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Journal of Hazardous Materials B127 (2005) 33–39

Journal of Hazardous Materials

www.elsevier.com/locate/jhazmat

Using exterior building surface films to assess human exposure and health risks from PCDD/Fs in New York City, USA, after the World Trade Center attacks

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Received 24 October 2003; received in revised form 16 January 2004; accepted 5 April 2004
Available online 3 August 2004

Abstract

Concentrations of tetra- through octa-chlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs) were determined in exterior window films from Manhattan and Brooklyn in New York City (NYC), USA, 6 weeks after the World Trade Center (WTC) attacks of 11 September 2001. High concentrations of the 2,3,7,8-substituted congeners (P_{2378}CDD/Fs) were observed, at levels up to 6600 pg-TEQ g^{-1} nearest the WTC site. An equilibrium partitioning model was developed to reconstruct total gas + particle-phase atmospheric concentrations of P_{2378}CDD/Fs at each site. The reconstructed atmospheric and window film concentrations were subsequently used in a preliminary human health risk assessment to estimate the potential cancer and non-cancer risks posed to residents of lower Manhattan from these contaminants over the 6 week exposure period between the WTC attacks and sampling dates. Residents of lower Manhattan appear to have a slightly elevated cancer risk (up to 1.6% increase over background) and increased P_{2378}CDD/F body burden (up to 8.0% increase over background) because of above-background exposure to high concentrations of P_{2378}CDD/Fs produced from the WTC attacks during the short period between 11 September 2001, and window film sampling 6 weeks later.

Keywords: World Trade Center attacks; Building surface films; Polychlorinated dibenzo-*p*-dioxins and dibenzofurans; Atmospheric concentration reconstruction; Risk assessment

1. Introduction

The World Trade Center (WTC) attacks in New York City (NYC) on 11 September 2001 destroyed the twin towers and adjacent structures of this complex and resulted in the death of nearly 3000 persons. Previous work has shown increased exposure to numerous contaminant classes, both organic and inorganic, for residents and worker in affected areas of NYC from these terrorist attacks [1–3]. Among the various organic contaminants of concern are polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs) which are the focus of the current study. Six weeks following the attacks of 11 September 2001, window films on building exteriors at seven NYC sites in Lower Manhattan and one site in Brooklyn were sampled [4]. Window films such as these provide a sample of the complex mixture of semivolatile organic compounds (SVOCs; e.g. PCBs, PCDD/Fs, PAHs) that humans and biota are exposed to in urban and rural environments. Previous analyses of these window films demonstrated extremely high concentrations of PCDD/Fs, at levels near the WTC that are among the highest ever reported PCDD/F concentrations in abiotic compartments and are in the upper range of even the most contaminated incinerator ash [4]. However, the potential human health risks of these high PCDD/F concentrations, particularly the 2,3,7,8-substituted congeners (P_{2378}CDD/F), were not considered in detail in this previous work. Thus, the present study aims to provide a more complete estimate of potential atmospheric P_{2378}CDD/F levels in lower Manhattan following the WTC attacks of 11 September 2001, and to

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doi:10.1016/j.jhazmat.2004.04.022

examine the potential health risks posed by such high levels of these contaminants residing in close proximity to residents of the affected areas.

2. Methods

2.1. Sampling and analysis

Organic film samples were collected from the outside of windows at eight sites (see Fig. 1 for locations) in lower Manhattan and Brooklyn, NYC, USA, by scrubbing the surfaces with pre-cleaned laboratory Kimwipes® soaked in HPLC grade isopropanol. All sampling locations were on window surfaces facing the WTC site. Sampling was conducted between 27 and 29 October 2001. Average air temperatures over the period from 11 September 2001 to 27 October 2001 were calculated using meteorological data provided by the National Weather Service and available at http://www.nws.bnl.gov/climate.html. The average temperature of 290.1 K is the geometric mean of daily average temperatures over this period. Samples were collected from either ground level or second story windows and a 10 cm border was left on all windows to prevent direct contamination from building materials. Following collection, samples were stored in pre-cleaned glass jars in the dark and frozen at −20 °C until analysis. Further details on the collection of urban organic films using this method, along with quality control/quality assurance (QA/QC) protocols and validation for SVOCs, and sample processing and analysis by high-resolution gas-chromatography with high-resolution mass spectrometry (HRGC–HRMS) are provided elsewhere [4,5].

