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ORIGINAL ARTICLE

Analysis of NHANES measured blood PCBs in the general US population and application of SHEDS model to identify key exposure factors

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Studies have shown that the US population continues to be exposed to polychlorinated biphenyls (PCBs), despite their ban more than three decades ago, but the reasons are not fully understood. The objectives of this paper are to characterize patterns of PCBs in blood by age, gender, and ethnicity, and identify major exposure factors. EPA's Stochastic Human Exposure and Dose Simulation (SHEDS)-dietary exposure model was applied, combining fish tissue PCB levels from a NYC Asian Market survey with National Health and Nutrition Examination Survey (NHANES) dietary consumption data, and then linked with blood biomarkers for the same NHANES study subjects. Results reveal that the mean concentration of total PCBs in blood was higher with increasing age; however, for the same age, gender, and ethnicity, the blood PCB concentrations measured in the later NHANES survey were significantly lower than those in the earlier one. The decrease within an age group between the two survey periods lessened with increasing age. Blood PCBs among different ethnicities ranked differently between the older and the younger age groups within each survey. Non-Hispanic Blacks had significantly higher blood PCBs for the >30 year age group. For the 12 to ≤ 30 year age group, the "Asian, Pacific Islander, Native American or multiracial" group had the highest values, with patterns fairly consistent with fish consumption and modeled PCB exposure patterns. We conclude that for younger people, patterns correspond to reduced environmental contamination over time, and are strongly associated with fish consumption and dietary exposures. Higher PCB concentrations in blood of the older population may partially reflect past exposures to higher environmental PCB concentrations, particularly before the ban.

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INTRODUCTION

Polychlorinated biphenyls (PCBs) are synthetic organochlorine compounds representing an important class of anthropogenic xenobiotics. PCBs released into the environment tend to bioaccumulate and persist in ecosystems for a long time.[1] As lipophilic compounds, PCBs often bioconcentrate in the food chain and accumulate in the adipose tissues of animals.[2–5] The exposure to PCBs by the human populations is widespread and has been associated with a variety of health risks.[6] The US Environmental Protection Agency (EPA) and the International Agency for Research on Cancer classified PCBs as probable carcinogens.[7,8] Other health risks include adverse effects on neurodevelopment,[9] neuropsychological function,[10,11] and contribution to metabolic syndrome[12] and diabetes[13,14] have been reported.

In the US, PCBs were manufactured between 1929 and 1977 by Monsanto and distributed under a trade name of Aroclor.[15] PCBs released from their industrial and residential applications moved into environments via different routes[16] and resulted in environmental accumulation.[1,17] As early as 1967, PCBs were detected in the US human populations.[18,19] While a ban of open use of PCBs in 1973 and a shutdown of PCBs production in 1979 in the US has had a very significant role in curtailing the progression of PCBs contamination,[20–22] the persistence of PCBs[23,24] still presents health problems, and PCBs have been recognized as an important class of persistent organic pollutants.[25–28] Studies have shown that much of the US human population is still exposed to PCBs.[22,29–31]

To better characterize the impact of PCBs in the general US human population, US Centers for Disease Control and Prevention began to include measurements of PCBs in serum samples of human subjects participating the National Health and Nutrition Examination Survey (NHANES) since 1999.[32] Analyses of NHANES data have yielded some interesting observations.[9,32–36] For example, PCBs detected in the blood of the general US population exhibit an age-related trend, with older individuals displaying higher concentrations.[35] Higher total PCBs levels were associated with older age and non-Hispanic Black race.[33] Regression models were used for estimating total PCBs and confirmed the pattern of increasing blood PCBs with increasing age as well as some ethnicity difference in the blood levels of PCBs.[36]

However, it has not been well addressed what exposure route(s) and vehicle(s) have had major roles in shaping the observed levels and patterns of the PCBs in the general US population at the

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post-ban era of PCBs. Human exposure to PCBs can happen from inhalation of polluted air, dermal exposure to dusts, and ingestion of contaminated food. Because the pollution of PCBs in the air in the post-ban era of PCBs is limited to certain specific situations and general population is more likely exposed to PCBs via dietary consumption of foods contaminated by PCBs,[37] we focused our study in understanding the patterns of PCBs in participants of NHANES by analyzing the food consumption patterns of the US population and assessing their likely contribution to the patterns of PCBs in the blood. We also used the EPA's Stochastic Human Exposure and Dose Simulation (SHEDS) model[38–40] to predict dietary PCBs exposures from fish consumption. Our study helps to identify major exposure routes and vehicles in the post-ban era of PCBs that contribute significantly to the observed biomonitoring pattern of PCBs in the blood of general US population.

