Evaluation of the Relative Health Risk Impact of Atmospheric PCDD/Fs in PM$_{2.5}$ in Taiwan

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ABSTRACT

Many studies have indicated that the largest amounts of particle-bound polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs) are found on small particles, which result in them having the highest PCDD/F TEQ concentrations. Epidemiological studies have found associations between PCDD/F exposure and development of many chronic diseases such as cardiovascular disease or cancers even in low exposure populations. Recently, in many countries, the concentrations of PM$_{2.5}$ and PCDD/Fs have been one of the main foci of air monitoring systems. Several researches have suggested an association between air pollutants and health outcomes. However, publications about how PM$_{2.5}$ and PCDD/Fs influence the health outcome in Taiwan are still limited. In this research, the spatial concentrations of PCDD/Fs in the vapor phase and the solid phase (TSP and PM$_{2.5}$) were investigated during the winter seasons from 2012 to 2014 at different sites in Taiwan. The mean PCDD/F concentrations ranged from 10.8 ± 11.0 to 135 ± 64.3 fg I-TEQ m$^{-3}$, with the highest concentrations found in the samples collected at industrial parks. Over 45.5% to 73.8% of the total PCDD/F concentrations were partitioned in the solid phase, while about 35.2% to 86.3% were distributed in PM$_{2.5}$. The total quantities of the PCDD/F TEQs adsorbed onto PM$_{2.5}$ measured at one northern urban sampling site (1,180 ± 294 pg I-TEQ g$^{-1}$PM$_{2.5}$) and one northern suburban sampling site (1,110 ± 574 pg I-TEQ g$^{-1}$PM$_{2.5}$) were higher than at other sites due to the influence of local municipal solid waste incinerators and long-range transport. Moreover, a significantly higher mortality risk was found for people living in areas with higher exposure rates of PM$_{2.5}$ and PCDD/Fs.

Keywords: PM$_{2.5}$; Dioxin; Relative risk; Long-range transport.

INTRODUCTION

Many of the epidemiological studies have indicated significant correlation between the exposure to fine particulate matter (PM$_{2.5}$) and adverse effects on human health (Dockery et al., 1993; Pope and Dockery, 2006; Corbett et al., 2007; Perez et al., 2009; Samoli et al., 2014; Mohammadi et al., 2016). The fine particles can retain deep in the lungs for long time, and can cause some inflammations (Slezakova et al., 2013), while some chemical in the PM$_{2.5}$ can enhance these effects. A study in the USA found that the mortality of all-cause mortality (1.18%), cardiovascular disease (1.03%), myocardial infarction (1.22%), stroke (1.76%), and respiratory deaths (1.71%) increased for each 10 µg m$^{-3}$ increase in the PM$_{2.5}$ concentrations (Dai et al., 2014). A research done by Taiwan government also found association between monthly PM$_{2.5}$ concentrations and all-cause, cardiovascular diseases, cerebrovascular disease, respiratory diseases, and pneumonia (Cheng et al., 2010). Other studies done in Taiwan also indicated that there was significant correlation between PM$_{2.5}$ and the rate of asthma (Bell et al., 2007), arrhythmia (Tsai et al., 2009), and myocardial infarction (Hsieh et al., 2010).

Polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs) are persistent organic pollutants (POPs), which are among the most ubiquitous and toxic organic pollutants in the environment. PCDD/Fs are formed and released unintentionally from anthropogenic sources, and capable of being transported through long distances to other environmental compartments. Atmosphere is a major pathway for PCDD/Fs to transport and deposit (Kouimtzis et al., 2002). Larger amounts of particle-bound PCDD/Fs were found on smaller particles which had the higher PCDD/Fs TEQs concentrations. Research found that 70% to 80% of the PCDD/Fs in the atmosphere were bound to particles (Oh et al., 2002), and about 50% of the total PCDD/Fs were found on small size particles with aerodynamic particle size less than 1.1 µm, and providing over 47% of the total PCDD/F TEQs (Kurokawa et al., 1998). A study in China...
found the total PCDD/F concentrations and TEQs in the PM$_{2.5}$ samples to be about 66.8% to 108% of the TSP samples, showed that the fine particles contained higher levels of PCDD/Fs than coarse particles (Wen et al., 2011).

