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Health Risk Assessment of PCDD/PCDF Exposure for the Population Living in the Vicinity of a Municipal Waste Incinerator

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Abstract. Emissions of polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs) by municipal solid waste (MSW) incinerators cause concern to the populations living in the vicinity of these facilities. In this study, the health risks of PCDD/F exposure were assessed for adults and children living 500 and 1,000 m from the MSW incinerator. A comparative analysis was performed before (1998) and after (2000) pronounced decreases in PCDD/F air emissions from the stack were noted as a consequence of technical improvements in the facility. At 500 m, total environmental exposure to PCDD/Fs diminished from 5.102×10^{-5} to 1.271×10^{-5} ng I-TEQ/kg/day for adults, and from 8.131×10^{-5} to 2.656×10^{-5} ng I-TEQ/kg/day for children, which means a reduction of 75.1% for adults and 67.3% for children between 1998 and 2000. At 1,000 m, total environmental PCDD/F exposure diminished from 4.087×10^{-5} ng I-TEQ/kg/day in 1998 to 0.995×10^{-5} ng I-TEQ/kg/day in 2000 and from 6.294×10^{-5} ng I-TEQ/kg/day in 1998 to 1.983×10^{-5} ng I-TEQ/kg/day in 2000 for adults and children, respectively. However, these reductions are almost imperceptible compared with the contribution of dietary intake of PCDD/F to total exposure to these contaminants. The present results corroborate that for MSW incinerators with modern technologies, human PCDD/F exposure is mainly due to background contamination.

In recent decades, because of advantages such as volume reduction and energy recovery, incineration of municipal solid waste has been frequently preferred to other treatment or disposal alternatives. However, emissions from municipal solid waste (MSW) incinerators contain, among other pollutants, polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs) (Olie *et al.* 1977; Lisk 1988). These organic compounds, especially the 2,3,7,8-substituted congeners, are among the most hazardous environmental contaminants (Travis and Hattemer-Frey 1991; Birnbaum 1994; McGregor *et al.* 1998; Mukerjee 1998; Kogevinas 2001). In addition, since 1997 2,3,7,8-TCDD is also considered by the IARC as a known human carcinogen (IARC 1997). Consequently, it means that much concern and debate has arisen about human exposure to PCDD/Fs emitted by MSW incinerators.

One of the main reasons for this notable opposition to MSW incinerators has probably been the perception that stack emissions of PCDD/Fs can be a real and serious threat to human health, which means that the public health impact associated with PCDD/F emissions from MSW has become (and remains) a major subject of concern. This atmosphere of intense political debate and strong public opposition to incineration as treatment of choice to process diverse wastes seriously complicates or even impedes the construction of new MSW incinerators. Moreover, the dioxin food crisis in 1999 in Belgium reactivated the debate about these contaminants and their emission sources.

Today's major air emissions of PCDD/Fs into the environment come from combustion processes (UNEP 1999; Quass *et al.* 2000). However, in recent years the relevance of MSW incinerators as the major source of PCDD/F emissions has remarkably diminished. In a recent inventory of PCDD/F air emissions in Tarragona Province (Catalonia, Spain), we found that the only MSW incinerator in the province contributed approximately 0.04% to the total PCDD/F emissions in 1999 (Fuster *et al.* 2001), whereas, for example, diesel motors or the vinyl chloride facility in which this product is manufactured and transformed contributed 18% and 6%, respectively, to the total PCDD/Fs to air flux. Although many people are quite concerned about MSW and PCDD/Fs, specific and direct information concerning the health risks of PCDD/F emissions from MSW incinerators is rather scarce (Hattemer-Frey and Travis 1989; Travis and Hattemer-Frey 1989a, 1989b; Valberg *et al.* 1996; Boudet *et al.* 1999; Nouwen *et al.* 2001).

Since 1975, a MSW incinerator has been operating in Montcada (Barcelona, Spain). In recent years, the concentrations of PCDD/Fs were determined in soil and herbage samples collected near the facility (Schuhmacher *et al.* 1997, 1998; Domingo *et al.* 1999a, 1999b, 2001). In 1999, an acid gas (HCl/SO_2) and metal emission limit equipment was installed in the facility. An active carbon adsorption filter, which could also complement the control of PCDD/F emissions, was added to the fabric filter. As a consequence of these technical improve-

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ments, PCDD/F air emissions via the stack have been reduced from average values higher than 10 ng I-TEQ/nm^3 to the current levels under 0.1 ng I-TEQ/nm^3.

