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Effect of Iron ir. NO Production and Mitochondria Function of Rat Peritoneal Macrophage

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The effect of iron-overload was observed in rat peritoneal macrophages. NO production and iNOS were elevated in macrophages from iron-injected rat. Lipid peroxidation was increased considerably in iron-injectedrat, compared with slight increase of ROS. Cytochrome c oxidase activity and mitochondrial membrane potential were decreased in iron treated rat suggesting that mitochondria were one of the major target suffered from iron-overload.

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Inhibitory Effect of Some Known and Probable Endocrine Disrupting Chemicals on Steroidogenic Acute Regulatory Gene Expression in Testis Leydig K28 Tumor Cells

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The effects of some known endocrine disrupting chemicals (EDCs) and suspect on the viability and steroidogenesis of Leydig K28 tumor cells were examined. Cells were treated with 10-13~10-3M of DES, Bisphenol-A, DEHP and Benzophenone. Cell viabilities were measured at 72hr after treatment using MTT assay. The viabilities of K28 cells treated with 5 x 10-5 M of DES and Bisphenol-A, and 2.5 x 10-4M of Benzophenone were reduced to 60% of nontreated cell. DHEP had no effect. To test whether EDCs affect steroidogenesis in K28 cells, StAR gene expression was examined with northern blot analysis. DES, Bisphenol-A and Benzophenone inhibited the (Bu)2 cAMP-induced StAR mRNA expression at 10^{-13}~10^{-4} M, but DEHP inhibited steroidogenesis only at high concentration of 10-4M. Because StAR gene expression is critical in sterodogenesis, inhibiting effect of DES, Bisphenol-A and Benzophenone on this gene expression suggests that these chemicals are inhibitors on steroidogenesis in testis Leydig cells.

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Transforming Growth Factor-βs Induced Apoptosis of Leg Bud Mesenchymal Cells via Up-regulation of p38 MAPK

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Transforming growth factor-βS (TGF-βS) control growth, differentiation and apoptosis of cells, and have important functions during embryonic development. In limb bud mesenchymal cells of chick embryo, TGF-β1 or -β3 stimulated phosphorylation of MAPKs (ERK and p38) as well as smad2/3. However, TGF-β1 or 3 showed opposite effect on cell condensation and chondrogenesis of leg and wing bud mesenchymal cells. Among those signaling molecules, ERK inhibited chondrogenesis but p38 showed opposite effect on cell survival of wing and leg bud cells. Moreover, TGF-βs inhibited cell proliferation and differentiation of leg bud cells, whereas stimulated those of wing bud cells. TGF-β arrested chondroblast cells of leg bud at G2M phase up to 30% at day 1, and then induced apoptosis at day 3 of the culture. Pretreatment of p38 MAPK inhibitor, SB 203580 decreased TGF-β-induced growth arrest resulting in apoptosis and thus significantly recovered from inhibition of chondrogenesis of leg bud cells. Interestingly, inhibition of p38 MAPK activity also decreased phosphorylation of GSK-3β resulting in stimulation of enzymatic activity. Co-treatment of 2 mM LiCl, a potent inhibitor of GSK-3β activity with TGF-β significantly increased growth arrest at G2M phase. Taken together, our data indicate that regulation of chondrobalst cell survival by TGF-βs occur via up-regulation of p38 MAPK signaling pathway, and p38 MAPK and GSK-3β pathways are interactively involved in this regulation of growth arrest.

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Induction of Apoptosis by the Extracts of *Ailanthus altissima* Swingle in Leukemia Cell Line HL60, K562

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An increasing number of natural occuring chemical compounds have been identified to be potent in the treatment of various human dieases including cancer. Many of these substances are found in oriental herbs or plants. Recently, some chemopreventive extracts of herbs or plants have been shown to be anti-tumorigenic. Mixed extract of herbs or plants might contain different chemopreventive or chemotherapeutic effects. It is therfore very attractive and important to identify combination effects of mixed extract of herbs or plants. This study was designed to further investigate the anti-tumor effects of extracts of *Ailanthus altissima Swingle* elucidate the potential mechanisms using an in vitro system. We found that the extracts of *Ailanthus altissima Swingle* was able to induce apotosis in human leukemai cell line HL60, K562 cells. The mechanisms might be mediated through the NF-kB dependent pathway in human leukemia K562 cells. In the human leukemia cancer cell line HL60 caused activation of caspase 3, cleavage of PARP, and subsequently cell death. These results indicate that the induction of apoptosis by the extracts of Ailanthus altissima Swingle may account for its anti-tumor activity and such activity is required for the activation of the mitochondrial pathway.