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PCBs IN HUMANS:
WHAT WE KNOW AND WHAT WE DON'T KNOW*

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ABSTRACT

PCBs are found in the adipose tissue of humans through environmental, dietary, and occupational exposure. Measurement of PCB concentrations in human adipose, blood, and other tissues has begun to give a picture of the human body burden. Total aggregate PCB concentrations are of some use, but the fingerprint chromatograms from the individual congeners tell much more about the exposure (e.g., which Aroclor is involved), uptake, and metabolism of PCBs.

TOTAL PCB LEVELS

Tables 1 and 2 present summaries of the literature values for blood and adipose, respectively. The literature available through the mid-1970s was reviewed by Wassermann et al. (1979); those studies are not repeated in these tables. While these tables oversimplify some of the studies, they provide an overall comparison of the PCB levels reported.

Blood

The limited data presented in Table 1 indicate that the mean PCB levels for an unexposed (ambient) population are less than 30 ppb, but are present at detectable levels in almost all people. In some, but not all cases, exposed people have much higher levels. Similar results were presented by Wassermann et al. (1979) in their review of studies from the early 1970s.

Adipose

As shown in Table 2, virtually all adipose samples contain measurable PCB levels. Most of the mean levels from several studies worldwide are near 1 ppm. Despite the differences in geography, exposure potential, analytical method, reporting basis, and other variables, the data are most remarkable in their consistency. Wassermann et al.'s (1979) review of 26 early studies found generally similar mean levels, although studies by one German group found mean levels in the 5-10 ppm (wet tissue) range.

Other Tissues

The mean PCB concentrations in whole milk tended to be around 10 to 50 ppb in the 21 studies from 1969 to 1977 reviewed by Wassermann et al. (1979), although one study reported a high of 390 ppb. In the same review, PCB concentrations of milk-extracted liquid tended to average 1 to 3 ppm with a high of 19 ppm. The U.S. National Human Adipose Tissue Survey (NHATS) network analyzed 1033 human milk samples from across the U.S., presumably collected in the mid-to-late 1970s (Lucas et al. 1980), and 99.1% of the samples contained PCBs. The highest reported concentration of 0.56 ppm (basis not reported) was from Texas.

INDIVIDUAL CONGENER LEVELS

It is well recognized that the individual PCB congeners have different physical and chemical properties and, thus, will have different biological half-lives. Thus, even assuming (incorrectly) that the ingested PCBs have the composition of the commercial products (e.g., Aroclors), there is every reason to believe that the PCB composition will change with time and that the PCBs in blood, adipose, and other human samples will not resemble given Aroclor patterns. Recognizing this, several researchers have reported PCBs in human samples by congener. This markedly increases the amount of information available but also makes comparisons that much more difficult.

Unfortunately, there is little agreement among the published reports on the identification of key congeners in blood and adipose. Wolff et al. (1982) identified 40 congeners, of which eight had relatively high concentrations (>1 mg/g for adipose and >4 ng/mL for blood plasma). Fait et al. (1989) identified seven major chromatographic peaks, most of which were two- or three-congener peaks, and a total of 89 peaks in adipose and serum. Only two of Fait's seven major peaks corresponded to Wolff's eight largest. Kuroki and Masuda (1977) assigned eight peaks in samples of blood from Yusho patients. Luotamo et al. (1988) identified 19 congeners in blood serum. Focardi et al. (1986) identified 31 congeners in adipose. The congeners identified in these studies range from di- through decachlorobiphenyls. Although many congeners are common to more than one study, only one congener (2,2-,4,4-,5,5-hexachlorobiphenyl) was reported in all five studies. Furthermore, "key" high concentration congeners reported in one study are notably absent in others. This lack of agreement implies that the identities and amounts reported are suspect in at least some of the studies.

