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Apigenin Ameliorates Oxidative Stress-induced Neuronal Apoptosis in SH-SY5Y Cells

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Abstract

The overproduction of reactive nitrogen species (RNS) and reactive oxygen species (ROS) causes oxidative damage to neuronal cells, leading to the progression of neurodegenerative diseases. In this study, we determined the nitric oxide radical (NO), hydroxyl radical (\cdot OH), and superoxide anion radical (O_2^-) scavenging activities of apigenin. Our results showed that apigenin exhibited remarkable, concentration-dependent \cdot OH, O_2^- , and NO radical scavenging activities. Particularly, apigenin indicated the strongest \cdot OH radical scavenging activity with 93.38% in the concentration of 100 μ M. Furthermore, we also investigated the protective effects of apigenin against hydrogen peroxide (H_2O_2)-induced oxidative stress in SH-SY5Y cells. The H_2O_2 treatment resulted in a significant decrease in cell viability, as well as an increase in lactate dehydrogenase (LDH) release and ROS production compared with the H_2O_2 -nontreated SH-SY5Y cells. However, the cell viability significantly increased in the apigenin-treated group, as well as inhibited ROS generation and LDH release compared with the H_2O_2 -induced control group. To elucidate the protective mechanisms of apigenin against oxidative stress in SH-SY5Y, we analyzed the apoptosis-related protein expression. The apigenin treatment resulted in the downregulated expression of apoptosis-related protein markers, such as cytochrome C, cleaved caspase-3, poly (ADP)-ribose polymerase (PARP), and B-cell lymphoma 2-associated X (Bax), as well as the upregulated expression of anti-apoptosis markers such as B-cell lymphoma 2 (Bcl-2). In this study, we report that apigenin exhibits a neuroprotective effect against oxidative stress in SH-SY5Y cells. These results suggest that apigenin may be considered as a potential agent for neurodegenerative disease prevention.

Author Keywords

Hydrogen peroxide, neuronal, neurodegenerative disease, neuroprotection, oxidative stress

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