substrates including some glutathione–metal complexes they might protect cells by extrusion of metal complexes. The ubiquitously expressed mammalian ABC-halftransporter (UMAT) has high sequence similarity to the fisson yeast heavy metal tolerance protein hmt1, thus it is assumed to be involved in metal ion homeostasis.

We had the rare opportunity to conduct long-term experiments with primary cultures of NHBEC from three patients (B182/1, B201/1, B222/1) which we incubated with As(III), Cu(II) and Hg(II) in concentrations proven to be non-toxic in the MTT-assay before. Cells were cultivated for 6 weeks in the presence of the investigated metals and RNA was isolated after each splitting. The expression of different MRP-isoforms and UMAT was determined by real-time RT-PCR.

The first week of treatment with As(III) down-regulated MRP1 (0.5-fold), MRP3 (0.6-fold; $p < 0.01$) and MRP4 (0.6-fold; $p < 0.05$) in B201/1. In cultures derived from B182/1 treatment with As(III) led to an up-regulation of MRP3 (1.3-fold), MRP4 (1.8–2-fold) and MRP5 (2.3-fold). After the second week of treatment with As(III) the expression of MRP1 (0.4-fold; $p < 0.01$), MRP3 (0.5-fold; $p < 0.01$; 2.5 μM), MRP4 (0.4-fold) and MRP5 (0.5-fold) was decreased in B201/1. In the cultures derived from B182/1 MRP1 was down-regulated significantly (0.75-fold, $p < 0.05$). The first week of incubation with Cu(II) significantly down-regulated the expression of MRP1 (0.6-fold; $p < 0.05$) in B201/1. In B182/1 incubation with Cu(II) slightly increased expression of MRP3. Incubation (5 days) of NHBEC B201/1 with Hg(II) significantly down-regulated MRP1-expression (0.5-fold; $p < 0.01$; 2.5 μM), whereas in B182/1 Hg(II) increased expression of MRP3 significantly (2.1-fold, $p < 0.05$). Comparing the results of cultures derived from different patients clear inter-individual differences in the reactions to As(III), Cu(II) and Hg(II) become obvious.

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Evaluation of biomarkers of exposure and effects of mercury using machine-learning methods

Darija Gibičar[1], Dragi Kocev[2], Bernard Zenko[2], Milena Horvat[1], Saso Dzeroski[2], Vesna Fajon[1], Barbara Mazzola[3]

[1] *Institute "Jozef Stefan", Department of Environmental Sciences, Jamova 39, 1000 Ljubljana, Slovenia;* [2] *Institute "Jozef Stefan", Department of Knowledge Technologies, Ljubljana, Slovenia;* [3] *Scuola Superiore Sant'Anna, CRIM Laboratory, Pisa, Italy*

During the EU funded project European Mercury Emissions from Chlor Alkali Plants (EMECAP) we evaluated the exposure to mercury in subjects living close to the mercury cell chlor-alkali (MCCA) plant and in occupationally exposed MCCA workers, compared to controls from the reference areas. Beside urinary mercury as a biomarker of exposure also biomarkers of effect were assessed: albumin, alpha-1-microglobuline (A1M) and *N*-acetyl-beta-D-glucosaminidase (NAG) in urine as indicators of kidney function damage and 8-hydroxydeoxyguanine (8-OH-dG) in urine as an indicator of DNA damage. In addition selenium in urine was analysed because of its known antioxidant protective role.

The dataset consisted of total 269 subjects: 57 chlor-alkali plant workers, 94 subjects living in 1.5 km diameter from the chlor-alkali plant, and 118 controls living 20 km south of the plant. All subjects completed a questionnaire about the location of their residence and workplace, occupational history including possible exposure to mercury, the number of teeth with amalgam fillings, as well as consumption of various types of fish, smoking habits, consumption of vegetables from their backyard gardens and questions connected to their medical history. Subjects with kidney diseases, diabetes, hypertension or extreme levels of creatinine were excluded from the data analysis.

