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Exposure assessment of residents living near a wood treatment plant ☆

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Abstract

We report the results of environmental sampling and modeling in a neighborhood adjacent to a wood processing plant. This plant used creosote and pentachlorophenol (PCP) to treat wood for over 70 years. Between 1999 and 2001, environmental samples were obtained to quantify the level of environmental contamination from the wood processing plant. Blood from 10 residents was measured for chlorinated dioxins and dibenzofurans. Soil sediment samples from drainage ditches and attic/dust samples from nearby residents' homes were tested for polychlorinated dioxins, furans, and polycyclic aromatic hydrocarbons (PAH). The dioxin congeners analysis of the 10 residents revealed elevated valued for octachlorodibenzo-*p*-dioxin and heptachlorodibenzo-*p*-dioxin compatible with PCP as the source. The levels of carcinogenic PAHs were higher than background levels and were similar to soil contamination on wood preserving sites. Wipe sampling in the kitchens of 11 homes revealed that 20 of the 33 samples were positive for octachlorinated dioxins with a mean value of 10.27 ng/m^2. The soil, ditch samples, and positive wipe samples from the homes indicate a possible ongoing route of exposure to the contaminants in the homes of these residents. Modeled air exposure estimated for the wood processing waste chemicals indicate some air exposure to combustion products. The estimated air levels for benzo(*a*)pyrene and tetrachlorodibenzodiozin in this neighborhood exceeded the recommended levels for these compounds in some states. The quantitative data presented suggest a significant contamination of a neighborhood by wood processing waste chemicals. These findings suggest the need for more stringent regulations on waste discharges from wood treatment plants.

Keywords: Creosote; Wood processing waste; Pentachlorophenol (PCP); Environmental pollution; Contamination

1. Introduction

Industrial sites, which may produce environmental pollution, can result in adverse effects in nearby residents (NIOSH, 1977). In this paper we present results of measurements of wood processing waste (WPW) chemical contaminants adjacent to a wood treatment plant including biomonitoring results from blood polychlorinated dioxins and furans. In a companion paper we report the results of a health study of this population. Residents in a small southern town filed a lawsuit against the wood treatment plant because of concerns about the effects of the contamination. These nearby neighbors complained that a strong odor of creosote was associated with the occurrence of symptoms including skin itch, headache, eye burning, sore throat, nausea, cough, and chest tightness. In addition, they reported oily ditch water and visible airborne particulates emanating from the wood treatment plant. On numerous occasions there were releases of oily, black specks that damaged automobile paint, requiring repainting. The wood treatment plant paid the repainting expense. Several drainage ditches flowed from the factory into the neighborhood. The company discharged WPW into the drainage ditches. During rain storms the ditches frequently overflowed, carrying WPW into the yards of the neighbors. Flooding of the neighborhood occurred on numerous occasions during the years.

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Children played in the ditch water during the summer. Some residents used scrap wood from the plant for home fireplace wood. A large fire in 1974 caused an acute exposure to levels of chemically contaminated smoke and run-off water, adding to the neighborhood contamination.

The plant operated from 1929 to the time of the writing of this article using coal-derived creosote continuously and pentachlorophenol (PCP) from the 1950s until 1974. Heat and pressure were applied to facilitate penetration of the creosote and PCP into the wood. The creosote was dehydrated on a regular basis to keep the moisture content below 3%. The dehydrating process required additional heating of the creosote, which released more vapor into the air. Creosote is a complex mixture containing a large percentage of polycyclic aromatic hydrocarbons (PAHs). Creosote constituents are numerous, but naphthalene and alkyl derivatives are the main components. Creosote contains significant amounts of aromatic hydrocarbons, including benzene, toluene, xylene, trimethylbenzene, methyl ethyl benzenes, styrene, phenol, and methyl styrene. The polycyclic aromatic hydrocarbons include benzo(*a*)pyrene, naphthalene, methyl naphthalene, dimethyl naphthalene, ethyl naphthalene, acenaphthene, fluorene, dibenzothiophene, acenaphthylene, benzocalpyrene, benzo(*ghi*)perylene, anthracene, pyrene, penanthrene, chrysene, benzo(*e*)pyrene, dibenzo(*ah*)anthracene, benzo(*k*)fluoranthrene, and benzo(*a*)fluorene. Other related compounds in creosote include benzofuran, dibenzofuran, benzonitrile, methyl benzonitrile, benzothiophenes, cresols, indenes, methyl indenes, xylenols, quinoline, isoquinoline, and diphenyl (Benedetti et al., 2001). PCP is contaminated with polychlorinated aromatic hydrocarbons (PCAHs) (i.e., chlorinated dioxin and furans) (ATSDR, 1999a). High pressure and heat were applied to facilitate penetration of the PCP into the wood, which potentially increased the formation of dioxins. Discharge of contaminants into the air, which occurred from normal operations, is believed to have created low-level air exposure on a daily basis. Unusual events such as a fire at the wood treatment plant and floods over the years have caused transient elevated levels of chemical contamination.

