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