These PCDD/F compounds are known to cause wide range of health effects in the immune, endocrine, nervous and reproductive systems of humans and animals (Vallack et al., 1998; Roots et al., 2004). According to the epidemiological studies, there are associations between PCDD/F exposure and circulatory disease, hepatobiliary cancer, lung cancer, colorectal cancer, non-Hodgkin’s lymphoma, female breast cancer, and cardiovascular disease among high- and low-exposure populations (Bertazzi et al., 1993; United States Environmental Protection Agency, 1994; Mitrou et al., 2001; Eskenazi et al., 2004; Consolini et al., 2008; Collins et al., 2009). A research about PCDD/Fs in the fly ash from an MSWI plant found the risk of having bad health outcome (carcinogenic and non-carcinogenic risk) for on-site workers is higher than other groups (Hsieh et al., 2018).

Due to the toxicity, endocrine disturbing effect, carcinogenicity and bioaccumulation, PCDD/F contents in PM$_{2.5}$ have raised great public concern worldwide. Therefore, understanding of the PCDD/F concentrations and distributions in atmosphere at different areas is important. The objective of this study is to monitor the atmospheric PCDD/Fs in vapor phase and solid phase (TSP and PM$_{2.5}$) and compare the association between the PCDD/Fs in PM$_{2.5}$ and compare the mortality between high PCDD/F-exposure group and low PCDD/F-exposure group in Taiwan.

**METHODS**

**Sampling Sites**

In this study, nine sampling sites were selected in the northern, northeastern, and central Taiwan with different characteristic. Nine sampling sites included northern urban area (Site A), northern urban area (Site B), northern traffic-affected zone (Site C), northern suburban area (Site D), northern suburban area (Site E), northeastern urban area (Site F), northeastern industrial park (Site G), central industrial park (Site H), and central industrial park (Site I) (Fig. 1). In northern Taiwan, there are six municipal solid waste incinerators (MSWIs) in the vicinity of sampling stations. The formation and emission of PCDD/F during start-up procedures of MSWIs is considered one of the main sources of PCDD/Fs (Cheruiyot et al., 2016). On the other hand, one MSWI and several electric arc furnace steelmaking processes were located close to northeastern Taiwan sampling station. Meanwhile, the samples collected in central industrial park sampling stations could be affected by the electric arc furnace steelmaking process, coal-fired power plant, waste incinerator, and metal manufacturing.

**Sample Collection and Analysis**

In this study, ambient air samples were collected using the high-volume total suspended particle (TSP) sampler (Shibata, HV-700) and high-volume PM$_{2.5}$ sampler (Analytica), following by the main guidelines of the Taiwan Environmental Protection Administration NIEAA80911B and European Union EN-14907, respectively. The samples were collected during winter season from 2012 to 2014. The samplers were equipped with quartz fiber filters for collecting the PCDD/F compounds in TSP and PM$_{2.5}$ (particle phase), while polyurethane foam (PUF) plugs were used for retaining the PCDD/F compounds in the vapor phase. Both TSP and PM$_{2.5}$ samplers were operating at a flow rate of 500 L min$^{-1}$ for 48 hours per sample; three samples were collected at each sampling site. Before sampling, the quartz fiber filters were baked at 450°C for 5 hours and stored in the aluminum foil packages. Before and after sampling, the quartz fiber filters were weighed in order to obtain the TSP and PM$_{2.5}$ weight. Prior to weighing, the quartz fiber filters were conditioned for 24 to 48 hours at ambient temperature of 20 ± 1°C and relative humidity 35 ± 5%.