We report here a representative sample of the measurements of WPW chemical contamination in the neighborhood adjacent to the wood treatment plant. Exposure pathways for the residents included air, soil, and surface water contaminated with WPW, including chlorinated dioxins and furans. Quantitative data were obtained on human blood, drainage ditch sediment, and yard soil levels for polychlorinated dioxin and furans. Semiquantitative wipe samples from kitchen countertops and baseboards were obtained and analyzed for polychlorinated dioxin and furans by the wood treatment plant management. Measurements were performed on ditch sediment, soil, and house dust for polycyclic aromatic hydrocarbons and petroleum hydrocarbons. Air exposure estimates for some of the residents' homes were modeled for PAHs, polychlorinated dioxins, and creosote volatile organic chemicals (VOCs), and particulates on nine of the homes near the wood treatment plant were also analyzed.

2. Materials and methods

Between 1999 and 2001 a variety of environmental samples were obtained to assess the presence and quantify the level of contamination from the wood processing plant. Biomonitoring studies were carried out on 10 African American adults who were, nearby residents of the plant and who were chosen at random from the initial study cohort of 1269 nearby residents based upon their having lived in the neighborhood for over 25 years. The subjects of the study were fully informed and signed an informed consent to participate in the study. Whole blood was collected in November 2000 in chemically cleaned glass containers prepared by the analytic laboratory with anticoagulant and also with Teflon tops containing no paper products for the nearby residents and for a Dallas, Texas comparison group. Blood was frozen and sent frozen on dry ice to Hamburg, Germany for polychlorinated dioxin and furan analysis at the ERGO Laboratory. Analysis was performed by high-resolution gas chromatography/high-resolution mass spectrometry by methods previously described (Paepke et al., 1989). Two sediment samples were collected simultaneously from the ditch adjacent to Mill Street (see Fig. 1) and were also performed by the ERGO Laboratories.

Some sediment, soil, and all house/attic dust samples were collected by Environmental Technologies Inc. Laboratories (Magnolia, TX) and analyzed in accordance with Environmental Protection Agency (EPA) and American Society of Testing Materials (ASTM) methods.

Surface soil samples were collected using hand trowels and augers. Subsurface sampling was conducted with the Geoprobe soil sampling system, and settled dust samples were collected using a microvacuum system and a 0.8-μm cellulose filter as described in ASTM methods #E 1973-99 and #D 5438-94 and EPA method #747-R-95-01. All samples were preserved as required and express-shipped to the analytical laboratory.

The house dust analysis was of total settled dust. Particles of up to 100 μm in size can enter the human airway. Inhalable particles are considered when they can affect health, regardless of their size. Even though fume-sized particles can enter the body though the lungs, the larger particles are swallowed. Thoracic particulates are generally 10 μm or smaller. Respirable particulates are

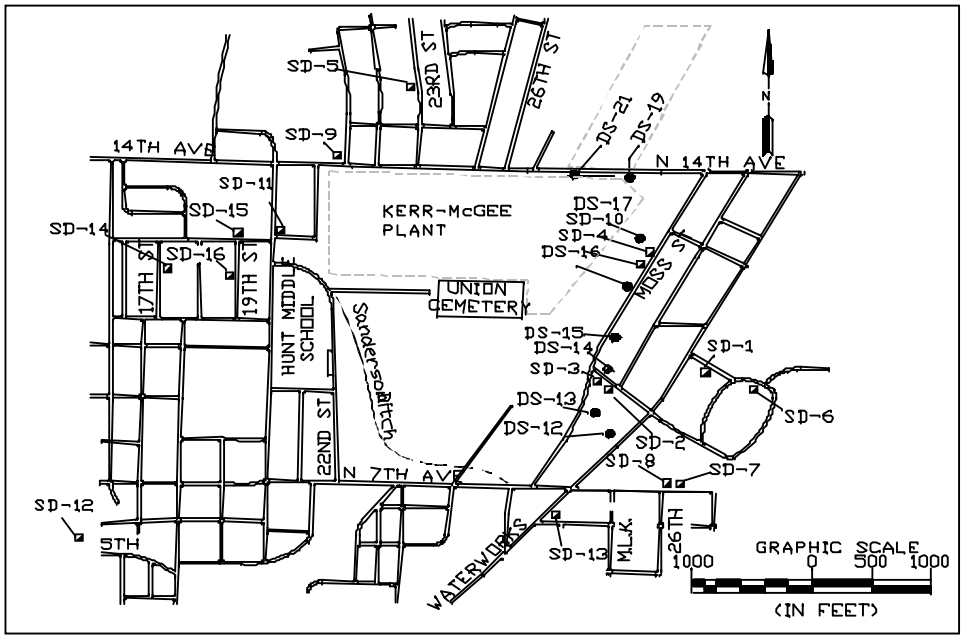

Fig. 1. Map of neighborhood with numbered sampling sites.

generally smaller than 4 μm. Therefore, the filter of 0.8 μm was appropriate for swallowable (thoracic) and respirable dust.

The soil samples were analyzed by Xenco Laboratories (Houston, TX). The settled dust samples were split and analyzed by both standard analytical methods.

Other soil and ditch sediment samples were collected at the surface and to 2.5 ft below grade surface depths by 3TM International (Houston, TX). The DS number denotes the collection locations of the ditch and nearby soil samples shown in Fig. 1. The letter A identifies samples from the middle of the ditch, whereas soil samples 10 ft east of the ditch are identified by the letter B. The samples were collected into laboratory-supplied collection vessels and sent to the laboratory on ice. Xenco Laboratories in analyzed the PAHs utilizing standard laboratory techniques. The SD number denotes the collection locations of the dust wipe samples from a template of 2 in. × 12 in. The kitchen surface wipe samples were collected by consultants to the plant management and analyzed by Triangle Laboratories (Research Triangle Park, NC). The method used was believed to be an EPA-approved method.