METHODS

Collection and Analysis of Blood PCBs Data

We obtained blood PCBs concentration data from the NHANES (http://www.cdc.gov/nchs/nhanes.htm) and performed our analyses by taking into the account of the varying sampling weight for the respective NHANES data sets. Four-year subsample weights were used in the analyses; we tried both 2-year and 4-year subsample weights and results were very similar. We used all congeners (30 congeners in Supplementary Table 3) measured in each survey and combined their concentrations into the total PCBs. Our initial analyses explored the total concentrations of blood serum PCBs in the general US population between the 2001–2002 and 2003–2004 survey times and between two major age groups considered, that is, >30 years old and 12 to ≤ 30 years old at the time of the survey. The selection of 30 years as a time division was based on the assumption that the ban on open PCBs use in the US ~ 30 years before the first survey might exert some impact on the release of PCBs into the environment, and thus exposure to PCBs by the US population.

Because the big difference in the limit of detection (LOD) between the 2001–2002 and the 2003–2004 NHANES measurements of blood PCBs (Supplementary Table 3) may artificially impact the study of the changes in blood levels of PCBs over the two survey periods, we adopted an approach of reducing the impact of LOD difference on the change of levels of PCBs between the two surveys. We hypothesized that, if we use the same sets of LOD for both surveys, the calculated concentrations of PCBs would be influenced by other factors other than a difference in LODs. Thus, we used the maximum LOD for each congener of 30 congeners in the 2001–2002 survey as the reference values for selecting all the data of PCBs whose 2003–2004 levels are below the reference values and calculated their mean values as "new adjusted LODs" for each congener. These new adjusted LODs are used for filling in all the 2001–2002 and 2003–2004 values reported as below their respective maximum LODs from 2001–2002. In this way we "artificially increased" LODs of the 2003–2004 survey, which will be consistent with LODs in 2001–2002 survey. New adjusted LODs were used to fill values below detection limits (see Supplementary Table 3). For example, in the NHANES blood PCBs data for 2001–2002, congener 66 has 17 measurements valued less than 2003–2004 NHANES maximum LOD (0.0018 ng/g) for this congener. There are 1864 measurements that have values between the 2003–2004 maximum LOD (0.0018 ng/g) and the 2001–2002 maximum LOD (0.059 ng/g) for the same congener. Thus, the mean of those ($17+1864$) values were calculated to be 0.01 ng/g. This mean value serves as the adjusted LOD of PCBs congener 66. This kind of congener-specific adjusted LODs is used for filling PCBs congener blood concentrations in the 2001–2002 and 2003–2004 NHANES data, which was lower than 2001–2002 maximum LOD (see Supplementary Table 3).

The impact of using unadjusted LODs and using adjusted LODs is shown in Supplementary Table 3a. For total PCBs, the mean and standard deviation are 1.185 and 1.542 with the unadjusted LODs and 1.409 and 1.518 with the adjusted LODs, respectively. This represents a 20% increase for the mean value and a 2% decrease in the standard deviation.

To examine the effect of ethnicity, we analyzed the blood total PCBs within the two major age groups by combining the two NHANES survey data sets. Regression and Tukey–Kramer analyses were conducted to explore the upward age-related trend, ethnicity patterns in the blood levels for the two age groups, and the degree of ethnicity-related differences within each age group.

Collection and Analysis of Food Consumption Data

Because dietary exposure from fish, meat, milk, and fat consumption is a major route for PCBs exposure by the general human population in the post PCBs-ban era,[41,42] we conducted an analysis to assess whether food consumption differences may explain the varied blood PCBs among different ethnicities and age groups. For dietary consumption inputs, we used 1999–2006 NHANES data, containing instantly recorded consumption of food and water by each survey participant. Recipe files in the US EPA's Food Consumption Intake Database (FCID) were used for breaking down consumed foods reported in NHANES into raw agricultural commodities (RACs), following methodologies detailed in previous publications.[43,44] Because the food consumption surveys in NHANES are only snapshots of food consumption in a short time window but the blood levels of PCBs should reflect the end results of some long-term exposure, we used food consumption data from all participants over a wider time window (1999–2006) than that shown for the data on blood PCBs (2001–2004) to compensate the lack of long-term observation of food consumption of the same survey participants. More importantly, with a larger set of data used in the analysis, the results are more stable and reliable.

