CLINICAL AND METABOLIC ABNORMALITIES IN OCCUPATIONAL EXPOSURE TO POLYCHLORINATED BIPHENYLS (PCBs)

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ABSTRACT

A cross-sectional study of 120 male workers was conducted to determine the prevalence of increased PCB absorption as well as the presence of potentially related clinical and metabolic abnormalities. Three exposure categories ("exposed", "nominally exposed", "non-exposed") were defined. Complete work histories, clinical histories, physical exams and lab tests were obtained including PCB plasma level determinations. In addition, PCB fat levels were determined in a portion of each exposed group.

Findings of dermatoxicity were observed and elevated PCB levels more frequently noted in the exposed group ($p<0.0001$) correlating well with age and duration of employment. These correlations were stronger for fat ($p<0.001$) than for plasma ($p<0.01$) PCB levels. In the exposed group, a significant correlation was found between PCB plasma and serum triglyceride ($p<0.0001$) and SGOT levels ($p<0.01$). These correlations remained significant after controlling for either age or length of employment. No significant correlations were found between PCB levels and cholesterol, HDL cholesterol or liver function tests other than SGOT.

Further analyses relating frequency of reported direct contact with PCB levels suggested a dermal route of exposure. An analysis by union affiliation demonstrated that crafts with greater direct exposure had correspondingly higher elevations of PCB levels.

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BACKGROUND

Polychlorinated biphenyls (PCBs) comprise a group of synthetic chlorinated hydrocarbon compounds with considerable industrial utility based on their resistance to oxidation and dielectric properties. They have been used world-wide in capacitors and transformers and many other applications described elsewhere (1). The contamination of the general environment as well as the work-place by PCBs was appreciated only recently leading to the ban of their continued manufacture under the Toxic Substances Control Act of 1976 (2). This action was promoted by greater awareness of animal toxicity studies (3), the stability and persistence of PCBs in the environment, the tendency of PCBs to accumulate in human tissues (4,5) and the documented chronic toxicity of PCBs in humans following accidental oral ingestion in the "Yusho" incident (6).

In spite of legislative and regulatory actions to phase out the use of PCBs, solutions to problems of effective disposal have not been forthcoming. In 1977 Hecht (7) estimated that of the 1.25 billion pounds produced in this country since the 1930s, 750 million pounds were still in use and 450 million pounds had found their way into the general environment. A review (3) by the National Institute for Occupational Safety and Health (NIOSH) of clinical and metabolic abnormalities in both environmentally and occupationally exposed groups revealed inconsistent relation-ships between tissue PCB levels and abnormal findings. A more recent publication (9) suggests alterations of lipid metabolism at PCB levels less than those usually reported in association with findings of clinical toxicity.

An opportunity to re-examine these relationships was recently provided in a railroad passenger car and locomotive maintenance facility at which some employees have been exposed to PCB-containing transformer fluids (Pyranol, Inerteen, Askerel) for nearly 40 years. Inasmuch as the potential health hazards of PCBs were not apparent until the past few years, the handling of transformer fluids at this facility was on a casual basis with few or no special handling procedures in effect for the greater part of this exposure period. A change in management followed by the documentation of elevated PCB levels in the work environment in early 1979 provided the impetus for developing more conservative handling procedures as well as a medical monitoring program which included the design and execution of an epidemiologic study herein described. It was the purpose of this study to determine the prevalence of increased PCB absorption among exposed workers as well as the prevalence of findings compatible with PCB toxicity. In addition two control groups were defined with either nominal or non-exposure against which comparisons could be made. Lastly, through detailed work histories, an attempt was made to determine those factors most predictive of elevated PCB levels as well as to shed light on the most probable route of exposure.

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MATERIALS AND METHODS

One hundred and twenty male employees participated in the study according to the following exposure groups:

Group I -- Exposed (86 subjects)

Group II -- Nominally Exposed (15 subjects)

Group III -- Non-Exposed (19 subjects)

Subjects within each of these three groups were randomly selected by a computer program from the corresponding underlying populations. Group I subjects were defined as "exposed" on the basis of their frequent opportunity for direct contact with PCB-containing transformer fluids in their assigned shop areas. Group II subjects were defined as "nominally exposed" on the basis of their employment at the same facility but without the opportunity (work assignment) characterizing Group I subjects. Group III subjects were defined as "non-exposed" on the basis of the absence of PCB-containing fluids in their work environment and were employed elsewhere. All three groups reside in the same geographic region.