The main goal of the present study was to assess the health risks of PCDD/F exposure for the population living in the vicinity of the MSW incinerator of Montcada. A comparative analysis was also performed of health risks before and after the pronounced decreases in the air emissions of PCDD/Fs from the facility were observed. The results are here presented and discussed.

Materials and Methods

Data about the MSW incinerator, prevailing winds in the area, as well as the location of the sampling points for soils and vegetation were previously reported (Schuhmacher *et al.* 1997, 1998). Duplicate soil and herbage samples were taken at 100, 250, 500, 750, 1,000, 1,500, 2,000, and 3,000 m from the stack in each of the three main directions of the wind in the area (S, NW, NE). Soil samples were collected from the upper 3 cm of soil in amounts consisting of a minimum of 500 g per sample. Herbage samples (*Pipatherum paradoxum L.*) were obtained by cutting at a height of approximately 4 cm from the soil. About 50 g (dry weight) were used for analytical purposes (Domingo *et al.* 1999a, 1999b, 2001). PCDD/F analyses were performed by HRGC/HRMS as previously detailed (Domingo *et al.* 1999a, 1999b). Good conditions of the instruments were regularly maintained by mass calibration and proved by standard runs. Analytical procedures included the use of ^{13}C-labeled internal standards. To achieve the best possible quality with the analytical process, internal standards were calibrated against references at regular intervals to ensure longtime accuracy. In turn, longtime reproducibility of analytical methods was ensured by analysis of internal and external reference materials. Recoveries of added surrogates were determined and evaluated according to the European norm EN-1948 part 1/2/3 for PCDD/F analyses in emissions. For I-TEQ calculations, in the case of values under the detection limit, the congener was assumed to be present at one-half of that limit of detection.

To assess the health risks of PCDD/F exposure for the general population living in the area under potential influence of the MSW incinerator, the following routes should be evaluated: (1) direct contact from inhalation of air and particles, as well as ingestion and dermal contact with soil and dust; and (2) exposure from the consumption of locally grown vegetables, local meats, and local dairy products, which could be contaminated by PCDD/Fs from exposure to polluted soils and herbage.

However, in the area under current evaluation other emission sources of PCDD/Fs were not expected. Consequently, a worst-scenario model was established for the health risk assessment. It was assumed that the total concentrations of PCDD/Fs in the environmental matrices air, soils, and herbage, were exclusively due to air emissions from the incinerator. Moreover, according to the urban location of the facility, there are neither pasture grounds nor crops of vegetables, grains, or fruits within a radius of at least 2 km from the incinerator. It would be highly unlikely that deposition of PCDD/Fs emitted by the MSW incinerator could cause contamination of significant quantities of vegetables, fruits, and cereals, which were consumed at significant amounts by the population living in the area. Therefore, additional exposure from ingestion of local products that might be potentially contaminated by PCDD/Fs was excluded.

Inhalation exposure was calculated by assuming that individuals were exposed to polluted air 24 h/day and that indoor air exposure was equal to outdoor exposure. The daily PCDD/F doses for adults and children were calculated by means of the following equation (Nouwen *et al.* 2001):

$$\mathrm{In}h = V_r \cdot C_{\mathrm{air}} \cdot f_r \cdot t_f/\mathrm{BW}$$

in which In*h* is the inhalation exposure in ng I-TEQ/kg/day; V_r is the ventilation rate (20 m^3/day for adults and 7.6 m^3/day for children); C_{air} is the air concentration in ng I-TEQ/m^3; f_r is the alveolar fraction retained in the lungs (0.75 for adults and children); t_f is the exposed time fraction; and BW is the body weight (70 kg for adults and 15 kg for children).

