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# Assessing the Health Effects of Potential Exposure to PCBs, Dioxins, and Furans from Electrical Transformer Fires: The Binghamton State Office Building Medical Surveillance Program

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**ABSTRACT. A medical surveillance program has been established for 482 persons who were potentially exposed to polychlorinated biphenyls (PCBs), dibenzo-*p*-dioxins, and dibenzofurans from an electrical transformer fire in a Binghamton, NY office building in 1981. Blood samples were analyzed for serum concentrations of PCBs and for biochemical and hematologic parameters at the time of the fire and 9 to 12 mo later. Firefighters and individuals who were in the building for 25 hr or more were also asked about post-fire symptomatology and examined after 1 yr for disorders of the skin, eyes, liver, and neurologic system. The results indicated that reported exposure was positively related to mean serum PCB levels ($p = .004$). The means and individual values, however, were within the range reported by other studies of persons with no unusual exposures. Significant correlations were observed between serum PCB concentrations and levels of liver enzymes and lipids, but mean levels of these biochemical parameters were not associated with reported exposure after adjustment for relevant covariables. Approximately one-half of those examined had skin lesions, but no cases of chloracne were detected, and there was no clinical evidence of any other exposure-related systemic disorder. The data suggest that exposure to contaminants from the building did not result in substantial absorption or cause any major short-term health effects.**

POLYCHLORINATED BIPHENYLS (PCBs), polychlorinated dibenzo-*p*-dioxins (PCDDs), and polychlorinated dibenzofurans (PCDFs) are aromatic hydrocarbons whose human health effects include chloracne,[1-3] hepatomegaly,[4] peripheral neuropathies,[5-6] ocular disorders,[7] emotional and psychological disturbances,[8] and elevated blood levels of liver enzymes and lipids.[9-11] Malignant neoplasms and congenital malformations have been induced among experimental animals by the administration of these chemicals,[12-14] but the evidence concerning human carcinogenicity and teratogenicity is equivocal.[15-18]

Most of these data have been gathered from studies of industrial workers occupationally or accidentally exposed. Other investigations include those in Japan and Taiwan of mass poisonings which occurred when persons ingested rice oils containing PCBs and PCDFs,[5,7,19] and those in Italy of the consequences of widespread environmental contamination by PCDDs from an industrial accident near Seveso.[6,20] This report describes a program to assess the health risks of potential exposure to all three chemicals from an office building electrical transformer fire. We present our methods and findings after 1 yr of medical screening, focusing on short-term symptoms and clinical disorders of the skin, eyes, liver, and neurologic system and on blood chemistries.

**Background.** The State Office Building is an 18-story structure in the center of Binghamton (Broome County), NY. At 5:30 A.M. on February 5, 1981, a fire, in conjunction with several explosions, occurred in the basement mechanical room. It originated in the switch gear of the secondary electrical power distribution system. Approximately 180 gallons of askarel, a dielectric fluid composed of 65% PCBs (Aroclor 1254) and 35% polychlorinated benzenes, leaked from a transformer. Pyrolysis of the askarel led to the formation of a fine oily soot which was spread throughout the building via two ventilation shafts.

Samples taken several days after the fire showed that the average concentration of PCB-1254 in air was 1.5 $\mu g/m^3$.[21] The average result for surfaces ranged from 4.6 $\mu g/m^2$ to 162.2 $\mu g/m^2$, depending on whether the area was open or enclosed and horizontal or vertical. In contrast, the levels found in outside air and on surfaces in neighboring buildings were generally at or below detection limits. Soot samples also were analyzed for pyrolysis products of PCBs and polychlorinated benzenes. The 2,3,7,8 isomer of tetrachlorodibenzo-*p*-dioxin (TCDD), the most toxic form of PCDD, was identified with an average concentration of 3 parts per million (ppm), and its PCDF counterpart, 2,3,7,8-tetrachlorodibenzofuran (TCDF), was measured at an average level of 199 ppm.[22]

Restoration efforts were suspended on February 26, 1981, after TCDD and TCDF were found in the soot samples. Further entry was allowed only with very stringent safeguards. This action alleviated concerns about exposure after February 26, but the possibility of adverse health effects remained for the firefighters, police officers, clean-up workers and other persons who had entered before that date. In addition, the building could not remain indefinitely in a contaminated state, although it was clear that a successful restoration would be very difficult, costly, and time-consuming.

Resolution of these questions regarding worker health and building safety required the establishment of medical and environmental monitoring programs. An advisory panel of 15 scientists from North America and Europe was formed to help in the design and implementation of a plan to clean and reoccupy the building. They also suggested that a medical surveillance plan be developed for the persons who had entered the building before it was closed. The National Institute of Occupational Safety and Health directs the program, while the New York State Department of Health administers it.

## Methods

**Program participants.** Individuals who entered the State Office Building on or after the day of the fire formed the target population. Participants were recruited through a variety of means, including direct contact by medical staff of the Broome County Health Department, letters to companies and agencies which were known to have employed persons who had entered the building, and announcements in the news media. These efforts identified 327 persons who had been in the building for a few minutes to more than a thousand hours. The median length of stay was 8 hr.

The medical surveillance program also includes 155 persons who did not enter the building. These individuals were primarily city and county employees who worked in the area and were concerned about possible exposure through secondary sources. The total number of participants therefore was 482. Their mean age at the time of the fire was 41 yr, 84% were male, and 98% were white.

