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DIOXIN AND DIBENZOFURAN CONGENERS IN BLOOD OF NEIGHBORS OF A WOOD TREATMENT PLANT 25 YEARS AFTER ENDING PENTACHLOROPHENOL (PCP) USE

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Introduction

Ten African American neighbors in a southern US location who lived near a wood treatment plant for many years had blood analysis for dioxin and dibenzofuran congeners. Exposure pathways included air, soil and surface water contaminated with chlorinated dioxins and furans. The wood treatment plant is believed to have used pentachlorophenol (PCP) between 1928 and the mid 1970s. High pressure and heat was applied to facilitate placing the PCP in wood. Discharge of contaminants into the air occurred from normal operations created low-level exposure on a daily basis. Unusual events such as fires and floods caused brief high levels of exposure. After cessation of PCP use its contaminants remain in the neighborhood in sediment of drainage ditches and in soil. Ten blood samples from residents drawn and analyzed in 2000 found elevation of higher chlorinated dioxins. Comparison with PCP workers reveals levels in these residents that are similar to exposed workers^{5,6}. The elevated higher chlorinated dioxins, especially OCDD, seem consistent with previous low level of chronic exposure to PCP, which is characterized by dioxin contamination with the higher chlorinated ccngeners.³ Comparison with a pooled blood collected at about the same time from 200 Dallas, Texas residents is shown to document current congener levels and elevated dioxin levels in the general population. Levels in the current Dallas blood show lower dioxin levels than previously reported¹⁻².

Methods

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, for the workers and for the Dallas comparison group. Blood was frozen and sent frozen on dry ice to Germany for dioxin analysis at ERGO Laboratory. Analysis was performed by high resolution gas chromatography-high resolution mass spectrometry by methods previously described.⁴

Results

Data from the residents and neighborhood drainage ditch sediment are shown in Table 1, which presents dioxin congeners specific analyses in 10 residents and two seciment samples collected near the plant and analyzed by the same laboratory. Elevations of higher chlorinated dioxins, especially hepta- and octachlorinated dioxins are seen in blood of residents, as well as in sediment. Presumably

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the furans with shorter half-lives of elimination may have been excreted during the 25 years following cessation of PCP use prior to blood sampling.

Table 2 compares the PCP wood processing plant neighbors with the Dallas general population blood and previous tissue levels in PCP exposed German, Canadian, and Chinese workers.^{3, 5-7} Elevation of OCDD is especially seen in the PCP exposed wood treatment plant workers. As expected, the Canadian worker who died of acute PCP poisoning and reported by Ryan⁷, had quite high levels of those congeners found in PCP. The German workers reported by Paepke³ had higher levels also presumably due to more current exposure. In rural China, the levels of dioxins in general is lower than in more industrialized countries, and PCP exposure may not have been as high as the other cohorts presented.

Conclusions and Discussion

The findings of elevated OCDD in blood of residents environmentally exposed to PCP 25 years prior is consistent with an elevated body burden of dioxins from PCP. The lack of elevation of some other congeners of PCDD/F found in PCP is consistent with excretion of dioxins and dibenzofurans over time. These findings seem consistent with the first report of elevated dioxins in PCP exposed persons as long as 25 years after exposure, to the best of our knowledge. Exposure to chlorinated dioxins and dibenzofurans after the 1970s from neighborhood contamination is also a possible explanation for the results. Further research is needed to document wood treatment plant neighbors to dioxin and dibenzofuran exposure to various technical mixtures of PCP at various times during and after exposure.

