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Choline is an important membrane phospholipid constituent and a neurotransmitter precursor that is minimally synthesized in brain. The long-term maintenance of brain choline concentration is dependent on choline transport from plasma, which occurs via saturable transport system at the blood-brain barrier. In the present study, we examined to elucidate the characteristics of transport of cationic amines, especially choline which is one of cationic amines, to BBB using conditionally immortalized rat brain capillary endothelial cell line (TR-BBB) in vitro. The [<sup>3</sup>H] choline in the TR-BBB was increased by time dependently but independent on Na ion concentration. The uptake of [<sup>3</sup>H]choline is susceptible for inhibition by various organic cationic compounds including tetraethylammonium chloride (TEA), L-carnitine, betaine, and drugs of the treatment of Alzheimer's disease such as L-acetyl carnitine, donepezil hydrochloride (Aricept), tacrine, and antioxidant, N-tert-butyl- $\alpha$ -phenyl-nitron (PBN). Also, hemicholinium-3 is strongly inhibited the uptake of [<sup>3</sup>H]choline. The kinetic parameters for the saturable transport of choline were estimated to be the apparent Michaelis-Menten constant, Km, and the maximal velocity, Vmax. The choline transport in this cell line existed high- and low affinity systems.

[PB3-7] [ 04/18/2003 (Fri) 09:30 – 12:30 / Hall P ]

### **Brain-to-blood efflux transport of taurine at the blood-brain barrier in rats**

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The purpose of this study is to examine whether a brain to blood efflux system for taurine is present on the blood-brain barrier (BBB) or not and this efflux transport system is regulated by CNS cell damage with oxidative stress agent such as diethyl maleate (DEM) or tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), by using the brain efflux index (BEI) method. The brain efflux index value is defined as the relative amount of test compound effluxed from cerebrum compared with that of a reference compound, [<sup>14</sup>C]carboxyininulin, which has limited BBB permeability. [<sup>3</sup>H]Taurine was microinjected into parietal cortex area 2 (Par2) of the rat brain, and was eliminated from the brain with an apparent elimination half-life of 57 min and an efflux transport rate of  $1.22 \times 10^{-2}$ /min. This process was inhibited by taurine transport inhibitors, such as unlabeled taurine,  $\beta$ -alanine, betaine, nipecotic acid and GABA, significantly. In addition, the effect of DEM or TNF- $\alpha$  on [<sup>3</sup>H] taurine efflux transport was investigated. [<sup>3</sup>H]Taurine efflux transport was reduced by pre-treatment with DEM or TNF- $\alpha$ . In conclusion, the efflux pump for taurine at the BBB acts to reduce taurine concentration in the brain interstitial fluid and this process was carrier mediated and also, was regulated by oxidative cell damage.

[PB3-8] [ 04/18/2003 (Fri) 09:30 – 12:30 / Hall P ]

### **15-Deoxy-PGJ<sub>2</sub> Stimulates Neuronal Differentiation of Embryonic Midbrain Cells by Up-regulation of PPAR- $\gamma$ Activity via the JNK-dependent Pathway**

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