**Interview data.** Four hundred and fifty persons (93%) were interviewed 6-12 mo after the fire to obtain data on the nature and extent of exposure, sociodemographic characteristics, medical problems present before the fire, alcohol consumption, cigarette smoking, and previous and subsequent occupational exposures. The resultant information updated and was merged with similar data gathered by the Broom County Health Department soon after the fire.

**Clinical laboratory data.** A follow-up blood screen was conducted 9-12 mo after the fire on all persons. It consisted of tests for 20 biochemical parameters.* A complete blood count with platelets and differential also was performed. Four hundred and thirty persons (89%) participated. Each was instructed to fast for 12 hr before venipuncture.

Similar tests had been ordered previously by the Broome County Health Department for 431 persons (initial blood screen). Approximately 15% of those who were in the State Office Building had this specimen taken before entry, while the median for the others was 6 days after entry. Because of the emergency atmosphere at the time of the fire, some participants had their first blood sample drawn despite the fact that they had not fasted. The results regarding triglycerides may be uninterpretable for such specimens.

All but 5 individuals took part in at least one of the blood screens, and 383 persons (79%) participated in both. The laboratory of Binghamton General Hospital analyzed the samples from each screen, and their reference ranges were used to determine if a test result was elevated or depressed.

*Glucose, blood urea nitrogen (BUN), creatinine, total and direct bilirubin, total protein, albumin, uric acid, alkaline phosphatase, serum glutamic oxalacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), gamma glutamyl transpeptidase (GGTP), lactic dehydrogenase (LDH), cholesterol, triglycerides, calcium, phosphorus, acid phosphatase, serium iron, and total iron binding capacity.

**Serum PCB determinations.** A total of 771 serum samples from 470 program participants (98%) were analyzed by a commercial laboratory for PCB-1254. Included were sera saved from the follow-up blood screen for each of the 430 persons who participated and from the initial blood screen for the 341 individuals who had available 2.5 ml of serum, the minimum quantity for analysis. Three hundred and one individuals (62%) had samples from both screens analyzed for PCB level. The specimens were prepared and analyzed using standard techniques.[23] The method of quantitation was that of Webb and McCall,[24] and the detection limit was one part per billion (ppb).

Our quality assurance program consisted of randomly inserting among the 771 serum samples 168 blind and bench control specimens which were mixed in exact proportion with the serum of a PCB-fed sheep to contain 10, 20, 30, or 50 ppb of PCB-1254. The bias, as determined by the difference between the mean and target values, varied in magnitude from 0.4% for the 30 ppb control samples to 9.1% for the 10 ppb group. The precision, as indicated by the coefficient of variation, ranged from 12.1% for the 20 ppb samples to 19.9% for the 50 ppb controls. These figures are similar to those reported by Lawton et al.,[25] who, in their study of the comparability and precision of serum PCB measurements, used the same commercial analyst.

**Symptomatology and physical examinations.** Firefighters and persons who were in the building for 25 hr or more were also questioned about symptoms and examined for physical abnormalities 9-12 mo after the fire. The rationale was that this group would be the most likely to experience adverse health effects if any occurred. The total eligible for this assessment was 156 persons, and 147 (94%) took part.

Staff physicians and nurses from Binghamton General Hospital administered the 34-item symptom checklist and conducted the 38-item examination. Individuals with rashes suggestive of chloracne were referred to a dermatologist from the Yale University Occupational Health Clinic in New Haven, CT, for a more definitive diagnosis. Nerve conduction velocity tests were not included in the study design, but persons with neurologic conditions were referred to a board-certified neurologist in Binghampton for further evaluation.

**Exposure index.** We developed an index to quantify the duration and intensity of each person's reported exposure to the State Office Building. It was a function of five factors: (1) location in building, (2) activity in building, (3) use of protective gear, (4) total number of hours in building, and (5) PCB-1254 air levels for the days a person was in the building.

Each factor was weighted by a number which reflected its relative magnitude, and the five weights were multiplied to obtain a total exposure score. The 155 persons (33%) who were not in the State Office Building were assigned a score of zero. Because the index probably does not measure degree of reported exposure on an interval scale, we grouped the scores into three categories for most analyses. The first category was comprised of persons who did not enter the building. They were considered the "low" exposure class. The remainder were divided along the median to form two other categories, the "medium" ($N$ = 156) and "high" ($N$ = 162) exposure classes.

**Statistical analysis.** We used analysis of variance (ANOVA) with repeated measures to compare mean serum levels of PCB and of the biochemical and hematologic parameters across the three exposure categories initially and at follow-up.[26] Pairwise contrasts were performed to identify which groups were different, and trend analysis was conducted by partitioning the variation in serum PCB due to exposure into its linear and nonlinear components. Since persons in the low exposure category were not asked about symptoms or examined and because we wished to maximize our ability to detect significant differences given small cell frequencies, we employed $t$ tests to compare the mean serum PCB levels and exposure index scores of persons with and without symptoms or abnormal physical findings instead of calculating risk ratios. Covariance adjustment was used in the ANOVA and $t$ tests to statistically control for the effect of potential confounders. Multiple linear regressions of the laboratory tests at each time period and of change in these variables over time on serum PCB were also performed. To control for possible confounding, covariables were added to the regression models before the effect of serum PCB was estimated.[27]

The serum PCB and clinical laboratory data were logarithmically transformed to normalize the residuals and stabilize the variances for significance testing. We report the data, however, in their original units to facilitate interpretation. Since it assumes a less restrictive form for the covariance matrix, multivariate analysis of variance (MANOVA) was performed in addition to repeated measures ANOVA.[28] The results of the two procedures were essentially identical. SPSSX and BMDP computer programs were used.[29,30] A finding was considered statistically significant if its two-tailed probability level equalled or was less than .05.

