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Exposure of Dioxin-like Chemicals in Participants of the Anniston Community Health Survey Follow-Up

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Abstract

The 2014 follow-up of the Anniston Community Health Survey (ACHS II) consisted of 338 surviving participants from the 2005–2007 baseline study (ACHS) who had previous polychlorinated biphenyl (PCB) measurements, were not pregnant, and were not institutionalized. Questionnaires and blood samples provided the demographic, personal history, and chemical concentration data of the Anniston residents. Approximately 51% of participants were African American, 72% were female, and the mean age was 63 years old. The objectives of this study were to provide an exposure assessment of dioxin-like chemicals in the ACHS II participants and compare the measurements with the general United States (U.S.) population via the National Health and Nutrition Examination Survey (NHANES). Stratified analyses revealed significantly higher average total dioxin toxic equivalencies (TEQs) among African Americans compared to Whites (33.1 vs. 19.2 pg/g lipid), and in females compared to males (29.8 vs. 17.0 pg/g lipid). When adjusting for age, sex, and race in linear regression, we found ACHS II participants to have significantly higher total dioxin TEQ than the general 2014 U.S. population that we estimated for using half-life and NHANES 2003/04 data (most recent NHANES individual samples data), by 16.7 pg/g lipid. Principal component analyses showed that non-ortho and mono-ortho PCBs were separated from the other dioxin-like chemicals among the Anniston residents, whereas the chemicals were all clustered together for estimated NHANES 2014. The concentrations of dioxin-like chemicals, especially non-ortho and mono-ortho PCBs, in Anniston residents who resided

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Conflicts of Interest Statement

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

near the former PCB production plant were higher than those in the general U.S. population. Although data strongly supported this difference, these inferences are limited because NHANES 2013/14 data were unavailable and we used estimated NHANES 2014 levels that we imputed from NHANES 2003/04 data in conjunction with half-life values estimated from Milbrath et al., 2009.

Keywords

Anniston; Blood levels; Polychlorinated dibenzo-p-dioxins; Polychlorinated dibenzofurans; Polychlorinated biphenyls; Total dioxin toxic equivalent

1. Introduction

Anniston, Alabama was the site of a production facility where approximately half of the total United States (U.S.) production of polychlorinated biphenyls (PCBs) occurred, from the 1930s to 1970s. Earlier ATSDR investigations detailing the extent of exposure to PCBs in Anniston communities found high concentrations of PCBs present in the environment and in local residents (ATSDR, 2000). We conducted the Anniston Community Health Survey (baseline ACHS 2005–2007) to investigate further PCB exposure and potential health effects in 765 participants (Pavuk et al., 2014a, 2014b). Several studies conducted in the ACHS cohort found positive associations between PCBs and diabetes, hypertension, and lipids (Silverstone et al., 2012, Goncharov et al., 2010, Aminov et al., 2013).

The follow-up of ACHS (ACHS II) was conducted in 2014 to expand on the chemical and health studies. Polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and non-ortho PCBs were added to ACHS II in addition to mono-ortho PCBs, which were also measured during ACHS, to provide a more extensive exposure profile. The inclusion of these dioxin-like chemicals was supported by the results of a small, nested pilot study (subset, n=65) conducted within the ACHS baseline population. We found significantly higher concentrations of the non-ortho PCBs 126 and 169 when compared to NHANES 2001/02 (Pavuk et al., 2014c); this finding was the impetus for the current analyses of samples collected in 2014 for ACHS II. Another reason for including dioxin-like chemicals in ACHS II was that commercially produced PCBs have been shown to be contaminated with small amounts (10–1,000 ng/g) of PCDFs. The heating or burning of PCBs is also known to produce PCDFs, small amounts of polychlorinated terphenyls, polychlorinated quaterphenyls, and traces of PCDDs (Kannan et al., 1987; Kodavanti et al., 2001). Today, uncontrolled burning of residential waste (outdoor/backyard trash burning) is considered to be the single largest source for releasing dioxin-like chemicals, such as PCB 169, in the U.S., in contrast to a larger contribution of historical releases by various industrial operations in the past (EPA, 2013; Brown et al., 1995; NIH, 2016).

The concentration of dioxin-like chemicals in the environment and humans has declined over time in most industrial countries (Lakind et al., 2009; Schecter, 2003; Consonni et al., 2012) because of improved environmental controls and reduction in emissions. Over the past three decades, exposure to dioxin-like chemicals has been assessed in various background adult populations across the world. Meta-analyses performed on the concentration levels revealed a significant decrease over time from 1985 to 2008 among PCDDs and PCDFs

(Consonni et al., 2012). However, no significant decreases were found for non-ortho PCBs, while only a few mono-ortho PCB congeners exhibited clear, significant declines (Consonni et al., 2012). The National Health and Nutrition Examination Survey (NHANES) has also measured dioxin-like chemicals since 1999. NHANES found PCDDs, PCDFs, non-ortho PCBs, mono-ortho PCBs, and total dioxin toxic equivalencies (TEQs) to have decreased from 2001 to 2008 (Ferriby et al., 2007; Patterson et al., 2009).

There is a strong need to continue studying and monitoring dioxin-like chemical concentrations (to prevent background exposure from reaching hazardous levels), especially in localities where environmental deposits of organochlorine chemicals may lead to bioaccumulation and associated health consequences. We present here the concentrations of individual dioxin-like chemicals and the TEQs of PCDD, PCDF, non-ortho PCB, and mono-ortho PCB groups measured from the human sera of ACHS II participants. We also compared ACHS II chemical concentrations to those of NHANES. The objectives of this study were to measure the concentrations of dioxin-like chemicals in the Anniston residents for providing internal comparisons and making external comparisons with the general U.S. population.

2. Methods

2.1 Data Collection

ACHS II in 2014 accounted for the follow-up of the residential cohort of Anniston, Alabama from baseline ACHS in 2005–2007. The methods on how ACHS participants were followed over time are described by Birnbaum et al., 2016. All surviving participants with valid PCB results, who were neither pregnant nor incarcerated individuals were eligible to participate in the follow-up. From the initial 765 people, we attained the mortality status of 114 via the Social Security Index (Birnbaum et al., 2016). Another 69 participants were not re-contacted because they had moved to new locations outside the study area, which was confirmed through site visits and phone calls (Birnbaum et al., 2016). Out of 438 participants successfully reached, 359 were enrolled in ACHS II. Health questionnaires, medications, demographic information, lifestyle factors, and occupational and family medical history were collected to attain covariate data for regression analyses. We had 338 participants with available chemical concentration data and covariate information to study in ACHS II.

2.2 Laboratory Analysis

The sera were isolated by centrifugation using red top vacutainer tubes from each participant and shipped on dry ice to the Division of Laboratory Sciences at the Centers for Disease Control and Prevention (CDC). Until measurements of persistent organic pollutants (POPs) were conducted on the samples, they were stored at -70°C . The serum samples were measured first for ortho-substituted PCBs, persistent pesticides, and polybrominated diphenyl ethers (PBDEs) according to previously published methodology (Sjödén et al., 2004; Jones et al., 2012) using 2 grams of serum (median 2.0 grams; 1.0 – 2.0 grams; 10th percentile was 2.0 grams). The samples were then measured for PCDDs, PCDFs, and non-ortho PCBs based on published methodology (Turner et al., 1997) using 20 grams of serum (median 20 grams; 2.5–20.7 grams; 10th percentile was 14.0 grams). Each analytical batch

for ortho-substituted PCBs, persistent pesticides, and PBDEs was defined as twenty-four unknowns, three quality controls, and three method blanks while in the case of PCDDs, PCDFs, and non-ortho PCBs, each analytical batch was defined as eight unknowns, two quality controls, and two method blanks. Measurements of target organohalogenes were made by gas chromatography isotope dilution high-resolution mass spectrometry (GC/ID-HRMS). In HRMS, the accurate mass of a labeled or unlabeled compound is known and calculated. The limit of detection (LOD) was defined as the higher value calculated by two methods, (i) the lowest point of the calibration curve having a signal-to-noise ratio of greater than 10:1 and (ii) three times the standard deviation of method blanks analyzed in parallel with the unknown samples after subtracting the median blank value. A few criteria had to be fulfilled for quality assurance and quality control: the target analyte's measurement in the sample had to be three standard deviations or less away from the mean of the quality assurance/quality control (QA/QC) samples; at least ten consecutive measurements of the QA/QC sample could not be outside the range of the mean of the QA/QC samples if one QA/QC sample failed the previous criterion. Regarding criteria for each measurement of a set of samples, the ratio of the two ions monitored for each analyte and ^{13}C -labeled internal standard could not be less than or greater than 20% of the theoretical value; the ratio of the retention time of the analyte and corresponding ^{13}C -labeled internal standard needed to fall within 0.99 and 1.01.