PARTITIONING BETWEEN COMPARTMENTS

PCBs are long-lived in the body; however, they do exchange between adipose and blood. In a study of 26 capacitor manufacturing facility workers, Wolff et al. (1982) reported ratios of PCB concentrations in adipose to blood for 37 key congeners from 60 to 300, with an average of 190. The range of ratios was attributed to differences in structure which affect lipophilic and other physical and chemical properties of the PCB congeners. Wolff et al. (1982) noted that this ratio is consistent with limited data from animal studies. Although not calculated in their work, the results of the Government Services Association (GSA) workers' study can be similarly compared. For the 94 total PCB concentrations calculated using the Webb-McCall (1973) method, adipose-to-blood PCB ratios ranged from 21 to 1300 with a mean of 190 (Erickson et al. 1981).

POPULATION STRATIFICATION

Not all humans will have the same PCB body burden. Exposures from food, occupation, environment, and other sources will vary markedly. Total body burden should increase with age if there is a steady exposure. Elimination rates will vary according to complex metabolic factors such as weight (amount, variability, etc.), diet, and activity level. Other factors such as sex and race may or may not be significant factors.

Several studies of "normal" population (i.e., no known non-ambient exposure) have affirmed the relatively even distribution of PCBs throughout the world. As shown on Tables 1 and 2, except for some known occupational or acute accidental exposures, there are no significant differences by sex or region in most studies. The studies summarized in Tables 1 and 2 are perhaps most remarkable for their consistency.

The blood and adipose PCB patterns were compared for patients exposed to Yusho rice oil (degraded Kanechlor 600) with "normal persons" in 1975 to 1976 (Kuroki and Masuda). The Yusho patients' blood tended to have over twice the concentration (all averaged less than one ppb/congener) of many PCB congeners as the controls eight years after the exposure to the rice oil.

EFFECTS OF ANALYTICAL METHODS

In theory, the reported values should be comparable regardless of what analytical method was used or which laboratory performed the work; i.e., the methods should all be accurate. However, several factors combine to degrade the accuracy of PCB analysis. The fundamental problem is that PCB analyses are, in reality, a simultaneous determination of roughly one hundred analytes followed by some sort of aggregation of the individual results. To further complicate the task for adipose and blood samples, the standard mixtures (whether an Aroclor or a congener mixture) do not qualitatively resemble the PCB mixtures found in the samples, nor are the PCB mixtures consistent among samples.

Most of the "total PCB" studies reviewed here employed "standard" methods common across the pesticide residue analysis community and should obtain reasonably comparable results; however, the lack of reported quality control (QC) information and the lack of a central QC testing program for PCBs force us to accept the data without critical evaluation. One critical evaluation, noted above for the large NHATS adipose data base, found the results unusable because of analytical problems.

A direct comparison of three quantitation methods was conducted using adipose and blood samples from 26 workers in a capacitor manufacturing facility (Wolff et al. 1982). For both plasma and adipose, the sum of areas determined with packed column gas chromatography/electron capture detection (GC/ECD) was over 50% higher than the quantitation using the Webb and McCall (1973) method, which, in turn, gave slightly higher results than the sum of individual congener quantitation with capillary GC/ECD chromatograms. Although the authors did not comment on the differences in quantitation, it appears that the major source of the higher values for the sum of areas on the packed chromatograms results from the significant difference between the Aroclor standard and (metabolized) sample PCB patterns. This difference is poorly handled by this method.

A second direct comparison of analytical methods was conducted in the "GSA workers" study (Fait et al. 1987, 1989; Erickson et al. 1981). Extracts of serum and adipose samples from 108 individuals were analyzed by the Webb-McCall packed column GC/ECD method (Webb and McCall 1973) and also by a congener-specific capillary GC/ECD method. In contrast to the results of Wolff et al. discussed in the previous paragraph, the GSA workers' capillary results were consistently higher than the packed column results, with mean results for some

groups differing by a factor of four for serum. Insufficient QA data are available to judge the relative accuracy of the two methods of quantitation. There is no evidence to support the conclusion by Fait et al. (1989) that the packed GC analyses "greatly underestimate those obtained by" capillary GC. Quantitation of the same chromatogram using different standards can also yield vastly different results. Luotamo et al. (1988) studied serum samples from different Finnish groups exposed to PCBs. In this study they compared quantitation of the total PCB concentration using a Chlophen A30 standard with two congener mixtures. The Chlophen A30 consistently gave the best quantitation as defined by the closeness of fit to a model. This result is not too surprising, since the serum patterns most closely matched (by visual inspection and by chemometric pattern recognition) the commercial mixture. Luotamo et al. (1988) noted the main reason for the discrepancies in quantitation was the inconsistency of the PCB patterns among exposure groups and individual samples within groups.

