

POLYCHLOROBIPHENYL (PCB), POLYCHLORODIBENZODIOXIN (PCDD), AND POLYCHLORODIBENZOFURAN (PCDF) EXPOSURE OF FIREFIGHTERS INVOLVED IN THE PCB FIRE AT ST-BASILE-LE-GRAND, QUEBEC, CANADA

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ABSTRACT:

On August 23, 1988, 110 firemen were exposed to a fire involving approximately 8 000 liters of PCB<sub>s</sub> and 7 000 liters of PCB contaminated oil. To assess their exposure to PCB<sub>s</sub>, PCDD<sub>s</sub> and PCDF<sub>s</sub>, plasma concentrations of these substances in the 29 most exposed firefighters were compared to those measured in an unexposed group of 30 firemen. The results of this study clearly indicate that exposure to the PCB fire did not result in significant absorption of PCB<sub>s</sub>, PCDD<sub>s</sub> and PCDF<sub>s</sub>.

INTRODUCTION:

On August 23, 1988, a fire broke out at a polychlorobiphenyl (PCB) storage facility located in St-Basile-Le-Grand, a rural municipality (population: 8 000) located 25 kilometres east of Montreal, Canada. At the time of the fire, it was believed that the warehouse contained approximately 152 000 kilograms of commercial PCB<sub>s</sub> (95% Aroclor 1242 [42% Cl], 5% Aroclor 1254 [54% Cl]), 10 000 to 35 000 litres of PCB-contaminated oil, and an unknown quantity of organic solvents. An inventory of the warehouse contents performed five hours later, after the fire had been extinguished, indicated that 8 000 litres of PCB<sub>s</sub> and 7 000 litres of oil had been consumed; it was not possible to estimate the quantity of solvents burnt up.

Approximately 110 firefighters were involved in combatting the fire and organizing the evacuation of the population residing under the emission plume. Upon recommendation of a medical advisor, firefighters wore positive-pressure self-contained breathing apparatus. However, the medical advisor did not reach the site until 2½ hours after the fire had started, and some of the firefighters were therefore unprotected during this initial period.

In the days following the fire, 70% of the firefighters complained of at least one of the following symptoms: throat irritation (40%), headache (24%), eye irritation (23%), coughing (17%), skin irritation (15%), taste alteration (13%) and nausea (9%). At least one objective clinical sign was present in 15% of cases; the most common signs were pharyngitis, conjunctivitis, and contact dermatitis. These clinical features may have been caused by caustic substances such as HCl and Cl<sub>2</sub>, likely to have been present in the smoke. Three days after the event (August 26, 1988), firefighters underwent a health assessment which included a medical examination, clinical chemistry work-up (CBC, biochemical profile), plasma PCB measurements, and a questionnaire on exposure, symptoms and lifestyle factors.

The purpose of this study was to quantify PCB<sub>s</sub>, PCDF<sub>s</sub> and PCDD<sub>s</sub> absorption by firemen who were involved in fighting this fire, and consequently, to assess the health risk of this exposure. Plasma levels of PCB<sub>s</sub>, PCDD<sub>s</sub> and PCDF<sub>s</sub> were compared in exposed and unexposed firefighters, with particular attention to congeners identified in environmental samples as being specific markers of exposure to this PCB fire.

In a preliminary investigation performed by the Quebec Toxicology Center, no exposed firefighters were found to have plasma PCB levels above 5 µg/l (5 ppb), for Aroclors 1242 and 1254 contained in the warehouse. However, the composition of a representative soot sample found to contain polyaromatic chlorinated hydrocarbons suggested that further investigation of PCDF and PCDD levels was nevertheless warranted (Table I). The soot in this sample, taken from the top of a truck located within 50 metres of the warehouse, contained significant amounts of PCDF<sub>s</sub>, and was found to have a PCB/PCDF ratio of 250:1, rather than the 50 000:1 ratio commonly found in commercial PCB<sub>s</sub>. With a PCB/PCDF ratio of this magnitude, PCDF absorption may be significant, even though PCB levels remain relatively low. Given a serum lipid concentration of 0.5% and a PCB/PCDF ratio of 250:1, for example, a plasma PCB level of 3 µg/l (600 µg/kg lipid) translates into a plasma-lipid-based PCDF level of 2.4 µg/kg, compared to the current Canadian average of 0.06 µg/kg (Ryan et al 1986).

#### MATERIAL AND METHODS

The exposed group was composed of 29 of the 110 firefighters present at the fire. Selection was on the basis of a high probability of intense and/or lengthy exposure. The exposed firefighters were further classified into heavily-exposed and moderately-exposed sub-groups, with the former group having spent at least 4 hours at the site. The control group was composed of 30 firemen from neighbouring municipalities located upwind of St-Basile, with no previous exposure to PCB fires. Participation in the control group was voluntary. All firefighters studied were male.