## Results

**Serum PCB.** Figure 1 displays initial and follow-up serum PCB concentrations as percentage barcharts. The means for each time period were respectively 6.90 and 6.50 ppb, with standard deviations of 3.52 and 4.12 ppb. Initially, the values ranged from 1.2 to 21.4 ppb, while at follow-up, the minimum was less than 1 and the maximum was 47.0 ppb. A total of 10 persons (2.1%) had one determination of at least 20 ppb.

Table 1 shows mean serum PCB concentrations by exposure category and time period with and without adjustment for age, sex, race (white or non-white), social class,[31] alcohol consumption (drink-years), cigarette smoking (pack-years), and reported occupational exposures to PCBs or PCDDs. Included were 258 persons who had serum PCB assessed at both time periods, complete data concerning exposure and the covariables, and an initial blood sample which was taken after they had first entered the State Office Building.

After covariance adjustment, the main effect for exposure was significant ($p$ = .004) and according to trend analysis primarily linear ($p$ < .001). Pairwise contrasts

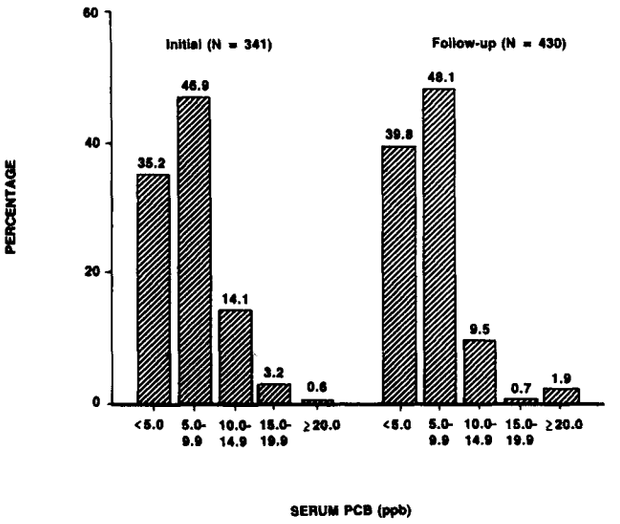


**Fig. 1. Percentage distribution of concentrations of serum PCB according to time period.**

showed that the adjusted mean serum PCB level of the high group was significantly greater than that for the low ($p$ = .001) and medium ($p$ = .046) categories, but the latter two were not significantly different from each other. The increment due to exposure in the proportion of variance explained was 3.85%. The main effect for time period and the interaction of exposure category and time period were nonsignificant. The results were unchanged when triglyceride level was added to the list of covariates to partially control for any differences in fasting.

Table 2 gives crude and adjusted initial and follow-up mean serum PCB concentrations according to whether the initial blood sample was drawn before, 1-4 days after, or 5 days or more after a participant first entered the State Office Building. The significant two-way interaction ($p$ = .001) indicated that separate analyses of the effect of when the first blood specimen was taken were necessary for each time period. They revealed that the three groups differed in adjusted mean serum PCB initially ($p$ = .001) but not at follow-up ($p$ = .953). Specifically, persons whose first blood sample was taken 1-4 days post-entry

**Table 1.—Crude and adjusted* mean concentrations of serum PCB (ppb), according to exposure category and time period**

| Exposure category† | N | Time period‡§ | | | |
|---|---|---|---|---|---|
| | | Initial | | Follow-up | |
| | | Crude | Adjusted | Crude | Adjusted |
| Low | 90 | 6.15 | 6.46 | 5.10 | 5.19 |
| Medium | 76 | 6.85 | 6.59 | 6.25 | 6.08 |
| High | 92 | 7.92 | 7.84 | 6.89 | 6.94 |

*Adjusted for age, sex, race, social class, previous and subsequent occupational exposures, alcohol consumption, and cigarette smoking.
†$F(2,185) = 5.60$, $p = .004$ for exposure category (adjusted).
‡$F(1,185) = 0.11$, $p = .916$ for time period (adjusted).
§$F(2,185) = 1.11$, $p = .333$ for exposure category × time period (adjusted).

**Table 2.—Crude and adjusted* mean concentrations of serum PCB (ppb), according to date of initial blood sample relative to entry and time period**

| Date of initial blood sample† | N | Time period‡§ | | | |
|---|---|---|---|---|---|
| | | Initial | | Follow-up | |
| | | Crude | Adjusted | Crude | Adjusted |
| Before entry | 25 | 8.01 | 7.91 | 7.01 | 6.72 |
| One to four days post-entry | 74 | 8.22 | 8.48 | 6.44 | 6.53 |
| Five days or more post-entry | 94 | 6.82 | 6.64 | 6.72 | 6.73 |

*Adjusted for age, sex, race, social class, previous and subsequent occupational exposures, alcohol consumption, and exposure index score.
†$F(2,127) = 1.93$, $p = .150$ for date of initial blood sample (adjusted).
‡$F(1,127) = 2.13$, $p = .146$ for time period (adjusted).
§$F(2,127) = 6.96$, $p = .001$ for date of initial blood sample × time period (adjusted).

had a significantly greater initial mean PCB level than those whose blood was drawn 5 days or more after entry ($p = .001$).

