Environmental Health Perspectives
Vol. 60, pp. 133-138, 1985

Occupational Exposure to Polychlorinated Biphenyls (PCBs)

by Mary S. Wolff*

Human occupational exposure to polychlorinated biphenyls (PCBs) with varying chlorine content has been reported by several investigators, using analyses of blood or adipose samples or skin wipes to evaluate levels in the body. The intensity of occupational exposure is related to both duration and intensity of exposure. The qualitative nature of occupational exposure, as well as casual environmental exposure, has been shown to consist of less readily metabolized PCB congeners. The pattern of PCB congeners in human tissues, determined by gas chromatography, may or may not be readily ascribed to specific PCB standard mixtures.

The average occupational exposure, as depicted in several studies of blood, plasma or serum concentrations, is approximately 10 to 1000 times that observed in nonoccupationally exposed persons. Currently used methods of PCB quantitation and pattern identification vary widely, with no uniformly administered criteria being applied to characterize human PCB exposure.

Although industrial use and manufacture of polychlorinated biphenyls (PCBs) had ended in the United States by 1977, the opportunity for human exposure still exists. PCB-containing transformers and capacitors remain in use, and exposure may occur during repair or in accidents involving electrical equipment. NIOSH estimated that 12,000 workers had potential occupational exposure from 1970 to 1976, when 40 to 85 million pounds per year were being produced in the U.S. (*1*). Most (70%) was used in capacitor manufacture: 95% of 100 million capacitors contained PCBs prior to 1977. The remainder (30%) was used in transformers; an estimated 5% of 135,000 transformers in the U.S. in 1975 contained PCBs (*1*).

Dermal and Respiratory Exposure to PCBs

There have been several studies of PCB-exposed workers reported since 1976 which will be discussed. In some cases, estimates of exposure, from ambient air measurements, were available.

The threshold limit values for PCBs, stipulated by the American Conference of Government Industrial Hygienists (ACGIH) have been 0.5 μg/m for PCBs with 54% chlorine and 1.0 μg/m for PCBs with 42% chlorine (8-hr time-weighted average). These values are similar to those of several other countries (*1*).

*Environmental Sciences Laboratory, Mount Sinai School of Medicine, One Gustave L. Levy Place, New York, NY 10029.

The importance of dermal, as well as respiratory exposure, has been appreciated, although the relative contribution of these routes of exposure is not known.

The exposures reported in Table 1, involving studies of workers prior to 1977, are well within the ACGIH values. However, significant levels were found on the palms of hands (*2*) and other skin surfaces (*3*). Even at fairly low levels of exposure, when PCB use had been discontinued for 2.5 years, Aroclor 1016 was found on the skin of workers, using passive collection after some time away from the workplace (*4*). A dermal exposure of 5 μg/cm over the hands and face (ca. 200 cm) or the entire body (ca. 20,000 cm), with 100% absorption into the main body reservoir (10 kg adipose), would represent 0.2 to 20% of a 50 μg/g adipose level, typical of that reported among exposed capacitor workers (*5*). Since dermal absorption could obviously be incremental over time, this exposure route could easily account for PCBs in the bodies of such workers.

Table 1. PCBs determined from skin of exposed workers (Aroclor 1242 or 1016).

Air levels, $\mu g/m^3$	Skin wipe, $\mu g/cm^2$	Exposure	Reference
80–275	2–22	Capacitor manufacture	(*2*)
0–264	0.1–7	Electrical equipment manufacture	(*3*)
0.4–9	0.05–5	Transformer inspection	(*3*)
< 100	0.002–0.019	Capacitor manufacture, off worksite	(*4*)

Table 2. PCB levels in blood of exposed workers (Aroclor 1016/1242/1248).

Air levels, mg/m³	Blood levels, ng/mL Mean	High	N		Reference
0.3–2	1060	3500	19	"Inside"	(6)[a]
	440	1400	14	"Out"	(2)[a]
0.05–0.275	130	407	60		(2)[a]
0–0.26[b]	355	3330	26	Exposed	(3)
	149	1500	55	Ever exposed	
	89	370	140	Never exposed	
0.1–1[b]	118	2530	110	High exposure	(5)
	48	604	180	Other	

[a]Whole blood.
[b]Geometric means; serum or plasma.