For PCDD/Fs analysis, the ambient air samples were then spiked with known amounts of internal quantification standards according to USEPA Method 23. Afterward, the ambient air samples were Soxhlet extracted with toluene for 24 hours, before being treated with concentrated sulfuric acid, then passed through a series of clean-up columns containing sulfuric acid-silica gel, acidic aluminum oxide and celite/carbon. Finally, the seventeen 2,3,7,8-substituted PCDD/F congeners were analyzed with high-resolution gas chromatography (HRGC)/high-resolution mass spectrometry.

![Fig. 1. Relative locations of sampling Sites in Taiwan.](image-url)
(HRMS) (Waters AutoSpec-Ultima and JEOL JMS-700) equipped with a fused silica capillary column DB-5 MS (60 m × 0.25 mm × 0.25 μm, i&W). For PCDD/Fs data analysis, the International Toxic Equivalent Factors (I-TEFs) were adopted to compare the potential toxicity of each PCDD/F congeners in a mixture with the toxicity of TCDD, the most toxic member of the group (Bellin and Barnes, 1987). The I-TEFs of each PCDD/F congeners present in a mixture was multiplied by the respective mass concentration, and the products were then summed to the 2,3,7,8-TCDD International Toxic Equivalence (I-TEQ) of the mixture.

**Statistical Analysis**

The relationship between PM$_{2.5}$ mass concentrations, PCDD/F TEQs concentrations and mortality was analyzed based on Taiwan national data. The data of automatically monitoring PM$_{2.5}$ concentrations from Taiwan Environmental Protection Administration from 2006 to 2014 was collected. The PCDD/Fs TEQs concentrations in PM$_{2.5}$ over that period of time was also estimated using the formula shown in Section 3.4. In addition, the mortality data at the same period of time was collected from National Mortality Registry data of the Ministry of Health and Welfare of Taiwan. The causes of death were categorized using the International Classification of Diseases, Tenth Revision (ICD-10). In this study, the mortality cause including the all causes of death (A00-Y98); cerebrovascular disease (I60-I69); pneumonia (J12-J18); malignant neoplasms (C00-C97); cancers of liver and intrahepatic bile ducts (C22); cancers of trachea, bronchus and lung (C33-C34); cancers of colon, rectum and anus (C18-C21); and female breast cancer (C50). The relationship between the pollutants concentrations and the diseases mortality were evaluated with the Poisson-regression model by SAS 9.3.

**RESULTS AND DISCUSSION**

**TSP and PM$_{2.5}$ Mass Concentration in Ambient Air at Different Areas**

In this study, the mass concentrations of TSP were $130 ± 26.7, 71.2 ± 16.5, 183 ± 33.1, 135 ± 28.3, 43.5 ± 16.0, 25.7 ± 11.1, 31.8 ± 21.7, 134 ± 49.3, and 150 ± 42.3$ µg m$^{-3}$ at Site A, B, C, D, E, F, G, H, and I, respectively (Table 1). On the other hand, the mass concentrations of PM$_{2.5}$ were $25.5 ± 4.74, 30.3 ± 4.95, 12.1 ± 8.85, 12.2 ± 3.75, 21.7 ± 3.57, 10.6 ± 4.21, 11.5 ± 7.16, 46.2 ± 2.09, and 60.4 ± 7.39$ µg m$^{-3}$ at those nine sampling sites (Table 1). A study found the annual average PM$_{2.5}$ concentrations in Taiwan to be around 20 µg m$^{-3}$. These concentrations were found to be lower in remote areas (Tsai et al., 2015) and elevate in urban and industrial cities (Lee et al., 2016; Lu et al., 2016; Lee et al., 2018). In our study, the TSP and PM$_{2.5}$ mass concentrations found at urban areas ranged from 25.7 ± 11.1 to 130 ± 26.7 µg m$^{-3}$ and 10.6 ± 4.21 to 30.3 ± 4.95 µg m$^{-3}$, respectively. The concentrations of TSP and PM$_{2.5}$ at central industrial park were higher at 134 ± 49.3 µg m$^{-3}$ and 46.2 ± 2.09 µg m$^{-3}$, especially at Site I, the concentrations of TSP and PM$_{2.5}$ were 150 ± 42.3 and 60.4 ± 7.39 µg m$^{-3}$. The ratios of PM$_{2.5}$ and TSP mass concentrations were 19.6% to 42.6% for urban areas, 6.61% for traffic-affected zone, 9.04% and 49.9% for suburban areas, 34.5% to 40.3% for industrial park. The results indicated that the coarse particles were dominant at all sampling sites, especially at traffic-affected zone. At northern traffic-affected zone (Site C), no industrial activity was found in the vicinity, hence, the traffic pollutions and road dusts were the major emission sources for this site. During the procedure of sampling at all sampling areas, we also collected the automatic monitoring of PM$_{2.5}$ concentrations from Taiwan Environmental Protection Administration (Taiwan EPA) at the nearby monitoring stations. The manual monitoring of PM$_{2.5}$ concentrations at different sampling sites, and the ratio of the sampling method between our field measurement and Taiwan EPA were shown in Table 1. A high corresponding rate between data collected in the field and the Taiwan EPA measurement was found (except traffic affected zone which is quite specific and far away from the nearest monitoring station). This finding provides us confidence to assume the constancy of samples collected in this research with data from Taiwan EPA (which will be further explained in Section 3.4).

