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The grape fruit flavanoid, naringin protects iron induced oxidative stress *in vitro*

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Iron is plays a very important role in the cell metabolism and it also induces oxidative stress and several diseases. Naringin's abilitity to inhibit the generation of various free radicals including hydroxyl, superoxide, 2, 2-diphenyl-1-picryl hydrazyl radicals and 2, 2-azino-bis-3-ethyl benzothiazoline-6-sulphonic acid was studied and naringin inhibited these radicals in a concentration dependent manner. The isolated mouse liver mitochondria were incubated with various concentrations of naringin before 50 μM ferric chloride treatment. The iron overloading of caused an increase in lipid peroxidation, protein oxidation, and DNA damage in the mitochondria, and conversely, it reduced glutathione (GSH) concentration, glutathione-S-transferase (GST), glutathione peroxidase (GSHPx), catalase and superoxide dismutase (SOD) activities. Treatment of mitochondrial fraction with naringin before iron overloading inhibited lipid peroxidation, protein oxidation, and DNA damage. Naringin supplementation also alleviated iron-induced depletion in the GSH concentration, GSHPx, GST, SOD and catalase activities significantly. To understand the mechanism of action of naringin, ferric iron reduction assay was carried out, where naringin was unable to reduce ferric iron into ferrous iron indicating that it did not exhibit prooxidant activity. Iron free coordination site assay indicated that naringin was unable to occupy all the active sites of iron indicating that naringin did not completely chelate iron. The present study demonstrates that naringin was able to share the burden of endogenous oxidants by inhibiting free radicals and suppressing the iron-induced depletion of all important antioxidants and it may be good remedy to treat iron-induced oxidative stress.

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Chronic unpredictable mild stress impairs erythrocyte immune function and changes T-lymphocyte subsets in a rat model of stress-induced depression

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Stress has been shown to suppress immune function and increase susceptibility to inflam-matory and psychiatric diseases. This study sought to investigate the changes in erythrocyte immune functions and T-lymphocyte subsets and to explore the mechanism implicated in the process of stress-induced depression by employing a rat depression model induced by chronic unpredictable mild stress (CUMS). The body weights and behavioral changes of the rats were recorded, and plasma corticosterone levels were determined by radioimmunoassay. Erythrocyte immune function and T-lymphocytes subsets were respectively measured by the method of yeast rosette and flow cytometry at different time intervals, and their relationship was analyzed. Results indicated that a reduction was observed in the following: the rats' crossing and rearing movement times, the volume of sucrose intake and the preference for sucrose in the depression model group. Plasma corticosterone levels were elevated; the rate of E-C3bR decreased, and E-IC was increased. Some alterations in the percentage of T-lymphocytes and IL-2 appeared in the depression model group and some relationships existed between these parameters. Collectively, these findings disclose that long-time stress could induce changes in rat behavior and activities through an effect on erythrocyte immune functions and T-lymphocyte subsets.

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