CHARACTERIZATION AND QUANTIFICATION OF DIOXIN-LIKE COMPOUNDS IN FLUE GAS CONDENSATES OF MUNICIPAL SOLID WASTE INCINERATORS

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Introduction

The toxic effects of mixtures of polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/PCDF) congeners are described by weighing the concentration of the 17 toxic congeners by toxicity equivalence factors (TEF). TEF also have been established for PCB congeners showing dioxin-like toxicity. A direct measurement of dioxin-like toxicity is feasible by measuring enzyme induction in cell cultures, e.g in the EROD-test[1, 2]. Dioxins can be distinguished from other compounds inducing the same cytochrome P450 related enzyme systems by their persistent (>72h) induction effects. PAH and other biodegradable enzyme inducing compounds are assumed to be metabolised under the test conditions within that time.

Extracts from municipal solid waste incineration fly ashes showed 2 - 5 times higher dioxin-like toxicity in enzyme induction tests than calculated according to TEQ schemes[3, 4]. Also aqueous condensates from MSWI flue gas samples showed higher toxic effects than explained by the PCDD/PCDF concentrations[5]. The authors assumed unknown toxic compounds to cause the differences observed.

As emissions of unknown toxic compounds from MSWI plants are an important issue in discussions on waste incineration, we investigated which compound groups might be responsible for the differences.

Methods

The flue gas condensates of two municipal waste incinerators (plant “B”; plant “C”)were sampled for a period of approx. 10 days. Water contained in the flue gas was condensed in a cooled probe and in a high-efficiency cooler at max. 20°C (Fig. 1). Only the condensate was collected because quantitatve

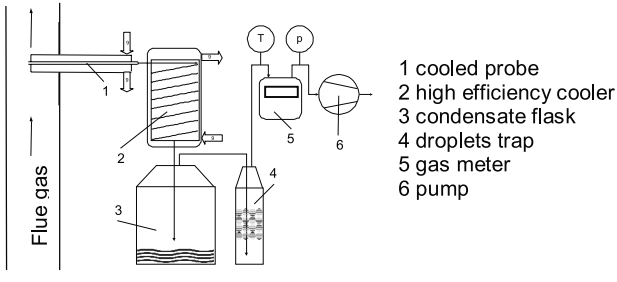

Figure 1. Sampling device

sampling was not necessary and the condensate at the low temperatures contains most of the semi- and non volatile compounds. At plant B two samples were taken, the other plant (C) was sampled once.

The condensates (7-30L) were extracted in triplicate with dichloromethane and the unified extract was concentrated to 50 mL. PCDD/PCDF were analysed in aliquots of the samples. Other aliquots of the original extract were analysed without clean-up by micro-EROD test[2] and fractionated by extraction with K_2CO_3-solution and by chromatography on alumina B super I (ICN) and step elution with hexane/dichloromethane 2 %, hexane/dichloromethane 10%, hexane/dichloromethane 50 % and pure dichloromethane (Fig. 2). The K_2CO_3-solution was acidified and re-extracted with hexane. Fractionating for EROD-test was done without addition of isotope labelled internal standards

All fractions were tested by the EROD test. Fractions 1,2, and 3 were analysed on PCDD/PCDF and coplanar PCB.

Results

The PCDD/PCDF concentrations in the flue gas condensates ranged between 3.2 and 18 ng/L corresponding to 0,7 to 1.2 ng/Nm³ in the flue gas. This is approx. 1/3rd of concentrations which were measured at plant B at another time.

All three flue gas condensates showed significant higher toxicity in the EROD test than explained by PCB and PCDD/PCDF concentrations (WHO TEF-scheme[6]).

The results of the EROD test ranged between 9,5 and 25ng TEQ/L. At plant B, 43 respectively 83% of the EROD could be explained from the PCB+PCDD/PCDF concentration. At plant C, PCB+PCDD/PCDF correspond to 34% of the EROD value. The differences are somewhat lower than reported for fly ash.

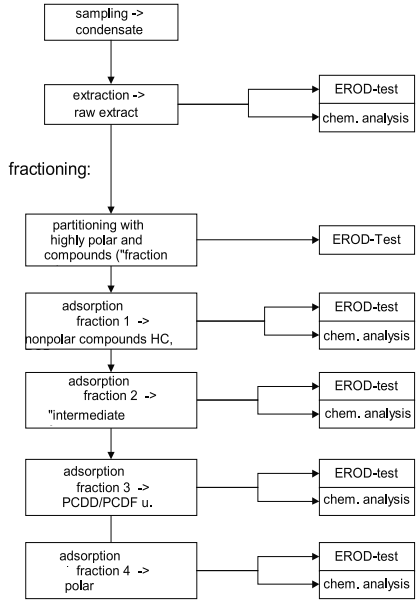

Figure 2. Fractionating scheme

The fractions F1 and F2 of all samples did not contribute significantly to total toxicity, PCB values were very low.