In turn, dermal exposure was calculated according to the following equation (Nouwen *et al.* 2001):

$$\mathrm{Der}_{\mathrm{total}} = \mathrm{Der}_{\mathrm{soil}} + \mathrm{Der}_{\mathrm{dust}}$$

$$\mathrm{Der}_{\mathrm{soil}} = \mathrm{Der}_{\mathrm{o}} \cdot \mathrm{Der}_r \cdot f_m \cdot 24 \cdot SA_{\mathrm{o}} \cdot t_{\mathrm{fso}} \cdot C_{\mathrm{soil}}/\mathrm{BW}$$

$$\mathrm{Der}_{\mathrm{dust}} = \mathrm{Der}_i \cdot \mathrm{Der}_r \cdot f_m \cdot f_{ri} \cdot 24 \cdot SA_i \cdot t_{\mathrm{fsi}} \cdot C_{\mathrm{soil}}/\mathrm{BW}$$

in which Der is the dermal absorption in ng I-TEQ/kg/day; $\mathrm{Der}_{\mathrm{o}}$ is the skin coverage with soil outside (0.0375 kg/m^2 for adults, and 0.0051 kg/m^2 for children); $\mathrm{Der}_{\mathrm{i}}$ is the skin coverage with dust inside (0.00056 kg/m^2 for both adults and children); $\mathrm{Der}_{\mathrm{r}}$ is 0.01 L/h; f_m is the matrix factor (here 0.15); and f_{ri} is the soil fraction in the inside dust (here 0.8); t_{fso} and t_{fsi} are the exposed time fractions: 0.158 for adults and 0.151 for children, and 0.458 for adults and 0.306 for children, respectively; SA_{o} is the exposed skin surface area for forehands and hands (adults, 0.17 m^2; children, including legs, 0.28 m^2); SA_i is exposed skin surface for hands (adults, 0.9 m^2; children, 0.05 m^2); C_{soil} is the soil concentration in ng I-TEQ/kg dry matter; and BW is the body weight (70 kg for adults and 15 kg for children).

For calculations of soil and dust ingestion the following equation was used (Nouwen *et al.* 2001). A bioavailability of 100% (worst approximation) was estimated.

$$\mathrm{Ing}_{\mathrm{total}} = \mathrm{Ing}_{\mathrm{soil}} + \mathrm{Ing}_{\mathrm{dust}}$$

$$\mathrm{Ing}_{\mathrm{soil}} = I_{\mathrm{sp}} \cdot [24/(24 - t_s)] \cdot t_{\mathrm{fso}} \cdot C_{\mathrm{soil}}/\mathrm{BW}$$

$$\mathrm{Ing}_{\mathrm{dust}} = I_{\mathrm{sp}} \cdot [24/(24 - t_s)] - f_{ri} \cdot t_{fsi} \cdot C_{\mathrm{soil}}/\mathrm{BW}$$

in which Ing is the ingestion of contaminated soil/dust in ng I-TEQ/kg/day; I_{sp} is the ingestion of soil particles (2.6×10^{-5} kg/day for adults and 6.3×10^{-5} kg/day for children); t_s is the sleeping time (8 h/day for adults and 12 h/day for children); t_{fso} and t_{fsi} are the exposed time fractions: 0.158 for adults and 0.151 for children, and 0.458 for adults and 0.306 for children, respectively; f_{ri} is the soil fraction in the inside dust (here 0.8); C_{soil} is the soil concentration in ng I-TEQ/kg dry matter; and BW is the body weight (70 kg for adults and 15 kg for children).

On the other hand, data on PCDD/F intake from food by the population of Montcada corresponding to 1998 were taken from Domingo *et al.* (1999c), whereas those corresponding to 2000 were recently calculated (unpublished data). In both studies, food samples were randomly obtained from local markets, supermarkets, and grocery stores from various towns in Catalonia. For all groups of analyzed samples, the quantity of each food was estimated according to the dietary habits of the population living in the area under assessment (Arija *et al.* 1996; Capdevila *et al.* 2000). Soil and air concentrations were taken from previous reports (Domingo *et al.* 2001), or were recently determined (unpublished data). All calculations were carried out for two possible scenarios, which were established depending on the distance from the incinerator to the place of residence of the potentially exposed population: 500 and 1,000 m.