All participants completed a detailed work history questionnaire in which tasks entailing potential exposure to PCB-containing fluids were characterized and quantified. Information related to potential exposure to other toxic substances was also obtained. Complete medical histories and physical examinations were performed on all participants. A comprehensive battery of clinical laboratory tests were performed in the fasting state (12 hours) as outlined in Table 1. Plasma PCB levels were determined on all subjects. In addition, 50 randomly selected subjects (36 in Group I, 5 in Group II and 9 in Group III) underwent subcutaneous fat biopsies upon which fat PCB levels were determined. Analytical determinations for PCBs in heparinized human plasma and fat specimens were performed as specified by EPA methods (10). In brief, hexane extraction was followed by specified silica gel and/or florisil cleanup to remove pesticides. The isolated PCB fraction was assayed by electron capture gas chromatography according to Webb/McCall (11). All of the laboratory testing was performed by Professional Clinical Laboratories, Inc. in Wilmington, Delaware. This laboratory participated in the Center for Disease Control (CDC) trial PCB testing program conducted during the course of this study.

Student's t-tests were used to compare average values of selected variables among the three groups. Correlation between selected variables in Group I was assessed using Pearson's simple correlation coefficients, and partial correlation coefficients to adjust for confounding variables. The chi-square procedure was used to test for association in the contingency tables.

RESULTS

Table 2 shows the age distribution of the three groups. The two exposed groups are older than the non-exposed group. The average age for the exposed group was 41.4 years; the nominally exposed group 37.2; and the non-exposed group 30.7. The difference in average age between the exposed group and the non-exposed group is statistically significant ($p < 0.05$). This age difference should be taken into consideration when comparisons between the groups are made, since many of the parameters vary with age.

Table 3 presents the distribution of length of employment by exposure category. The average lengths of employment were 17 years, 3 years and 9 months, and 4 years and 3 months for the exposed, nominally exposed, and non-exposed groups, respectively. The difference between the exposed group and the nominally exposed group or the non-exposed group is highly significant ($p < 0.0001$). This was not surprising in view of the traditionally stable crafts represented by the exposed group.

Review of medical histories and physical findings failed to reveal findings of organ toxicity in the exposed group although several cases of chloracne were encountered in this group with none being found in the other groups. Other dermatological findings were more prevalent in the exposed group as well, but no significant association between these and PCB levels could be demonstrated within the group. Historically, there was a common reporting in Group I of transient skin and eye irritation attributed to accidental spills in which there was direct contact with PCB-containing fluids.

The averages of some selected biochemical parameters by exposure category are shown in Table 4. There is no statistically significant difference among the groups in triglycerides, cholesterol, HDL, SGOT, SGPT. For γ-glutamyl transpeptidase (γGTP), the highest value, 37.1 IU/ℓ, was reported among the exposed employees, the intermediate value, 34.1, among the nominally exposed, and the lowest value, 29.8, among the non-exposed: an indication of a trend by exposed category. However, because of the considerable variabilities in the measurement and the relatively small sample sizes of both the nominally exposed and the non-exposed groups, these differences are not statistically significant.

For PCB levels, the exposed group showed a clear elevation when compared to either the nominally exposed or the non-exposed groups (Table 5). The average plasma PCB level (33.4 ppb) of the exposed group was more than twice that of either the nominally exposed group (14.2 ppb) or the non-exposed group (12.0 ppb). The differences in plasma PCB level between the exposed group and the other two groups are highly significant ($p < 0.0001$), but the difference between the nominally exposed and the non-exposed group is not statistically significant. The discrepancy in fat PCB level for the exposed group was 5.6 ppm, more than three

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times that of either the nominally exposed (1.4 ppm) or the non-exposed group (1.3 ppm). Again the difference between Group I (exposed) and either Group II (nominally exposed) or Group III (non-exposed) is highly significant ($p < 0.0001$), but that between Group II and Group III is not significant.

In an attempt to determine any association or correlation between PCB exposure and other variables in Group I, Pearson's correlation coefficients were calculated. Similar analyses were not performed for Groups II or III as the PCB levels were well within the normal range (12,13) and the sample sizes for these groups were relatively small. Table 6 shows the correlation coefficients and the corresponding p values for selected biochemical parameters, length of employment and age for the exposed group. Apart from the obvious expected association between length of employment and age, significant correlations were found between length of employment and plasma PCB level, as well as between length of employment and fat PCB level. However, the correlation between length of employment and fat PCB level ($r = 0.54$, $p < 0.001$) was stronger than that between length of employment and plasma PCB level ($r = 0.27$, $p < 0.01$). Similar significant correlations were observed between age and plasma PCB as well as fat PCB levels. A discussion on the possible confounding effect between age and length of employment is given below.

