Biomonitoring for Creosote and Pentachlorophenol in Nearby Residents of a Wood Treatment Plant

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Introduction:
Contaminated wood treatment sites can result in adverse health effects to nearby residents. Environmental exposure can be estimated by measuring concentrations of pollutants in air, water, food, or wipe tests. This environmental exposure value can be used as a surrogate to estimate individual exposure. The objective of this study was to determine whether or not pentachlorophenol (PCP) could be found in potentially exposed residents and if the dioxin levels are consistent with PCP exposure. A further objective of the study was to determine whether or not PAH-DNA adducts could be found in the potentially exposed residents.

We present results of biomonitoring studies in residents living near a wood treatment plant that used coal-derived creosote and PCP to process and treat wood for over 100 years. The plant was built in 1904 and used creosote and PCP. Creosote is a complex mixture that contains numerous polycyclic aromatic hydrocarbons (PAHs)[1]. PCP is contaminated with polychlorinated dioxin and furans[2]. The residents' exposure pathways include air, soil and surface water.

Methods and Materials:
In 2003 biomonitoring studies were performed on a total of 29 subjects who were nearby residents of the wood treatment plant. The residents are part of an ongoing litigation against the wood treatment plant due to their concern about associated health problems. Subjects selected for biomonitoring were chosen at random from a total of 103 residents with health problems. Inclusion criteria consisted of subjects living within 2 miles of the plant; above 18 years old; and living in the same residence for at least 5 years. All exposed subjects in the study were fully informed and signed an informed consent to participate in the study. All control and exposed samples were blinded to the laboratory.

Pentachlorophenol and Chlorinated Dioxins and Furans:
Whole blood was collected in chemically cleaned glass containers prepared by the analytic laboratory with anticoagulant and also with Teflon ® tops containing no paper products. Blood was frozen and sent frozen on dry ice to Germany for PCP and polychlorinated dioxin and furan analysis at ERGO Laboratory (World Health Organization certified). Analysis was performed by gas chromatography/high-resolution mass spectrometry by methods previously described[3]. Pooled blood from two hundred adults chosen at random from patients having routine blood testing in a hospital in Dallas, Texas served as controls[4]. Measured levels have been converted to dioxin toxic equivalents (TEQ) using the 1998 WHO toxic equivalent factors (TEFs).

DNA Adducts:
Whole blood was collected in Vacutainer CPT tubes (Becton Dickinson) from only 24 of the 29 subjects. Five (5) subjects failed to appear to have their blood drawn for the adduct testing. These tubes are Ficoll-containing tubes that are drawn under vacuum. The tubes were then transported to a laboratory for centrifugation and separation (by Ficoll Method) of mononuclear cells within 24 hours. The samples were kept on dry ice and sent for DNA adduct analysis to England. PAH-DNA adducts were measured utilizing the ^{32}P-post labeling technique[5]. The PAH-DNA adduct levels have been shown to quantify PAH exposure[6]. The PAH-DNA adducts in lymphocytes are compared to those in an unexposed population in Florida.

Results:
The mean age of the controls is 36 years old. The mean age of the exposed group is 51 years old. The mean PAH-DNA adducts in the control group was 0.87 per 10^8 nucleotides (range = 0.54 – 0.99 per 10^8 nucleotides) whereas the exposed subjects had a mean adduct level of 4.11 per 10^8 nucleotides (range = 1.72 – 8.52 per 10^8 nucleotides).

Table 1 presents the chlorinated dioxins and dioxin-like PCBs (PCDDs, PCDFs, non-ortho PCBs, and mono-ortho PCBs) results. Elevated OCDD (1049 ppt for exposed and 374 ppt for controls) and hepta CDD (132 ppt for exposed and 45.1 ppt for controls) are consistent with contamination of PCP in this exposed group.

Table 2 presents the polychlorinated biphenyls (6 marker PCBs), predominant polybrominated diphenyl ether (PBDEs), hexachlorobenzene, and pentachlorophenol (PCP) levels. PCP levels are twice as high in the exposed residents as compared to the controls (3.0 ug/L and 1.5 ug/L respectively). The range of the PCP values for the exposed residents ranged from 0.8 ug/L to 26 ug/L. Nineteen (19) out of 29 residents had PCP values higher than the control population mean (n=200).

Discussion:
The dioxin, PCB, and PBDE results for the residents are similar to the control population mean values. However, there are some notable increases in the 1,2,3,4,6,7,8-hepta-CDD and OCDD. The exposed residents increase in 1,2,3,4,6,7,8-hepta-CDD and OCDD compared to the control population mean is indicative of past exposure to PCP. Nearly 2/3 of the exposed residents had higher PCP values than the control population mean. These residents have been exposed to PCP and the dioxin pattern (increase in hepta-CDD and OCDD) supports the PCP exposure.

Our PAH-DNA adduct test are the first reported results on residents living near a creosote plant. DNA adduct levels in white blood cells reflect environmental exposure to PAHs[6, 7]. The exposed residents have a mean level of PAH-DNA adducts 4.7 times higher than that of the control population. These results indicate that the residents are being exposed to higher levels of PAH contaminants than the controls. Transformed or activated PAHs can bind to DNA forming adducts, which is widely believed to be the initiating step in chemical carcinogenesis[8, 9]. Few studies have attempted to explain a dose relationship with the PAH and DNA adduct levels[10]. Higher PAH-DNA adduct levels are believed to predict a higher risk of cancer[11].