Devraj Sharma of Principe Mathematica (Lakewood, CO) performed the air modeling. A systematic mathematical modeling analysis of air dispersion in the vicinity of Kerr-McGee's (KMC's)wood preserving facility site was conducted. This analysis was based upon a historical reconstruction of the facility operations and used estimated air emissions from significant sources during the time period from January 1929 through December 1999. This included 23 point sources and 10 area sources at different elevations. The hourly emission rates from each of these sources estimated for the seven-decade time period were modeled. The sources included the consequences of a fire which engulfed the KMC facility units for approximately 8 h on September 30, 1974. Influences of the uncertainties in available data were evaluated. The results of air dispersion modeling analysis were then utilized in evaluating nearby residents' exposures.

The computer program utilized for air dispersion modeling analysis of the KMC site is the US EPA's recommended code for analyzing industrial source complexes, **ISCST3**. The input data sets for this program, the receptor grid, the source representations and meteorological data used have all been selected, prepared, and utilized for modeling in accordance with the EPA's recommended procedures. In this respect, wind data from the local US Air Force Base were supplemented with upper air data and climatic information collected at locations nearby. Methods of filling relatively small gaps in available measurement data were devised and tested.

The receptor grid used for air dispersion modeling consisted of two parts: an individual receptor including approximately 27 locations and a polar coordinate system comprising more than 5000 grid nodes. Model calculations at all these receptor locations were saved and used to calculate exposures at each appropriate location and for each individual's appropriate exposure time span. The grid information was used to prepare contour diagrams of breathing-level concentration

plumes for a number of chemical species, including combustion products. The individual receptor information was used to prepare a set of tables that present the calculated air doses, i.e., multiplications of exposure concentrations and exposure duration, for each individual exposure location under several different scenarios. Each of these scenarios employed conservative approximations in order to overcome limitations in the available data; hence, the calculated doses represent lower limits of historical exposures. The cooling tower, which was identified as a major source of air contamination, was not modeled because of the lack of data from the plant management. Plant management first denied that the data ever existed; later the plant claimed that the data had been destroyed.

3. Results

Data from the resident's blood samples and two neighborhood drainage ditch's sediments are shown in Table 1. The dioxin congeners analyses in 10 resident sediment samples both reveal elevations of higher chlorinated dioxins, especially hepta- and octachlorinated dioxins in some persons. A similar pattern is seen in the blood of residents. A similar pattern is also seen in Table 4, which depicts environmental PCAH samples analyzed by a different laboratory. The pattern reveals a higher proportion of the higher chlorinated dioxin congeners. Presumably, chlorinated dibenzo-furans with shorter half-lives of elimination may have been excreted during the 26 years following cessation of PCP use prior to blood sampling.

Table 1 further compares the PCP wood processing ditch sediment samples with the recently published results of ditch sediment samples in Vietnam (Schecter et al., 2001). The Hanoi control sample is from northern Vietnam and the Bien Hung Lake 2 sample is close to a former air base.

Table 2 compares the PCP wood processing plant neighbors with the Dallas general population blood levels and with previous tissue levels in PCP-exposed German, Canadian, and Chinese workers (Paepke et al., 1992; Ryan et al., 1987; Schecter et al., 1996, 1994). Elevation of octachlorodibenzo-*p*-dioxin (OCDD) is the predominant congener noted in the PCP-exposed wood treatment plant neighbors. The mean level for OCDD is 2.6 times, that for heptachlorodibenzo-*p*-dioxin (HpCDD) is 1.3 times, and that for hexachlorodibenzo-*p*-dioxin (HxCDD) is 1.6 times higher in the wood processing plant neighbors than in the general population. As expected, the Canadian worker who died of acute PCP poisoning reported by Ryan et al. (1987) had quite high levels of those congeners found in PCP. The German workers reported by Paepke et al. (1992) had higher levels also presumably due to more current exposure than that of the persons reported here. In rural China, the levels of dioxins in general in people are lower than in more industrialized countries, and PCP exposure may not have been as high as in the other cohorts presented.

Tables 3 and 4 report the results of sampling for dioxins along the drainage ditch, which parallels North 14th Avenue and then turns down Moss Street (Fig. 1). Table 3 also compares total dioxin/furan/PCB levels, expressed as toxicity equivalent factors (TEQs), to illustrate how our study group compared to another control group and another exposed population (Hanoi Control Sample and Bein Hung Lake). The levels are markedly elevated and again reflect the pattern expected from PCP contamination, namely higher chlorinated congeners, especially OCDD. Total dioxin/furan TEQs are within ranges seen in another dioxin-contaminated site, Times Beach, Missouri, where tetrachlorodibenzo-*p*-dioxin (TCDD) levels were in the 1 ppb range. Those levels of dioxin exerted a toxic effect in laboratory animal systems (Lucier et al., 1986).

Wipe sampling in the kitchen of 11 homes revealed that 20 samples of 33 were positive for octachlorinated dioxin with a mean value of 10.27 ng/m^2. The detection limit ranged from 0.517 to 2.583 ng/m^2. The two kitchen wipe samples with the highest values are shown in Table 5 which reports the results for selected congeners of chlorinated dioxins and furans. The wood processing company performed the wipe sampling. Unfortunately, there was no estimate of the area from which the wipe sample was taken so quantification is not precise. The results are presented here because they are consistent with PCP-derived contamination entering the home and persisting. The kitchen contamination suggests an ongoing route of exposure for these residents.

