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 group1, group2A 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.

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 networks, a gene–gene interaction database, biological processes analysis and functional annotation analysis to explore system effects for prioritising candidate genes to biomarkers to evaluate benzene exposure.
Methods We selected 96 genes targets with altered expression in occupational exposed to benzene (2009 to 2014). The analysis was performed using the multiple association network integration algorithm for predicting gene function, which identifies known gene-gene interactions among a genes list and provides additional genes. Topological properties of network were calculated by MCODE, BINGO and Centiscape,
Results A network of 114 genes and 2415 interactions were obtained. After topological analysis, a minor network composed by 16 nodes was identified, which is composed by most relevant nodes of major network. In this sub-network, KLF6, KLF4 and JUN are the most interconnected nodes, they being been considered a putative biomarker in which the exclusion of one node could produce a strong perturbation in the signalling network.
Conclusion The biological interaction network method presented probabilities of interactions between genes, demonstrating the potential of the use and application of the multiple association network integration algorithms for predicting gene function and for the observation of multiple genes in the system, using theoretical data to building clusters for identification of possible genes as biomarker.

886 AUDITORY DYSFUNCTION IN WORKERS FROM A PRINTING PRESS EXPOSED TO ORGANIC SOLVENTS

A Juarez, Aguilar, O Trujillo*, D Fierro, L Trejo, C Madriga. *Occupational health research*

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Introduction There are various chemical agents such as organic solvents (OS), which can cause hearing loss. The objective of this study was to determine the presence of auditory dysfunction in a mixture of DO and noise-exposed workers from a printing press.

Cross-sectional study was conducted including 176 from a printing press in Mexico City, exposed to noise and an OS mixturesWe categorised workers within 2 groups I. Exposed for <10 and II. Exposed ≥10 years, we estimated hearing loss through a multiple linear regression model.
Results The mean age of group I was 32±9.3 [19–62] years and for group II was 41. 6±6. 5 [29–58] years. The mean noise was 78.10±10.6 dB58.1 dB and 93.8 dB, group I. showed a threshold fall in the 4 kHz up to 25 DB in both ears, with an average recovery of 5 dB at 8 kHz Right ear: 2000 Hz: II β=4.2 (p=0.003), 4000 Hz: II β=5.6 (p=0.002), 8000 Hz II β=3.8 (p=0.5); Left ear: 2000 Hz: II. =4.1 (p=0.002), 4000 Hz: II β=5.2 (p=0.006), 8000 Hz: II β=5.2 (p=0.002) the second model high frequencies (2, 4, and 8 KHz) in right ear was II β=4.4 (p=0.002) and in the left ear was II β=4.8 (p<0.001).
Discussion Our studied population, showed an overall prevalence of auditory dysfunction of 3.94%, group II was the most affected Workers with a concomitant exposure to noise and DO >10 years have a higher auditory dysfunction prevalence, compared with workers without exposure to these agents.

722 INDUCTION OF METABOLIC SHIFT FROM GLYCOLYSIS TO PENTOSE PHOSPHATE PATHWAY IN HUMAN BLADDER CANCER CELLS EXPOSED TO BENZO[A] PYRENE

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Introduction Benzo[a]pyrene (B[a]P), a well-known polyaromatic hydrocarbon, is known for its lung carcinogenicity, however, its role in bladder cancer development is still discussed. The present

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investigations 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.
Methods We applied the two-dimensional blue native SDS-PAGE (2D BN/SDS-PAGE) technique to elucidate the network of protein-protein interactions that regulate cellular metabolism. In order to analyse the effects of B[a]P-induced protein alterations at the metabolite level, untargeted metabolomic profiling of B[a]P-exposed cells was carried out by using gas chromatographic mass spectrometric analysis (GC-MS).
Results It appeared that B[a]P exposure led to a repression of enzymes (fructose-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.

49 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, hepatotoxic, 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, hepatotoxic, 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 parameters of pulmonary function were measured both, prior to exposure and at the end of work-shift.
Results Both groups were similar as far as their demographic variables and smoking habits were concerned. The association between exposure to asphalt fumes and changes in most liver and kidney function tests and complete blood count parameters was statistically significant. Mean values of FEV1, both prior to the exposure (89.58% [SD 18.69%] predicted value) and at the end of shift (85.38% [SD 19.4%]), were significantly ($p<0.05$) smaller than those of the comparison subjects (93.88% [SD 13.93%]). Similarly, pre-shift (87.05 [SD 8.57]) and postexposure (89.95 [SD 6.85]) FEV1/FVC ratio were both significantly ($p<0.01$) lower than those of the unexposed employees (107.56 [SD 9.64]). The pattern of changes in parameters of lung function in asphalt workers was consistent with that of chronic obstructive lung disease.
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-Glucuronidase 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

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