Also in Group I, statistically significant correlation was demonstrated between plasma PCB levels and triglycerides ($r = 0.56$, $p < 0.0001$) but not between fat PCB levels and triglycerides. Plasma PCB levels were also significantly correlated with SGOT ($r = 0.28$, $p < 0.01$) but not with SGPT or γGTP. Fat PCB levels were not significantly correlated with SGOT, SGPT, or γGTP.

Since many of the variables examined in Table 6 are interrelated, partial correlation coefficients, which take into account confounding factors, were calculated and are presented in Table 7. The correlation between plasma PCB levels and triglycerides remained highly significant even after adjusting for either age or length of employment ($r = 0.54$ and 0.55 respectively, $p < 0.001$). On the other hand, there was no significant correlation between fat PCB levels and triglycerides, adjusting for age or length of employment. Among the liver function parameters, SGOT was found to remain significantly correlated with plasma PCB levels even after adjusting for age ($r = 0.26$, $p < 0.05$).

To determine the possible relationship between the frequency of direct contact with PCB-containing fluids and PCB levels, all 120 subjects in the study were asked whether or not they ever had direct contact with such fluids and, if so, to report the frequency with which this had occurred. Table 8 shows the crosstabulation of plasma PCB levels and reported frequency of direct contact. The figures clearly demonstrate a direct relationship between the two parameters. Among those who reported no direct contact with PCB-containing fluids, 41% had PCB plasma levels less than or equal to 10 ppb, whereas only 4% of those with frequent contacts had plasma PCB levels in this range. On the other hand, only 3% of those who reported no direct contact had plasma PCB levels greater than 30 ppb, compared to 44% among those with frequent such contacts. This association is highly significant (chi-square = 28.83, $p < 0.005$). Table 9 demonstrates a similar relationship between fat PCB levels and reported frequency of direct contact.

As it was evident from the work histories that certain crafts inherently provided greater opportunity than others for direct contact with PCB-containing fluids, an attempt was made to correlate plasma PCB levels with union affiliation for those subjects employed at the maintenance facility (Groups I and II). The result was highly significant (chi-square = 42.89, $p<0.001$) and is presented in Table 10. Similar calculations were not performed for fat PCB levels as the number of subjects with recorded levels was too small to permit same.

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DISCUSSION

In this study Groups I (exposed) and II (nominally exposed) consisted of male subjects employed at a railroad passenger car and locomotive maintenance facility, whereas Group III (non-exposed) subjects were employed elsewhere. All subjects were of the same sex, shared the same employer, and lived in the same geographic area. The greater length of employment noted in Group I subjects was attributed to the traditional stability in the crafts represented by that group. These employees have remained at the same facility for a mean duration of 17 years with a range of a few months to 40 years.

Comparable occupational PCB exposure studies have variously shown findings of dermatotoxicity, hyperpigmentation and liver function abnormalities but without consistent relationships to PCB levels when measured (8). The transient symptoms of skin and eye irritation associated with acute exposures, as well as the occasional finding of chloracne reported in this study, are in keeping with these other studies. A recent comprehensive study of capacitor manufacturing workers by Fischbein et al. (14) was similarly notable for its relative absence of physical findings outside of a high prevalence of dermatologic abnormalities in spite of a mean plasma PCB level of 172 ppb (n=289). Even more recently Baker et al. (9) reported no symptoms or physical findings of organ toxicity in a different group of PCB exposed capacitor workers whose mean plasma PCB level was 75.1 ppb (n=18).

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Although no evidence of overt hepatotoxicity was evident in the exposed group, the association between plasma PCB levels and SGOT was statistically significant, a finding which has been recently reported by others (14). It is tempting to incriminate the liver as a target organ for PCB exposure responsible for both the triglyceride and SGOT abnormalities as has been suggested in animal studies (16), but this requires further testing of alternative hypotheses.

The demonstrated relationship between PCB levels and reported frequency of direct contrast with PCB-containing fludis suggests a dermal route of exposure. As PCBs generally exhibit low vapor pressures (19), an airborne route of exposure is not likely unless the PCB-containing fluids are heated to a significant degree. There is thus theoretical support for the use of suits impervious to PCBs in protecting the worker from further exposure to PCBs and the potential, yet still unknown, risks for same.