A total of 28 dioxin-like chemicals with the World Health Organization's (WHO) assigned toxic equivalency factors (TEFs, Van den Berg et al., 2006) were selected for further statistical evaluation in ACHS II. These included seven PCDD congeners (2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, 1,2,3,4,7,8-HxCDD, 1,2,3,6,7,8-HxCDD, 1,2,3,7,8,9-HxCDD, 1,2,3,4,6,7,8-HpCDD, and OCDD), ten PCDFs (2,3,7,8-TCDF, 1,2,3,7,8-PeCDF, 2,3,4,7,8-PeCDF, 1,2,3,4,7,8-HxCDF, 1,2,3,6,7,8-HxCDF, 1,2,3,7,8,9-HxCDF, 2,3,4,6,7,8-HxCDF, 1,2,3,4,6,7,8-HpCDF, 1,2,3,4,7,8,9-HpCDF, and OCDF), three non-ortho PCBs (PCBs 81, 126, and 169), and eight mono-ortho-PCBs (PCBs 105, 118, 156, 157, 167, 189, 114, and 123).

2.3 Statistical Analysis

Based on the inclusion criteria and having covariate data, 338 participants remained for statistical analyses. Serum total lipids were attained from triglyceride and total cholesterol measurements using the enzymatic "summation" method (Bernert et al., 2007). We stratified by race when analyzing the various demographic variables to be consistent with the baseline ACHS study (Pavuk et al., 2014a). Regarding missing demographic variable data, there were less than four missing (<1.2%) for waist size, years residing in Anniston, and high school graduation status. 29 people did not have data for fish consumption. Multiple imputation methods as described in Pavuk et al., 2014b were used to account for the missing demographic variable data. Numerical variables were compared between African Americans and Whites race using the two-tailed t-test. Categorical variables were analyzed by race using the chi-square test. Statistical significance was determined by a p-value of less than 0.05.

We provided statistics on PCDD, PCDF, non-ortho PCB, and mono-ortho PCB congeners in lipid weight form as opposed to wet weight because the majority of studies present results using lipid weight (Fierens et al., 2005; Garabrant et al., 2009; Sjödin et al., 2014). We also did not find any notable differences in the analyses when using wet weight data (Supplemental Table 1). Each lipid weight congener was assigned a potency relative to 2,3,7,8-TCDD (TEF). Based on the WHO 2005 guidelines, we multiplied the TEF values by the associated congener concentration to attain specific TEQs (Van den Berg et al., 2006). PCDD, PCDF, non-ortho PCB, and mono-ortho PCB TEQs were the sum of the individual congener-specific classifications. Total Dioxin TEQ was the sum of all individual dioxin-like chemical TEQs. For internal ACHS II analyses, we did not impute for the non-detects in order to provide the most representative chemical concentrations in the population. Their arithmetic means and percentiles were stratified by race and sex. In addition to keeping analyses consistent with the baseline ACHS study, which revealed substantial differences in chemical concentrations by race, and also sex to a lesser extent (Pavuk et al., 2014), our use of these strata was further justified by Sjödin et al., 2014 who found significant differences in various POPs such as PBDEs, PCBs, and pesticides between various combinations of age, sex, and race groups. Fierens et al., 2005 and Garabrant et al., 2009 analyzed dioxin-like chemicals specifically and also found a link to sex, race, and age. In our analyses, we chose not to stratify by age groups specifically because the age distribution was heavily skewed towards the older population (>60 years old). To test statistical significance (p -value<0.05) when comparing the dioxin-like chemical levels by race and sex, we used linear regression with the concentrations as the outcome and age, race, and sex as the predictors.

When making external comparisons with the general U.S. population, measured by NHANES, we imputed for the non-detect chemical concentrations in the ACHS II participants. For ACHS II participants with analyte values below the detection limits, we used congener-specific LOD values and divided by the square root of 2 (LOD/sqrt(2)) in order to approximate those levels (Hornung et al., 1990). Each participant had a congener-specific LOD based on the serum volume they were able to provide. NHANES used this same method to impute for samples below the detection limits and provided data to the public in this form (Sjödin et al., 2014). Newer methods include reverse Kaplan-Meier estimator (Gillespie et al., 2010) and multiple imputation and trending approach (Bichteler et al., 2017). However, we continued the use the LOD/sqrt(2) approach of NHANES in our analyses in order to be consistent when making comparisons to NHANES data.

The last cohort of NHANES that was measured using an individual sampling approach was from 2003/04 (n=1546); we restricted the NHANES 2003/04 population to individuals at least 20 years old to better match the age range of ACHS II. In order to properly compare with the period of the ACHS II participants (2014), we used adult reference half-life values of dioxin-like chemicals provided by Milbrath et al., 2009 to estimate the 2014 chemical measurements of NHANES 2003/04 participants. They compared congener-specific elimination rates from over 30 studies and extracted the data to develop predictive equations based on the assumed linear relationship between half-life of dioxin-like chemicals and age, adjusting for body fat, smoking, and breast-feeding (Milbrath et al., 2009). With half-life values calculated from their models, we estimated NHANES 2014 levels using the exponential decay formula, $N(t)=N_0e^{-\lambda t}$, where N_0 is the NHANES 2003/04 congener-

specific concentration, e is the Euler's number (~ 2.72), λ is the congener-specific half-life from Milbrath et al., 2009, and t is 11 for the number of years from 2003 to 2014. We assumed that the elimination rate used in this formula was stable over time and did not account for background exposure.

We provided ACHS II arithmetic means to compare with the estimated NHANES 2014 survey means; we accounted for weights, primary sampling units, and strata (Curtin et al., 2012) for the estimated NHANES 2014 data in R 3.3.0. Regarding statistical variance, we provided 95% confidence intervals (CIs) and ranges for all the individual dioxin-like chemicals and TEQs. To directly compare ACHS II and the estimated NHANES 2014 levels in a statistical test, we performed linear regression, adjusting for age (years), sex (reference group=male), and race (reference group=people who are not African American or White). Each dioxin-like chemical and TEQ was the outcome in its individual models. The main predictor was "population group", with NHANES participants as the reference group. We provided the slope, 95% CI, and p-value of the "population group" variable from each model. The slope shows the pg/g lipid difference between the ACHS II and NHANES populations for each dioxin-like chemical. All direct comparisons between ACHS II and estimated NHANES 2014 levels were conducted in R 3.3.0.

Principal component analyses (PCA) were used to analyze potential clustering patterns of the dioxin-like chemicals in both ACHS II and estimated NHANES 2014 (Johnson, 2002; Jolliffe, 2002; Megson et al., 2013). For ACHS II PCA, we removed the dioxin-like chemicals confirmed to have less than 60% of measurements above the LOD. The variables were centered and scaled by subtracting the means (Kuhn and Johnson, 2013). The loadings, which correspond to each dioxin-like chemical variable, of the first two principal components were plotted, one plot for ACHS II and another for the estimated NHANES 2014. R 3.3.0 was used for PCA and for creating all the plots.

3. Results

Demographics of ACHS II, stratified by race, are exhibited in Table 1. The participants consisted of 172 (50.9%) African Americans; females accounted for 245 (72.5%). The mean age was 61.3 (range of 26 to 86) among African Americans and 64.1 (range of 27 to 87) among Whites. 59.5% of the participants were at least 60 years or older. Body mass index (BMI) average and the proportion of current smokers were similar between the two race groups; both had an average BMI above 30 (obese) and had about 20% who currently smoked. Approximately 76.2% were high school graduates. Both African Americans and Whites resided in Anniston, Alabama for approximately 52 years. There were significantly more (p -value <0.05) African Americans who resided in west Anniston and had ever eaten fish from local waterways (Snow Creek, Choccolocco Creek, or Lake Logan Martin under Alabama PCB fish consumption advisories), than Whites.

