TP255 Organophosphate Flame Retardants in Car Indoor Environment

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Most people in modern societies spend approximately 90% of their time indoors. Among indoor environments, a car indoor environment has become increasingly essential, since a car has become the main form of transportation in our society. People spend 5% of their time in the car, which proportion is next to home and office. The air in the car cabin is polluted by the emission of organic compounds from interior materials within the car. In the car, carpets, paints, plastics and foam in the car seat can be an air pollution sources. In the car interior materials, organophosphate flame retardants (OPFRs) are added to prevent fire hazards. Nature of the car cabin construction, high surface area compared to small air volume, easily leads to the increase of the chemical concentration emitted from car interior materials. Because OPFRs possess toxicity such as delayed neurotoxicity, carcinogenicity and brain degenerative lesions, high concentration of OPFRs may cause adverse health effects on passengers. In this study, the concentrations of OPFRs in the gas phase and in dust were measured in 26 unoccupied cars. As target OPFRs, 2-ethylhexyldiphenyl phosphate (EHDP), tris(2-butoxyethyl) phosphate (TBEP), tris(n-butyl) phosphate (TBP), tris(2-chloroethyl) phosphate (TCEP), *o*-tricresyl phosphate (*o*-TCP), *p*-tricresyl phosphate (*p*-TCP), tris(1-chloro-2-propyl) phosphate (TCPP), tris(1,3-dichchloroisopropyl) phosphate (TDCPP), tris(ethyl) phosphate (TEP), tris(2-ethylhexyl) phosphate (TEHP), tris(propyl) phosphate (TPrP), tris(phenyl) phosphate (TPP) were selected. In the air in car cabins, TCPP was the most frequently detected (100%) and had the highest concentration (mean concentration of 178.5 ng/m^3; highest concentration of 1502 ng/m^3) among the OPFRs measured in this study. The results of OPFRs concentration in dust collected from car cabins show that TCPP, TCEP, TBP, and TBEP were detected with high frequency (< 50%). TBEP showed the highest mean concentration in dust (86.8 mg/g). Due to low-volatility of OPFRs, OPFRs prefer dust rather than the gas phase. Therefore, various OPFRs were detected in dust.

TP256 Elucidating the effects of alternative flame retardants, TBB and TBPH on thyroid hormone synthesis, steroidogenesis, and estrogen receptor binding

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TBB (2-ethylhexyl-2,3,4,5-tetrabromobenzoate) and TBPH (2-ethylhexyl-2,3,4,5-tetrabromophthalate) are compounds widely used as flame retardants in several commercial products, e.g., Firemaster® 550, BZ-45, and DP-54. Both have been detected in various environments such as indoor dust, atmosphere, municipal sewage, sediment, and even in wildlife. Since TBB and TBPH are highly lipophilic (LogKow 8.75 and 11.95, respectively), they have been suspected to accumulate and persist in the body. However, limited information is available on their toxicological mechanisms as endocrine disruptors. Therefore we sought to investigate whether these chemicals have adverse impacts on endocrine system including thyroid and sex hormone regulations. To this end, we exposed TBB and TBPH to GH3 (rat pituitary cell), H295R (human adrenocortical carcinoma cell), and MVLN (human breast cancer cell), which are model systems for in vitro assessment of endocrine disruption. Exposure concentrations were determined by performing cell viability test. In GH3, exposure to TBB (0.05, 0.5, 5, and 50 mg/L) and TBPH (0.5, 5, 50, and 500 mg/L) changed the mRNA expression of *TSH*β, *TR*α, and *TR*β genes involved in thyroid hormone synthesis. In general, exposure to TBB caused the decreasing trends in mostmRNA levels and the transcription of *TR*α significantly decreased at the highest concentration (50 mg/L). TBPH also changed the mRNA expressions though it was not significant. In H295R exposed to 0.00001, 0.0001, 0.001, 0.01, 0.1, and 1 mg/L of TBB, estradiol production significantly increased in a dose-dependent manner. MVLN was also employed to elucidate the estrogenic effect of TBB and TBPH with affinity for estrogen receptor. The results of this study contribute to the general understanding of endocrine disrupting potential of TBB and TBPH and its related mechanisms.

TP257 Gene expression analysis of saturated aliphatic aldehydes reveals carbon number-specific molecules involved in pulmonary toxicity

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The aim of this study was to determine the transcriptomic responses and identify specific molecular signatures of Low-molecular-weight saturated aliphatic aldehydes (LSAAs). To evaluate the change in gene expression levels, A549 human alveolar epithelial cells were exposed to six LSAAs (propanal, butanal, pentanal, hexanal, heptanal, and octanal) for 48 h. Through clustering analysis of gene expression profiles, we found that the low carbon number group (LCG), relating to propanal, butanal, and pentanal, was distinguished from the high carbon number group (HCG), relating to hexanal, heptanal, and octanal. Also, transcriptomic profiling shows higher sensitivity to gene alteration in LCG exposure group than HCG group. Supervised analysis revealed 703 LCG specific genes and 55 HCG specific genes. After Gene Ontology (GO) analysis on LCG specific genes, we determined several key pathways which are known as related to increase pulmonary toxicity such as cytokine-cytokine receptor interaction and chemokine signaling pathway. But we did not find pulmonary toxicity-related pathways through GO analysis on HCG specific genes. Genes that are expressed in only low carbon LSAAs exposure group were regarded as biomarker of aldehydes-induced pulmonary toxicity. In conclusion, this study describes changes in gene expression profiles in in vitro respiratory system in response to exposure to 6 LSAAs with different carbon number and relates these gene expression changes to pulmonary toxicity related pathways. Moreover, novel carbon number-specific genes and pathways can be more widely implemented in combination with more traditional technique for assessment and prediction of exposure to environmental toxicants.

TP258 Comparing predicted and measured concentrations of neurological drugs in the effluents of selected health institutions

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Due to demographic change human population gets more affected to neurological diseases. This is accompanied by an increasing use of neurological drugs (NDs). There is a discussion about wastewater sources from households and health institutions (HIs) as major contributors of active pharmaceutical ingredients (APIs) in influents of sewage treatment plants (STPs). Recently, strategies are requested to extend the focus from end of pipe solutions at STPs to integration of strategies for reducing pollution at the source. To address this research question, this study demonstrates that the calculation of predicted wastewater concentrations (PWWCs) in HIs could be a feasible approach to identify point sources and their potential API contributions. PWWCs of six highly consumed NDs (pregabalin, gabapentin, levetiracetam, amisulpride, doxepin and quetiapine) were calculated by means of consumption data of a psychiatry, a nursing home and a general hospital, the corresponding not-metabolized fraction for the APIs and the annual water consumption within the years 2010 and 2012. A range for PWWC was given with respect to interindividual differences in human metabolism and consumption variations during investigated years. Additionally, these selected APIs were analytically quantified in the HIs' effluents for three different seasons of the year to validate PWWC. Samples were taken every two hours during the day to obtain 24 h-mixed samples. Analytical quantification was conducted by SPE-LC-MS/MS with the standard addition method to compensate varying matrix effects. Analytically measured concentrations for APIs were almost congruent with PWWCs. The predicted range for five APIs was mostly less than one order of magnitude different from analytical results. Only PWWCs of Gabapentin were estimated up to almost 40 times higher. Since only Gabapentin was estimated wrong, this indicates that analytical measurements could