A 500 ml blood sample was drawn from each fireman by Canadian Red Cross personnel on January 10, 1989. The frozen plasma was analyzed by the Midwest Research Institute, Kansas City, Missouri. Plasma concentrations of PCB<sub>s</sub>, PCDD<sub>s</sub> and PCDF<sub>s</sub> were determined using a modification of a standard HRGC/HRMS (High Resolution Gas Chromatography/High Resolution Mass Spectrometry) analytical protocol developed by the Environmental Protection Agency (EPA) and the Centres for Disease Control (CDC).

#### RESULTS

##### 1. Characteristics of the exposed and control groups

The potentially confounding effects of age, weight, and sex were analyzed for both exposed sub-groups and the control group by an analysis of variance (Tables II, III).

##### 2. PCB<sub>s</sub>

###### 2.1 Congener profile

The following congeners were detected in the plasma of firefighters (numbers in brackets correspond to the classification system devised by Ballschmiter et al. 1989): 2,3',4,4',5 PeCB (118); 2,2',3,4,4',5' HxCB (138); 2,2',4,4',5,5' HxCB (153); 2,3,3',4,4',5 HxCB (156); 2,2',3,3',4,4',5 HpCB (170); 2,2',3,4,4',5,5' HpCB (180); 2,2',3,4',5,5',6 HpCB (187) and 2,2',3,4,4',5',6 HpCB (183).

Previous analyses have demonstrated that only congeners 118, 138, and 153 are present in Aroclor 1242, where they account for 0.03%, 0.08% and 0.02%, respectively, of the commercial product (Albro and Parker 1979). In contrast, congeners 138 and 153 are the principal components of Aroclor 1260 (11.4% and 9.9%, respectively) (Duinker and Hillebrand 1983), and congener 118 is one of the principal components of Aroclor 1254. No Aroclor 1242-specific congener was detected in the blood of any of the firefighters.

#### 2.2 Plasma PCB levels

As table IV indicates, no statistically significant difference was found between the exposed and control groups for total PCB concentrations, which are generally lower than national levels. Specific PCB congeners results (not shown) do not differ significantly either.

#### 3. PCDF<sub>s</sub> and PCDD<sub>s</sub>

The principal congeners found in the soot sample (Table I) were the TeCDF<sub>s</sub>, PeCDF<sub>s</sub>, and HxCDF<sub>s</sub>, with HpCDF<sub>s</sub>, OCDF, and PCDD<sub>s</sub> all present at much lower concentrations. Interestingly, this soot sample was the only environmental sample in which 2,3,7,8-TCDD was detected.

No significant difference was observed between the levels of any of the individual congeners or the sum of tetra- to hexa- PCDF<sub>s</sub> ( $\Sigma$  TePeHx-CDF) in the exposed and control groups (Table V).

Exposed firefighters, and more particularly those in the heavily-exposed sub-group ( $\geq 4$  hours on-site), have a significantly higher level of total PCDD<sub>s</sub> (Table VI), probably attributable to an increased level of HpCDD<sub>s</sub> and OCDD. However, given the measured PCDF/PCDD ratio of 360:1, and the absence of a similarly increased plasma level of total PCDF<sub>s</sub>, it is not possible to attribute this increase in PCDD to fire-related exposures.

As table VII indicates, no correlation was observed between age, weight and smoking, and the concentrations of PCDF<sub>s</sub> or PCDD<sub>s</sub> in either group. Finally, the TCDD equivalent toxicity of PCDF<sub>s</sub>, PCDD<sub>s</sub>, and  $\Sigma$  TePeHx-CDF was calculated in both groups, using the EPA (1987) and NATO-COMS (1988) weighting factors listed in table VIII. No significant difference was found (Table IX).

#### DISCUSSION

The results of this study clearly indicate that firefighters involved in this PCB fire did not significantly absorb PCB<sub>s</sub>, PCDD<sub>s</sub> or PCDF<sub>s</sub>. This finding may have two explanations. Firstly, it may well be that toxic releases from the fire were indeed low. This hypothesis is supported by the relatively low concentrations of contaminants found in environmental samples. Secondly, firefighters may have adopted mitigating strategies such as maintaining a minimum distance from the fire or staying upwind, before respiratory protective devices become available for all.

PCDD and PCDF values measured in this study are consistent with those previously observed in the Canadian population (Ryan 1986). The mean  $\Sigma$  TePeHx-CDF values measured in the firefighters (34.4 ng/kg lipid) is far less than that found to cause disease in one Yusho and one Yu-Cheng patients, where levels of 9 300 ng/kg and 12 700 ng/kg, respectively, have been observed (Kuroki and Masuda 1978, Chen et al. 1985). The ratio of concentrations found in these Yusho and Yu-Cheng patients to those observed in the firefighters examined in this study is 270:1 and 369:1, respectively.

