervous system. In this disease, failure of remyelination could cause persistent disability."

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Our news journalists obtained a quote from the research from Yunnan University, "Cordycepin, also known as 3'-deoxyadenosine, exerts anti-inflammatory, anti-oxidic, anti-apoptotic and neuroprotective effects. The cuprizone (CPZ) model has been widely used to study MS as it mimics some characteristics of demyelination disease. To determine whether cordycepin promotes remyelination and functional recovery after CPZ-induced demyelination, we administered cordycepin to the CPZ-induced demyelination mice. Cordycepin reversed CPZ-induced loss of body weight and rescued motor dysfunction in the model mice. Cordycepin effectively promoted remyelination and enhanced MBP expression in the corpus callosum. Cordycepin also inhibited the CPZ-induced increase in the number of Iba1-positive microglia, GFAP-positive astrocytes and Olig2-positive oligodendroglial precursor cells in the corpus callosum and cerebral cortex. Pro-inflammatory cytokine expression (IL-1 beta and IL-6) was inhibited while anti-inflammatory cytokine IL-4 and neurotrophic factor BDNF release was elevated in the corpus callosum and hippocampus after cordycepin treatment. In addition, we also found that cordycepin ameliorated CPZ-induced body weight loss, motor dysfunction, demyelination, glial cells activation and pro-inflammatory cytokine expression in the corpus callosum and hippocampus."

According to the news editors, the research concluded: "Our results suggest that cordycepin may represent a useful therapeutic agent in demyelination-related diseases via suppression of neuroinflammation."

For more information on this research see: Cordycepin (3 '-deoxyadenosine) Promotes Remyelination Via Suppression of Neuroinflammation In a Cuprizone-induced Mouse Model of Demyelination. International Immunopharmacology, 2019;75():. International Immunopharmacology can be contacted at: Elsevier, Radarweg 29, 1043 Nx Amsterdam, Netherlands. (Elsevier - www.elsevier.com; International Immunopharmacology - www.journals.elsevier.com/international-immunopharmacology/)

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