2.2. Atmospheric concentration reconstruction

Concentrations of the 17 P_{2378}CDD/F analytes at each sampling location in units of pg analyte per gram of exterior window film (pg g^{-1}) are provided in Table 1, and are also available elsewhere [4]. Analyte concentrations in pg g^{-1} were converted to toxic equivalents (TEQs) through multiplication by the respective World Health Organization toxic equivalence factors (TEFs) for each analyte [6,7] and are also provided in Table 1. Window film concentrations in units of pg analyte per square meter of window surface (pg m^{-2}), available in previous work from our group [4], were converted to units of pg analyte per cubic meter of window film (pg m^{-3}) through multiplication by the average window film thickness of 1.2×10^{-7} m (120 nm) and are presented in Table 2. Octanol-air partition coefficients at the average daily temperature in NYC (290.1 K) over the period from 11th September through 25 October 2003, were calculated for each analyte using the regression equations provided elsewhere [8] and are presented in Table 2. Gas phase atmospheric concentrations of each analyte were subsequently calculated using the following relationship [4],

$$C_{\text{gas-phase}} = \frac{C_{\text{film}}}{K_{\text{oa}} \times F_{\text{om}}}$$

where $C_{\text{gas-phase}}$ and C_{film} are the analyte concentrations (in pg m^{-3}) in the gas phase of the atmosphere and the exterior window film, respectively, K_{oa} is the octanol-air partition coefficient at the temperature of interest (i.e. 290.1 K), and f_{om} is the fraction of organic matter in the film. Previous work has shown exterior building surface films to contain approximately 20% organic matter by weight [5,9], hence f_{om} was assumed to equal 0.2 for the present study. To convert the

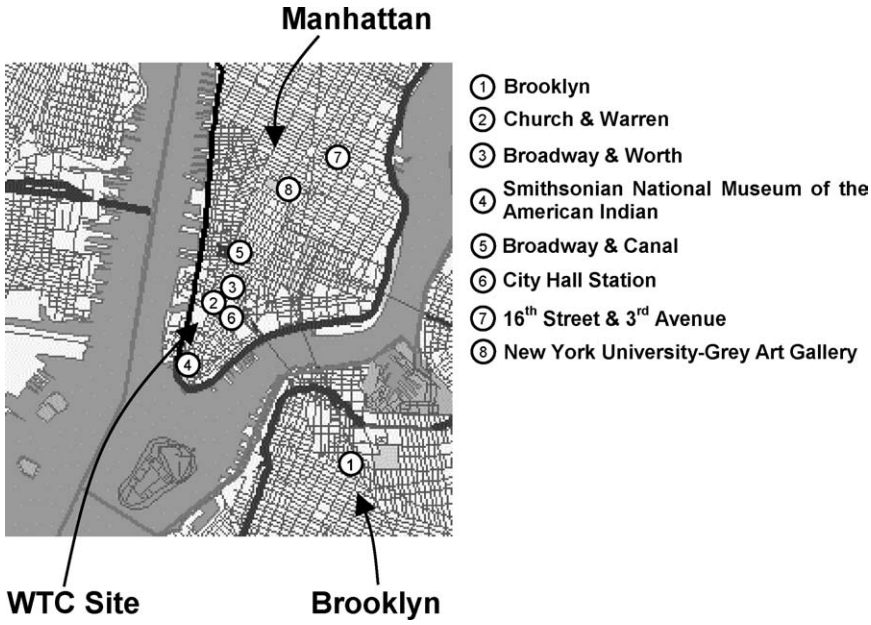

Fig. 1. Exterior window film sampling locations in New York City, USA.

gas phase concentrations into gas + particle-phase concentrations, the following relationship was used [8],

$$C_{\text{gas+particle-phase}} = \frac{C_{\text{gas-phase}}}{(1-\phi)}$$

where φ is the fraction of total contaminant on particulates (i.e. $(1-\varphi)$ is the fraction of total contaminant in the gas phase). To calculate $(1-\varphi)$, the particle–gas partitioning constant (K_P) was first calculated using the following relationship: $\log K_P = \log K_{oa} + \log f_{om} - 11.91$ [8]. Assuming an average total suspended particulate concentration (TSP) of 70 μg m^{-3} in the sampling region over the period of interest [2], $(1-\varphi)$ may be calculated using the following formula [8],

$$1-\phi = 1 - \left[\frac{K_P \times \text{TSP}}{(K_P \times \text{TSP}) + 1}\right]$$

where φ is the fraction of contaminant on suspended particulates, K_P is the particle–gas partition coefficient, and TSP is the total suspended particulate concentration. To convert the gas phase analyte concentrations calculated above into gas + particle-phase concentrations, the gas phase concentrations were divided by $(1-\varphi)$ as follows:

$$C_{\text{gas+particle-phase}} = \frac{C_{\text{gas-phase}}}{(1-\phi)}$$

The reconstructed gas + particle-phase concentrations in both units of fg m^{-3} and fg-TEQ m^{-3} are presented in Table 2.

