E103

Lactate Dehydrogenase and Mitochondrial LDH Inhibitor in Tissues of Vertebrate

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The mitochondria of vertebrate were isolated with the density gradient centrifuge with Percoll and it was identified with the measurement of monoamine oxidase (EC 1.4.3.4) activity. Lactate dehydrogenase (EC 1.1.1.27, LDH) inhibitors of muscle in Syrian hamster (*Mesocricetus auratus*) and Korean cow (*Bos taurus coreanae*) were successfully separated in the treatment of 175 mM NaCl and ultrasonication. The LDH inhibitor of muscle in Korean cow was determined heat-resistant form and LDH A_4 isozyme was more inhibited than other isozymes. The molecular weight of the inhibitor was 22,000 Dalton. The inhibitor played a major role in the binding of LDH with the mitochondria in tissues of muscle, kidney and liver except heart.

E104

Induction of Apoptosis in Human Head and Neck Cancer Cell Line, HTB 43 by Vitamin E-succinate

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Vitamin E-succinate (VES) treatment induced human head and neck cancer cell line, HTB 43 to undergo apoptosis. After 48 hr of VES treatment at 10 μg/ml, more than 50% of cells appeared as apoptotic cells. Evidence for the induction of apoptosis by VES treatment is based on the DAPI staining for detection of chromatin condensational fragmentation and electrophoretic DNA ladder formation. Western blot analysis showed a transient increase in cdc 2 protein level at 6-12 hr after VES treatment, which resulted in cell cycle arrest at M-phase leading to a rapid cell death. VES treatment also induced activation of caspase-3 that is consistent with the pattern of cleavages of caspase-3 cascade including PARP and lamin B. Taken together, our data suggest that induction of apoptosis by VES treatment involves a transient enhancement of cell division which caused a sudden death of HTB 43 cells.

E105

Effect of Tea Fungus/Kombucha Beverage on Lipid Metabolism and Enzyme Activities in Streptozotocin-Induced Diabetic Female Rats

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The effect of tea fungus/kombucha (TF) beverage on serum lipid concentrations and enzyme activities in growing female rats was investigated. Diabetic groups were divided into D-control (TF free water), 20 or 40% TFD (20% or 40% TF in water) according to the level of TF supplementation for 6 weeks. The body weight gains were lower in all diabetic groups than that of the control group. The levels of fasting serum glucose were higher in all diabetic groups than that of the control group. The concentrations of total lipid, cholesterol, triglyceride and hemoglobin of rats fed 20% TFD and 40% TFD groups were similar to those of the control group. The

levels of serum HDL-cholesterol and liver triglyceride were significantly decreased in all diabetic groups compared with those of the control group. In all diabetic groups, there were a significant increase of alkaline phosphatase, GPT and GOT activities.

E106

Effects of 17 β -Estradiol (E2) and Polyamines on TNF α or Tamoxifen-induced Apoptosis in MCF-7 Human Breast Cancer Cells

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Effects of 17 β -estradiol (E2) and polyamines on TNF α or tamoxifen-induced apoptosis were investigated using MCF-7 breast cancer cells. Polyamines (putrescine, spermidine and spermine) are ubiquitous low-molecular polycationic amines that play multifunctional roles in cell growth and differentiation. The possible roles of polyamines include protecting DNA fragmentation, inhibiting DNA damage and preventing apoptosis. E2 induces proliferation of the estrogen receptor (ER) -positive MCF-7 breast cancer cell line. In addition, E2 has been known to suppress TNF α -induced apoptosis in ER-positive MCF-7 breast cells. Cell viability on TNF α and tamoxifen treatment was performed using MTT assay. Reactive oxigen species (ROS) generation was measured using 2',7'-dichlorofluorescin diacetate (DCFDA) by Fluorescence Plate Reader. TNF α and tamoxifen had a significant dose-and time-dependent inhibitory effect on the growth and viability of MCF-7 cells, as determined by the MTT assay. TNF α -induced ROS generation in MCF-7 cells was increased to 150% at 48 h. However, tamoxifen had no ROS generation in MCF-7 cells. TNF (10 ng/ml) treatment for 48 h induced strong DNA fragmentation. Pretreatment of E2 (10 nM) or spermine (0.1 mM) for 48 h suppressed TNF α -induced DNA fragmentation. An anti-estrogen, tamoxifen (5 μM), treatment induced DNA fragmentation at 96 h. However, pretreatment of E2 or putrescine (1 mM) for 24 h completely suppressed tamoxifen-induced DNA fragmentation. These results demonstrate that E2 and polyamines can prevent TNF α or tamoxifen-induced apoptosis in MCF-7 human breast cancer cells.

E107

Induction of Antioxidant Enzymes and Lipid Peroxidation in the Hepatic and Brain Tissues of Guinea Pigs after Exposure to 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin

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The present study was designed to investigate whether 2,3,7,8-tetrachlor-dibenzo-*p*-dioxin (TCDD) induce the production of lipid peroxidation and antioxidant enzymes in the hepatic and brain regions of male guinea pigs after single exposure to TCDD. For this study, male guinea pigs were treated with TCDD (1 μg TCDD/kg body weight, single i.p. administration), sacrificed at 4 weeks after the treatment, and dissected with hepatic tissues and brain regions (hypothalamus, thalamus, hippocampus, cortex, cerebellum, and striatum). TCDD induced the marked