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Cucurbitacin B mitigates experimental autoimmune encephalomyelitis by inhibition of IL-17/IL-23 immune axis

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Pharmacological approaches to inhibit brain acute inflammation may represent important strategies for the control of autoimmune diseases. Multiple sclerosis (MS) is a chronic, inflammatory, demyelinating and autoimmune disease of the central nervous system (CNS). Cucurbitacin B (CuB), an oxygenated tetracyclic triterpenoid compound extracted from Cucurbitaceae plant species, is a bioactive agent by disruption of microtubule polymerization and inhibition of JAK/STAT signaling. However, there has been little information about impact of CuB on MS treatment. In this research, for the first time we examine effects of CuB (specific STAT3 blocker), in experimental autoimmune encephalomyelitis (EAE) mouse model of MS. EAE was induced by subcutaneous immunization of MOG35-55 in 8-week-old C57BL/6 mice. CuB was administered at different doses (0.25, 0.5 and 1 mg/kg body weight/day/i.p) from the first day of the experiment. Inflammatory responses were examined using qRT-PCR, western blot and immunohistochemistry (IHC) analysis of specific markers such as p-STAT3, IL-17A, IL-23A, CD11b and CD45. CuB reduced STAT3 activation, leukocyte trafficking, and also IL-17/IL-23 immune axis in this model. Treated mice with lower doses of CuB exhibited a considerable depletion in the EAE clinical score which correlated with decreased expression of IL-17, IL-23 and infiltration of CD11b+ and CD45+ cells into the CNS. Our *in vivo* results suggest that STAT3 inhibition by CuB will be an effective and new approach for the treatment of neuro-inflammatory disease such as MS.

Recent Publications:

1. Doan V, Kleindienst A M, McMahon E J, Long B R, Matsushima G K and Taylor L C (2013) Abbreviated exposure to cuprizone is sufficient to induce demyelination and oligodendrocyte loss. J Neurosci Res. 91: 363-73.
2. Kastelein R A, Hunter C A and Cua D J (2007) Discovery and biology of IL-23 and IL-27: related but functionally distinct regulators of inflammation. Annu. Rev. Immunol. 25: 221-242.
3. Haines C J et al (2013) Autoimmune memory T helper 17 cell function and expansion are dependent on interleukin-23. Cell Rep. 3: 1378-1388.
4. Ramroodi N, Khani M, Ganjali Z, Javan M R, Sanadgol N, Khalseh R and Abdollahi M (2015) Prophylactic effect of BIO-1211 small-molecule antagonist of VLA-4 in the EAE mouse model of MS. Immunological Investigations. 44: 694-712.

Biography

Nima Sanadgol is expert in field of Cell and Molecular Neurobiology. His recent research emphasis is in treatment of neurodegenerative disease with use of new natural compounds. He has particular interest in evaluation of mechanisms of neuron-glia interactions, in order to fascinating myelin repair and control of neuro-inflammatory and neuro-degenerative diseases (Multiple sclerosis, Alzheimer, Parkinson, etc.). He has already gained so much experience in neuro-immune and circuit-specific signaling in glial-neuron networks (T cell biology, NF-κB, Nrf2, MAP kinase, AMP kinase, apoptosis and autophagy).

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