Regarding internal ACHS II comparisons, we did not impute for the non-detect dioxin-like chemical concentrations. Tables 2a–b exhibit the summary TEQs stratified by race and sex. African Americans and females had higher TEQs than Whites and males. The African American/White and female/male arithmetic mean ratios were higher for the mono-ortho

and non-ortho PCB TEQs than they were for PCDD/F TEQs (1.9–2.9 vs. 1.2–1.5). All TEQ groups were significantly higher (p -value <0.05) among African Americans and females when compared to Whites and males, which were tested using linear regression adjusted for age, race, and sex. Tables 3a–b show arithmetic means and selected percentile distributions of the individual dioxin-like chemicals, stratified by race and sex. Of the 28 lipid adjusted dioxins, arithmetic means were calculated for 21 in which over 60% of the samples were above the LOD. The seven congeners in which $<60\%$ of the samples exceeded the LOD were 2,3,7,8 TCDF, 1,2,3,7,8 PeCDF, 1,2,3,7,8,9 HxCDF, 1,2,3,4,7,8,9 HpCDF, OCDF, PCB 81, and PCB 123. When comparing the congener levels by race and sex, we used linear regression adjusting for age, sex, and race to determine statistical significance (p -value <0.05). In general, we found PCBs to have significantly higher concentrations (p -value <0.05) in African Americans and females than in Whites and males. All PCDDs were significantly higher (p -value <0.05) in females than in males. PCDFs however, showed little difference by race and sex. Among PCDDs, OCDD had the highest mean concentration (358.9 pg/g lipid in African Americans and 304.6 in Whites; 377.6 in females and 212.7 in males). Among PCDFs, the congener with the highest mean concentration was 2, 3,4,7,8 PeCDF (7.1 pg/g lipid in African Americans and 5.6 in Whites; 6.8 in females and 5.1 in males). Of the non-ortho PCBs measured, PCB 126 had the highest average concentration in participants (114.5 pg/g lipid in African Americans and 35.4 in Whites; 92.5 in females and 31.2 in males). The mono-ortho PCB with the highest average concentration was PCB 118 (80510 pg/g lipid in African Americans and 24550 in Whites; 63290 in females and 25990 in males).

Table 4 shows comparisons of ACHS II arithmetic means and estimated NHANES 2014 survey means of dioxin-like chemicals and TEQ groups. ACHS II had considerably higher concentrations than estimated NHANES 2014. Total dioxin TEQ was 26.3 pg/g lipid for ACHS II and 6.2 pg/g lipid for estimated NHANES 2014. PCDD and PCDF TEQs in ACHS II were approximately twice as high (PCDD TEQ (pg/g lipid): 11.5 in ACHS II vs. 5.0 in estimated NHANES 2014; PCDF TEQ (pg/g lipid): 2.9 in ACHS II vs. 0.93 in estimated NHANES 2014). Moreover, non-ortho PCB TEQ and mono-ortho PCB TEQ were 45.8 times (8.7 pg/g lipid in ACHS II vs. 0.19 pg/g lipid in estimated NHANES 2014) and 14.8 times (3.1 pg/g lipid in ACHS II vs. 0.21 pg/g lipid in estimated NHANES 2014) higher in ACHS II, respectively. PCDD, PCDF, non-ortho PCB, and mono-ortho PCB congener concentrations for ACHS II ranged from being 1.9 to 4.3, 2.8 to 6.5, 6.4 to 346.4 (PCB 126 highest), and 5.9 to 102.6 (PCB 105 highest) times higher than those of estimated NHANES 2014, respectively.

Table 5 provides a direct statistical test to compare the two cohort groups while adjusting for age, sex, and race. ACHS II consistently had significantly higher levels (p -value <0.0001) compared to estimated NHANES 2014. After controlling for age, sex, and race, we found PCDD, PCDF, non-ortho PCB, mono-ortho PCB, and total dioxin TEQs to be 4.7, 1.8, 7.4, 2.5, and 16.7 pg/g lipid higher in ACHS II than in estimated NHANES 2014.

Figure 1a shows PCA loading plot of the individual congeners in ACHS II. The first principal component accounted for about 54.1% variance and the second one accounted for 19.0%. PCDDs and PCDFs were close together, with the latter showing greater variance.

PCBs were all clustered near each other, with the exception of PCB 169, indicative of congener 169 possibly stemming from a different source than the other PCBs. There was a clear separation between PCBs (non-ortho and mono-ortho) and the other dioxin-like chemicals, which is indicative of the two classes of compounds coming from different sources. Within the PCDD/F cluster, 2, 3, 4,6,7,8 HxCDF and 1,2,3,4,6,7,8 HpCDF were the only congeners that were outside the grouping. Figure 1b shows the PCA plot for estimated NHANES 2014. The first principal component accounted for 46.9% variance while the second component accounted for 14.2%. Distinct from ACHS II data, no clear separation of clusters was visible in estimated NHANES 2014; PCDDs and PCBs were more clustered together while PCDFs showed the greatest variance.

4. Discussion

4.1 Summary of Findings

This study provides an extensive exposure profile of the Anniston residents, building on the baseline ACHS study from 2005–2007. In addition to ortho-substituted PCBs, which were previously analyzed in ACHS, we added dioxin-like chemicals in the follow-up study (Pavuk et al., 2014c; Birnbaum et al., 2016). In ACHS II, we found that concentrations of dioxin-like chemicals were significantly higher in African Americans than in Whites, adjusting for age and sex. African Americans had about 3 times higher non-ortho PCB and mono-ortho PCB TEQs than Whites; PCDD and PCDF TEQs were only higher among African Americans by 1.2 and 1.3 times, respectively. We also found significantly higher levels of dioxin-like chemicals in females compared to males, when adjusted for age and race. Females had higher non-ortho PCB and mono-ortho PCB TEQs than males by 1.9 and 2.5 times, respectively, while PCDD and PCDF TEQs were higher by 1.5 and 1.3 times, respectively. PCA showed non-ortho and mono-ortho PCBs to be in a separate cluster from PCDD/Fs, indicating that they could potentially come from a different source. When comparing to the estimated 2014 levels of NHANES, ACHS II showed significantly higher concentrations of dioxin-like chemicals. After adjusting for age, race, and sex, we found PCDD, PCDF, non-ortho PCB, mono-ortho PCB, and total dioxin TEQs in ACHS II to be 4.7, 1.8, 7.4, 2.5, and 16.7 pg/g lipid higher than those of estimated NHANES 2014.

4.2. Internal Comparisons

Regarding internal ACHS II comparisons, similar to ACHS findings, ACHS II revealed higher concentrations of dioxin-like chemicals, especially for non-ortho and mono-ortho PCBs, in African Americans after adjusting for age and sex. This could be due to the significantly higher proportion of African Americans than Whites who resided in west Anniston, where the former PCB plant operated. We also found a significantly higher proportion of African Americans who have ever eaten fish from Snow Creek, Choccolocco Creek, or Lake Logan Martin, which was consistent with ACHS (Pavuk et al., 2014a, and 2014b). Potential pollution of PCBs in the environment could have led to bioaccumulation in the fish over time (Durfee, 1976). Past consumption of locally produced foods could also have potentially contributed to the racial difference in PCB concentrations (Pavuk et al., 2014b). The significantly higher levels of PCDDs and PCDFs found in African Americans could be directly associated with the PCB trends, because PCDDs and PCDFs can form

from the burning of PCBs (Kannan et al., 1987; Kodavanti et al., 2001). Garabrant et al., 2009 found that living in the Midland and Saginaw counties near a Dow Chemical Company discharge site, along with hunting and fishing in contaminated areas, were significantly associated with higher levels of dioxin-like chemicals, consistent with our findings regarding close proximity and conducting activities near the contamination sites. Contrary to ACHS findings, which showed no consistent pattern in mono-ortho PCB concentrations when stratified by sex (Pavuk et al., 2014a), ACHS II showed significantly higher chemical concentrations in females than in males, even when adjusting for age and race. Previous research showed that among people who have never smoked, women reported higher levels of PCDDs, PCDFs, and non-ortho PCBs than men (Fierens et al., 2005). Patterson et al., 2008 found higher TEQs for all dioxin-like chemicals when comparing females and males using NHANES 2001/02. Overall, race appears to be a stronger indicator for dioxin-like chemical concentrations than sex, particularly with higher levels being present in African Americans (Pavuk et al., 2014a; Patterson et al., 2008).

