THE USE OF MULTIMEDIA MODELS FOR HEALTH RISK ASSESSMENT TO PCDD/FS

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Introduction

Chemicals emitted from an atmospheric source as air emissions are directly transmitted to humans through air inhalation. However, these chemicals can also cross environmental media boundaries, becoming distributed in different media: soil, vegetation, water, biota, etc. Consequently, human health can be indirectly affected through different exposure pathways such as drinking water or groundwater, skin absorption of the chemicals present in water (i.e., bath, river, lake), eating foodstuffs, as well as ingestion and skin absorption of chemicals adsorbed to soil. Hence, for accurate health risk estimates, chemical concentrations in each of these environmental media must be determined.[1,2]

There are still considerable uncertainties about the relative importance of atmospheric sources of PCDD/Fs and their fluxes into the environment. This uncertainty undermines efforts to relate sources to air concentrations and will hamper future efforts to further reduce human exposure to these pollutants. There is evidence that atmospheric source reductions of PCDD/Fs have been occurring in recent decades, with atmospheric concentrations in "atmospherically impacted" media (e.g., vegetation, cow's milk), human dietary intake, and body burdens all showing significant declines. It is interesting to note that data from sediment cores and archived samples suggest that peak inputs of PCDD/Fs to the environment probably occurred in the late 1960s/early 1970s.[3,4,5] However, the large-scale efforts at primary source reduction were not initiated until later in Spain (and other countries), with efforts to tackle emissions from municipal solid waste incinerators (MSWI) and other important primary sources. It has important implications, because it suggests that we do not adequately understand why PCDD/Fs levels have been reduced and which sources/measures were responsible for the observed declines.

Since 1975, a MSWI is operating in Montcada (Barcelona, Spain). Until recently, an electrostatic precipitator was used as emission control device. In March 1999, a modernisation of the flue gas cleaning system was carried out. An acid gas (HCl/SO_2) and metal emission limit equipment was installed and an active-carbon adsorption filter was added to the fabric filter. As a consequence of this, PCDD/F emissions have been notably reduced.[6] However, the MSWI is located in an active industrial zone, which has also a heavy traffic. Therefore, other PCDD/F emissions additional to those from the MSWI should be also present in the area near the facility. These emissions sources could mask and/or distort the environmental effects of the pronounced reduction in the emissions of PCDD/Fs from the MSWI. The aim of this study was to calculate the incremental lifetime-risk to PCDD/Fs for the residents living in the surroundings of the MSWI.

Methods and Materials

Analytical (24 soil and 24 herbage samples) and modelled data were obtained. PCDD/F concentrations in air near the MSWI due to the plant emission were quantified by the application of a

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Gaussian dispersion model (ISCST-3). PCDD/F concentrations in the environmental media were determined by means of a simple-compartment-multimedia model (air-soil-vegetation model). Since different physical and chemical properties have been demonstrated for the 17 toxic congeners, they have been considered as different compounds. Soil and vegetation models were validated and a quite good agreement for I-TEQ values was obtained.[7] Predicted and measured dioxin concentrations in soils and vegetation were compared, and the effect of the MSWI emissions in the environmental media was determined. Finally, human health risks due to PCDD/F emissions from the incinerator were estimated based on I-TEQ measured and modelled in various environmental media. The quantitative estimation of health risks due to PCDD/F exposure is generally considered to be a combination of five pathways: 1) ingestion of contaminated soil, 2) ingestion of vegetables from the area, 3) inhalation of re-suspended soil particles, 4) air inhalation, and 5) dermal absorption. Although ingestion through the diet is known to be the major pathway of exposure to PCDD/Fs, it has not been here considered since the area of study is an industrial area with no agricultural or animal production.

Results and discussion

Tables 1, 2 and 3 summarise the mean air, vegetation and soil PCDD/F concentrations, respectively. Simulated and measured mean concentrations due to MSWI emissions, corresponding to 24 sampling points within a radius of 3 km from the stack are shown.

Table 1. Immision levels of PCDD/Fs (fg/m^3).