Occupational Exposures to Lower Chlorinated Biphenyls

The magnitude of human occupational exposure has been evaluated by measurement of blood and adipose concentrations of PCBs. For lower chlorinated PCBs (LPCB) of the type Aroclors 1016, 1242 or 1248, a range of means of whole blood, serum or plasma levels from 100 to 1000 ng/mL has been reported (Table 2). The highest means, approximately ten times the others in Table 2, were associated with an ambient exposure that was also ten times higher. In one study, serum PCB levels were correlated with personal air sample concentrations (*3*).

Factors other than exposure intensity may contribute to the range of reported values. The analysis of whole blood, rather than serum or plasma, was utilized in one study, where it was reported that whole blood concentrations were generally higher (1.7 times average) than serum, with a wide range (1.1–2.2) (*2*). However, animal experiments with polybrominated biphenyls (*8*) as well as analysis of nonoccupational human blood samples for PCBs in our laboratory (unpublished) have shown that over 90% of such chemicals in blood are found in plasma. This may represent physiological equilibrium, so that the observations reported (*2*) show higher levels in whole blood in persons with very recent exposure. The potential utility of this hypothesis for characterization of human exposures is great and warrants fuller investigation.

Variations in analytical technique contribute to interlaboratory differences in reported blood PCB levels. Intralaboratory variation probably limits reproducibility to 10 to 20% (*5,7*). Different methods of sample preparation, particularly solvent extraction, introduce further wide variations (*9*). The method of calculation of PCB concentration can cause variations of 100% or more (*7,10*). The absence of clearly stipulated calculation procedures in many reported studies prevents quantitative comparison of data. Appendix I provides an example of differences in concentration that can occur for typical data using different calculation methods. Variation by a factor of two may be common.

Adipose concentrations of LPCBs were reported (as Aroclor 1248) in one study (*5*). Workers with direct exposure to PCBs (> 100 μg/m) had 44 μg/g LPCB in adipose (geometric mean, GM; n = 33). Plasma levels among such workers were 118 ng/mL (GM; n = 110). Indirectly exposed workers had 15 μg/g in adipose (GM; n = 28) and 48 ng/mL in plasma (GM; n = 180). The correlation of adipose with plasma levels suggested a fairly uniform adipose–blood partition (*5,10*). However, the partition was found to vary for individual PCB congeners with certain substitution patterns, and those with lower partition ratios were present in significant amounts only among higher exposures (*10*). Nevertheless, given the preponderance of PCB congeners with higher partition ratios, the contribution of those isomers (with lower partition) to the overall body burden is probably negligible in their effect on total adipose–plasma partition.

Occupational Exposure to Higher Chlorinated Biphenyls

For higher chlorinated PCBs, there have been four reports since 1980 of workers exposed to Aroclor 1254, although the actual dates of the studies were several years earlier. In Table 3, the data for analysis of blood or serum from these studies are compared with that from accidental ingestion of PCB-contaminated rice oil in Taiwan, where analysis was undertaken within a year of the episode (*11*). We have also analyzed Aroclor 1260 in serum of transformer manufacture workers (n = 33), with average levels of 86 ng/mL, high 378 ng/mL (unpublished). The average blood levels for the studies in Table 3 are similar, with one exception (*2*). The lowest value in this study (56 ng/mL) (*2*), exceeds the mean of the other studies, and the mean is similar to the upper limit of the other reports. It appears unlikely that analytic methodology can readily account for a factor of 10 difference in blood levels (*7,10*) (Appendix I), and the relatively nonpolar extraction technique used in the study (*2*) would tend to lower (rather than elevate) the values. Air sampling data (as Aroclor 1254) were reported in one example (*3*). The study of Maroni et al. (*2*) found no air levels of higher chlorinated biphenyls, but workplace surfaces and skin wipes from workers showed significant contamination. The other studies (*3,4,12*) apparently involved past exposure or minimal current exposure. Thus, the higher levels in one study (*2*) probably reflect current as well as accumulated past exposure. In other studies, where some historical categorization of exposure was made, higher blood (or adipose) concentrations were seen in higher exposure groups (Tables 3 and 4).

