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Serum concentrations of polychlorinated biphenyls (PCBs) in participants of the Anniston Community Health Survey☆,☆☆

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Abstract

Serum concentrations of 35 ortho-substituted polychlorinated biphenyl congeners (PCBs) were measured in 765 adults from Anniston, Alabama, where PCBs were manufactured between 1929 and 1971. As part of the Anniston Community Health Survey (ACHS), demographic data, questionnaire information, and blood samples were collected from participants in 2005–2007. Forty-six percent of study participants were African-American, 70% were female, and the median age was 56 years. The median concentration of the sum of 35 PCB congeners (ΣPCBs) was 528 ng/g lipid, with a 90th percentile of 2600 ng/g lipid, minimum of 17.0 ng/g lipid, and maximum of 27,337 ng/g lipid. The least square geometric mean ΣPCBs was more than 2.5 times higher for African-American participants than for White participants (866 ng/g lipid vs. 331 ng/g lipid); this difference did not change materially after adjustment for age, sex, body mass index (BMI) and

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Conflict of interest statement

J.R. Olson has served as an expert witness for the plaintiffs in legal actions relating to exposure of residents of Anniston, Alabama, to PCBs. The other authors declare that they have no competing interests.

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current smoking. In spite of large differences in absolute PCB levels, relative contributions of individual congeners to ΣPCBs were quite similar between race groups. Nevertheless, while percent contributions to ΣPCBs for most of the most abundant penta- to heptachlorobiphenyls were higher among African-Americans, the percentages were higher in Whites for the lower-chlorinated PCBs 28 and 74 and for octa- to decachlorinated PCBs. No major differences were observed in geometric mean ΣPCBs between women and men when adjusted for age, race, BMI and current smoking (516 ng/g lipid vs. 526 ng/g lipid). Principal component analysis revealed groups of co-varying congeners that appear to be determined by chlorine substitution patterns. These congener groupings were similar between ACHS participants and the National Health and Nutrition Examination Survey (NHANES) 2003–04 sample of the general United States population, despite ACHS participants having serum concentrations of ΣPCBs two to three times higher than those in comparable age and race groups from NHANES.

Keywords

Polychlorinated biphenyls; Anniston; Exposure

1. Introduction

Anniston, Alabama, is the site of a production facility that produced polychlorinated biphenyls (PCBs) from 1929 to 1971. In 1935 the facility was purchased by the Monsanto Company, which owned and operated the plant until 1997. PCB production in the Anniston plant ceased in 1971. Monsanto also owned the only other facility that produced PCBs in the United States (Krummrich plant, Sauget, Illinois). Fairly detailed data on sales volumes were published (Nisbet and Sarofim, 1972), and the EPA estimates that 1.4 billion pounds was produced in total (EPA, 1976; ATSDR, 2000a). It is estimated that both facilities produced about the same amount of PCBs.

Specific details on production, sources of PCB emissions into the environment, or PCB-containing waste sites in Anniston are based on court documents from the series of litigations in Anniston (1996–2003) as published by the lay press (Grunwald, 2001; Love, 2007). Direct discharges of liquid waste containing PCBs from the plant to sewers and discharge ditches leading to Snow Creek seemed to be the main source of contamination in Anniston. Monsanto estimated surface water discharges to range from less than a pound per day up to 250 lb (114 kg) per day. In 1970, the average discharge was estimated to be 88 lb (40 kg) per day, and PCB releases to air may have amounted to 0.5–2 lb (0.2–0.9 kg) per day (Grunwald, 2001). Monsanto undertook extensive efforts to reduce surface water discharges from 1969 through the end of production in 1971 (Durfee, 1976). During PCB production in Anniston, there were no federal or state regulations governing the manufacture, sale, distribution, disposal or cleanup of PCBs. An estimated 1 million pounds of PCB-containing solid and liquid waste was deposited in unlined and previously uncapped landfills south and west of the Monsanto facility. PCBs may have dispersed in Anniston via air transport (Hermanson et al., 2003; Hermanson and Johnson, 2007) and movement of contaminated soils and water (ADPH, 1996; ATSDR, 2000b,c).

Ingestion of contaminated food products, especially fish and livestock, is the most important pathway of PCB exposure in non-occupationally exposed persons (Hovinga et al., 1992; Humphrey and Budd, 1996; Kreiss et al., 1981; Schecter et al., 2001; Startin, 1994). Exposures by way of inhalation and dermal contact have also been studied (DeCaprio et al., 2005; Löffler and van Bavel, 2000) and generally contribute much less to the body burden. In Anniston, records of high PCB contamination of locally raised hogs, chickens and other animals were presented during litigation (Chemical Industry Archives, 2001; Love, 2007). The contamination of local fish (above the U.S. Food and Drug Administration's published tolerance of 2 ppm [edible portion]; 21CFR109.30, 2012) resulted in "no consumption" fish advisories in the 1990s that are still in effect (ADPH, 1995, 1996, 2011).

In 1995, ATSDR evaluated whether PCB levels in residents of Anniston were elevated in a sample of 103 volunteers living in the immediate vicinity of the plant (ADPH, 1996). Total PCB blood levels (based on Aroclor standards) ranged from <5 ppb to 303 ppb (ng/g whole weight); 31 of 103 persons had undetectable levels, while 5 persons had levels above 100 ppb. Age was a major determinant of PCB levels; 61% of those older than 50 years had levels above 20 ppb, while no person younger than 28 had PCB levels above 20 ppb.

Data collected between 1996 and 1998 from more than 3000 plaintiffs in one of the Anniston litigation efforts (*Abernathy vs. Monsanto*) were submitted for ATSDR review in 1999 (ATSDR, 2000b). PCB concentrations in almost half of the blood specimens collected were below the detection limit (3 to 5 ng/g whole weight). The samples were analyzed for total PCBs based on Aroclor standards. Over 17% of the participants had PCB levels above 20 ppb (ng/g whole weight). Data verification and quality control were limited in the dataset provided to the agency. Whereas earlier ATSDR assessments did not include congener-specific PCB results, a smaller study (Hansen et al., 2003) identified up to 47 congeners or pairs of congeners in one or more of 12 blood samples collected from the Anniston residents in 2000. The sum of these PCBs ranged from 0.68 ng/g whole weight in an 8-year-old child to 71.6 ng/g whole weight in a 69-year-old woman.

The results of exposure investigations that documented elevated levels of PCBs in some Anniston residents and environmental matrices caused community concerns over potential health effects due to exposure. In response to those concerns and following congressional hearings, ATSDR funded the Anniston Environmental Health Research Consortium to conduct exposure and health studies in Anniston. The results from human health studies of environmental PCB exposure provide some evidence of associations between PCB serum levels and a variety of health outcomes, including diabetes and its precursors (Codru et al., 2007; Langer et al., 2002; Lee et al., 2007, 2011; Rylander et al., 2005; Vasiliu et al., 2006; Wang et al., 2005), hypertension (Everett et al., 2008; Ha et al., 2009), adverse thyroid and metabolic health signs (Langer et al., 2009; Persky et al., 2001; Turyk et al., 2007), immune system effects (Heilmann et al., 2006; Park et al., 2008), and some cancers (De Roos et al., 2005; Hardell et al., 2004). The results from Anniston on PCBs and health outcomes such as hypertension, blood pressure, and diabetes (Goncharov et al., 2010, 2011; Silverstone et al., 2012) are generally supportive of these findings. Evaluations of prospective cohort studies of PCBs and associations with health outcomes found suggestive support for diabetes

(Taylor et al., 2013), inconclusive findings for thyroid outcomes (Salay and Garabrant, 2009), and a lack of evidence for breast cancer (Laden et al., 2001).

The goal of the present study was to characterize PCB exposure in a refined assessment by measuring 35 ortho-substituted PCB congeners in serum samples collected from 18- to 93-year-old Anniston residents living in close proximity to the former PCB production facility and to provide percentile distributions of ΣPCBs and individual PCB congener profiles.

2. Methods

2.1. Study design and population

The Anniston Community Health Survey (ACHS) is a cross-sectional community-based study of residents of Anniston, Alabama (population of approximately 24,000). The study was reviewed and approved by the University of Alabama Institutional Review Board. At CDC, analyses were conducted as technical assistance and in compliance with the Research Determination procedure; an exemption from the CDC IRB was obtained. A pool of 3320 eligible addresses was randomly selected from a commercial list of all residential properties in Anniston with intentional oversampling (two thirds of all eligible) of residences in west Anniston—the location of the former PCB manufacturing facility (Silverstone et al., 2012). Oversampling in west Anniston resulted in a stratified sample that facilitated enrollment of residents with residences closer to the plant and thus with higher potential for PCB exposure.

Current Anniston residents 18 years of age and older were eligible to participate and 1823 households were successfully contacted. Written informed consent was obtained from all participants at the time of the household survey and the survey questionnaire was administered by a trained interviewer. A total of 1110 randomly selected participants – one per household – completed the interview, resulting in a survey response rate of 39% (1110/2831 = 0.39. (2831 = 3320 total addresses—489 vacant or nonresidential addresses.)); 774 of those volunteered to provide blood samples during 2005–2007. The age, sex, and race distributions of those who did not provide blood samples did not differ materially from those of the participants who did. A standardized questionnaire solicited information on demographics; sex-specific and general health histories; nutritional, occupational, and health behaviors data; environmental perceptions; and litigation knowledge. We present only selected demographic and health behavior data in this paper.

2.2. Chemical analyses

Ten milliliters of blood (fasting) was collected from each participant by venipuncture as part of a study office visit in 2005–2007. After centrifuging, 2 mL of serum from each participant was frozen at the ACHS study office in Anniston. The specimens were then sent on dry ice to CDC's National Center for Environmental Health (NCEH) laboratory (Atlanta, GA), where they were stored at −70 °C until chemical analysis. Eight samples failed to meet quality control requirements and were discarded.

Thirty-five ortho-substituted PCBs were measured in serum by CDC's NCEH laboratory using high-resolution gas chromatography/isotope-dilution high-resolution mass

spectrometry (HRGC/ID-HRMS) as reported previously (Sjödin et al., 2004). The congeners measured were PCBs 28, 44, 49, 52, 66, 74, 87, 99, 101, 105, 110, 118, 128, 138–158, 146, 149, 151, 153, 156, 157, 167, 170, 172, 177, 178, 180, 183, 187, 189, 194, 195, 196–203, 199, 206, and 209 (Supplemental Table 1). Briefly, serum specimens (2 mL) 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) using silica and silica/sulfuric acid lipid degradation were performed on the Rapid Trace SPE work station (Caliper Life Sciences Inc.; Hopkinton, MA). Samples were then injected into a Hewlett-Packard 6890 gas chromatograph equipped with a DB-5 ms capillary column (30 m × 0.25 mm× 0.25 μm film thickness) coupled to a Thermo Finnigan MAT95 XP mass spectrometer operated in EI mode using selected ion monitoring at 10,000 resolving power. The concentration of each analyte was calculated from its calibration curve. Analytical results were reported on both a whole-weight and lipid-adjusted basis. Serum total lipids were calculated using an enzymatic “summation” method using triglyceride and total cholesterol measurements (Bernert et al., 2007). Median limits of detection (LOD) ranged from 0.4 ng/g lipid to 1.3 ng/g lipid (Supplemental Table 1). Repeatability of measurements was excellent; the coefficients of variations between blinded QC replicate samples ranged from 2.4 to 11.2 for each congener.