**Clinical laboratory data.** Descriptive statistics for the levels of liver enzymes and serum lipids are displayed by time period in Table 3. The person with the follow-up gamma glutamyl transpeptidase (GGTP) level of 580 IU/L also had a follow-up triglyceride concentration of 2,350 mg/dl. He was found to have pre-existing liver disease. Another individual with a follow-up triglyceride level of 1,175 mg/dl had a familial hyperlipidemia.

for time period, and the interaction of exposure and time period was also nonsignificant for each of these five tests.

**Symptoms and physical findings.** As Table 5 illustrates, almost one-half of the 147 examined firefighters and persons in the building for 25 or more hr reported at least one skin problem during the year after the fire. The median number of symptoms was two, and the maximum number was 19. Eight participants (5.4%) had at least one symptom in each of the four major systems.

Of the five participants who reported hepatitis or liver problems, four admitted that they were heavy consumers

**Table 3.—Descriptive statistics for liver enzymes and serum lipids, by time period**

| Time period | Liver enzyme/ lipid | *N* | X | *SD* | Min | Max | Median | % Elevated‡ |
|---|---|---|---|---|---|---|---|---|
| Initial | SGOT* | 430 | 26.0 | 12.8 | 6.1 | 168.0 | 23.6 | 10.5 |
| | SGPT* | 427 | 20.8 | 14.5 | 2.9 | 105.0 | 16.7 | 17.3 |
| | GGTP* | 426 | 20.5 | 17.4 | 1.0 | 96.0 | 15.0 | 10.3 |
| | Triglycerides† | 427 | 181.1 | 137.7 | 35 | 1,129 | 139 | 35.5 |
| | Cholesterol‡ | 427 | 213.6 | 46.0 | 97 | 420 | 209 | 1.6 |
| Follow-up | SGOT* | 430 | 24.9 | 13.5 | 5.9 | 204.0 | 22.8 | 9.3 |
| | SGPT* | 430 | 23.7 | 16.4 | 1.4 | 157.0 | 19.5 | 21.2 |
| | GGTP* | 428 | 25.2 | 35.1 | 2.2 | 580.0 | 17.0 | 11.7 |
| | Triglycerides† | 430 | 179.4 | 176.1 | 28 | 2,350 | 133.5 | 31.4 |
| | Cholesterol‡ | 430 | 226.7 | 46.3 | 105 | 475 | 223.5 | 4.0 |

Notes: SGOT = serum glutamic oxalacetic transaminase, SGPT = serum glutamic pyruvic transaminase, and GGTP = gamma glutamyl transpeptidase.
*Measured in IU/L.
†Measured in mg/dl.
‡Defined by contract laboratory as 36.0 IU/L for SGOT, 32.0 IU/L for SGPT, 42.0 IU/L for GGTP, 180 mg/dl for triglycerides, and 318 mg/dl for cholesterol.

Table 4 presents partial regression (*b*) and correlation (*r*) coefficients for serum PCB by time period from the multiple regression analyses of serum glutamic oxalacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), GGTP, triglycerides, and cholesterol on serum PCB, adjusting for age, sex, race, social class, alcohol consumption, cigarette smoking, and prior liver disease. The 250 persons who had serum PCB and these five biochemical parameters measured twice, who had complete data on the covariables, and whose initial blood sample was drawn after they first entered the building formed the subgroup analyzed. Serum PCB showed the strongest relationship to triglycerides, but it was also positively and significantly correlated with the four other biochemical parameters at each time period. When changes in liver enzymes or lipids were regressed on change in serum PCB over time, the only statistically significant result was that for triglycerides ($b = 0.211$, $p = .035$, $r = 0.170$).

Mean initial and follow-up levels of SGOT, SGPT, GGTP, triglycerides, and cholesterol did not differ significantly by exposure category after covariance adjustment. No statistically significant main effects were found

of alcohol. Their age-adjusted initial mean serum PCB level of 13.01 ppb was significantly greater than the age-adjusted mean of 8.09 ppb for individuals who did not report hepatitis or liver problems ($p = .026$). The group that complained of itching skin had a significantly higher ($p = .018$) age-adjusted mean exposure index score ($17.24 \times 10^3$) than those without that symptom ($11.54 \times 10^3$). Serum PCB and exposure index score did not vary significantly by any other symptom.

Table 6 summarizes the results of the physical examinations that were conducted 9-12 mo after the fire. More than one-half had at least one skin abnormality. The observed acne-like lesions consisted of open and closed comedones, pustules, papules, and scarring. The 15 persons with the most serious skin conditions were referred to the occupational dermatologist. His diagnoses included acne vulgaris, contact dermatitis, actinic keratoses, psoriasis vulgaris, and folliculitis. No cases of chloracne were detected.

