6-72 hr with no effect in male rats. In the cerebellum, AchE was significantly inhibited by 20-34% at 6, 12, and 24 hr in female rats and by 9-13% in male rats. In the plasma, AchE was significantly inhibited by 52% at 6 hr, 58% at 12 hr, 30% at 24 hr and 17% at 72 hr after administration, compared to 29% at 6 hr and 33% at 12 hr post-dose in male rat. Neuropathy target esterase activity was significantly decreased at 6 and 24 hr in the entorhinal cortex, and 6 and 12 hr in the liver of male rats. Taken together, TBF treatment results in more vulnerability to female than male rats in brain and blood AchE activity.

[PA4-22] [04/18/2002 (Thr) 14:00 - 17:00 / Hall E]

The effect of PCBs (polychlorinated biphenyls) on EROD activity in vivo.

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PCBs are wide spread persistent environmental pollutants that exert a broad spectrum of toxic effects. PCBs includes 209 possible congeners differing in extent and position of chlorination of their aromatic rings. In order to understand the toxic mechanism of PCBs, we have tested the effect of PCB on the EROD activity in vivo. Among the AhR gene battery, CYP4501A is a well understood parameter for the potency of AhR agonists. The 7-ethoxyresolufin O-deethylase(EROD) activity of CYP4501A isozyme is widely accepted marker to measure the inducibility of dioxin-like compounds on CYP1A gene expression. The bioaccumulated diortho-chloro-substituted PCB congeners, PCB118(2,3',4,4'5-pentachlorobiphenyl), PCB138 (2,2',3',4,4'5-hexachlorobilhenyl), PCB153 (2,2',4,4',5,5'-hexachlorobilhenyl), PCB180 (2,2'3,4,4'5,5'-heptachlorobiphenyl) and commercial (the technical mixture) PCBs, Aroclor 1254 were treated SD rats and ERDO activity of rat liver microsome was examined. Also in order to evaluate the possible cross talk between these chemicals and estrogen by comparing the effect of a series of diortho-substituted PCB congeners alone treatment and cocomitant treatment with estrogen on CYP1A-catalyzed EROD. As expected, PCBs and Aroclor showed the induction of CYP1A-catalyzed EROD activity in rat liver microsome. Aroclor1254 treatment showed the dose-dependent increase of EROD activity in SD rat liver microsome and the effect of Aroclor1254 was inhibited by E2 concomitant treatment. Also PCBs-induced EROD activity and this stimulatory effect was inhibited by E2 concomitant treatment. In the present study, we demonstrate that PCBs are inducers of CYP1A gene espression and these effects cen be inhibited by estrogen.

Poster Presentations - Field B1. Physiology

[PB1-1] [04/19/2002 (Fri) 10:00 - 13:00 / Hall E]

Effects of Protein Kinase Inhibitors on Melanin Production in B16 Cells Stimulated via cAMP-dependent Pathway

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To investigate the effect of protein kinase on melanin production via cAMP-dependent pathway, we measured the melanin amount and tyrosinase activity in B16 melanoma cells stimulated by alpha-melanocyte stimulating hormone (MSH), forskolin and 8-Br-cAMP. MSH, forskolin and 8-Br-cAMP significantly increased both melanin production and tyrosinase activity in B16 cells. Bisindolmaleimide (1 μ M), protein kinase C inhibitor, significantly inhibited melanin production and tyrosinase activity stimulated by MSH, forskolin and 8-Br-cAMP with the following order of potency: MSH>forskolin>8-Br-cAMP. Tyrosine kinase inhibitor, genistein and DHC, significantly inhibited both, but the inhibitory effect was more potent in 8-Br-cAMP-stimulated B16 cells than MSH-stimulated cells. Both melanin production and tyrosinase

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