Those crafts with known direct exposure to PCBs are at greater risks for PCB absorption as has been shown earlier (Table 10). The predominant crafts in this study are represented by the following

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unions:

BMWE -- Brotherhood of Maintenace of Way Employees

BRAC -- Brotherhood of Railway and Airline Clubs

JCC -- Joint Council of Carmen

IAM -- International Association of Machinists

SMWIA - Sheet Metal Workers International Association

IBEW -- International Brotherhood of Electrical Workers

Analyses of this type provide a rational basis for the assignment of protective equipment and the development of appropriate handling procedures for PCB-containing fluids.

(8) National Institute for Occupational Safety and Health: Criteria for a Recommended Standard...Occupational Exposure to PCBs, NIOSH Pub. No. 77-225, Washington, D.C. Government Printing Office, 1977.

(9) Baker EL, Landrigan PJ, Glueck CJ, et al: Metabolic consequences of exposure to polychlorinated biphenyls (PCBs) in sewage sludge. Am J. Epidemiology 112: 553-563, 1980.

(10) EPA 600/8-80-038 6/80 Manual of Analytical Methods for the Analysis of Pesticides in Humans and Environmental Samples. U.S. EPA Health Effects Research Lab, Research Triangle Park, N.C. 27711. Ed. by R.R. Watts.

(11) R.G. Webb and A.C. McCall, J. of Chromatographic Sciences 11:366-373, 1973.

(12) Price HA, Welch RL: Occurrence of polychlorinated biphenyls in humans. Environ Hlth Perspective 1:73-78, 1972.

(13) Yobs, AR: Levels of polychlorinated biphenyls in Adipose tissue of the general population of the nation, Environ Hlth Perspective 1:79-81, 1972.

(14) Fischbein A, Woolf MS, Libis R, et al: Clinical findings among PCB-exposed capacitor manufacturing workers. Ann NY Acad Sci 320:703-715, 1979.

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(15) Ouw Hk, Simpson GR, Siyali Os: Use and health effects of Arochlor 1242, a polychlorinated biphenyl, in an electrical industry. Arch Env Health 31:189-194, 1976.

(16) Holub Bj, Pierkarski J, Nillson K: The effect of a PCB (2,4,2',4'-tetra-chlorobiphenyl) on lipid synthesizing enzymes in rat liver microsomes. Bull Environ Contam Toxicol 14:415, 1975.

(17) Kuratsune, M, Masuda Y, Nagayama J: Some of the recent findings concerning Yusho, in Proceedings of a National Conference on Polychlorinated Biphenyls. EPA-560/6-75-004, Washington, D.C. Environmental Protection Agency. March 1976, pp. 14-29.

(18) Carlson, LA, Kolmadin-Hedman B: Hyper-alpha-lipoproteinemia in men exposed to chlorinated hydrocarbon pesticides. Acta Med Scand 192:29-32, 1972.

(19) National Institute for Occupational Safety and Health, Health Hazard Evaluation Determination Report No. 76-52-386, April, 1977.

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Table 1. - Laboratory Tests Determined in Study Subjects

Complete Blood Count	A/G Ratio
Urinalysis	Total Bilirubin
Glucose	SGPT
BUN	SGOT
Uric Acid	LDH
Creatinine	γ-Glutamyl Transpeptidase
Calcium	Triglycerides
Phosphorous	Cholesterol
Alkaline Phosphatase	HDL Cholesterol
Total Protein	LDL Cholesterol
Albumin	VLDL Cholesterol
Globulin	Phenotype

Table 2. - Age Distribution of 120 Workers by PCB Exposure Category

Age (years)	Exposed No.	Exposed %	Nominally Exposed No.	Nominally Exposed %	Non-exposed No.	Non-exposed %
15-19	1	1.2	0	0	2	10.5
20-24	14	16.3	1	6.7	3	15.8
25-29	17	19.8	2	13.3	6	31.6
30-34	5	5.8	6	40.0	4	21.1
35-39	7	8.1	1	6.7	2	10.5
40-44	6	7.0	2	13.3	0	0
45-49	2	2.3	1	6.7	1	5.3
50-54	11	12.8	1	6.7	0	0
55-59	17	19.8	1	6.7	1	5.3
60+	6	7.0	0	0	0	0
Total (N)	86	71.7	15	12.5	19	15.8
Average	41.4		37.2		30.7	

Table 3. - Distribution of Length of Employment of 120 Workers by PCB Exposure Category.

