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Health Risk from Exposure to PCDD/Fs from a Waelz Plant in Central Taiwan

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ABSTRACT

This study combined ambient air sampling and biomonitoring data to determine the PCDD/Fs exposure levels and health risk for residents near a Waelz plant. In total, 457 adults completed questionnaires and 48 subjects also gave blood samples. We used the USEPA multimedia model to estimate lifetime exposure and cancer risk. Analytical results show that average atmospheric PCDD/Fs concentrations were 149–285 fg I-TEQ/m^3 and deposition flux was 0.57–116 pg I-TEQ/m^2-d. The serum PCDD/Fs concentrations were from 5.30–50.91 WHO1998-TEQ with an average of 18.73 ± 10.05 pg/g lipid. The estimated lifetime average daily doses (LADDs) were 1.20 and 1.09 pg WHO1998-TEQ/kg/d, which are below the tolerable daily intake (TDI) range of 1–4 pg TEQ/kg bw (World Health Organization). However, the concentration was higher than concentrations for the general population in Taiwan. The USEPA multimedia model showed that the median of lifetime exposure to PCDD/Fs was 1.90×10^{-2} pg/kg bw/day and 3.19×10^{-2} pg/kg bw/day for Area U (upwind site) and Area D (downwind site), respectively. The carcinogenic risk was 2.96×10^{-6} (95th percentile = 1.61×10^{-5}), and 4.97×10^{-6} (95th percentile = 2.59×10^{-5}) for Area U and Area D, respectively. In summary, residents near the plant had a higher body burden of PCDD/Fs than the general population in Taiwan. The health risk from exposure, including cancer mortality caused by chronic exposure to PCDD/Fs, needs to be clarified further.

Keywords: PCDD/Fs; Health risk assessment; Waelz plant; Biomonitoring; Estimated lifetime average daily doses (LADD).

INTRODUCTION

Polychlorinated dibenzo-para-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are environmental pollutants comprising a group of dangerous chemicals that are potentially highly toxic. They can cause enzyme induction, reproductive and developmental toxicity, immunotoxicity, adverse endocrine effects, chloracne, and tumor promotion (Birnbaum, 1994; Alaluusua and Lukinmaa, 2006; King-Heiden *et al.*, 2012). The anthropogenic sources of dioxins include byproducts of industrial processes, as well as municipal and industrial waste incinerators (Abad *et al.*, 1997; Colombo *et al.*, 2009; Suzuki and Kawamoto, 2012). More than 90% of human exposure is reported via food (Charnley and Doull, 2005). Consequently, protecting food supplies from contamination is critical. The most effective approach includes source-directed measures to reduce dioxin emissions.

It was estimated that over 150,000 tons of electric arc furnace (EAF) dusts contain relatively high concentrations of heavy metals and PCDD/Fs, and are sources of dioxins in Taiwan. A Waelz plant was established in central Taiwan in 2000 to treat and decontaminate these hazardous wastes. The PCDD/Fs concentration measured in stack gas at the Waelz plant exceeded 190 ng-I-TEQ/N m^3 (Chi *et al.*, 2006). To meet the stringent emission limit in Taiwan (stage 1 was 9.0 ng-I-TEQ/Nm^3 in 2005 and stage 2 was 1.0 ng-ITEQ/Nm^3 in 2006), installing additional air pollution control devices (APCDs) was necessary. Therefore, the Waelz plant used activated carbon (AC) injection followed by a double baghouse filter (BF) to control dioxin emissions. Chi *et al.* (2008) reported that the total PCDD/Fs and polychlorinated

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biphenyl (PCB) emission flows at the plant (per kg of electric arc furnace dust treated) reduced to 0.235 ± 0.04 ng-I-TEQ/(N m^3) after the modified APCDs were installed. Additionally, atmospheric PCDD/Fs concentrations measured in the vicinity area of the Waelz plant were also greatly reduced from 568–1465 to 48.9–130 fg-I-TEQ/m^3.

This study combined air dispersion models, ambient air sampling, and biomonitoring data to determine the PCDD/Fs exposure level of residents near this Waelz plant and their health risk. This study had four major objectives. 1. Measure the serum PCDD/F levels in residents near the plant. 2. Estimate body burden and average daily exposure dosage using serum PCDD/F levels. 3. Estimate the lifetime daily PCDD/Fs exposure dosage and carcinogenic risk using monitoring data and the multimedia model, and compare the consistency with body burden data. 4. Explore the cancer-related mortality of residents using registered data.

MATERIALS AND METHODS

Study Area and Air Sampling

The coastal Waelz plant is located in central Taiwan. A steady-state Gaussian plume dispersion model (Industrial Source Complex, ISCST3) was employed to define the plant's upwind (Area U) and downwind (Area D) areas (Fig. 1). Emission rates and distribution of PCDD/Fs in the surrounding ambient air were monitored.

To measure atmospheric PCDD/Fs deposition and determine the ambient air concentration of PCDD/Fs near the Waelz plant, four sampling sites (A, B, C, and D) were established based on meteorological records and location to the plant. Fig. 1 shows the relative locations of the four sampling sites. Site A was on the roof of an office building at the Waelz plant (about 300 m from the stack); Site B was close to a duck farm located about 1.8 km directly downwind from the plant; Site C was near a junior high school about 3.5 km from the plant (slightly downwind); and site D was close to a primary school about 4.2 km upwind of the plant. Due to topographical features, a north wind predominates in this area all year except for during summer. The atmospheric PCDD/F samples in this study were taken on May (2009/5/25–6/24) and June (2009/6/25–7/24) during the summer and October (2009/10/22–11/24) during the winter season.