Studies in animals demonstrate that the levels of DNA adducts are related to PAH contaminated sites[10] as compared to reference sites[12].

Limitations in our study include smoking[13] and dietary intake[15], which are possible confounders. We are currently investigating those issues in more detail but the prevalence of smoking and eating PAH contaminated food should be similar in both exposed and control groups.

Although the TEQ average for the exposed and controls are similar, HpCDD and OCDD are higher than expected with PCP exposure.

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Table 1. Comparisons of dioxins and dioxin-like PCB congeners profiles

Congener	**Exposed Residents (n=29)** **Mean Concentration* [range]**	**Controls-Dallas (n=200)** **Mean Concentration***
2,3,7,8-tetra-CDD	3.4 [1.0-9.7]	3.8
1,2,3,7,8-penta-CDD	8.0 [1.2-23.7]	8.5
1,2,3,4,7,8-hexa-CDD	10.6 [1.4-46.8[	7.5
1,2,3,6,7,8-hexa-CDD	51.2 [7.1-231.0]	41.3
1,2,3,7,8,9-hexa-CDD	9.5 [1.5-60.1]	5.7
1,2,3,4,6,7,8-hepta-CDD	**132.0 [16.5-847.2]**	**45.1**
OCDD	**1049 [117.7-8892.5]**	**374**
2,3,7,8-tetra-CDF	1.1 [1.0-1.9]	n.d. (LOD 1.0)
1,2,3,7,8-penta-CDF	n.d (LOD 1.0-2.3; mean=1.0)	n.d. (LOD 1.0)
2,3,4,7,8-penta-CDF	3.4 [1.0-104.4]	5.0
1,2,3,4,7,8-hexa-CDF	7.8 1.6-32.0]	7.4
1,2,3,6,7,8-hexa-CDF	4.5 [1.4-18.8]	4.0
1,2,3,7,8,9-hexa-CDF	n.d. (LOD 2.0-99.4; mean=30.4)	n.d. (LOD 48.1)
2,3,4,6,7,8-hexa-CDF	1.0 [1.08-6.8]	n.d. (LOD 1.4)
1,2,3,4,6,7,8-hepta-CDF	11.6 [3.6-47.6]	4.1
1,2,3,4,7,8,9-hepta-CDF	n.d. (LOD 1.0-3.2; mean=1.4)	n.d. (LOD 1.1)
OCDF	n.d. (LOD 1.2-7.1; mean=2.6)	2.5
3,3',4,4'-TCB-77	n.d. (LOD 27-100; mean=53)	n.d. (LOD 25)
3,4,4',5-TCB-81	1[n.d.-5]	5
3,3',4,4',5-PeCB-126	44 [9-184]	42
3,3',4,4',5,5'-HxCB-169	18 [3-50]	31
2,3,3',4,4'-PeCB-105	2208 [522-9732]	3333
2,3,4,4',5-PeCB-114	8261 [126-1914]	1338
2,3',4,4',5-PeCB-118	11508 [2855-40565]	17283
2',3,4,4',5-PeCB-123)	280 [n.d.-1197]	307
2,3,3',4,4',5-HxCB-156	5832 653-25954]	7852
2,3,3',4,4',5'-HxCB-157	1251 [139-4740]	1753
2,3',4,4',5,5'-HxCB-167	1571 [287-5019]	2044
2,3,3',4,4',5,5'-HpCB-189	457 [40-1782]	685
Total_PCDD/PCDF	1303	509
Total_non-ortho-PCB	63	78
Total_mono-ortho-PCB	23724	34595
TEQ (WHO) based on PCDD/F	23	22
TEQ (WHO) based on PCB	10	12
TEQ(WHO)?	33	34

*values in pg/g (ppt) lipid based.
n.d. = not detected
LOD = Limit of detection range in ( )

Table 2. Mean Concentrations of HCB, PCBs, PBDEs, p,p'-DDE and PCP in exposed and control residents.

Congener	Exposed Residents n=29	Controls n=200
PCP*	**3.0**	**1.5**
HCB*	**0.040**	**0.073**
PCB #28*	0.009	n.d.(LOD 0.04)
PCB #52*	n.d. (LOD 0.01)	n.d. (LOD 0.01)
PCB #101*	n.d. (LOD 0.01)	n.d. (LOD 0.03)
PCB #138*	0.128	0.12
PCB #153*	0.149	0.15
PCB #180*	0.123	0.16
PBDE #28**	1.2	1.9
PBDE #47**	28.4	44
PBDE #99**	13.0	13
PBDE #100**	5.3	5.2
PBDE #153**	5.4	12
PBDE #154**	1.3	0.82
PBDE #183**	0.3	0.3
PBDE #209**	1.7	1.4
ppDDE*	4.6	3.3

*other chlorinated parameters in ug/l, original weight based
**data in ng/g, lipid based
n.d. = not detected
LOD = Limit of detection