Table 6 reveals the levels in ppm of carcinogenic PAHs (cPAHs) in ditch sediment samples. Table 7 shows the carcinogenic PAHs toxicity equivalent factors (TEFs) for eight sampling sites. The cPAHs are significantly above background levels and similar to the soil contamination on wood preserving sites (Tables 5.3 and 5.4 in ATSDR, 1995).

Table 8 reports the modeled air exposure estimates for naphthalene, dibenzofuran, carcinogenic benzo(*a*)pyrene (B(*a*)P) TEQs, creosote VOCs and particulates. Table 9 report modeled air exposure estimates for chlortinated dioxin/furans TEQs.

Table 10 reports the house/attic dust results for petroleum hydrocarbons (PHC). PHC is similar to total petroleum hydrocarbons (TPH), but does not use freon in the analysis process. PHC was estimated from chromatographs EPA Method 8270, the background level expected in house dust is not established.

Table 1
Dibenzodioxin, dibenzofuran and PCB levels in residents, neighborhood drainage ditches, and Vietnamese drainage ditches

Congeners	TEQ equivalence	Resident 1	Resident 2	Resident 3	Resident 4	Resident 5	Resident 6	Resident 7	Resident 8	Resident 9	Resident 10	Drainage ditch sediment 1	Drainage ditch sediment 2	Hanoi control sample[a]	Bien Hung lake 2A[b]
PCDDs															
2,3,7,8-TCDD	1	5.9	4.2	4.1	5.5	3.1	7.4	3.1	3.2	1.9	2.5	7	0.7	ND	177
1,2,3,7,8-PeCDD	0.5	10.5	12.5	12	12.2	10.3	6.8	8.3	8.4	6.8	5.1	13	0.6	0.9	4.2
1,2,3,4,7,8-HxCDD	0.1	14.2	15.1	12.7	10.3	9.3	4.9	8.1	5.9	5.9	4	87	4.1	0.6	4
1,23,6,7,8-HxCDD	0.1	72	69.3	79.1	55.2	68.4	31.6	37.4	51.1	52.9	25	1202	31.7	1.7	7.5
1,2,3,7,8,9-HxCDD	0.1	12.8	10.5	7.2	11.7	4.7	5.1	6.1	5.6	3.9	3.4	159	9.7	1	7.1
1,2,3,4,6,7,8-HpCDD	0.01	155.1	99.3	46.5	95.6	58.3	54.8	81.3	28.8	34.3	28.3	48500	2364	25.7	160
OCDD	0.001	2152.7	1038	744.2	1015.7	644.3	711.7	822.9	505.1	679.5	277.5	931200	302097	373	1610
PCDFs															
2,3,7,8-TCDF	0.1	0.8	0.8	0.75	1.05	1.1	0.85	5	0.7	0.7	0.75	17	0.5	9.8	62.3
1,2,3,7,8-PeCDF	0.05	0.6	ND	ND	ND	1	ND	ND	ND	ND	5	84	0.5	5.7	3
2,3,4,7,8-PeCDF	0.5	5.1	6.3	5.8	3.8	7.2	3.1	4.2	3.8	3.9	ND	98	0.9	4.3	1.3
1,2,3,4,7,8-HxCDF	0.1	12.9	9.2	11.5	10	15.9	5.1	6.8	6.3	9.3	5	679	14.3	9.7	2
1,2,3,6,7,8-HxCDF	0.1	7.7	6.9	6.6	5.1	10.2	3.4	4.3	4	5	3.4	288	2	3.4	4.4
1,2,3,7,8,9-HxCDF	0.1	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	71	0.6	1.2	1
2,3,4,6,7,8-HxCDF	0.1	3.5	6.9	9.1	4.5	10.1	2.4	4.1	4.3	3.3	3	NA	NA	1.8	2.3
1,2,3,4,6,7,8-HpCDF	0.01	12.3	8.9	12.9	19.5	14.9	7.4	6.3	6.7	16.3	5.9	11098	173.4	8.2	13.2
1,2,3,4,7,8,9-HpCDF	0.01	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1402	15.9	2.7	2
OCDF	0.001	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	76280	918.1	23.5	42.3
Coplanar PCBs															
3′44′TCB 77	0.0005	33	NA	NA	NA	31	NA	NA	30	35.5	28.5	1228	28	NA	NA
3′44′5-PCB 126	0.1	69	40	39	70	36	29	67	26	28	23	7	NA	NA	NA
3′44′55′-HCB 169	0.01	19	36	49	15	42	11	21	19	28	13	1	NA	NA	NA
Total PCDDs		2423.2	1248.9	905.8	1206.2	798.4	822.3	967.2	608.1	785.2	345.8	981168	304508	403	1970
Total PCDFs		45.4	41.5	49.2	46.5	62.9	24.8	33.2	28.3	41	25.6	90158	1128	70.3	134
Coplanar PCBs		121.0	76.0	88.0	85.0	109.0	40.0	88.0	75.0	91.5	64.5	1236	28	NA	NA
Total PCDD/furans and PCB		2589.6	1366.4	1043.0	1337.7	970.3	887.1	1088.4	711.4	917.7	435.9	1072562	305664		
Total TEQ		37.1	32.0	31.4	32.6	29.3	22.0	25.1	20.8	19.6	12.8	1936	336	6.5	193

Results are in ng/kg (ppt).
ND, not detected; NA, not available.
[a]Hanoi sample is the northern Vietnam control sample (Schecter et al., 2001).
[b]Bien Hung Lake 2 sample is close to the former air base (Schecter et al., 2001).