Ambient air (n = 6) and atmospheric deposition (n = 12) PCDD/Fs samples were obtained. Ambient air samples for

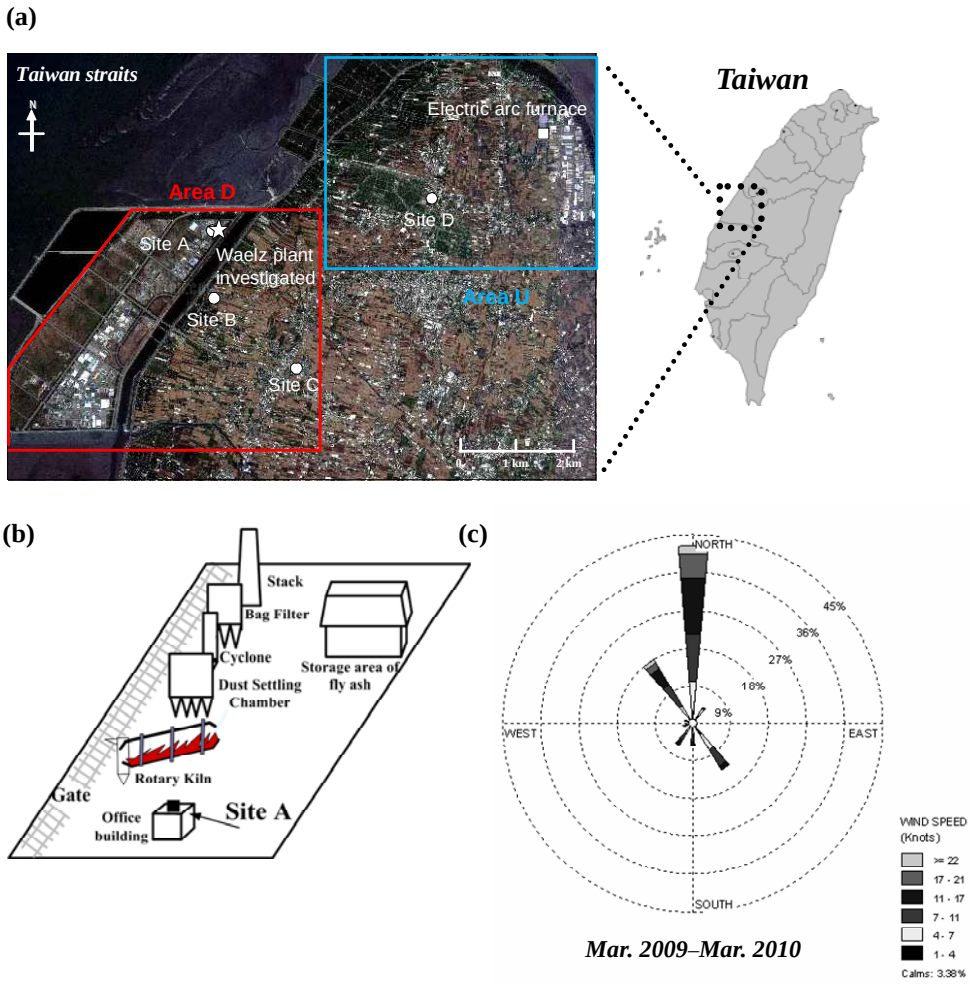


Fig. 1. (a) Location of four sampling sites in this study (satellite image provided by http://www.urmap.com/), (b) Location of sampling site A in the Waelz plant investigated and (c) wind rose plot during sampling periods.

both vapor and solid phases of PCDD/Fs were collected using sampling trains (Shibata HV-700F) for semi-volatile compounds. Fiberglass filters were utilized to collect all particle-bound compounds, while polyurethane foam (PUF) plugs were utilized to retain PCDD/Fs compounds in the vapor phase. Total volume of sampled air exceeded 1,000 m^3 for a typical sampling duration of 7 days (gas flow rate: 100 L/min). At the same location, atmospheric deposition of PCDD/Fs was measured monthly (30 days) using mirror-polished stainless-steel cylindrical vessels (D: 500 mm, H: 600 mm). Prior to sampling, approximately 10 L of deionized water were added to the vessel to cover the surface of the bottom.

Study Subjects and Demographic Data

One township was chosen in Area U and Area D. Population density was 1604 and 941 per km^2, respectively, which are low compared to the average of 2844 per km^2 in urban areas. Stratified sampling was conducted to recruit subjects. Each subject gave informed consent and completed a questionnaire to provide demographic information, subjectively assessed air pollution levels, time-activity patterns, and dietary records. Approximately 30 ml blood was collected in a vacutainer tube without any anticoagulant from 48 volunteers who also donated blood for serum dioxin measurement. Whole blood was allowed to clot for 1 hour and then centrifuged for 15 min at 2500 rpm. The serum was transferred to rinsed glass vials and was stored at –80°C until analysis.

