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.