ACHS dioxin-like chemical concentrations of the participants from the pilot study compared with ACHS II suggested that they were similar for the most part, with a slight decrease in the follow-up (Supplemental Table 2). There were several congeners that showed a slight increase from baseline to follow-up. They consisted of 2,3,4,7,8 PeCDF from 7.9 to 8.6 pg/g lipid, PCB 169 from 42.3 to 55.2 pg/g lipid, PCB 156 from 31800 to 34200 pg/g lipid, PCB 167 from 13270 to 13800 pg/g lipid, and PCB 189 from 2960 to 3170 pg/g lipid. We were unable to definitively explain what could have caused these increases for some congeners but decreases for others. The small sample size of Anniston participants with both baseline and follow-up dioxin-like chemical data (n=35), however, could explain some of these occurrences. We could not exclude ongoing exposures in Anniston; this will be examined in further detail in a follow-up paper on changes in PCB concentrations (Pavuk et al., 2015). Dioxin TEQs from the pilot study showed a pattern of slight (<10%) decrease from 2007 to 2014; PCDD TEQ decreased from 16.5 to 15.9 pg/g lipid, PCDF TEQ remained the same around 3.7–3.8 pg/g lipid, non-ortho PCB TEQ decreased from 15.8 to 13.4 pg/g lipid, mono-ortho PCB TEQ decreased from 5.4 to 5.1 pg/g lipid, and total dioxin TEQ decreased from 41.4 to 38.1 pg/g lipid, indicating that the few congeners that showed increases over time could have occurred by chance. The general temporal trend shown in this study was consistent with previous literature documenting a decrease in dioxin-like chemical exposure around the world over time (Consonni et al., 2012; Fang et al., 2013; Lakind et al., 2009).

4.3. External Comparisons

Analyses showed that dioxin-like chemical concentrations were significantly higher in ACHS II than in the general U.S. population measured by estimated NHANES 2014, which were imputed from NHANES 2003/04 individual data and adult reference half-lives calculated by Milbrath et al., 2009. This was supported from our comparison of means, 95% CIs, range, and from linear regression analyses, which adjusted for age, sex, and race. The results of higher dioxin-like chemicals were consistent with previous findings from the ACHS, where we found significantly higher levels of the sum of mono-ortho and other ortho-substituted PCBs when comparing the Anniston cohort (2005–2007) with NHANES 2003/04 (Pavuk et al., 2014a, 2014c; Sjödin et al., 2014). Although concentrations of dioxin-

like chemicals in Anniston residents appear higher than those of the general U.S. population, we cannot definitively provide a quantitative measure at this point in time. NHANES 2013/14 data was not available for analyses. When it becomes available, it would likely be pooled data, making direct comparisons with individual samples complicated at the least (Bichteler et al., 2017; Heffernan et al., 2014; Caudill, 2011). Our measures were estimates based on half-life curves, which could very likely underestimate the actual concentrations because the general U.S. population should already be on the lower end of the half-life exponential decay curve since they should be from background exposure (Lakind et al., 2009). Temporal trends found in Anniston residents (7–9 years) suggested that any decrease in concentrations of dioxin-like chemicals should be relatively small. The Milbrath et al., 2009 half-life formula assumed that the NHANES 2003/04 concentrations would start the elimination on the beginning, steep part of the curve. What made the differences between ACHS II and estimated NHANES 2014 levels substantially large were the short half-lives of various dioxin-like chemicals used to impute for estimated NHANES 2014 values, such as 2,3,7,8 TCDF (2.1 years), 2,3,4,6,7,8 HxCDF (2.8 years), 1,2,3,4,6,7,8 HpCDF (3.1 years), OCDF (1.4 years), PCB 77 (0.1 years), PCB 81 (0.7 years), PCB 126 (1.6 years), PCB 105 (2.4 years), and PCB 118 (3.8 years) (Milbrath et al., 2009). Supplemental table 3 shows the dioxin-like chemical concentrations of NHANES from 1999 to 2010. The decreases in chemical concentrations were more modest on the population level and did not clearly follow the half-life values of Milbrath et al., 2009. However, each NHANES year used different participants, making any conclusions about the half-life curves difficult to support. Another concern was that NHANES 2005–2010 used pooled samples (Bichteler et al., 2017). Ongoing or current exposures in Anniston cannot be completely excluded but would likely be limited to a few individuals (Franzblau et al., 2009). Although the inference of ACHS II having higher levels than estimated NHANES 2014 is limited due to the latter being calculated from half-life estimates, there is still a strong likelihood that ACHS II levels were higher because NHANES measurements showed background levels (Lakind et al., 2009) and previous literature supported Anniston residents having higher PCB levels in earlier years (Pavuk et al., 2014a).

PCA revealed that PCDD, PCDF, non-ortho PCB, and mono-ortho PCB congeners within estimated NHANES 2014 were all clustered together indicating that they potentially come from a similar source, most likely from consumption of food contaminated with background concentrations of dioxin-like chemicals (Health Canada, 2006a; Health Canada, 2006b; Startin, 2003; Sjödin et al., 2014). On the other hand, the ACHS II plot revealed a distinct separation of PCDD and PCDFs in one cluster and mono-ortho PCBs and non-ortho PCBs in the other, indicating that the PCBs potentially originated from a different source. However, among the PCBs, non-ortho PCB 169 was distinctly farther away from the PCB cluster. Most Aroclor® mixtures contained relatively large amounts of mono-ortho PCBs in percent weight (Frame et al., 1996; ATSDR, 2000), so the contamination of environmental and local food sources historically represent a likely pathway which resulted in elevated mono-ortho PCB concentrations in Anniston residents. Small amounts of PCB 126 and to a lesser extent PCB 169 have only been detected in Aroclor® products by applying more sensitive analytical methods (Johnson et al., 2008; Kodavanti et al., 2001; Rushneck et al., 2004). Some increases in non-ortho PCB 169 could also be partly due to the burning of

residential waste in Anniston, which is still a common practice, in neighborhoods close to the plant (Brown et al., 1995; EPA, 2013, 2016). However, no major increases in PCDFs were observed in ACHS II, which can also be formed during burning waste (EPA, 2013). The cause for the clustering patterns for the ACHS II PCA plot could stem from the Anniston residents being exposed to elevated PCBs in the food and environment while living in close proximity to the former PCB production plant, which may also explain the considerable difference in non-ortho PCB and mono-ortho PCB TEQ levels comparing ACHS II and NHANES data, much larger than the difference in PCDD and PCDF levels.

4.4. Strengths and Limitations

This study had a balanced racial distribution, approximately equal between African Americans and Whites. It also contained in-depth, congener-specific analyses and comparisons between the PCB-exposed population of Anniston and the general U.S. population. Race and sex stratified comparisons of ACHS II dioxin-like chemical concentrations were adjusted for either race or sex, and age. When comparing ACHS II participants' demographics from phase I with those ACHS participants who did not participate in the follow-up, we found the sex distribution and averages of age, total lipids, and mono-ortho PCB concentrations to be similar. ACHS II had very low LODs compared to NHANES 2003/04 (Supplemental Table 4), enabling us to quantify dioxin-like chemical concentrations for most of the participants without imputing for non-detects; over 60% of our samples for 21 out of 28 dioxin-like chemicals were above the LOD. This study also provided a direct, statistical comparison with estimated NHANES 2014 values, using linear regression adjusted for age, sex, and race.

Limitations included some small sample bias in sex-stratified analyses from the internal ACHS II comparisons; males made up only 27.5% of the sample. From comparisons between ACHS II participants' demographics at baseline and demographics of ACHS participants who did not participate in the follow-up, there was a higher proportion of African Americans and a lower proportion of smokers among ACHS II participants than the ACHS participants who were not part of the follow-up study. Over half of the ACHS II population was obese and over 60 years old; with this issue, comparisons between different cohorts can be complicated. Regarding BMI, some ACHS II individuals who had dropped in BMI from baseline to follow-up showed an increase in dioxin-like chemical concentrations due to the decrease in lipids releasing the stored chemicals; the overall trend however, still remained a decrease over time. The initial study design intentionally oversampled areas closer to the plant with higher potential for PCB exposure and was not intended to represent the city population. Half of the PCDFs had over 60% of measurements below LOD. One of the non-ortho PCBs, congener 77, could not be measured in this study, and we had only 3% of measurements of PCB 81 above the LOD. This made summary TEQ comparisons for non-ortho PCB TEQs and total dioxin TEQs more difficult with studies that included PCBs 77 or 81. However, these rapidly eliminated congeners with low concentrations contributed little to the summary non-ortho PCB TEQ. Most of the non-ortho PCB TEQ was comprised of PCBs 126 and 169, which were measured in over 79% and 88% of ACHS II samples, respectively.