This study was approved by the Institutional Review Board at National Yang-Ming University.

Determination of Dioxin Concentrations

For PCDD/Fs analysis, the ambient (air and deposition) samples were then spiked with known amounts of internal quantification standards according to USEPA method 23. Thereafter, the PUF and filter samples were Soxhlet extracted with toluene for twenty-four hours. The toluene extract was then concentrated to about 1 mL by rotary evaporation and was replaced by 5 mL hexane for pretreatment process. Having been treated with conc. sulfuric acid, the sample was then subjected to a series of clean-up columns including sulfuric acid silica gel column, acidic aluminum oxide column and Celite/Carbon column. For the serum sample, each serum sample was spiked with a mixture containing PCDD/F standards as defined in the analysis methods 1613B of USEPA. In addition, the sample is mixed with sodium sulfate, allowed to dry for 12–24 hours, and extracted for 24 hours using methylene chloride: hexane (1:1) in a Soxhlet extractor. The extract is evaporated to dryness, and the lipid content is determined. The lipid content is determined by extraction of serum with the same solvent system (methylene chloride: hexane) that was used in Methods 1613B. Serum samples were enriched and fractionated by silica, and highly selective adsorbent magnesium-silica gel cartridges prior to analysis. Finally, the recovery standard solutions were spiked with known amounts Method 23 (ambient samples) and Method 1613B (serum sample) and then analyzed for 17 PCDD/Fs with high-resolution gas chromatography (HRGC)/high-resolution mass spectrometry (HRMS) (Thermo DFS) equipped with a fused silica capillary column DB-5 MS (60 m × 0.25 mm × 0.25 μm, J&W).

Quality Control and Data Analysis

A laboratory blank and matrix spike sample (40–400 pg PCDD/Fs) were used in the analytical procedure for every eight samples for quality control. Method detection limits (0.04–1.3 pg/g) were determined from the blanks and quantified as three times the standard deviation of the mean concentration in the blanks. In this study, the concentrations of all laboratory blank samples were < 1.0 pg (PCDD/Fs). Mean recoveries of standards for all $^{13}C_{12}$-2,3,7,8-chlorosubstituted PCDD/Fs were 52–109%. All analytical results were within the acceptable range of 40–130% in the USEPA Method 23 and Method 1613. For data analysis, International Toxic Equivalent Factors (I-TEFs) were adopted to compare the potential toxicity of each atmospheric PCDD/Fs congener in a mixture to the well-studied and understood toxicity of TCDD, the most toxic member of the PCDD/Fs group (USEPA, 1989). The I-TEF of each congener in a mixture was multiplied by the respective mass concentration, and products were summed to yield the 2,3,7,8-TCDD International Toxic Equivalence (I-TEQ) of the mixture. To determine concentrations of PCDD/Fs in serum samples, WHO_{1998}-TEQ and WHO_{2005}-TEQ were used to describe a mixture for which PCDD and PCDF congeners were determined, and for which TEQ was calculated based on the WHO 1998 and WHO 2005 scheme, respectively (Van den Berg *et al.*, 1998, 2006). All sera dioxin concentrations were lipid adjusted (pg/g lipid).

Calculation of Lifetime Exposure and Cancer Risk

This study used the monitored airborne PCDD/Fs concentrations to estimate lifetime exposure and cancer risk. A USEPA multimedia risk assessment procedure was applied to determine the fate and transport of PCDD/Fs emissions from the plant and the PCDD/F concentrations in various foodstuffs. Exposure scenarios were generated based on the above-mentioned surveillance and annual report of national agricultural products consumption. Lifetime daily intakes of PCDD/Fs (in mg/kg-day) through multiple exposure pathways were estimated by summing the exposure dosage from each pathway. Lifelong cancer risk of PCDD/Fs exposure was computed as follows:

$$\text{Risk} = \text{Intake dosage in pg-TEQ/kg/day} \times \text{slope factor in } (\text{pg-TEQ/kg/day})^{-1} \quad (1)$$

The carcinogenic slope factor in this study was 1.56×10^{-4} $(\text{pg-TEQ/kg/d})^{-1}$ (USEPA, 1999).

A Monte Carlo simulation was used to describe probability distributions of cancer risks (Oracle Crystal Ball, Fusion Edition 11.1.2.1.0). Normal probability distribution was selected for each parameter in running the Monte Carlo simulation.

Cancer Mortality of the Population in Study Area

This study compared cancer mortality at these two

townships with that of other townships nearby using the Atlas of cancer mortality in Taiwan. Mortality rates used were for the period 1971–2010 and were all age-adjusted.