PCB congeners have several possible patterns of chlorine atom substitution around two phenyl rings that determine their chemical and toxicological properties; non-ortho, mono-ortho, di-, tri- and tetra-ortho chlorine substitutions are possible (Safe, 1993; Hansen, 1998). Out of 209 possible congeners, these 35 were chosen for analysis because of their relevance in human exposure assessment from multiple exposure pathways and because they represent the majority of steady-state and episodic PCB congeners that are selectively found in humans (Hansen, 2001). Most of the congeners measured in the Anniston sample were di- and higher ortho-substituted congeners. We also measured mono-ortho-substituted PCBs 101, 118, 156, 157, 167, and 189, which are dioxin-like and act through the aryl hydrocarbon receptor (AhR) as dioxins do (Hansen, 1998; IARC, 1997). Mono-ortho-substituted congeners have much weaker dioxin-like activity than do the non-ortho co-PCB congeners (Van den Berg et al., 2006).

2.3. Statistical analysis

Congener-specific LODs were reported from the laboratory for each participant based on serum specimen volume and weight. For calculation of geometric means (GM) and selected percentiles, any concentration that was less than the congener- and participant-specific LOD and had no measured value (i.e., a value of zero) was assigned a value equal to the LOD divided by the square root of 2 (Hornung and Reed, 1990). When measured concentrations below the LODs were reported from the laboratory, those measurements (rather than substituted values) were included in the dataset and analyses because a reported result is the best available estimate of the true value (ASTM, 1989). If the proportion of results below the LODs was greater than 40%, geometric means were not calculated for that congener. For each congener, if the median LOD was above a given percentile estimate, the percentile was denoted as “<LOD” and not reported.

The participants were classified by self-identification as African-American or White. We computed the sum of 35 PCBs (ΣPCBs) on both a lipid-adjusted (ng/g lipid) and whole-weight basis (ng/g whole weight). For estimates of ΣPCBs, the individual congeners with non-detectable results were assigned a value of LOD/√2. There were very few missing results for individual congeners, which were not counted in the PCB totals. Percentiles of distribution were compared among race and age groups by examining whether or not the 95% confidence intervals overlap, which is a conservative method of rejecting the null hypothesis (Schenker et al., 2002).

Congener-specific results are presented by race and by sex as well, with the percentage each congener contributes toward ΣPCBs and the ratio of the level of each congener to that of PCB 153. Least square geometric means from the analyses of covariance (SAS/STAT® 9.1) are presented unless denoted otherwise. Geometric means were adjusted for age, BMI, current smoking status, and either race or sex, respectively, for ease of comparison by race and sex, as well as with results from other studies.

Principal component analysis (PCA) was also used to explore relationships between serum concentrations of individual PCB congeners in this sample. After use of PCA to reduce the dimensionality of the dataset by transforming it to a set of new, uncorrelated eigenvectors (principal components) (Johnson et al., 2002; Joliffe, 2002), PCA results were displayed in a scores plot to show distribution of the original data by the principal components, as well as in a loadings plot to show groups of co-varying congeners. For the PCA, PCB concentrations were standardized by dividing the concentration of each congener by ΣPCBs; the data were then centered by subtracting their averages and scaled to unit variance. These transformations were conducted in order to prevent high-concentration congeners from dominating the analysis (Johnson et al., 2002). All PCA and plots were generated using SIMCAP (Umetrics; Umeå, Sweden).

3. Results

Demographic characteristics of participants stratified by race are summarized in Table 1. The study sample was 46% African-American and 70% female. Mean age was about 54 years (range of 18 to 92) for African-American participants and 56 years (range of 19 to 93) for White participants. Body mass index (BMI), number of pregnancies, and proportion of current smokers were similar across race groups. White participants resided in Anniston for about 5 years longer and drank less alcohol in the past 30 days. A higher proportion of African-American participants (89.2%) than of Whites (79.4%) lived in west Anniston, the neighborhood adjacent to the former PCB plant. The mean distance of the current residence from the plant was 2.4 km for African-Americans and 4.2 km for Whites. More African-Americans than Whites completed at least a high school education.

Geometric means, medians, and selected percentiles for 35 measured ortho-substituted PCB congeners are shown in Table 2 for African-Americans and in Table 3 for Whites. Most PCB congeners were measured with detection rates of at least 80% of results above the individual LOD in both race groups (26 congeners among African-Americans and 24 among Whites). Among the White participants, several congeners were detected with less than 26%

of results above the congener-specific LODs. Among African-Americans, PCBs 44 and 49 were the only two congeners for which such small proportions of results (approximately 13% and 11%, respectively) were above the LODs. (Supplemental Table 1 lists the median LOD for each congener.)

Median PCB congener levels were generally two to three times higher in African-Americans than in Whites for most congeners. The seven most abundant congeners, with the highest median levels, contributed over 65% of ΣPCBs (70.9% in African-American participants, 66.2% in Whites). Among African-American participants, these congeners were PCBs 153 (contributing a relative 22% to ΣPCBs), 138–158 (15.3%), 180 (12.9%), 187 (6.2%), 118 (5.8%), 170 (4.8%), and 199 (3.9%). For White participants, these congeners were PCBs 153 (19.8%), 180 (16.5%), 138–158 (13.1%), 187 (4.9%), 199 (4.6%), 194 (4%), and 196–203 (3.3%).

The geometric mean for ΣPCB congeners was 516 ng/g lipid and the median was 528 ng/g lipid (range = 17.0–27,337 ng/g lipid) (Table 4). African-Americans had ΣPCBs that were more than two times higher than Whites had at the geometric mean (866 ng/g lipid vs. 331 ng/g lipid) and median, with ΣPCBs about three times higher at the 75th, 90th and 95th percentiles.

The results of the current study showed that PCB levels were age- and race-dependent. Lipid-adjusted geometric means and the 95% confidence intervals for ΣPCBs are presented by race for three age groups (20–39, 40–59, and ≥60 years old) in Table 5. African-Americans had higher geometric mean ΣPCBs than Whites did in each age group—two times higher in the youngest age group and almost three times higher in the older age groups. The highest ΣPCBs were observed in the oldest age group (≥60 years old) of African-Americans at a geometric mean of 1874 ng/g lipid, compared to 683.8 ng/g lipid among Whites.

No major differences by sex were observed, but after adjustment for age, BMI, race and current smoking, ΣPCBs was marginally higher in men than in women (526 ng/g lipid vs. 516 ng/g lipid) (Table 6). However, levels of lower-chlorinated congeners were somewhat higher in women, with almost twofold higher percentage contributions to ΣPCBs from congeners 74, 99, and 118. Levels of more highly chlorinated congeners were slightly lower in women.

The largest differences in PCB concentrations for any covariate studied were by race, with ΣPCBs almost three times higher among African-American participants than among Whites, after adjustment for age, BMI, sex, and current smoking (Table 6; Fig. 1). In general, the differences between concentrations of individual congeners were similar in proportion to the difference seen for ΣPCBs. We should note, though, that percentages of contribution to ΣPCBs and ratios to PCB 153 were similar across race groups and not uniformly higher in African-Americans (Table 6; Supplemental Fig. 1). For certain congeners, including low-chlorinated PCBs 28 and 74 and highly chlorinated PCBs 194, 195, 196, 199, 206, and 209 (9 or 10 chlorines each), percent contributions to ΣPCBs and ratios to PCB 153 were higher in White participants. While the percent contributions to ΣPCBs for most of the most

abundant penta- to heptachlorobiphenyls were similar but higher among African-Americans, the percentages were marginally higher in Whites for PCBs 156, 157, 170 and 180.

When stratified by race, no substantial differences by sex in ΣPCBs were observed in African-Americans (903 ng/g lipid for men vs. 849 ng/g lipid for women, $p = 0.67$) or Whites (349 ng/g lipid in men vs. 324 ng/g lipid for women, $p = 0.54$) (adjusted for age, results not shown).

Relationships between serum concentrations of individual PCB congeners were examined using PCA. Principal component 1 (PC1) appears to be strongly related to participant age (Fig. 2). Together, PC1 and PC2 account for 41% of the variance in these data. PC1 accounts for 27% and PC2 for 14% of the variance, respectively. The variance described cumulatively by adding additional principal components is shown in Supplemental Fig. 2.

The loading plot in Fig. 3 shows patterns in the congener profiles seen in the participants. It shows a tight grouping of congeners with eigenvector values for $PC1 > 0$ and for $PC2 < 0$ (Group A, in the bottom right quadrant), mainly including congeners with 2,5 substitution (PCBs 44, 49, 52, 101, an 151) and 2,3,6 substitution (PCBs 110, 149). Group A also includes the tri- and tetrachlorinated but more persistent PCBs 28, 66, and 74. Also on the right side ($PC1 > 0$) but in the upper quadrant ($PC2 > 0$), Group B mostly consists of penta- to heptachlorinated and 2,4,5-substituted congeners, including PCBs 99, 118, 138, 146, 153, 167, 183, and 187. Groups C1 and C2, on the left side of the plot ($PC1 < 0$), include more highly chlorinated congeners with the core 3,4,5 substitution: 3,4,5-substituted PCB 157; 2,3,4,5-substituted PCBs 156, 170, 180, 189, 194, 196, 199, 206, and 2,3,4,5,6-substituted 209. Also in Group C1 are 2,3,5,6 substituted PCBs 177 and 178. The separation between C1 and C2 appears to be on the basis of chlorination, with the most highly chlorinated congeners are in C2 and the rest in C1. The location of Groups C1 and C2 on the left side of Fig. 3 ($PC1 < 0$), matching the distribution of older participants across PC1 (Fig. 2), suggests that enrichment of Group C congeners is greatest in older participants; this pattern is most pronounced for Group C2, which contains the most highly chlorinated congeners.

4. Discussion

4.1. Anniston, Alabama, PCB production site

This manuscript adds to the body of literature that examines environmental exposures to PCBs in Anniston, Alabama (ATSDR, 2000b, 2006; Carpenter et al., 2003; Orloff et al., 2003; Hansen et al., 2003; Martín-Jiménez and Hansen, 2008). However, several aspects of the Anniston PCB site not specifically addressed in any of the previous documents are important to the context and conclusions of this study.

