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The pleiotropic effects of fisetin and hesperetin on human acute promyelocytic and chronic myeloid leukemia cells

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The studies elucidating the roles and the mechanisms of action of fisetin and hesperetin, plant flavonoids, in acute promyelocytic leukemia (APL) and chronic myeloid leukemia (CML) are absent. In this study, we investigated the mechanism of the antiproliferative and apoptotic actions exerted by fisetin and hesperetin on human HL60 APL and human K562 CML cells. The viability of HL60 and K562 cells was evaluated using the MTT assay, apoptosis by annexin V/propidium iodide (PI) staining and cell cycle distribution using flow cytometry, and changes in caspase-3 enzyme activity and mitochondrial transmembrane potential were determined. Moreover, we performed whole-genome microarray gene expression analysis to reveal genes and biological networks affected by KEGG and IPA analysis. For APL cells, both flavonoids showed a concentration- and time-dependent inhibition of proliferation and induced G2/M arrest and G0/G1 arrest for hesperetin at only the highest concentrations. There was a disruption of mitochondrial membrane potential together with increased caspase-3 activity. Furthermore, findcution of apoptosis was confirmed by annexin V/PI analysis. The microarray gene profiling analysis revealed some important biological pathways including MAPK and inhibitor of DNA binding (ID) signaling pathways altered by fisetin and hesperetin treatment as well as gave a list of genes modulated involved in cell proliferation, cell division, and apoptosis. For CML cells, fisetin and hesperetin inhibited cell proliferation and triggered programmed cell death. The latter was confirmed by mitochondrial membrane depolarization and an increase in caspase-3 activation. In addition to that, we have detected S and G2/M cell cycle arrests and G0/G1 arrest upon fisetin and hesperetin treatment, respectively. The microarray gene profiling analysis revealed some altered important signaling pathways including JAK/STAT pathway, KIT receptor signaling. They both significantly modulated the expression of genes involved in cell proliferation and division, apoptosis, cell cycle regulation, and other significant cellular processes such as replication, transcription, and translation.

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Investigation of ferulic acid effects on autophagy

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Coffee is particularly rich in bound phenolic acids, such as caffeic acid, ferulic acid, and p-coumaric acid. Over the past years, several studies have shown that Ferulic acid acts as a potent antioxidant by scavenging free radicals. Autophagy has recently been considered as a protective mechanism during the development of atherosclerosis because of its ability to stabilize plaques through the processing of oxidatively modified proteins. Our objective was to study the effect of ferulic acid on autophagy in cultured human umbilical vein endothelial cells (HUVECs) undergoing oxidative stress. LC3B and Beclin1 protein expression levels were measured by western blott and immunocytochemistry in oxidative stress conditions induced by administration of 200 microM hydrogen peroxide (H_2O_2) for 1 hour. After H_2O_2 administration cells were washed with DMEM and incubated 24 hours with 100 microM ferulic acid (FA). Ascorbic acid, a known antioxidant, has been used as positive control. Ferulic acid decreased the ratio of LC3II/LC3I and Beclin-1 protein levels in oxidative stress conditions. We found similar results in immunocytochemistry studies. Our preliminary data suggest the novel cardioprotective properties of ferulic acid mediated by targeting the autophagic pathway. Ferulic acid may have the potential for use as an autophagic-related antioxidant for prevention and treatment of oxidative stress.

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