Scutellariae radix, and balicalein. However, the other ingredient did not show any ameliorating effects on the total infarction volume. The inhibiting effect of Scutellariae radix on the total infarction volume was more potent than that of the others. In addition, HHDT, Scutellariae radix, and baicalein significantly inhibited myeloperoxidase (MPO) activity, an index of neutrophil infiltration in ischemic brain tissue at about same rate (30%). There was marked mismatch between total infarction volume and MPO activity in the Scutellariae radix-treated rats but not in the HHDT- and baicalein-treated group. Our findings suggest that Scutellariae radix as an ingredient of HHDT plays a crucial protective role in ischemia-induced brain injury by inhibiting neutrophil infiltration. In addition, it is apparent that the effect of Scutellariae radix is the result, in part, of baicalein, a compound contained in Scutellariae radix.
[Supported by MOHW grant HMP-00-CO-04-0004]

[PB3-9] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

The neuroprotective activities of the Panax ginseng in the transient ischemic model in rats.

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Panax ginseng C. A. Meyer, a traditional chinese herb, has many pharmacological effects on memory, learning, physical stress, fatigue, etc. However, several lines of evidences suggest that ginseng root plays a role in the neuroprotection. Therefore, we studied to investigate the possible neuroprotective activities of various ginseng extracts and its chemical processed compounds in ischemia-reperfusion brain injury. They were orally administered one time (100 mg/kg), promptly prior to reperfusion. Rats were subjected to 120 min of focal cerebral ischemia by means of the filament method of middle cerebral artery occlusion (MCAo). After 120 min transient-MCAo, reperfusion was achieved by pulling the filament out of the ICA under the anesthetic conditions. After 22 hr of reperfusion, infarct size was measured and neurological function was quantified. Metabolites fraction of Ginseng BuOH extract and Ginseng BuOH extract-treated with mild acid showed significant decreases of infarct size. The neuroprotecting effects of other materials are under study.[Spported by NACF grant].

[PB3-10] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Regulation of taurine transporter, TAUT, in a brain endothelial cell lines

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The activity of taurine transport in the brain endothelial cells was investigated using conditionally immortalized rat brain capillary endothelial cell lines (TR-BBB). The uptake of [^{3}H]taurine in the TR-BBB was increased by time-dependently and was dependent on both sodium and chloride ion. Furthermore, β-alanine strongly inhibited the uptake of [^{3}H]taurine in the TR-BBB. Taurine transporter (TAUT) was expressed in TR-BBB using RT-PCR and TAUT expressed at about 70 kDa was revealed by Western blot analysis in TR-BBB.
Considering taurine neuroprotective and osmoregulatory functions in brain endothelial cells, experiments were performed to study the effects of TNF-α, taurine or raffinose on taurine uptake in TR-BBB. TR-BBB exposed to 20 ng/ml of TNF-α for 12h showed 1.7 fold increase in taurine uptake and significant uptake increase was observed after 24h exposure. But taurine uptake was significantly decreased time-dependently by incubating the cells in the same medium containing exogenous taurine. Aiso, the uptake of
[^{3}H]taurine in the TR-BBB was 3.2 fold increased by hypertonic condition after 24h exposure. The

mRNA level of TAUT in TNF-α treatment and hypertonic cells was markedly higher than that in control cells, but in taurine treatment cells was lower than that in control cells. In conclusion, the regulation of taurine transport was associated with the amount of the TAUT presents at the BBB by biological factors.

[PB3-11] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

15-Deoxy-Δ12,14-Prostaglandin J2, a Ligand of Peroxisome Proliferator-Activated Recepter-γ Induced Apoptosis Through G2/M Phase Arrest in Neuroblastoma Cells

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15-Deoxy-Δ12,14-prostaglandin J2 (15-deoxy-PGJ2), a peroxisome proliferator-activated receptor (PPAR-γ) ligand, has been shown to stimulate differentiation and induced apoptosis in several cancer cells including breast, prostate and lung cancer cells. In this study, we examined whether 15-deoxy-PGJ2 could inhibit cell growth through induction of apoptosis in neuroblastoma cells (SK-N-MC and SK-N-SH). We also investigated the expression of (anti-) apoptosis-related genes and activation of transcription factors. 15-Deoxy-PGJ2 inhibited neuroblastoma cells growth and induced apoptosis in a dose (2-16 μM) and time-dependent manner. Consistent with the induction of apotosis, 15-deoxy-PGJ2 reduced the expression of anti-apoptotic Bcl-2 but increased the expression of pro-apoptotic Bax, caspase 3 and caspase 9. Flow cytometric analysis showed that these cells were arrested in G2/M phase after 15-deoxy-PGJ2 treatment. Furthermore, 15-deoxy-PGJ2 significantly increased the expression of cyclin B1, but decreased the expression of cdk4, cyclin D1 cdk2 and cdc2. It was also found that PPAR-γ was expressed by 15-deoxy-PGJ2 in these cells. Taken together, these results suggest that 15-deoxy-PGJ2 may be a candidate for a preventive or a therapeutic agent for neuroblastoma.

[PB3-12] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

The pharmacokinetics of taurine in Senescence-Accelerated Mouse

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The senescence-accelerated mouse (SAM) strains show senescence acceleration and age-associated pathological phenotypes similar to geriatric disorders seen in humans. Among these strains, we used SAMP8 and SAMR1. This study compared the blood-brain barrier (BBB) permeability of [^{3}H]taurine in SAM and normal mice with common carotid artery perfusion (CCAP) method. Also, for evaluation of pharmacokinetic parameters of [^{3}H]taurine in SAM and normal mice, we used intravenous injection technique.

In the result of CCAP method in SAM at perfusion flow-rate of 2 ml/min, the brain volume of distribution (V_D) of [^{3}H]taurine was reduced to that of the normal mice.

Brain distribution volume of [^{3}H]taurine in SAMP8 right brain by CCAP method reduced by 85% compared with that in normal mice. Brain distribution volume of [^{3}H]taurine after CCAP at a rate of 2 ml/min for 15, 30 second in anesthetized SAM was obtained by linear regression. We found from result by intravenous injection technique, the sucrose space in SAMP8 was significantly decreased compare than that of normal mice.

These results suggest that aging may have any effect on the brain transport activity of taurine in disease state model animal.