Similarly, the TCDD equivalent toxicity concentrations found in the firefighters' plasma corresponded to average Canadian values (Ryan 1986), and was much lower than those measured in 8 children who developed chloracne within 1 month of the Seveso accident (CDC 1988). While, in the firefighters' plasma, the average TCDD equivalent toxicity concentration is 12.4 or 24.38 ng/kg lipid, depending on the method of calculation employed, that of the children varied from 828 to 27 825 ng/kg.

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Table I

Composition of a soot sample taken from a truck roof located 50 metres from the fire

Species	Conc. (ng/g)
Total PCB <sub>g</sub>	1 723 219
2,3,7,8 TCDF	2 858
Total TCDF <sub>g</sub>	5 824
PeCDF	761
HeCDF	216
IpCDF	22
OoCDF	30
Total PCDF <sub>g</sub>	6 053
2,3,7,8 TCDD	6
Total TeCDD <sub>g</sub>	6
PeCDD	ND
HeCDD	ND
IpCDD	6
OoCDD	7
Total PCDD <sub>g</sub>	19
Total PCB <sub>g</sub> /Total PCDF <sub>g</sub>	250: 1
Total PCB <sub>g</sub> /Total PCDD <sub>g</sub>	360: 1

ND not detected

Table II

Average age, weight, and tobacco consumption of subjects

Group	N	Age (years)			Weight (kg)			Tobacco consumption (pack-years)		
		$\bar{x}$	SD	p	$\bar{x}$	SD	p	$\bar{x}$	SD	p
Exposed	29	32.8	8.2	0.037	83.83	15.1	0.074	176.69	121.3	0.897
Control	30	28.7	6.4		84.41	12.7		170.77	194.0	

N number of subjects per group

$\bar{x}$  mean

SD standard deviation

Table III

Analysis of variance of age, weight and tobacco consumption

Group	N	Age (years)			Weight (kg)			Tobacco consumption (pack years)		
		$\bar{x}$	SD	p	$\bar{x}$	SD	p	$\bar{x}$	SD	p
Exposed high	18	33.4	9.04	0.163	86.08	16.4	0.299	224.44	309.06	< 0.0001
moderate	11	31.7	6.87		80.16	12.6		106.45	149.07	
Control	30	28.7	6.45		84.41	12.7		170.77	194.84	
Total	59	30.7	7.58		84.13	13.8		175.15	262.30	

N number of subjects per group

$\bar{x}$  mean

SD standard deviation

Table IV

Whole plasma and plasma-lipid-based total PCB concentrations

Group	N	Whole plasma ( $\mu\text{g/l}$ )			Lipid ( $\mu\text{g/kg}$ )		
		$\bar{x}$	SD	p	$\bar{x}$	SD	p
Exposed	29	1.40	0.899	0.344	230.24	136.38	0.539
Control	29	1.88	1.324		260.41	224.17	

N number of subjects per group

$\bar{x}$  mean

SD standard deviation

Table V  
 Plasma-lipid-based concentrations (ng/kg) of PCDF<sub>s</sub> and PCDD<sub>s</sub>  
 in exposed and control subjects

Congener	Status	N	$\bar{x}$	SD	p
<b>FURANS</b>					
* 2,3,7,8 - TCDF	E	24	6,3117	4,366	0,145
	C	28	8,5761	6,559	
* 1,2,3,7,8 - PeCDF	E	0	0	N.A.	N.A.
	C	1	2,61	N.A.	
* 2,3,4,7,8 - PeCDF	E	21	10,9543	5,189	0,431
	C	26	9,8654	4,205	
* 1,2,3,4,7,8 - HxCDF	E	27	7,6363	3,459	0,9
	C	30	7,5353	3,088	
* 1,2,3,6,7,8 - HxCDF	E	28	7,2929	3,42	0,809
	C	28	7,0857	2,959	
* 2,3,4,6,7,8 - HxCDF	E	24	3,0075	0,996	0,944
	C	28	3,0296	1,214	
* 1,2,3,7,8,9 - HxCDF	E	0	0	N.A.	N.A.
	C	0	0	N.A.	
1,2,3,4,6,7,8 - HpCDF	E	29	24,270	11,291	0,785
	C	30	23,445	11,822	
1,2,3,4,7,8,9 - HpCDF	E	0	0	N.A.	N.A.
	C	1	2,71	N.A.	
1,2,3,4,6,7,8,9 - OCDF	E	0	0	N.A.	N.A.
	C	1	13,8	N.A.	
* 2 TePeHx - CDF	E	29	32,2848	13,933	0,267
	C	30	36,4453	14,568	
Total PCDF <sub>s</sub>	E	29	56,5548	22,659	0,519
	C	30	60,4407	23,347	
<b>DIOXINS</b>					
2,3,7,8 - TCDD	E	17	4,3	2,83	0,586
	C	13	3,81	1,69	
1,2,3,7,8 - PeCDD	E	25	8,026	2,198	0,316
	C	24	7,181	2,601	
1,2,3,4,7,8/ 1,2,3,6,7,8 - HxCDD	E	29	72,847	32,257	0,666
	C	30	69,436	27,954	
1,2,3,7,8,9 - HxCDD	E	28	12,643	7,120	0,772
	C	30	12,156	5,608	
1,2,3,4,6,7,8 - HpCDD	E	29	106,46	57,159	0,191
	C	30	85,642	57,791	
1,2,3,4,6,7,8,9 - OCDD	E	29	587,965	516,436	0,336
	C	30	479,067	327,057	
Total PCDD <sub>s</sub>	E	29	788,92	602,20	0,309
	C	29	649,56	412,67	

