Cord blood levels of PCBs, *p,p'*-DDE and HCB in infants born in communities adjacent to a PCB-contaminated hazardous waste site.

Larisa Altshul,[1] Susan Korrick,[1,2] Paige Tolbert[3], Richard Monson.[1,4]

[1] Department of Environmental Health, Harvard School of Public Health, 665 Huntington Ave., Boston, MA 02115, USA.
[2] Channing Laboratory, Brigham and Women's Hospital, 181 Longwood Ave., Boston, MA 02115, USA.
[3] Department of Environmental and Occupational Health, Rollins School of Public Health, Emory University, 1518 Clifton Rd., NE Atlanta, GA 30322, USA.
[4] Department of Epidemiology, Harvard School of Public Health, 665 Huntington Ave., Boston, MA 02115, USA.

Introduction

Cord blood is currently the only available *direct* measure of prenatal exposure to PCBs, *p,p'*-DDE, and HCB. Prenatal exposure, in turn, is believed to be the key determinant of PCBs' neurodevelopmental toxicities and is potentially critical to the hypothesized reproductive toxicities of PCBs and DDE. The objective of this study was to characterize intrauterine exposures to PCBs, *p,p'*-DDE, and HCB among a sample of US infants for whom the neurodevelopmental toxicities of prenatal exposure to these compounds are being studied.
The concentrations of 51 individual PCB congeners, the sum of these PCB congeners (ΣPCB), *p,p'*-DDE, and HCB were measured in cord serum of 755 infants born to mothers residing in towns adjacent to a PCB-contaminated harbor in southeastern Massachusetts, USA.

Materials and Methods

Study participants were infants born between March 1993 and December 1998. They were recruited at the time of birth from mothers residing in the towns (New Bedford, Acushnet, Fairhaven, Dartmouth) bordering the PCB-contaminated site.

Laboratory Analysis. Cord serum samples were analyzed for 51 individual PCB congeners and two chlorinated pesticides (*p,p'*-DDE and HCB). The extraction used procedures developed by the Centers for Disease Control (Centers for Disease Control, undated) with modifications to conform to ultra trace level analyses. The serum extracts were analyzed by capillary HRGC/ECD and quantitated based on the response factor of each individual PCB congener or pesticide relative to the internal standard (PCB #166). PCB concentrations were reported as individual congeners and as the sum of all congeners assayed (ΣPCB). All final concentrations were reported after

subtracting the amount of the analyte measured in the procedural blank associated with the analytic batch. Strict quality control and quality assurance procedures were followed in preparation for and during study sample analyses.

Results

Analytic Sensitivity, Reproducibility and Accuracy. Percent recoveries for cord blood samples spiked with low concentrations of PCBs and pesticides ranged from 81 to 123% with the exception of HCB where percent recoveries were lower. The method detection limits (MDL) for targeted individual PCB congeners ranged from 0.002 to 0.036 ng/g serum, with most MDLs < 0.01 ng/g serum (Table 1). After subtracting procedural blank values, *p,p'*-DDE was above MDL in 96% of samples, HCB was ≥MDL in 66% of samples, and 13 of the PCB congeners were measured at values ≥ MDL in at least half of the study infants.

Study Population. The majority (75%) of study infants were born to mothers residing in New Bedford, Massachusetts, the largest of the four towns adjacent to the contaminated site and the study mothers had lived in the area an average of 20 years. The mean (±SD) maternal age at the time of the study infant's birth was 26±5 years. Most (63%) infants were born to multiparous mothers, approximately half of whom had breastfed previous infants.

Levels of PCBs, *p,p'*-DDE and HCB in New Bedford Infants' Cord Serum Samples. All final concentrations of PCBs, *p,p'*-DDE and HCB were reported after subtracting the level of these analytes in procedural blanks analyzed with each batch (Table 1). The levels of *p,p'*-DDE were generally higher than the levels of individual PCB congeners. The median ΣPCB was 0.39 ng/g; the median *p,p'*-DDE and HCB cord serum concentrations were 0.29 and 0.02 ng/g serum, respectively. Despite low concentrations, maternal age was strongly and significantly (p=0.0001) correlated with both ΣPCB (Spearman r=0.43) and *p,p'*-DDE (Spearman r=0.46) concentrations. The procedural blank concentrations were very close to the sample concentrations. For example, the ΣPCB in procedural blanks averaged 0.34 ng/g compared with a final mean sample ΣPCB concentration (*after* subtraction of the procedural blank) of 0.56 ng/g serum (Table 1). Furthermore, despite sensitive methods (most MDLs < 0.01 ng/g serum), only 13 of the 51 PCB congeners assayed were above the MDL for at least half of the study infants. We retained measurable values below the MDL in our dataset and assigned a value of zero only to samples in which the analyte was not detectable or was at or below the level of contamination in procedural blanks.

Discussion

Despite its importance, there is limited information available on the concentrations of PCBs, *p,p'*-DDE, and HCB in cord blood. This is one of the largest studies of the distribution of PCBs and pesticides in cord blood samples, and we were able to demonstrate that cord blood concentrations

of PCBs and organochlorine pesticides can be measured with high sensitivity (most MDLs < 0.01 ng/g serum) and minimal laboratory error (within run CVs of 7.5%, 5%, and 6% for ΣPCB, *p,p′*-DDE, and HCB, respectively).