The PCDD/PCDF concentrations were similar to the EROD-results of the same fraction (F3). In figure 3 the residual difference (delta F3) is highlighted.

The polar fractions F4 showed no enzyme induction in the EROD-test.

A small part of the total EROD-activity was found in the fraction F5 which contains very polar and acidic compounds. This fraction did not contain PCB or PCDD/PCDF.

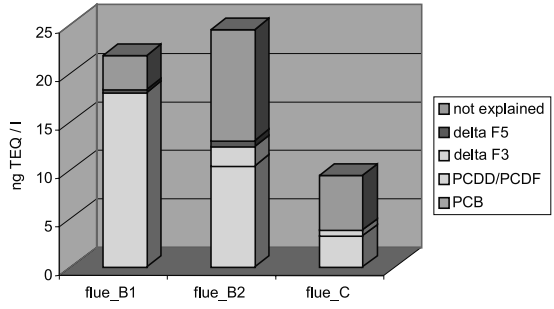

Figure 3. Results of EROD tests and partitioning to the fractions F1-F5

The sum of EROD activities in the fractions is much lower than the total EROD activity of the unfractionated extract. This gap in the EROD balance of the fractionation test cannot be explained yet. Additional tests showed the first step in the fractionation scheme to remove much of the EROD activity although it is not possible to recover the activity upon re-extraction.

Fraction F3 should contain not only polychlorinated dibenzodioxins and dibenzofurans but also mixed halogenated PXDD and PXDF. Additional target analyses have been run by GC-HRMS on the fraction F3 samples. Only small amounts of bromine containing PXDD and PXDF were detected. Most abundant homologues were BrCl3DF and BrCl4DF. Br2Cl4DF was detected in very small traces, not allowing a confirmation by fragment ions. Additionally in the samples from plant B, traces of ICl3DF were identified. Concentrations of ICl3DD were too low to confirm the identity by fragment ions.

Assuming the toxicity of 2,3,7,8-BrCl3DF to be similar to the toxicity of 2,3,7,8-Cl4DF and estimating the amount of the 2,3,7,8-substituted congeners to be max. 30 % of the total BrCl3DF, a maximum of 0,5 ng TEQ/L can be attributed to mixed halogenated PXDD/PXDF in sample A2. As shown in figure 4, this is only 5 % of the PCDD/PCDF-TEQ in fraction F3.

Target analyte search for other substances, which might contribute to enzyme induction, such as olychloroxanthenes, polychloronaphthalenes and polychlorodiphenylethers had no positive result. Additionally, scanning GC-LRMS chromatograms were examined without finding a indication for

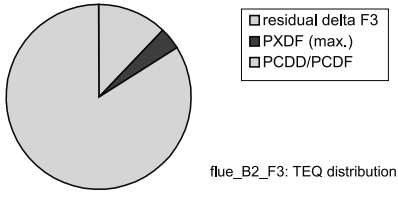

Figure 4. Contributions to TEQ in sample B2, fraction F3

other compounds which might exhibit dioxin-like effects (figure 5). Remarkable are the large peaks caused by mercury iodide in the raw extracts of all samples. HgI_2 usually is not identified in flue gas. Possibly the formation of HgI_2 occured from $HgCl_2$ and traces of iodides during the long sampling period. HgI_2 is eliminated from the extract upon the first fractionating step.

Conclusions

Examination of flue gas condensate extracts gave an estimation of the influence of mixed halogenated PXDD/PXDF to total dioxin-like toxic effects. The comparison of EROD-test results with GC-HRMS analysis shows total dioxin-like toxicity being approx. 2.5 times the WHO-TEQ of the samples. It has not been possible to identify single compounds as a cause for this deviation. Considering the effectiveness of the flue gas cleaning system applied this gives no indication for large amounts of unknown dioxin-like toxic compounds in the stack gas.

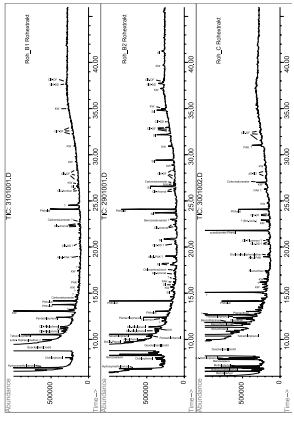

Figure 5. Reconstructed total ion current chromatograms of the raw flue gas condensate extracts

Acknowledgement

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