

SERUM DIOXIN AND PCB LEVELS AMONG PCB WASTE PROCESSING PLANT WORKERS

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Introduction

A clinical investigation and a biological monitoring were carried out to evaluate the occupational exposure of workers in contact with pyralen in a PCB-transformer and capacitor decontamination plant in France. The aim of this study was to assess the body burden of polychlorinated dibenzo-*p*-dioxin (PCDDs), polychlorinated dibenzofurans (PCDFs), dioxin-like (DL) PCBs and marker PCBs for those workers. We examined 8 representative workers (34-44 years old). The subjects of this study worked from 5 years to 19 years at the plant. All workers reported daily multiple contacts with various metal and wood parts of dismantled transformers during working hours. On a yearly basis, the exposed subjects were examined by the occupational health physician for a check-up. Basic parameters such as liver function and lipid statement were performed.

Materials and Methods

Eight workers were referred to the University Hospital Occupational Health Department. They underwent medical examination including an interview through a questionnaire focused on their dietary habits, the self-production of foods and the intake of locally grown food.

100 ml of blood were collected in blood bags by the French National Blood Transfusion Center in Nantes and left overnight in a fridge at 4°C. Serum was then harvested from blood by centrifugation, transferred in 100 ml amber glass vials, frozen and stored at -20°C. Specimens were later shipped on dry ice to the dioxin laboratory CART in Liège, Belgium. They were stored at -20°C prior analysis. Thirty-five target compounds (the 17 2,3,7,8-PCDD/Fs, the 12 dioxin-like PCBs and the 6 marker PCBs) were measured under accredited procedures. The extraction, purification and analysis methods are described elsewhere¹. The serum total lipids (TL) were estimated by enzymatic colorimetric assays. The total cholesterol, free cholesterol, triglyceride and phospholipids were individually determined by enzymatic colorimetric tests. The TL estimate was then obtained by summation according to Akins et al.² formula.

Results and discussion

Table 1 presents the current dioxin levels for the eight PCB waste processing plant workers who took part in the study. It highlights the high levels of PCDFs, DL-PCBs and non DL-PCBs in their serum. The mean TEQ for the comparisons was 8.4 pg/g lipids for PCDDs, 157.5 pg/g lipids for PCDFs and 1272.8 pg/g lipids for DL-PCBs yielding the total mean TEQ of 1438.7 pg/g lipids using 1998 World Health Organisation toxic equivalent factors (WHO

TEFs)³. Corresponding mean TEQs in these workers were 8.4 pg/g lipids for PCDDs, 103.1 pg/g lipids for PCDFs and 394.4 pg/g lipids for DL-PCBs for a total TEQ of 505.9 pg/g lipids when using 2005 WHO TEFs⁴ (Table 1). The PCDDs TEQ did not change substantially. The PCDFs TEQs in comparison decreased by 35% to 103.1 pg/g lipids. The most significant effect of using the re-assessed TEFs was seen in DL-PCBs with 69% decrease to 394.4 pg/g lipids. The eight patients showed neither chloracne nor liver abnormality. These PCDD/Fs TEQ₁₉₉₈ levels correspond to the mean TEQ levels measured in 83 Yusho patients 27 years after the incident (i.e. 156 pg/g lipids)⁵. Compared to the mean TEQ levels from the French national study⁶ organised in 2005, the exposed subjects have PCDFs and DL-PCBs TEQ levels higher by one or two orders of magnitudes, respectively (Table 1). It is also worthwhile to underline the atypical congener's profile in their serum compared to the one observed in background exposed population. Ryan et al. reported that, in the case of Yusho and Yucheng patients who ingested contaminated PCB rice oil, 2,3,4,7,8-PnCDF and 1,2,3,4,7,8-HxCDF were the two main PCDD/F persistent toxic congeners in their blood with a contribution of about 70% of 2,3,4,7,8-PnCDF to the total PCDD/Fs TEQ₁₉₉₈⁷. Such a manifest pattern is noticed in Table 1 and graphically illustrated in Figure 1. The contribution of 2,3,4,7,8-PnCDF to the total PCDD/Fs TEQ₁₉₉₈ in this study is 80% versus 36% in the French national study (Figure 1 A and B).

When comparing the data from the French national study to the exposed workers for marker PCBs, higher levels by two orders of magnitude are observed (Table 1). Recently, Dalghren et al. reported blood marker PCB levels in transformers manufacturing workers in the range of 3 to 6 ppb⁸ which is much lower than what observed in our study.