N number of subjects per group  
 $\bar{x}$  mean  
 SD standard deviation  
 NA not applicable  
 E exposed  
 C control  
 \* marker congeners for this PCB fire

Table VI  
 Analysis of the effect of exposure duration on  
 PCDF and PCDD plasma-lipid-based concentrations (ng/kg)

Group	Status	N	$\bar{x}$	SD	p
Σ TePeHx-CDF	E (h)	18	35.11	15,296	0,47
	E (m)	11	27.66	10,391	
	C	30	36.44	14,568	
	All	59	34.4	14,29	
Σ PCDF	E (h)	18	62.65	25.58	0.141
	E (m)	11	46.57	12.18	
	C	30	60.44	23.34	
	All	59	58.53	22.90	
Σ PCDD	E (h)	18	929.49	712.387	<0.0001
	E (m)	11	558.89	242.76	
	C	30	649.56	412.67	
	All	59	719.24	516.47	

N number of subjects per group  
 $\bar{x}$  mean  
 SD standard deviation  
 E (h) : heavily-exposed  
 E (m) : moderately-exposed  
 C : control

Table VII  
 Correlation matrix of selected variables

Correlations	Σ TePeHx-CDF	Σ PCDF	Σ PCDD
Σ TePeHx-CDF	1.0000	.9026*	.5954*
Σ PCDF	.9026*	1.0000	.7774*
Σ PCDD	.5954*	.7774*	1.0000
Age	-.0515	-.0490	.1708
Weight	.0062	.0495	.0891
Tobacco consumption	-.1237	-.0877	-.0700

\* p < 0.001

Table VIII  
TCDD-equivalent weighting factors

Congener	Weighting factors	
	EPA 1987	NATO-CCMS 1988
<b>FURANS</b>		
2,3,7,8 - TCDF	0,1	0,1
1,2,3,7,8 - PeCDF	0,1	0,05
2,3,4,7,8 - PeCDF	0,1	0,5
1,2,3,4,7,8-HxCDF	0,01	0,1
1,2,3,6,7,8-HxCDF	0,01	0,1
2,3,4,6,7,8-HxCDF	0,01	0,1
1,2,3,7,8,9-HxCDF	0,01	0,1
1,2,3,4,6,7,8-HpCDF	0,001	0,01
1,2,3,4,7,8,9-HpCDF	0,001	0,01
1,2,3,4,6,7,8,9-OCDF	0	0,001
<b>DIOXINS</b>		
2,3,7,8-TCDD	1	1
1,2,3,7,8-PeCDD	0,5	0,5
1,2,3,4,7,8/1,2,3,6,7,8-HxCDD	0,04	0,1
1,2,3,7,8,9-HxCDD	0,04	0,1
1,2,3,4,6,7,7-HpCDD	0,001	0,01
1,2,3,4,6,7,8,9-OCDD	0	0,001

Table IX  
TCDD equivalent dose (ng/kg) in exposed and control subjects

Congeners		EPA 1987				NATO-CCMS 1988			
		N	$\bar{x}$	SD	p	N	$\bar{x}$	SD	p
Σ PCDF	E	29	1,68	0,75	0,176	29	7,20	3,15	0,575
	C	30	2,18	0,93		30	7,65	2,95	
Σ PCDD	E	29	10,52	4,64	0,276	29	17,18	7,61	0,341
	C	29	9,36	3,30		29	15,49	5,66	
Σ PCDF + Σ PCDD	E	29	12,40	5,24	0,507	29	24,38	9,88	0,641
	C	29	11,59	3,89		29	23,27	8,03	

N number of subjects per group  
 $\bar{x}$  mean  
 SD standard deviation  
 E exposed  
 C control