Introduction In order to prevent neurodegenerative diseases such as chronic solvent encephalopathy (CSE), it is important to elucidate the underlying mechanism. Cells are able to respond to occupational stressors by turning on or off specific genes mediated through epigenetic alterations. Consequently, epigenetic mechanisms can play an important role in mediating the effects of solvent exposure on the development of neurobehavioral disorders.

Methods We have set-up several studies in a translational design using cell lines, mice and humans exposed to solvents and CSE patients. DNA methylation was measured with liquid chromatography coupled with tandem mass spectrometry for the assessment of 2'-deoxycytidine, 5-methyl-2'-deoxycytidine, 5-hydroxymethyl-2'-deoxycytidine, 5-formyl-2'-deoxycytidine, 5-carboxy-2'-deoxycytidine and pyrosequencing.

Results Global DNA methylation and hydroxymethylation was mostly negatively associated with solvent exposure in both *in vitro* as *in vivo* studies. DNMT1 CpG1 gene-specific methylation levels were significantly higher in CSE patients as compared to controls, associations that persisted after adjustment for age, gender, alcohol abuse, smoking, level of education and the use of psychotropic medication ($p=0.004$ and $p=0.017$, respectively). In addition, in the group of CSE patients there was a positive correlation between the duration of solvent exposure and the percentage of BDNF CpG1 methylation ($rs=0.374$, $p=0.017$), which was maintained after adjustment for age and smoking ($p<0.001$).

Conclusion The study of epigenetic alterations gives us the possibility to study the immediate consequences of exposure and make projections for disease development. These epigenetic marks can be used to develop a set of biomarkers and used in the follow-up of humans.

1713d OCCUPATIONAL EXPOSURE TO SOLVENTS AND RISK OF PARKINSON DISEASE IN FINLAND

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Background Prior epidemiologic and animal studies indicate that occupational exposure to solvents, particularly trichloroethylene, may increase risk of Parkinson disease (PD).

Methods We constructed a population-based case-control study of incident PD in Finland using a medication database maintained by the Social Insurance Institution of Finland, along with the Population Information System, which includes census records for all Finnish residents. PD cases were diagnosed (first approved to receive PD medications) between 1995–2014. We randomly selected controls from the underlying population while matching on diagnosis year, birth year (1930–1950), and sex. Individual occupational census data from 1990, allowing for a minimum of five years of exposure lagging, were available for 11,757 PD cases and 23 236 controls. These data were linked to the Finnish Job Exposure Matrix (FINJEM) to identify potential for occupational exposure to four classes of solvents. We estimated PD-solvent odds ratios (ORs) and 95% confidence intervals (CIs) using unconditional logistic regression, while adjusting for matching variables and socioeconomic status as a proxy for tobacco smoking.

Results Cases and controls were age 45–89 years at diagnosis/reference and age 40–60 years at occupational exposure

assessment. There was no evidence that PD was associated with occupational exposure to solvents overall (19% cases and 20% controls), or to aromatic hydrocarbon solvents in particular, with ORs very close to null. In contrast, there was a modestly higher PD risk in relation to exposure to chlorinated hydrocarbon solvents, most notably trichloroethylene (OR=1.11, 95% CI: 0.98 to 1.26). Occupation as a mechanic (OR=1.20, 95% CI: 0.97 to 1.49) or as an electronic or telecommunications worker (OR=1.65, 95% CI: 1.07 to 2.54) contributed to this association.

Conclusion Consistent with prior literature, occupational exposure to trichloroethylene may at least modestly increase risk of PD.

1716 ENVIRONMENTAL INTOLERANCE – A CONTINUUM FROM ANNOYANCE TO SEVERE ILLNESS

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Aim of the special session Intolerance may develop to various everyday environmental exposures at levels that are well below those known to cause adverse health effects. Reactions initiate typically from odorous substances, but also from non-perceivable factors considered harmful to health, e.g. electrical devices. Reactions range from unpleasant sensations and annoyance to multi-organ symptoms, severe disability, and major restrictions in daily life and work. Symptoms often lead to exposure assessments at work and may result in excessive actions to eliminate minor exposures. In environmental intolerance, occupational health care is the front line actor in primary and secondary prevention, and support of recovery.

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1716a ENVIRONMENTAL INTOLERANCE – DEFINITIONS AND RELEVANCE TO OCCUPATIONAL HEALTH

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Number of people attribute symptoms to everyday environmental exposures at levels that are well below those known to cause adverse health effects and that are tolerated by the majority. Intolerance may develop to various environmental