5. Conclusion

This is the first report from a large population sample on measurements of dioxin-like chemicals from Anniston, Alabama. African Americans had higher levels of exposure than Whites, after adjusting for age and sex. The reason for this was likely because a higher proportion of African Americans lived in west Anniston, where the former PCB plant operated, and a higher proportion of them had eaten contaminated local foods including fish. Females had higher concentrations of dioxin-like chemicals when compared to males after adjusting for race and sex. However, the sex difference was not as pronounced as the race comparisons.

The concentrations of dioxin-like chemicals, especially non-ortho and mono-ortho PCBs, in Anniston residents who lived in close proximity to the former PCB production plant were higher than those in the general U.S. population. However, this conclusion is limited due to the fact that the NHANES 2013/14 data were unavailable and we estimated NHANES 2014 levels from NHANES 2003/04 data in conjunction with half-life values estimated from Milbrath et al., 2009.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

PCB	polychlorinated biphenyls
PCDD	polychlorinated dibenzo-p-dioxins
PCDF	polychlorinated dibenzofurans
LOD	limit of detection
PCA	principal component analysis
ACHS II	Anniston Community Health Survey follow-up

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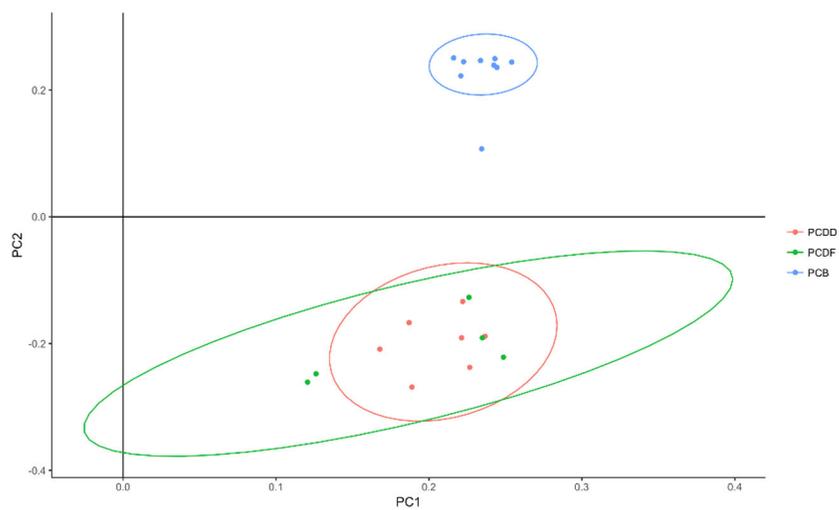


Figure 1a.

PCA loading plot for ACHS II showing groupings of PCDD, PCDF, non-ortho PCB, and mono-ortho PCB congeners based on type. PC1 accounts for 54.1% of variance and PC2 accounts for 19.0% of variance.

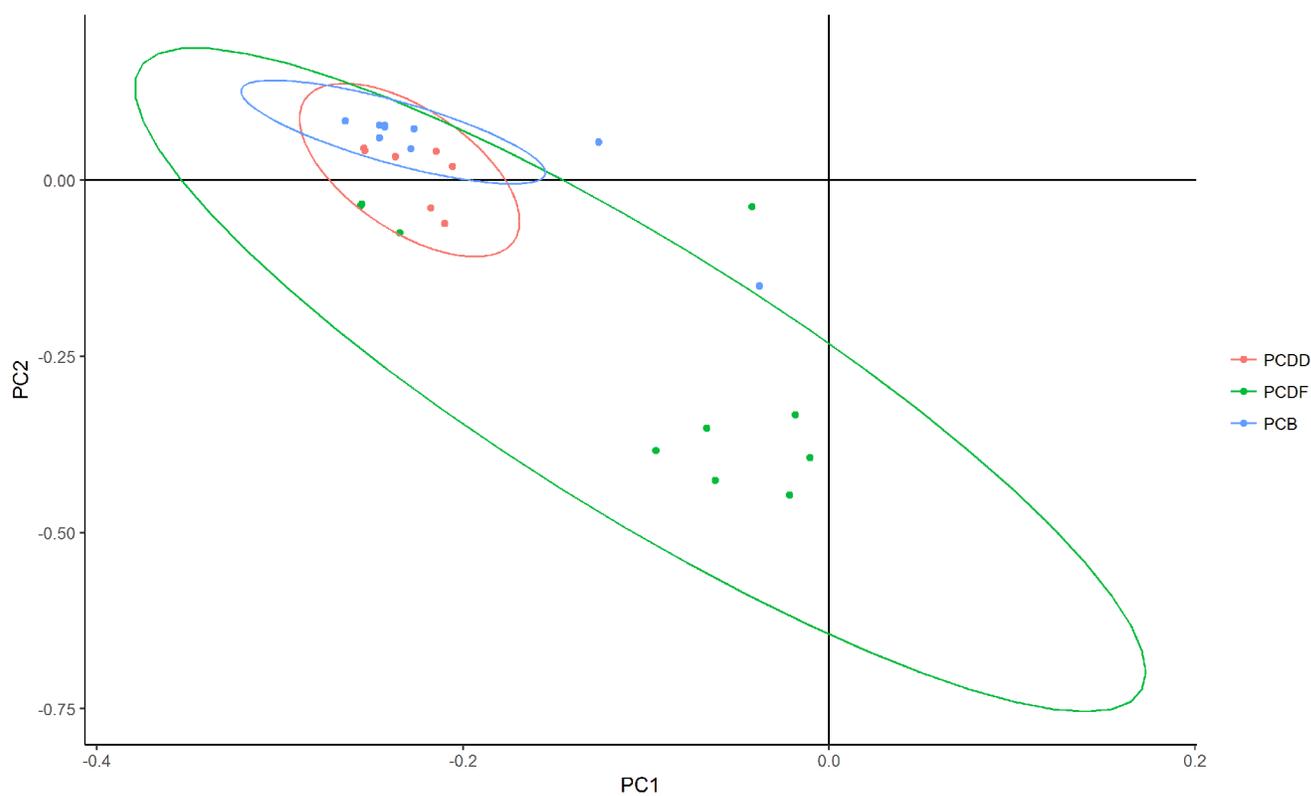


Figure 1b. PCA loading plot for estimated NHANES 2014 showing groupings of PCDD, PCDF, non-ortho PCB, and mono-ortho PCB congeners based on type. PC1 accounts for 46.9% of variance and PC2 accounts for 14.2% of variance.

Table 1.

Demographic characteristics (mean ± SE or n (percent)) of the participants of ACHS II.

Characteristic	African Americans (n=172)	Whites (n=166)	Total (n=338)
	Mean ± Standard Error		
Age in years	61.3 ± 0.91	64.1 ± 1.1	62.7 ± 0.71
BMI – kg/m ²	32.4 ± 0.59	30.8 ± 0.66	31.6 ± 0.44
Total lipid (mg/dL)	612.2 ± 12.3	634.0 ± 11.5	622.9 ± 8.4
Waist size (inches)	42.1 ± 0.47	41.6 ± 0.49	41.9 ± 0.34
Years residing in Anniston	51.8 ± 1.2	51.9 ± 1.4	51.8 ± 0.92
	Count (Percentage non-missing)		
Female	132 (76.7%)	113 (68.1%)	245 (72.5%)
Age groups (years)			
18–39	8 (4.7%)	11 (6.6%)	19 (5.6%)
40–59	68 (39.5%)	50 (30.1%)	118 (34.9%)
60	96 (55.8%)	105 (63.3%)	201 (59.5%)
BMI classification			
Normal < 25	30 (17.4%)	38 (22.9%)	68 (20.1%)
Overweight 25–29	45 (26.2%)	51 (30.7%)	96 (28.4%)
Obese 30	97 (56.4%)	77 (46.4%)	174 (51.5%)
Smoking status	37 (21.6%)	34 (20.5%)	71 (21.1%)
Reside in west Anniston	162 (94.2%) ^a	132 (79.5%)	294 (87.0%)
High school graduate	135 (79.0%)	121 (73.3%)	256 (76.2%)
Ever eaten fish from Snow Creek, Choccolocco Creek, or Lake Logan Martin	124 (78.0%) ^a	90 (60.0%)	214 (69.3%)
Ever exposed at a job to PCBs	37 (21.5%)	46 (27.7%)	83 (24.6%)

^a p < 0.05 for African Americans compared to Whites using Chi-square test.