2.3. Human exposure and risk assessment

The daily inhalation exposure dose can be calculated as $DD_{in} = [IN \times HRD \times C \times ABS]/BW$ where DD is the daily dose (pg-TEQ kg^{-1} d^{-1}), IN is the inhalation rate (m^3 h^{-1}), HRD is the hours per day of inhalation (h), C is the analyte concentration (pg-TEQ m^{-3}; equal to $C_{\text{gas+particle-phase}}$ as defined above), ABS is the fraction of contaminant inhaled which is absorbed (dimensionless; assumed to equal 0.8), and BW is the body mass (kg) assumed to be 70 kg for the average adult) [2]. The soil ingestion exposure dose can be calculated as $DD_{si} = SIR \times C \times ABS/BW$ where SIR is the soil ingestion rate (g d^{-1}; assumed to equal 0.050 g d^{-1}), C is the analyte concentration (pg-TEQ g^{-1}; equal to C_{film} as defined above), ABS is the fraction of contaminant ingested with soil which is absorbed (dimensionless; assumed to equal 1.0), and BW is the body mass (assumed to be 70 kg for the average adult) [7]. The soil dermal contact absorption dose can be calculated as $DD_{sdc} = SCR \times C \times ABS/BW$ where SCR is the soil contact rate (g d^{-1}; assumed to equal 12 g d^{-1}), C is the analyte concentration (pg-TEQ g^{-1}; equal to C_{film} as defined above), ABS is the fraction of contaminant contacted dermally in soil which is subsequently absorbed (dimensionless; assumed to equal 9.93×10^{-4}), and BW is the body mass (assumed to be 70 kg for the average adult) [7]. The average daily dose (ADD; pg-TEQ kg^{-1} d^{-1}) for each of

Table 1
Concentrations (in pg-TEQ g^{-1}) of the 17 P_{2378}CDD/F congeners in exterior window films from New York City, USA

	Brooklyn	Manhattan (Church & Warren)	Manhattan (Broadway & Worth)	Manhattan (Smithsonian NMAI)	Manhattan (Broadway & Canal)	Manhattan (City Hall Station)	Manhattan (16th Street & 3rd Avenue)	Manhattan (NYU-Grey Art Gallery)
2378DF	2.5(25)	790(7,900)	170(1,700)	210(2,100)	60(600)	50(500)	24(240)	0.76(7.6)
12378DF	0.15(3.0)	0.97(19)	0.18(3.6)	1.7(33)	0.12(2.4)	0.27(5.4)	0.42(8.4)	0.090(1.8)
23478DF	24(48)	10(20.0)	1.8(3.6)	16(32)	1.2(2.4)	2.7(5.4)	330(650)	0.90(1.8)
123478DF	5.4(54)	490(4,900)	280(2,800)	180(1,800)	100(1,000)	81(810)	58(580)	1.7(17)
123678DF	0.20(2.0)	0.96(9.6)	0.34(3.4)	0.64(6.4)	0.18(1.8)	0.48(4.8)	0.78(7.8)	0.080(0.80)
234678DF	3.6(36)	250(2,500)	170(1,700)	120(1,200)	67(670)	53(530)	37(370)	1.3(13)
123789DF	0.20(2.0)	41(410)	22(220)	16(160)	8.4(84)	5.8(58)	3.4(34)	0.080(0.80)
1234678DF	3.3(33)	860(8,600)	50(5,000)	35(3,500)	19(1,900)	16(1,600)	11(1,100)	0.56(56)
1234789DF	0.24(24)	11(1,100)	7.8(780)	4.6(460)	3.1(310)	2.4(240)	0.072(7.2)	0.010(1.0)
OCDF	0.040(400)	0.33(3,300)	0.11(1,100)	0.14(1,400	0.049(490)	0.040(400)	0.034(340)	0.0030(30)
2378DD	3.8(3.8)	910(910)	260(260)	260(260)	82(82)	5.4(5.4)	15(15)	1.4(1.4)
12378DD	14(14)	3,010(3,010)	1,600(1,600)	960(960)	540(540)	464(464)	250(250)	9.4(9.4)
123478DD	3.0(30)	216(2,160)	150(1,500)	77(770)	50(500)	46(460)	29(290)	1.4(14)
123678DD	4.8(48)	305(3,100)	210(2,100)	120(1,200)	72(720)	66(660)	42(420)	2.0(20)
123789DD	3.6(36)	360(3,600)	170(1,700)	110(1,100)	83(830)	72(720)	33(330)	0.16(1.6)
1234678DD	5.4(540)	86(8,600)	41(4,100)	36(3,600)	16(1,600)	16(1,600)	13(1,300)	0.86(86)
OCDD	0.41(4,100)	3.0(30,000)	0.34(3,400)	1.3(13,000)	0.15(1,500)	0.18(1,800)	0.36(3,600)	0.035(3,500)
ΣP_{2378}CDD/F	75(5,700)	6,600(80,000)	3,100(28,000)	2,100(32,000)	1,100(11,000)	880(9,900)	850(9,500)	21(620)

