**Introduction**

According to 2011 annual report of national cancer registration program, age-standardised incidence of non-hodgkin lymphoma, leukaemia, multiple myeloma, Hodgkin lymphoma is 6.8, 5.0, 1.4, 0.5 per 1,00,000. Although incidence rate is low, social attention is increasing due to the fatality. While there has been many foreign studies on association between occupational risk factor and lymphatic, haematopoietic cancer, a study reflecting the domestic situation is insufficient. So we conducted case-control study using data from occupational cancer monitoring system to assess risk factor.

**Methods**

Cases were 384 leukaemia, 523 non-hodgkin lymphoma, 218 multiple myeloma patients reported from occupational cancer monitoring system from 2011 to 2014. Controls were selected randomly matched on age, sex, residence. All participants were interviewed for lifestyle habits, exposure or occupational history of group 1, group 2A carcinogen. Analysis was performed using chi-square test primarily, and logistic regression to adjust for smoking status.

**Results**

Analysing by chi-square test, excess risks were shown for exposure to benzene, formaldehyde, TCE, PAH in leukaemia, to benzene, formaldehyde, TCE in non-hodgkin lymphoma, to benzene, formaldehyde in multiple myeloma. Analysing by logistic regression to adjust for age, sex, smoking status, excess risk were shown for exposure to benzene, formaldehyde, pesticide in non-hodgkin lymphoma, to benzene in multiple myeloma. Other exposures were associated with lymphatic or haematopoietic cancer, but were not significant.

**Conclusion**

Increased risk of lymphatic or haematopoietic cancer were associated with some occupations and chemicals. But other exposures showed no statistically significant association due to insufficient number of samples. There is a need for sufficient number of samples to obtain additional association between exposure and cancer risk.

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**1500 PREDICTION AND CHARACTERISATION OF BIOMARKER NETWORK FOR BENZENE EXPOSURE**

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**Introduction**

Benzene is identified as a carcinogen. Long-term exposure to benzene causes haematological alterations, including an increased risk of acute myeloid leukaemia. However, the molecular mechanisms of Benzene systems effects remain poorly understood. Hence, a better understanding of the molecular mechanisms involved in this condition is a priority. Here, we employed a joint the integration of molecular network of possible genes as biomarker.

**Methods**

We selected 96 genes targets with altered expression between exposure and cancer risk. We selected 96 genes targets with altered expression between exposure and cancer risk. Analysing by logistic regression to adjust for smoking status, excess risk were shown for exposure to benzene, formaldehyde, pesticide in non-hodgkin lymphoma, to benzene in multiple myeloma. Other exposures were associated with lymphatic or haematopoietic cancer, but were not significant.

**Conclusion**

Increased risk of lymphatic or haematopoietic cancer were associated with some occupations and chemicals. But other exposures showed no statistically significant association due to insufficient number of samples. There is a need for sufficient number of samples to obtain additional association between exposure and cancer risk.
HEALTH EFFECTS FOLLOWING OCCUPATIONAL EXPOSURE TO PAVING ASPHALT FUMES

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Introduction: Controversy exists as to the potential of asphalt fumes to induce health effects including respiratory, hepatoxic, nephrotoxic, or hematotoxic responses. The main purpose of this study was to ascertain whether occupational exposure to asphalt fumes, under normal working conditions, is associated with any respiratory, hepatoxic, nephrotoxic, or hematotoxic response.

Methods: In this cross-sectional study in which 210 subjects (80 exposed and 130 reference subjects) were investigated. Using standard methods, atmospheric concentrations of total particulate and benzene-soluble fractions of asphalt fumes, as well as total particulate were measured. Additionally, urine and blood samples were taken from subjects for complete blood count, white blood cell differential test, urinalysis, and routine biochemical tests of kidney and liver function. For the prevalence of respiratory symptoms among subjects was investigated by a standard questionnaire. Additionally, the investigation involves analysis of the shift in cellular metabolism that the bladder epithelia cells (RT4) undergo to sustain the hostile environment generated by B[a]P-induced toxicity.

Results: It appeared that B[a]P exposure led to a repression of enzymes (fructos bisphosphate aldolase A, glucose-6-phosphate isomerase, lactate dehydrogenase) involved in glycolysis, and an up-regulation of proteins (glucose-6-phosphate 1-dehydrogenase, 6-phosphogluconolactonase) catalysing the pentose phosphate pathway and one carbon metabolism (10-formyltetrahydrofolate dehydrogenase, bifunctional purine biosynthesis protein). Untargeted metabolomics analysis revealed, lower concentration of glycolytic metabolites, as compared to glutamine, xylulose and fatty acids. The analysis of the glutathione and nucleotide content of the cells revealed a significant increase of these cofactors. Concomitantly, we did not observe any detectable increase in the production of ROS.

Discussion: The study provides new insights into a B[a]P-induced shift in cellular metabolism towards processes involved in NADPH generation. B[a]P exposure causes oxidative DNA damage and hence cellular perturbations. To overcome these effects, the cells undergo a metabolic flux change from glycolysis to the pentose phosphate pathway. This shift leads to the generation of the redox cofactor NADPH that is essential for the activity of many antioxidant enzymes and intermediates necessary for the de novo generation of nucleotides (purine and pyrimidine) and for the normal functioning of the cells. The study provides preliminary indication of changes in cellular metabolism upon B[a]P exposure.

Conclusion: This study showed that exposure to sub-threshold limit value levels of total particulate and benzene-soluble fractions is associated with early liver and kidney dysfunction as well as haematological disorders. Also, significant decrements in the parameters of pulmonary function as well as a significant increase in the prevalence of respiratory symptoms in asphalt paving workers compared to their unexposed counterparts provided evidence in favour of a significant association between exposure to asphalt fumes and lung function impairments.

515 USE OF URINARY BIOMARKERS AND BIOASSAYS TO EVALUATE CHEMICAL EXPOSURE AND ACTIVATION OF CANCER PATHWAYS IN FIREFIGHTERS

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Introduction: Cancer is a leading cause of fire service morbidity and mortality. Measurement of urinary polycyclic aromatic hydrocarbons (PAHs), a group which includes known carcinogens, provides a means of evaluating absorption from all exposure routes. Activation of the aryl hydrocarbon receptor (AhR) and p53 pathways is associated with cancer, and their evaluation through in vitro urinary bioassays provides measures of toxicity of the chemical mixtures to which firefighters are exposed.

Methods: Urine was collected at baseline and two hours after responding to fires in 80 Tucson firefighters. Urine contaminants were de-conjugated using β-Glucoroniidase and extracted using Focus Solid Phase Extraction (SPE) cartridges. Quantification of hydroxylated PAH (PAH-OH) target analytes was conducted with GC-MS/MS. In addition, the urinary extracts were evaluated using AhR and p53 in vitro bioassays.

Results: Compared to baseline, structural firefighting was associated with an increase in urinary PAH-OH concentrations. Increased concentrations were also found in training fires when self-contained breathing apparatus (SCBA) were used assiduously, suggesting a primary route of dermal exposure in that setting. Contrary to expectations, engineers (vehicle drivers) also demonstrated increased urinary PAH-OH concentrations, which was felt to be due to inhalation exposure as they generally did not wear SCBA. AhR and p53 activation occurred in general with higher concentrations of PAH-OHs.