Comparing the New Bedford infants' serum levels of PCBs, *p,p′*-DDE, and HCB with other birth cohorts is complicated by at least three sources of variability: (1) temporal declines in the population's exposure to organochlorine compounds since these chemicals were banned from industrial use in most nations, (2) differences among populations (both within and between countries) regarding risk factors for exposure to PCBs and pesticides, and (3) differences in analytic technique.

If we compare our results to studies using similar analytic techniques, the New Bedford cord serum concentrations were generally lower than those observed in relatively contemporaneous populations of infants from other industrialized nations. For example, Koopman-Esseboom and colleagues (1994) measured the levels of four individual congeners (IUPAC 118, 138, 153, and 180) in cord blood samples and reported an average sum of PCBs among their Dutch infants of 0.45 ng/g. In our study the mean sum of the same congeners was 0.26 ng/g. Among western populations, the Dutch are considered to be at substantially increased risk for PCB exposure as evidenced by breast milk levels. However, for less chlorinated PCB congeners (for example, congeners 99 and 118) study samples had concentrations comparable to those seen in other populations, including groups at risk for high dietary PCB exposure. Of note, the contaminated harbor sediment has a relatively high proportion of less chlorinated PCB congeners (Brown and Wagoner, 1990). Thus, although the overall ΣPCB in study infants was not higher than concentrations available for infants living elsewhere in the United States and Europe, the relative predominance of less chlorinated congeners was generally consistent with the characteristics of the contaminated site.

Acknowledgements

Support for this research was provided by NIEHS P42-ES05947.

References

1. Brown, J.F. and R.E. Wagoner. PCB movement, dechlorination, and detoxification in the Acushnet River Estuary. 1990. Environ. Toxicol. Chem. 9:1215-1233.
2. Centers for Disease Control (CDC). Polychlorinated biphenyl determination at parts-per-billion level in serum. Laboratory Update 81-108, (L.L. Needham, undated).
3. Koopman-Esseboom, C., M. Huisman, N. Weisglas-Kuperus, C.G. Van der Paauw, J.G.M. Tuinstra, E.R. Boersma, and P.J.J. Sauer. 1994. PCBs and dioxin levels in plasma and human milk of 418 Dutch women and their infants. Predictive value of PCB congener level in maternal plasma for fetal and infant exposure to PCBs and dioxins. Chemosphere 28:1721-1731.

Concentrations (ng/g serum) of PCBs, p,p'-DDE, and HCB in cord serum samples from 755 New Bedford area infants

Pesticide or PCB	MDL[†]	% samples ≥MDL[†]	Mean (all samples)	Mean (samples≥MDL)[†]	Range
p,p-DDE	0.070	96%	0.475	0.492	0 - 14.93
HCB	0.016	66%	0.028	0.034	0 - 0.66
ΣPCB[*]	NA[‡]	NA[‡]	0.559	NA[‡]	0.07 - 18.14
8	0.006	16%	0.006	0.027	0 - 0.28
16	0.002	14%	0.002	0.015	0 - 0.79
28	0.021	16%	0.021	0.091	0 - 2.05
49	0.004	10%	0.003	0.016	0 - 0.30
66	0.009	16%	0.009	0.045	0 - 1.37
74	0.008	91%	0.039	0.042	0 - 1.20
87	0.005	11%	0.002	0.013	0 - 0.14
95	0.007	10%	0.005	0.031	0 - 0.48
99	0.018	51%	0.032	0.050	0 - 1.42
101	0.005	47%	0.011	0.021	0 - 1.09
105	0.008	28%	0.009	0.025	0 - 0.66
118	0.011	89%	0.055	0.061	0 - 2.05
128	0.003	10%	0.001	0.009	0 - 0.09
138	0.013	88%	0.062	0.069	0 - 0.90
141	0.004	13%	0.003	0.019	0 - 0.43
149	0.006	11%	0.007	0.057	0 - 1.17
153	0.014	90%	0.088	0.096	0 - 1.34
156	0.010	30%	0.010	0.024	0 - 0.43
167	0.003	53%	0.005	0.008	0 - 0.08
170	0.018	37%	0.024	0.054	0 - 0.43
171	0.004	23%	0.003	0.009	0 - 0.15
177	0.001	55%	0.003	0.005	0 - 0.05
180	0.014	78%	0.046	0.056	0 - 0.40
183	0.003	50%	0.005	0.008	0 - 0.04
187	0.008	52%	0.014	0.023	0 - 0.17
194	0.002	62%	0.005	0.008	0 - 0.05
195	0.002	37%	0.002	0.004	0 - 0.03
196/203	0.003	55%	0.006	0.009	0 - 0.07
199	0.026	29%	0.029	0.077	0 - 1.49

[*] Sum of all PCB congeners evaluated. Individual PCB congeners are identified by IUPAC number.

[†] Method detection limit based on the average sample size (3 grams). Congeners were not included in Table if they were detected (>MDLs) in less than 10% of samples.

[‡] Not applicable.