Table 2a.

Arithmetic means (AMs) and selected percentiles for the summary TEQs (pg/g lipid) of the ACHS II participants stratified by race.

TEQ	Race	n	AM	AM Race Ratio	Min.	10 th %	50 th %	90 th %	95 th %	Max.
PCDD ^a	AA	172	12.5	1.2	1.8	4.6	9.4	21.6	31.1	76.5
	W	166	10.5		1.4	4.2	9.4	16.8	22.6	39.0
PCDF ^a	AA	172	3.3	1.3	0.57	1.3	2.5	5.6	7.4	26.4
	W	166	2.6		0.24	1.3	2.4	4.1	5.2	8.5
Mono-ortho PCB ^a	AA	172	4.5	2.8	0.15	0.60	2.5	11.8	13.5	29.4
	W	166	1.6		0.076	0.25	0.88	3.3	4.6	20.2
Non-ortho PCB ^a	AA	172	12.8	2.9	0.14	0.93	5.8	32.4	48.4	124.8
	W	166	4.4		0.11	0.42	2.3	10.3	13.2	80.2
Total Dioxin ^a	AA	172	33.1	1.7	2.9	8.4	21.7	74.3	99.5	177.4
	W	166	19.2		1.9	6.6	14.7	32.9	43.2	131.5

^aStatistically significant difference ($p < 0.05$) in TEQ by race in a linear regression model adjusting for age and sex.

Table 2b.

Arithmetic means (AMs) and selected percentiles for the summary TEQs (pg/g lipid) of the ACHS II participants stratified by sex.

TEQ	Race	n	AM	AM Sex Ratio	Min.	10 th %	50 th %	90 th %	95 th %	Max.
PCDD ^a	F	245	12.6	1.5	1.4	4.9	10.4	22	29.8	76.5
	M	93	8.6		2.7	3.6	7.4	14.4	17.7	42.4
PCDF ^a	F	245	3.1	1.3	0.24	1.3	2.6	5.4	6.9	26.4
	M	93	2.4		0.57	1.2	2	3.9	4.5	20.1
Mono-ortho PCB ^a	F	245	3.6	1.9	0.076	0.48	2	10	12.6	29.4
	M	93	1.9		0.097	0.29	0.84	4.4	8.9	19.8
Non-ortho PCB ^a	F	245	10.5	2.5	0.11	0.73	4.5	29.5	39.9	124.8
	M	93	4.2		0.11	0.54	1.7	6.1	20.9	58.2
Total Dioxin ^a	F	245	29.8	1.8	1.9	8.3	20.5	69.6	83.2	177.4
	M	93	17.0		4.2	6.2	12.5	26	53.7	90

^aStatistically significant difference ($p < 0.05$) in TEQ by sex in a linear regression model adjusting for age and race.

Table 3a.

Dioxin-like chemicals (pg/g lipid) in ACHS II participants stratified by race.

Dioxin Congener	Race	n	% > LOD	AM	AM Race Ratio	Min	10 th %	50 th %	90 th %	95 th %	Max
2,3,7,8 TCDD ^a	AA	171	82.5	1.7	1.2	<LOD	<LOD	1.3	3.3	4.8	15.6
	W	166	86.1	1.4		<LOD	<LOD	1.2	2.7	3.3	7.1
1,2,3,7,8 PeCDD ^a	AA	172	96.5	5.8	1.2	<LOD	2.1	4.8	10.2	13.7	25.3
	W	166	98.2	5.0		<LOD	1.9	4.5	8.9	11.2	17.5
1,2,3,4,7,8 HxCDD ^a	AA	172	97.7	4.4	1.1	<LOD	1.3	3.2	9.2	11.6	19.1
	W	166	99.4	3.9		<LOD	1.4	3.2	7.2	8.9	22.3
1,2,3,6,7,8 HxCDD ^a	AA	172	99.4	31.6	1.1	<LOD	13.3	25.2	56.1	73.6	148.3
	W	166	100	28.7		3.1	11.7	26.8	48.5	56.7	96.9
1,2,3,7,8,9 HxCDD	AA	172	75.6	4.2	1.1	<LOD	<LOD	3.7	8.8	13.5	20.0
	W	166	79.5	4.0		<LOD	<LOD	3.7	7.3	9.6	21.1
1,2,3,4,6,7,8 HpCDD	AA	172	76.2	28.9	1.1	<LOD	<LOD	22.1	61.9	91.9	176.9
	W	166	77.7	26.2		<LOD	<LOD	19.5	52.3	71.2	299.7
OCDD ^a	AA	172	87.2	358.9	1.2	<LOD	<LOD	298.6	693.8	945.9	1557
	W	166	86.8	304.6		<LOD	<LOD	263.3	602.7	704.9	1962
2,3,7,8 TCDF	AA	172	26.2	--		<LOD	<LOD	<LOD	0.9	1.3	19.3
	W	166	18.1	--		<LOD	<LOD	<LOD	0.7	1.1	2.7
1,2,3,7,8 PeCDF	AA	172	7.6	--		<LOD	<LOD	<LOD	<LOD	0.5	19.0
	W	166	10.8	--		<LOD	<LOD	<LOD	0.5	1.1	3.1
2,3,4,7,8 PeCDF ^a	AA	171	98.3	7.1	1.3	<LOD	2.2	5.2	12.6	16.8	78.6
	W	166	99.4	5.6		<LOD	2.4	5.1	9.6	11.9	22.7
1,2,3,4,7,8 HxCDF ^a	AA	172	98.8	4.1	1.2	<LOD	1.9	3.5	7.4	9.5	15.8
	W	166	99.4	3.5		<LOD	1.8	3.2	5.7	6.5	10.1
1,2,3,6,7,8 HxCDF	AA	172	98.3	3.9	1.0	<LOD	1.7	3.5	6.1	8.3	16.2
	W	166	99.4	3.8		<LOD	1.8	3.3	6.2	8.0	11.1
1,2,3,7,8,9 HxCDF	AA	172	1.2	--		<LOD	<LOD	<LOD	<LOD	<LOD	17.4
	W	166	0.6	--		<LOD	<LOD	<LOD	<LOD	<LOD	0.7
2,3,4,6,7,8 HxCDF	AA	172	64.5	0.76	1.0	<LOD	<LOD	0.7	1.6	1.9	17.4
	W	166	72.3	0.79		<LOD	<LOD	0.7	1.8	2.4	3.0
1,2,3,4,6,7,8 HpCDF ^a	AA	172	89.5	5.4	1.4	<LOD	<LOD	4.6	8.7	10.8	57.8
	W	166	82.5	3.8		<LOD	<LOD	3.7	6.9	8.5	20.0
1,2,3,4,7,8,9 HpCDF	AA	172	5.8	--		<LOD	<LOD	<LOD	<LOD	0.3	15.3
	W	166	3.6	--		<LOD	<LOD	<LOD	<LOD	<LOD	0.6
OCDF	AA	172	1.2	--		<LOD	<LOD	<LOD	<LOD	<LOD	18.5
	W	166	0.6	--		<LOD	<LOD	<LOD	<LOD	<LOD	2.2

