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**SALIVARY OXIDATIVE STRESS BIOMARKER: 8-HYDROXYGUANOSINE**

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**Introduction** Oxidative stress leads to many kinds of diseases. Various chemicals in workplaces induce oxidative stress. In addition, psychological stress also increases oxidative stress. To prevent diseases potentiated by oxidative stress, a method for the appropriate assessment of the oxidative stress status is needed. Currently, urinary 8-hydroxydeoxyguanosine (8-OHdG) is widely measured as an oxidative stress biomarker. There is a specific advantage if saliva can be used as the sample to measure the oxidative stress biomarker, because saliva is much easier to collect than urine. However, the accurate measurement of 8-OHdG in saliva is impractical, because the quantity of 8-OHdG in saliva is quite low. In this study, we investigated the measurement of 8-hydroxyguanine (8-OHGua) in saliva, as an oxidative stress marker.

**Methods** The 8-OHGua levels in saliva were analysed by a column switching HPLC system equipped with an electrochemical detector (HPLC-ECD).

**Results** The 8-OHGua in saliva could be detected as a single peak by HPLC-ECD. The salivary 8-OHGua levels of smokers were significantly higher than those of non-smokers.

**Conclusion** Salivary 8-OHGua may be a useful biomarker in the human population, in relation to the assessment of the oxidative stress induced by various factors in working environments.

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**OCCUPATIONAL TOLUENE DIISOCYANATES EXPOSURE AND CANCER MORTALITY: 12-YEAR FOLLOW-UP STUDY FOR TEN THOUSAND MALE WORKERS IN KOREA**

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**Introduction** An International Agency for Research on Cancer (IARC) evaluated that the TDI was possibly carcinogenic to humans (Group 2B) based on inadequate evidence in humans and sufficient evidence in experimental animals. However after the IARC review, some epidemiologic study suggested that TDI was related to the cancer occurrence, especially lung cancer. So we analysed the cancer mortality of methanol exposed male workers in Korea.

**Methods** A cohort was comprised of 10,526 TDI exposed workers working between January 1, 2000, and December 31, 2004. These cohort members were matched with the mortality data of the Korean National Statistical Office to follow-up for cancer mortality between 2000 and 2011. Standardised Mortality Ratios (SMRs) of methanol exposed workers with reference to Korean men were calculated. Also controlling age, calendar year and other carcinogen exposure including hepatitis B and C, the Adjusted Hazard Ratios (AHRs) of workers categorised by the TDI-exposure duration (over 10 years) with reference to workers with less than 10 years were calculated.

**Result** There were no significantly increased or decreased SMRs. But, non-significantly increased SMRs were observed in lung cancer (SMR=1.11, 95% CI: 0.41 to 2.41) with over 10

years exposure. There were no significantly increased or decreased AHRs of cancer mortalities in workers exposed to TDI with over 10 years exposure compared to workers with less than 10 years.

**Discussion** In this study short follow-up periods and healthy worker effect (HWE) may hamper observation for increasing cancer mortality of TDI exposed workers comparing to that of Korean male. Continuous follow-up to overcome HWE and cancer morbidity study are needed to confirm this study result.

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**TISSUE DISTRIBUTION OF INDIUM AFTER AN INTRATRACHEAL OR A SUBCUTANEOUS ADMINISTRATION OF INDIUM OXIDE IN RATS**

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**Introduction** Indium oxide ( $\text{In}_2\text{O}_3$ ), a raw material from which indium tin oxide (ITO) is produced, causes indium lung disease, and there is a possibility of inhalation exposure during the manufacturing of ITO. It is, however, not known whether indium is more widely distributed throughout the body after  $\text{In}_2\text{O}_3$  exposure. The aim of this study was to clarify the tissue distribution of indium in rats after intratracheal instillation or subcutaneous injection of  $\text{In}_2\text{O}_3$ .

**Methods** Male rats were divided into three groups: an  $\text{In}_2\text{O}_3$  intratracheal group (n=25), an  $\text{In}_2\text{O}_3$  subcutaneous group (n=25), and a control group (n=30). In both  $\text{In}_2\text{O}_3$  groups, rats received a single 10 mg In/kg body weight dose of  $\text{In}_2\text{O}_3$ . A subset of rats was periodically euthanized throughout the study from 1 day to 36 weeks after treatment. Indium concentrations in the organs were determined using inductively coupled plasma-mass spectrometry.

**Results** In both  $\text{In}_2\text{O}_3$  groups, very low concentrations of indium were detected in the main organs on day 1 after treatment. Although the content of indium in the lungs for the subcutaneous group gradually increased over the 36 week observation periods, that for the intratracheal group decreased slowly with clearance half-life of approximately 22 weeks. In both  $\text{In}_2\text{O}_3$  groups, the indium concentration in each intraperitoneal organ gradually increased over time until 36 weeks, with levels being higher in the subcutaneous group than in the intratracheal group at 36 weeks.

**Discussion** Although the indium accumulation ratio in each intraperitoneal organ for the total administration doses was very low, indium accumulation in these organs indicated that translocation from the lungs or subcutaneous tissue occurred. This study clarifies that when  $\text{In}_2\text{O}_3$  is administered intratracheally or subcutaneously in rats, indium becomes widely distributed in the body and is excreted very slowly.

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**PLASMATIC MICRORNAS PROFILE EXPRESSION IN MALIGNANT PLEURAL MESOTHELIOMA**

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