pathway were also investigated. Nitric oxide production was not increased by CKD-501 treatment and CKD-501 induced glucose uptake was not inhibited by L-NAME, a nitric oxide synthase (NOS) inhibitor. Intracellular Ca2+ depletion abolished the increase in glucose transport induced by either insulin or CKD-501.
In conclusion, CKD-501 might improve the hyperglycemia by increasing GLUT-4 translocation, leading to the stimulation of glucose transport and this stimulation might be at least partially caused by the increase in intracellular calcium.

[PA3-2] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Induction of Apoptosis by N-nitrosocarbofuran, via Cytochrome c-Mediated Activation of Caspase-3 protease

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Carbofuran(CF) is one of the most widely used carbamate pesticides in the world applied for insect and nematode control. Due to its widespread use in agriculture and households, contamination of food, water, and air has become serious, and consequently adverse health effects are inevitable in humans, animals, wildlife and fish, it has reported that CF alone or in combination with other carbamate insecticides influences the level of reproductive and metabolic hormones such as thyroxine and corticosterone, and results in impairment of endocrine, immun behavioral functions. we investigated the effects of NOCF on the Chinese hamster lung fibroblast (CHL) induction of apoptosis. The treatment of CHL cells with NOCF caused activation of caspase-3 , 8 protease. NOCF did not affect the expression of proapoptotic protein Bid but did cause a release of mitochondrial cytochrome c into cytosol. In conclusion, our results demonstrate that NOCF induced apoptotic cell death of CHL cells via cytochrome c dependent pathway.

[PA3-3] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

EFFECTS OF CADMIUM CHLORIDE ON GLUCOSE TRANSPORT IN 3T3-L1 ADIPOCYTES

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Cadmium is well known as a toxic metal and has insulin mimicking effects in rat adipose tissue. To investigate the effect of CdCl2 on glucose transport and its mechanism, this study was performed in 3T3-L1 adipocytes.
10 and 25mM of CdCl2 exposed to cells for 12 hours increased 2-deoxyglucose uptake to 2.2 and 2.8 fold, respectively. Nifedipine, a calcium channel blocker, inhibited the 2-deoxyglucose uptake stimulated by CdCl2. This indicates that CdCl2 enters into the cell through the Ca2+ channel to affect glucose transport. Wortmannin, PI3 kinase inhibitor, and PD98059, MEK inhibitor, did not affect 2-deoxyglucose uptake. From these results, it is thought that CdCl2 may act on glucose uptake via insulin independent pathway.
ROS were also considered to increase glucose transport. To examine the relationship between Cd-induced glucose uptake and Cd-induced ROS production, [ROS]i and GSH level were measured. The fluorescence signal of reduced form of DHDCF-DA by cellular ROS,was measured with confocal microscope and was found to be dramatically increased by CdCl2

treatment. CdCl2 induced elevation of [ROS]i was inhibited by N-acetylcystein, GSH precursor. Total GSH level was decreased by CdCl2 treatment. but ratio of GSSG/GSH was not changed. Simultaneous treatment of BSO, a GSH synthesis inhibitor,with CdCl2 showed further decrease of total GSH levels. But, NAC treatment resulted in the reduction of Cd-induced depletion of total GSH and GSH/GSSG ratio. Cd-induced 2-deoxyglucose uptake was inhibited in NAC or BSO treated group.
All these results suggest that Cd-stimulated glucose transport might be based on the activation of pentose phosphate pathway of the cells as an antioxidant defense mechanism

[PA3-4] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Effect of β-carotene on DNA damage by gamma radiation in mice

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This study deals with the radiation protection effect of the pretreatment of β-carotene and combination with selenium on the DNA damage in mice after whole body γ-irradiation. This was obtained the radioprotective effect by evaluation of DNA damage levels in mice spleen and blood after irradiation. Six-week-old ICR male mice were administrated with β-carotene and combination with selenium orally once a day for 5 days and then irradiated with 8.0 Gy of γ-ray at a dose rate of 1.0 Gy/min. After that, the mice were sacrificed 3 days later to prepare splenic lymphocytes and blood lymphocytes. Spleen and blood were collected aseptically and isolated the lymphocytes by Ficoll-histopaque gradient centrifugation. Cells embedded in agarose are lysed, subjected briefly to an electric field, stained with a fluorescent DNA binding stain and viewed using a fluorescence microscope. The tail moment(TM) of DNA single-strand breaks in mice splenic and blood lymphocytes were evaluated by single cell gel electrophoresis assay (Comet assay). In splenic lymphocytes, TM values in high administration doses of β-carotene and plus selenium reduced the most compared to low administration dose group and those of all experimental groups in blood lymphocytes showed similar. These results indicate that β-carotene had a little protective effects on the radiation induced DNA damage of the mice splenic and blood lymphocytes but it did show a little difference in radioprotective effectivness according to the administration dose and combined effect of β-carotene and selenium of high administration dose in splenic lymphocytes was the most effective compared with all experimental groups including blood lymphocytes.

[PA3-5] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Effect of selenium on DNA damage of radiation in mice splenic and blood lymphocyte

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The aim of this study was to investigate the protective effects of selenium and its combination with β-carotene treatments prior to whole-body irradiation in mice. This was obtained the radioprotective effect of selenium and its combination with β-carotene by evaluation of DNA damage levels in mice spleen and blood after irradiation. Six-week-old ICR male mice were administrated with selenium(low dose : 0.5 mg/kg, high dose : 2.0 mg/kg) and plus β-carotene (low dose : 3.0 mg/kg, high dose : 12mg/kg) orally once a day for 5 days and then irradiated with 8.0 Gy of γ-ray. After that, the mice were sacrificed 3 days later. Spleen and blood were