Dioxin Congener	Race	n	% > LOD	AM	AM Race Ratio	Min	10 th %	50 th %	90 th %	95 th %	Max
PCB 81	AA	172	5.2	--		<LOD	<LOD	<LOD	<LOD	4.7	71.4
	W	166	1.8	--		<LOD	<LOD	<LOD	<LOD	<LOD	9.9
PCB 126 ^a	AA	172	86.6	114.5	3.2	<LOD	<LOD	44.9	293.2	449	1196
	W	166	71.1	35.4		<LOD	<LOD	15.7	81.7	121.7	760.1
PCB 169 ^a	AA	172	89.5	43.4	1.5	<LOD	<LOD	31.1	94.9	122.6	363.6
	W	166	86.8	28.7		<LOD	<LOD	23.7	53.9	74.4	175.5
PCB 105 ^a	AA	172	97.7	14700	3.7	<LOD	1000	7000	35700	56800	101700
	W	166	89.2	3920		<LOD	<LOD	1364.5	10500	14300	60430
PCB 118 ^a	AA	172	100	80510	3.3	1694	6200	40200	202900	277500	532600
	W	166	100	24550		716.9	2400	10475	53800	83700	382200
PCB 156 ^a	AA	172	100	28120	2.0	700	4900	19285	64100	88400	170600
	W	166	98.2	13930		<LOD	1817	9885	29810	40500	94600
PCB 157 ^a	AA	172	97.1	7570	2.2	<LOD	1100	5003.5	17300	23100	44800
	W	166	91.0	3510		<LOD	483.4	2500	8000	10600	30200
PCB 167 ^a	AA	172	98.8	12700	2.7	<LOD	1300	7816	32600	39800	73310
	W	164	92.7	4660		<LOD	633.1	2433.5	10810	14500	70900
PCB 189 ^a	AA	168	92.3	2900	2.2	<LOD	500	2000.5	5800	9700	21650
	W	164	84.2	1300		<LOD	<LOD	1000	2700	3800	8900
PCB 114 ^a	AA	162	92.6	4430	2.2	<LOD	700	2622	12700	14300	31850
	W	151	88.1	1990		<LOD	<LOD	1300	3700	6107	18600
PCB 123	AA	151	32.5	--		<LOD	<LOD	<LOD	2300	3400	9086
	W	138	15.2	--		<LOD	<LOD	<LOD	793.3	1264	11400

% > LOD—percent over the limit of detection

AA—African American

W—White

AM—arithmetic mean

Arithmetic means were only calculated for congeners with 60% >LOD

^aStatistically significant (p < 0.05) by race when adjusted for age and sex in linear regression.

Table 3b.

Dioxin-like chemicals (pg/g lipid) in ACHS II participants stratified by sex.

Dioxin Congener	Sex	n	% > LOD	AM	AM Sex Ratio	Min	10 th %	50 th %	90 th %	95 th %	Max
2,3,7,8 TCDD ^a	F	244	88.1	1.7	1.8	<LOD	<LOD	1.4	3.3	4.3	12.7
	M	93	74.2	0.94		<LOD	<LOD	0.72	1.6	2.3	15.6
1,2,3,7,8 PeCDD ^a	F	245	97.1	5.9	1.4	<LOD	2.1	5.0	10.3	13.1	25.3
	M	93	97.9	4.3		<LOD	1.9	3.9	7.2	9.0	17.0
1,2,3,4,7,8 HxCDD ^a	F	245	98.8	4.6	1.5	<LOD	1.5	3.7	8.9	11.6	22.3
	M	93	97.9	3.1		<LOD	1.1	2.5	5.3	8.1	14.7
1,2,3,6,7,8 HxCDD ^a	F	245	99.6	32.7	1.4	<LOD	12.9	28.5	56.1	74.5	148.3
	M	93	100	23.4		4.9	9.4	21.5	42.8	49.1	69.3
1,2,3,7,8,9 HxCDD ^a	F	245	84.5	4.8	2.0	<LOD	<LOD	4.3	8.9	12.9	21.1
	M	93	59.1	2.4		<LOD	<LOD	2.4	5.6	6.3	16.0
1,2,3,4,6,7,8 HpCDD ^a	F	245	78.8	31.4	1.8	<LOD	<LOD	24.4	63.7	91.9	299.7
	M	93	72.0	17.6		<LOD	<LOD	16.5	35.4	46.2	94.9
OCDD ^a	F	245	91.0	377.6	1.8	<LOD	112.9	317.1	702.7	920.6	1962.0
	M	93	76.3	212.7		<LOD	<LOD	200.4	424.1	626.5	765.4
2,3,7,8 TCDF	F	245	24.1	--		<LOD	<LOD	<LOD	0.88	1.3	5.1
	M	93	17.2	--		<LOD	<LOD	<LOD	0.60	1.0	19.3
1,2,3,7,8 PeCDF	F	245	8.2	--		<LOD	<LOD	<LOD	<LOD	0.69	3.1
	M	93	11.8	--		<LOD	<LOD	<LOD	0.40	0.79	19.0
2,3,4,7,8 PeCDF ^a	F	245	98.8	6.8	1.3	<LOD	2.6	5.6	12.3	14.3	78.6
	M	92	98.9	5.1		<LOD	2.2	4.1	9.1	10.7	34.0
1,2,3,4,7,8 HxCDF ^a	F	245	99.2	4.1	1.3	<LOD	1.9	3.6	7.2	8.8	14.1
	M	93	98.9	3.2		<LOD	1.8	2.8	4.8	5.7	15.8
1,2,3,6,7,8 HxCDF ^a	F	245	98.8	4.0	1.2	<LOD	1.8	3.6	6.5	8.5	13.0
	M	93	98.9	3.3		<LOD	1.8	3.0	5.0	5.8	16.2
1,2,3,7,8,9 HxCDF	F	245	0.82	--		<LOD	<LOD	<LOD	<LOD	<LOD	0.74
	M	93	1.1	--		<LOD	<LOD	<LOD	<LOD	<LOD	17.4
2,3,4,6,7,8 HxCDF	F	245	69.8	0.76	0.92	<LOD	<LOD	0.70	1.8	2.0	3.0
	M	93	64.5	0.83		<LOD	<LOD	0.69	1.4	1.6	17.4
1,2,3,4,6,7,8 HpCDF	F	245	86.9	4.6	1.0	<LOD	<LOD	4.1	8.2	9.9	23.9
	M	93	83.9	4.6		<LOD	<LOD	3.9	6.9	9.2	57.8
1,2,3,4,7,8,9 HpCDF	F	245	3.7	--		<LOD	<LOD	<LOD	<LOD	<LOD	2.0
	M	93	7.5	--		<LOD	<LOD	<LOD	<LOD	0.43	15.3
OCDF	F	245	0.41	--		<LOD	<LOD	<LOD	<LOD	<LOD	17.9

Dioxin Congener	Sex	n	% > LOD	AM	AM Sex Ratio	Min	10 th %	50 th %	90 th %	95 th %	Max
	M	93	2.2	--		<LOD	<LOD	<LOD	<LOD	<LOD	18.5
PCB 81	F	245	4.1	--		<LOD	<LOD	<LOD	<LOD	<LOD	40.5
	M	93	2.2	--		<LOD	<LOD	<LOD	<LOD	<LOD	71.4
PCB 126 ^{*a}	F	245	84.5	92.5	3.0	<LOD	<LOD	36.4	271.6	382.0	1196.0
	M	93	64.5	31.2		<LOD	<LOD	12.1	51.2	141.1	559.7
PCB 169	F	245	88.2	37.7	1.2	<LOD	<LOD	28.7	76.9	102.7	363.0
	M	93	88.2	32.3		<LOD	<LOD	23.9	69.1	94.9	235.9
PCB 105 ^a	F	245	95.5	11300	2.6	<LOD	700	5000	33800	43500	101700
	M	93	88.2	4370		<LOD	<LOD	1219	6900	25560	81200
PCB 118 ^a	F	245	100	63290	2.4	716.9	4800	29500	178700	235500	532600
	M	93	100	25990		900	2117	8242	55640	144900	409900
PCB 156	F	245	98.8	22600	1.3	<LOD	3300	14490	52700	67300	170600
	M	93	100	17300		400	3300	9600	42700	61100	129100
PCB 157	F	245	94.7	6040	1.4	<LOD	700	2500	10720	16000	32200
	M	93	92.5	4350		<LOD	900	3900	13100	18900	44800
PCB 167 ^a	F	244	96.7	9920	1.7	<LOD	1100	5250	27610	36200	73310
	M	92	93.5	5680		<LOD	668.5	2370.5	16710	26300	56500
PCB 189	F	241	87.6	2100	1.0	<LOD	<LOD	1400	4606	5800	21650
	M	91	90.1	2100		<LOD	399.8	1300	4185	6200	16800
PCB 114 ^a	F	229	93.5	3680	1.8	<LOD	600	2300	9700	13400	31850
	M	84	82.1	2080		<LOD	<LOD	1080.5	6498	9000	19400
PCB 123	F	212	29.7	--		<LOD	<LOD	<LOD	2300	3300	11400
	M	77	9.1	--		<LOD	<LOD	<LOD	<LOD	844.9	2312

% > LOD—percent over the limit of detection

F—Female

M—Male

AM—arithmetic mean

Arithmetic means were only calculated for congeners with 60% >LOD

^aStatistically significant (p < 0.05) by sex when adjusted for age and race in linear regression.