CONCLUSIONS

PCB levels in humans have been studied for over 20 years. General conclusions can now be made that PCBs are ubiquitous, with average levels in adipose somewhere around 1 ppm and in blood, 10 ppb. Exposed people tend to have higher levels in both adipose and blood. Detailed conclusions about congener-specific accumulation and partitioning are not warranted with available data.

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Table 1. PCBs in Human Blood

Geographic Area	No. of Samples	Year (Collected)	(PCB) ppb			Basis	% Detected	Exposure	Significant Findings	References
			High	Low	Mean \pm SD					
Washington DC	37	1980	300	<1	27 \pm 50	Serum	96	Current elect. eqpt. maint.	Current exp. higher than past	Fait et al. 1987-1989; Erickson et al. 1981
Washington DC	17	1980	30	<1	9.9 \pm 9.1	Serum	96	Past elect. eqpt. maint.	Past exp., control	Same
Washington DC	54	1980	15	<1	6.1 \pm 3.8	Serum	96	Control		Same
Finland	35	Late 1980s	24.2	1.1	7.6	Serum	100	1.Occup. 2.Capacitor accident 3.Ambient	Resembles Clophen A30	Luotamo et al. 1988

Table 2. PCBs in Human Adipose

Geographic Area	No. of Samples	Year (Collected)	(PCB) ppb				% Detected	Exposure	Significant Findings	References
			High	Low	Mean \pm SD	Basis				
U.S.A. (All regions)	8593	1972-81	NR	NR	NR	NR	65-99*	Unknown	% of population with detectable residues increasing	Lucas et al. 1980
U.S.A	136	Late 1980s	NR	NR	1.0 \pm 0.57	NR	100	Unknown	Most closely resembles Aroclor 1260	Peterson and Robinson 1988
Canada (All regions)	168	1970s	6.6	0.11	0.91 \pm 0.82	Wet	100	Ambient	Calc. as Aroclor 1260, no significant differences among region, sex, or age group	Mes et al. 1977
Canada (All regions)	99	Early 1980s	6.8	0.040	0.94 \pm 0.90	Wet	100	Ambient	Calc. as Aroclor 1260, no significant differences	Mes et al. 1982
Canada (All regions)	99	Early 1980s	2.3	0.050	0.31 \pm 0.27	Wet	100	Ambient	Calc. as Aroclor 1242, no significant differences except higher levels in Central Region	Mes et al. 1982

Table 2. (cont'd.)

Geographic Area	No. of Samples	Year (Collected)	(PCB) ppb				% Detected	Exposure	Significant Findings	References
			High	Low	Mean \pm SD	Basis				
Kingston Ontario	91	1971-81	28.3	0.090	3.0 \pm 3.6	Wet	100	Ambient	Males > females	Williams et al. 1984
Ottawa Ontario	84	1980-81	4.8	0.45	2.0 \pm 0.87	Wet	100	Ambient		Williams et al. 1984
Washington DC	36	1980	33	T	3.9 \pm 5.9	Lipid	100	Current elect. eqpt. maint.	Current exposure	Fait et al. 1987-89; Erickson et al. 1981
Washington DC	16	1980	5.1	0.3	1.1 \pm 1.2	Lipid	100	Past elect. eqpt. maint.	Past, controls	Same
Washington DC	53	1980	3.0	T	0.8 \pm 0.68	Lipid	100	Control		Same
Siena Italy	26	1983-84	NR	NR	1.8 \pm 0.59	Dry	100	Ambient	Calc. as Aroclor 1260, no sex difference	Focardi et al. 1986

*Significant quantitation problems.

NR = Not reported.

T = Trace.