The group with acne-like lesions had a significantly higher ($p = .009$) age-adjusted mean exposure index score ($15.19 \times 10^3$) than did persons without such lesions ($12.91 \times 10^3$). The age-adjusted initial mean

**Table 4.—Summary of multiple linear regressions of liver enzymes and lipids on serum PCB (ppb), according to time period (*N* = 250)***

| Liver enzyme/ lipid | Time period | | | |
|---|---|---|---|---|
| | Initial | | Follow-up | |
| | *b* | *r* | *b* | *r* |
| SGOT (IU/L) | 0.215† | 0.280 | 0.219† | 0.259 |
| SGPT (IU/L) | 0.379† | 0.285 | 0.336‡ | 0.228 |
| GGTP (IU/L) | 0.343‡ | 0.205 | 0.482† | 0.285 |
| Triglycerides (mg/dl) | 0.438† | 0.321 | 0.524† | 0.351 |
| Cholesterol (mg/dl) | 0.075§ | 0.167 | 0.127† | 0.292 |

*Adjusted for age, sex, race, social class, alcohol consumption, cigarette smoking, and prior liver disease.
†$p < .001$.
‡$p < .01$.
§$p < .05$.

PCB concentration of 12.56 ppb among those with hyperpigmentation was significantly greater than the age-adjusted mean of 8.17 ppb for persons without the condition ($p = .025$). Serum PCB and exposure index score did not vary significantly by any other physical finding.

Abnormalities of the eyes included conjunctival injection (2.0%), various pigment deposits at the rim of the disc and at the limbus (2.0%), and swollen eyelids (1.4%). Of the five individuals (3.4%) with some sensory impairment, two had evidence of a bilateral peripheral neuropathy. Both were insensitive to vibration at either ankle, but their upper extremities were normal. They were under 45 yr old, but one reported heavy alcohol consumption for 25 yr. The two persons diagnosed as having hepatomegaly also admitted to a history of heavy alcohol use.

The median number of abnormal physical findings was one, while four individuals (2.7%) had six. No participant had one or more abnormal findings in each of the four major systems.

## Discussion

The finding that adjusted serum PCB concentrations varied significantly and directly with degree of reported exposure is consistent with the hypothesis that association with the State Office Building elevated body burdens through absorption of the contaminants formed or released by the fire. A corollary is the prediction that mean serum PCB levels should have decreased significantly over time, at least among the medium and high exposure groups, since PCBs are preferentially stored in adipose tissue.[32] The data, however, did not support this prediction, i.e., the main effect for time period and the interaction of time period and exposure category were nonsignificant after covariance adjustment.

One possible reason is that initial serum PCB levels may have already declined among the 50% of the medium and high exposure classes who had their blood first tested 6 days or more after entry. Such an occurrence would attenuate both initial differences in serum PCB among exposure categories and decreases in serum PCB from the initial to follow-up periods. Consistent with this interpretation are the observations that persons who were first tested 1-4 days after entry had significantly higher adjusted initial mean serum PCB concentrations than did those first tested later, and that no significant differences in mean serum PCB levels by date of initial blood drawing were found among samples taken 9-12 mo after the fire.

We also acknowledge the possibility that the relationship between exposure category and serum PCB was the result of an unaccounted or inadequately controlled confounder. A definitive test would be to compare the serum PCB levels of a group of persons immediately before and after entry, but such data were unavailable. Regardless of whether it is causal, the magnitude of the association was small. That is, exposure category accounted for less than 4% of the variance in serum PCB concentrations. This finding is consistent with the relatively brief duration of exposure for most persons and the fact that two-thirds wore protective gear. The mean and individual values for each exposure group were also within the ranges reported by Landrigan as typical of persons with no unusual exposure to PCBs.[33] In contrast, studies of occupationally exposed electrical capacitor workers have found means as high as 820 ppb and individual values as great as 1,900 ppb.[34]

Since interlaboratory variations in procedure can considerably influence apparent human PCB levels,[25] comparisons with the results of other investigations must be qualified. It is unlikely, however, that our statistically significant differences among exposure groups in mean serum PCB concentrations or our correlations with the biochemical parameters are artifacts of intralaboratory variability. To avoid systematic bias, initial and follow-up samples from persons in the three exposure categories were randomly assigned to each analytic batch. When the quality control data were plotted against time, the pattern indicated random variation around the target values, with no evidence of a consistent trend.

The association of serum PCB with the liver function tests implies hepatic involvement, including the possibility of microsomal enzyme induction.[35] The clinical significance nevertheless is unclear, especially in the

**Table 5.—Number (*N*) and Percentage (%) of 147 examined persons with selected symptoms by system**

| System | *N* | % |
|---|---|---|
| Skin | 68 | 46.3 |
| Rash or dermatitis | 49 | 33.6 |
| Itching | 47 | 32.4 |
| Thickening or scaling | 20 | 13.6 |
| Discoloration | 15 | 10.3 |
| Acne | 14 | 9.6 |
| Eyes | 46 | 31.3 |
| Pain or burning | 28 | 19.0 |
| Vision changes | 25 | 17.1 |
| Discharge or infection | 14 | 9.5 |
| Swollen eyelids | 8 | 5.4 |
| Neurologic | 40 | 27.2 |
| Numbness in extremities | 24 | 16.3 |
| Dizziness | 20 | 13.6 |
| Clumsiness | 8 | 5.5 |
| Gastrointestinal | 32 | 21.8 |
| Change in bowel habits | 17 | 11.6 |
| Abdominal pain | 14 | 9.5 |
| Nausea or vomiting | 6 | 4.1 |
| Hepatitis or liver problem | 5 | 3.4 |
| General | 68 | 46.3 |
| Nervousness or sleep problems | 38 | 25.9 |
| Headaches | 37 | 25.3 |
| Muscle pain | 24 | 16.3 |
| Unintentional weight loss or 10 lbs or more | 19 | 13.0 |
| None | 41 | 27.9 |