Table 4.

Dioxin-like chemical concentration and summary TEQ (mean (95% confidence interval) and range) comparisons between ACHS II and estimated NHANES 2014 (imputed from NHANES 2003/04 using half-life estimates of Milbrath et al., 2009).

Dioxin-like Chemicals (pg/g lipid)	ACHS II (2014) 26–87 years old (n=338)		Estimated NHANES (2014) 31–96 years old (n=1546)	
	Arithmetic Mean (95% CI)	Range	Survey Mean (95% CI)	Range
2,3,7,8 TCDD	1.7 (1.5, 1.9)	0.23–15.6	0.69 (0.61, 0.77)	0.10–9.9
1,2,3,7,8 PeCDD	5.6 (5.2, 6.0)	0.72–25.3	2.3 (2.1, 2.5)	0.20–18.4
1,2,3,4,7,8 HxCDD	4.2 (3.9, 4.5)	0.58–22.3	1.9 (1.8, 2.1)	0.46–15.2
1,2,3,6,7,8 HxCDD	30.2 (28.1, 32.3)	3.1–148.3	15.9 (14.5, 17.3)	0.61–115.1
1,2,3,7,8,9 HxCDD	4.2 (3.8, 4.5)	1.9–21.1	0.98 (0.91, 1.1)	0.16–7.6
1,2,3,4,6,7,8 HpCDD	28.8 (25.5, 32.1)	5.7–299.7	7.8 (6.9, 8.6)	0.27–96.2
OCDD	337.1 (306.7, 367.5)	71.0–1962	94.7 (82.2, 107.2)	10.0–1051
2,3,7,8 TCDF	--	--	0.043 (0.041, 0.045)	0.011–0.33
1,2,3,7,8 PeCDF	--	--	0.19 (0.19, 0.20)	0.057–2.2
2,3,4,7,8 PeCDF	6.5 (5.8, 7.1)	0.77–78.6	1.9 (1.8, 2.0)	0.24–22.0
1,2,3,4,7,8 HxCDF	3.9 (3.6, 4.2)	0.66–15.8	1.3 (1.2, 1.4)	0.18–9.8
1,2,3,6,7,8 HxCDF	3.9 (3.6, 4.1)	0.87–16.2	1.4 (1.3, 1.5)	0.17–12.5
1,2,3,7,8,9 HxCDF	--	--	0.68 (0.64, 0.72)	0.14–9.3
2,3,4,6,7,8 HxCDF	0.84 (0.71, 0.96)	0.35–17.4	0.13 (0.13, 0.14)	0.033–0.76
1,2,3,4,6,7,8 HpCDF	5.0 (4.4, 5.6)	1.8–57.8	0.79 (0.69, 0.90)	0.068–31.4
1,2,3,4,7,8,9 HpCDF	--	--	0.42 (0.38, 0.46)	0.095–10.2
OCDF	--	--	0.020 (0.013, 0.027)	0.0026–1.9
PCB 81	--	--	0.00011 (0.000094, 0.00012)	0.00002–0.013
PCB 126	76.2 (61.1, 91.3)	6.3–1196	0.22 (0.19, 0.25)	0.016–6.1
PCB 169	36.7 (32.6, 40.7)	5.1–363.6	5.7 (5.3, 6.1)	0.67–69.0
PCB 105	9410 (7760, 11100)	400–101700	91.7 (81.0, 102.5)	1.7–3204
PCB 114	3280 (2830, 3730)	300–31850	--	--
PCB 118	53030 (44270, 61780)	716.9–532600	1480 (1330, 1630)	94.1–47600
PCB 123	--	--	--	--
PCB 156	21150 (18570, 23740)	400–170600	3610 (3310, 3910)	24.8–227900
PCB 157	5590 (4890, 6290)	368.3–44800	906 (828.4, 983.6)	26.2–54990
PCB 167	8770 (7500, 10040)	255.6–73310	726.3 (670.0, 782.6)	15.9–26960
PCB 189	2130 (1860, 2400)	387.9–21650	260.2 (193.0, 327.4)	14.1–8485
Summary TEQs				
PCDD	11.5 (10.6, 12.4)	1.4–76.5	5.0 (4.5, 5.4)	0.55–38.8
PCDF	2.9 (2.7, 3.2)	0.24–26.4	0.93 (0.89, 0.98)	0.011–9.1
Non-ortho PCB	8.7 (7.1, 10.3)	0.11–124.8	0.19 (0.18, 0.21)	0–2.1
Mono-ortho PCB	3.1 (2.7, 3.6)	0.076–29.4	0.21 (0.20, 0.22)	0.0081–9.8

Dioxin-like Chemicals (pg/g lipid)	ACHS II (2014) 26–87 years old (n=338)		Estimated NHANES (2014) 31–96 years old (n=1546)	
	Arithmetic Mean (95% CI)	Range	Survey Mean (95% CI)	Range
Total Dioxin	26.3 (23.6, 29.0)	1.9–177.4	6.2 (5.7, 6.6)	0.016–48.0

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Table 5.

β s, 95% Confidence Intervals (CIs), and p-values of linear regression comparing ACHS II and estimated NHANES 2014 (imputed from NHANES 2003/04 using half-life estimates from Milbrath et al., 2009).

Dioxin-like Chemicals	β	95% CI	p-value
2,3,7,8 TCDD	0.65	0.50, 0.80	<0.0001
1,2,3,7,8 PeCDD	2.4	2.1, 2.7	<0.0001
1,2,3,4,7,8 HxCDD	1.7	1.5, 2.0	<0.0001
1,2,3,6,7,8 HxCDD	9.4	7.8, 11.0	<0.0001
1,2,3,7,8,9 HxCDD	2.9	2.7, 3.1	<0.0001
1,2,3,4,6,7,8 HpCDD	18.7	16.8, 20.7	<0.0001
OCDD	204.7	186.0, 223.5	<0.0001
2,3,7,8 TCDF	--	--	--
1,2,3,7,8 PeCDF	--	--	--
2,3,4,7,8 PeCDF	4.0	3.6, 4.4	<0.0001
1,2,3,4,7,8 HxCDF	2.2	2.1, 2.4	<0.0001
1,2,3,6,7,8 HxCDF	2.3	2.1, 2.5	<0.0001
1,2,3,7,8,9 HxCDF	--	--	--
2,3,4,6,7,8 HxCDF	0.71	0.64, 0.78	<0.0001
1,2,3,4,6,7,8 HpCDF	4.0	3.7, 4.4	<0.0001
1,2,3,4,7,8,9 HpCDF	--	--	--
OCDF	--	--	--
PCB 81	--	--	--
PCB 126	66.2	58.0, 74.4	<0.0001
PCB 169	27.6	25.4, 29.9	<0.0001
PCB 105	7990	7100, 8880	<0.0001
PCB 118	44100	39400, 48800	<0.0001
PCB 156	14600	13000, 16170	<0.0001
PCB 157	3870	3450, 4290	<0.0001
PCB 167	6700	6020, 7390	<0.0001
PCB 189	1660	1500, 1820	<0.0001
Summary TEQs			
PCDD	4.7	4.1, 5.3	<0.0001
PCDF	1.8	1.6, 1.9	<0.0001
Non-ortho PCB	7.4	6.6, 8.3	<0.0001
Mono-ortho PCB	2.5	2.2, 2.7	<0.0001
Total Dioxin	16.7	15.2, 18.1	<0.0001

The outcomes are lipid adjusted dioxin-like chemicals (pg/g lipid). The main predictor is cohort group, with estimated NHANES 2014 as the reference group. The β s show the number of pg/g lipid higher the concentration is in ACHS II than in NHANES 2014. Each model is adjusted for age, sex, and race.