QUANTITATIVE INHALATION EXPOSURE ASSESSMENT ON AIRBORNE PARAQUAT EXPOSURE OF HERBICIDE KNAPSACK SPRAYERS

Introduction
Paraquat is a hazardous chemical, widely used as herbicide, and was imported for 31,552 tons to Thailand in 2016. However, there was almost no report of inhalation risk assessment in Thailand. This cross-sectional analytic study was designed to quantify the inhalation exposure to airborne paraquat during spray operation of knapsack sprayers.

Methods
The study was conducted in 30 voluntary herbicide knapsack sprayers in a District of Khon Kaen province, Thailand. The airborne paraquat concentration, working and personal characteristics of sprayers were used for inhalation intake calculation following U.S. EPA (1991) equation. The selected concentration of airborne paraquat was from monitoring with active personal sampling using PTFE filter membrane and analysed with HPLC.

Results
The paraquat knapsack sprayers were farmers in sugar-cane, cassava, rice, and corn field. Paraquat dichloride was used at 0.1-2400 litres/year. Adverse symptoms related to respiratory system were throat/upper airway irritation, runny nose (not from flu), wheezing, and difficulty breathing. The inhalation intake of paraquat exposure in short term effect, long term effect, and specific effect of lung (chronic pneumonitis) were calculated by using paraquat concentration at 125.49 μg/m³. The intake estimations were between 0.00011 to 0.04610 mg/kg/day. The health risk was presented by hazard quotient (HQ>1). HQlong term was 0.263–115.25 when compared to recommended AOEILong term (0.0004 mg/kg/day). HQshort term was 0.231–92.202 when compared to recommended AOELShort term (0.0005 mg/kg/day). HQchronic pneumonitis was 0.023–10.245 when compared to the reference dose (0.0045 mg/kg/day).

Conclusion
It can be summarised that at the selected concentration and without using respirator of sprayers, this study found that 66.67%, 63.33% and 13.33% of Thai knapsack sprayers were under unacceptable risk of long term exposure, short term and chronic pneumonitis, respectively. This information should be communicated to the public health related institutions and farmers for seriously preventive regulation on inhalation exposure to paraquat.

1M.Sc. Program in Occupational Health and Safety, Khon Kaen University, Khon Kaen, Thailand; 2Department of Environmental Health, Occupational Health and Safety, Khon Kaen University, Khon Kaen, Thailand; 3Research and Training Centre for Quality of Life of Working Age People, Khon Kaen University, Thailand

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Methods 60 workers of a steel plant, aged 25–55 years, were enrolled in a panel study with two blood samples at the beginning and the end of a week. Gene expression was quantified by a 44-gene PCR Array and normalised by GAPDH housekeeping. Gene expression and PM10 data were log-transformed. Univariate correlation between each gene expression and PM10 is misleading because does not account for the association structure among genes. We instead focused on the entire association network among genes and the impact of PM10 exposure on it. Since our study is mainly explorative we deliberately choose a simple approach: the minimal BIC forest. This approach, which relies on linearity, multivariate Normality and the Adequacy of Linear Scores, is useful as a preliminary step towards understanding the overall dependence structure of high-dimensional discrete and/or continuous data.

Results The minimal BIC forest resulted in the identification of eight subgroups of correlated genes expression. The model including PM10 showed a negative association between PM10 exposure and NFKB1 expression. The sensitivity analysis on the assumptions was conducted as follows:

a. all possible [n=1892] Student t-statistics for squared terms were calculated;

b. all possible [n=39 732] choices of cross-product terms in the linear regressions.

No violation regarding the correlation between PM10 and gene expressions was found.

Conclusions The study was explorative and no a priori knowledge on gene expression pathways was considered. In conclusion we found an association of PM10 exposure with NFKB1 expression. The sensitivity analysis on the assumptions was conducted as follows:

Introduction Coachbuilding and painting shop workers are co-exposed to noise and ototoxic substances thereby producing synergistic adverse effects on hearing, which might increase the risk of professional hearing loss. A previous investigation showed that most of employers and workers ignore those risks due to occupational activities, or negligences.

Methods 60 volunteers were measured in 24 shops. We used dosimeters for evaluating the noise exposures, and passive badges for the amount of solvents inhaled by each worker during a workday.

COS DPOAE suppression (Echoscan) allowed auditory fatigue to be assessed by summing several sources: metabolic fatigue from hair cells, afferent and efferent fibres, central nuclei (SOC and facial), and obviously from middle-ear muscles. The trigger threshold variations of workers exposed to both noise and solvents were measured with Echoscan twice a day.

In the meantime, the wearing of auditory and respiratory individual protectors was monitored.

Results The Lex10H measurements were higher than 80, 85, 87 dB(A) for 33, 17, 12% of tested workers, respectively. Toluene, ethylbenzene and xylene constituted the most used ototoxics. These findings are in agreement with the data collected in the French database COLCHIC for carshops’ workers. Taken separately each solvent exposure was inferior to its legal threshold limit value, but an exposure to a chemical cocktail may affect the hair cells or the central nuclei. Results showed that data obtained with Echoscan were sensitive both to noise and solvents. At the end of a working day, trigger threshold variations of workers exposed to both noise and solvents were significantly lower than those measured in workers exposed to noise only.

Workers and shop observation showed significant neglects in collective and individual prevention.

Conclusion Difficulties to characterise workers’ effective exposure are numerous in car shops. Regarding employers and workers’ awareness, a primary prevention has been launched through information sessions. Auditory fatigue depends on the nature of the exposure. A co-exposure impacts the trigger threshold variations of COS DPOAEs.