Table 2
Comparisons of dioxin congener profiles in samples of pentachlorophenol-exposed workers and the general US population

Congeners	TEQ[a] equivalence	PCP wood treatment residents				General population (Dallas; Schecter, 2000)	PCP poisoning (Ryan et al., 1987)	PCP workers (Germany) (Paepke, 1992)	PCP direct contact, (China)
		$n = 10$				$n = 200$	$n = 1$	$n = 20$	$n = 26$
PCDDs		Mean	Median	Minimum	Maximum				
2,3,7,8-TCDD	1	4.1	3.7	1.9	7.4	2.6	15	4.5	3
1,2,3,7,8-PeCDD	0.5	9.3	9.4	5.1	12.5	6.3	32	28.3	7.2
1,2,3,4,7,8-HxCDD	0.1	9	8.7	4	15.1	6.4		47.9	22.1
1,23,6,7,8-HxCDD	0.1	54.2	54.1	25	79.1	32.8	321	240.6	9
1,2,3,7,8,9-HxCDD	0.1	7.1	5.9	3.4	12.8	4.9	159	110.3	2.9
1,2,3,4,6,7,8-HpCDD	0.01	68.2	56.6	28.3	155.1	49.2	7.2	2514.1	24.1
OCDD	0.001	859	727	277.5	2125.7	330.4	59300	33192	1148
PCDFs									
2,3,7,8-TCDF	0.1	1.3	0.8	0.7	5	1	ND{2.0}	2.6	1.5
1,2,3,7,8-PeCDF	0.05	2.2	1	0.6	5	0.05	NA	3.5	2.4
2,3,4,7,8-PeCDF	0.5	4.8	4.2	3.1	7.2	4.5	23	48.6	ND{1.0}
1,2,3,4,7,8-HxCDF	0.1	9.2	9.3	5	15.9	5.9		69.1	16.4
1,2,3,6,7,8-HxCDF	0.1	5.7	5.1	3.4	10.2	3.5	80	63.7	2.3
1,2,3,7,8,9-HxCDF	0.1	ND	ND			0.5	NA	1.2	ND {1.0}
2,3,4,6,7,8-HxCDF	0.1	5.1	4.2	2.4	10.1	1.5	17	12.6	1.2
1,2,3,4,6,7,8-HpCDF	0.01	11.1	10.6	5.9	19.5	6.7	2770	238.3	4.9
1,2,3,4,7,8,9-HpCDF	0.01	ND	ND			0.8	1860	2.8	ND {1.6}
OCDF	0.001	2.5	2.5	2.5	2.5	2.5	7060	8.7	5.2
Coplanar PCBs									
33′44′TCB 77	0.0005	31.6	31	28.5	35.5	NA			
33′44′5-PCB 126	0.1	42.7	37.5	23	70	34.5			
33′44′55′-HCB 169	0.01	25.3	20	11	49	24.5			
Total PCDDs		1010.9	865.4			536.7	66847	36137	1216
Total PCDFs		41.9	37.7			25.4	11810	450.3	35.7
Coplanar PCBs		99.6	88.5			59.0			
Total PCDD/Fs and PCB		1152.4	991.6			621.0	78657.0	36587.0	1252.0
Total TEQ		26.6	24.7			18.2	283.1	158.6	14.9

Results are in ng/kg (ppt).
ND, not detected [limit of detection]; NA, not available.
[a]Toxicity Equivalents.

4. Discussion

Ten blood samples from residents drawn and analyzed in the year 2000 found elevations in some persons of higher chlorinated dioxins. A comparison with PCP workers reveals levels and patterns in these residents that are similar to those of exposed workers (Schecter et al., 1996, 1994). The elevated higher chlorinated dioxins, especially OCDD, are consistent with previous exposure to PCP, which is characterized by dioxin contamination with the higher chlorinated PCDD/PCDF congeners (Paepke et al., 1992). A pooled blood sample from a general population of 200 Dallas,

Table 3
Total polychlorinated dioxins/furans in drainage ditch sediment samples at depths of 0–12 in

Sample site on map (Fig. 1)	Total polychlorinated dioxin/furans (ppt)	Total TCDD equivalents (TEQs)
DS-12A	615	1.04
DS-13A	439	0.44
DS-14A	ND	ND
DS-15A	55,537	66.77
DS-15B	377,377	2,593.17
DS-16A	1,393,786	9,854.16
DS-16B	4,552,708	10,096.9
DS-17A	326,456	1,214.19
DS-19A	2,739,054	4,579.12
DS-21A	221,014	311.51
Hanoi[a] control sample	473	6.5
Bein Hung Lake 2A[b]	2104	193

Results in ng/kg (ppt).
ND, none detected
[a] Hanoi sample is the northern Vietnam control sample (Schecter et al., 2001).
[b] Bein Hung Lake 2 sample is close to a former air base (Schecter et al., 2001).