Length of Employment (years)	Exposed No.	Exposed %	Nominally Exposed No.	Nominally Exposed %	Non-Exposed No.	Non-Exposed %
<5	30	34.9	13	86.7	17	89.5
5- 9	20	23.3	1	6.7	1	5.3
10-14	4	4.7	0	0	0	0
15-19	0	0	1	6.7	0	0
20-24	0	0	0	0	0	0
25-29	3	3.5	0	0	0	0
30-34	3	3.5	0	0	0	0
35-39	19	22.1	0	0	1	5.3
40-44	6	7.0	0	0	0	0
45+	1	1.2	0	0	0	0
Total (N)	86	71.7	15	12.5	19	15.8
Average	17.0		3.8		4.3	

Table 4. - Averages of Selected Biochemical Parameters in 120 Workers by PCB Exposure Category

Parameter	Exposed	Nominally Exposed	Non-Exposed
Triglycerides (mg%)	135.8	128.7	145.5
Cholesterol (mg%)	231.7	238.3	238.2
HDL Cholesterol (mg%)	58.8	57.2	61.7
SGOT (IU/ℓ)	19.6	19.1	19.4
SGPT (IU/ℓ)	21.7	21.6	21.3
γGTP (IU/ℓ)	37.1	34.1	29.8

Table 5. - Averages and Ranges of Plasma and Fat PCB Levels in 120 Workers by PCB Exposure Category.

	Exposed	Nominally Exposed	Non-Exposed
Plasma PCB Levels (ppb*)			
Average	33.4	14.2	12.0
Range	10-312	10-30	10-27
Fat PCB Levels (ppm**)			
Average	5.6	1.4	1.3
Range	1.0-21.6	1.0-1.8	1.0-2.0

*parts per billion
**parts per million

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Table 7. - Selected Partial Correlation Coefficients and Probabilities for Selected Variables in 86 Workers Exposed to PCBs.

Variables	Coefficient	Probability
Plasma PCB level and triglycerides adjusting for age	.54	<.001
Plasma PCB level and triglycerides adjusting for length of employment	.55	<.001
Fat PCB level and triglycerides adjusting for age	.20	ns*
Fat PCB level and triglycerides adjusting for length of employment	.23	ns
Plasma PCB level and SGOT adjusting for age	.26	<.05

*ns = not significant

Table 8. - Relationship Between Plasma PCB Levels and Frequency of Reported Direct Contact with PCB-Containing Fluids

Frequency of Reported Direct Contact		Plasma PCB Levels (ppb) ≤10	11-20	21-30	≥31	
	Never	16 (41%)*	17 (44%)	5 (13%)	1 (3%)	39 100%
	Rarely	7 (30%)	7 (30%)	6 (26%)	3 (13%)	23 100%
	Occasionally	5 (15%)	9 (27%)	9 (27%)	10 (30%)	33 100%
	Frequently	1 (4%)	8 (32%)	5 (20%)	11 (44%)	25 100%
		29	41	25	25	120

chi-square = 28.83, $p < .005$

*The figures in parentheses are row percentages.

Table 9. - Relationship Between Fat PCB Levels and Frequency of Reported Direct Contact with PCB-Containing Fluids

Frequency of Reported Direct Contact	Fat PCB Levels (ppm) <1.5	1.51-2.25	>2.26	
Never	8 (73%)*	2 (18%)	1 (9%)	11 100%
Rarely	5 (42%)	1 (8%)	6 (50%)	12 100%
Occasionally	5 (31%)	1 (6%)	10 (63%)	16 100%
Frequently	2 (18%)	0 (0%)	9 (82%)	11 100%
	20	4	26	

chi-square = 13.06, $p < .05$

*The figures in parentheses are row percentages.

Table 10. - Relationship Between Plasma PCB Levels and Union Affiliation of Workers at Maintenance Facility

Union Affiliation **		Plasma PCB Levels (ppb)				
		≤10	11-20	21-30	≥31	
	BMWE	5 (56%)*	2 (22%)	2 (22%)	0 (0%)	9 100%
	BRAC	2 (50%)	1 (25%)	1 (25%)	0 (0%)	4 100%
	MISC	4 (57%)	2 (29%)	0 (0%)	1 (14%)	7 100%
	JCC	1 (7%)	4 (27%)	8 (53%)	2 (13%)	15 100%
	IAM	4 (12%)	15 (45%)	7 (21%)	7 (21%)	33 100%
	SWMIA	0 (0%)	2 (29%)	2 (29%)	3 (43%)	7 100%
	IBEW	1 (4%)	9 (35%)	4 (15%)	12 (46%)	26 100%
		17	35	24	25	101

chi-square = 42.89, p<.001

* The figures in parentheses are row percentages.

** See text.