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.

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

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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

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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.

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