# POLYCHLORINATED BIPHENYLS (PCBS) IN SERUM OF CHILDREN LIVING IN THE VICINITY OF THE FORMER PCB PRODUCTION FACILITY IN ANNISTON, ALABAMA

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**Abstract**
We measured levels of 35 ortho-substituted polychlorinated biphenyl (PCB) congeners in a sample of 321 children from Anniston, Alabama. Demographic data and blood samples were collected in 2005-2006 from children aged 10 to 17 years who participated in the Neuro-PCB study, part of the Anniston PCB Health project conducted by the Anniston Environmental Health Research Consortium and the Agency for Toxic Substances and Disease Registry (ATSDR). Seven congeners (PCBs 99, 118, 153, 138-158, 187, 180, and 170) were measured above the detection limit in about 90% of participants. Mean sum of the three most predominant congeners PCBs 153, 138-158, and 180 was 25.0 ng/g lipid, with a range of 1.1-426 ng/g lipid, representing about 68% of the sum of the seven PCB congeners (mean=36.7 ng/g lipid, range 2.6-603 ng/g lipid). PCB levels reported here were similar to the NHANES (2001-2) study levels for the 12 to 19 year old age category.

**Introduction**
Polychlorinated biphenyls (PCBs) were produced in Anniston, Alabama, by Monsanto/Solutia Corporations between the early 1930s and 1975; over 1.5 billion pounds were manufactured. The city of Anniston, Alabama, has about 24,000 inhabitants and is located some 90 miles west of Atlanta, Georgia. In the early 1990s, community concerns over potential health effects due to PCB exposure led to the involvement of the U.S. Environmental Protection Agency and several litigation efforts. The Anniston Environmental Health Research Consortium which included participants from 13 academic institutions and community representatives was formed in 2003 by the ATSDR. The goal of the Consortium was to characterize the environmental exposure to PCBs in Anniston residents and examine associations with potential adverse health outcomes such as diabetes and neurobehavioral development in children. One of the Consortium's projects was the Neuro-PCB study conducted in collaboration with the University of Alabama. Children and their parents were enrolled and tested for neurobehavioral effects potentially associated with PCBs. The current study presents the exposure assessment, consisting of serum PCB data from the children who participated in this project.

**Methods**
Blood samples were collected in 2005-2006 from children 10 to 17 year old that were residents of Anniston, Alabama. Two milliliters of sera was sent to the laboratory within 2 weeks of the blood collection where it was stored at -70°C until chemical analysis. In total, 321 samples were collected. Two samples initially failed to meet quality control requirements and were reanalyzed. Thirty five ortho-substituted PCBs were measured in serum by CDC/NCEH laboratory by high-resolution gas chromatography/isotope-dilution high-resolution mass spectrometry (HRGC/ID-HRMS). The analytical methodology employed for the measurement of PCBs in serum has been reported, and hence is only briefly described below.[1] Serum specimens (2mL) were fortified with $^{13}C_{12}$-labeled

internal standards and diluted with concentrated formic acid and water using a 215 liquid handler (Gilson Inc.; Middleton, WI) for automation. Automated solid phase extraction (SPE) and silica: silica/sulfuric acid lipid degradation were performed on the Rapid Trace SPE work station (Caliper Life Sciences Inc.; Hopkinton, MA). Samples were injected into a Hewlett-Packard 6890 gas chromatograph equipped with a DB-5ms capillary column (30m x 0.25 mm x 0.25 μm film thickness) coupled to a Thermo Finnigan MAT95 XP mass spectrometer operated in EI mode using selected ion monitoring (SIM) at 10,000 resolving power. The concentration of each analyte was calculated from its linear calibration curve. Study specimens were analyzed in batches of 24 specimens intermixed with quality control (QC; n=3) and method blank samples (n=3), in accordance with the previously published procedure.[1] All data were reviewed using comprehensive quality assurance and quality control (QA/QC) procedures and the analytical results were reported on both a whole-weight and lipid-adjusted basis. We present only lipid-adjusted results in this paper. Serum total lipids were calculated using an enzymatic "summation" method using triglyceride and total cholesterol measurements.[2] All means were calculated using the limit of detection divided by the square root of 2 substituted for measurements below the detection limit.[3]

**Results and Discussion**

Generally low levels of PCBs were measured in a large sample (n=321) of 10 to 17 year old participants of the Neuro-PCB Study in Anniston, Alabama. Among the 35 PCB congeners measured, seven PCB congeners 99, 118, 153, 138-158, 187, 180, and 170, were measured above the limit of detection in over 87% of subjects (Table 2). The mean sum of 7 congeners was 36.7 ng/g lipid, median 20.1 ng/g lipid, with a range of 2.6 to 603 ng/g lipid. PCBs 74, 146, were detected in about 60% of participants and 3 congeners, PCBs 199, 196, 194, were detected in about 50% of the participants. Two mono-ortho substituted, dioxin-like congeners PCBs 118 and 105 were detected in levels above the detection limit in about 20% of participants. We did not calculate the sum of 35 congeners as 17 out of 35 congeners had more than 60% of measurements below the detection limit. The three predominant congeners (PCBs 153, 138-158, and 180) accounted for about 68% of the total for the sum of 7 congeners with a mean of 25.0 ng/g lipid, median 20.1 ng/g lipid, and a range of 1.1-426 ng/g lipid (Table 2). Congeners 153, 138-158, and 180 were highly correlated with the sum of 3 and 7 congeners (all $r>0.95$). Similarly, mono-ortho congeners 105 and 157 were highly correlated with PCB 153 ($r>0.65$ and $r>0.90$, respectively).