References

- 1. Schecter A (1994) Exposure assessment: Measurement of dioxins and related chemicals in human tissues. In: Schecter, A. (ed.). Dioxins and Health. New York: Plenum Press, 449-485
- 2. Schecter A., McGee H., Stanley J.S. and Brandt-Rauf P. (1996). Dioxins and dioxin-like chemicals in blood and semen of American Vietnam veterans from the state of Michigan. Am J Ind Med, 30(6):647-654
- 3. Paepke O., Ball M., Lis A. (1992) Various PCDD/PCDF patterns in human blood resulting from different occupational exposures. Chemosphere, 25(7-10): 1101-1108
- 4. Paepke O., Ball M., Lis A. and Sheunert (1989), PCDD and PCDF in whole blood samples of unexposed persons. Chemosphere 19, 941-948.
- 5. Schecter A.J., Li L., Ke J., Furst P., Furst C. and Papke O. (1996) Pesticide application and increased dioxin body burden in male and female agricultural workers in China. J Occup Environ Med., 38(9):906-11.
- 6. Schecter A., Jiang K., Papke O., Furst P., Furst C. (1994) Comparison of dibenzodioxin levels in blood and milk in agricultural workers and others following pentachlorophenol exposure in China. Chemosphere, 29(9-11):2371-80.
- 7. Ryan J. J., Lizotte R. and Lewis D. (1987) Human tissue levels of PCDDs and PCDFs from a fatal pentachlorophenol poisoning. Chemosphere, 16(8/9): 1989-1996.

Table 1. Dibenzodioxin, dibenzofuran and PCB levels in wood treatment plant workers exposed to pentachlorophenol and in sediments from plant's proximity

Congeners	I-TEFs I	Resident	Ditch	Drainage Ditch Sediment 2									
		1	2	3	4	5	6	7	8	9	10		
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
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
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
1,2,3,6,7,8-HxCDD	0.1	72	69.3	79.1	55.2	68.4	31.6	37.4	51.1	52.9	25	1202	31.7
1,2,3,7,8,9-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
1,2,3,4,6,7,8-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
OCDD	0.001	2152.7	1038	744.2	1015.7	644.3	711.7	822.9	505.1	679.5	277.5	931200	302097
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
1,2,3,7,8-PeCDF	0.05	0.6	ND	- ND	ND	1	ND	ND	ND	ND	5	84	0.5
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
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
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	2.0
1,2,3,7,8,9-HxCDF	0.1	nd	71	0.6									
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	141	2.1
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
1,2,3,4,7,8,9-HpCDF	0.01	nd	1402	15.9									
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
Coplanar PCBs													
33'44'-TCB 77	0.0005	33	NA	NA	NA	31	NA	NA	30	35.5	28.5	1228	28
33'44'5-PCB 126	0.1	69	40					67				7	NA
33'44'55'-HCB 169	0.01	19	36	49	15	42	11	21	19	28	13	1	<u>NA</u>
Total PCDDs		2423.2	1248.9					967.2		785.2		981168	304508
Total PCDFs		45.4	41.5					33.2				90158	1128
Coplanar PCBs		121.0	76.0									1236	28
Total PCDD/Fs&PCB		2589.6	1366.4	1043.0	1337.7	970.3	887.1	1088.4	711.4	917.7	435.9	1072562	
Total TEQ		37.1	32.0	31.4	32.6	29.3	22.0	25.1	20.8	19.6	12.8		

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ND - not detected, NA - not available

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Congeners	I-TEQ	PCI	P wood trea	tment reside		General population Dallas (Schecter 2000	PCP poisoning (Ryan 1987)	PCP workers Germany (Papke1992)	PCP direct contact, China (Schecter 1996)	
		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	51	12.5	6.3	32	28.3	\sim 7.2	
1,2,3,4,7,8-HxCDD	0.1	9	8.7	4	15.1	6.4		47.9	22.1	
1,2,3,6,7,8-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	7020	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	1.0	ND (2.0)	2.6	1.5	
1,2,3,7,8-PeCDF	0.05	22	1	0.6	5	0.5	NA	35	2.4	
2,3,4,7,8-PeCDF	0.5	4.8	4.2	3.1	7.2	45	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	35	80	63.7	23	
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	nđ	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(PCBs)		1152.4	991.6			621.0	78657	36587	1252	
Total TEQ		26.6	24.7			18.2	283.1	158.6	14.9	

Table 2. Comparison of dioxin congeners profiles in samples of pentachlorophenol exposed workers and general US population

ND - not detected (limit of detection), NA - not available