Table 1. PCDD/PCDF exposure for adults and children living 500 m from a municipal waste incinerator before (1998) and after (2000) pronounced reductions in the emissions of PCDD/PCDFs from the facility

	1998		2000	
	Adults	Children	Adults	Children
Body weight (kg)	70	15	70	15
Environmental exposure				
PCDD/F concentrations in air (ng I-TEQ/m^3)	35×10^{-5}		7.1×10^{-5}	
PCDD/F inhalation	4.62×10^{-5}	6.08×10^{-5}	9.37×10^{-6}	1.23×10^{-5}
Mean PCDD/F concentrations[a] in soil (ng I-TEQ/kg)	5.32		3.69	
Dermal PCDD/F exposure	3.261×10^{-6}	2.841×10^{-6}	2.262×10^{-6}	1.970×10^{-6}
PCDD/F ingestion from soils	1.554×10^{-6}	1.769×10^{-5}	1.078×10^{-6}	1.227×10^{-5}
Total environmental exposure (ng I-TEQ/kg/day)	5.102×10^{-5}	8.131×10^{-5}	1.271×10^{-5}	2.656×10^{-5}
Dietary exposure				
Intake of PCDD/Fs (ng I-TEQ/kg/day)	2.77×10^{-3}	11.4×10^{-3}	0.903×10^{-3}	3.81×10^{-3}
Total exposure to PCDD/Fs (pg I-TEQ/kg/day)	2.82	11.45	0.92	3.84

[a] Values correspond to the mean of PCDD/F concentrations in three samples collected 500 m from the incinerator in 1998 and again in 2000.

Results and Discussion

The results of the estimation of total PCDD/F exposure for adults and children living 500 m from the MSW incinerator of Montcada, 6 months before and 18 months after technical improvements in the facility were carried out are summarized in Table 1. The concentrations of PCDD/F in air were reduced from 35×10^{-5} to 7.1×10^{-5} ng I-TEQ/m^3, and on average PCDD/F levels in soils decreased from 5.32 to 3.69 ng I-TEQ/kg (dry matter). As a consequence of these reductions, the total environmental exposure to PCDD/Fs, under the case conditions established, diminished from 5.102×10^{-5} to 1.271×10^{-5} ng I-TEQ/kg/day for adults and from 8.131×10^{-5} to 2.656×10^{-5} ng I-TEQ/kg/day for children. It means a reduction of 75.1% for adults and 67.3% for children between 1998 and 2000.

Dietary intake of PCDD/Fs was estimated on the basis of PCDD/F concentrations in food items and the average daily intake of these items. It was calculated by the multiplication of the intake of the different food items and the PCDD/F concentration in I-TEQ/g food item on a wet-weight basis (Domingo *et al.* 1999c; unpublished data). Notable reductions in the dietary intake of PCDD/Fs were noted in the period 1998–2000 for both adults and children.

The total PCDD/F exposure for the population living 500 m from the incinerator decreased remarkably during the period 1998–2000. The 2000 values, 0.92 pg I-TEQ/kg/day (adults) and 3.84 pg I-TEQ/kg/day (children), were 67.4% and 66.5% lower than those corresponding to 1998. In addition, although the total exposure for children (3.84 pg I-TEQ/kg/day) was still relatively great, it is within the range established by WHO, 1–4 pg I-TEQ/kg/day as the tolerable daily intake (TDI) of PCDD/Fs for general toxicological effects (other than cancer) (van Leeuwen *et al.* 2000). The noncarcinogenic risk index was calculated by dividing the total exposure by the WHO TDI (1–4 pg I-TEQ/kg/day). For adults, the risk index decreased from the range 0.71–2.82 in 1998 to 0.23–0.92 in 2000, and for children it diminished from the range 2.86–11.45 in 1998 to 0.96–3.84 in 2000.

For carcinogenic effects, the risk is expressed as the probability of contracting cancer over a lifetime of 70 years (Schecter and Olson 1997; Han *et al.* 1998). A value of 1×10^{-3} per pg I-TEQ/kg/day was used as an estimator of the upper bound cancer risk (US EPA 2000). According to the U.S.