This study focused on residents who lived near the former plant, where the potential was expected to be highest for exposure to historical releases from the facility and to environmental PCB residues. During PCB production in Anniston, Monsanto produced a number of Aroclor mixtures (1016, 1221, 1232, 1242, 1248, 1254, 1260, 1262 + 1268, and possibly also 1270) for capacitor and transformer producers, as well as about 3000 other customers (Brown and Lawton, 2001). The Anniston population thus had the unique

potential to be exposed to a combination of PCBs from essentially all production and experimental Aroclors produced over time. By contrast, most other exposed populations in occupational settings or residential communities near industrial PCB waste sites were mostly exposed to a more limited spectrum of Aroclors (usually 3 or 4), with only one Aroclor mixture used during a given time period (Choi et al., 2006; Seegal et al., 2011; Wolff et al., 1982).

PCB production in Anniston also led to discharges of non-product PCBs into the environment at various stages of the manufacturing process, whether in spills, leaks into cooling water, or in solid waste as described for Monsanto's other PCB plant (Durfee, 1976). These discharges likely contained congeners or mixtures that do not match any Aroclor product profile. Disposing of Aroclor-grade product into landfills or wastewater made no commercial sense; this important point has often been overlooked in past studies of environmental PCB exposures. In any case, although analysis of Aroclor "fingerprinting" has been attempted at other sites (DeCaprio et al., 2005; Fitzgerald et al., 2007b), matching a specific pattern indicative of Aroclors that came from the Monsanto plant in Anniston to the congeners identified in the participants' serum would be difficult to do since we do not know the pattern.

4.2. PCB patterns observed in Anniston residents

The seven most abundant congeners in the study sample were PCBs 153, 138–158, 180, 187, 118, 170, and 199, although the order was slightly different for each race group. PCBs 153, 138–158, and 180 are considered to be the most consistently detected PCBs in human tissue around the world and present at the highest proportions relative to other congeners (Hansen, 1998). These highly chlorinated congeners are resistant to breakdown by the P450 enzyme system (Hansen, 1999; Safe, 1993), so their presence likely reflects lifetime exposures.

It could be argued that the most abundant congeners found in the Anniston participants were also present in large proportions in Aroclors 1260 and 1254 (Frame et al., 1996; ATSDR, 2000a). Specifically, those dominant among the African-American participants (PCBs 187, 118, 170, and 199) were components of Aroclors 1260 and 1254. The composition of Aroclor 1260 was up to 9.39% of PCB 153, 6.54% of PCB 138, 11.4% of PCB 180, 5.4% of PCB 187, 4.1% of PCB 170, 1.78% of PCB 199 and 0.48% of PCB 118 (ATSDR, 2000a). Although some of these congeners were also present in Aroclor 1248, each makes up less than 2.5% of the 1248 mixture. Notably, the lot of Aroclor 1254 in which PCB 118 was a major component (nearly 14%) was not produced in Anniston (ATSDR, 2000a). Meanwhile, the congeners more abundant among the White participants (PCBs 187, 199, 196–203, and 194) were found in Aroclor 1260 and to a far lesser extent (contributing less than 1% each) in Aroclor 1254. Due to widespread and long-term industrial use of PCBs in Anniston and elsewhere, and consequent contamination of food and environmental media, it is impossible to pinpoint specific origins of individual congeners in human samples.

Detailed PCB congener profiles of PCB-containing waste are rarely provided for soil and sediment samples from affected communities. Soil and sediment samples from Anniston have not been assessed for congener profiles (ATSDR, 2000b). However, previous studies

suggest very little direct contribution to PCB body burden from soil and dust exposure in general (Kimbrough et al., 2010) and in Anniston in particular (Orloff et al., 2003).

In order to explore further the patterns of congeners found in these Anniston residents, we used PCA to examine groups of co-varying congeners. As shown in the PCA loading plot for the first two principal components (Fig. 3), the groupings were most distinctly classified by the congeners' chlorination. As mentioned, congeners that are more highly chlorinated are generally more resistant to biotransformation and elimination from the body (Hansen, 1999; Safe, 1993; Seegal et al., 2011). Although there are exceptions to this broad observation (with PCB 28 and 74, for example, classified by Hansen (2001) as steady-state congeners), the general trend of longer half-lives for highly chlorinated congeners suggests that higher proportions of these congeners would be observed in older individuals, whereas the presence of less-chlorinated congeners – which are, generally, more readily eliminated – may reflect more recent exposure and so would be typically expected to contribute to ΣPCBs in greater proportions among younger individuals. Accordingly, the positions of Group A, C1 and C2 on the right and left sides of the loading plot align with the distribution of participants by age across PC1, with younger participants concentrated on the right and older ones on the left.

Beyond the distribution of the groups across PC1 by, in most cases, overall chlorination of congeners, several patterns emerged with regard to the positions of chlorine atoms on the grouped congeners. The underlying substitution patterns within congener groups are very similar between the groups identified in Fig. 3 and those found by Megson et al. (2013) in their application of PCA to the NHANES 2003–04 sample. Similar to Group 1 identified by Megson et al. (2013), our Group A included congeners with bonding in the 2,5, and 2,3,6 positions (PCBs 44, 49, 52, 101, 110, 149, and 151), which are likely to be quickly biotransformed and classified as episodic (Hansen, 2001). Group B included 2,4,5-substituted congeners, similar to those in Megson et al.'s (2013) Group 3. Differing from the position of Group 3 found by Megson et al. (2013) across both quadrants where $PC2 < 0$, our Group B is located where $PC2 > 0$ – where middle-aged participants are concentrated (Fig. 2) – and $PC1 < 0$. Their presence on the left side of the plot indicating enrichment in older participants, Groups C1 and C2 mainly included 2,3,4,5 and 2,3,4,5,6-substituted congeners, corresponding to those in Megson et al. (2013) Group 2. However, our Groups C1 and C2 are distinctly separated across quadrants, with the most-chlorinated congeners in C2, whereas Group 2 identified by Megson et al. (2013) falls almost entirely within a single quadrant ($PC1 < 0$, $PC2 > 0$). The similarities between congener groupings seen in Fig. 3 and by Megson et al. (2013) suggest that chlorine substitution patterns and structural activity of PCB congeners dictate these groupings, resulting in only minor differences despite much higher serum concentrations of PCBs in these Anniston residents than in the NHANES 2003–04 sample and demographic differences.

The descriptive groupings in our analyses do not necessarily correspond with toxicity groupings defined by structure and activity, such as those recently reviewed by Warner et al. (2012). We must emphasize that the groupings shown in Fig. 3 are suggestive and dependent on this specific sample's distribution, not confirmatory.

4.3. Internal comparisons

Among all participants (n = 765), the geometric mean concentration for ΣPCBs was 516 ng/g lipid with a range of 17.0 ng/g lipid to 27,337 ng/g lipid (Table 4). Higher concentrations of ΣPCBs were found in African-Americans than in White participants (Table 4) and much higher concentrations of ΣPCBs were found in the older age groups than among younger participants (Table 5). Geometric mean ΣPCBs in the ≥60-year-old groups were nearly three times higher for African-Americans than for Whites. In those under 40 years old, the difference was smaller, but ΣPCBs were still higher among African-American participants than in Whites.

In agreement with our assessment of unadjusted data, we did not see major differences in congener profiles by race. Stronger similarities than differences are observed with regard to proportion contributed to ΣPCBs and ratio to PCB 153 (Supplemental Fig. 1), in contrast with large differences in absolute levels (Fig. 1). This similarity in profiles appears to suggest that the sources of PCB exposure did not differ substantially between race groups. We calculated the ratio of the level of each congener to that of PCB 153 as it may provide a crude standardization (relative proportion) of PCB congener levels, because PCB “totals” presented in research studies vary widely and are highly inconsistent in regard to the congeners measured or included. These results may indicate that the retention of some individual congeners is tightly controlled in humans, irrespective of the sources of PCBs—independent of absolute levels of exposure and demographic differences.

4.4. External comparisons

We compared ΣPCB concentrations in Anniston to the NHANES 2003–2004 results stratified on age and race (Patterson et al., 2009). ACHS participants had serum concentrations of ΣPCBs two to three times higher than did the general US population in comparable age and race groups from the NHANES 2003–2004 (Table 5). While for African-Americans, this increase in comparison to the NHANES was still evident in the youngest age group (20–39 years old), the concentrations in White ACHS participants in this age group were similar to and lower than that reported from NHANES (68.2 ng/g lipid vs. 82.5 ng/g lipid).

Caution is necessary when comparing PCB results from other studies because of differences in populations, time periods and analytical methods (Longnecker et al., 2003). Selection and composition of the Anniston sample clearly differed from NHANES even when compared to similar race and age groups. However, the time frames when data were collected for the present study (2005–2007) and NHANES 2003–2004 were similar, and analyses of the same 35 congeners were conducted at the same laboratory using similar analytical methods.

We also contrasted Anniston PCB congener data to results from an older population of the upper Hudson River community living in close proximity to PCB-contaminated sites and General Electric (GE) industrial facilities that used large amounts of Aroclors (Fitzgerald et al., 2007a, 2012), and to a cohort of capacitor manufacturing workers from the same area who provided valuable information on congener profiles and half-lives of PCBs after 28 years of follow-up (Seegal et al., 2011).

The geometric mean for the sum of 30 PCB congeners in the upper Hudson study population older than 55 (with at least 25 years of residence) was 473.4 ng/g lipid in samples collected in 2000–2002; levels in the comparison group (residents of towns upriver from the GE plants) were almost identical (Table 7; Fitzgerald et al., 2007a). Aroclor 1254 was used in nearby capacitor-making facilities from 1946 to 1953, followed by Aroclor 1242 from 1953 to 1970, and then mostly Aroclor 1016 from 1971 to 1977 (Fischbein et al., 1979). These PCB concentrations are lower than the geometric means in Anniston for the 40- to 60-year-old African-American group and the ≥60-year-old White group (Table 5). The levels of congeners 28 and 74 in the Hudson group are higher than in the Anniston sample, while the levels of most other congeners were similar, generally falling closer to the levels for White participants in Anniston than for African-Americans. The vast majority of upper Hudson participants were White (97%), with a mean age of 64 years (Fitzgerald et al., 2007a). The levels of many of the more highly chlorinated congeners were almost twice as high among African-American participants in Anniston as among the Hudson cohort.

In capacitor workers occupationally exposed to PCBs (Seegal et al., 2011), high overall serum PCB levels were found 28 years after the end of PCB use in production (Table 7). A pattern of elevated congener levels emerged as well, with the lightly chlorinated congeners 28 and 74 observed to be more abundant in the serum PCB compositions of the capacitor workers than in individuals of the general population in the same towns (Fitzgerald et al., 2007a; Seegal et al., 2011). Compared to the concentrations found in Anniston participants, concentrations of PCBs 28 and 74 were 2 to 3 times higher among the Hudson older resident cohort and about 10 times higher among the former capacitor workers. Those two congeners were present in the highest proportion in Aroclor 1016, which was used in the GE plant in 1973 to 1977. In addition to PCBs 28 and 74, PCBs 105, 118 and 156 were also considered to be uniquely indicative of occupational exposure to PCBs from Aroclor mixtures in capacitor plants (Wolff et al., 1982; Seegal et al., 2011).

