whether ceramide formation plays a role in hypoxia-induced apoptosis. Ceramide level was measured in SH-SY5Y human neuroblastoma cells by metabolic labeling with [^{3}H]palmitic acid or [^{3}H]serine. Hypoxia resulted in the increase in ceramide production with subsequent evidence of apoptosis in SH-SY5Y cells. Both fumonisin B1 (FB1), a ceramide synthase inhibitor, and L-cycloserine, a serine palmitoyltransferase inhibitor, blocked hypoxia-induced ceramide generation while sphingomyelin levels remained unchanged. L-cycloserine, but not FB1, reduced hypoxia-induced apoptosis. This may be due to a known cytotoxic sphingolipid, sphinganine accumulated by cotreatment of hypoxia and FB1. Hypoxia-induced cell death and ceramide production were significantly potentiated by NOE (N-oleoylethanolamine), an inhibitor of ceramidase, and PDMP (DL-thero-1-phenyl-2-decanoylamino-3-morpholino-1-propanol), an inhibitor of glucosylceramide synthase (GCS). PARP cleavage and caspase 3 activation were accelerated and potentiated by treatment of PDMP but not NOE. This indicated that GCS is more important in hypoxia-induced apoptosis than ceramidase. Hypoxia-induced neuronal cell death was potently inhibited by an inhibitor of caspase, z-VAD-fmk (z-VAD-fluoromethylketone). Our results suggest that hypoxia-induced neuronal cell death may be caused by increase in the *de novo* synthesis of ceramide pathway and the subsequent activation of caspase.

Poster Presentations - Field B1. Physiology

No submitted abstract in the field B1 (Physiology)

Poster Presentations - Field B2. Pathology

[PB2-1] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Anti-proliferative Effects of Godulbaegi Extracts on Human Cancer Cells

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We investigated the anti-proliferative effects of godulbaegi (*Ixeris sonchifolia* H.) root extracts, luteolin (3', 4', 5, 7-Q-glucoside and 3', 4', 5, 7-tetrahydoxyflavone) and apigenin (3', 4', 5, 7-O-gluconic acid) on SK-MEL-2 human melamine cancer cells and HepG2 liver cell line. In MTT assay 3', 4', 5, 7-tetrahydoxyflavone showed the most efficient anti-proliferative effects. According to PI staining and DNA fragmentation assay, we postulated that this effects may be a result from cell cycle arrest, then we examined the changes of protein expression related cell cycle arrest and apoptosis. Western blotting data represented that the expression of p53, cyclin A and cyclin B1 were decreased against the increase of quantity of 3', 4', 5, 7-tetrahydoxyflavone in HepG2 cell line. However, luteolin induced apoptosis in SK-MEL-2 cells. Now we are performing more experiments to clear the potentiality of it's cell cycle arrest and/or apoptosis inducing effects on human cancer cells.

[PB2-2] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Inhibitory Action of Phenylpropanoids on Phospholipase A2 Activity in RAW 246.7 Cells

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