parts of the body would reduce skin contamination, a major route of pesticide exposure.

Work was supported by NIEHS grants ES017223 and ES016308.

1714 OCCUPATIONAL NEUROTOXICOLOGY – RECENT STUDIES

Markku A Sainio*. *Work Environment, Finnish Institute of Occupational Health, Helsinki, Finland*

10.1136/oemed-2018-ICOHabstracts.824

Aim of the special session International researchers summarise their studies on adverse nervous system effects of occupational exposure to neurotoxic compounds.

Brad Racette [1,2], Eric Amster [3,4,5], Qiao NIU [6]

[1]Washington University School of Medicine, St. Louis, Missouri, USA

[2]University of the Witwatersrand School of Public Health, Johannesburg, South Africa

[3]University of Haifa, School of Public Health, Department of Occupational Health, Israel

[4]Harvard University, T.H. Chan School of Public Health. USA

[5]Meuhedet HMO, Occupational Health Department, Israel

[6]School of Public Health, Shanxi Medical University, Taiyuan 030001, Shanxi, China

1714a PARKINSONISM PREDICTIVE MODEL IN MN-EXPOSED WORKERS

Brad A Racette. *Washington University School of Medicine and University of the Witwatersrand School of Public Health*

10.1136/oemed-2018-ICOHabstracts.825

Introduction Manganese (Mn) over-exposure in occupational settings is associated with basal ganglia toxicity and a movement disorder characterised by parkinsonism (i.e., the signs and symptoms of Parkinson disease). A simple test to help non-neurologists identify workers with clinical Mn neurotoxicity represents an unmet need.

Methods Using a cohort of 536 Mn-exposed workers, age ≤65 years, from welding worksites, with extensive clinical data, we developed a linear regression model to predict the Unified Parkinson Disease Rating Scale motor subsection part 3 (UPDRS3) score. We primarily considered factors easily obtained in a primary care or occupational medicine clinic, including timed motor task results and selected symptoms/conditions. Secondarily we considered other demographic variables and welding exposure. We selected the model based on simplicity for clinical application, biologic plausibility, and statistical significance and magnitude of regression coefficients.

Results The final model contained age, timed motor task scores for each hand, and indicators of action tremor, speech difficulty, anxiety, depression, loneliness, pain and current cigarette smoking. When we examined how well the model identified workers with clinically significant parkinsonism (UPDRS3 ≥15), the receiver operating characteristic area under the curve (AUC) was 0.72 (95% confidence interval [CI] 0.67, 0.77). With a cut point that provided 80% sensitivity, specificity was 52%, the positive predictive value in our cohort was 29%, and the negative predictive value was 92%. Using the same cut point for predicted UPDRS3, the AUC was nearly identical for UPDRS3 ≥10, and was 0.83 (95% CI: 0.76 to 0.90) for UPDRS3 ≥20.

Conclusion Since welding exposure data were not required after including its putative effects, this model may help identify workers with clinically significant Mn neurotoxicity in a variety of settings, as a first step in a tiered occupational screening program.

1714b EVALUATION OF PARKINSONISM AMONG MANGANESE EXPOSED WORKERS

[1,2,3]ED Amster*, [3]L Mazor. [1]*University of Haifa, School of Public Health, Department of Occupational Health, Israel*; [2]*Harvard University, T.H. Chan School of Public Health. USA*; [3]*Meuhedet HMO, Occupational Health Department, Israel*

10.1136/oemed-2018-ICOHabstracts.826

Introduction Accumulation of manganese in the brain may result in a neurological condition with cognitive, psychiatric, and movement abnormalities. The clinical and toxicological literature demonstrates that manganese accumulates in the basal ganglia which may result in parkinsonism. There is little published about the prevalence of Parkinsonism among manganese exposed workers. We present a case series of 6 workers from a single factory and discuss methods of neurological assessment of the manganese exposed worker for the occupational health provider.

Methods IH sampling of a large tire factory employing 527 production workers was conducted for heavy metals. A walkthrough was performed assessing safety, hygiene, ventilation and use of personal protective equipment. Workers in the departments with manganese concentrations above NIOSH REL completed a symptoms survey and were assessed by occupational medicine physicians, with a specific focus on neurological assessments.

Results Environmental sampling of manganese concentration was above 1 mg/m^3 in three departments; highest measurement was 6.7 mg/m^3. Walkthrough survey revealed inadequate ventilation in all three departments and improper PPE use among 72% workers. 27 exposed workers were evaluated with symptom questionnaire and clinical exam focusing on neuropsychologic and neuropsychiatric findings; 4 of those workers had evidence of parkinsonism on exam and symptom survey. Those workers were immediately removed from the worksite. Biomarkers were sent for evaluation and the workers were sent for neurological referral.

Conclusion Manganese exposure at work is associated with increased risk of Parkinsonism. We identified a cluster of manganese exposed workers with Parkinsonism in a factory with inadequate ventilation and poor hygiene practice. Based on the findings from this case study we are able to develop a simple neurological assessment tool for the exposed worker.

1714c ASSOCIATION BETWEEN H3K4ME3/BDNF AND THE COGNITIVE FUNCTION OF WORKERS OCCUPATIONALLY EXPOSED TO ALUMINIUM

Hengying Qiu. *School of Public Health, Shanxi Medical University, Taiyuan 030001, Shanxi, China*

10.1136/oemed-2018-ICOHabstracts.827

Introduction Brain-derived neurotrophic factor (BDNF) is involved in synaptic plasticity and may be modified by H3K4me3, thus affecting learning and memory. This study aims to investigate the influence of occupational aluminium exposure on cognitive function and its relationship with H3K4me3 and BDNF levels.