Table 5
Dioxins from wipe samples on kitchen surfaces in homes

ng/kg (ppt)	Kitchen on Moss Street	Kitchen on 23rd Street North
TCDD	0.02	0.04
12378 PCDD		0.20
123478 HxCDD		0.22
123678 HxCDD		0.25
123789 HxCDD		0.23
1234678 HpCDD	0.1	0.25
OCDD	0.59	0.50
TCDF		0.03
23478 PCDF		0.21
12378 PCDF		0.22
123478 HxCDF	0.01	0.22
123678 HxCDF		0.23
123789 HxCDF		0.21
234678 HxCDF		0.23
1234678 HpCDF	0.03	0.24
1234789 HpCDF		0.20
OCDF	0.05	0.45
Total TEQs (ppt)	0.02385	0.37845

Surface areas sampled are not specified. Twenty out of 33 kitchen wipe samples were positive for dioxins. Values are in ng/kg (ppt).

Table 4
Concentrations of polychlorinated dioxin congeners in three sites

	DS–16A	TEQs	DS–16B	TEQs	DS–17a	TEQs
TCDD	991	991			86	86
12378 PCDD	3841	1720			346	346
123478 HxCDD	4987	498			427	42
123678 HxCDD	7337	733	6562	656	706	70
123789 HxCDD	6921	692			667	66
1234678 HpCDD	12,038	120	285,445	2854	11,298	112
OCDD	1,272,616	1272	4,034,652	4034	302,075	302
Total dioxin TEQs		6026		7544		1024
TCDF	996	99			92	9.2
23478 PCDF					425	212
123478 HxCDF	4951	495	9988	998		
123678 HxCDF	5029	502	1468	146		
123789 HxCDF	3795	379	7484	748	227	22
234678 HxCDF					770	77
1234678 HpCDF	17,626	176	46,703	467	2551	25.51
1234789 HpCDF	4481	44	3325	33	615	6.15
OCDF	48,380	48	157,077	157	6164	6.16
Total furan TEQs		1651		2359		136.82
Total dioxin/furan ppt TEQs		7677		9903		1160

Results in ng/kg (ppt).

Table 6
Carcinogenic cPAHs and total PAHs in neighborhood ditch samples

Sample site on (Fig. 1)	Benzo(*a*)pyrene	Benzo(*a*)anthracene	Benzo(*b*)fluoranthene	Chrysene	Indeno(123-cd)pyrene	Total PAH[a]
DS-12A	ND	1.65	3.7	1.85	0.427	33.2
DS-13	0.963	3.0	4.1	2.85	0.498	38.2
DS-14	12.4	21.5	20.4	26.7	6.71	405
DS-14A	1.11	1.19	2.04	1.72	BRL	444
DS-15B	3.65	3.13	3.18	2.58	0.37	110
DS-16	24.5	70	33	61.5	10.5	1356
DS-16A	6.66	9.92	11.3	BRL	2.14	1355
DS-17A	1.9	3.81	2.56	BRL	BRL	245
DS-19	6.82	16.70	10.4	15.3	2.7	659
DS-21A	0.295	1.38	4.52	1.91	0.87	31.1
1009 Moss Street house dust sample	3.26	2.98	14.1	7.06	2.93	
1214 Moss Street house dust sample	0.099	0.11	0.22	0.11	0.094	
Background rural	0.002–1.3	0.005–0.02	0.02–0.03	0.038	0.01–0.015	
Wood preserving site surface soil	28	12	38	38	10	
MS DEQ target remediation goals	0.0875	0.875	0.875	87.5	0.875	
US EPA target remediation goals	0.062	0.62	0.62	62	0.62	

Comparisons are to ATSDR published background rural soil and contaminated site values (ATSDR, 1995). Results are in mg/kg (ppm).
[a] Includes noncarcinogenic and carcinogenic PAHs.

Table 7
Totals for carcinogenic PAHs in mg/kg with total toxicity equivalence factor

Sample site on (Fig. 1)	Benzo(*a*)pyrene	Benzo (*a*)anthracene	Benzo (*b*)fluoranthene	Chrysene	Indeno (123-cd)pyrene	Total TEF
DS-12A	0	0.165	0.37	0.0018	0.042	0.5795
DS-13A	0.963	0.300	0.41	0.0028	0.042	1.7186
DS-14A	1.11	2.150	3.990	0.0267	0.670	7.9467
DS-15A	3.65	0.313	0.318	0.0025	0.037	4.3205
DS-16A	6.66	7.000	6.790	0.0615	1.050	21.5615
DS-17A	1.9	0.947	0.890	0.0082	0.130	3.8752
DS-19A	1.43	1.670	1.790	0.0150	0.270	5.1750
DS-21A	0.295	0.138	0.452	0.0019	0.087	0.9739
Toxicity equivalence Factor (USEPA, 2000)	1.0	0.1	0.1	0.001	0.1	

Table 8
Modeled air exposure estimates for naphthalene, dibenzofuran, carcinogenic B(*a*)P toxicity equivalent factors, creosote VOCs, and particulates

Exposure period		Maximum annual average air exposure for 10 residents (ng/m^3)—(High range—MPRH)[a]				
Start	End	Naphthalene	Dibenzofuran	Benzo(*a*)pyrene TEF	Creosote (VOC)	Particulate matter
07/1952	06/1979	30.57	5.92	0.07	72.78	0.79
01/1990	10/1995	70.14	13.59	0.18	167.00	0.44
12/1990	10/1995	68.22	13.22	0.17	162.42	0.44
07/1978	12/1999	67.18	13.02	0.17	159.96	0.44
07/1969	12/1999	41.41	8.03	0.10	98.59	0.65
07/1983	06/1985	68.58	13.29	0.17	163.29	0.47
07/1985	12/1999	46.12	8.94	0.12	109.82	0.40
07/1990	12/1999	33.29	6.45	0.08	79.26	0.36
07/1982	06/1995	30.56	5.92	0.08	72.76	0.38
07/1976	06/1981	35.47	6.87	0.09	84.46	0.56

[a] Most Probable Range High.