However, concentrations of more highly chlorinated congeners among Anniston participants are similar to those seen in capacitor manufacturing workers (Seegal et al., 2011). In fact, adjusted geometric mean concentrations of PCBs 99, 118, 138, 146, 153, 167, 170, 180, 183, and 187 for African-American participants from Anniston are higher than those of the former capacitor workers (from whom serum was collected in 2003–2006). The occupationally related PCBs 105 and 156, along with PCB 177, were the only more highly chlorinated congeners for which serum concentrations were higher in the workers (Table 7).

4.5. Study strengths and limitations

The primary purpose of this report is to describe exposure among ACHS participants using congener-specific data and examine exposure by demographic factors such as age, sex, and race. A detailed evaluation of predictors of PCB exposure and possible exposure pathways is the focus of a separate report currently in production.

The current study improves upon previous exposure assessments in Anniston in several ways. First, households were selected as a large, random, stratified sample for the current study, whereas previous investigations involved self-selected volunteers (ADPH, 1996; ATSDR, 2000b) or small sample sizes (Hansen et al., 2003; Orloff et al., 2003). Due to

advances in analytical methodology, PCBs were detectable in the vast majority of serum samples in the present study; in past ATSDR reports, approximately one-third (1999) and half (2000b) of the samples had PCB levels below the LODs. Also, two earlier reports provided data on total PCBs only (ADPH, 1996; ATSDR, 2000b), while these congener-specific analyses provide a more comprehensive profile of human PCB exposure from multiple pathways.

Limitations include the possibility of bias introduced by distributions of the sample by sex and age that do not reflect the study area's population. After the original random selection of households to contact, a truly random sample of individual participants was not achieved; if the first person to be randomly selected from a household declined to participate, another adult in the household was selected at random to replace them. Women and older residents may be more likely to be at home for door-to-door surveys conducted during daytime working hours, which may explain the high percentages of female participants (70%) and those over age 60 (42%). Non-random selection of individuals within households resulted in underrepresentation of men and participants under age 40 in this sample, which limits generalizability of the results. However, the ACHS sample was not intended to be representative of the demographics of the city of Anniston, in that it was designed to oversample in west Anniston.

5. Conclusion

Age- and race-stratified comparisons revealed that the geometric means for ΣPCBs were 2 to 3 times higher for African-Americans than for Whites from Anniston who lived in areas near the former PCB production facilities. In spite of large absolute differences in ΣPCBs, the PCB congener patterns were quite similar across race groups, suggesting similar sources or routes of exposure but a higher quantity of exposure in African-American participants. Geometric mean serum concentrations for ΣPCBs were found to be 1.5 to 3.5 times higher in the ACHS participants than in most comparable age and race groups from the NHANES 2003–2004 sample. The results of PCA showed that congeners appeared to covary in serum within this sample based on chlorine substitution patterns, similar to groupings reported for NHANES 2003–2004. ACHS participants over 40 years of age had ΣPCB concentrations similar to those in other groups living close to PCB-contaminated waste sites elsewhere in United States, but PCB concentrations for African-American participants in Anniston were closer to those of former capacitor workers occupationally exposed to PCBs. Much lower concentrations in participants younger than 40 years old suggest limited exposure to PCBs from environmental deposits in Anniston after the end of PCB production, which can only be confirmed in follow-up exposure analyses.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Abbreviations

PCBs	polychlorinated biphenyls
ACHS	Anniston Community Health Survey
LOD	limit of detection
GM	geometric mean
CI	confidence interval
ATSDR	Agency for Toxic Substances and Disease Registry
CDC	Centers for Disease Control and Prevention
EPA	U.S. Environmental Protection Agency
NHANES	National Health and Nutrition Examination Survey

References

21 Code of Federal Regulations 109.30. Tolerances for polychlorinated biphenyls (PCB's). Apr 1. 2012

ADPH (Alabama Department of Public Health). Health consultation: Monsanto Company. Anniston, Calhoun County, Alabama: 1995. CERCLIS No. ALD004019048

ADPH (Alabama Department of Public Health). Health consultation: Cobbtown/Sweet Valley Community PCB exposure investigation. Anniston, Calhoun County, Alabama: 1996.

ADPH (Alabama Department of Public Health). [Accessed 01/27/2012] Alabama fish consumption advisory guidelines 2002-2011. 2011. http://adph.org/tox/index.asp?id=1360

ASTM (American Society for Testing and Materials). Committee on standards designation, D4210-89. ASTM; Philadelphia: 1989. p. 2-7.

ATSDR (Agency for Toxic Substances and Disease Registry). Toxicological profile for polychlorinated biphenyls (PCBs). U.S. Department of Health and Human Services; Atlanta: 2000a.

ATSDR (Agency for Toxic Substances, Disease Registry). Health consultation: evaluation of soil, blood & air data from Anniston, Alabama. Monsanto Company, Anniston, Calhoun County, Alabama. U.S. Department of Health and Human Services; Atlanta: 2000b. CERCLIS No. ALD004019048

ATSDR (Agency for Toxic Substances, Disease Registry). Health consultation: exposure investigation report. Monsanto Company, Anniston, Calhoun County, Alabama. U.S. Department of Health and Human Services; Atlanta: 2000c. CERCLIS No. ALD004019048

ATSDR (Agency for Toxic Substances, Disease Registry). Updated assessment of PCB exposures in Anniston, AL; Anniston PCB site, Anniston, Calhoun County, Alabama. U.S. Department of Health and Human Services; Atlanta: 2006. EPA Facility ID: ALD004019048

Bernert JT, Turner WE, Patterson DG Jr, Needham LL. Calculation of serum "total lipid" concentrations for the adjustment of persistent organohalogen toxicant measurements in human samples. Chemosphere. 2007; 68:824–31. [PubMed: 17408721]

Brown, JF., Jr; Lawton, RW. Factors controlling the distribution and levels of PCBs after occupational exposure. In: Robertson, LW.; Hansen, LG., editors. PCBs. University Press of Kentucky; Lexington, KY: 2001. p. 103-9.

Carpenter DO, Morris DL, Legator M. Initial attempts to profile health effects with types of exposures in Anniston, Alabama. Fresenius Environ Bull. 2003; 12:191–5.

Chemical Industry Archives, Environmental Working Group. [Accessed: February 15, 2013] Trial Transcript, Owens v. Monsanto CV-96-J-440-E, (N.D. Alabama April 5, 2001), pg. 551, line 1; a memo from E.S. Tucker to W.B. Papageorge, December 21, 1970 (both Monsanto, PCB results for hog's liver and fat samples). 2001. http://www.chemicalindustryarchives.org/dirtysecrets/annistonindepth/wildlife.asp

Choi AL, Levy JI, Dockery DW, Ryan LM, Tolbert PE, Altshul LM, et al. Does living near a Superfund site contribute to higher polychlorinated biphenyl (PCB) exposure? Environ Health Perspect. 2006; 114(7):1092–8. [PubMed: 16835064]

Codru N, Schymura MJ, Negoita S. Diabetes in relation to serum levels of polychlorinated biphenyls and chlorinated pesticides in adult Native Americans. Environ Health Perspect. 2007; 115(10): 1442–7. [PubMed: 17938733]

De Roos AM, Hartge P, Lubin JH, Colt JS, Davis S, Cerhan JR, et al. Persistent organochlorine chemicals in plasma and risk of non-Hodgkin's lymphoma. Cancer Res. 2005; 65(23):11214–26. [PubMed: 16322272]

DeCaprio AP, Johnson GW, Tarbell AM, Carpenter DO, Chiarenzelli JR, Morse GS, et al. Polychlorinated biphenyl (PCB) exposure assessment by multivariate statistical analysis of serum congener profiles in an adult Native American population. Environ Res. 2005; 98(3):284–302. [PubMed: 15910784]

Durfee, RL. Proceedings of the National Conference on Polychlorinated Biphenyls, Chicago, 1975. U.S. Environmental Protection Agency; Washington, DC: 1976. Production and usage of PCBs in the United States; p. 103-7.EPA-560/6-75-004

EPA (U.S. Environmental Protection Agency). PCBs in the United States: industrial use and environmental distribution. U.S. Environmental Protection Agency, Office of Toxic Substances; Washington, DC: 1976. NTIS PB252012

Everett CJ, Mainous AG, Frithsen IL, Player MS, Matheson EM. Association of polychlorinated biphenyls with hypertension in the 1999-2002 National Health and Nutrition Examination Survey. Environ Res. 2008; 108(1):94–7. [PubMed: 18606400]

Fischbein A, Wolff MS, Lilis R, Thornton J, Selikoff IJ. Clinical findings among PCB-exposed capacitor manufacturing workers. Ann N Y Acad Sci. 1979; 320:703–15. [PubMed: 110206]

Fitzgerald EF, Belanger EE, Gomez MI, Hwang SA, Jansing RL, Hicks HE. Environmental exposures to polychlorinated biphenyls (PCBs) among older residents of upper Hudson River communities. Environ Res. 2007a; 104:352–60. [PubMed: 17382313]

Fitzgerald EF, Hwang SA, Gomez M, Bush B, Yang BZ, Tarbell A. Environmental and occupational exposures and serum PCB concentrations and patterns among Mohawk men at Akwesasne. J Expo Sci Environ Epidemiol. 2007b; 17(3):269–78. [PubMed: 16736058]

Fitzgerald EF, Shrestha S, Gomez MI, McCaffrey RJ, Zimmerman EA, Kannan K, et al. Polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs) and neuropsychological status among older adults in New York. Neurotoxicology. 2012; 33(1):8–15. [PubMed: 22079442]

Frame GM, Cochran JW, Bowadt SS. Complete PCB congener distributions for 17 Aroclor mixtures determined by 3 HRGC systems optimized for comprehensive, quantitative, congener-specific analysis. J High Resolut Chromatogr. 1996; 19(12):657–68.

Goncharov A, Bloom M, Pavuk M, Birman I, Carpenter DO. Blood pressure and hypertension in relation to levels of serum polychlorinated biphenyls in residents of Anniston, Alabama. J Hypertens. 2010; 28(10):2053–60. [PubMed: 20644494]

Goncharov A, Pavuk M, Foushee HR, Carpenter DO, for the Anniston Environmental Health Research Consortium. Blood pressure in relation to concentrations of PCB congeners and chlorinated pesticides. Environ Health Perspect. 2011; 119(3):319–25. [PubMed: 21362590]

Grunwald, M. Monsanto Hid Decades Of Pollution But No One Was Ever Told. Washington Post: Jan 1. 2001

Ha MH, Lee DH, Son HK, Park SK, Jacobs DR. Association between serum concentrations of persistent organic pollutants and prevalence of newly diagnosed hypertension: results from the

National Health and Nutrition Examination Survey 1999-2002. J Hum Hypertens. 2009; 23:274–86. [PubMed: 18843279]

Hansen LG. Stepping backward to improve assessment of PCB congener toxicities. Environ Health Perspect. 1998; 106(Suppl. 1):171–89. [PubMed: 9539012]

Hansen, LG. Occurrence and disposition. Kluwer Academic Publishers; Boston: 1999. The ortho-side of PCBs.

