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Neuroprotective Effects of Antioxidants on Oxidative Stress and Neuronal Injury

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Description

Increased oxidative stress and lipid peroxidation in the brain are linked to aging, which damages neurons. Carotenoids with strong antioxidant qualities, such zeaxanthin and lutein, have been connected to improved cognitive function. Their precise impact on lipid peroxidation-induced neuronal injury is yet unclear, though. The purpose of this study was to investigate the neuroprotective effects of these carotenoids on neuronal damage caused by lipid peroxidation. We investigated the effects of lipid peroxidation on neuronal health using an in vitro oxidative stress model using differentiated neuronal cells generated from human neuroblastoma SH-SY5Y cells. Neuronal damage resulted from the combined administration of rotenone and RSL3, which markedly elevated lipid Reactive Oxygen Species (ROS) and mitochondrial oxidative stress. After a week of administration, lutein and zeaxanthin significantly decreased mitochondrial oxidative stress, inhibited lipid peroxidation and lessened neuronal damage in the cells they treated. Although these findings are encouraging, more investigation is required to elucidate the precise biochemical processes at play.

Cerebral ischemia-reperfusion damage

Nitric oxide: The synthesis of Nitric Oxide (NO) by neuronal Nitric Oxide Synthase (nNOS) contributes negatively to cerebral ischemia-reperfusion damage, which is a major cause of death and disability in ischemic stroke. Peroxynitrite ($ONOO^-$), a hazardous chemical that worsens neuronal damage by nitrating proteins, particularly tyrosine residues, is created when NO combines with superoxide (O_2-). Although its precise function in cerebral ischemia-reperfusion injury is unclear, one protein of interest, SIRT6, is essential in controlling cell division, death and aging.

In a rat model of four-artery cerebral ischemia-reperfusion, we found that higher nitration of SIRT6 decreased its enzymatic activity, exacerbating the damage to hippocampus neurons. Neuronal damage was lessened and SIRT6 activity was increased by lowering nitration. Moreover, SIRT6 nitration exacerbated hippocampal ischemia injury by influencing the activity of downstream enzymes. Neuronal damage was reduced by treatment with receptor antagonist MK801, nNOS inhibitor 7-NI, or the antioxidant resveratrol, which reduced SIRT6 nitration and the activity of these downstream molecules. Tyrosine 257 was also found to be essential for SIRT6 activation and nitration susceptibility. Under oxygen-glucose deprivation circumstances, neurocyte death was reduced when this residue was mutated to phenylalanine. Our knowledge of SIRT6's function in cerebral ischemia disorders is improved by these findings.

Vascular cognitive impairment

Cognitive deficits: One of the main causes of neuronal deterioration and cognitive dysfunction in Vascular Cognitive Impairment (VCI) is oxidative stress. In VCI, Isoamericanin A (ISOA), a naturally occurring lignan with many hydroxyl groups, has demonstrated promise in reducing oxidative stress. The pharmacological effects and antioxidative mechanisms of ISOA in the treatment of VCI were the main focus of this investigation.

For us *in vitro* and *in vivo* tests, we used N2a cells treated with Hydrogen Peroxide (H_2O_2) and a Temporary Bilateral Common Carotid Artery Occlusion (tBCCAO) animal model, respectively. According to behavioral tests, the tBCCAO mouse model's learning, memory and recognition were all markedly enhanced by ISOA therapy (5, 10 mg/kg). By boosting MAP-2 expression, decreasing TUNEL-positive cell density and increasing NeuN-positive cells, ISOA also lessened neuronal injury by safeguarding synapses and neuronal nuclei. In terms of mechanism, ISOA reduced oxidative pharmacological suppression or genetic knockdown of Nrf2 decreased the effects of ISOA on the reduction of superoxide, ROS and MDA levels, as well as the rise in ARE promoter activity and antioxidant enzyme expression. On the other hand, Keap1 knockdown in H_2O_2-treated cells increased these protective effects. These results demonstrate ISOA's potential as a treatment for cognitive deficits brought on by oxidative stress.