**Table 6.—Number (*N*) and Percentage (%) of 147 examined persons with selected physical findings by system**

| System | *N* | % |
|---|---|---|
| Skin | 82 | 55.8 |
| Rash | 26 | 17.7 |
| Acne-like lesions | 24 | 16.3 |
| Erythema | 10 | 6.8 |
| Thickening | 7 | 4.8 |
| Hyperpigmentation | 6 | 4.1 |
| Eyes | 11 | 7.5 |
| Conjunctive injection | 3 | 2.0 |
| Abnormal pigment | 3 | 2.0 |
| Swollen eyelids | 2 | 1.4 |
| Neurologic | 10 | 6.8 |
| Sensory impairment | 5 | 3.4 |
| Abnormal cranial nerves | 3 | 2.0 |
| Liver and Abdomen | 5 | 3.4 |
| Tenderness | 4 | 2.7 |
| Hepatomegaly | 2 | 1.4 |
| None | 49 | 33.3 |

absence of manifest disorders such as hepatomegaly. The correlation between concentrations of serum PCB and triglycerides and cholesterol may indicate that PCBs interfere with lipid metabolism. Brown and Lawton, however, have argued that the PCB—lipid association found in serum samples is an artifact of the tendency of lipophilic compounds such as PCBs to partition between adipose tissue and serum lipids in direct proportion to the lipid content of the serum.[36] In other words, the direction of the relationship may be reversed, with the level of PCB in the serum dependent upon the level of serum lipids.

In contrast to the results for serum PCB, there were no statistically significant differences among exposure groups in their mean concentrations of liver enzymes or lipids at either time period after covariance adjustment. The average difference of 2 ppb in serum PCB levels

between the low and high exposure categories, although statistically significant, was apparently too small to induce elevations in these biochemical parameters. The means and medians for the liver function tests and lipids were also within the range specified by the contract laboratory as normal with the exception of triglycerides. Outliers at each time period and failure to fast initially most likely contributed to the elevation of the means for that parameter to the upper limit of 180 mg/dl.

Among the firefighters and persons in the State Office Building for 25 hr or more, one-half reported or had a dermatologic condition upon examination 9-12 mo after the fire. Skin pathology, however, is also common in the general population. For example, prevalence rates of significant skin conditions in the National Health and Nutrition Survey for men of similar age range from 35.8% to 42.4%.[37] Since the participants and examining physicians were aware of both the exposure and of potential health effects, it is also possible that these results reflect reporting and diagnostic biases.[38] In addition, symptoms such as nervousness or sleep problems and headaches may be the result of the psychological stress that environmental accidents frequently engender.[39]

Further analyses revealed that mean levels of initial serum PCB were significantly greater among persons who complained of liver problems or hepatitis and who had hyperpigmentation, while those with itching skin and acne-like lesions had significantly higher mean exposure index scores. These findings may reflect exposure-related health effects, e.g., persons with alcohol-impaired livers could have difficulty clearing PCBs. The greater use of protective gear among persons who were in the building for many hours, however, may have led to the association between skin problems and exposure index score as an artifact of increased perspiration and local irritation. Background differences in factors unrelated to exposure may be responsible, since small cell frequencies allowed for the covariance adjustment of only age in these analyses. Chance may also be involved, given the multiple comparisons that were performed. That is, even if the null hypothesis of no effect was true, 3.60 of the 72 contrasts involving symptoms, physical findings, serum PCB levels, and exposure index scores would be significant at the 5% level and 0.72 significant at the 1% level, expectations which closely agree with the observed results.

It is also important to note that no cases of chloracne were detected. Chloracne is generally regarded as the *sine qua non* of substantial exposure to PCBs, PCDDs, and PCDFs,[40] and its absence among persons in this program is consistent with their relatively low serum PCB levels. In fact, other studies suggest a threshold effect, with chloracne rarely observed among persons who have serum PCB concentrations of less than 200 ppb.[34] Such a value exceeds by four times the maximum found in this program. There also was little clinical evidence of exposure-related liver or eye diseases and of neurologic conditions. This finding agrees with the general belief that systemic disorders are unlikely in man in the absence of chloracne.[41]

Seppalainen et al.[42] have recently reported similar results from an investigation of 15 men accidentally exposed to PCBs from a capacitor explosion in a Finnish cardboard plant. The highest serum PCB concentrations were measured 3 days after the explosion and were comparable in magnitude to our values; they decreased to baseline levels within 1-2 mo. Nausea, intense perspiration, and headaches were acute symptoms which cleared quickly. Blood chemistries and physical examination data were not reported, but nerve conduction velocity tests indicated a slight and reversible impairment of the peripheral nerves.