We also examined the TEQ serum levels of these workers versus their occupational exposure. Figure 2 demonstrated the longer their occupational exposure, the higher their serum PCDD/s and DL-PCB levels. One worker shows much higher level compared to the rest of the group. No evidence of additional occupational exposure was highlighted. However, a potential non-occupational exposure related to high fat dietary habits of exclusively self-production of foods such as pigs, poultry, eggs, fruits and vegetables for this worker living in the vicinity of the PCB waste processing plant was pointed out.

The very high levels of PCDFs and PCBs in workers serum made the factory aware how working environment and safety conditions are pivotal. In the past few years, a series of measures were taken to limit occupational exposure. It is very likely that most of the exposure occurred during the nineties. A medical follow-up of these workers will be carried out in a near future in order to understand what might be the health effects of such exposure.

References

1. Focant J.-F., Eppe G., Massart A.-C., Scholl G., Pirard C., De Pauw E. *Journal of Chromatography, Serie A*. 2006; 1130: 97-107
2. Akins J.-R., Waldrep K., Bernet Jr. *Clin. Chim Acta*. 1989;184:219-226
3. Van den Berg M., Birnbaum L.S., Bosveld A.T.C., Brunström B., Cook P., Feeley M., Giesy J.P., Hanberg A., Hasegawa R., Kennedy S.W., Kubiak T., Larsen J.C., van Leeuwen F.X.R., Liem A.K.D., Nolt C., Peterson R.E., Poellinger L., Safe S., Shrenk D., Tillitt D., Tysklind M., Younes M., Waern F., Zacharewski T., *Environ. Health Perspect.* 1998; 106: 775.
4. Van den Berg M., Birnbaum L.S., Denison M., DE Vito M., Farland W., Feeley M., Fiedler H., Hakanson H., Hanberg A., Haws L., Rose M., Safe S., Shrenk D., Tohyama C., Tritscher A., Tuomisto J., Tysklind M., Walker N., Waern F., Peterson R.E.
5. Masuda Y *Chemosphere*. 2001; 43 (4-7):925-930.
6. Fréry N., Zeghnoun A., Sarter H., Volatier J.-L., Falq G., Pascal M., Grange D., Schmitt M., Bérat B., Fabre P., Guillois-Becel Y., Noury U., Pouey J., Mathieu A., Heymann C., Lucas N., Thébault A., Eppe G., Focant J.-F., Le Strat Y., Pelletier B., Salines G., *Organohalogen compounds*. 2007; vol 69: 1013-1016.
7. Ryan J.J., Levesque D., Panopio L.G., Sun W.F., Masuda Y., Kuroki H. *Archives of Environmental Contamination and Toxicology*. 1993; 24 (4): 504-512.
8. Dalghren J.G., Takhar H., Warshaw R. *Organohalogen compounds*. 2007; vol 69: 739-741

Table 1: Comparison of mean congener levels in the present study (n=8) with the mean levels obtained during the French national survey (n=1030)⁶ expressed as pg/g lipids [min, max]

