A cohort study of 1599 men and women working exposed more recently, and at lower levels, than other cohorts of trichlorophenol process workers. 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 exposure group (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.

EXPOSURE TO 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN AND MORTALITY AT A TRICHLOROPHENOL PLANT IN NEW ZEALAND

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Objectives To describe how 2,3,7,8-tetrachlorodibenzodioxin (TCDD) exposure influenced mortality in a cohort of workers exposed more recently, and at lower levels, than other cohorts of trichlorophenol process workers. 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%CI) 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: 0.32–4.39, diabetes, SMR=1.27, 95% CI: 0.55–2.50 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.