Texas residents collected at about the same time documents current congener levels and dioxin levels in the general population of this region of the country. Levels in the current Dallas blood show lower dioxin levels than previously reported (Schecter, 1994; Schecter et al., 1996). The blood polychlorinated biphenyls

Table 9
Modeled air exposure estimates for chlorinated dioxin/furans TEQs[a]

Exposure period		Total air exposure (ng/m3)(years) (high range–MPRH)		
Start	End	Polychlorinated dibenzo-*p*-dioxins (CDD) TEQ	Polychlorinated dibenzo-*p*-furans (CDF) TEQ	2,3,7,8-Tetrachloro-dibenzo-*p*-dioxin (TCDD)
07/1952	06/1979	1.536E-04	3.711E-04	4.606E-07
07/1979	12/1999	2.242E-05	5.416E-05	6.722E-08
03/1960	06/1989	4.456E-05	1.077E-04	1.336E-07
07/1959	06/1966	4.142E-05	1.001E-04	1.242E-07
07/1978	12/1999	3.072E-05	7.422E-05	9.211E-08
07/1969	12/1999	7.787E-05	1.881E-04	2.335E-07
07/1976	06/1983	3.037E-05	7.339E-05	9.108E-08
07/1962	06/1966	3.199E-05	7.729E-05	9.592E-08
07/1968	06/1978	3.332E-05	8.050E-05	9.991E-08
07/1967	06/1971	2.302E-05	5.562E-05	6.903E-08

[a] TEQ—Toxicity Equivalents.

Table 10
Petroleum hydrocarbons house/attic dust levels in 16 homes near the wood processing plant in 2001

Sample site on (Fig. 1)	Address	Petroleum hydrocarbons[a] (mg/kg)
SD-4	1025 Moss Street	2230
SD-3	2304 Marvin Circle	14,600
SD-2	2306 Marvin Circle	2210
SD-6	267 Byrnes Circle	2610
SD-1	289 Byrnes Circle	4580
SD-5	1602, 23rd Street, North	1520
SD-7	2605, 7th Avenue North	2580
SD-8	2609-2619, 7th Avenue North	620
SD-9	1403, 21st Street, North	18,300
SD-10	1009 Moss Street	226,000
SD-11	1300, 20th Street, North	27,500
SD-12	514, 15th Street, North	6130
SD-13	2325 6th Avenue	9110
SD-14	1204, 17th Street, North	44,000
SD-15	1807, 12th Avenue North	46,400
SD-16	1213, 19th Street, North	10,900
	Average	26,206

[a] PHC—similar to TPH, estimated from chromatographs, EPA method 8270.

(PCBs) of the study group were not elevated above those of the Dallas reference group, which is expected, since PCB exposure in the wood processing neighbors would be similar to that of a normal background. After cessation of PCP use in 1974, its contaminants still remain in the neighborhood in the sediment of drainage ditches and in the soil at some locations. As a result, reexposure is possible, especially in children who are still playing in the ditches and on the adjacent soil. Dust from the contaminated soil can also be resuspended, giving rise to respirable dust and settling dust on surfaces in homes. Thus, ongoing exposure to the contaminants from prior PCP use is possible.

The finding of chlorinated dioxins from PCP indicates a pattern that is consistent with the neighbors of the wood processing plant having higher levels from neighborhood contamination than background. Whether the current blood levels reflect only prior exposure or a combination of prior exposure and ongoing exposure cannot be determined from the available information. It is probable that the levels of the dioxins in blood would have been higher in the decades during which the PCP was being used.

The PCP characteristic pattern of dioxins in the blood serves as a surrogate marker for the other contaminants. The PAHs are transient in the body, making dioxins the only biomarker of exposure available. One can assume that the contaminated soil and ditch sediment serve as a pathway of exposure for the residents living in this neighborhood. The quantitative internal dose that has occurred from this exposure to all the contaminants cannot be determined with assurance, but is likely to be significant, given the high levels of PHC in the attic dust. Since the house dust has high levels of petroleum hydrocarbon contaminants, it is probable that the inhabitants of those homes would be inhaling and ingesting carcinogenic and toxic chemicals in significant concentrations.

The residents report creosote odor on a daily basis even at the time of writing, although the strong odors from the plant are reported to occur less often. The strongest odors occur at night after 9 PM. Naphthalene is the largest creosote air constituent in wood treating plants (Heikkila et al., 1987). Heikkila and co-workers studied a creosote wood treatment plant and found naphthalene to constitute 2.2 mg/m^3 (0.42 ppm) of the total 3.7 mg/m^3 of airborne creosote vapor in the work area. This level of creosote exposure caused neurological and irritant symptoms in the exposed workers. The symptoms experienced by the near neighbors are similar to those in the exposed workers reported by Heikkila.