	<i>Mean (n=8)</i>		<i>Mean (n=1030)</i>	
Total PCDD (pg TEQ WHO₁₉₉₈/g lipids)	8.4	[4.9-16.4]	7.7	[1.1-59.9]
Total PCDD (pg TEQ WHO₂₀₀₅/g lipids)	8.4	[5.0-16.5]	7.7	[1.1-60.2]
2,3,7,8 - TCDD	1.0	[0.6-1.5]	0.7	[0.1-3.7]
1,2,3,7,8 - PnCDD	4.5	[2.0-8.1]	3.9	[0.1-23.1]
1,2,3,4,7,8 - HxCDD	2.6	[1.5-5.2]	2.5	[0.2-28.1]
1,2,3,6,7,8 - HxCDD	22.4	[13.4-46.6]	20.1	[4.0-269.0]
1,2,3,7,8,9 - HxCDD	4.8	[3.3-9.5]	2.8	[0.2-30.0]
1,2,3,4,6,7,8 - HpCDD	25.6	[7.1-71.1]	24.4	[2.7-384.0]
OCDD	147.9	[57.5-375.1]	195.3	[15.0-1409.7]
Total PCDF (pg TEQ WHO₁₉₉₈/g lipids)	157.5	[63.5-350.8]	5.9	[1.0-50.0]
Total PCDF (pg TEQ WHO₂₀₀₅/g lipids)	103.1	[43.7-232.8]	3.9	[0.5-35.2]
2,3,7,8 - TCDF	10.4	[2.8-26.2]	0.3	[0.0-3.5]
1,2,3,7,8 - PnCDF	7.4	[2.8-20.5]	0.1	[0.0-4.6]
2,3,4,7,8 - PnCDF	265.0	[98.3-588.3]	9.8	[0.8-85.5]
1,2,3,4,7,8 - HxCDF	178.0	[81.0-397.0]	2.9	[0.8-19.9]
1,2,3,6,7,8 - HxCDF	38.2	[17.0-91.4]	4.3	[1.2-32.6]
1,2,3,7,8,9 - HxCDF	1.5	[0.8-3.2]	0.2	[0.1-6.5]
2,3,4,6,7,8 - HxCDF	14.1	[5.8-32.0]	1.3	[0.1-17.1]
1,2,3,4,6,7,8 - HpCDF	34.8	[21.9-56.3]	4.2	[1.1-150.2]
1,2,3,4,7,8,9 - HpCDF	8.3	[5.2-13.9]		[0.3-2.3]*
OCDF		*		[2.8-33.5]*
Total DL-PCBs (pg TEQ WHO₁₉₉₈/g lipids)	1272.8	[520.5-3009.9]	13.6	[2.5-99.0]
Total DL-PCBs (pg TEQ WHO₂₀₀₅/g lipids)	394.4	[186.0-1119.0]	6.0	[0.6-68.8]
PCB 77	134	[89-237]		[93.0-450.9]*
PCB 81	124	[44.4-234]		[23.3-180.0]*
PCB105	346015	[166843-801480]	1878	[522-44097]
PCB114	115307	[37088-296727]	962	[69-9126]
PCB 118	1794264	[1019318-4936015]	12041	[1837-139349]
PCB 123	22381	[5551-84233]	72.7	[0.3-3699.8]
PCB126	2432	[726-7659]	34.3	[1.9-476.5]
PCB156	1288423	[280298-2684387]	11252	[1326-81521]
PCB157	162491	[32834-390949]	2489	[331-21418]
PCB167	488236	[167931- 1225144]	4213	[545-38792]
PCB169	632	[149-1171]	49.1	[6.9-348.8]
PCB189	190936	[31609-517430]	1812	[232-13835]
PCB markers (ng/g lipids)	41635	[12797-94839]**	348	[8-2466]**
PCB28	787.5	[65.5-2479.1]		*
PCB52	30.4	[21.3-56.7]		*
PCB101	169.3	[117.1-250.6]		*
PCB138	8675	[3336-11831]	55	[1.5-367]
PCB153	16269	[4397-34539]	120	[0.9-996]
PCB180	15703	[2631-43278]	154	[1.7-1039]

*: 99% of values were <LOQ

** : the sum of PCB 138, 153, 180

Figure 1: Percent Contribution of 2,3,4,7,8 PnCDF, PCDDs and PCDFs to the total mean PCDD/Fs TEQ using 1998 WHO TEFs, (A) French national study (InVS)⁶, (B) exposed PCB workers

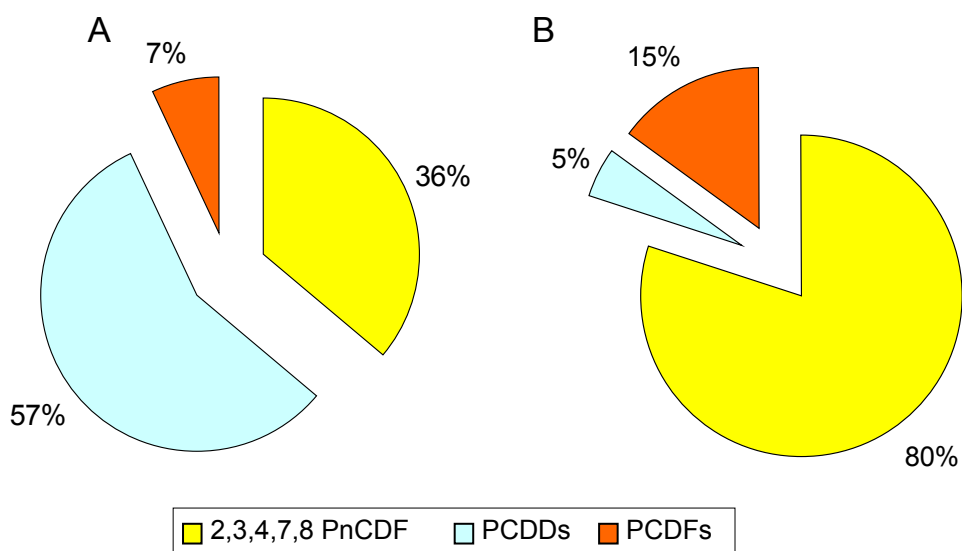


Figure 2: TEQs serum levels against length of service in the PCB plant for the 8 workers

