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

Lee BangWool°, Oh SeonHee, Lee ByungHoon

Collage of Pharmacy and Medicinal Resources Research Center WonKwang University

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

Kim MH, Kim KS°, Lee HB, Chae SH, Jung AY, Jo YY, Kim MH, Moon CK

Lab of hygienic chemistry, college of pharmacy, Seoul National University, Seoul, Korea

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