Adipose concentrations of HPCB have been reported in two studies (*5,12*). As with LPCB, a uniform adipose–plasma was demonstrated by their strong correlation (*5,10,12*). Summary data are included in Table 4.

Table 3. HPCB levels in blood of exposed workers and PCB-poisoned patients in Taiwan (Aroclor 1254).

Blood levels ng/mL			
Mean	High	N	Reference
238[a]	1032	64	(2)
49[a]	720	66	(2) (Yu-Cheng)
47[a]	250	26[c]	(3)
27[b]	250	39[d]	(3)
23[b]	130	195[e]	(3)
21[b]	546	290	(5)
33[f]	312	86	(12)

[a]Whole blood.
[b]Plasma or serum; geometric means.
[c]Electrical equipment manufacture, current exposure to Aroclor 1242.
[d]Utility company, current exposure 0–215 μg/m^3 as Aroclor 1254 or Aroclor 1260.
[e]Electrical equipment manufacture, no current exposure to Aroclor 1242.
[f]Plasma.

Table 4. PCB blood levels (Aroclor 1254) and duration of exposure.

Mean duration of employment, yr	Mean blood concentration, ng/mL	N	Mean adipose concentration, μg/g	N	Reference
12± 6	238	80			(2)
16± 8	24[a]	258	17	53	(5)
	6[b]	32	4	8	
17	33[c]	86	5.6	36	(13)
3.8	14[d]	15	1.4	5	
4.3	12[e]	19	1.3	9	

[a]Persons with more than 5 years employment; geometric means; geometric mean of 53 plasma samples which matched the adipose samples was 54 ng/mL.
[b]Persons with less than 5 years employment; geometric means.
[c]Persons exposed.
[d]Persons nominally exposed.
[e]Nonexposed.

Comparisons of Occupational Exposures to PCBs with Casual Environmental (General Population) Exposure

Much attention has been focused on the ubiquitous nature of human exposure to PCBs. One reason for assessing occupational exposure and its derived health effects has been to provide a basis for predicting the potential health hazards of less intense exposure among the general population. It has been recognized that the human body burden of PCBs represents a nonuniform distribution of many PCB congeners (*13*). Differentiation occurs by excretion of more readily metabolized PCB isomers and by retention of more persistent ones (*10,13*).

Smith et al. (*3*) compared their findings with those of community residents from another study (*14*). LPCB serum concentrations were 12 ng/mL for community residents (n=22, mean)* compared with geometric means of 89 to 355 for workers with varying degrees of exposure to Aroclor 1242 (see Table 2). For utility company workers, whether or not exposed to Aroclor 1254, the LPCB serum concentration was 17 ng/mL (geometric mean, n = 93; HPCB levels for this group are found in Table 3). However, the community residents presented for comparison here were apparently a historical control group, and no evidence is given to substantiate comparability of the data. Therefore all but the workers exposed to Aroclor 1242 may be said to have had similar LPCB serum levels.

In the same report (*3*) the community residents (*14*) were compared with exposed workers according to HPCB serum levels. The average level among the community residents was 13 ng/mL (n = 22); among nonexposed utility company personnel, 7 ng/mL (geometric mean, n = 51); among exposed groups, 23 to 47 ng/mL (geometric means) (*3*).

Other general population blood concentrations of HPCB have been reported elsewhere in this symposium. In our laboratory, we have used PCB serum levels among Michigan residents as baseline levels for the general population (*15*). Median concentration among adults (n = 963) was 7 ng/mL (range 1–66 ng/mL), except for one geographic area near Lake Michigan where the median was 18 ng/mL (n = 69, range 1–69 ng/mL). Median adipose levels were 1.1 μg/g (n = 724) and 2.1 μg/g (n = 71), respectively, for the two areas. In this population, recorded fish consumption was related to serum PCB levels, as reported elsewhere in this symposium. We have observed similar levels of HPCB in serum among various non-PCB-exposed groups in the New York–New Jersey area (over 200 subjects), median 5 ng/mL, range 2 to 21 ng/mL. Thus the upper limit of serum HPCB in nonoccupationally exposed persons overlaps the range of values seen in occupational exposures. This is not surprising, and may be attributed to fish consumption or to undocumented accidental or occupational exposure. However, it is obviously impossible to characterize such general population outliers unambiguously, in the absence of documented exposure information.

