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 tricholoethylene 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

factors, typically to odorous chemicals, but also to non-perceivable factors considered potentially harmful to health, e.g. electrical devices. Reactions due to intolerance range from unpleasant sensations or annoyance to disabling symptoms, which may lead to serious restrictions in daily life. Reactions often initiate exposure assessments in work, home or public environment and may lead to excessive actions to eliminate minor exposures.

In 1996, the WHO classified all medically unexplained conditions attributed to different environmental exposures under the term idiopathic environmental intolerance (IEI), regardless of the factor in question (IPCS 1996). The factors may include e.g chemicals, moulds. The term is most commonly used for multiple chemical sensitivity (MCS), but also nonspecific building-related symptoms (or sick building syndrome), hypersensitivity to electromagnetic fields (EMFs) are regarded as its subtypes. Although the various types of environmental intolerance (EI) share common core characteristics, there is no generally agreed definition of the condition.

The prevalence estimates are based on self-reporting. In adults, the prevalence estimates of EI attributed to chemicals vary from 9% to 52%, to EMFs from 1.5% to 21%, and to sounds from 8% to 39% (Karvala, *et al* 2017, submitted).

Environmental intolerance manifests as different degrees of annoyance, which shows a continuum with increasing symptoms, behavioural changes and disability.

IPCS/WHO (1996). Conclusions and recommendations of a workshop on Multiple Chemical Sensitivities (MCS). International Program on Chemical Safety/World Health Organisation. Regul Toxicol Pharmacol 24:188–189.

Keywords: Idiopathic environmental intolerance, reactions to environment, multiple chemical sensitivity, sick building syndrome, sensitivity to electromagnetic fields

1716b LOW LEVEL CHEMICAL EXPOSURES – WHY DO SOME INDIVIDUALS DEVELOP HEALTH SYMPTOMS WHEREAS OTHERS DO NOT?

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People are on a daily basis exposed to a variety of odorous and pungent substances. For a person with severe chemical intolerance (CI) or building-related intolerance (BRI) such exposure can result in substantial suffering and reduced quality of life. Symptoms are reported in relation to low-level chemical exposures and there is currently no established dose-response relationship between exposure to certain compounds and reports of symptoms. Most of the volatile organic compounds (VOCs) identified in indoor air are non-reactive and chemicals that might be more important for symptom reports require specific sampling and analytical methods and are therefore probably not included in the measurements. The overall aim was to investigate the role of individual factors in the development of health symptoms due to exposure to low levels of VOCs.

Data from a cross-sectional field study investigating individuals diagnosed with BRI will be presented as well as data collected from controlled exposures in an exposure chamber. By combining data on individual factors with data from new, more sensitive methods for measurement of organic compounds we are able to study indoor air health problems in a new light. There is a large individual variation in the response to exposures to certain reactive compounds. Exposure-related factors such as type of compound and duration of exposure are of importance. One example is the reactive compound acrolein that induced sensory irritation in a time-dependent manner at a concentration below previously reported detection levels and at half the Swedish occupational threshold limit. Factors related to the individual such as CI, stress or inflammation are also of importance for reports of sensory irritation due to low level chemical exposures. Further, negative affect and information about the exposure also mediate annoyance and symptoms. In order to understand sensory irritation from low-level exposure to VOCs we have to take both individual- and environmental factors into account.

1716c IS ENVIRONMENTAL INTOLERANCE RELATED TO CHEMICAL OR ODOUR HYPERSENSITIVITY? A SUMMARY OF EPIDEMIOLOGICAL AND EXPERIMENTAL EVIDENCE

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Multiple chemical sensitivities (MCS) nowadays more often called idiopathic environmental intolerance (IEI) is characterised by the recurrent occurrences of multiple symptoms involving in multiple organ systems. With respect to the aetiology of IEI toxico-, immune-, and psychogenic theories have been proposed but the exact physiological mechanisms underlying this syndrome are fare from being conclusive. The complex health complaints are triggered by environmental chemicals in very low doses and the upper respiratory tract and the nervous system are two of the most prominent organs of these health complains. Due to the fact that these 'trigger concentrations' are usually tolerated by most persons, genetic (e.g. slower detoxification) and acquired hypersensitivities (odour sensitivity) are thought to be characteristic features of IEI patients. In questionnaires assessing sub-clinical levels of chemical intolerance items addressing odour sensitivity are often used. Our experimental research showed that such scores predict a more unidimensional hedonic evaluation (e.g. malodors) of chemicals. Moreover, differentiation between odours was reduced. In line with other researchers we also showed that such self-descriptions are not associated with better olfactory acuity assessed with standardised psychophysical methods. When combining our own experimental findings with epidemiological studies on IEI we came to the conclusion that chemical or odour hypersensitivity is not an increased sensitivity of chemosensory pathways, but it appears to be a uniform and affective style of responding to low-level exposures to volatile chemicals. Such an undifferentiated response might also explain the fact the chemical triggers of IEI symptoms span from natural fragrances, to perfumes, to industrial chemicals like solvents.