Quercetin Exerts Differential Neuroprotective Effects Against H2O2 and A? Aggregates in Hippocampal Neurons: the Role of Mitochondria

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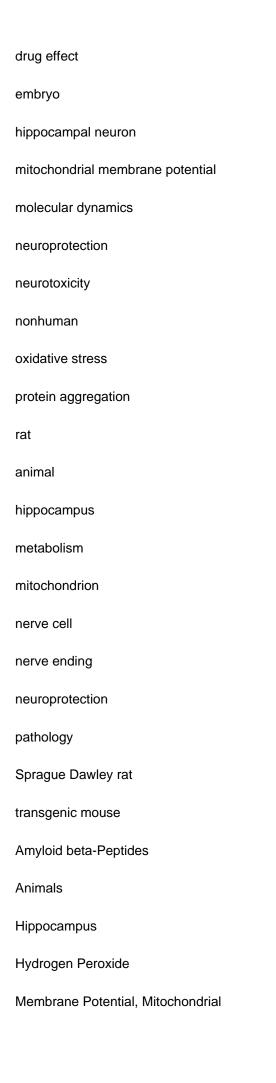
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Amyloid-? peptide (A?) is one of the major players in the pathogenesis of Alzheimer?s disease (AD). Despite numerous studies, the mechanisms by which A? induces neurodegeneration are not completely understood. Oxidative stress is considered a major contributor to the pathogenesis of AD, and accumulating evidence indicates that high levels of reactive oxygen species (ROS) are involved in A?-induced neurodegeneration. Moreover, A? can induce the deregulation of calcium homeostasis, which also affects mitochondrial function and triggers neuronal cell death. In the present study, we analyzed the effects of quercetin, a plant flavonoid with antioxidant properties, on oxidative stress- and A?-induced degeneration. Our results indicate that quercetin efficiently protected against H2O2-induced neuronal toxicity; however, this protection was only partial in rat hippocampal neurons that were treated with A?. Treatment with guercetin decreased ROS levels. recovered the normal morphology of mitochondria, and prevented mitochondrial dysfunction in neurons that were treated with H2O2. By contrast, quercetin treatment partially rescued hippocampal neurons from A?-induced mitochondrial injury. Most importantly, quercetin treatment prevented the toxic effects that are induced by H2O2 in hippocampal neurons and, to a lesser extent, the A?-induced toxicity that is associated with the superoxide anion, which is a precursor of ROS production in mitochondria. Collectively, these results indicate that quercetin exerts differential effects on the prevention of H2O2- and A?-induced neurotoxicity in hippocampal neurons and may be a powerful tool for dissecting the molecular mechanisms underlying A? neurotoxicity. © 2016,

Springer Science+Business Media New York. Alzheimer?s disease Oxidative stress Quercetin Sod?/+ mice ?? aggregates amyloid beta protein hydrogen peroxide quercetin agents interacting with transmitter, hormone or drug receptors amyloid beta protein hydrogen peroxide neuroprotective agent protein aggregate quercetin reactive oxygen metabolite superoxide dismutase animal cell animal experiment animal tissue Article brain mitochondrion brain nerve cell cell viability controlled study

drug determination



Neurons
Neuroprotection
Neuroprotective Agents
Neurotransmitter Agents
Oxidative Stress
Presynaptic Terminals
Protein Aggregates
Quercetin
Rats, Sprague-Dawley
Reactive Oxygen Species
Superoxide Dismutase

Mice, Transgenic

Mitochondria