Cumulative Nature of PCB Body Burden

The association of HPCB concentrations in blood with age has been reported both in occupational and general environmental exposure cirucmstances (*3,5,12-14*). However, in occupational exposures, the corre lation with duration of employment was at least as important as age (*2,5,14*). In Table 4, three occupational groups with exposure to Aroclor 1254 are listed, with similar duration of employment. The high blood HPCB levels (*2*) have been discussed. For longer employed persons (*5,12*), the mean values are similar for both duration

*Smith et al. (*3*) reported n = 89, but our reading of Baker et al. (*14*) indicates that n = 22.

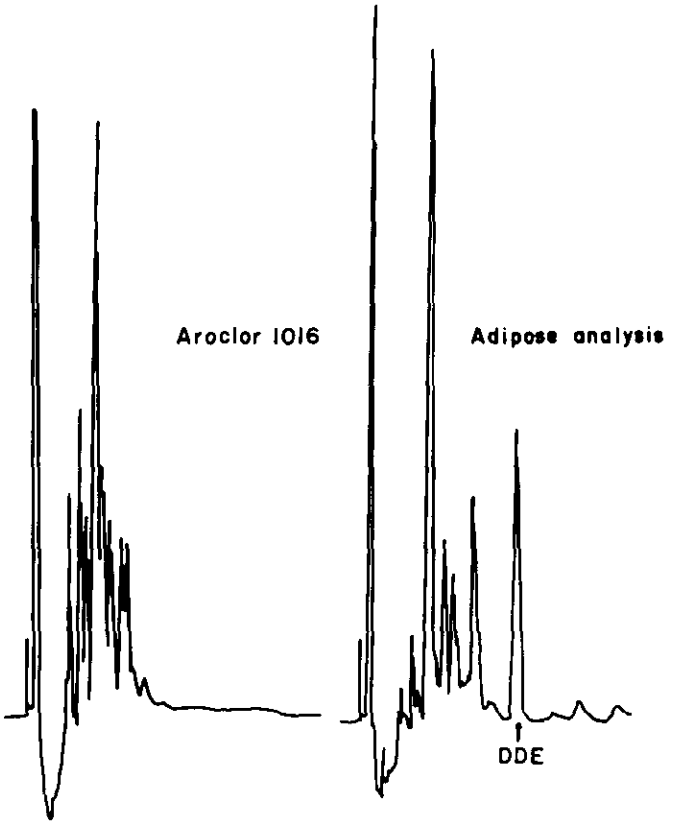

FIGURE 1. Adipose sample from an exposed worker, with 18 μg/g LPCB, has a PCB pattern of peaks similar to Aroclor 1016. The analysis data for this sample, shown here by packed column gas chromatography, obtained by using high resolution gc is given elsewhere (*10*).

employment (16, 17 years) and mean plasma HPCB concentration (24, 33 ng/mL). Persons with shorter-term employment had lower levels. Aroclor 1254 had not been used within the past five years (*5*), and persons employed only during that time had serum HPCB levels comparable to those in the general population. In Chase's study (*12*), nominally or nonexposed persons also had much shorter terms of employment, with similar HPCB serum levels.

