with 10mg/kg fumonisin B1(FB1), and kidney, liver, heart, lung, brain and serum were collected for sphingolipid analysis. Free sphingosine and free sphinganine were determined by HPLC. The concentrations of free sphingoid bases in control rats were approximately 1595 pmol > 898 pmol > 651 pmol > 642 pmol > 563 pmol/100mg wet weight in lung > kidney > liver > brain > heart, while free sphinganine were 294 pmol > 99 pmol > 81 pmol > 76 pmol > 63 pmol in lung > heart > brain > liver > kidney, respectively. FB1-treated rats showed that amounts of elevated free sphinganine were 10.6 nmol > 5.3 nmol > 3.4 nmol > 2.2 nmol > 0.2 nmol in kidney > liver > lung > heart > brain, respectively. Thus, these results indicate that 1) *de novo* sphingolipid biosynthesis is the most active in lung and 2) the most sensitive organ of fumonisin B1 is kidney, while the least sensitive one is brain. Sphinganine 1-phosphate (Sa1P) elevation in FB1 exposure to rats was highest in kidney and lung, and lowest in brain. FB1 increased Sa1P conentration by 678 pmol/100μl serum compared to 6 pmol in control serum. In coclusion, FB1 sensitivity to sphingolipid metabolism are organ-specific and related to the fumonisin toxicity.

[PA3-12] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Distribution of Arsenic in Korean Human Tissues

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Arsenic is a ubiquitous element that ranks 20th in abundance in the earth's crust, 14th in the sea water, and is a component of several hundred minerals. Arsenic and its compounds are mobile in the environment. Groundwater contamination by arsenic is a serious threat to mankind all over the world and it can also enter food chain. Humans are exposed to this toxic arsenic from air, food and water. The current study was performed to investigate the levels of arsenic in the internal organs and to find out correlation with age and interrelationship between tissues in Korean human bodies who had lived in Seoul or Kyonggi do and Honam district. The tissues from 43 Korean cadavers were digested with microwave digestion system and arsenic was determined by ICP-MS. The mean recovery percentages of arsenic in liver were about 80% and arsenic concentrations in human tissues were almost uniform. The mean level of arsenic in internal tissues were as follow ; liver 44.556±25.199ppb, kidney cortex 42.652±22.082ppb, lung 31.020±17.504ppb, cerebrum 35.703±22.591ppb, muscle 43.415±26.619ppb and skin 42.106± 25.831ppb. No significant difference was found in the levels of arsenic between sexes. Significant differences between districts where they had lived were found in all tissues tested. The levels of arsenic in the tissues of cadavers who had lived in Seoul Kyonggi do were higher than those of Honam district. And Positive correlation with age was observed only in the cerebrum($p<0.05$). A significantly high correlations between tissues were observed in all tissues tested. This result also shows that the distribution of arsenic is uniform in internal tissues.

[PA3-13] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Effect of Arsenic on Acetylcholine-Induced Relaxation in Blood Vessels

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Several epidemiological studies suggested that arsenic exposure was strongly correlated with the development of cardiovascular disease such as hypertension. In order to examine whether

arsenic affects vasomotor tone in blood vessels, we investigated the effect of arsenic on agonist-induced vasorelaxation using the isolated rat aortic rings in in vitro organ bath system. Treatment with arsenite inhibited acetylcholine-induced relaxation of aortic rings in a concentration-dependent manner. The inhibitory effects by arsenic were also observed in the relaxation induced by sodium nitroprusside, a NO-donor. Consistent with these findings, the cGMP levels stimulated by acetylcholine in blood vessels were reduced significantly by arsenite treatment. In addition, higher concentration of arsenite decreased the relaxation by 8-Br-cGMP, a cGMP analog, in aortic rings without endothelium. These in vitro results indicated that arsenite was capable of suppressing acetylcholine-induced relaxation in blood vessels by inhibiting production of nitric oxide in endothelial cells and by impairing the relaxation machinary in smooth muscle cells. In vivo studies revealed that the reduction of blood pressure by acetylcholine infusion was significantly suppressed after arsenite was administered intravenously to rats. These data suggest that vasomotor tone impaired by arsenite exposure may be one of the contributing factors in development of cardiovascular disease.

[PA3-14] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Evidence of TCDD-like activities in crude and fractionated extracts of PM 2.5 diesel particle material using EROD-microbioassay.

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Diesel motors exhaust particulate material, which is known to be mutagenic, has caused heavy air pollution. PM 2.5 diesel exhaust of vehicle was collected using a high-volume sample equipped with a cascade impact. The crude extract was fractionated according to EPA recommended procedure into seven fractions by acid-base partitioning and silica gel column chromatography. We examined Ah receptor-mediated activities of fractionated samples using EROD microbioassay in H4IIE rat hepatoma cell line and HepG2 human hepatoma cell line. EROD microbioassay was conducted to determine cytochrome P4501A activity in environmental samples, and the TCDD equivalent concentration(TEQ) was calculated for the quantitative assessment. The biological TEQ was calculated by comparing the concentration response curve of the sample with those of the TCDD calibration curve. In the results, we confirmed that a large quantity of TCDD-like components was presented in PM 2.5 diesel exhaust particulate materials. Higher potency was observed in crude extract and nonpolar fraction. Since, it is reported that aliphatic and aromatic compounds such as chlorinated hydrocarbons, PAH and their alkylderivatives are contained nonpolar fraction, we presume that these chemicals may relate to TCDD-like activities.

[PA3-15] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Inorganic Arsenic Increases Vasoconstriction through Calcium-Sensitization in Vascular Smooth Muscles

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