Exposure Modeling

Because the fish consumption appeared to be a significant factor affecting the blood PCBs in the studied US population, and we were able to use available residue data, we applied the SHEDS dietary exposure model to explore dietary contributions and patterns related to the observed blood levels. SHEDS dietary is a probabilistic, population-based dietary exposure assessment model that can simulate individual exposures to chemicals in food and drinking water over different time periods (http://www.epa.gov/heasd/products/sheds_multimedia/sheds_mm.html). Model inputs were NHANES consumption data, FCID recipe files, and PCBs concentration data in fish from a New York city Asian market survey.[45] Detailed descriptions of the SHEDS model methodology for estimating dietary exposure can be obtained from previous publications.[43,44] SHEDS modeled results were compared against NHANES measurements of total PCBs in blood. This model has undergone extensive review by expert panels, and has been evaluated with comparison to both measured and modeled data for various chemical classes. All statistical analyses were performed with SAS 9.2.

RESULTS

Age and Ethnicity Patterns for Blood Levels in Different Time Periods

While the age and ethnicity composition were similar between the two NHANES survey time periods (see Table 1), the mean total blood concentrations of PCBs (30 common congeners in two surveys) in the study population decreased statistically significantly from the earlier time period to the later one for a given age group (Figure 1a; Supplementary Figure 1). Total blood PCBs of the 2003–2004 survey decreased by 18 and 19% (mean) and 10 and 20% (geometric mean, GM) for the 12 to ≤ 30 years and $30+$ year age groups, respectively, in comparison with the 2001–2002 survey (Figure 1a).

Total blood PCBs for the 2003–2004 survey decreased by 8%, 26%, 25% and 20% (mean) and 5%, 16%, 23% and 22% (GM) for the 12–19, 20–30, 31–49 and $50+$ age groups, respectively, in comparison with the 2001–2002 survey levels. Figure 1b shows that blood PCBs concentrations increased as age increased in the both surveys. For these four age groups, the absolute reductions of total blood PCBs between the two surveys are 0.03, 0.16, 0.29 and 0.46 ng/g.

Because many PCBs congeners showed low detection rates in the surveys, we analyzed the blood PCBs of two specific PCB congeners (138 and 153), whose detection rates were the highest and, which were comparable between the two surveys, to see if our results on total blood PCBs reflect some genuine trends of decreasing blood PCBs over time among different age groups. The detection rates for PCB 138 are 79% and 100% and for PCB 153 are 84% and 100%, for the NHANES surveys 2001–2002 and 2003–2004, respectively. Analysis on PCB 153 (Figure 1c and d) and PCB

138 (Supplementary Figure 3) yielded similar results of decreasing blood PCBs over time for the same age groups as seen for the total blood PCBs.

Ethnicity Patterns for Blood Concentrations

The patterns of ethnicity rankings of total blood PCBs were different between the two major age groups. For the older age group (>30 years at the survey time), non-Hispanic Black had the highest blood total PCBs, followed by non-Hispanic White, other Hispanic, Asian/Pacific Islander/Native American/other Multiracial (A/P/N/M), and then Mexican American (Figure 2). For the younger age group (12 to ≤30 years at the survey time), the ranking of total blood PCBs from high to low concentration was: A/P/N/M, non-Hispanic White, other Hispanic, non-Hispanic Black, and Mexican American.

Figure 3 shows more linear regression analyses than Figure 2 to emphasize that the concentrations of total blood PCBs exhibited an upward age-related trend, the differences in ethnicity-related patterns for the two age groups, and the degree of ethnicity-related differences within each age group. A/P/N/M had significantly higher blood PCBs for the younger age group (12 to ≤30 years at the time of the surveys) (Figure 3a), and had the highest rate of change in total PCBs with age. Non-Hispanic Blacks had significantly higher blood PCBs for the >30 year age group (see Figure 3b), and the highest rate of change by age within that group. The similar pattern is shown with log-transformed measurement data (Supplementary Figure 4). General linear model (GLM) analyses revealed that age group, survey year (2001–2002 *vs* 2003–2004), ethnicity, and interaction of age group with ethnicity are statistically significant factors for total blood PCBs, whereas gender is not (Supplementary Tables 1 and 2).

Table 1. Age and ethnicity composition by survey year.