RESULTS AND DISCUSSION

Atmospheric PCDD/Fs Concentrations and Deposition Fluxes near the Waelz Plant

Measurement results indicate that average atmospheric PCDD/Fs concentration and deposition flux at the four sampling sites were as follows: 285 fg I-TEQ/m^3 and 116 pg I-TEQ/m^2-d at Site A; 1.95 pg I-TEQ/m^2-d at Site B (only deposition flux was measured because of no power supply at this site); 149 fg I-TEQ/m^3 and 0.57 pg I-TEQ/m^2-d at Site C; and 229 fg I-TEQ/m^3 and 0.97 pg I-TEQ/m^2-d at Site D. The analytical results in our previous study (Chi *et al.*, 2008) indicated that atmospheric PCDD/Fs concentrations in an urban area in Taiwan were 20–110 fg I-TEQ/m^3. In some Asian countries, such as Korea and Japan, atmospheric PCDD/F concentrations in urban areas were 28–120 fg I-TEQ/m^3 (Lee *et al.*, 2007; Makiya, 1999). The atmospheric PCDD/Fs concentrations measured near the Waelz plant (especially at Site A) were considerably higher than those measured in other countries. Compared to that in other studies in Taiwan, atmospheric PCDD/Fs depositions (116 pg I-TEQ/m^2-d) at Site A were significantly higher than that measured in an industrial area (18–26 pg I-TEQ/m^2-d) in northern Taiwan (Chi *et al.*, 2009) and urban area (3.1–19 pg I-TEQ/m^2-d) in southern Taiwan (Shih *et al.*, 2006). We speculate that the high deposition flux of PCDD/Fs at site A is due to the strong re-suspension of fly ash particles from the storage area at the Waelz plant (Fig. 1).

Subjective Response on Living Quality and Health Status

In total, 457 adults completed questionnaires and only those who lived in the study area for more than 5 years were presented. Table 1 lists the demographic data of subjects. Mean ages were 38.5 ± 7.8 and 47.7 ± 13.9 years for subjects residing in Area U and Area D, respectively. Subjects in Area D were significantly older, less educated, had higher BMI (body mass index), and less exposure to second-hand smoke than subjects in Area U. Area U also had more females than Area D. About 70% and 50% of participants complained of air pollution and malodor. Area U complained significantly more and rated air pollution higher than those in Area D. The incidence rate of self-reported disease was higher in Area U, especially for respiratory diseases and dermal diseases. Area U and Area D are both located downwind of an industrial area and Area U is closer to it than Area D. The impact of the industrial area on residents was greater than that of the Waelz plant.

Cancer Mortality

During the period 2001–2010 when the plant started in operation, the total age-adjusted cancer mortality for males was 173.56 and 188.75/10^5 and that for females was 83.65 and 86.70/10^5 in Area D and Area U, respectively. The cancer mortality rate for males was higher than that nationwide (156.38/10^5). The leading three morality-causing cancers in the study areas were liver and intra-hepatic bile duct cancer, tracheal, bronchial, and lung cancer, and stomach cancer. The cancer mortality rates of liver and lung for males were consistently higher in Area D than in Area U or nationwide. Fig. 2 compares temporal trend of cancer mortality in Area U, Area D and nationwide in the past forty years. Mortality rates of two other townships next to Area U (Area Un) and Area D (Area Dn) but farther to the Waelz plant were also shown in the figure. We found a sharp increase of cancer mortality rates of all sites combined-, lung-, and liver- for female and of lung cancer for male in Area D. The increase was particularly during the period of 2001–2010 when the plant started in operation. Exposure to PCDD/Fs is reportedly associated with the above-mentioned cancers. However, because of the multiple risk factors (such as smoking and polycyclic aromatic hydrocarbons) for carcinogenesis, the contribution of exposure to PCDD/Fs to cancer mortality in these areas needs to be clarified (Motorykin *et al.*, 2013; Svecova *et al.*, 2013).

Serum PCDD/Fs Concentrations

Table 2 lists the concentrations of 17 congeners of PCDD/Fs for the 48 subjects living in the study areas. Subjects living downwind of the plant did not have significantly higher dioxin levels (mean WHO_{2005}-TEQ, 16.72 pg/g lipid and WHO_{1998}-TEQ, 19.37 pg/g lipid), as compared to subjects living upwind (mean WHO_{1998}-TEQ, 15.42 pg/g lipid and WHO_{1998}-TEQ, 17.96 pg/g lipid). In order to adjust the smoking effect, the subjects were stratified by the smoking status. Similar serum PCDD/Fs concentrations were found for subjects in both areas.