	$C_{estimated}$			$C_{measured}$*
	C_{gas}	$C_{particle}$	C_{Total}	
2,3,7,8-TCDD	0.0004	0.001	0.001	4.1
1,2,3,7,8 PeCDD	0.001	0.003	0.004	7.2
1,2,3,4,7,8-HxCDD	0.002	0.003	0.005	7.2
1,2,3,6,7,8-HxCDD	0.005	0.009	0.014	13.6
1,2,3,7,8,9-HxCDD	0.001	0.011	0.012	27.9
1,2,3,4,6,7,8-HpCDD	0.009	0.078	0.087	70.7
OCDD	0.009	0.099	0.108	158.0
2,3,7,8-TCDF	0.004	0.001	0.005	91.2
1,2,3,7,8 PeCDF	0.001	0.001	0.002	10.2
2,3,4,7,8 PeCDF	0.002	0.002	0.005	35.8
1,2,3,4,7,8-HxCDF	0.004	0.005	0.008	78.7
1,2,3,6,7,8-HxCDF	0.003	0.005	0.008	29.9
1,2,3,7,8,9-HxCDF	0.000	0.000	0.000	1.8
2,3,4,6,7,8-HxCDF	0.003	0.013	0.017	44.8
1,2,3,4,6,7,8-HpCDF	0.006	0.032	0.038	131.0
1,2,3,4,7,8,9-HpCDF	0.001	0.006	0.006	10.5
OCDF	0.002	0.054	0.056	101.0
I-TEQ	0.005	0.009	0.014	57.99

Data provided by the Generalitat de Catalunya

Table 2. Levels of PCDD/Fs in vegetation (ng/kg).

Congeners	$C_{simulated}$	$C_{measured}$	% variation
2,3,7,8-TCDD	0.0001	0.0550	0.21
1,2,3,7,8 PeCDD	0.0005	0.2000	0.26
1,2,3,4,7,8-HxCDD	0.0002	0.1000	0.15
1,2,3,6,7,8-HxCDD	0.0003	0.3500	0.09
1,2,3,7,8,9-HxCDD	0.0004	0.2500	0.17
1,2,3,4,6,7,8-HpCDD	0.0035	3.1000	0.11
OCDD	0.0076	7.5000	0.10
2,3,7,8-TCDF	0.0010	0.7000	0.14
1,2,3,7,8 PeCDF	0.0007	0.4500	0.16
2,3,4,7,8 PeCDF	0.0010	0.5000	0.20
1,2,3,4,7,8-HxCDF	0.0009	0.4000	0.23
1,2,3,6,7,8-HxCDF	0.0006	0.4000	0.16
1,2,3,7,8,9-HxCDF	0.0000	0.0250	0.19
2,3,4,6,7,8-HxCDF	0.0011	0.4000	0.27
1,2,3,4,6,7,8-HpCDF	0.0024	1.6000	0.15
1,2,3,4,7,8,9-HpCDF	0.0002	0.1500	0.12
OCDF	0.0007	1.3000	0.06
I-TEQ	0.0014	0.7040	0.20

Table 3. Levels of PCDD/Fs in soils (ng/kg).

Congeners	$C_{simulated}$	$C_{measured}$	% variation
2,3,7,8-TCDD	0.0005	0.09	0.52
1,2,3,7,8 PeCDD	0.005	0.50	1.00
1,2,3,4,7,8-HxCDD	0.011	0.55	2.02
1,2,3,6,7,8-HxCDD	0.026	1.00	2.58
1,2,3,7,8,9-HxCDD	0.040	0.90	4.39
1,2,3,4,6,7,8-HpCDD	0.319	13.50	2.36
OCDD	0.513	64.00	0.80
2,3,7,8-TCDF	0.022	1.10	2.00
1,2,3,7,8 PeCDF	0.005	0.90	0.54
2,3,4,7,8 PeCDF	0.036	1.15	3.16
1,2,3,4,7,8-HxCDF	0.061	1.45	4.23
1,2,3,6,7,8-HxCDF	0.038	1.35	2.78
1,2,3,7,8,9-HxCDF	0.008	0.10	7.98
2,3,4,6,7,8-HxCDF	0.092	1.85	4.96
1,2,3,4,6,7,8-HpCDF	0.116	9.25	1.25
1,2,3,4,7,8,9-HpCDF	0.038	0.80	4.74
OCDF	0.066	8.75	0.75
I-TEQ	0.056	2.15	0.01

Table 4. PCDD/Fs carcinogenic and non-carcinogenic risk.

	RISK $C_{simulated}$	RISK $C_{measured}$	% Variation
Non-Cancer risk	4.51E-4	6.49E-2	0.70
Cancer risk	4.06E-11	5.84E-9	0.70

In turn, the carcinogenic and non-carcinogenic risks of PCDD/Fs for the population living in the area under potential influence of the MSWI are given in Table 4.

PCDD/F emissions from the MSWI contributed in a 0.024%, 0.20% and 0.004% of the total air, vegetation and soil I-TEQ, respectively, in the area under evaluation. It means a 0.70% of the total carcinogenic and non-carcinogenic risks due to the total emissions of PCDD/Fs in this area.

According to the above, it can be concluded that in comparison with other emission sources of PCDD/Fs in the same area of the MSWI, the health risks due to the current PCDD/F emissions from the MSWI would be of a small significance for the population living in the neighbourhood of the facility.

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