Astaxanthin

Astaxanthin (ATX) is a carotenoid pigment with pleiotropic pharmacological properties that is seen as a possible drug for Cerebral ischemia treatment and subarachnoid hemorrhage.

Emerging evidence suggests that astaxanthin may also have additional biological activities relating to neurogenesis and synaptic plasticity.

Grimmig et al., investigated the potential for astaxanthin to modulate cognitive function and neuroplasticity in young and aged mice. They showed that feeding astaxanthin to aged mice for 1 month improves performance on several hippocampal-dependent cognitive tasks and increases long-term potentiation. However, they did not observe an alteration in neurogenesis, nor did we observe a change in microglial-associated IBA1 immunostaining. This demonstrates the potential for astaxanthin to modulate neural plasticity and cognitive function in aging [1].

Na+-K+-2Cl- co-transporter-1 (NKCC1), an intrinsic membrane protein expressed by many cell types, is activated by various insults, leading to the formation of cell swelling and brain edema.

Zhang et al., previously established that ATX attenuated brain edema and improved neurological outcomes by modulating NKCC1 expression after traumatic brain injury in mice. This paper explored the molecular mechanism of ATX-mediated inhibition of NKCC1 utilizing an in vitro astrocyte stretch injury model.

RESULTS: Stretch injury in cultured astrocytes lowered cell viability time-dependently, which was substantially reducing by pretreating with ATX (50 μmol/L). Stretch injury increased Bax level and cleaved caspase-3 activity, and decreased Bcl-2 level and pro-caspase 3 activity, resulting in the apoptosis of astrocytes. Additionally, stretch injury substantially raised the gene and protein expressions of interleukin (IL)-1β, IL-6, and tumor necrosis factor (TNF)-α and prompted the expression and nuclear translocation of NF-κB. Pretreatment with ATX remarkably prevented the trauma-induced initiation of NF-κB, expressions of pro-inflammatory cytokines, and cell apoptosis. Moreover, stretch injury markedly elevated the gene and protein expression of NKCC1, which was partly blocked by co-treatment with ATX (50 μmol/L) or an NF-κB inhibitor (PDTC, 10 μmol/L). Cleaved caspase-3 activity was partially reduced by PDTC (10 μmol/L) or an NKCC1 inhibitor (bumetanide, 50 μmol/L).

CONCLUSIONS: ATX attenuates apoptosis after stretch injury in cultured astrocytes by inhibiting NKCC1 expression, and it acts by reducing the expression of NF-κB-mediated pro-inflammatory factors [2].

1)

Grimmig B, Hudson C, Moss L, Peters M, Subbarayan M, Weeber EJ, Bickford PC. Astaxanthin supplementation modulates cognitive function and synaptic plasticity in young and aged mice. Geroscience. 2019 Feb 9. doi: 10.1007/s11357-019-00051-9. [Epub ahead of print] PubMed PMID: 30739297.

2)

Zhang M, Cui Z, Cui H, Wang Y, Zhong C. Astaxanthin protects astrocytes against trauma-induced apoptosis through inhibition of NKCC1 expression via the NF-κB signaling pathway. BMC Neurosci. 2017 May 10;18(1):42. doi: 10.1186/s12868-017-0358-z. PubMed PMID: 28490320; PubMed Central PMCID: PMC5425995.