P.2.14 PULMONARY DYSFUNCTION IN INDIUM TIN OXIDE EXPOSED WORKERS
1Soou-hsing Lou*, 2Yoon-Ting Hsu, 3Wei-jin Li, 4Wei-Te Wu. 1National Health Research Institutes, Miaoli County, Taiwan; 2Graduate Institute of Life Sciences, National Defense Medical Center, Taipei, Taiwan; ROC

to investigate the relationship between indium exposure and lung effects markers among indium tin oxide (ITO) manufacturing workers without job change.

Methods We enrolled 179 male workers from ITO target manufacturing and recycling factories in Taiwan. Plasma indium (P-In), urine indium (U-In) and creatinine adjusted U-In (U-In/Creat.) were used as internal dose of inductium exposure. Plasma Krebs von den Lungen-6 (KL-6) and surfactant protein D (SP-D) were used as markers of interstitial pneumonitis. Forced vital capacity (FVC), forced expiratory volume at 1st second (FEV1), and FEV1/FVC were also evaluated by spirometry.

Results After adjusted for covariates by linear regression, plasma, urinary and creatinine adjusted indium were increased in high exposure group (P-In: β=1.13, p<0.001; U-In: β=0.54, p<0.05; U-In/Creat: β=0.63, p<0.01) and low exposure group (P-In: β=0.75, p<0.05; U-In/Creat: β=0.52, p<0.05) with comparison to reference group. Plasma KL-6 was higher in high exposure group (β=0.24, p<0.05) compared to reference group, but not for surfactant protein D (SP-D). Furthermore, FVC and FEV1 were reduced in both high and low exposure groups (FVC: β=−0.08, p<0.01; FEV1: β=−0.05, p<0.05) and low exposure group (FVC: β=−0.06, p<0.05) compared to reference group.

Conclusion Our findings indicate indium exposure was related to restrictive lung dysfunction, decreased lung function for both FEV1 and FVC test but not for FEV1/FVC ratio. Meanwhile, increased plasma KL-6 in high exposure group also supports that indium exposure results in increased risk of interstitial pneumonitis among direct indium exposure workers. Our study provided an explanation to the consequence of indium exposure-interstitial pneumonitis-restrictive lung dysfunction.

P.2.15 EXPOSURE TO 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN AND MORTALITY AT A TRICHLOROPHENOL PLANT IN NEW ZEALAND
1David McBride*, 2James Collins, 3Thomas Bender, 4Kenneth Bodner, 5Lesa Aylward. 1University of Otago, Dunedin, New Zealand; 2Saginaw Valley State University, Saginaw, USA; 3Dow Chemical, Midland, USA; 4Summit Toxicology, Falls Church, USA

Methods A cohort study of 1599 men and women working between January 1, 1969 and November 1, 1988 at a plant producing the herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) with TCDD as a contaminant.

A toxicokinetic model developed in a previous follow up was updated to estimate cumulative TCDD exposure for each individual in the study. Calculation of cause-specific standardized mortality ratios (SMRs) and 95% confidence intervals (95%CIs) compared those never and ever exposed to TCDD. Dose-response trends were assessed firstly through SMRs stratified in quartiles of cumulative TCDD exposure, and secondly with a proportional hazards model.

Results The toxicokinetic model intercept of 5.1 parts per trillion (ppt) of TCDD was consistent with background New Zealand TCDD concentrations among older members of the population. Exposed workers had non-significant increases in all cancer deaths SMR=1.08, 95% CI 0.86–1.34, deaths from soft tissue sarcoma, SMR=2.38, 95% CI: 0.06–13.26, non-Hodgkin lymphoma, SMR=1.57, 95% CI: 0.32–4.59, diabetes, SMR=1.27, 95% CI: 0.53–2.50 and ischaemic heart disease, SMR=1.21, 95% CI: 0.96–1.50. Lung cancer deaths SMR=0.95, 95% CI: 0.56–1.53, were fewer than expected. Neither the stratified SMR nor proportional hazard analysis showed a dose response relationship.

Conclusion We found neither an excess of all cancers, or any specific cancer, nor a trend with TCDD exposure. This argues against the carcinogenicity of TCDD at lower levels of exposure.

P.2.16 HEALTH AND PRODUCTIVITY: A FIVE YEAR STUDY (2010–14) IN A LARGE AUTOMOBILE INDUSTRY IN INDIA
1Gautham Sukumar*, 2Kowshik Kupatira, 3Radeep Banandur, 1G Gururaj. 1National Institute of Mental Health and Neuro Sciences, Bangalore, India; 2Leading automobile industry, Bangalore, India

Introduction This ‘Health and productivity’ project was implemented to identify priority health problems and health related productivity loss in a large automobile industry in India. It hopes to re-emphasize that ‘OH department’ is a partner in productivity.

Objectives
1. To estimate prevalence, incidence rates , trends and risk for health problems among employees (year 2010–14)
2. To identify leading causes of hospitalization and out-patient care among employees (year 2010–14)
3. To quantify loss in productive work time attributed to health related absence in year 2014 and forecast to year 2025

Methods A five year records analysis (2010–14) was conducted in a leading automobile industry in year 2015–16. Data was pooled from clinic visit records, annual medical examination , insurance claims and leave records, systematically using employee ID. Trends and incidence rates of leading health risks were computed per 1000 person-months of employment. Leading causes and trends in out-patient and in-patient care of employees were determined. Total work time (man-hours) was computed and health related loss in work-time was derived. Cox-regression was used to assess risk between work department and morbidity. ARIMA method was used to forecast productivity loss till year 2025.

Results Between 2010–14, overweight and hypertension were identified as leading health risks. Respiratory, musculoskeletal and digestive disorders were leading causes for clinic visits. Infectious diseases are leading cause for hospitalization. Health related absence accounted for 1.87% of total productive time. Forecasting indicates an increase to 9.31% by year 2025.