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

increase of the formation of thiobarbituric acid-reactive substances (TBARS) as a measurement of lipid peroxidation in the hepatic cytosol fractions. The activities of cytosolic glutathione *S*-transferase (GST), glutathione reductase (GR), and copper/zinc-superoxide dismutase (Cu/Zn-SOD) were also significantly induced. However, The catalase and glutathione peroxidase activities in hepatic cytosol were no changed in TCDD treated group compared with the control group. Treatment of TCDD caused the overall increase of the Cu/Zn-SOD activity in the all brain regions, especially showing the marked increase in hippocampus, cerebellum, and striatum. On the other hand Mn-SOD activity showed no characteristic change in the brain regions by TCDD administration. When compared with the control, administration of TCDD led to the regional specific increase of GR activity especially in cortex and striatum. Our result further showed that lipid peroxidation was increase in all the brain regions, especially statistic increase in thalamus. These results provide strong evidence that, even though the vulnerability to oxidative stress by TCDD is non-specific to brain region, exposure to TCDD induce an oxidative stress in the hepatic and brain tissues.

E108

Induction of Oxidative Stress and Cell Death in Neuronal SK-N-MC Cells by Stimulation with 2,2',5,5'-Tetrachlorobiphenyl (PCB 52)

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Polychlorinated biphenyls (PCBs) are large scale industrial chemicals which are using in diverse applications, such as in dielectric fluids, in transformers and capacitors, in hydraulic fluids, and as sealants. The goal of this study was to determine if exposure to 2,2',5,5'-Tetrachlorobiphenyl (PCB 52) leads to an increase in the production of active oxidants, and subsequently promotes apoptosis of neuronal SK-N-MC cells. Upon treatments with PCB 52, the time- and concentration-dependent inhibition of cell viability were observed. The capability of PCB 52 to induce apoptosis was associated with proteolytic cleavage of specific target proteins such as poly (ADP-ribose) polymerase and beta-catenin proteins suggesting the possible involvement of caspases. Reactive oxygen species (ROS) formation was examined in SK-N-MC cells after treatment of PCB 52 by concentrations (5, 10, 15, and 20 μg/ml) and incubation times (15, 30, 45, 60, 75, 90, 120 min), respectively. It showed that the rate of ROS production in the cells was increased in a does-dependent manner to 45 min, followed by a return towards control levels after 120 min treatment. ROS formation was also measured in the presence of superoxide dismutase (inhibitor of oxygen free radical production) and mannitol (hydroxyl radical scavenger). Mannitol significantly inhibited ROS generation in PCB 52-treated group. We examined the association of PCB-induced apoptosis with the modulation of biomakers of oxidative damage to lipids (malondialdehyde [MDA]) in SK-N-MC cells. Increased MDA was observed in cytosol treated with 10, 15, and 20 μg/ml of PCB 52 for 12 h and in media treated with 10, 15, and 20 μg/ml of PCB 52 for 24 h. The activities of antioxidant enzymes, catalase, CuZn-Superoxide Dismutase (CuZn-SOD), were also examined. The cells did not show any significant increase in the rate in CuZn-SOD activity. On the other hand, when