	12–30 years old		*30+ years old*	
	2001–2002	*2003–2004*	*2001–2002*	*2003–2004*
Ethnicity composition (%)				
Mexican American	31.2	27.2	19.4	19.1
Non-Hispanic White	34.1	32.9	54.6	56.2
Non-Hispanic Black	26.5	32.0	18.6	17.0
Other Hispanic	4.3	3.2	4.0	3.2
A/P/N/M	3.9	4.7	3.5	4.5
Averaged age (years)	18.2	18.5	53.8	55.2

Food and Fish Consumption Patterns, and Dietary Exposures by Ethnicity and Age Group

Although the consumption of meat and of skin and fat was similar among different ethnicities, the consumption of fish and milk varied significantly (Figure 4). The greatest variation among ethnicities occurred with the fish consumption. A/P/N/M showed the highest fish consumption in both age groups, and the older people (>30 years) consumed larger amounts of fish than the younger people (12 to ≤30 years).

The distribution of mean (Figure 5a) and 95th percentile (Figure 5b) of SHEDS modeled PCBs exposures from fish consumption, generated by 100 simulations for variability analyses,

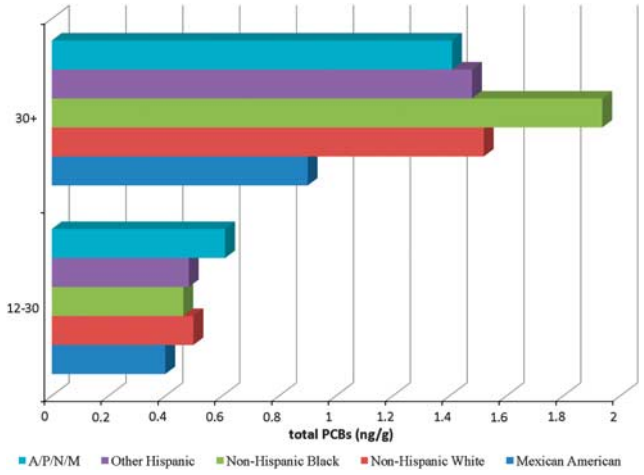

Figure 2. Total blood PCBs by age group (years) and ethnic from 2001–2004 NIHANES data.

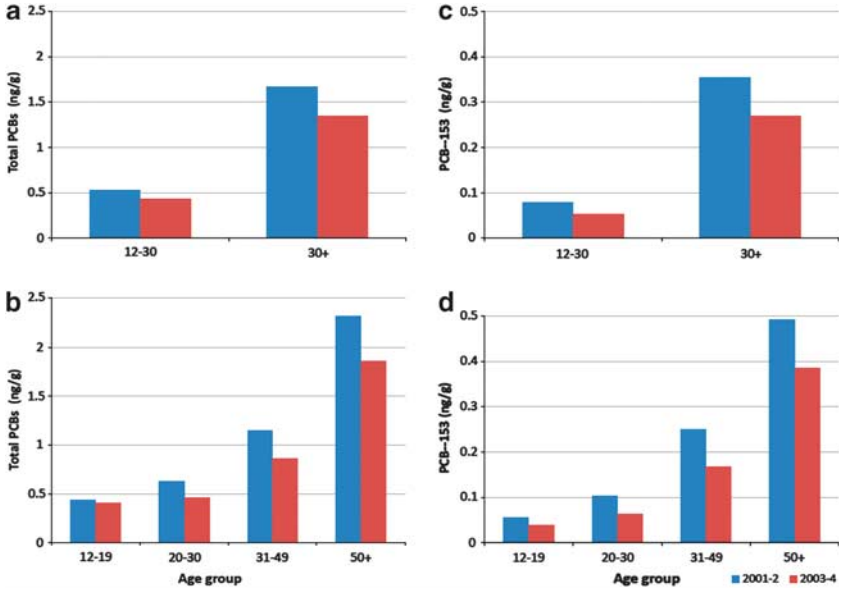

Figure 1. Averaged total blood PCBs in NHANES by age group (years) and survey year.

displayed a predicted ethnicity ranking (from high to low) as: A/P/N/M, non-Hispanic Black, other Hispanic, non-Hispanic White, and Mexican American for the older age group; and A/P/N/M, other Hispanic, non-Hispanic Black, Mexican American, and non-Hispanic White for the younger age group. For the younger age group (12 to $\leq$30), the ethnicity-specific PCBs blood concentration patterns in NHANES (Figure 2) are fairly consistent with patterns for NHANES fish consumption (Figure 4) and SHEDS modeled exposure estimates (Figure 5), but not for the older age group.