Correlations (r) of blood and adipose levels with duration of employment have also been reported. A duration of exposure index was significantly correlated with LPCB (n = 13, r = 0.6–0.9) or HPCB (n = 18, r = 0.7–0.8) blood concentrations (*2*). Among exposed persons, the correlation (r) of length of employment with serum HPCB was 0.27 (n = 86) and HPCB, 0.54 (n = 36) (*13*). The correlations with age were similar (n = 0.28 and 0.51, respectively), since age and length of employment were strongly associated (r = 0.84). The correlation of age and duration of employment was 0.64 in another study (*5*). Here, the correlation of age with serum or adipose HPCB (r = 0.35, n = 290; r = 0.43, n = 61, respectively) was lower than that of employment (r = 0.46, r = 0.45). Among persons currently exposed to LPCB, blood levels were not correlated with term of employment (*5,6*), although the correlation was significant for persons exposed in the past (*5*).

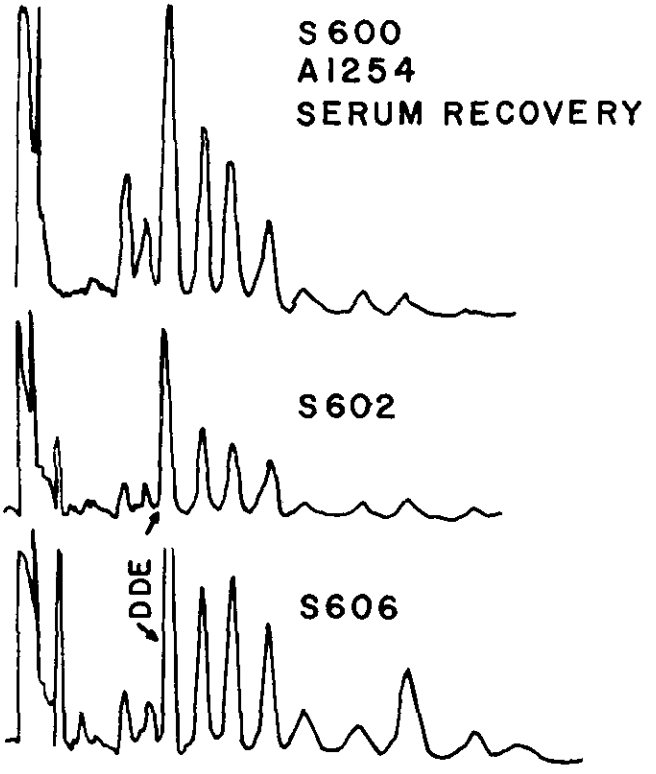

FIGURE 2. Serum PCB analyses from persons exposed to Aroclor 1254, showing a pattern of peaks after p,p'-DDE similar to a serum sample fortified with 50 ng/mL Aroclor 1254.

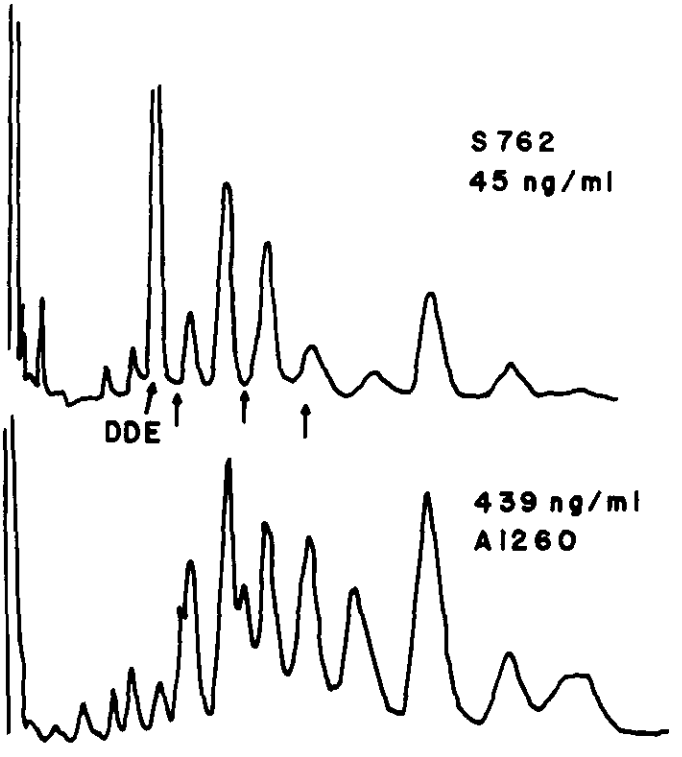

FIGURE 3. Serum PCB analysis from a worker in transformer manufacture, showing a pattern similar to Aroclor 1260 with peaks 117, 160, 203 (arrows) significantly diminished.