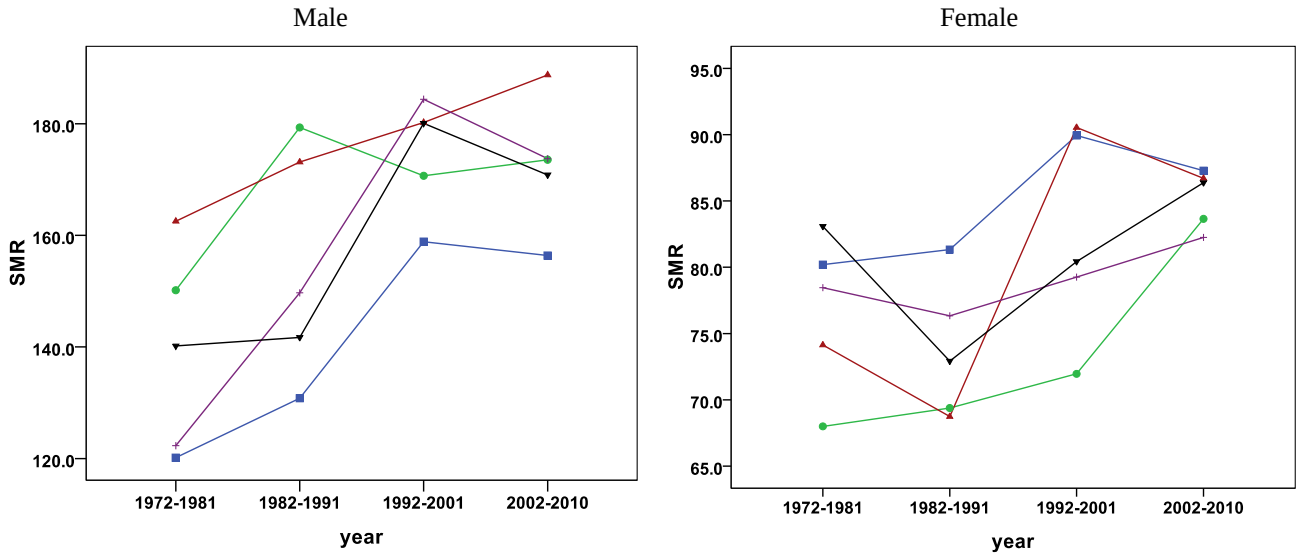
Comparisons with available data in Taiwan indicate that average serum dioxin levels were higher in study subjects than that in non-occupationally exposed populations. Hsu *et al.* (2010) reported that the mean serum TEQ level of 17 congeners of PCDD/Fs in 251 Taiwanese was 10.9 ± 3.5 pg WHO_{2005}-TEQ/g lipid. Domestic reports in Taiwan indicated the average serum dioxin levels for residents near 18 municipal incinerators were 14.7–29.2 pg WHO_{1998}-TEQ/g lipid (Taiwan EPA, 2001, 2002a, b, 2003). A study of aluminum smelting workers in Taiwan revealed that average serum PCDD/F levels were 19.4 pg WHO_{1998}-TEQ/g lipid (Liu *et al.*, 2005). Lin *et al.* (2012) reported that the median serum dioxin-like chemical concentrations (including those of 17 PCDD/Fs and 9 PCBs) for 2361 American subjects aged 40 or older was 19.2 WHO_{2005}-TEQpg/g lipid. According to many studies of concentrations of dioxin-like in foodstuffs, the contribution of PCDD/Fs and dioxin-like PCBs to WHO-TEQ varied (Marin *et al.*, 2011; Storell *et al.*, 2011; Wei *et al.*, 2011; Chang *et al.*, 2012; Lin *et al.*, 2012; Zhang *et al.*, 2013). Therefore, one cannot compare this study's analytical results with those for American populations because of the different components in TEQ. Given that the study areas were coastal and rural, few pollution sources should exist and air dispersion should be good. The higher PCDD/Fs concentrations in sera from study subjects than in the general population in Taiwan is indicative of the adverse impact of the Waelz plant and the nearby industrial site.

Table 1. Socio-demographic characteristics of the study population.

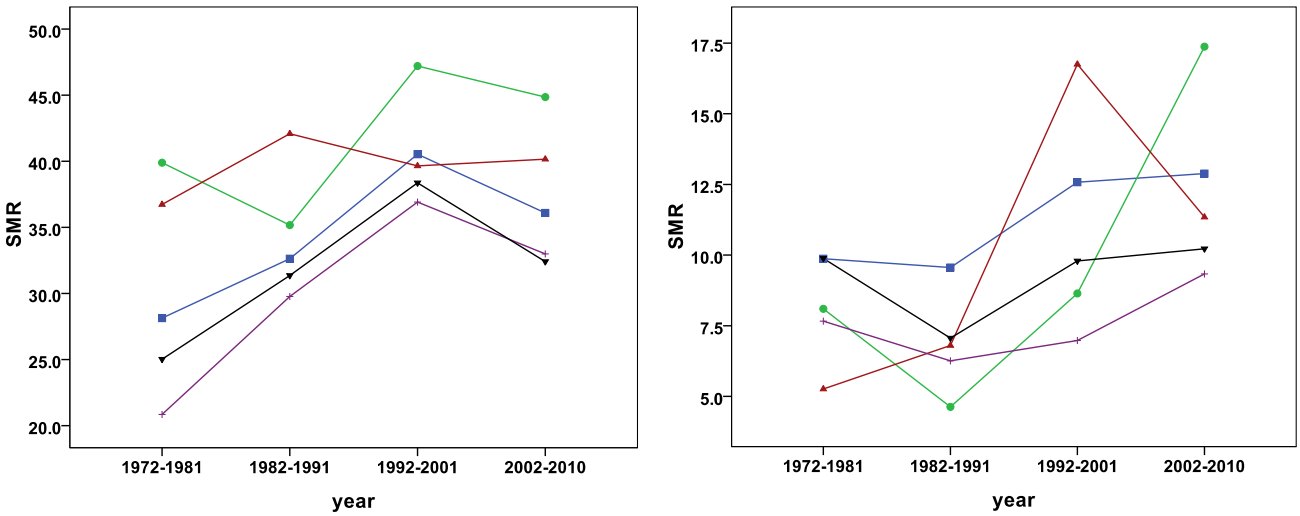
Variable	Area U (n = 200)	Area D (n = 257)	Total (n = 457)	p value
Gender				0.19
female	122(61.0)	145(56.4)	267(58.4)	
male	78(39.0)	112(43.6)	190(41.6)	
Age[a]	38.5 ± 7.8	47.7 ± 13.9	43.7 ± 12.5	< 0.01
BMI (kg/m^2) [a]	22.9 ± 3.7	24.9 ± 14.5	24.1 ± 11.1	0.05
Education				< 0.01
< college	84(42.2)	152(60.6)	236(52.4)	
> college	115(57.8)	99(39.4)	214(47.6)	
Live year	18.4 ± 11.7	29.0 ± 16.3	24.4 ± 15.4	< 0.01
Current smoke state				0.51
no smoking	177(88.5)	224(88.9)	401(88.7)	
smoking	23(11.5)	28(11.1)	51(11.3)	
Second-hand Smoke				< 0.01
no	117(59.4)	177(71.7)	294(66.2)	
yes	80(40.6)	70(28.3)	150(33.8)	
Air pollution				< 0.01
no	42(21.3)	91(36.1)	133(29.6)	
mild	104(52.8)	119(47.2)	223(49.7)	
moderate	38(19.3)	28(11.1)	66(14.7)	
heavy	13(6.6)	14(5.6)	27(6.0)	
Malodor				0.10
No	95(49.0)	126(50.8)	221(50.0)	
mild	76(39.2)	92(37.1)	168(38.0)	
moderate	19(9.8)	15(6.0)	34(7.7)	
heavy	4(2.1)	15(6.0)	19(4.3)	
Medical History				
Heart disease				0.33
no	168(87.5)	213(85.5)	381(86.4)	
yes	24(12.5)	36(14.5)	60(13.6)	
Respiratory disease				0.10
no	146(76.0)	201(81.7)	347(79.2)	
yes	46(24.0)	45(18.3)	91(20.8)	
Renal disease				0.14
no	184(95.3)	229(92.3)	413(93.7)	
yes	9(4.7)	19(7.7)	28(6.3)	
Liver disease[§]				0.33
no	171(88.1)	222(89.9)	393(89.1)	
yes	23(11.9)	25(10.1)	48(10.9)	
Dermal disease				0.02
no	170(87.6)	232(93.5)	402(91.0)	
yes	24(12.4)	16(6.5)	40(9.0)	
Cancer				0.39
no	192(98.5)	244(99.2)	436(98.9)	
yes	3(1.5)	2(0.8)	5(1.1)	
Other disease				0.43
no	175(96.7)	205(95.8)	380(96.2)	
yes	6(3.3)	9(4.2)	15(3.8)	