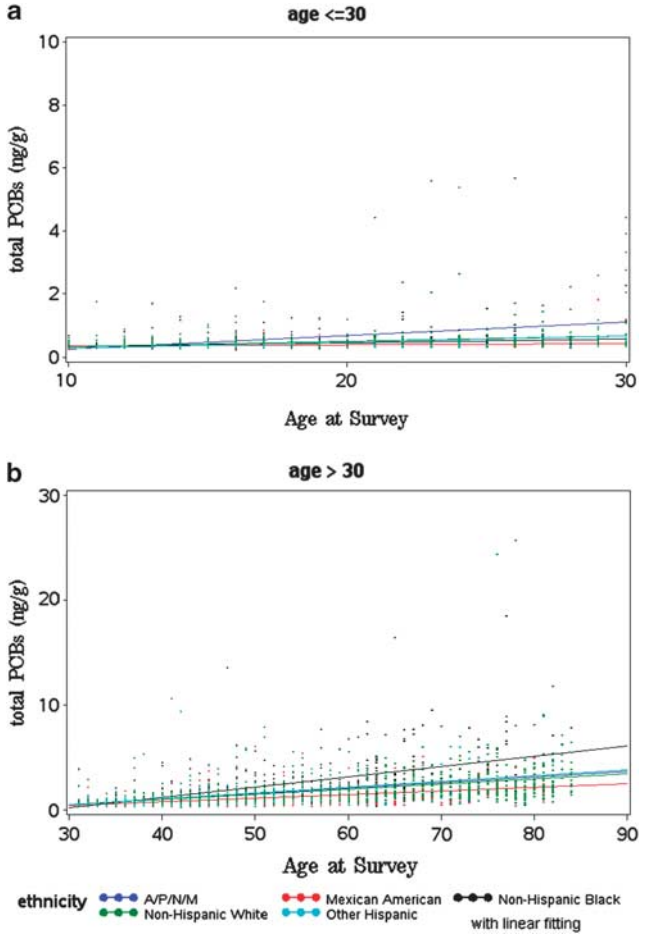

Figure 3. Total blood PCBs by age group (**a**, $\leq$30; **b**, $>$30) and ethnicity from 2001–2004 NHANES data.

Using the SHEDS model, we further analyzed the exposures of total PCBs within each ethnicity (Table 2). The highest level of fish consumption-contributed PCBs exposure was 0.015 μg/kg/day for A/P/N/M females and 0.017 μg//kg/day for A/P/N/M males in the $>$50 year age group. The averaged PCBs exposures of males are higher than those of females, and as age increased, the exposures increased: A/P/N/M averaged exposures were 0.006, 0.009, 0.11, 0.015 μg/kg/day (female) and 0.011, 0.012, 0.016, 0.017 μg/kg/day (male) for the 12–20, 21–30, 31–49, and 50+ year age groups, respectively.

A side-by-side comparison between age group segregated and ethnicity-specific total PCBs measured in blood and intake estimated by SHEDS modeling is shown (Supplementary Table 4). This comparison shows that with the increase in age total PCB intake estimated with SHEDS modeling and total PCBs measured in blood both increase. An interesting point is that for the 50+ years age group, the non-Hispanic Black group has the highest total PCBs in blood (3.08 ng/g) even though its estimated intake is at the same level (1.05 μg/day) as the Tribal-Asian-Pacific group, which has much lower blood total PCBs (1.78 ng/g).

Correlation analyses between fish consumption and concentrations of total blood PCBs at participant level show a Pearson correlation coefficient of 0.07 (*P*-value $<$0.01) between fish consumption data from the dietary survey and blood PCBs concentration data. However, there is no positive correlation between consumption of either meat or milk with the blood PCBs.

