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