Values in parentheses are concentrations in units of pg g^{-1}.

Table 2
Calculated logarithmic octanol-air partition coefficients (log K_{oa}), gas-particle partitioning coefficients (K_p), fraction of analyte in the gas phase ($1-\varphi$), and atmospheric gas + particle-phase TEQ concentrations (in fg TEQ m^{-3}) of the 17 P_{2378}CDD/F congeners at each exterior window film sampling site in New York City, USA

	log K_{oa}	K_p	$1-\varphi$	Brooklyn	Manhattan (Church & Warren)	Manhattan (Broadway & Worth)	Manhattan (Smithsonian NMAI)	Manhattan (Broadway & Canal)	Manhattan (City Hall Station)	Manhattan (16th Street & 3rd Avenue)	Manhattan (NYU-Grey Art Gallery)
2378DF	10.28	0.0047	0.75	3.6(36)	1,100(11,000)	250(2,500)	300(3,000)	87(870)	72(720)	34(340)	1.1(11)
12378DF	10.74	0.013	0.51	0.11(2.2)	0.72(14)	0.13(2.7)	1.2(24)	0.090(1.8)	0.20(4.0)	0.31(6.2)	0.065(1.3)
23478DF	10.86	0.018	0.45	16(31)	6.5(13)	1.2(2.3)	10(21)	0.78(1.6)	1.8(3.5)	210(420)	0.58(1.2)
123478DF	11.28	0.047	0.23	2.5(25)	230(2,300)	130(1,300)	84(840)	48(480)	38(380)	27(270)	0.78(7.8)
123678DF	11.30	0.049	0.23	0.093(0.93)	0.45(4.5)	0.16(1.6)	0.30(3.0)	0.083(0.83)	0.22(2.2)	0.36(3.6)	0.037(0.37)
234678DF	11.39	0.061	0.19	1.6(16)	110(1,100)	76(760)	53(530)	30(300)	24(240)	17(170)	0.59(5.9)
123789DF	11.46	0.070	0.17	0.086(0.86)	18(180)	9.7(97)	6.8(68)	3.6(36)	2.5(25)	1.5(15)	0.035(0.35)
1234678DF	11.80	0.155	0.085	1.3(130)	34(3,400)	20(2,000)	14(1,400)	7.5(750)	6.2(620)	4.1(410)	0.22(22)
1234789DF	11.81	0.160	0.082	0.094(9.4)	4.4(440)	3.1(310)	1.8(180)	1.22(121.93)	0.93(93)	0.028(2.8)	0.0039(0.39)
OCDF	12.30	0.492	0.028	0.015(150)	0.12(1,200)	0.041(410)	0.053(530)	0.018(180)	0.015(150)	0.013(13)	0.0011(11)
2378DD	10.41	0.006	0.69	4.4(4.4)	1,100(1,100)	310(310)	300(300)	96(96)	6.3(6.3)	18(18)	1.6(1.6)
12378DD	10.96	0.022	0.39	8.4(8.4)	1,800(1,800)	940(940)	570(570)	320(320)	270(270)	150(150)	5.54(5.54)
123478DD	11.48	0.075	0.16	1.3(13)	93(930)	65(650)	33(330)	21(210)	20(200)	12(120)	0.60(5.99)
123678DD	11.50	0.078	0.16	2.0(20)	130(1,300)	88(880)	50(500)	31(310)	28(280)	18(180)	0.85(8.5)
123789DD	11.54	0.085	0.14	1.5(15)	150(1,500)	73(730)	47(470)	35(350)	30(300)	14(140)	0.067(0.67)
1234678DD	11.84	0.170	0.078	2.1(210)	33(3,300)	16(1,600)	14(1,400)	6.4(640)	6.3(630)	5.1(510)	0.33(33)
OCDD	12.45	0.697	0.020	0.15(1,500)	1.1(11,000)	0.12(1,200)	0.47(4,700)	0.056(560)	0.066(660)	0.13(1,300)	0.013(130)
ΣP_{2378}CDD/F				45(2,200)	4,800(41,000)	2,000(14,000)	1,500(15,000)	680(5,200)	510(4,600)	510(4,200)	12(250)