DISCUSSION

Our analyses confirmed previous analyses of NHANES biomonitoring data for PCBs, and revealed important new findings on how age, ethnicity, and diet influence PCBs exposures and blood concentrations. Within each of the two NHANES survey periods considered, a trend of increased average total blood PCBs with increased age group was observed; however, for the same age, gender, and ethnicity, the blood PCBs measured in the later NHANES survey were significantly lower than those measured in the earlier one. Variations of PCB concentrations in blood among different ethnicities ranked differently between the older and

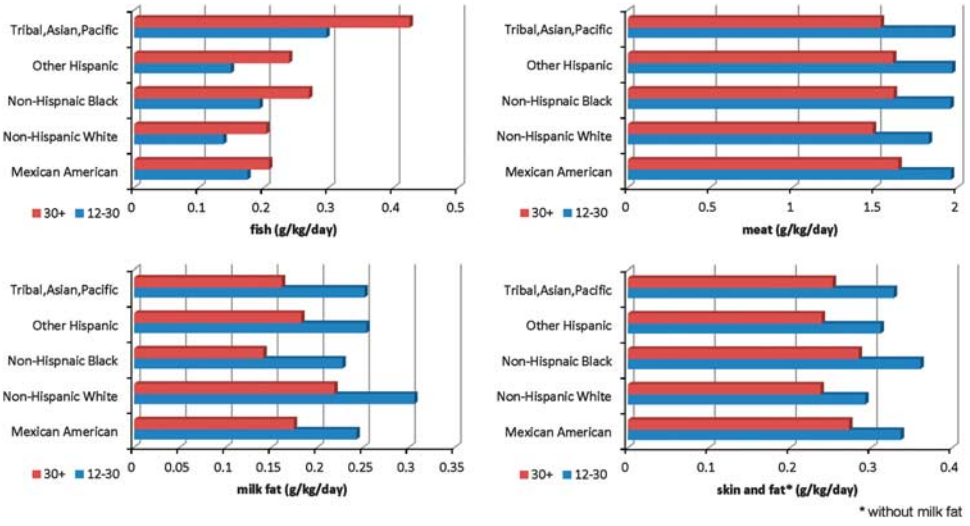

Figure 4. Daily averaged fish, meat, fat, consumption by ethnicity and age group (years) from 1999–2006 NHANES data.

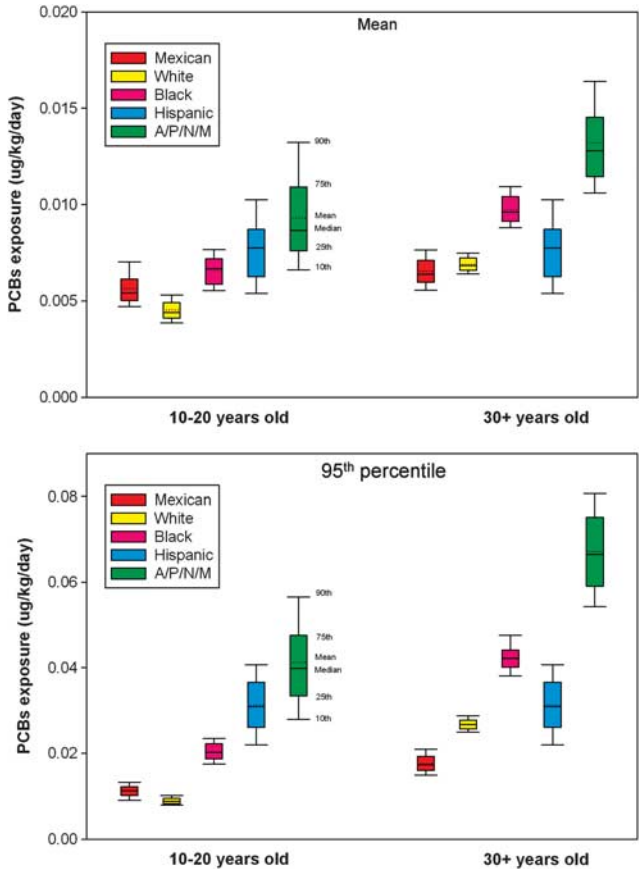

Figure 5. Distribution of modeled dietary PCBs exposure from fish consumption, by ethnicity and age group: 100 SHEDS runs using 1999–2006 NHANES consumption data.

younger age groups within each survey. Analyses of available food and fish consumption data, and SHEDS dietary exposure modeling helped to explain these differences and patterns for the younger age group (12 to $\leq$30 years).

Age and Ethnicity Patterns for Blood Concentrations

Our analyses (Figure 1 and Supplementary Figure 3) confirmed previous findings of apparent increase in PCBs in blood with increasing age, and longitudinal studies that have shown decreasing blood PCBs over time for given individuals.[42] The analyses on PCB congeners 138 and 153, whose detection rates are very close between two NHANES surveys, indicate that the observed trend of decreasing total blood PCBs over time for the same age groups is not a measurement bias but a true reflection of a decline, especially in the over-30-years-old groups. This trend may be explained by one or several of the following possibilities: (a) bioaccumulation naturally leads to higher body burden with age, that is, persistence of higher concentrations of blood PCBs in the older subjects might be a reflection of continued mobilization of some internally stored PCBs;[46] (b) people are eating more fish as they get older, as shown in Figure 4; and (c) fish PCB concentrations have declined in recent years, so older individuals have higher blood levels because of higher exposures in the past or disappearance of some exposure pathways.