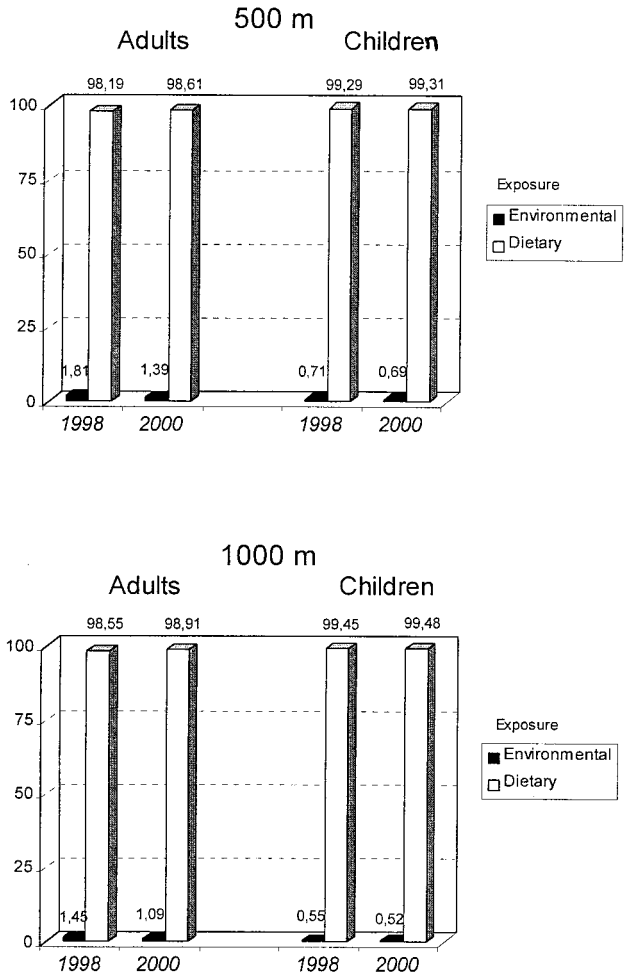

Fig. 1. Percentages of environmental and dietary PCDD/F exposure for adults and children living 500 and 1,000 m from a municipal solid waste incinerator (Montcada, Spain) before (1998) and after (2000) pronounced reductions in PCDD/F emissions from the facility

Table 2. PCDD/PCDF exposure for adults and children living 1,000 m from a municipal waste incinerator before (1998) and after (2000) pronounced reductions in the emissions of PCDD/PCDFs from the facility

	1998		2000	
	Adults	Children	Adults	Children
Body weight (kg)	70	15	70	15
Environmental exposure				
PCDD/F concentrations in air (ng I-TEQ/m^3)	28.6×10^{-5}		5.8×10^{-5}	
PCDD/F inhalation	3.78×10^{-5}	4.97×10^{-5}	7.66×10^{-6}	1.01×10^{-5}
Mean PCDD/F concentrations[a] in soil (ng I-TEQ/kg)	3.44		2.53	
Dermal PCDD/F exposure	2.109×10^{-6}	1.837×10^{-6}	1.551×10^{-6}	1.351×10^{-6}
PCDD/F ingestion from soils	1.005×10^{-6}	1.144×10^{-5}	7.392×10^{-7}	8.412×10^{-6}
Total environmental exposure (ng I-TEQ/kg/day)	4.087×10^{-5}	6.294×10^{-5}	0.995×10^{-5}	1.983×10^{-5}
Dietary exposure				
Intake of PCDD/Fs (ng I-TEQ/kg/day)	2.77×10^{-3}	11.4×10^{-3}	0.903×10^{-3}	3.81×10^{-3}
Total exposure to PCDD/Fs (pg I-TEQ/kg/day)	2.81	11.44	0.91	3.83

[a] Values correspond to the mean of PCDD/F concentrations in three samples collected 1,000 m from the incinerator in 1998 and again in 2000.

EPA (2000), although there are uncertainties associated with the epidemiological evidence that could have influenced the risk estimates rendering these data limited, the overall weight of evidence from the epidemiological studies suggests that the generally increased risk of overall cancer is more likely than not due to exposure to TCDD and its congeners.