[a] mean ± SD, t test.

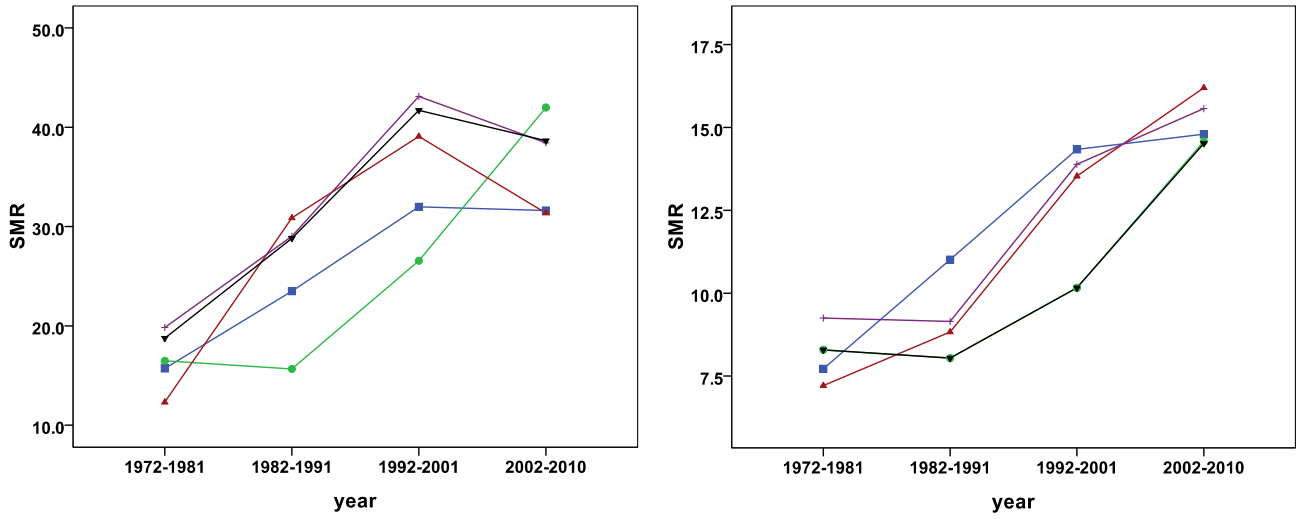
Based on serum concentrations of PCDD/Fs, estimated body burden was 4.26 (Area D) and 3.95 (Area U) ng WHO_{1998}-TEQ/kg of body weight (bw) for subjects, assuming dioxins are equally distributed in body fat and an adult has 22% body fat (DeVito *et al.*, 1995). Their estimated lifetime average daily doses (LADDs) were 1.20 and 1.09 pg WHO_{1998}-TEQ /kg/d, respectively. The WHO has established a tolerable daily intake (TDI) range of 1–4 pg TEQ/kg bw for dioxins (van Leeuwen, 2000) and a tolerable weekly intake (TWI) of 14 pg WHO-TEQ/kg bw has been determined by