**The Distribution of PCDD/F Concentrations in Solid Phase and Vapor Phase in Ambient Air at Different Sampling Areas**

The total PCDD/Fs (vapor + TSP) concentrations showed the differentiation among sampling stations (Table 2). The
Table 2. Average PCDD/F concentrations in vapor and solid phases at different sampling sites.

<table>
<thead>
<tr>
<th>Sampling Sites</th>
<th>Site A (Northern urban area)</th>
<th>Site B (Northern urban area)</th>
<th>Site C (Northern traffic-affected zone)</th>
<th>Site D (Northern suburban area)</th>
<th>Site E (Northern suburban area)</th>
<th>Site F (Northeastern urban area)</th>
<th>Site G (Northeastern industrial park)</th>
<th>Site H (Central industrial park)</th>
<th>Site I (Central industrial park)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ΣPCDDs, fg I-TEQ m–3</td>
<td>2.20 ± 1.80</td>
<td>3.75 ± 1.45</td>
<td>5.25 ± 3.48</td>
<td>0.49 ± 0.24</td>
<td>0.98 ± 0.74</td>
<td>2.11 ± 0.98</td>
<td>4.16 ± 3.30</td>
<td>2.08 ± 0.98</td>
<td>1.67 ± 0.60</td>
</tr>
<tr>
<td>ΣPCDFs, fg I-TEQ m–3</td>
<td>7.97 ± 2.33</td>
<td>13.1 ± 5.88</td>
<td>8.65 ± 3.94</td>
<td>6.52 ± 10.7</td>
<td>10.7 ± 10.7</td>
<td>3.27 ± 1.10</td>
<td>6.25 ± 10.7</td>
<td>6.52 ± 10.7</td>
<td>6.25 ± 10.7</td>
</tr>
<tr>
<td>ΣPCDD/Fs, fg I-TEQ m–3</td>
<td>10.4 ± 3.49</td>
<td>17.2 ± 5.78</td>
<td>11.3 ± 4.37</td>
<td>4.54 ± 3.30</td>
<td>7.95 ± 7.04</td>
<td>3.34 ± 1.30</td>
<td>7.76 ± 10.7</td>
<td>8.94 ± 10.7</td>
<td>8.94 ± 10.7</td>
</tr>
<tr>
<td>ΣPCDD/Fs (Vapor + TSP), fg I-TEQ m–3</td>
<td>17.6 ± 5.80</td>
<td>56.8 ± 9.65</td>
<td>10.8 ± 11.0</td>
<td>21.9 ± 35.0</td>
<td>31.5 ± 16.1</td>
<td>15.9 ± 31.5</td>
<td>24.3 ± 35.0</td>
<td>33.0 ± 16.1</td>
<td>33.0 ± 16.1</td>
</tr>
</tbody>
</table>

