activity stimulated by MSH, forskolin and 8-Br-cAMP were not affected by KN-62 (calmodulin-dependent protein kinase II inhibitor), PD098059 (mitogen-activated protein kinase kinase inhibitor, MAPKK) and wortmannin (phosphatidylinositol 3-kinase inhibitor). These results suggest that protein kinase C and tyrosine kinase are involved in melanin production via cAMP-dependent pathway and their action site on cAMP-dependent melanin production may be different from each other.

[PB1-2] [04/19/2002 (Fri) 10:00 - 13:00 / Hall E]

Sodium Chloride Regulates Alpha Epithelia Sodium Channel through Unknown Pathway(s)

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The epithelial amiloride-sensitive sodium channel is a heteromultimer composed of three subunits that plays a central role in sodium homeostasis and blood pressure control. The molecular effect of high sodium on the epithelial sodium channel gene is not well known. This study examined the effect of high salt intake on alpha epithelia sodium channel gene transcription in Sprague-Dawley rat kidney. Seven-week-old female Sprague-Dawley rat were injected intraperitoneally with hypertonic (1.5M NaCl) or normal saline solution (3 rats/group). The plasma sodium concentration of rats in the hypertonic saline injected group was found to increase significantly at 30 min after injection. At 3 h after injection, plasma sodium decreased but remained above the control value. The plasma aldosterone concentration was slightly decreased at 3 h after hypertonic saline injection. The kidney cortex was dissected macroscopically mRNA was isolated at 1.5 h and 3 h after treatment. Levels of mRNA were determined by semi-quntitative RT-PCR. Following hypertonic saline treatment, alpha sodium channel mRNA levels were dramatically reduced compared with levels observed in either rats injected with normal saline, or uninjected rats. Under these experimental conditions, no changes in mineralocorticoid receptor mRNA levels were observed, suggesting that transcription factors other than the mineralocorticoid receptor may be responsible for epithelial sodium channel gene regulation. Inhibition of protein synthesis by cycloheximide co-injection (1.5 mg/kg of body mass) blocked the sodium chloride-induced alpha epithelial sodium channel mRNA down-regulation at 3h of treatment. This indicates that synthesis of new, uncharacterized protein(s) is required for sodium chloride-mediated inhibition of alpha epithelial sodium channel gene transcription. This work was supported in part by grants from the Korean Ministry of Health and Welfare (01-PJ1-Pg1-01CH06-0003, YJL).

Poster Presentations - Field B2. Pathology

[PB2-1] [04/19/2002 (Fri) 10:00 - 13:00 / Hall E]

Alteration of MAP kinase activity in experimental esophagitis

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In inflammation condition, it was reported that the level and/or activity of MAP kinase was/were changed in the response of immune mediators. Using two models of experimental esophagitis, we assessed the activity of p38 MAP kianse, p44/42 MAP kinase and JNK. First, we performed the repeated perfusion of the cat esophagus with 0.1 N hydrochloric acid for three days to make feline acute experimental esophagitis. Western blotting of normal and esophagitis-induced smooth muscle with each types of MAP kinase antibodies revealed that decrease of phosphorylated form of p38 MAP kinase. JNK activity was also decreased, but the amount of change was less than that of p38 MAP kinase. The level of phosphorylated form of p44/42 MAP kinase in esophagitis-induced smooth muscle showed no differences, compared with normal muscle. Second, surgically induced reflux esophagitis of rats showed time-dependent increase of ulcer index (UI), resulting in UI 4 after 6 hours. After the increase of phosphorylation of p38 MAP kinase in 4

hours (UI = 1), it was decreased below the basal level in 6 hours. The activity of JNK was increased with accordance with the progression of esophagitis. The level of phosphorylation of p44/42 MAP kinase was increased in 1 hour and decreased in 4hours. After 6 hours, it was recovered to the basal level. With these results, we suggest that the each type of MAP kinases shows different features of activation and deactivation in experimental esophagitis models.

Poster Presentations - Field B3. Neuroscience

[PB3-1] [04/19/2002 (Fri) 10:00 - 13:00 / Hall E]

METHAMPHETAMINE SELF-ADMINSTRATION INDUCED C-FOS AND GFAP EXPRESSION IN RAT BRAINS.

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(+)-Methamphetamine (METH) is a psychostimulant, which has been the most popular abused drug in Korea. In this study, we investigated the behavioral changes in rats administered repeated or self-administered METH, and the effects of METH self-administration on the expression of c-fos, glial fibrillary acidic protein (GFAP) and tyrosine hydroxylase (TH) in brain. The repeated administration of 1.0 mg/kg/day METH for 12 days increased locomotor activities, and there was no difference between i.v. and i.p. treatment. Rats had acquired actively METH self-administration for 3 weeks at 0.1 or 0.2 mg/kg/injection. Whereas, it was taken few days to acquire sucrose pellet self-administration. The dose-response relationship for METH demonstrated a typical inverted U-shaped function. Rats were injected about 1.0 mg/kg/day for 27~53 days in intravenous self-administration training course. METH self-administration increased dose-dependently the protein expression of c-fos in prefrontal cortex, hippocampus, striatum and ventral tagmental area (VTA). GFAP expression was also increased dose-dependently in hippocampus, striatum and VTA. However, TH expression was not changed in striatum and VTA. These results suggest that low dose of METH may induced neurotoxicity in rats self-administrated for long periods.

[PB3-2] [04/19/2002 (Fri) 10:00 - 13:00 / Hall E]

Inhibitory Effects of Aporphine Alkaloids on Dopamine Biosynthesis in PC12 cells

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The effects of aporphine isoquinoline alkaloids such as boldine, isocorydine, liliodenine, anonaine and asimilobine on dopamine biosynthesis in PC12 cells were investigated. Treatment of PC12 cells with boldine, liliodenine, anonaine and asimilobine showed 5~50 % inhibition of dopamine content at a concentration of 1~20 μM for 24 h. However, Isocorydine did not show an inhibitory effect. The IC50 values of boldine, liliodenine, anonaine and asimilobine were 19.6 μM, 7.7 μM, 0.35 nM and 0.13 nM, respectively. Dopamine conetent decreased at 6 h and reached minimal level at 24h after the exposure to aporphine isoquinoline alkaloids described above. Tyrosine hydroxylase (TH) activities were also inhibited by aporphine alkaloids. Treatment of PC12 cells with aporphine alkaloids showed 60~85 % inhibition of TH activities at a concentration of 1~20 μM for 6 h. However, Aromaic amino acid decarboxylase activities did not.TH activities reached minimal level at 6~12h following the treatments of boldine, liliodenine, anonaine and asimilobine (84.0 % at 24.4 μM, 85.4 % at 12 μM, 67.4 % at 1.51 μM, 87.5 % at 1.48 μM, respectively), and maintained at a reduced level for up 36 h in PC12 cells. These results suggest that the inhibition of TH activities by each aporphine isoquinoline alkaloids might be involved in at least one component of the reduction of dopamine biosynthesis in PC12 cells. Intracelluar mechanisms need further studies of