While the blood PCBs decreased from the earlier to the later NHANES survey period, the decrease between the survey times for 12–19 year olds is the smallest (8% for mean and 5% for GM; Figure 1b). This implies that the body burden of PCBs is a key contributor to the blood concentrations, especially for older people. The relatively small change of absolute total blood PCBs between the two surveys indicates long half-life of PCBs in human body. Mobilization of internal PCBs may be accelerated with the aging process.[47]

Our age-stratified analysis of the ethnicity variation in NHANES blood PCBs showed different rankings between the older age group considered ($>$30 years at the survey time) and the younger age group (12 to $\leq$30 years at the survey time) (Figure 2). This suggests that some exposure pathway(s) changed after the ban of PCBs. For the younger age group, A/P/N/M had the highest levels: this new finding warrants follow-up investigation to determine approaches for reducing exposures in this group.

For the older age group, non-Hispanic Black had the highest blood total PCBs; this has been seen in previous studies that have also reported ethnic/racial differences for PCBs body burden.[33] Another study on two separate data sets of blood PCBs in the NHANES also showed a consistent pattern of ethnicity ranking between two survey times when all ages were combined.[36]

Food and Fish Consumption Patterns and Dietary Exposures by Ethnicity and Age Group

Our analyses confirm that fish consumption is a key factor in PCBs exposure, and reveal new findings on ethnicity- and age-related patterns. Ethnicity-related fish consumption patterns were fairly consistent with fish dietary exposure levels for both age groups. For the younger age group, both the fish consumption and modeled exposure patterns matched reasonably well with the pattern and rankings of blood PCBs among the studied ethnicities.

Detailed analysis of PCBs exposure from specific fish species would be useful as follow up research, but challenging due to limited data. We did find that the distribution of PCBs concentrations in fish tissue for various fish species is different than for methyl mercury.[44] Also, PCBs concentrations in bottom-dweller fish are higher than predator fish, but the opposite is seen for methyl mercury.

The SHEDS modeling predictions are consistent with other studies showing that fish eaters usually have higher serum PCBs than non fish eaters in the same region.[30] Our study reveals that A/P/N/M generally had higher PCBs exposures than other ethnicities because of the dietary exposure route via fish consumption. Similar findings were reported in our earlier study,[44] which reported a SHEDS exposure modeling analysis for methyl mercury and NHANES blood concentrations. However, the higher blood total PCBs in the non-Hispanic Black group than the other ethnicities (Supplementary Table 4) may indicate a higher earlier environmental exposure to PCBs and the release of some internally stored PCBs into the blood. It is possible that a dietary, genetic, or other vulnerability factor is related to this disparity.

The fish PCBs concentrations used in our exposure model came from a 2007 Asian market survey in New York City.[45] This may introduce some bias, as the types of fish consumed by other ethnicities may be different.[41] Owing to the lack of available PCBs residue data in meat, skin, fat, and milk, we were not able to model contributions to dietary PCBs exposures from these foods.

Dynamic Model Evaluation, Considering Trends of PCBs in Fish over Time

A WHO report has disclosed the decreasing trend of PCBs exposure over time via dietary intake.[37] Studies have shown that overall, PCBs levels decreased in fish tissues over time from 1980 to 2004.[20,48] Because SHEDS inputs for fish PCBs levels and consumption data were from fixed time periods, we conducted additional dynamic model evaluation analyses, using fish PCBs data from EPA's National Lake Fish Tissue study collected at different years as exposure model inputs.

Table 2. SHEDS PCBs exposure predictions by ethnicity and age group from dietary fish consumption with 1999–2006 NHANES data (μg/kg/day).

