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Activation of hepatic stellate cell has been identified as a critical step in hepatic fibrogenesis and is regulated by several factors including cytokines and oxidative stress. In this study, we assayed effects of saponin (CKS), inulin (CKI) and oligo-sugars (CKO) isolated from Platycodon grandiflorum A. DC, changkil (CK) on experimental cell cycle arrest and apoptosis in hepatic stellate cell line (HSC-T6). CKS induced cell arrest at G_1. CKS also reduced intercellular reactive oxygen species and collagen synthesis in hydrogen peroxide-induced oxidative stress and acetaldehyde-stimulated collagen synthesis, respectively, in HSC-T6 cells. However, both CKI and CKO were no effects. CKS induced the sustained activation of the extracellular signal-regulated kinase induced the expression of $p21^{Cip1/WAF1}$, the cell cycle-dependent kinase inhibitor, and mediated cell growth arrest through the p53 transcription activator-dependent mechanism. In conclusion, the suppression of collagen synthesis by CKS may be due to an overriding of the cell cycle arrest. These results provide that hepatic stellate cell cycle arrest by CKS be useful in the theoretical basis for clinical approaches in therapies of liver fibrosis.

[PA3-6] [2003-10-11 09:00 - 12:30 / Grand Ballroom Pre-function]

Effect of Cinnamon and Rhodiola rosea treatment on blood Glucose, Triglyceride, Total cholesterol and Glycohemoglobin in db/db mouse

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The purpose of this study is to investigate the effect of the samples on the blood levels of glucose, glycohemoglobin(HbAlc), total cholesterol and triglyceride. The samples have been used in the treatment of a type 2 diabetic animal model (C57BlKsj db/db). The samples were administrated orally before each meal for 6 weeks. Cinnamon dose was 50mg/kg/day, 100mg/kg/day, 150mg/kg/day and 200mg/kg/day, respectively. Rhodiola rosea dose was 50mg/kg/day, 100mg/kg/day, 150mg/kg/day and 200mg/kg/day, respectively.Overnight fasting and 30, 60, 90, 120 minutes postprandial blood levels of glucose were measured at 2 weeks intervals. The blood levels of HblAc, total cholesterol, and triglyceride were measured after the supplements. After 6 weeks of supplements, the blood levels of glucose and HbAlc tended to decrease in all experimental groups. However, the changes in the blood levels of total cholesterol and triglyceride were not observed after the supplement. In conclusion, the present study has demonstrated that sample have a tendency to decrease 6 Week postprandial blood glucose levels and HbA1c.

[PA3-7] [2003-10-11 09:00 - 12:30 / Grand Ballroom Pre-function]

Identification of Proteineous Biomarkers for Cadmium- and Ceramide- Induced Toxicity in Human Brain Cells through Display Proteomic Analysis

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Cadmium is an environmental pollutant and exhibits nephrotoxicity, hepatotoxicity and immunotoxicity. Recently, cadmium was found to induce DNA fragmentation, a biochemical hallmark of apoptosis, in cultured renal cells, hepatocytes and neuroblastoma cell. Therefore, the various toxicities of cadmium are thought to be caused by the induction of apoptosis. Lipids-derived pro-apoptotic ceramide has emerged as an important intracellular signaling molecule that mediates diverse cellular effects, of which programmed cell death, or apoptosis, has attracted significant interest. Although the biochemical mechanism by which ceramide triggers apoptosis is not fully understood, there are considerable lines of evidence that they are the key mediator of this response. In this study, we examined to the change of protein level in cadmium or ceramide-induced apoptosis using a high-resolution two-dimensional gel electrophoresis (2-D). The fifteen different proteins stained by Coomassie G, were identified by mass spectrometry analyses combined with peptide fingerprinting. Among them, vimentin exhibited marked accumulation in cadmium and ceramide-induced apoptotic cells. Our data show that vimentin could be a biomarker of cadmium and ceramide-induced cell death or a critical pathway to lead to the toxicity.