**AP-33**

**DNA damage evaluation using comet assay in workers occupationally exposed to lead**

Ahmet SAYAL¹, İlknur YAVUZ², Zeliha KAYAALTI², Ayşegül BACAKSIZ², Esma SÖYLEMEZ², Tülin SÖYLEMEZOĞLU²

¹Division of Pharmaceutical Toxicology, Department of Pharmaceutical Sciences, Gulhane Military Medical Academy, Turkey, ²Forensic Sciences Institute, Ankara University, Turkey

Lead is a non-biodegradable and significant environmental chemical contaminant. Lead decreases the accuracy of DNA synthesis or repairs and inhibits the activity of DNA polymerase, thus it causes DNA damage including DNA base modifications, DNA strand breaks, chromosome rearrangements. Comet assay is a recently developed technique in order to monitor human populations that have been exposed to environmental toxicants. The aim of this study was to investigate the association between the DNA damage and lead levels in individuals who are exposed to lead due to occupational reasons. For this purpose, 61 workers exposed to lead were monitored in the way of DNA damage in blood lymphocytes. The levels of DNA damage were measured according to three comet assay parameters including tail intensity (TI), tail moment (TM) and DNA tail (DNAt) by BAB Bs Comet Assay system and Pb levels were analyzed with the atomic absorption spectrometer system. As a result, statistically significant positive correlation was found between the Pb levels and TI, TM and DNAt parameters (r=0.515, p<0.01; r=0.507, p<0.01; r=0.343, p<0.01, respectively) in peripheral lymphocytes. Smoking had independent effects on TI, DNAt and TM. Significant difference was observed between the smokers and non-smokers in relation to DNA damage parameters (p<0.05). In addition, when the Pb levels were compared with smokers and non-smokers groups, Pb levels in smokers were found significantly higher than non-smokers (17.96±13.27 ppb versus 4.77±2.47 ppb; p<0.05). In summary, our results show that exposure to Pb induces genotoxic effect in peripheral lymphocytes, as measured by comet assay.

**AP-34**

**Induction of SLC39A8 expression by FGF-2 in vascular endothelial cells**

Emi HACHISUKA¹, Chika YAMAMOTO², Toshiyuki KAJI¹

¹Faculty of Pharmaceutical Sciences, Tokyo University of Science, Japan, ²Faculty of Pharmaceutical Sciences, Hokuriku University, Japan

FGF-2 is released from injured endothelial cells and then promotes the repair of the cell layer by stimulation of migration and proliferation of the cells near the damaged site in an autocrine fashion. Essential trace element zinc enhances the activity of FGF-2, suggesting that there is a strong interrelationship between intracellular zinc metabolism and FGF-2 activity. The present study was undertaken to clarify the regulation of zinc transporter expression by FGF-2 in vascular endothelial cells. Bovine aortic endothelial cells were treated with FGF-2 and the expression of SLC39A8 (a zinc transporter) was determined by Western blot analysis. The expression level of zinc transporter mRNAs was analyzed by real time RT-PCR. It was shown that FGF-2 increased the level of SLC39A8 that transports zinc into the cytosol from the extracellular space or intracellular compartments at both protein and mRNA levels. In contrast, the growth factor decreased the mRNA expression of SLC30A1 that transports zinc from the cytosol to the extracellular space or into intracellular compartments. The mRNA expression of the other zinc transporters including the SLC39A family and SLC11A2 was not increased by FGF-2. The present results suggest that FGF-2 regulates the expression of zinc transporters and raises the cytosolic zinc concentration by induction of SLC39A8 expression and suppression of SLC30A1 in vascular endothelial cells.