(a) All site combined (ICD-9: 140-208)

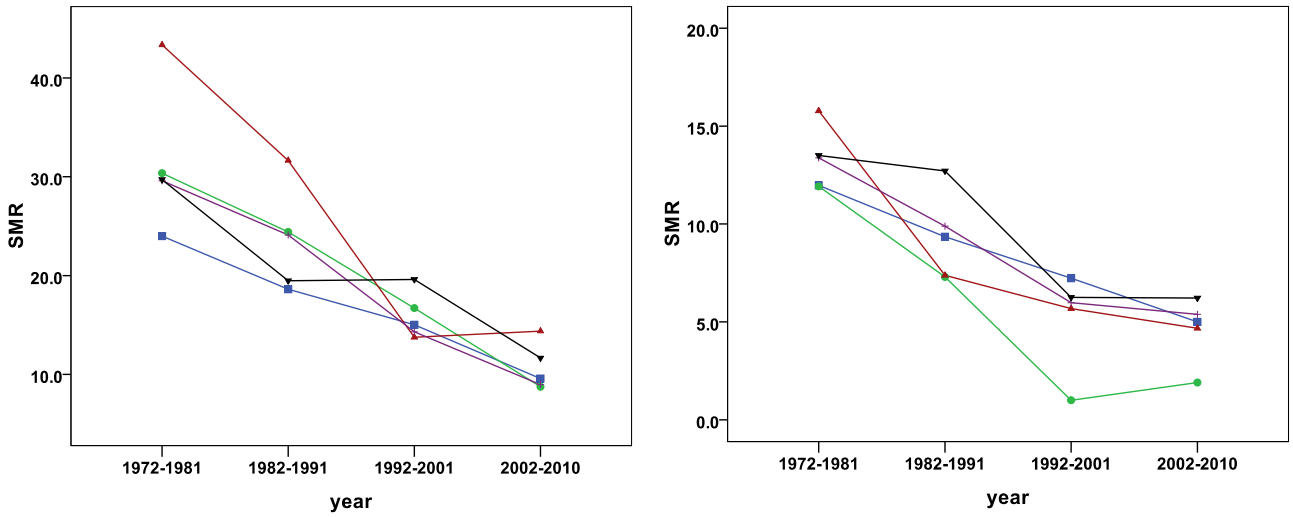


(b) Liver & intrahepatic bile duct (ICD-9: 155)



(c) Tracheal, bronchial & lung (ICD-9: 162)

Fig. 2. Gender- and age- standardized cancer mortality (per 100,000 population mortality) from 1972–2010 (■Nationwide, ●Area D,▲Area U,+Area Un,▼Area Dn).



(d) Stomach (ICD-9: 151)

Fig. 2. *(continued).*

Table 2. Serum PCDD/F concentrations of the subjects.

Variable	Area U Mean ± SD (n = 22)	Area D Mean ± SD (n = 26)	Total Mean ± SD	p value
Blood				
PCDD/F (pg-$TEQ_{WHO2005}$/g)	15.42 ± 7.42	16.72 ± 9.36	16.12 ± 8.46	0.082
PCDD/F (pg $TEQ_{WHO1998}$/g)	17.96 ± 9.08	19.37 ± 10.94	18.73 ± 10.05	0.090
Body burden				
(pg-$TEQ_{WHO1998}$/kgBW)	3.95	4.26	4.12	
Daily intake				
(pg $TEQ_{WHO1998}$/kg/day)	1.09	1.20	1.15	
Stratified by smoking status				
Smoker combined with Second-hand Smoker	n = 0	n = 2 29.27 ± 0.16		
Either Smoker or Second-hand Smoker	n = 4 14.82 ± 5.60	n = 11 15.38 ± 8.8		
Neither smoker nor Second-hand Smoker	n = 18 15.55 ± 7.89	n = 13 15.93 ± 9.43		

the European Union (EU) through the European Commission Scientific Committee on Food (SCF) (Commission, 2001). Assuming the dl-PCBs were negligible, the estimated PCDD/Fs intake level of study subjects was within the tolerable limit. Notably, the USEPA (2012) has reanalyzed its reference dose and has recommended 0.7 pg $TEQ_{WHO1998}$/kg/d based on the increase in neonatal TSH and a decreased sperm count and motility. This study found that the estimated intake level of PCDD/Fs for study subjects exceeded this recommendation. The body burden of 13 ng TEQ/kg bw (calculation included PCDD/Fs and dl-PCB) for the general population of America.