Hansen, L. Identification of steady state and episodic PCB congeners from multiple pathway exposures. In: Robertson, LW.; Hansen, LG., editors. PCBs. University Press of Kentucky; Lexington, KY: 2001. p. 47-56.

Hansen LG, DeCaprio AP, Nisbet ICT. PCB congener comparisons reveal exposure histories for residents of Anniston, Alabama, USA. Fresenius Environ Bull. 2003; 12(2):181–90.

Hardell L, van Bavel B, Lindström G, Carlberg M, Eriksson M, Dreifaldt AC, et al. Concentrations of polychlorinated biphenyls in blood and the risk for testicular cancer. Int J Androl. 2004; 27(5): 282–90. [PubMed: 15379968]

Heilmann C, Grandjean P, Weihe P, Nielsen F, Budtz-Jørgensen E. Reduced antibody responses to vaccinations in children exposed to polychlorinated biphenyls. PLoS Med. 2006; 3(8):9.

Hermanson MH, Johnson GW. Polychlorinated biphenyls in tree bark near a former manufacturing plant in Anniston, Alabama. Chemosphere. 2007; 68:191–8. [PubMed: 17307226]

Hermanson MH, Scholten CA, Compher K. Variable air temperature response of gas-phase atmospheric polychlorinated biphenyls near a former manufacturing facility. Environ Sci Technol. 2003; 37:4038–42. [PubMed: 14524433]

Hornung RW, Reed DR. Estimation of average concentration in the presence of nondetectable values. App Occup Environ Hyg. 1990; 5(1):46–51.

Hovinga ME, Sowers M, Humphrey HE. Historical changes in serum PCB and DDT levels in an environmentally-exposed cohort. Arch Environ Contam Toxicol. 1992; 22(4):362–6. [PubMed: 1489385]

Humphrey HE, Budd ML. Michigan's fisheater cohorts: a prospective history of exposure. Toxicol Ind Health. 1996; 12(3-4):499–505. [PubMed: 8843566]

IARC (International Agency for Research on Cancer). Polychlorinated Dibenzo-*para*-dioxins and polychlorinated dibenzofurans. IARC Monogr Eval Carcinog Risks Hum. 1997:69.

Johnson, GW.; Ehrlich, R.; Full, W. Principal component analysis and receptor models in environmental forensics. In: Murphy, BL.; Morrison, RD., editors. Introduction to environmental forensics. Academic Press; 2002.

Joliffe, IT. Principal component analysis. 2nd ed.. Springer; 2002.

Kimbrough RD, Krouskas CA, Carson ML, Long TF, Bevan C, Tardiff RG. Human uptake of persistent chemicals from contaminated soil: PCDD/Fs and PCBs. Regul Toxicol Pharmacol. 2010; 57:43–54. [PubMed: 20035816]

Kreiss K, Zack MM, Kimbrough RD, Needham LL, Smrek AL, Jones BT. Association of blood pressure and polychlorinated biphenyl levels. JAMA. 1981; 245(24):2505–9. [PubMed: 6785463]

Laden F, Collman G, Iwamoto K, Alberg AJ, Berkowitz GS, Freudenheim JL, Hankinson SE, Helzlsouer KJ, Holford TR, Huang HY, Moysich KB, Tessari JD, Wolff MS, Zheng T, Hunter DJ. 1,1-Dichloro-2,2-bis(pchlorophenyl)ethylene and polychlorinated biphenyls and breast cancer: combined analysis of five U.S. studies. J. Natl. Cancer Inst. 2001; 93(10):768–76. [PubMed: 11353787]

Langer P, Tajtáková M, Guretzki HJ, Kocan A, Petrík J, Chovancová J, et al. High prevalence of anti-glutamic acid decarboxylase (anti-GAD) antibodies in employees at a polychlorinated biphenyl production factory. Arch Environ Health. 2002; 57(5):412–5. [PubMed: 12641181]

Langer P, Kočan A, Tajtáková M, Sušsienková K, Rádiková Ž , Koška J, et al. Multiple adverse thyroid and metabolic health signs in the population from the area heavily polluted by organochlorine cocktail (PCB, DDE, HCB, dioxin). Thyroid Res. 2009; 2(3)

Lee DH, Lee IK, Jin SH, Steffes M, Jacobs DR Jr. Association between serum concentrations of persistent organic pollutants and insulin resistance among nondiabetic adults: results from the National Health and Nutrition Examination Survey 1999-2002. Diabetes Care. 2007; 30(3):622–8. [PubMed: 17327331]

Lee DH, Lind PM, Jacobs DR Jr, Salihovic S, van Bavel B, Lind L. Polychlorinated biphenyls and organochlorine pesticides in plasma predict development of type 2 diabetes in the elderly: the prospective investigation of the vasculature in Uppsala Seniors (PIVUS) study. Diabetes Care. 2011; 34(8):1778–84. [PubMed: 21700918]

Löffler G, van Bavel B. Potential pathways and exposure to explain the human body burden of organochlorine compounds: a multivariate statistical analysis of human monitoring in Würzburg, Germany. Chemosphere. 2000; 40(9-11):1075–82. [PubMed: 10739048]

Longnecker MP, Wolff MS, Gladen BC, Brock JW, Grandjean P, Jacobson JL, et al. Comparison of polychlorinated biphenyl levels across studies of human neurodevelopment. Environ Health Perspect. 2003; 111(1):65–70. [PubMed: 12515680]

Love D. My city was gone: one American town's toxic secret, its angry band of locals, and a $700 million day in court. Harper Perennial Publishers. 2007

Martín-Jiménez, T.; Hansen, LG. Toxicokinetic extrapolation of PCB exposure in Anniston, Alabama. In: Robertson, LW.; Hansen, LG., editors. PCBs: human and environmental disposition and toxicology. University of Illinois Press; Champaign, IL: 2008. p. 70-9.

Megson D, O'Sullivan G, Comber S, Worsfold PJ, Lohan MC, Edwards MR, et al. Elucidating the structural properties that influence the persistence of PCBs in humans using the National Health and Nutrition Examination Survey (NHANES) dataset. Sci Total Environ. 2013; 461-462:99–107. [PubMed: 23712120]

Nisbet IC, Sarofim AF. Rates and routes of transport of PCBs in the environment. Environ Health Perspect. 1972; 1:21–38. [PubMed: 17539082]

Orloff KG, Dearwent S, Metcalf S, Kathman S, Turner W. Human exposure to polychlorinated biphenyls in a residential community. Arch Environ Contam Toxicol. 2003; 44:125–31. [PubMed: 12434227]

Park HY, Hertz-Picciotto I, Petrik J, Palkovicova L, Kocan A, Trnovec T. Prenatal PCB exposure and thymus size at birth in neonates in Eastern Slovakia. Environ Health Perspect. 2008; 116(1):104–9. [PubMed: 18197307]

Patterson DG Jr, Wong LY, Turner WE, Caudill SP, Dipietro ES, McClure PC, et al. Levels in the U.S. population of those persistent organic pollutants (2003-2004) included in the Stockholm Convention or in other Long-Range Transboundary Air Pollution Agreements. Environ Sci Technol. 2009; 43(4):1211–8. [PubMed: 19320182]

Persky V, Turyk M, Anderson HA, Hanrahan LP, Falk C, Steenport DN, et al. The effects of PCB exposure and fish consumption on endogenous hormones. Environ Health Perspect. 2001; 109:1275–83. [PubMed: 11748036]

Rylander L, Rignell-Hydborn A, Hagmar L. A cross-sectional study of the association between persistent organochlorine pollutants and diabetes. Environ Health. 2005; 4:28–33. [PubMed: 16316471]

Safe S. Toxicology, structure-function relationship, and human and environmental health impacts of polychlorinated biphenyls: progress and problems. Environ Health Perspect. 1993; 100:259–68. [PubMed: 8354174]

Salay E, Garabrant D. Polychlorinated biphenyls and thyroid hormones in adults: a systematic review appraisal of epidemiological studies. Chemosphere. 2009; 74(11):1413–9. [PubMed: 19108870]

Schecter A, Cramer P, Boggess K, Stanley J, Päpke O, Olson J, et al. Intake of dioxins and related compounds from food in the US population. J Toxicol Environ Health A. 2001; 63:101–18. [PubMed: 11393797]

Schenker N, Gentleman JF, Rose D, Hing E, Shimizu IM. Combining estimates from complementary surveys: a case study using prevalence estimates from national health surveys of households and nursing homes. Public Health Rep. 2002; 117(4):393–407. [PubMed: 12477922]

Seegal RF, Fitzgerald EF, Hills EA, Wolff MS, Haase RF, Todd AC, et al. Estimating the half-lives of PCB congeners in former capacitor workers measured over a 28-year interval. J Expo Sci Environ Epidemiol. 2011; 21:234–46. [PubMed: 20216575]

Silverstone AE, Rosenbaum PF, Weinstock RS, Bartell SM, Foushee HR, Shelton C, et al. Polychlorinated biphenyl (PCB) exposure and diabetes: results from the Anniston Community Health Survey. Environ Health Perspect. 2012; 120(5):727–32. [PubMed: 22334129]

Sjödin A, Jones RS, Lapeza CR, Focant JF, McGahee EE, Patterson DG. Semiautomated high-throughput extraction and cleanup method for the measurement of polybrominated diphenyl ethers, polybrominated biphenyls, and polychlorinated biphenyls in human serum. Anal Chem. 2004; 76:1921–7. [PubMed: 15053652]

Startin, JR. Dioxins in food. In: Schecter, A., editor. Dioxins and health. Plenum Press; New York: 1994. p. 115-37.

Taylor KW, Novak RF, Anderson HA, Birnbaum LS, Blystone C, Devito M, et al. Evaluation of the association between persistent organic pollutants (POPs) and diabetes in epidemiological studies: a national toxicology program workshop review. Environ Health Perspect. 2013; 121(7):774–83. [PubMed: 23651634]

Turyk ME, Anderson HA, Persky VW. Relationships of thyroid hormones with polychlorinated biphenyls, dioxins, furans, and DDE in adults. Environ Health Perspect. 2007; 115:1197–203. [PubMed: 17687447]

Van den Berg M, Birnbaum LS, Denison M, De Vito M, Farland W, Feeley M, et al. The 2005 World Health Organization reevaluation of human and mammalian toxic equivalency factors for dioxins and dioxin-like compounds. Toxicol Sci. 2006; 93(2):223–41. [PubMed: 16829543]

Vasiliu O, Cameron L, Gardiner J, Deguire P, Karmaus W. Polybromated biphenyls, polychlorinated biphenyls, body weight, and incidence of adult-onset diabetes mellitus. Epidemiology. 2006; 17(4):352–9. [PubMed: 16755267]

Wang S-L, Su P-H, Jong S-B, Guo YL, Chou W-L, Päpke O. In utero exposure to dioxins and polychlorinated biphenyls and its relations to thyroid function and growth hormone in newborns. Environ Health Perspect. 2005; 113(11):1645–50. [PubMed: 16263525]

Warner J, Osuch JR, Karmaus W, Landgraf JR, Taffe B, O'Keefe M, et al. Common classification schemes for PCB congeners and the gene expression of *CYP17, CYP19, ESR1 and ESR2*. Sci Total Environ. 2012; 414:81–9. [PubMed: 22119029]

Wolff MS, Fischbein A, Thornton J, Rice C, Lilis R, Selikoff IJ. Body burden of polychlorinated biphenyls among persons employed in capacitor manufacturing. Int Arch Environ Health. 1982; 49:199–208.