Preliminary investigations show that age was a significant predictor of PCB levels even with the range of only 10 to 17 years (p-value 0.02). Younger children had lower PCB levels than older children but there was no difference between the two older age groups (geometric means, sum of 7 congeners; 18.9 ng/g lipid for 10 to 12 year old; 24.9 ng/g lipid for 13-14 year old, and 24.9 ng/g lipid for 15-17 year old, p-values for difference 0.04 and 0.02, respectively). Similarly, after adjustment for age, children who resided in Anniston less than 5 years had lower PCB levels (14.6 ng/g lipid) than children who lived there longer (23.3 ng/g lipid for 5-10 years, and 24.1 ng/g lipid for more than 10 years, p-values for difference 0.01 and 0.003, respectively) but there was little difference between the 5-10 years and more than 10 years categories (p-value 0.77). African-American children had higher PCB levels than children who were White (26.8 ng/g lipid versus 16 ng/g lipid, p value <0.01), but both these levels were at background range and a large proportion of parents did not report their child's race (21.3 ng/g lipid for non-reported race).

PCBs measured in this study in samples collected in 2005-6 were similar to those reported for children of comparable age group, 12-19 year old, enrolled in the NHANES, 2001-2002 cohort.[4] 90$^{th}$

percentiles for congeners 153, 138-158, and 180 were similar in Anniston and the NHANES samples, 25.2 ng/g lipid versus 23.1 ng/g lipid, 18.5 ng/g lipid versus 17.0 ng/g lipid, and 13.6 ng/g lipid versus 12.2 ng/g lipid, respectively (Table 3). Much higher levels were observed in the serum collected in 2001-2 from children also living in the vicinity of a former PCB production facility in Slovakia (236 ng/g lipid, 149 ng/g lipid, and 212 ng/g lipid for PCBs 153, 138, and 180; 8-10 year old).[5] It should be noted that the PCB production continued for more than a decade longer in Slovakia than in the United States, and higher consumption of locally grown food in Slovakia may account for higher PCB levels observed.

In conclusion, background levels of PCBs were found in samples collected from children in Anniston, Alabama, where PCBs were manufactured in large quantities from the 1930s to the 1970s. These results suggest that bioaccumulation of PCBs in children through exposure to local environmental deposits did not translate into elevated PCB levels. Within the background range, we observed higher PCB levels in older children and after adjustment for age, associations with the length of residence and race.

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**References**

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**Table 1.** Demographic characteristics of children, Neuro-PCB Study, Anniston, Alabama.

| Variable | | N=321 |
|---|---|---|
| Age[a], Mean (Range) years | | 13.3 (10.4-16.5) |
| Race | African-American | 154 (48%) |
| | White | 76 (23%) |
| | American Indian | 2 ( 1%) |
| | Multi-Racial | 5 ( 2%) |
| | Unreported | 84 (26%) |
| Resident of Anniston | <5 years | 34 (11%) |
| | 5-10 years | 92 (29%) |
| | >10 years | 168 (52%) |
| | Unreported | 27 ( 8%) |

[a.] Age as of Jan 1, 2006.

**Table 2.** PCBs congeners in 321 children enrolled in Anniston Neuro-PCB study (ng/g lipid).

| PCB congener | % >LOD[a] | Mean | Median | GeoMean | 90% | Max |
|---|---|---|---|---|---|---|
| PCB 99 | 96.0 | 2.7 | 1.6 | 1.8 | 5.3 | 39.8 |
| PCB 118 | 99.1 | 3.6 | 2.3 | 2.5 | 7 | 37.4 |
| PCB 153 | 98.4 | 10.8 | 5.6 | 6.1 | 25.2 | 193 |
| PCB 138-158 | 97.2 | 8.6 | 5.0 | 5.1 | 18.5 | 128 |
| PCB 187 | 87.2 | 3.2 | 1.3 | 1.4 | 7.3 | 60.4 |
| PCB 180 | 98.8 | 5.6 | 2.7 | 3.1 | 13.6 | 105 |
| PCB 170 | 86.9 | 2.2 | 1.1 | 1.2 | 5.2 | 39.3 |
| Sum of 3 PCBs [b] | | 25.0 | 13.6 | 14.6 | 52.7 | 426 |
| Sum of 7 PCBs [c] | | 36.7 | 20.1 | 22.2 | 80.8 | 603 |

[a] % >LOD - Percent above the limit of detection; [b] Sum of 3 PCBs – congeners 153, 138-158, and 180; [c] Sum of 7 PCBs – congeners 99, 118, 153, 138-158, 187, 180, and 170.

**Table 3.** Comparison of 90$^{th}$ percentiles and 95% confidence intervals in samples from Anniston, Alabama (2005-6) and NHANES (2001-2) samples (ng/g lipid).

| | Anniston, 2005-6 | NHANES, 2001-2 |
|---|---|---|
| Age group (years) | 10-17 | 12-19 |
| Number | 321 | 748 |
| 90$^{th}$ Percentile (95% CI) ng/g lipid | | |
| PCB 153 | 25.2 (19.5-30.2) | 21.2 ( 17.4-26.7) |
| PCB 138-158 | 18.5 (14.4-24.9) | 17.0 ( 13.7-20.2) |
| PCB 180 | 13.6 (10.6-16.1) | 12.2 (<LOD-14.8)[a] |
| Sum 3 PCBs (138-158, 153 & 180) | 52.7 (44.1-70.3) | 50.5 ( 40.3-60.9) |

[a] LOD – limit of detection.