Age group	*Ethnicity*	*Female*					*Male*				
		n	*Mean*	*std*	*p95*	*p99*	n	*Mean*	*std*	*p95*	*p99*
12–20 years	Mexican American	2287	0.004	2.360	0.004	0.082	2187	0.004	3.389	0.004	0.073
	Non-Hispanic White	1787	0.004	6.833	0.005	0.092	1791	0.003	4.519	0.003	0.070
	Non-Hispanic Black	2121	0.006	4.065	0.014	0.149	2241	0.005	2.995	0.011	0.157
	Other Hispanic	257	0.003	3.379	0.002	0.091	229	0.004	3.238	0.010	0.095
	A/P/N/M	312	0.006	6.743	0.014	0.161	256	0.011	11.259	0.019	0.370
21–30 years	Mexican American	805	0.005	3.863	0.014	0.120	608	0.009	10.808	0.015	0.237
	Non-Hispanic White	1338	0.004	7.772	0.012	0.111	867	0.006	10.306	0.018	0.206
	Non-Hispanic Black	568	0.008	9.075	0.034	0.160	444	0.009	9.552	0.031	0.211
	Other Hispanic	143	0.009	10.178	0.037	0.221	94	0.002	4.003	0.004	0.138
	A/P/N/M	121	0.009	5.649	0.070	0.140	105	0.012	10.573	0.068	0.330
31–49 years	Mexican American	989	0.008	6.835	0.019	0.263	927	0.008	7.482	0.029	0.173
	Non-Hispanic White	1984	0.004	8.392	0.015	0.113	1877	0.006	12.103	0.019	0.144
	Non-Hispanic Black	1044	0.008	9.720	0.031	0.153	919	0.009	9.654	0.040	0.199
	Other Hispanic	208	0.002	1.940	0.015	0.043	185	0.014	17.925	0.067	0.258
	A/P/N/M	206	0.011	11.297	0.076	0.221	159	0.016	27.264	0.060	0.228
50 + years	Mexican American	1097	0.005	2.500	0.015	0.157	1051	0.004	2.343	0.012	0.160
	Non-Hispanic White	3236	0.008	10.116	0.035	0.204	3401	0.008	10.548	0.035	0.184
	Non-Hispanic Black	1098	0.008	7.220	0.041	0.180	1053	0.014	9.360	0.057	0.343
	Other Hispanic	184	0.005	5.368	0.030	0.116	160	0.003	2.745	0.027	0.056
	A/P/N/M	196	0.015	12.412	0.096	0.333	158	0.017	14.068	0.092	0.436

Abbreviations: NHANES, National Health and Nutrition Examination Survey; PCBs, polychlorinated biphenyls; SHEDS, Stochastic Human Exposure and Dose Simulation.

The additional SHEDS simulations for dynamic evaluation confirmed the decline in exposures because of lower levels of PCBs in fish, and indicated that an accelerated decline started in 1996. The mean modeled dietary PCBs exposures using residue data from the National Lake Fish Tissue study was 0.0087 μg/kg/day, which is 1.3 times higher than the modeled mean exposure using the 2007 NYC Asian market survey data; the 95th and 99th percentiles were 1.2 times higher (see Supplementary Figure 2).

CONCLUSIONS

The reduction in total blood PCB concentrations in each NHANES age group over two survey periods (2001–2002 and 2003–2004) is evidence of the effectiveness of the US control measures on PCB release and production. Currently, the major route of PCBs exposure is dietary via consumption of fish and other foods with that tend to bioaccumulate PCBs present in the environment. This is particularly evident in A/P/N/M populations. Analysis of food consumption explained the blood concentrations of PCBs for the younger (12 to $\leq$30 years) age group and the A/P/N/M ethnicity group within that younger subpopulation. Dietary ingestion, particularly *via* fish consumption, remains a major route of exposure to environmental PCBs for the general human population.

Additional research is also needed to consider cumulative exposures of fish consumption (i.e., from multiple exposure routes and chemicals including PCBs, methyl mercury, and other persistent pollutants), vulnerable groups such as tribes and children, and other PCBs sources (e.g., old schools, other food types such as meat and dairy).

ABBREVIATIONS

A/P/N/M, Asian/Pacific Islander/Native American/Other Multiracial; EPA, U.S. Environmental Protection Agency; FCID, EPA's Food Consumption Intake Database; GLM, general linear model; GM, geometric mean; NHANES, National Health and Nutrition Examination Survey; ORD, Office of Research and Development; PCBs, polychlorinated biphenyls; RAC, raw agricultural commodity; SHEDS, Stochastic Human Exposure and Dose Simulation (model)

CONFLICT OF INTEREST

All authors declare no conflict of interests.

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DISCLAIMER

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Supplementary Information accompanies the paper on the Journal of Exposure Science and Environmental Epidemiology website (http://www.nature.com/jes)