Methods By cluster random sampling method, 235 male workers from Shanxi Aluminium Company who occupationally exposed to aluminium were recruited in the study. A group of cognitive tests were performed, in which includes Mini-Mental State Examination (MMSE), CDT, DST, FOM and VFT. Concentration of Aluminium in plasma was tested by graphite furnace atomic absorption spectrometry. The subjects were divided into three groups by the 25, 50 and 75 percentile of the blood aluminium concentration, as low, middle and high aluminium concentration group. The contents of H3K4me3 in lymphocyte and BDNF in plasma were determined by enzyme-linked immunosorbent assay.

Results The levels of aluminium in plasma were 100.19, 134.36 and 178.96μg/L respectively. The scores of MMSE, DSFT, DST of high blood aluminium concentration group were lower than those of low and middle blood aluminium groups (27.98□1.53 vs 28.68□1.54, 27.98□1.53 vs 28.23□1.53, 9.19□2.00 vs 10.61□2.90, 9.19□2.00 vs 9.95□2.32, 15.27□3.11 vs 17.59□4.63, 15.27□3.11 vs 16.17□3.86, $p<0.05$), The scores of CDT, DSBF, FOM, VFT among three groups had no statistical significance ($p>0.05$). The expression levels of H3K4me3 and BDNF of high blood aluminium group were lower than those of the low (20.95□3.91 vs 28.18□8.79 ng/μg protein, 26.07□10.18 vs 31.15□9.85 μg/L, $p<0.05$) and middle blood aluminium groups (20.95□3.91 vs 25.78□6.30 ng/μg protein, 26.07□10.18 vs 26.91□10.27 μg/L, $p<0.05$). Multiple correlation analysis showed that Blood aluminium concentration was negatively correlated to H3K4me3, BDNF, MMSE, DSFT, DST, respectively ($r=-0.307$, –0.175, -0.229, –0.206, –0.173, $p<0.05$).

Discussion Long-term occupational exposure to aluminium may impair cognitive function, along with the decreasing of H3K4me3 level in lymphocyte and BDNF protein expression in plasma.

(This work is supported by NSFC 81430078, 81372968)

180 MYOCLONIC SEIZURE AFTER ACUTE AND CHRONIC OCCUPATIONAL EXPOSURE TO 'THINNER' PRIOR TO DIAGNOSIS OF CHRONIC TOXIC ENCEPHALOPATHY – BELGIAN CASE-REPORT

WLC Van Hooste*. *MD, Mediwet Occupational Health Services, External Service for Prevention and Protection at Work, Ghent, Belgium*

10.1136/oemed-2018-ICOHabstracts.828

Introduction 'Thinner' is an industrial mixture of organic solvents (OS). Exposure to OS is usually not considered as possible cause of epilepsy. A case that shows a remarkable coincidence between exposure to OS mixture and occurrence of epileptic seizure is reported.

Methods We present the case of a Belgian woman, occupationally exposed to organic solvents for more than 20 years. She was admitted to the hospital after a seizure with myoclonic movements at the workplace.

Result This event was her first epileptic insult. Classic signs and symptoms of acute solvent intoxication (e.g. headache, nausea, asthenia, feeling of drunkenness, sleeping disorder) were present since she worked permanently for over 2 months with 'Thinner', containing high toluene levels, neither with proper collective nor individual protection in a poorly ventilated workplace. Electroencephalogram and computed tomography of the head were within limits. Non-toxicological causes (craniocerebral trauma; infection; familial disposition; known history of prenatally, perinatally, or childhood disease) and some toxicological causes (alcohol or addictive substances abuse) were excluded. No anti-epileptic drugs were started because of this first epileptic episode. Seizures did not reappear after work removal, although intoxication signs and symptoms remained. Two years after the seizure, she was diagnosed with Chronic Toxic Encephalopathy (CFE) type 2b.

Discussion Being volatile, OS rapidly contaminate the working environment and pose a major health risk in occupational settings. Myoclonic encephalopathy has been reported in toxic conditions, e.g. after exposure to solvent Trichloroethylene. Epileptic discharges have been described on EEG recordings among CFE patients. This case emphasises a possible unusual neurological presentation of occupational exposure to toluene. This clinical picture may be explained by lowering of the threshold for seizures by the same mechanism as seen for alcohol. It should be kept in mind that OS exposure in badly-ventilated spaces and/or without appropriate protective measures may cause seizures.

57 LITERATURE REVIEW ABOUT THE EFFECTS OF CHEMICAL EXPOSURE TO LEAD AND CADMIUM IN THE SLEEP-WAKE CYCLE

Teixeira LR[1], Coutinho GBF[1], Cozendey-Silva EN[1], Silva ICO[1], Franco WM[1], [1]A Ferreira, Fischer FM[2]. *[1]Center of Studies of Occupational Health and Human Ecology, Escola Nacional de Saúde Pública Sergio Arouca, Fundação Oswaldo Cruz CESTEH/ENSP/FIOCRUZ-RJ; [2]Department of Environmental Health, Faculdade de Saúde Pública, Universidade de São Paulo*

10.1136/oemed-2018-ICOHabstracts.829

Introduction The world of work has undergone significant changes, with the adoption of new technologies and the use of various chemicals and minerals in their processes and production methods, expanding and increasing the environmental hazards. A bibliographic survey about deleterious effects on exposure to the chemicals lead and cadmium was conducted in order to verify how this exposure may influence the population's sleep-wake cycle.

Methods This is an exploratory study through open access literature review, of systematic type. Search was conducted in the period from 2001 through October 2016. Search strategy has included the use and combination of descriptors and terms: Chemical Exposure; Exposure to Metals; Exposure to Lead; Exposure to Cadmium; Sleepiness; Sleep Disorders; Sleep Disturbances; Sleep-Wake Cycle.