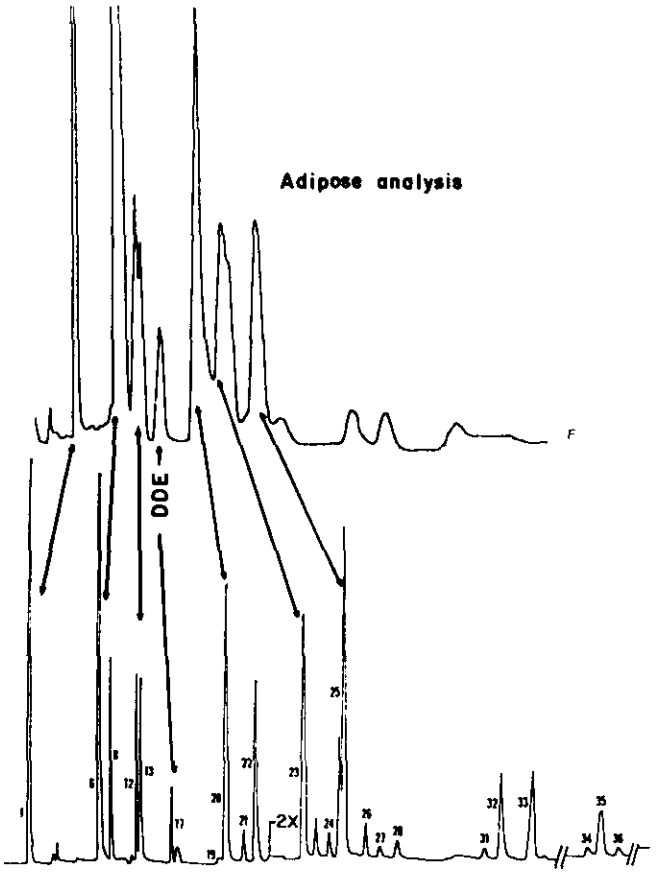

FIGURE 4. Comparison of packed and high resolution gas chromato grams of adipose tissue from a worker with mixed LPCB and HPCB (Aroclor 1254) exposure. Numbered peaks on lower chromatogram are identified elsewhere (*10*).

Qualitative Nature of PCB Exposure

The differentiation of PCB congeners in humans, compared with the commercial product, has been mentioned. Investigators characterize the pattern of PCB peaks appearing in gas chromatograms as resembling a commercial PCB used as a standard. However, there is no widely accepted means of objectively designating PCB patterns in human samples. The problem is particularly difficult with mixed occupational exposures. Even in the case of nonoccupational exposures, our experience has been that combinations of Aroclor 1254 and 1260 are apparent, but impossible to characterize definitively as predominantly one or the other, using various reported methods. Samples of tissue and serum from three types of readily identifiable occupational exposures are depicted in Figures 1–3; packed column gas chromatography (*5*) was used. An occupational exposure to Aroclor 1016, (Fig. 1) produces an adipose tissue extract having a PCB pattern similar to the standard, with some preferential retention of later-eluting peaks. Exposure to Aroclor 1254 is evident in serum extracts (Fig. 2). In cases we have seen, the relative concentration of the first peak after DDE (peak 125; mainly 2,4,5,3′,4′-pentachlorobiphenyl) (*10,16*) varies widely, with lower levels presumably attributable to preferential metabolism over time.