Air levels of creosote vapor sufficient to cause symptoms are also accompanied by significant carcinogenic PAH exposure (Heikkila et al., 1987). The odor threshold for naphthalene is 0.437 mg/m3 (0.084 ppm), with a standard error of 9.88 mg/m^3 (1.9 ppm) (Amoore and Hautala, 1983). Thus, the presence of a detectable odor of naphthalene associated with symptoms would indicate that significant airborne exposure to the creosote vapor is occurring and this at levels ranging from 0.437 to 2.2 mg/m^3. Tables 8 and 9 provide results of air modeling of the vapors and particulates from the plant calculated as the concentration that would be present at the mailbox of the studied home. The results provide a quantitative estimate of exposures over time. The modeled exposures provide values consistent with annual exposures in range of nanograms per cubic meter. Such values would not result in strong odors or in symptoms. The reason that the modeled data are less lower than expected than the odors and symptoms reported by the residents is that the real-world conditions of plant operation differ from the assumptions of the model. The air modeling values are conservative and probably underestimate this route of exposure, since many activities of the plant, plus uncontrolled releases, cannot be considered in the calculations.

The air modeling reveals that there are annual exposures to a variety of airborne contaminants. The air modeling suggests doses of individual exposure, which are very conservative. It does not take into account the residents' exposure from events such as the dehydrating of the creosote. This is a process in which the creosote is heated to reduce the water content. This dehydrating process is carried out at night, at which time the residents note a strong odor. If the dehydrating process were taken into account, residents who lived close to the plant (closer than 1 mile) would have an estimated 15–50% higher exposure. Residents who lived further away (1 mile or further) would have an estimated 0–20% higher exposure. Even so, the annual doses of PAHs and dioxins are higher than some states' regulatory levels for inhalation and ingestion of these compounds. Michigan, for example, has an annual acceptable air exposure for dioxin (TCDD) of 2.3E–5 ng/m^3 (ATSDR, 1998). If the TCDD equivalence factors were used rather than the TCDD alone, the values for some homes would be above the Michigan standard. The acceptable level of annual ambient air exposure for B(*a*)P in New York and Maryland is zero. The acceptable annual ambient air level for B(*a*)P in Vermont is 3E–11 ng/m^3 (ATSDR, 1998).

The current EPA recommendation for ingestion of TCDD TEQs is that it is not to exceed 0.1 pg/kg/day of dioxin TEQs (USEPA, 2000). This established guideline relates to cancer risk. ATSDR has derived an acute-duration oral Minimum Reportable Limit (MRL) of 0.0002 μg/day. Risk of exceeding these values upon exposure to the house dust, air, run-off water, sediment, and soil matrices exists in this neighborhood. The ingestion of 200 mg of soil from site DS-16B with 7.544 ppb TEQs dioxin would be highly dangerous and likely to have an adverse health impact. Site 16B is a vacant lot next to an occupied home. A 20-kg child playing in this yard could easily ingest 200 mg of soil (USEPA, 1989), for a dose of 75.4 pg/kg/day. Since this same soil also has high levels of PAHs, the population living in this neighborhood is sustaining a very high dose of carcinogenic and toxic chemicals. When all routes of exposure, inhalation, ingestion, and dermal, are included, higher than normal background exposure to dioxins and PAH has most likely occurred.

The finding of high levels of PHC in the house/attic dust indicates that contamination has occurred and is occurring. The Commonwealth of Massachusetts considers 10,000 ppm to be the upper concentration of TPH allowed in soil (ATSDR, 1999b). House/attic dust does not usually have significant TPH as a normal contaminant. Seven of the homes exceed the Massachusetts State allowable level for soil. House dust should probably have a lower acceptable level because of the greater likelihood that it will be ingested or inhaled. In addition, the make-up of this neighborhood's TPH would be expected to contain significant cPAHs as well as chlorinated dioxins based of what is present in the neighborhood kitchens, soil, and ditch sediment. Because there are high levels of cPAHs and dioxins in the soil and ditch sediment near these homes, it is possible that the attic dust also contains high levels of these carcinogenic and toxic pollutants. One thousand nine Moss Street, the house with the highest level of PHC (226,000 ppm), is between DS-16 and DS-15, where very high levels of contamination are present. The contaminants outside the home would be similar to the contaminants inside the home. TPH is probably a helpful surrogate of exposure intensity. It may also be an economical analytical agent for tracking the level of greatest pollution in the neighborhood.

The Agency for Toxic Substances and Disease Registry (ATSDR) has determined an action level of greater than 1 ppb TEQ for dioxin and dioxin-like compounds (ATSDR, 1998). The ATSDR recommends surveillance, research, health studies, community education, physician education, and exposure investigations (ATSDR, 1998). The toxicity levels documented here exceed the action level of the ATSDR and thus warrant a public health action concerning the residents living near the wood processing plant.

We conclude that the data presented here document a substantial contamination of a neighborhood by wood processing waste chemicals. That contamination has not remained in only ditches or in soil but has also reached the bodies of the residents. The predicted health risk to the residents from this contamination is considerable.

Our companion paper reports findings on the health effects found in the nearby residents. The findings suggest the need for more stringent controls on discharges from wood treatment plants to protect the public health.

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