HIGHLIGHTS

- We measured levels of 35 PCBs in 765 adults living near a former PCB production plant.
- Median PCB levels were about 2 to 3 times higher in African-Americans than in Whites.
- Relative contributions of congeners to sum of PCBs were similar across race groups.
- No major differences were observed in the sum of PCBs between women and men.

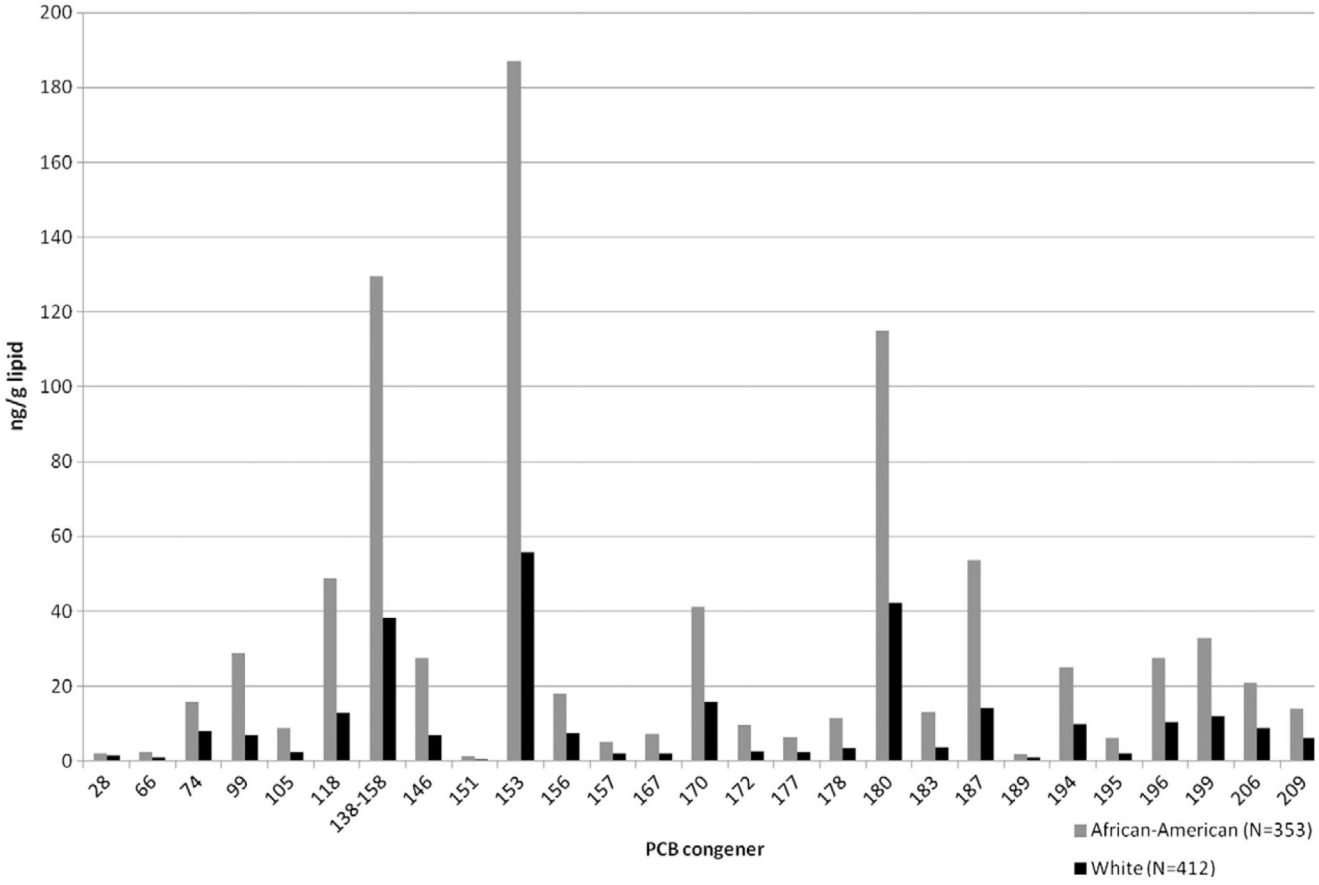

Fig. 1.

Least square geometric mean serum PCB concentrations (ng/g, lipid-adjusted) for selected congeners, by race. Geometric means (GM) were adjusted for age, sex, BMI, and current smoking status. All congeners shown here had >60% of results above LOD.

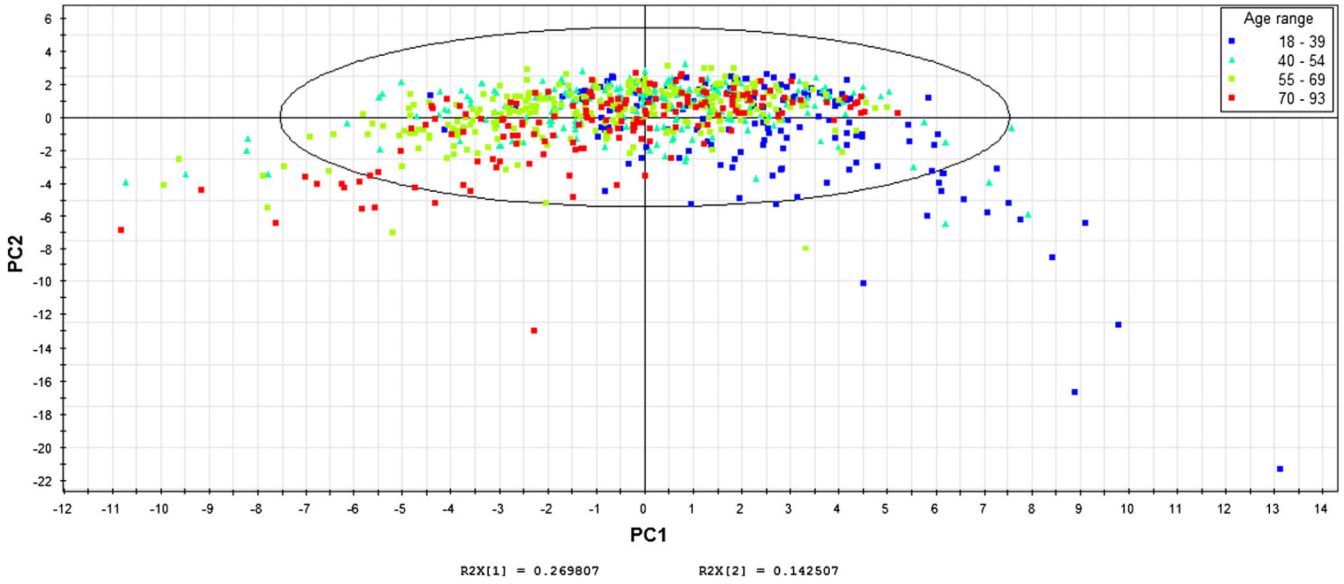

Fig. 2.

PCA score plot of PC1 (accounting for 32.6% of variance) and PC2 (accounting for 15.3% of variance), with age group indicated by color. Younger participants are most concentrated in the bottom right corner of the plot (PC1 > 0, PC2 near or <0), while older participants dominate the center and right side of the plot. PC1 accounts for 27.0% of variance and PC2 accounts for 14.3% of variance.

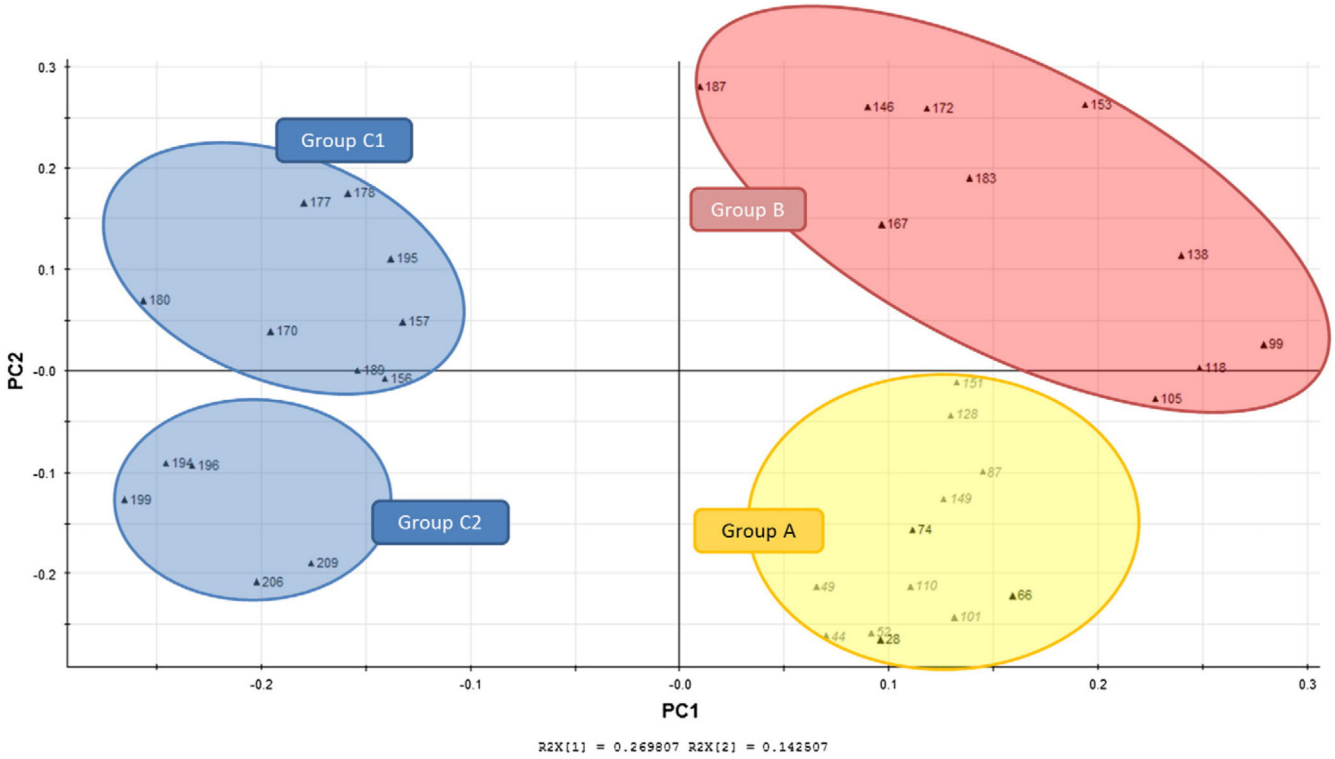

Fig. 3.