Values in parentheses are atmospheric gas + particle-phase concentrations in units of fg m^{-3}.

these exposure pathways is the geometric mean of daily doses over the period of exposure. For the purposes of preliminary exposure modeling, the individual resident/worker at each site was exposed 24 h per day, 7 days per week, over the 43 day period from 11th September through 25 October, 2003.

Cancer risks were estimated as LADD = ADD × [ED/LT] where LADD is the lifetime daily dose (pg-TEQ kg^{-1} d^{-1}), ADD is the average daily dose during the period of exposure (pg-TEQ kg^{-1} d^{-1}), ED is the exposure duration (d; equal to 43 days), and LT is the individual's lifetime (d; equal to 25,568 days). The cancer risk may then be calculated as Risk = LADD × SF where Risk is the upper bound incremental excess cancer risk that results from an exposure described by LADD, and SF is the upper bound cancer slope factor expressed in inverse units to LADD (kg d pg-TEQ^{-1}; equal to 1.56×10^{-4} kg d pg-TEQ^{-1}) [2].

For non-cancer risks, the United States Environmental Protection Agency (USEPA) recommends a concentration based, or body burden, approach where the contaminant concentration is expressed on a lipid weight basis. With the assumption that humans are 25% lipids by weight, whole weight concentrations can be converted to lipid weight through division by 0.25. Assumptions inherent in this approach are that P_{2378}CDD/Fs have a half-life of 7 years in humans, and that the current estimated P_{2378}CDD/F body burden in adults from the United States is 18 pg-TEQ per gram of body lipid [2]. A one-compartment, first order pharmacokinetic model can be used to estimate the body burden resulting from a defined intake regime. For an exposure of finite time, the non-steady state form of the model to predict an increment in body burden (IBB) from a constant intake dose is

$$\mathrm{IBB} = \frac{\mathrm{ADD}}{(k \times \mathrm{LW})} \times (1 - \mathrm{e}^{-kt})$$

where IBB is the increment of body burden on a lipid basis (pg g^{-1} LW), ADD is the average daily dose expressed not on a body weight basis (pg-TEQ d^{-1}), k is the first order dissipation rate constant (d^{-1}), LW is the weight of body lipids (g; equivalent to full body weight multiplied by 0.25), and t is the exposure duration (d). Values of 17,500 g for lipid weight (i.e. 70 kg × 0.25) and a k of 2.67×10^{-4} d^{-1} (equivalent to a 7.1 years half-life) are used for an average adult. The percent increase in body burden can then be calculated as [IBB/BK] × 100% where BK is the background body burden of 18 pg-TEQ per gram of body lipid [10]. This method explicitly accounts for how the limited duration exposure of residents and workers in NYC to PCDD/Fs produced by the WTC attacks affects an otherwise normal lifetime PCDD/F exposure.