Our results should be interpreted cautiously. For instance, the program lacks a comparison group of randomly selected community controls. This omission is a result of the fact that this primary goal of the program is public service, i.e., to assess the health status of the workers and to provide them and their physicians with the results. It is only secondarily a research project addressing the question of whether a statistical association exists between exposure and any adverse outcomes.

We also did not measure body burdens of PCDDs and PCDFs in addition to PCBs. Our reasons included the lack of appropriate general population data to use as background and the inherent difficulties of the analytic methodology.[43] It therefore was not possible to ascertain whether reported exposure was correlated with PCDD and PCDF fat levels or to determine the independent health effects of the three chemicals.

Despite such limitations, the general pattern of the findings suggests that exposure to contaminants from the building did not result in substantial absorption or cause major short-term health effects. One may also speculate that the future morbidity and mortality experience of the group should not differ from that for the general population, but follow-up will continue, since so little is known about the long-term effects of human exposure to PCBs, PCDDs, and PCDFs, particularly at low levels.[44]

PCB-containing coolants and fluids have been banned from electrical transformers manufactured in the United States since 1977, but approximately 130,000 such transformers placed in operation before that date were still in use in 1981.[45] The Binghamton episode is probably the leading example of transformer fire contamination and since the accident, San Francisco, Santa Fe, and other cities across the nation have employed the procedures developed for Binghamton to assist in their post-fire clean-up and medical surveillance. The methods described in this report should continue to be a useful guide in dealing with future episodes. The results also contribute to our understanding of how low level exposures to PCBs, PCDDs, and PCDFs affect human health.