Hung et al. (2004) investigated the particle size distribution of PCDD/Fs in ambient air and indicated that over 47% of the total PCDD/F TEQs were found on small particles with the diameters less than 1.1 µm, and providing over 47% of the total PCDD/F TEQs (Kurokawa et al., 1998). The I-TEQ concentration of PCDD/Fs in PM2.5 at Site C (northern traffic-affected zone) was 22.7-fold more than PM >2.5 (11.1 ± 8.10 pg I-TEQ g-PM2.5). This huge difference suggested that further evaluation about PCDD/Fs in PM2.5 emitted from mobile sources should be conducted in the future research. The PCDD/Fs in PM2.5 ranged from 447 ± 137 to 1,180 ± 294 pg I-TEQ g-PM2.5 at urban areas (Site A, B, F), 925 ± 205 pg I-TEQ g-PM2.5 at Site C, while at suburban areas (Site D and E), the PCDD/Fs contents in PM2.5 were 214 ± 350 and 1,110 ± 574 pg I-TEQ g-PM2.5, respectively. On the other hand, in the industrial park (Site G, H, I), the PCDD/Fs contents in PM2.5 were 1,090 ± 3.74, 833 ± 149, and 1,060 ± 488 pg I-TEQ g-PM2.5, respectively. The higher level of PCDD/Fs compared to coarse particles. The I-TEQ concentrations of PCDD/Fs in PM2.5 samples were 60.0% to 86.3% of that of TSP samples. A previous study investigated the particle size distribution of PCDD/Fs in atmosphere and indicated that over 50% of PCDD/Fs were found on small particles with the diameters less than 1.1 µm, and providing over 47% of the total PCDD/F TEQs (Kurokawa et al., 1998). Fig. 2 shows the PCDD/F congener distributions in particle phase and vapor phase at different sampling sites. The distribution of 2,3,7,8-substituted PCDD/F congeners was calculated based on the mass concentration. In all three matrices (TSP phase, PM2.5 phase, vapor phase), higher contribution of I-TEQ concentration was from PCDF congeners comparing with that of PCDD congeners. The distribution of 2,3,7,8-substituted PCDD/F congeners was calculated based on the mass concentration. In all three matrices (TSP phase, PM2.5 phase, vapor phase), higher contribution of I-TEQ concentration was from PCDF congeners comparing with that of PCDD congeners. Our results also indicated that the high chlorinated-level of PCDD/Fs tend to be the most abundant congeners in solid phase, while less chlorinated-level of PCDFs tend to be distributed in vapor phase.

Particle-bound PCDD/Fs in Different Size of Particulate Matter in Ambient Air

The particle-bound PCDD/Fs in TSP, PM2.5, and PM1.0 at different sampling sites in Taiwan are shown in Fig. 3. Comparing the results of PCDD/F TEQ contents, the total amount of PCDD/Fs in PM2.5 were 2.04 to 22.7 time higher than that of PM1.0. At Site C (northern traffic-affected zone), the particle-bound PCDD/Fs in PM1.0 (251 ± 205 pg I-TEQ g-PM1.0) was 22.7-fold more than PM2.5 (11.1 ± 8.10 pg I-TEQ g-PM2.5). This huge difference suggested that further evaluation about PCDD/Fs in PM1.0 emitted from mobile sources should be conducted in the future research. The PCDD/Fs bound to PM2.5 ranged from 447 ± 137 to 1,180 ± 294 pg I-TEQ g-PM2.5 at urban areas (Site A, B, F), 251 ± 205 pg I-TEQ g-PM2.5 at Site C, while at suburban areas (Site D and E), the PCDD/Fs contents in PM2.5 were 214 ± 350 and 1,110 ± 574 pg I-TEQ g-PM2.5, respectively. On the other hand, in the industrial park (Site G, H, I), the PCDD/Fs contents in PM2.5 were 1,090 ± 3.74, 833 ± 149, and 1,060 ± 488 pg I-TEQ g-PM2.5, respectively.
Fig. 2. PCDD/F congener distribution observed in the atmosphere at different sampling Sites.

