THE DISTRIBUTION ASSOCIATION BETWEEN PREDICTED AMBIENT PCDD/FS AND SERUM PCDD/FS OF RESIDENTS FROM THE VICINITY OF INCINERATOR

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Introduction

Municipal waste incineration (MWI) has been considered the most common source of environmental polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs) over the past decades. PCDD/Fs often found in fly ash, soil, air and emissions from MWI [1-5]. Most PCDD/Fs are also known to be resistant to environmental and biological degradation, and dispersed into environment throughout atmosphere, water, soil, sediments and food [6].

Human exposure to PCDD/Fs has been detected by analyzing samples collected from air, soil, food and other environmental media. Researches have examined the association between the human body burden and ambient exposure of PCDD/Fs from MWI[7-10]. Nevertheless, most of the reported studies have been rough with the definition of ambient exposure, and that deficiency has suggested the need of better characterization of ambient exposures.

The aim of this study was to evaluate serum PCDD/Fs concentration distribution of residents from the vicinity of incinerators and their association with annual ambient dioxin exposures predicted by application of atmospheric dispersion model.

Materials and methods

A MWI in northern Taiwan was chosen for this study. It had been operated for 6 years at the time when this study took place. The “exposed areas”(zone A, B, and C)and “background area”(zone D) around the MWI were identified by the simulated ambient annual averaged PCDD/Fs concentrations, established by applying Industrial Complex Sources (ISC) model on measured PCDD/Fs data emitted from stack and meteorological data from 1995-1997. Figure 1 shows 4 zones(A, B, C, and D) were separated by equal concentration line of 14×10^{-3}, 4×10^{-3}, and 2×10^{-3} pg I-TEQ/Nm^3 respectively.

Participants in this study have all lived within the radius of 5 km of the incinerator for at least 5 years, and were selected based on the non-occupational exposure potential and population distribution of each district. Ninety-five volunteers (47 male, 48 female) were recruited. At least 60 mL of blood was drawn from the invited subjects, and the serum was separated by centrifugation and stored at -70 ^{o}C until analysis, and all values were adjusted to the percentage of lipid content.

The sample enrichment and cleanup procedures used in this study were similar to the procedures reported by Chang [11]. Serum samples were enriched and fractionated by C18, SCX, silica, and Florisil cartridges before HRGC/HRMS Analysis. Quality assurance/quality control protocols were established, according to those defined in USEPA method 1613, in our laboratory to ensure positive identification and the quality of the measurements.

Levels of PCDD/Fs in serum were reported in pg/g lipid and pg I-TEQ/g lipid. The EXCEL package was used for data management and JMP (version 3.2.6, based on SAS program) for statistical analysis.

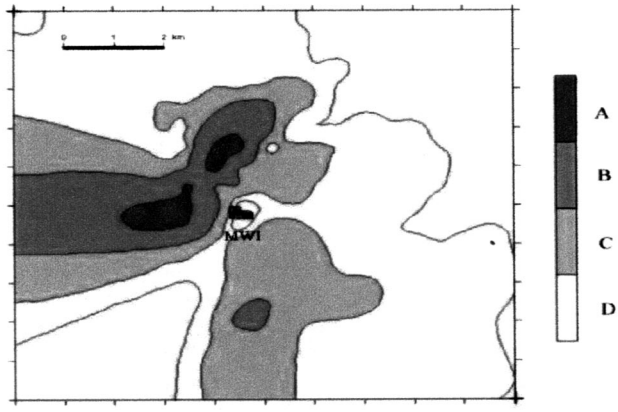

Figure1. Locations of the incinerator, areas A, B, C, and D base on the atmosphere dispersion model.

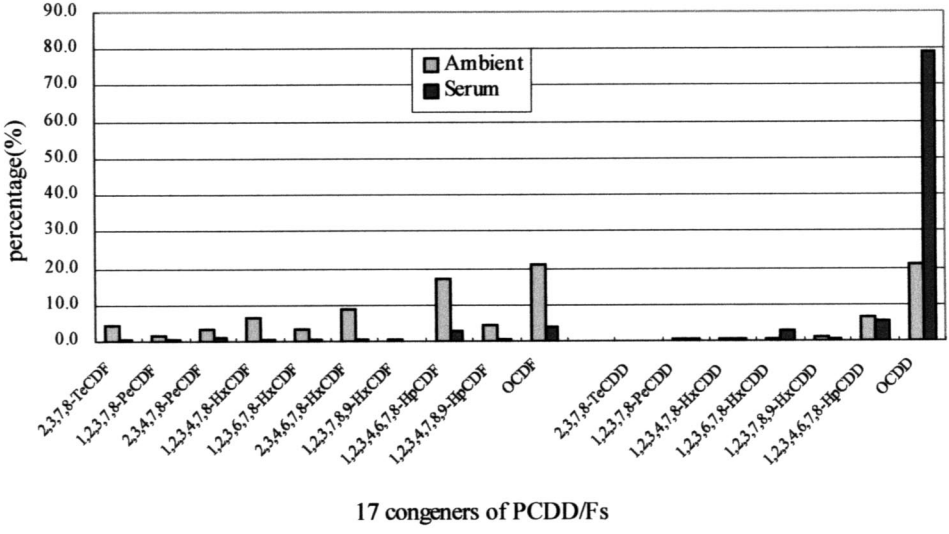

Figure 2. Percentages of 17congeners of PCDD/Fs in predicted ambient levels and serum levels

Results and discussions

Table 1 showed the overall average concentration of serum PCDD/Fs of subjects was 14.1 pg I-TEQ/g lipid, and 12.3, 14.8, 15.6, and 13.6 pg I-TEQ/g lipid for those living in zone A, B, C, and D. The highest level of serum PCDD/Fs was observed in zone C and the lowest in zone A, but there is no significant difference was observed for serum PCDD/Fs level of each group compared with D group by Wilcoxon test. The distribution of serum levels of 4 areas was not consistent with predicted ambient levels.