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## References


1. Taylor, J.S. 1979. Environmental chloracne: Update and overview. *Annals NY Acad Sci* 320: 295-307.
2. Suskind, R.R., and Hertzberg, V.S. 1984. Human health effects of 2,4,5-T and its toxic contaminants. *JAMA* 251: 2372-80.
3. Moses, M.; Lilis, R.; Crow, K.D.; et al. 1984. Health status of workers with past exposure to 2,3,7,8-Tetrachlorodibenzo-p-dioxin in the manufacture of 2,4,5-Trichlorophenoxyacetic acid: Comparison of findings with and without chloracne. *Am J Ind Med,* 5: 161-82.
4. Maroni, M.; Colombi, A.; Arbosti, G.; et al. 1981. Occupational exposure to polychlorinated biphenyls in electrical workers. II. Health effects. *Br J Ind Med* 38: 55-60.
5. Murai, Y., and Kuroiwa, Y. 1971. Peripheral neuropathy in chlorophenyl poisoning. *Neurology* 21: 1173-76.
6. Pocchiari, F.; Silano, V.; and Zampieri, A. 1979. Human health effects from accidental release of tetrachlorodibenzo-p-dioxin (TCDD) at Seveso, Italy. *Annals NY Acad Sci* 320: 311-20.
7. Kuratsune, M.; Yoshimura, T.; Matsuzaka, J.; et al. 1972. Epidemiologic study on yusho, a poisoning caused by ingestion of rice oil contaminated with a commercial brand of polychlorinated biphenyls. *Environ Health Perspect* 1: 119-28.
8. Pazderova-Vejlupkova, J.P.; Nemcova, M.; Pickova, J.; et al. 1981. The development and prognosis of chronic intoxication by tetrachloro-dibenzo-p-dioxin in men. *Arch Environ Health* 36: 5-11.
9. Oliver, R.M. 1975. Toxic effects of 2,3,7,8 tetrachlorodibenzo 1,4 dioxin in laboratory workers. *Br J Ind Med* 32: 49-53.
10. Baker, E.L.; Landrigan, P.J.; Glueck, C.J.; et al. 1980. Metabolic consequences of exposure to polychlorinated biphenyls (PCB) in sewage sludge. *Am J Epidemiol* 112: 553-63.
11. Chase, K.H.; Wong, O.; Thomas, D.; et al. 1982. Clinical and metabolic abnormalities associated with exposure to polychlorinated biphenyls (PCBs). *J Occup Med* 24: 109-14.
12. Courtney, K.D., and Moore, J.A. 1971. Teratology studies with 2,4,5-T and 2,3,7,8-TCDD. *Toxicol Appl Pharmacol* 20: 396-403.
13. Kimbrough, R.D.; Squire, R.A.; Linder, R.E.; et al. 1975. Introduction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. *J Natl Cancer Inst* 55: 1453-59.
14. Kociba, R.J.; Keyes, D.G.; Beyer, J.E.; et al. 1978. Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rats. *Toxicol Appl Pharmacol* 46: 279-303.
15. Hardell, L., and Sandstrom, A. 1979. Case-control study: Soft-tissue sarcomas and exposure to phenoxyacetic acids or chlorophenols. *Br J Cancer* 39: 711-17.
16. Brown, D.P., and Jones, M. 1981. Mortality and industrial hygiene study of workers exposed to polychlorinated biphenyls. *Arch Environ Health* 36: 120-29.
17. Townsend, J.C.; Bodner, K.M.; Van Peenen, P.F.D.; et al. 1982. Survey of reproductive events of wives of employees exposed to chlorinated dioxins. *Amer J Epidiol* 115: 695-713.
18. Erickson, J.D.; Mulinare, J.; McClain, P.W.; et al. 1984. Vietnam veterans' risks for fathering babies with birth defects. *JAMA* 252: 903-12.
19. Kuratsune, M., and Shapiro, R.E., Eds. 1984. PCB poisoning in Japan and Taiwan. *Am J Ind Med* 5: 1-155.
20. Caramaschi, F.; Del Corno, G.; Favaretti, C.; et al. 1981. Chloracne following environmental contamination by TCDD in Seveso, Italy. *Intl J Epidiol* 10: 135-43.
21. Schecter, A.J.; Haughie, G.F.; Rothenberg, R.; et al. 1981. PCB transformer fire—Binghamton, NY. *MMWR* 30: 187-93.
22. Smith, R.M.; O'Keefe, P.W.; Hilker, D.R.; et al. 1982. Analysis for 2,3,7,8-tetrachlorodibenzofuran and 2,3,7,8-tetrachlorodibenzo-p-dioxin in a soot sample from a transformer explosion in Binghamton, NY. *Chemosphere* 12: 325-32.
23. Centers for Disease Control. 1981. *Polychlorinated Biphenyl Determination at Parts—Per—Billion Level in Serum.* Atlanta: CDC Laboratory Update 81-108.
24. Webb, R.G., and McCall, A.C. 1973. Quantitative PCB standards for electron capture gas chromatography. *J Chromatogr Sci* 11: 366-73.
25. Lawton, R.W.; Brown, J.F.; Ross, M.R.; et al. 1985. Comparability and precision of serum PCB measurements. *Arch Environ Health* 40: 29-37.
26. Winer, B.J. 1973. *Statistical Principles in Experimental Design,* 2nd ed. New York: McGraw-Hill.
27. Kleinbaum, D.G., and Kupper, L.L. 1978. *Applied Regression Analysis and Other Multivariable Methods.* Belmont, CA: Wadsworth Publishing Company.
28. Morrison, D.F. 1976. *Multivariate Statistical Methods.* New York: McGraw-Hill.
29. SPSS, Inc. 1983. *SPSSX User's Guide.* New York: McGraw-Hill.
30. Dixon, W.J., Ed. 1983. *BMDP Statistical Software.* Berkeley: University of California Press.
31. Hollingshead, A.B. 1957. *Two Factor Index of Social Position.* New Haven: Mimeograph.
32. Lutz, R.J.; Dedrick, R.L.; Matthews, H.B.; et al. 1977. A preliminary pharmacokinetic model for several chlorinated biphenyls in the rat. *Drug Metab Disp* 5: 336-96.
33. Landrigan, P.J. 1980. General population exposure to environmental concentrations of halogenated biphenyls. In *Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products,* R.D. Kimbrough, Ed. Amsterdam: Elsevier/North-Holland Biomedical Press.
34. National Institute of Occupational Safety and Health. 1977. *Criteria for a Recommended Standard: Occupational Exposure to Polychlorinated Biphenyls (PCB),* U.S. Department of Health, Education and Welfare, HEW (NIOSH) Publication No. 77-225. Washington, DC: US Government Printing Office.
35. Smith, A.B.; Schloemer, J.; Lowry, L.K.; et al. 1982. Metabolic and health consequences of occupational exposure to polychlorinated biphenyls. *Br J Ind Med* 39: 361-69.
36. Brown, J.F., and Lawton, R.W. 1984. Polychlorinated biphenyl (PCB) partitioning between adipose tissue and serum. *Bull Environ Contam* 33: 277-80.
37. Johnson, M.-L.T. 1978. *Skin Conditions and Related Need for Medical Care Among Persons 1-74 Years, United States, 1971-1974.* Vital and Health Statistics: Series 11, Data from the National Health Survey; No. 212. DHEW Publication No. (PHS) 79-1660. Washington, DC: U.S. Government Printing Office.
38. Sackett, D.L. 1979. Bias in analytic research. *J Chron Dis* 32: 51-63.
39. Logue, J.N.; Melick, M.E.; Hansen, H. 1981. Research issues and directions in the epidemiology of the health effects of disasters. *Epidiol Rev* 3: 140-62.
40. Young, A.L.; Calcagni, J.A.; Thalken, C.E.; et al. 1978. *The Toxicology, Environmental Fate, and Human Risk of Herbicide Orange and Its Associated Dioxin.* Brooks Air Force Base, TX: US Air Force, Office of Occupational and Environmental Health Laboratory, Aerospace Medical Division, Technical Report TR-78-92.
41. Beljan, J.R.; Irey, N.S.; Kilgore, W.W.; et al. 1981. *The Health Effects of Agent Orange and Polychlorinated Dioxin Contaminants.* Chicago, IL: American Medical Association.
42. Seppalainen, A.M.; Vuojohahti, P.; Elo, O. 1985. Reversible nerve lesions after accidental polychlorinated biphenyl exposure. *Scand J Work Environ Health* 11: 91-95.
43. Rappe, C. 1984. Analysis of polychlorinated dioxins and furans. *Environ Sci Technol* 18: 78A-90A.
44. Kimbrough, R.D., Ed. 1980. *Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products.* Amsterdam: Elsevier/North-Holland Biomedical Press.
45. U.S. Environmental Protection Agency. 1982. Polychlorinated biphenyls (PCBs) manufacturing, processing, distribution in commerce and use prohibitions, use in electrical equipment. *Fed Reg* 47: 37342-60.