Aroclor 1260 exposure is apparent in a serum sample from a transformer manufacture worker (Fig. 3). However, this PCB pattern also closely resembles what we have most commonly observed in the general population. Figure 4 shows packed and high resolution gas chromatograms from a person with occupational exposure to both higher and lower chlorinated biphenyls. The exposure origin has been discussed in detail elsewhere (*10*). Peaks 1 and 6 represent a metabolically well-differentiated exposure to lower chlorinated PCBs, while peaks 6 and later suggest Aroclor 1254 exposure. Without detailed investigation, visual matching of a PCB pattern such as this to an Aroclor standard presents obvious problems. Utilization of individual PCB congeners as quantitative standards may be suitable for such cases. However, comparability to other analyses may then be lost. Furthermore, except in well defined exposures, discrimination of PCB peaks from potentially coeluting pesticide residues may require both specific sample preparation techniques and verification by gas chromatography–mass spectrometry.

Appendix I

For the purposes of comparing methods of calculation, 20 samples have been used, from workers with a wide range of lower and higher chlorinated PCBs (LPCB, HPCB). LPCBs were calculated by use of Aroclor 1248 as the standard. The sum of the integrated peak areas was used to calculate a response factor (method A: concentration of Aroclor 1248 in ng/mL ÷ total area for peaks 28, 32, 37, 40, 47, 54, 58, 70, 78, 84). Peaks are designated related to *p,p′*-DDE[2,2-bis(4-chlorophenyl)-1,1-dichloroethylene, RRT = 100] (*16*). For method B, individual peak response factors were calculated by using their proportional weight contributions to the standard (*16*), and the resulting peak concentrations were added. For method A, the same response factor was used, regardless of the number of matching peaks (usually 7–9/10). Method B used only corresponding peaks in the sample and the standard. Method C used individual peak responses of authentic PCB standards: 4,4′-dichloro-CB for peaks 28, 32; 2,4,4′-trichloro-CB for peak 37; 2,4,2′,4′-tetrachloro-CB for peaks 40–70; 2,4,5,2′,5′-pentachlorobiphenyl for peaks 78, 84. The results were, for method A, 4 to 251 ng/mL, mean 85, standard deviation (SD) 67, geometric mean (GM) 59. For method B, the range was 3 to 201 ng/mL; mean 67, SD 53, GM 46. For method C the range was 3 to 208 ng/mL; mean 67, SD 54, GM 45.

For linear regression of the values of method A versus method B, the slope was 1.3, $r = 0.999$. For method C versus A, the slope was 1.02, $r = 0.999$. The response factor for Aroclor 1016 was 1.15 times that of Aroclor 1248, using method A. Thus, LPCB levels, using Aroclor 1016 as the standard with method A, would be 1.5 times the levels using method B, and 1.15 times the levels using method A with Aroclor 1248 as the standard. Methods B and C gave almost identical results.

HPCBs were calculated six different ways, with Aroclor 1254 or Aroclor 1260 as the standard, using the following response factors.

Method A: concentration of Aroclor 1254 (ng/mL) sum of areas of peaks 104, 125, 146, 160, 174, 203, 232.

Method B: concentration of Aroclor 1254 0.515 sum of peaks 104 to 232 (0.515 is the relative weight contribution of peaks 104 to 232 from reference 11).

Method C: individual peak response factors for Aroclor 1254 were calculated for peaks 104 to 232 (*16*) and the resulting individual sample peak concentrations were added.

Method D: individual peak response factors for Aroclor 1260 were calculated for peaks 117 to 372 (*16*).

Method E: as for method D, using peaks 117 to 232.

Method F: peak response factors were calculated for peaks 125 to 232 for purchased PCB congeners. 2,4,5,2′,5′-Pentachlorobiphenyl was used for peak 125 since a standard was not available.

The results were (range, mean, SD, GM in ng/mL): Method A: 3–250, 52, 58, 33; Method B: 1.5–129, 27, 30, 24; Method C: 1.5–110, 23, 26, 14; Method D: 3–232, 40, 52, 17; Method E: 2–166, 33, 38, 20; Method F: 2–115, 25, 28, 16.

From visual inspection the pattern of HPCB peaks most often resembled Aroclor 1254. The linear regression of the values for the methods against values of method C showed slopes of 2.2, method A; 1.2, method B; 2.0, method D; 1.5, method E; 1.1, method F. Therefore other methods gave values 20 to 120% higher than method C, except for method F, which closely approximated method C. Analysis of variance of the geometric means showed that method A (GM 33 ng/mL) was significantly different from method C (GM 14 ng/mL). The remaining results (B, D, E) were not significantly different from any others.