For calculations of total PCDD/F intake, inhalation and dermal exposure were assimilated to oral exposure. The risk level for adults over a lifetime of 70 years decreased from 2,820 in 1998 to 920 in 2000. These figures mean 40 and 13 cases of cancers per year due to total PCDD/F exposure (inhalation, dermal, and oral) in an adult population of 1 million. Although it is likely that for children PCDD/F metabolism yields a shorter half-life, data on adverse health effects, in particular cancer development later in life, are not available and cannot be estimated (Becher *et al.* 1998).

In relation to total PCDD/F exposure, the percentage of environmental exposure was reduced from 1.81 to 1.39 for adults, whereas for children it diminished from 0.71 to 0.69 before and after the pronounced decreases in PCDD/F emissions from the facility were recorded (Figure 1, top). These reductions are almost insignificant in relation to background contamination in the area, especially with respect to the dietary PCDD/F exposure.

Similar results were obtained in the estimation of health risks for the population living 1,000 m from the MSW incinerator (Table 2). For adults and children, total environmental PCDD/F exposure diminished from 4.087×10^{-5} ng I-TEQ/kg/day in 1998 to 0.995×10^{-5} ng I-TEQ/kg/day in 2000 and from 6.294×10^{-5} ng I-TEQ/kg/day in 1998 to 1.983×10^{-5} ng I-TEQ/kg/day in 2000, respectively. Taking into account that the dietary PCDD/F exposure was the same for those living 500 and 1,000 m from the incinerator, during the period 1998–2000 total PCDD/F exposure for the population living 1,000 m from the facility was reduced from 2.81 to 0.91 pg I-TEQ/kg/day for adults and from 11.44 to 3.83 pg I-TEQ/kg/day for children. Although these values are analogous to those estimated for the population living 500 m from the incinerator, in adults the percentage of environmental exposure to PCDD/Fs was reduced from 1.45 to 1.09 and for children it diminished from 0.55 to 0.52 (Figure 1, bottom), showing that the contribution of dietary exposure to PCDD/Fs to the total PCDD/F exposure decreases when the distance to the MSW incinerator increases.

Similar findings were previously reported by other investigators. In a perspective on dioxin emissions from MSW incinerators, Travis and Hattemer-Frey (1989a, 1989b) and Hattemer-Frey and Travis (1989) estimated that approximately 99% of human exposure to PCDD/Fs was from background contamination (including exposure from food), even for those individuals living near a modern MSW incinerator. In a comparative analysis of health risk assessments of municipal waste combustors, Levin *et al.* (1991) concluded that the contribution to total risk of all indirect routes of exposure to pollutants emitted by the facilities clearly exceeded that of the direct inhalation route for most studies reviewed. In turn, Boudet *et al.* (1999) showed that the relative contribution of a MSW incinerator with modern technology to population exposure to significant health-related pollutants is small. However, neither PCDD/Fs nor mercury were analyzed in that study.

Our results are also in agreement with those of a recent study by Nouwen *et al.* (2001), who performed a risk assessment of dioxin emissions from two incinerators placed in the vicinity of a residential area in Belgium. Clinical and toxicological measurements, as well as model calculations, were used for the health risk assessment. According to the results, it was concluded that just residing in the impact area of the facility did not result in a meaningful risk.

It is obvious that from the analysis of a particular MSW incinerator, it cannot be concluded that the health risks associated with PCDD/Fs emitted is negligible. However, the current results in accordance with those from previous investigations (Travis and Hattemer-Frey 1989a, 1989b; Levin *et al.* 1999; Nouwen *et al.* 2001) seem to clearly indicate that modern incinerators or ones equipped with modern technologies that emit less than 0.1 ng I-TEQ/m^3 are not mainly responsible for the health risks of PCDD/Fs for the populations living near the facilities because the dietary PCDD/F exposure is quantitatively much more important. Only a significant reduction in the levels of PCDD/F in food will guarantee a significant decrease in human exposure to these pollutants. For that, the continuous efforts to reduce the environmental levels of PCDD/Fs must be continued and/or intensified. These efforts should not be con-

centrated only in MSW incinerator emissions but especially in those sources of PCDD/Fs, which, according to recent inventories, seem to be more relevant.

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