3. Results and discussion

Sampling of exterior window films in New York City approximately 6 weeks after the terrorist attacks of 11 Septem-

Table 3
Estimated PCDD/F-TEQ based human exposure and assessed health risks for residents in New York City following the World Trade Center attacks

Exposure route	US average background intake	Brooklyn	NYC resident						
			Manhattan (Church & Warren)	Manhattan (Broadway & Worth)	Manhattan (Smithsonian NMAI)	Manhattan (Broadway & Canal)	Manhattan (City Hall Station)	Manhattan (16th Street & 3rd Avenue)	Manhattan (NYU-Grey Art Gallery)
Soil ingestion	0.47	3.7	330	160	110	55	44	42	1.0
Soil dermal contact	0.11	0.89	78	37	26	13	11	10	0.25
Freshwater fish and shellfish	13	13	13	13	13	13	13	13	13
Marine fish and shellfish	4.9	4.9	4.9	4.9	4.9	4.9	4.9	4.9	4.9
Inhalation	1.6	1.1	120	49	37	17	13	13	0.31
Milk	8.4	8.4	8.4	8.4	8.4	8.4	8.4	8.4	8.4
Dairy	9.8	9.8	9.8	9.8	9.8	9.8	9.8	9.8	9.8
Eggs	3.0	3.0	3.0	3.0	3.0	3.0	3.0	3.0	3.0
Beef	13	13	13	13	13	13	13	13	13
Pork	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5	3.5
Poultry	5.3	5.3	5.3	5.3	5.3	5.3	5.3	5.3	5.3
Vegetable fat	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6	1.6
Water	0.00077	0.00077	0.00077	0.00077	0.00077	0.00077	0.00077	0.00077	0.00077
Total	65	69	590	306	230	150	130	130	64
Percent of daily intake via soil	0.9	6.8	69.1	63.4	57.2	46.2	42.1	41.1	2.0
Percent of daily intake via food + water	96.6	91.6	10.6	20.4	26.9	42.2	48.1	48.9	97.5
Percent of daily intake via inhalation	2.5	1.6	20.3	16.1	15.9	11.5	9.8	10.0	0.5
ADD	0.92	0.97	8.4	4.4	3.3	2.1	1.9	1.8	0.91
LADD	0.0016	0.0016	0.014	0.0073	0.0056	0.0036	0.0031	0.0031	0.0015
Cancer risk	2.4×10^{-7}	2.6×10^{-7}	2.2×10^{-6}	1.1×10^{-6}	8.7×10^{-7}	5.5×10^{-7}	4.9×10^{-7}	4.8×10^{-7}	2.4×10^{-7}
Percent cancer risk increase	0.17	0.18	1.6	0.82	0.62	0.40	0.35	0.34	0.17
IBB	0.16	0.17	1.4	0.75	0.57	0.36	0.32	0.31	0.16
Percent IBB increase	0.88	0.93	8.0	4.2	3.2	2.0	1.8	1.7	0.87

ber 2001 revealed high concentrations of P_{2378}CDD/Fs close to the former site of the World Trade Center (Fig. 1 and Table 1). Exterior window film concentrations near the WTC site ranged up to 6600 pg-TEQ g^{-1}, in sharp contrast to background samples in mid-Manhattan and Brooklyn having concentrations of 75 and 21 pg-TEQ g^{-1}. The concentrations of all P_{2378}CDD/F congeners were greatly elevated in exterior window films near the WTC site, as discussed more extensively elsewhere [4], ranging up to 80,000 pg g^{-1} compared to background concentrations of 620–5700 pg g^{-1}. A model for reconstructing atmospheric contaminant concentrations from exterior building surface films was subsequently developed to estimate the total gas + particle-phase P_{2378}CDD/F concentrations in regions of lower Manhattan and Brooklyn near where sampling sites were located. Using this model, total gas + particle-phase atmospheric P_{2378}CDD/F concentrations exhibited a similar spatial pattern to that observed in the exterior window films, with high concentrations near the WTC site (up to 4800 fg-TEQ m^{-3} or 41,000 fg m^{-3}) declining to background levels of 12 fg-TEQ m^{-3} (or 250 fg m^{-3}) and 45 fg-TEQ m^{-3} (or 2200 fg m^{-3}) in mid-Manhattan and Brooklyn, respectively (Table 2). These reconstructed atmospheric P_{2378}CDD/F concentrations are in the range of values given by the USEPA monitoring stations near the WTC site (340–139,000 fg-TEQ m^{-3}) [2], appearing to lend credibility to the modeling approach. Thus, estimated atmospheric concentrations of P_{2378}CDD/Fs near the WTC site at the time of sampling 6 weeks after the building collapses were up to 2.6 orders of magnitude higher than background levels only a few kilometers away. A rudimentary human health risk assessment was then performed using the exterior window film and reconstructed gas + particle-phase atmospheric P_{2378}CDD/F concentrations to examine the potential cancer and non-cancer risks in regions impacted by the WTC contaminant plume from this limited duration contaminant exposure. Assuming a continuous (i.e. 24 h per day) exposure duration of 43 days from 11 September to 25 October 2001 (first of the 2 days of window film sampling) a percent cancer risk increase of 1.6% is estimated for residents closest to the WTC site (Table 3). In contrast, residents of mid-Manhattan and Brooklyn would only experience an estimated 0.17% increase in cancer risk from P_{2378}CDD/F exposure over this time period. In addition, residents nearest the WTC site would experience an estimated 8.0% increase in P_{2378}CDD/F body