REFERENCES

1. NIOSH. Criteria for a recommended standard. Occupational exposure to polychlorinated biphenyls. USDHEW. (NIOSH) publication No. 77–225, September 1977.
2. Maroni, N., Columbi, A., Cantoni, S., Ferioli, E., and Foa, V. Occupational exposure to polychlorinated biphenyls in electrical workers. I. Environmental and blood polychlorinated biphenyls concentrations. Brit. J. Ind. Med. 38: 49–54 (1981).
3. Smith, A. B., Schloemer, J., Lowry, L. K., Smallwood, A. W., Ligo, R. N., Tanaka, S., Stringer, W., Jones, M., Hervin, R., and Glueck, C. J. Metabolic and health consequences of occupational exposure to polychlorinated biphenyls. Brit. J. Ind. Med. 39: 361–369 (1982).
4. Wolff, M. S. Analysis of skin lipids for halogenated hydrocarbons. Anal. Chem. 56: 1492–1496 (1984).
5. Wolff, M. S., Fischbein, A., Thornton, J., Rice, C., Lilis, R. and Selikoff, I. J. Body burden of polychlorinated biphenyls among persons employed in capacitor manufacturing. Int. Arch. Occup. Environ. Health. 49: 199–208 (1982).
6. Ouw, H. K., Simpson, G. R., and Siyali, D. S. Use and health effects of Aroclor 1242, a polychlorinated biphenyl, in an electrical industry. Arch. Environ. Health. 31: 189–194 (1974).
7. Sawyer, L. D. Quantitation of polychlorinated biphenyl residues by electron capture gas-liquid chromatography: collaborative study. J. Assoc. Off. Anal. Chem. 61: 282–291 (1978).
8. Tuey, D. B., and Matthews, H. B. Distribution and excretion of 2,2′,4,4′,5,5′-hexabromobiphenyl in rats and man: pharmaco kinetic model predictions. Toxicol. Appl. Pharmacol. 53: 420–431 (1980).
9. Albro, P. W. Problems in analytic methodology: sample handling, extraction, and cleanup. Ann. N.Y. Acad. Sci. 320: 19–27, 1979.
10. Wolff, M. S., Thornton, J., Fischbein, A., Lilis, R., and Selikoff, I. J. Disposition of polychlorinated biphenyl congeners in occupationally exposed persons. Toxicol. Appl. Pharmacol. 62: 294–306 (1982).
11. Chen, P. H., Gaw, J. M., Wong, C. K., and Chen, C. J. Levels and gas chromatographic patterns of polychlorinated biphenyls in the blood of patients after PCB poisoning in Taiwan. Bull. Environ. Contam. Toxicol. 25: 325–329 (1980).
12. Chase, K. H., Wong, O., Thomas, D., Berney, B. W., and Simon, R. K. Clinical and metabolic abnormalities associated with occupational exposure to polychlorinated biphenyls (PCBs). J. Occup. Med. 24: 109–114 (1982).
13. Jensen, S., and Sundstrom, G. Structures and levels of most chlorobiphenyls in two technical PCB products and in human adipose tissue. Ambio 3: 70–76 (1974).
14. Baker, E. L., Landrigan, P. J., Glueck, C. J., Zack, W. M., Liddle, J. A., Burse, V. W., Housworth, W. J. and Needham, L. . Metabolic consequences of exposure to polychlorinated biphenyls (PCB) in sewage sludge. Amer. J. Epidemiol. 112: 553–563 (1980).
15. Wolff, M. S., Anderson, H. A., and Selikoff, I. J. Human tissue burdens of halogenated aromatic chemicals in Michigan. J. Amer. Med. Assoc. 247: 2112–2116 (1982).
16. Webb, R. G., and McCall, A. C. Quantitative PCB standards for electron capture gas chromatography. J. Chromatogr. Sci. 11: 363–373 (1973).