PCA loading plot showing groupings of congeners based on chlorination. Shown in gray and labeled in italics are the 9 congeners with less than 75% of measurements above the analytical limits of detection. PC1 accounts for 27.0% of variance and PC2 accounts for 14.3% of variance.

Table 1

Demographic characteristics (mean ± SE or percent) of the participants of the Anniston Community Health Survey.

Characteristic (number of missing responses)[a]	African-Americans (n = 353)	Whites (n = 412)	Total (n = 765)
	Mean ± std. Error		
Age in years	53.6 ± 0.8	55.9 ± 0.8[b]	54.9 ± 0.6
BMI—kg/m^2	31.9 ± 0.4	30.7 ± 0.4[c]	31.2 ± 0.3
Total years of residence	26.4 ± 1.0	31.1 ± 1.0[c]	28.9 ± 0.7
Residential distance from the plant—km	2.4 ± 0.06	4.2 ± 0.1[c]	3.4 ± 0.07
	Percentage (non-missing)		
Female	70.3%	70.2%	70.2%
Age groups—years			
18–39	17.9%	19.4%[d]	18.7%
40–59	45.6%	34.7%	39.7%
≥60	36.5%	45.9%	41.6%
BMI classification			
Normal <25	17.4%	22.6%[d]	20.2%
Overweight 25–29	22.5%	29.4%	26.2%
Obese ≥30	60.1%	48.1%	53.6%
Reside in west Anniston	89.2%	79.4%[d]	83.9%
Current smoker	31.3%	31.8%	31.6%
Drank alcohol in the past 30 days	37.1%	21.4%[d]	28.6%
High school graduate	71.3%	66.6%	68.8%
Ever eaten fish from Snow Creek, Choccolocco Creek, or Lake Logan Martin	74.8%	50.5%[d]	61.7%
Ever exposed at a job to PCBs	27.0%	27.3%	27.1%

Variables with missing values: BMI (2); residential distance from plant (25); current smoking (9); drank alcohol in the past 30 days (13); high school completion (6); ever eaten locally raised hogs, chicken or other livestock (51); ever eaten fish from Snow Creek, Choccolocco Creek, or Lake Logan Martin (63); ever eaten local clay (34); ever eaten locally grown vegetables (9); ever occupationally exposed to PCBs (109); ever exposed to transformer fluids (45); ever occupationally exposed to ionizing radiation (70); ever occupationally exposed to lead, boron, mercury or cadmium (81); ever occupationally exposed to fertilizers (37); ever occupationally exposed to pesticides (40); ever occupationally exposed to herbicides (52); and ever occupationally exposed to solvents (50).

[a] A response of don't know or refusal is counted as missing.

[b] $p < 0.05$ for African-Americans compared with Whites using *t*-test.

[c] $p < 0.05$ for African-Americans compared with Whites using analysis of covariance adjusting for age.

[d] $p < 0.05$ for African-Americans compared with Whites using Chi-square test of independence.

Table 2

PCB congeners in African-American participants of the ACHS (n = 353; ng/g lipid).

PCB	% >LOD	GM	Min	10th %	25th %	50th %	75th %	90th %	95th %	Max
28	88.1	2.0	<LOD	<LOD	1.1	1.9	3.4	5.6	7.4	34
44	12.8	–	<LOD	<LOD	<LOD	<LOD	<LOD	0.5	0.8	5
49	10.5	–	<LOD	<LOD	<LOD	<LOD	<LOD	0.4	1	22.1
52	33.1	–	<LOD	<LOD	<LOD	<LOD	0.6	1.8	3.3	62.3
66	90.9	2.4	<LOD	0.5	0.9	2.2	5.2	14.2	17.7	124
74	99.7	15.1	<LOD	2.9	6.5	16.4	34.7	62.8	88.7	503
87	58.1	1.0	<LOD	<LOD	<LOD	0.8	2.5	5.1	7.6	60
99	100	27.6	1.3	5.6	11.4	30.3	70.8	127	163	1470
101	39.7	–	<LOD	<LOD	<LOD	<LOD	0.9	3.3	5.8	63.6
105	92.9	8.4	<LOD	1.3	2.7	8.9	24.3	52.2	78.8	716
110	35.7	–	<LOD	<LOD	<LOD	<LOD	0.7	1.9	3.8	38.9
118	100	46.5	1.5	6.4	15.4	51.1	133	274	390	3250
128	47.0	–	<LOD	<LOD	<LOD	<LOD	1.8	4.8	6.8	131
138–158	100	123	5.8	25.1	53.3	135	296	501	731	4890
146	99.2	25.7	<LOD	4.7	11.6	29.2	71.1	117	168	905
149	52.1	–	<LOD	<LOD	<LOD	0.5	1.7	3.8	6.3	44.6
151	61.8	0.82	<LOD	<LOD	<LOD	1	3	7.2	11.2	58.8
153	100	176	5.8	35	77.8	194	459	718	1070	5650
156	99.2	16.6	<LOD	3.2	8.1	18	36.8	73	99.5	471
157	93.2	4.6	<LOD	0.7	2.2	5	10.4	20.2	27.2	147
167	94.1	6.8	<LOD	1	2.5	7.5	20.3	33.6	44.3	304
170	99.7	38.4	<LOD	6.8	20.3	42.1	85.4	165	253	981
172	93.9	6.0	<LOD	0.9	3	6.6	14.4	27.8	44	162
177	98.3	9.2	<LOD	1.6	4	9.7	22.7	41.9	63	326
178	95.5	10.5	<LOD	1.6	5.3	12	26.1	49.3	81.3	310
180	100	106	3.2	17.8	54.7	114	250	489	778	2770
183	98.3	12.4	<LOD	2.3	5.6	13.1	31.9	51.8	78	494
187	100	50.1	1.7	8	24.1	54.9	129	235	372	1720

PCB	% >LOD	GM	Min	10th %	25th %	50th %	75th %	90th %	95th %	Max
189	81.6	1.8	<LOD	<LOD	0.8	1.9	3.6	7.5	11.5	38.9
194	97.2	22.7	<LOD	3.3	11.7	25.4	56.2	109	176	755
195	92.4	5.6	<LOD	0.8	2.8	6.3	13.6	25.5	38.4	139
196–203	98.9	25.3	<LOD	4.1	12.7	28.9	60	108	177	520
199	98.9	30.0	<LOD	4.3	14	34.7	73.9	151	225	771
206	99.2	19.2	<LOD	2.6	7.8	19.5	51.7	107	172	1220
209	98.3	12.4	<LOD	1.3	3.9	12.7	39.5	90.6	152	1640

% > LOD—Percent over the limit of detection.

GM—Geometric mean.

Table 3

PCB congeners in White participants of the ACHS (n = 412; ng/g lipid).

PCB	% >LOD	GM	Min	10th %	25th %	50th %	75th %	90th %	95th %	Max
28	83.1	1.7	<LOD	<LOD	0.9	1.5	2.9	5.8	9.7	116
44	8.7	–	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	0.6	14.7
49	4.1	–	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	6.1
52	13.8	–	<LOD	<LOD	<LOD	<LOD	<LOD	0.6	1.1	13.9
66	75.8	1.0	<LOD	<LOD	0.4	0.9	1.8	3.7	5.6	37.9
74	99.8	8.4	<LOD	2.3	4.4	9.1	16.9	25.9	36.5	125
87	32.2	–	<LOD	<LOD	<LOD	<LOD	0.6	1.2	2.0	13.3
99	98.8	7.0	<LOD	1.7	3.4	6.8	14.8	28.8	42.2	364
101	25.2	–	<LOD	<LOD	<LOD	<LOD	0.15	0.8	1.4	5
105	92.5	2.3	<LOD	0.6	0.9	2.2	5.0	10.3	15.5	106
110	12.1	–	<LOD	<LOD	<LOD	<LOD	<LOD	0.4	1.0	3.8
118	99.8	13.2	<LOD	2.9	5.3	12.8	30.2	60.1	94.7	580
128	8.2	–	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	0.8	7.5
138–158	99.5	39.8	<LOD	8.5	20.6	44.2	88	149	207	1440
146	96.9	7.2	<LOD	1.4	3.9	8.2	15.2	31.7	46.3	300
149	13.6	–	<LOD	<LOD	<LOD	<LOD	<LOD	0.5	1.0	5.6
151	11.6	–	<LOD	<LOD	<LOD	<LOD	<LOD	0.5	1.1	5.4
153	99.8	58.5	<LOD	11.5	31	67	126	225	319	2,470
156	98.1	8.0	<LOD	1.6	4.7	9.5	16.5	28	38.4	157
157	87.9	2.2	<LOD	<LOD	1.2	2.4	4.3	8.0	10.3	45.3
167	83.5	2.3	<LOD	<LOD	1.1	2.6	5.3	9.8	14.1	90.4
170	100	16.7	0.6	3.10	10.4	20.2	34.1	58.9	78.1	426
172	86	2.4	<LOD	<LOD	1.4	3	5	9.6	12.4	68.7
177	89.8	2.6	<LOD	<LOD	1.2	2.8	5.9	11	16.2	134
178	89.1	3.7	<LOD	<LOD	1.9	4.5	8.1	16.9	22.6	132
180	99.5	45.1	<LOD	7.3	27.2	55.8	97.9	168	248	1340
183	92.3	3.9	<LOD	0.6	2	4.5	8.8	15.7	21.9	214
187	98.8	15	<LOD	2.9	8	16.7	33.2	68.1	105	695

PCB	% >LOD	GM	Min	10th %	25th %	50th %	75th %	90th %	95th %	Max
189	71.7	0.9	<LOD	<LOD	<LOD	0.9	1.6	3.1	4.1	16.6
194	94.9	10.8	<LOD	1.3	6.1	14	25.4	45.9	70.3	590
195	85.7	2.9	<LOD	<LOD	1.1	2.6	5	9	13.7	77.3
196–203	98.3	11.2	<LOD	2	6	13.4	24.1	45.3	71.7	543
199	98.1	13.0	<LOD	1.9	6.4	15.4	31.4	60.9	102	1050
206	98.3	9.6	<LOD	1.3	3.7	9.8	24	60	116	788
209	93.5	6.7	<LOD	0.7	2	7.1	21.4	59.7	131	533

% >LOD—percent over the limit of detection.

GM—geometric mean.

Table 4

Geometric means, selected percentiles and 95% confidence intervals for the sum of 35 PCBs (ΣPCBs) in the participants of the Anniston Community Health Survey (ng/g lipid and ng/g whole weight).

