Nurr1-overexpressing human neural stem cells (Nu-NSCs). Immortalized human NSCs were generated from embryonic human brain cells via a retroviral vector encoding v-myc. Neurotoxins, 6-OHDA and MPP+, induced an extensive cell death in NSCs and Nu-NSCs, but resulting cytotoxicity are different. Nurr1 expression increased the vulnerability of NSC-Nurr1 to 6-OHDA-induced cell death. In contrast to 6-OHDA, Nu-NSC were more resistant to MPP+-induced cell deaths than parental cells. Annexin-V staining, mitochondrial membrane potential and electron microscopy indicated that 6-OHDA but not MPP+-mediated cell death was apoptotic. These results suggest that neuronal cell deaths in response to 6-OHDA and MPP+ progress through different mechanisms, which can be differentially regulated by Nurr1 protein.(Supported by grants from KOSEF/BDRC)

E140 Analysis of Biochemical Alteration in Glial Cells by Iron

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As an essential nutrient and a potential toxin, iron poses an exquisite regulatory problem in biology and medicine. An important cellular site of both the generation of oxygen radical and oxidative damage is the mitochondria. The generation of oxygen radical by iron causes mitochondrial damage. Alteration of the MPT triggered by calcium has increasingly been implicated in ischemic and apoptotic cell death, especially in brain cells. So we examined the physiological change of glial cell mitochondria using rhodamine 123 and several biochemical parameters such as free radical production, protein oxidation, lipid peroxidation, NO production, iron and calcium content after iron treatment to glial cells. First we examined the calcium content using Arsenaso III in control and iron-treated cells because calcium accumulation make mitochondria expand. In addition, the membrane potential measured using rhodamine 123 was decreased in iron-treated cells. From these results, we suggest that iron overload alter calcium regulation in cells and it can affect the mitochondrial function. Other biochemical parameter were increased in iron-treated cells than control.

E141 Effect of Okcheonsan Powder on Concentrations of Glucose, Lipid and Protein in Streptozotocin-Induced Diabetic Female Rats

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The effects of Okcheonsan powder on the body weight, the organ weight, and the concentrations of glucose, lipid and protein were studied in the diabetic rats. Female rats (Sprague-Dawley, mean weight 313.618.5 g) were randomly assigned to one normal and two diabetic groups. They were fed experimental diets for 5 weeks. The diabetic groups were divided into the diabetic control (D-control) and 3% Okcheonsan groups. Rats were injected with streptozotocin intraperitoneally (i.p.) to induce diabetes. The Okcheonsan powder feeding could decrease the pancreatic weight, the concentrations of the triglyceride, the total cholesterol and LDL-cholesterol of serum, and the cholesterol of liver in the diabetic rats. But the concentrations of the glucose, the hepatic triglyceride and the atherogenic index seems to be not affected by it.

E142 8-Cl-cAMP Increases CD98 Heavy Chain Expression via Protein Kinase C Pathway.

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8-Cl-cAMP has been known to induce growth inhibition and differentiation in a variety of cancer. To further understand the mechanisms of anticancer activity of 8-Cl-cAMP, we tried to find the genes whose expressions are influenced by 8-Cl-cAMP treatment. DT cells were treated with 8-Cl-cAMP for 72 h and then the RNA expression levels were compared with those of control cells through DD-PCR. By analyzing the expression patterns, we were able to select several genes whose expressions are increased or decreased after 8-Cl-cAMP treatment. Among these genes, we chose CD98 heavy chain for further analysis. CD98 is a type II integral membrane protein consisting of an 80 kDa heavy chain and a 40 kDa light chain. CD98 is strongly expressed on the surface of activated lymphocytes and various tumor cells. The expression of CD98 heavy chain is augmented up to 3 folds after 72 h-treatment of 8-Cl-cAMP, which was reversed to the basal level by PKC inhibition but was not affected by PKA or MAPK inhibition. Furthermore, a potent PKC activator PMA alone could increase CD98 heavy chain expression. The present results suggest that 8-Cl-cAMP increases CD98 heavy chain expression via PKC pathway.

E143 Lipid Transfer Particle Mediates the Delivery of Diacylglycerol from Lipophorin to Fat Body in Larval Manduca sexta

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This work analyzed the process of lipid storage in fat body of larval *Manduca sexta*, focusing in the role of lipid transfer particle(LTP). The transfer of DAG from Lp to fat body and its storage as TAG was significantly inhibited(60%) by preincubation of the tissue with anti-LTP antibody. Lipid transfer was restored to control values by the addition of purified LTP to fat body that had been treated with anti-LTP antibody. Incubation of fat body with dual labeled-DAG-Lp or the tissue treatment with ammonium chloride showed that neither a membrane-bound lipoprotein lipase nor Lp endocytosis are relevant pathways to tranfer or to storage lipids into fat body, respectively. The treatment of fat body with lipase after its incubation with [^{3}H]-DAG-Lp significantly reduces the amount of labeled-DAG associated with the tissue, suggesting the lipid is still on the external surface of the membrane. These results indicate that the main pathway for the transfer DAG from Lp to fat body is via a LTP-mediated process.

E144 Effects of ATP on the stability of liver and pectoral muscle enzymes in 6-aminonicotinamide treated quail against heat and trypsin digestion

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The stabilities of liver and pectoral muscle enzymes in 6-aminonicotinamide (6-AN) treated quail against heat and trypsin treatment in the presence and absence of ATP were investigated. In the thermal stability of liver enzymes, ATP provided a high level of protection for glyceraldehydes-3-phosphate dehydrogenase over the incubation temperature but virtually exerted no effects for lactate dehydrogenase and acetylcholinesterase. Similar observations were also made with the pectoral muscle but the degree of protection was greater. In the susceptibility towards trypsin digestion there was a substantial protection against trypsin digestion by ATP of liver glyceraldehydes-3-phosphate dehydrogenase and lactate dehydrogenase but almost no protection of acetylcholinesterase. In the