We used one-way ANOVA to evaluate the differences between the groups in all observed parameters. In order to find associations between the attributes, machine-learning methods were used. We used WEKA's model trees and regression trees, which were validated using 10-fold cross-validation.

Results have shown significantly higher concentrations of mercury and NAG in the urine of chlor-alkali workers and lower concentration of their urinary selenium, compared to the subjects living close to the MCCA and the controls.

Urinary mercury was positively associated with number of teeth with amalgam fillings and negatively associated with age of the subjects and working years in the plant. Associations between A1M and NAG concentrations, teeth with amalgam fillings and urinary selenium were found.

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W3 Current Issues in Air Pollution and Health

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Toxicity of automotive fine and ultrafine particles

F.R. Cassee

National Institute for Public Health and the Environment, Bilthoven, The Netherlands

Ambient particulate matter (PM) has been linked with augmentation of respiratory and cardiovascular diseases and increased mortality rates. In particular diseased people (e.g. asthmatics) and elderly are at increased risk. Traffic exhaust, but also wood smoke has been suggested to contribute in particular to these health effects. The wealth of recent toxicological studies provides sufficient plausibility to explain the modes of action and underlying mechanism of these health effects. However, there has been little or at least conflicting evidence that the toxic effects also occur at lower, closer to ambient levels of PM. In addition, toxicology has shown that at an equal mass basis PM from different places or sampled at different seasons have different toxic properties. It has also been shown that size matters: particles <0.1 um seem to be more toxic than larger, yet still inhalable particles. There is evidence for source specific particle toxicity. The currently dominated discussion relates to combustion type particles resulting from various sources such as traffic (gasoline and diesel engine exhaust, lubrication oil, tire and brake ware dust). Besides that there is evidence that mineral dusts re-dispersed from, e.g. road surfaces express different toxic potentials. Even though there still are admitted gaps of understanding, the general principle of induction of the viscous circle of oxidative stress and inflammation provides a reasonable causality of almost all respiratory and cardiovascular diseases associated with PM exposure. This paper will review recent toxicological information on traffic related ambient PM.

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School children's exposure to ambient air pollution in Bangkok, Thailand

Mathuros Ruchirawat

Chulabhorn Research Institute, Bangkok 10210, Thailand

Urban air pollution resulting from traffic is a major problem in many cities in Asia including Bangkok, Thailand. These rapidly expanding cities tend to have significantly more traffic congestion than most others. Such pollution originates mainly from incomplete fossil fuel combustion, the composition of which is very complex and some elements of which are carcinogenic in experimental animals and in man. Polycyclic aromatic hydrocarbons (PAHs) and benzene are among the major carcinogenic compounds found in urban air pollution from motor vehicle emissions.

In major cities in Asia, the levels of PAHs and benzene are relatively high compared with those in Europe or in the United States. People living in such cities, therefore, are exposed to higher levels of these carcinogenic pollutants.

The potential health effects of exposure to PAHs and benzene in air pollution have been studied in school children attending schools in inner-city Bangkok compared with those attending schools in the rural areas.

Bangkok school children are exposed to total PAHs at levels more than six-fold higher than those in the rural area. Urinary 1-hydroxypyrene, a metabolite of PAH, was also significantly higher, while PAH-DNA adducts in lymphocytes were four-fold higher in Bangkok children than rural school children.

Benzene exposure in Bangkok school children was approximately two-fold higher than in rural school children. This is in agreement with the levels of biomarkers of internal benzene dose, i.e. blood benzene and urinary *t,t*-muconic acid.

The potential health risks from exposure to genotoxic substances were assessed through DNA-damage levels and DNA repair capacity. DNA strand breaks and 8-OHdG levels were significantly higher, whereas DNA repair capacity was significantly reduced in Bangkok children. This indicates that children living in major cities may have an increased health risk of the development of certain diseases due to exposure to genotoxic substances in air pollution.

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