Table 1. Predicted ambient levels of PCDD/Fs of four districts and serum levels of PCDD/Fs of residents who lived near the incinerator

Areas	Total	A	B	C	D
Number of subjects	89	23	19	28	19
Predicted airborne levels of PCDD/Fs (10^{-3}pg I-TEQ/Nm^3)	3.2±4.2*	20.6±3.9	6.9±2.5	2.8±0.6	1.3±0.4
Serum levels of PCDD/Fs (10^{-3}pg I-TEQ/Nm^3)	14.1±8.9	12.3±8.7	14.8±10.2	15.6±9.0	13.6±7.7
P value#		0.343	0.965	0.448	

*: Mean±standard deviation.
#: Wilcoxon test of serum PCDD/Fs level of each group compare with D group

Table 2. PCDD/Fs levels in the serum and the percentage of each congener compared with total concentration of PCDD/Fs

Congeners	PCDD/Fs levels (pg/g lipid)				Percentage (%)			
	A	B	C	D	A	B	C	D
2,3,7,8-TeCDF	3.8	4.0	3.8	3.8	0.5	0.6	0.5	0.4
1,2,3,7,8-PeCDF	7.2	7.9	16.6	2.2	0.8	0.7	1.3	0.4
2,3,4,7,8-PeCDF	7.4	7.0	6.3	9.3	1.0	1.0	0.7	1.2
1,2,3,4,7,8-HxCDF	6.9	8.3	8.5	6.8	0.9	0.9	0.8	0.9
1,2,3,6,7,8-HxCDF	6.0	6.3	6.9	5.9	0.8	0.8	0.6	0.8
2,3,4,6,7,8-HxCDF	3.0	2.0	4.5	2.4	0.4	0.3	0.4	0.3
1,2,3,7,8,9-HxCDF	17.1	11.7	13.0	10.5	2.7	1.6	1.5	1.5
1,2,3,4,6,7,8-HpCDF	2.1	2.6	3.4	2.0	0.3	0.3	0.3	0.3
1,2,3,4,7,8,9-HpCDF	0.3	0.4	1.2	0.3	0.1	0.1	0.1	0.1
OCDF	37.4	35.0	62.2	19.5	4.7	3.9	4.2	2.9
2,3,7,8-TeCDD	1.1	1.0	1.1	1.3	0.2	0.2	0.1	0.2
1,2,3,7,8-PeCDD	2.3	4.8	3.6	2.5	0.3	0.5	0.3	0.4
1,2,3,4,7,8-HxCDD	3.3	4.4	5.2	3.9	0.4	0.5	0.5	0.8
1,2,3,6,7,8-HxCDD	18.5	24.9	26.0	22.6	2.4	2.7	2.2	3.1
1,2,3,7,8,9-HxCDD	3.3	5.4	6.3	4.2	0.5	0.5	0.5	0.6
1,2,3,4,6,7,8-HpCDD	38.7	54.4	79.9	46.8	4.9	5.1	6.0	6.1
OCDD	626.7	1029.5	1168.0	640.1	79.1	80.4	80.1	80.2
Total PCDF	91.3	85.1	126.3	62.5	12.1	10.1	10.3	8.7
Total PCDD	694.0	1124.4	1289.9	721.3	87.9	89.9	89.7	91.3
Total PCDD/Fs	785.2	1209.5	1416.2	783.8				
TEQ of PCDD/Fs (pg I-TEQ/g lipid)	12.3	14.8	15.6	13.6				

Table 2 showed serum levels of 17 congeners of PCDD/Fs of study subjects. Higher levels of 1,2,3,7,8,9-HxCDF and OCDF were identified compared to the other 8 PCDF congeners. In addition, levels of 1,2,3,6,7,8-HxCDD, 1,2,3,4,6,7,8-HpCDD and OCDD were higher than the other 4 PCDD congeners. Among them, 1,2,3,4,6,7,8-HpCDD represented almost 5 % of the total concentration of all congeners included, while OCDD would make up about 80 % of the total. Results are partly consistent with other studies[7-10]. In general, the sum of all PCDD/Fs congeners averaged from 783.77pg/g lipid to 1416.21pg/g lipid across the 4 zones, and the residents of zone B and C showed higher serum PCDD/Fs levels than the others. Total PCDDs appeared to contribute 87.9-91.3 % of total PCDD/Fs in these study subjects. Figure 2 showed percentage composition of 17congeners of PCDD/Fs in predicted ambient levels and serum levels. The significant difference was observed between the patterns of 17 congeners in predicted ambient levels and serum levels of neighboring residents.

Results showed, similar with other investigation, that people living closest to MWI might not be necessary those who experienced the highest levels of serum PCDD/Fs. We concluded that PCDD might be the more predominant congener in human serum, but PCDF in ambient samples. Residents exposed to PCDD/Fs contamination of MWI emission didn't seem to present higher PCDD/Fs concentration in serum. It might be explained by the fact that ambient exposure was not the most important contributor to serum concentrations when compared to other sources of exposure such as dietary intake.

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