			Selected percentile (95% confidence intervals)							
	Sample size	**GM[a] (95% CI)**	**Min.**	**10th%**	**25th%**	**50th%**	**75th%**	**90th%**	**95th%**	**Max.**
(ng/g lipid)										
Total[b]	765	516 (472–564)	17.0	90.0 (75.5–106)	233 (208–266)	528 (475–589)	1172 (1041–1362)	2600 (2315–2945)	3703 (3327–4883)	27,337
African-American	353	866 (767–978)	40.1	159 (109–211)	409 (321–512)	948 (853–1081)	2141 (1681–2483)	3581 (3149–4734)	5312 (4490–8062)	27,337
White	412	331 (296–371)	17.0	70.2 (52.1–82.3)	185 (131–208)	372 (341–426)	680 (599–815)	1232 (1021–1479)	1768 (1467–2212)	10,025
(ng/g whole weight)										
Total	765	3.04 (2.83–3.34)	0.11	0.51 (0.42–0.59)	1.43 (1.28–1.63)	3.28 (2.96–3.66)	7.41 (6.53–8.56)	15.4 (13.6–17.2)	22.2 (19.4–27.5)	170.4
African-American	353	5.03 (4.44–5.69)	0.21	0.84 (0.56–1.11)	2.43 (1.76–2.92)	5.76 (4.73–6.63)	12.2 (10.6–14.2)	21.1 (18.9–24.8)	31.3 (24.1–37.6)	170.4
White	412	2.05 (1.82–2.30)	0.11	0.36 (0.30–0.47)	1.11 (0.86–1.34)	2.24 (2.04–2.50)	4.26 (3.74–4.86)	7.75 (6.66–9.89)	10.8 (9.84–14.5)	47.9

[a] GM—geometric mean.

[b] Median ages for total, African-American and White Anniston participants are 55,54, and 56 years, respectively.

Table 5

Geometric means for the sum of 35 PCBs and their corresponding 95% confidence limits in the Anniston Community Health Survey and NHANES 2003–2004 by race and age group (ng/g lipid).

Anniston 2005–2007	**20–39 years**[a]	**Age group 40–59 years**	**≥60 years**
African-American	175.1 (139.9–219.2)	870.9 (756.6–1002)	1874 (1602–2193)
White	68.8 (58.7–80.4)	306.1 (272.2–344.1)	683.8 (617.5–757.2)
NHANES 2003–2004[b]			
Non-Hispanic Black	83.9 (72.7–96.9)	246.6 (209.2–290.6)	630.1 (491.0–808.7)
Non-Hispanic White	82.8(76.5–89.7)	181.5 (161.9–203.3)	332.8 (312.8–354.1)

[a] Four persons younger than 20 years old included in the Anniston samples (1 was 18 years old, 3 were 19 years old).

[b] NHANES 2003–2004 results from Table S4 (supplement) in Patterson et al., 2009.

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Table 6

Least square geometric mean serum PCB concentrations (ng/g, lipid-adjusted) for selected congeners, by sex and race. Geometric means (GM) were adjusted for age, BMI, and current smoking status, as well as either race or sex, respectively. Congeners shown in Table 6 all had > 60% of results above LOD.

Congener	African-American participants (*N* = 353)			White participants (*N* = 412)			Male participants (*N* = 228)			Female participants (*N* = 537)		
	Adj. GM	% of ΣPCBs	Ratio to 153	Adj. GM	% of ΣPCBs	Ratio to 153	Adj. GM	% of ΣPCBs	Ratio to 153	Adj. GM	% of ΣPCBs	Ratio to 153
PCB 28	2.037	0.22%	0.011	1.662**	0.53%	0.030	1.547	0.29%	0.016	1.960**	0.38%	0.020
PCB 66	2.441	0.26%	0.013	0.958**	0.30%	0.017	1.192	0.23%	0.012	1.620**	0.31%	0.017
PCB 74	15.868	1.71%	0.085	8.089**	2.57%	0.145	7.964	1.51%	0.081	12.711**	2.46%	0.131
PCB 99	28.674	3.10%	0.153	6.840**	2.18%	0.123	11.792	2.24%	0.119	14.004*	2.71%	0.144
PCB 105	8.785	0.95%	0.047	2.218**	0.71%	0.040	2.884	0.55%	0.029	4.807**	0.93%	0.049
PCB 118	48.716	5.26%	0.260	12.740**	4.06%	0.229	16.756	3.19%	0.170	27.530**	5.34%	0.283
PCB 138–158	129.700	14.01%	0.693	38.118**	12.13%	0.684	63.785	12.13%	0.645	68.852	13.35%	0.707
PCB 146	27.366	2.96%	0.146	6.896**	2.20%	0.124	13.767	2.62%	0.139	12.796	2.48%	0.131
PCB 151	1.213	0.13%	0.006	0.358**	0.11%	0.006	0.611	0.12%	0.006	0.639	0.12%	0.007
PCB 153	187.084	20.21%	1.000	55.724**	17.74%	1.000	98.835	18.80%	1.000	97.338	18.87%	1.000
PCB 156	17.880	1.93%	0.096	7.544**	2.40%	0.135	11.320	2.15%	0.115	11.237	2.18%	0.115
PCB 157	4.929	0.53%	0.026	2.092**	0.67%	0.038	2.964	0.56%	0.030	3.180	0.62%	0.033
PCB 167	7.213	0.78%	0.039	2.184**	0.70%	0.039	3.149	0.60%	0.032	4.119**	0.80%	0.042
PCB 170	41.265	4.46%	0.221	15.778**	5.02%	0.283	26.941	5.12%	0.273	23.745*	4.60%	0.244
PCB 172	9.689	1.05%	0.052	2.527**	0.80%	0.045	4.665	0.89%	0.047	4.738	0.92%	0.049
PCB 177	6.481	0.70%	0.035	2.285**	0.73%	0.041	4.191	0.80%	0.042	3.520**	0.68%	0.036
PCB 178	11.431	1.24%	0.061	3.464**	1.10%	0.062	6.828	1.30%	0.069	5.720*	1.11%	0.059
PCB 180	115.054	12.43%	0.615	42.271**	13.46%	0.759	75.401	14.34%	0.763	64.120**	12.43%	0.659
PCB 183	13.087	1.41%	0.070	3.733**	1.19%	0.067	6.711	1.28%	0.068	6.672	1.29%	0.069
PCB 187	53.672	5.80%	0.287	14.185**	4.52%	0.255	28.867	5.49%	0.292	25.299	4.90%	0.260
PCB 189	1.907	0.21%	0.010	0.881**	0.28%	0.016	1.423	0.27%	0.014	1.198**	0.23%	0.012

Congener	African-American participants (*N* = 353)			White participants (*N* = 412)			Male participants (*N* = 228)			Female participants (*N* = 537)		
	Adj. GM	% of ΣPCBs	Ratio to 153	Adj. GM	% of ΣPCBs	Ratio to 153	Adj. GM	% of ΣPCBs	Ratio to 153	Adj. GM	% of ΣPCBs	Ratio to 153
PCB 194	24.871	2.69%	0.133	9.973**	3.17%	0.179	18.921	3.60%	0.191	13.880**	2.69%	0.143
PCB 195	6.000	0.65%	0.032	2.172**	0.69%	0.039	3.780	0.72%	0.038	3.353	0.65%	0.034
PCB 196	27.462	2.97%	0.147	10.485**	3.34%	0.188	19.115	3.64%	0.193	15.362**	2.98%	0.158
PCB 199	32.866	3.55%	0.176	12.088**	3.85%	0.217	22.890	4.35%	0.232	17.865**	3.46%	0.184
PCB 206	21.078	2.28%	0.113	8.839**	2.81%	0.159	14.409	2.74%	0.146	12.753	2.47%	0.131
PCB 209	13.814	1.49%	0.074	6.104**	1.94%	0.110	9.781	1.86%	0.099	8.577	1.66%	0.088
ΣPCBs	925.527			314.164**			525.795			515.889		

*p < 0.01.

**p < 0.05.

Table 7

Mean serum PCB concentrations (ng/g, lipid-adjusted) for selected congeners.

Congener	Upper Hudson River study area, 2000–2002[a] (*N* = 128) ppb			Former capacitor workers in New York, 2003–2006[b]; men (*N* = 129) ng/g			Former capacitor workers in New York, 2003–2006[b]; women (*N* = 112) ng/g		
	Adj. GM	% of total PCBs	Ratio to 153	Unadj. GM	% of total PCBs	Ratio to 153	Unadj. GM	% of total PCBs	Ratio to 153
PCB 28	5.5	1.2	0.07	10	0.8	0.07	20	2.3	0.20
PCB 56	NR	–	–	20	1.7	0.14	10	1.2	0.10
PCB 66	NR	–	–	20	1.7	0.14	30	3.5	0.30
PCB 74	29.9	6.3	0.38	160	13.4	1.14	80	9.3	0.80
PCB 99	11.4	2.4	0.14	20	1.7	0.14	20	2.3	0.20
PCB 101	NR	–	–	50	4.2	0.36	40	4.7	0.40
PCB 105	5.2	1.1	0.07	10	0.8	0.07	10	1.2	0.10
PCB 118	22.6	4.8	0.29	30	2.5	0.21	30	3.5	0.30
PCB 138	60.1	12.7	0.76	120	10.1	0.86	80	9.3	0.80
PCB 146	NR	–	–	10	0.8	0.07	10	1.2	0.10
PCB 153	78.7	16.6	1.00	140	11.8	1.00	100	11.6	1.00
PCB 156	NR	–	–	30	2.5	0.21	20	2.3	0.20
PCB 167	NR	–	–	4	0.3	0.03	4	0.5	0.04
PCB 170	23.9	5.0	0.30	40	3.4	0.29	20	2.3	0.20
PCB 172	NR	–	–	10	0.8	0.07	10	1.2	0.10
PCB 174	NR	–	–	10	0.8	0.07	10	1.2	0.10
PCB 177	NR	–	–	10	0.8	0.07	10	1.2	0.10
PCB 178	NR	–	–	10	0.8	0.07	4	0.5	0.04
PCB 180	67.3	14.2	0.86	80	6.7	0.57	60	7.0	0.60
PCB 183	6.2	1.3	0.08	10	0.8	0.07	10	1.2	0.10
PCB 187	14.0	3.0	0.18	20	1.7	0.14	20	2.3	0.20
PCB 194	14.1	3.0	0.18	NR	–	–	NR	NR	NR
PCB 199	NR	–	–	20	1.7	0.14	10	1.2	0.10
PCB 203	NR	–	–	20	1.7	0.14	10	1.2	0.10
Total PCBs	473.4			1190			860		

NR = not reported; NC = not calculated because >40% of results were below individual limits of detection.

[a] Fitzgerald et al., 2007a. Total PCBs calculated as sum of 30 congeners. Geometric means adjusted for age, BMI, and cigarette smoking. Five persons from the study area were missing lipid determinations.

[b] Seegal et al., 2011. Total PCBs calculated as sum of 27 congeners. Unadjusted geometric means.