

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-hexachlorobiphenyl), PCB153 (2,2',4,4',5,5'-hexachlorobiphenyl), 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  $\mu$  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