

20 and 50 µg/ml) dose dependently increased ROS generation and underwent apoptotic death. Aβ treatment also led to the increased formation of 8-oxo-dG, a form of oxidative DNA, and NF-κB activation. Green tea extract dose dependently attenuated Aβ (50 µM)-induced cytotoxicity, apoptic features, and intracellular ROS in addition to the reduction of NF-κB activation. These data demonstrate that green tea extract may be useful agent to prevent or reduce of AD development and progression.

[PA1-22] [ 2003-10-10 14:00 - 17:30 / Grand Ballroom Pre-function ]

### **Alteration of Gβ Expression in Rat Brain by Stress**

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The heterotrimeric G protein βγ subunits (Gβγ) are region-specifically expressed in brain such as hypothalamus and pituitary gland in abundant, suggesting that is may be associated with "stress-axis". This study was designed to examine the effect of stress on the region-specific expression of various Gβ subunits in rat brain. The localization of mRNAs encoding seven of Gβ and striking region-specific patterns of expression were observed in 12 different regions of both non-stressed and stressed rat brain; (1) frontal cortex area, (2) cerebral cortex area, (3) striatum, (4) hippocampus area, (5) thalamus, (6) brain stem, (7) cerebellum area, (8) hypothalamus, (9) septum, (10) amygdala, (11) preoptic area, and (12) pituitary gland. Animals were exposed to stress by immobilizing restrain method with 2 hr/day for 8 days. The results show that Gβ1, Gβ4, Gβ5, and Gβ5L were expressed in several regions of rat brain such as cortex, hippocampus, thalamus, hypothalamus, amygdala, cerebellum and pituitary gland in non-stressed rat brain. In contrast, stress altered the expression of Gβ1 and Gβ5 in these regions of rat brain. Interestingly, the expression of Gβ1 subunits in the regions associated with stress-axis such as hypothalamus and pituitary glands was lessed by stress. In contrast, the expression of Gβ5 was increased by stress. Therefore, these results suggest that these Gβ subunits play a key role of Gβγ signaling in regulating stress in brain.

[PA1-23] [ 2003-10-10 14:00 - 17:30 / Grand Ballroom Pre-function ]

### **Mechanism of leptin-induced catecholamine secretion in the perfused rat adrenal medulla**

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It has been demonstrated the presence of leptin receptors (Ob-Ra) on epinephrine-secreting chromaffin cells in rat adrenal medulla, suggesting that leptin may directly affect the adrenal medulla (Cao et al., 1997). Leptin is found to stimulate catecholamine (CA) synthesis in cultured bovine adrenal medullary cells (Utsumomiya et al., 2001; Shibuya et al., 2002) and cultured porcine adrenal medullary cells (Takekoshi et al., 2001). Thus, the present study was designed to examine the effect of leptin on CA release from the isolated perfused rat adrenal gland, and to establish its mechanism of action. Leptin (1 ~ 100 ng/ml), when perfused into an adrenal vein of the rat adrenal gland for 60 min, enhanced a dose-dependently the secretory responses of CA evoked by ACh (5.32 x 10<sup>-3</sup> M), excess K<sup>+</sup> (5.6 x 10<sup>-2</sup> M, a membrane depolarizer), DMPP (10<sup>-4</sup> M, a selective neuronal nicotinic N<sub>n</sub>-receptor agonist) and McN-A-343 (10<sup>-4</sup> M, a selective M<sub>1</sub>-muscarinic agonist). leptin alone produced a weak secretory response of the CA. Moreover, leptin (100 nM) in to an adrenal vein for 60 min also augmented the CA release evoked by BAY-K-8644, an activator of the dihydropyridine L-type Ca<sup>2+</sup> channels, and cyclopiazonic acid, an inhibitor of cytoplasmic Ca<sup>2+</sup> ATPase. However, in the presence of anti-leptin (10 ng/ml), an antagonist of Ob receptor, leptin (10 ng/ml) no longer enhanced the CA secretion evoked by ACh and DMPP. Collectively, these experimental results suggest that leptin enhances the CA secretion from the rat adrenal medulla evoked by cholinergic stimulation (both nicotinic and muscarinic receptors) and membrane depolarization. It seems that this facilitatory effect of leptin may be mediated by activation of leptin receptors located on the rat adrenomedullary chromaffin cells.