burden from this initial 43 day exposure period, compared to increases of 0.87 and 0.88% for residents of mid-Manhattan and Brooklyn, respectively. Hence, the WTC attacks appear to have increased incremental cancer and non-cancer risks by up to one order of magnitude over normal background levels for those residents near the WTC site.

Several assumptions have been made in both reconstructing atmospheric P_{2378}CDD/F concentrations and in assessing exposure routes that may have led to either overall under- or over-estimates of actual risk in the current study. As an equilibrium partitioning model was used to reconstruct atmospheric concentrations, equilibrium between the atmosphere and the exterior window films had to be assumed. Previous work has shown such equilibrium to be achieved rather quickly (i.e. within a few days) [11]; thus, the equilibrium assumption appears to be reasonably valid for the purpose served. In addition, all exterior building surfaces near each site were assumed to have organic films with approximately the same P_{2378}CDD/F concentrations as were observed in the window films. In the event that windows were preferential "kinetic sinks" for atmospheric phase P_{2378}CDD/Fs, and nearby exterior surfaces (e.g. concrete, wood, plastic, metal, asphalt) did not accumulate the contaminants to the same degree, then local equilibrium would not be attained and a continuing redistribution of P_{2378}CDD/Fs would be occurring over time to equilibrate concentrations in all local surface films. With these assumptions regarding equilibrium between the atmosphere and window films, and between different exterior building surface films, atmospheric contaminant concentrations would be expected to be relatively constant at any given time within a local area and up to a height approximately equivalent to that the mean value for nearby buildings. Diurnal and daily temperature variations would also influence the atmospheric concentrations through a changing octanol-air partition coefficient (K_{oa}) value, which decreases with increasing temperature. However, given the other assumptions within the model, use of an overall daily average air temperature over the exposure period of interest would likely not introduce any unreasonable error. It must be noted that because of nocturnal cooling and daytime heating, the estimated nighttime and daytime atmospheric P_{2378}CDD/F concentrations would be expected to be higher and lower, respectively, than the mean values presented above.

Furthermore, the actual atmospheric particulate concentrations play a major role in estimating the particle-phase P_{2378}CDD/F concentrations shown in Table 2, with higher TSP concentrations resulting in correspondingly higher P_{2378}CDD/F concentrations as illustrated above in the relevant governing equations. The mean TSP concentration of 70 $\mu g\,m^{-3}$ used in the model is based on USEPA monitoring data throughout Manhattan during the period of interest [2], and is likely a conservative estimate because several "spikes" of TSP concentrations >100–200 $\mu g\,m^{-3}$ were observed in lower Manhattan in the first few days and weeks following the WTC attacks. Indeed, the TSP value of 70 $\mu g\,m^{-3}$ used in the present model is only moderately greater than typical ambient levels of 30 $\mu g\,m^{-3}$ at rural sites [8]. Hence, overall atmospheric P_{2378}CDD/F concentrations are likely underestimated as the model does not account for the extremely high TSP concentrations in the first few days after the WTC attacks.

An additional important assumption is the temporal pattern of P_{2378}CDD/Fs within the window films. The reconstructed atmospheric concentrations assume that the window films had unchanging P_{2378}CDD/F concentrations over the 6 weeks after the WTC attacks. Monitoring data from the USEPA showed clearly that atmospheric P_{2378}CDD/F concentrations declined by nearly an order of magnitude within several weeks of 11 September 2001. Assuming a short time for equilibrium to be established between the atmosphere and window films suggests that the window films acted as a P_{2378}CDD/F "sink" for the initial few weeks after the WTC attacks, and may have been acting as a P_{2378}CDD/F "source" around the time of sampling. Thus, the P_{2378}CDD/F concentrations in the window films could have been significantly higher during the initial portion of the 6 week exposure period modeled in the current study, and hence, the risk assessment presented here may in fact significantly underestimate the actual exposure to P_{2378}CDD/Fs by residents of lower Manhattan.