Fig. 3. The particle-bound PCDD/Fs in total suspended particle (TSP), coarse particle (PM$_{>2.5}$) and fine particle (PM$_{2.5}$) at different sampling Sites.

The PCDD/F TEQs contents at Site B (1,180 ± 294 pg I-TEQ g-PM$_{2.5}$$^{-1}$) and Site E (1,110 ± 574 pg I-TEQ g-PM$_{2.5}$$^{-1}$) were much higher than that measured at other sampling sites. An MSWI located in the upwind of the two sampling stations might be the cause of high concentration of PM$_{2.5}$-bound PCDD/Fs in these two stations. On the other hand, Site B and E were located in the northern part of Taiwan which was affected by winter monsoon during sampling period. Many previous research found that, during monsoon period, pollutants can be transported from other areas to Taiwan (Chi et al., 2016; Chi et al., 2017). Our previous studies (Chi et al., 2013) indicated that during the winter
monsoon period, the quantity of PCDD/Fs in TSP at background site, was found to increase from 300 ± 127 to 630 ± 115 pg I-TEQ g-TSP−1, hence, the winter monsoon not only brings cold air but also transports air pollutants for long distances from other places to northern Taiwan (Chi et al., 2017). The study in 1997 found that the PCDD/F contents in suspended particles measured in rural and urban areas were 320 and 630 pg I-TEQ g−1, respectively (Wallenhorst et al., 1997), which was higher than those measured at urban areas of this study. In addition, the highest PM2.5 mass concentrations was observed at Site I (60.4 ± 7.39 µg m−3), an industrial park sampling station, but highest PCDD/F contents was measured at Site B (1,180 ± 294 pg I-TEQ g-PM2.5−1), an urban area. The result indicated that the high mass concentration of PM2.5 was not corresponding to high PCDD/F contents.

The Relative Risk of Mortality Associated with Groups Exposed to High and Low PM2.5 and PCDD/F Concentrations

In order to estimate the PCDD/Fs TEQs concentrations in PM2.5, we assumed that variations of the concentrations of PCDD/Fs in ambient air at different areas were constant, and the ratio of sampling method between EN-14907 and U.S. EPA FEM was also stable. The total quantity of PCDD/Fs in PM2.5 (pg I-TEQ g-PM2.5−1) measured in this study, the ratio of EN-14907/U.S. EPA FEM, and the automatically monitored PM2.5 concentrations from EPA of Taiwan were used to estimate the PCDD/F concentrations over the ten past years through the following formula:

\[ \text{Conc.} = A \times B \times C \]  

where \( \text{Conc.} \): PCDD/F estimated concentrations (fg I-TEQ m−³). \( A \): The total quantity of PCDD/Fs in PM2.5 in this study (pg I-TEQ g-PM2.5−1). \( B \): The ratio of manual monitoring and automatic monitoring in PM2.5 (EN-14907/U.S. EPA FEM). \( C \): The automatic monitoring of PM2.5 concentrations from EPA, Taiwan (µg m−³).

According to the automatic monitoring of PM2.5 concentrations from EPA, Taiwan, the highest PM2.5 mass concentrations was measured at Site H, the lowest PM2.5 mass concentrations were obtained at Site D and Site G. For the PCDD/F concentrations, the highest TEQs concentrations were measured at Site I, the lowest TEQs concentrations were obtained at Site D and Site F. In this study, we evaluated the relative risk of mortality between groups exposed to high and low PM2.5 mass concentrations and PCDD/F TEQ concentrations. Table 3 showed the results of the relative risk of mortality associated with PM2.5 concentrations between Site H and Site D. In the whole population or population stratified by genders, deaths of all causes are significantly higher in Site H (places with higher PM2.5 mass concentration) comparing to Site D. In other specific cause of death, significant elevations of relative risk were found for death caused by cerebrovascular disease; malignant neoplasms; cancers of liver and intrahepatic bile ducts; cancers of colon, rectum and anus; cancers of trachea, bronchus and lung (in male); pneumonia (in male); and...
Table 4. The relative risk of mortality between high and low PCDD/Fs TEQs concentrations exposure group.