Lifetime Dioxin Exposure and Cancer Risk Assessment

Based on the monitoring data, concentrations of PCDD/Fs in various foodstuffs were calculated using the USEPA multimedia model procedure. Table 3 summarizes the exposure parameters used in health risk assessment. Combined with exposure data and time-activity patterns, the daily intake of PCDD/Fs was obtained (Table 4). Analytical results show that the median lifetime exposure to PCDD/Fs was 1.90×10^{-2} pg/kg bw/day and 3.19×10^{-2} pg/kg bw/day for the Area U and Area D, respectively. Monte Carlo simulation was used to describe probability distributions of exposures and risks. Results show a 2.96×10^{-6} (95th percentile = 1.61×10^{-5}), and 4.97×10^{-6} carcinogenic risk (95th percentile = 2.59×10^{-5}) for Area U and Area D, respectively (Table 4). The calculated daily intake of PCDD/Fs was two orders of magnitude lower than estimates from serum concentrations. This discrepancy might be the result of several factors: the Waelz plant is only one of many sources of PCDD/Fs; serum PCDD/Fs concentrations result from long-term exposure and the Waelz plant had much higher PCDD/Fs emissions before installation of additional APCDs; monitoring data were not representative of emissions because of the limited number of temporal

Table 3. Summary of exposure factors used in health risk assessment.

Variable name			Value	Source
Human body	Exposure time	(yr)	12	The industrial areas have reached 12 years.
	The averaged body weight	(kg)	64.56	Note 1
	Life time	(yr)	70	Note 1
Intake	(1) Exposure pathway & consumption			
	Respiration	(m^3/d)	9.29	Note 1
	Water intake	(L/kg/day)	0.0235	
	(2) Crops consumption			
	Rice	(g/d)	170.99	Note 1
	Root vegetables	(g/d)	29.56	
	Non-roots vegetables	(g/d)	195.63	
	Fruit	(g/d)	137.66	
	(3) Livestock products			
	Beef	(g/d)	5.57	Note 1
	Milk	(g/d)	57.12	
	Pork	(g/d)	85.66	
	Chicken	(g/d)	26.54	
	Egg	(g/d)	25.95	
	Fish	(g/d)	51.12	
Frequency of contact	(1) Exposure pathway			
	Respiration	(d/year)	365	Assumption
	Water intake	(d/year)	365	Assumption
Food local-supply rate (%)	Rice		91.90	Note 2
	Root vegetables		25.90	
	Non-roots vegetables		88.90	
	Fruit		88.20	
	Beef		5.50	
	Milk		32.30	
	Pork		92.00	
	Chicken		83.90	
	Egg		100	
	Fish		100	
Dioxin residual rate (%)	Root vegetables		47.1	Note 3
	Non-roots vegetables		84.6	
	Beef		54.9	
	Pork		64.0	
	Chicken		70.0	
	Fish		81.1	
Absorption rate (%)	: Inhalation		100	
	Ingestion		50	

Note 1: from: “Compilation of Exposure Factors”, Center for Health Risk Assessment and Policy, College of Public Health, National Taiwan University, Taiwan, 2008.
Note 2: from: “Food supply and demand report 2010”, Council of Agriculture, Taiwan, 2010.
Note 3: from: “Exposure and Human Health Reassessment of 2,3,7,8-Tetrachloro Dibenzo-*p*-Dioxin (TCDD) and Related Compounds” (Draft), U.S. EPA, 2000.

and spatial samples; the USEPA multimedia model may be unsuited to Taiwan; and some foodstuffs were not produced and consumed locally. Compared with dietary exposure data in Taiwan (Wang *et al.*, 2009; Hsu *et al.*, 2010; Chang *et al.*, 2012; Lin *et al.*, 2012), lifetime dioxin exposure based on the combination of monitoring data and the USEPA multimedia model seemed under-estimated. Wang *et al.* (2009) estimated the daily intake of PCDD/Fs in Taiwan at 34.4 and 25.1pg WHO-TEQ for males and females, respectively, and the main contributor to dietary intake for adults were fish and shellfish. Assuming that a person’s body weight is 60 kg, daily intake would be 0.57 pg WHO-TEQ/kg bw/day and 0.41 pg WHO-TEQ/kg bw/day for males and females, respectively. Chang *et al.* (2012) measured the dietary intake of PCDD/Fs and dl-PCBs from fresh foods and estimated it at 0.33 pg WHO-TEQ/kg bw/day for males and 0.31 pg WHO-TEQ/kg bw/day for females. Marin *et al.* (2011) estimated the dietary intake of

Table 4. The life-time average daily dose (LADD) of dioxin (Unit: pg/kg/d).

Pathway	Region					
	Area U		Area D		Average	
	Exposure dose	%	Exposure dose	%	Exposure dose	%
Inhalation	3.52×10^{-3}	18.53	6.00×10^{-3}	18.82	4.76×10^{-3}	18.68
Ingestion	1.55×10^{-2}	81.47	2.59×10^{-2}	81.18	2.07×10^{-2}	81.32
Carcinogenic risk	2.96×10^{-6}		4.97×10^{-6}		3.97×10^{-6}	
Sensitivity analysis						
50%	2.66×10^{-6}		4.42×10^{-6}			
75%	4.83×10^{-6}		8.07×10^{-6}			
95%	1.61×10^{-5}		2.59×10^{-5}			

dioxins and dl-PCBs in Spain at 2.86 pg WHO-TEQ/kg bw/day for adults, higher than those estimated in Taiwan.

In summary, the installation of APCDs in the Waelz plant reduced emissions of PCDD/Fs. However, residents near the plant still had a higher body burden of PCDD/Fs than the general population in Taiwan. Serum concentrations are more reliable than environmental monitoring data in representing overall exposure level to PCDD/Fs. The contribution to the health risk from chronic exposure to PCDD/Fs, including the associated cancer mortality rate, needs further clarification.

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