An additional assumption with regard to the preliminary risk assessment is that the P_{2378}CDD/Fs produced by the WTC attacks did not affect the intake quantities of P_{2378}CDD/Fs for nearby residents from food and water sources. Because there is no large-scale food production in lower Manhattan, most residents are likely acquiring the vast majority of their food as imports from regions which were not affected by the WTC attacks. However, those residents consuming "home-grown" vegetables in areas near the WTC attacks may have experienced elevated dietary intakes as P_{2378}CDD/Fs are well known to partition onto exterior vegetative surfaces. Thus, as Table 3 indicates, exposure intakes from food sources were set to equal the US average background intake values for residents of NYC. As well, NYC receives much of its water supply from upper New York State in the Catskill/Delaware and Croton watersheds (see http://www.nyc.gov/html/dep/html/wsmaps.html), an area not affected by contaminants from the WTC attacks, and thus P_{2378}CDD/F intake from drinking water was set to equal the US average background intake values for residents of New York City. Perhaps the most influential assumption within the risk assessment assumes that soil concentrations of P_{2378}CDD/Fs at each site equaled concentrations within the window films. Exterior window films and most soil types contain fairly substantial quantities of organic matter onto which hydrophobic contaminants may adsorb; however, it is not clear how the preferential surficial deposition of PCDD/Fs onto soil surfaces, and not to any appreciable depth absent any mixing mechanisms, would influence the calculations for soil ingestion and soil dermal absorption. For the present assessment, soil concentrations for the soil ingestion and soil dermal exposure routes were assumed to equal the window film

concentrations. As there is of yet no work reporting PCDD/F levels in soils from near the WTC site, it is unclear how using window films as a surrogate for soils influences the risk assessment results. Furthermore, polychlorinated biphenyls (PCBs) may contribute to TEQ exposure and should be considered in any risk assessment once the data becomes available. While total PCB concentrations in dusts and the atmosphere have been reported elsewhere [1,2], the congener specific concentrations needed to calculate PCB–TEQ exposure from the WTC attacks for residents of varying regions of NYC are not available.

Inhalation, soil ingestion, and soil dermal exposures to P_{2378}CDD/Fs are all increased in approximately equal amounts near the WTC site, each by up to 2.6 orders of magnitude over background levels in mid-Manhattan and Brooklyn. Whereas the average US resident receives ∼97% of his daily intake via food and (to a much lesser extent) water, with only 1.0% from soil ingestion or dermal exposure and 2.5% via inhalation, residents nearest the WTC are estimated to receive up to 70% of their daily intake via soil derived routes and up to 20% via inhalation during the time period under study. Thus, the PCDD/Fs produced from the WTC attacks are expected to shift exposure dominance from food to soil and inhalation intake routes for this short duration period. Background regions of Brooklyn and Manhattan have very similar intake patterns and quantities to the US average over the study period, with from 91.6 to 97.5% arriving via food + water, from 2.0 to 6.8% via soil contact and/or ingestion, and from 0.5 to 1.6% via inhalation.

The results presented above suggest that PCDD/Fs residing in exterior surface films and soils from lower Manhattan produced from the WTC attacks may pose a human health threat, with residents nearest the WTC site potentially experiencing P_{2378}CDD/F intakes via soil and inhalation up to 2.6 orders of magnitude higher than typical background levels measured in mid-Manhattan and Brooklyn during the time period under study. Furthermore, total exposures at sites located near the WTC are estimated to increase cancer risks by up to 1.6% and to increase total P_{2378}CDD/F body burdens by up to 8.0% over background levels. Continuing exposure to high P_{2378}CDD/F levels after the initial 6 week period following the WTC attacks, and the inability to fully account for the even higher P_{2378}CDD/F levels immediately following the building collapses, may have resulted in greater cancer and non-cancer health risks to residents and workers near the WTC site.

Acknowledgments

Thanks are extended to Matthew Lorber of the United States Environmental Protection Agency for helpful discussions and encouragement on the manuscript. The author is grateful to those residents and building owners of New York City who allowed our research group to sample at the site locations described above.

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