<table>
<thead>
<tr>
<th>Mortality cause (ICD-10*)</th>
<th>Site I vs. Site D</th>
<th>ALL</th>
<th>RR (95% CI)</th>
<th>P-value</th>
<th>Male</th>
<th>RR (95% CI)</th>
<th>P-value</th>
<th>Female</th>
<th>RR (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>All causes of death (A00-Y98)</td>
<td></td>
<td>ALL</td>
<td>1.029 (1.028–1.030)</td>
<td>&lt; .0001</td>
<td>1.028 (1.026–1.029)</td>
<td>&lt; .0001</td>
<td>1.030 (1.029–1.032)</td>
<td>&lt; .0001</td>
<td></td>
<td></td>
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<tr>
<td>Cerebrovascular disease (I60-I69)</td>
<td></td>
<td>ALL</td>
<td>1.027 (1.022–1.031)</td>
<td>&lt; .0001</td>
<td>1.020 (1.016–1.025)</td>
<td>&lt; .0001</td>
<td>1.034 (1.029–1.039)</td>
<td>&lt; .0001</td>
<td></td>
<td></td>
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<tr>
<td>Pneumonia (J12-J18)</td>
<td></td>
<td>ALL</td>
<td>1.022 (1.017–1.028)</td>
<td>&lt; .0001</td>
<td>1.022 (1.018–1.027)</td>
<td>&lt; .0001</td>
<td>1.025 (1.019–1.032)</td>
<td>&lt; .0001</td>
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<td></td>
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<tr>
<td>Malignant neoplasms (C00-C97)</td>
<td></td>
<td>ALL</td>
<td>1.026 (1.023–1.028)</td>
<td>&lt; .0001</td>
<td>1.028 (1.026–1.030)</td>
<td>&lt; .0001</td>
<td>1.020 (1.018–1.023)</td>
<td>&lt; .0001</td>
<td></td>
<td></td>
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<tr>
<td>Cancers of liver and intrahepatic bile ducts (C22)</td>
<td></td>
<td>ALL</td>
<td>1.019 (1.013–1.025)</td>
<td>&lt; .0001</td>
<td>1.019 (1.014–1.024)</td>
<td>&lt; .0001</td>
<td>1.006 (0.998–1.015)</td>
<td>0.140</td>
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<tr>
<td>Cancers of trachea, bronchus and lung (C33-C34)</td>
<td></td>
<td>ALL</td>
<td>1.009 (1.004–1.014)</td>
<td>0.001</td>
<td>1.013 (1.008–1.017)</td>
<td>&lt; .0001</td>
<td>0.999 (0.993–1.006)</td>
<td>0.799</td>
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<tr>
<td>Cancers of colon, rectum and anus (C18-C21)</td>
<td></td>
<td>ALL</td>
<td>1.021 (1.015–1.028)</td>
<td>&lt; .0001</td>
<td>1.015 (1.008–1.021)</td>
<td>&lt; .0001</td>
<td>1.025 (1.017–1.032)</td>
<td>&lt; .0001</td>
<td></td>
<td></td>
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<tr>
<td>Female breast cancer (C50)</td>
<td></td>
<td>ALL</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>0.987 (0.978–0.996)</td>
<td>0.006</td>
<td></td>
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</tbody>
</table>

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<tr>
<td>All causes of death (A00-Y98)</td>
<td></td>
<td>ALL</td>
<td>1.046 (1.044–1.048)</td>
<td>&lt; .0001</td>
<td>1.043 (1.041–1.047)</td>
<td>&lt; .0001</td>
<td>1.049 (1.047–1.052)</td>
<td>&lt; .0001</td>
<td></td>
<td></td>
</tr>
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<td>Cerebrovascular disease (I60-I69)</td>
<td></td>
<td>ALL</td>
<td>1.028 (1.021–1.035)</td>
<td>&lt; .0001</td>
<td>1.018 (1.011–1.023)</td>
<td>&lt; .0001</td>
<td>1.039 (1.032–1.047)</td>
<td>&lt; .0001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pneumonia (J12-J18)</td>
<td></td>
<td>ALL</td>
<td>1.026 (1.017–1.035)</td>
<td>&lt; .0001</td>
<td>1.031 (1.023–1.039)</td>
<td>&lt; .0001</td>
<td>1.030 (1.019–1.041)</td>
<td>&lt; .0001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant neoplasms (C00-C97)</td>
<td></td>
<td>ALL</td>
<td>1.046 (1.042–1.051)</td>
<td>&lt; .0001</td>
<td>1.048 (1.045–1.052)</td>
<td>&lt; .0001</td>
<td>1.041 (1.036–1.046)</td>
<td>&lt; .0001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cancers of liver and intrahepatic bile ducts (C22)</td>
<td></td>
<td>ALL</td>
<td>1.059 (1.046–1.072)</td>
<td>&lt; .0001</td>
<td>1.068 (1.057–1.079)</td>
<td>&lt; .0001</td>
<td>1.018 (1.001–1.035)</td>
<td>0.038</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cancers of trachea, bronchus and lung (C33-C34)</td>
<td></td>
<td>ALL</td>
<td>1.042 (1.030–1.053)</td>
<td>&lt; .0001</td>
<td>1.041 (1.031–1.051)</td>
<td>&lt; .0001</td>
<td>1.038 (1.023–1.053)</td>
<td>&lt; .0001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cancers of colon, rectum and anus (C18-C21)</td>
<td></td>
<td>ALL</td>
<td>1.038 (1.026–1.050)</td>
<td>&lt; .0001</td>
<td>1.032 (1.020–1.043)</td>
<td>&lt; .0001</td>
<td>1.039 (1.027–1.052)</td>
<td>&lt; .0001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female breast cancer (C50)</td>
<td></td>
<td>ALL</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>0.987 (0.971–1.004)</td>
<td>0.136</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

ICD-10: International Classification of Disease, Tenth Revision. RR: Relative Risk, CI: Confidence interval.
female breast cancer in the group exposed to high PM$_{2.5}$ mass concentrations (Site H) comparing to the group exposed to low PM$_{2.5}$ mass concentrations (Site D). The model was also performed to relative risk of death due to PM$_{2.5}$ between Site H and Site G, however, due to the lack of data, the result of the analysis was not shown. For the correlation between the PCDD/F TEQ concentrations and mortality, the group exposed to high PCDD/F TEQ concentrations had significantly higher relative risk for death of all causes; cerebrovascular disease; pneumonia; malignant neoplasms; cancers of liver and intrahepatic bile ducts; cancers of trachea, bronchus and lung; cancers of colon, rectum and anus; and female breast cancer for both males and females than the group exposed to low PCDD/F TEQ concentrations for two cases (Site I versus Site D, and Site I versus Site F) (Table 4).

Because of the lack of background data of people at the areas, the health risk analysis could not include adjustment for some important confounders such as smoking status or alcohol using. However, this research did give a preliminary suggestion of the relationship between PM$_{2.5}$ and human health risk. Furthermore, the inclusion of PCDD/Fs suggested that it is important to include the composition of PM$_{2.5}$ when doing the health risk analysis of this type of pollutant.

CONCLUSIONS

This research analyzed the ambient air PCDD/Fs at nine sampling sites in Taiwan during the winter seasons from 2012 to 2014. It was found that the mean PCDD/F concentrations measured at an industrial park ($135 \pm 64.3$ fg I-TEQ m$^{-3}$, Site I) were significantly higher than those measured in other areas. More than half (45.5%–73.8%) of total PCDD/F concentrations were allocated in the solid phase, among other areas. More than half (45.5%–73.8%) of total PCDD/F concentrations were allocated in the solid phase, among other areas. More than half (45.5%–73.8%) of total PCDD/F concentrations were allocated in the solid phase, among other areas. More than half (45.5%–73.8%) of total PCDD/F concentrations were allocated in the solid phase, among other areas.

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