Organochlorines, Lead, and Mercury in Akwesasne Mohawk Youth

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Most humans have detectable body burdens of polychlorinated biphenyls (PCBs), hexachlorobenzene (HCB), and *p,p′*-dichlorophenyldichloroethylene (*p,p′*-DDE), a metabolite of *p,p′*-dichlorodiphenyltrichloroethane (DDT). Native American communities may be at increased risk of exposure through subsistence-based diets and greater physical contact with contaminated soil and water. In this article we describe the levels of toxicants (PCBs, *p,p′*-DDE, HCB, mirex, lead, and mercury) among youth 10–17 years old (n = 271) of the Akwesasne Mohawk Nation. Ultratrace, congener-specific PCB analysis of human serum quantitated 83 PCB congeners (plus 18 as pairs/triplets), in addition to *p,p′*-DDE, HCB, and mirex, and included all major Aroclor-derived congeners typically present in human samples. Twenty congeners (in 16 chromatographic peaks) were detected in 50% or more of the individuals sampled [geometric mean (GM) of the sum of these congeners = 0.66 ppb]. Thirteen congeners (in 10 peaks) were detected in 75% or more of the samples (GM = 0.51 ppb). Of the 20 congeners detected in 50% or more of the samples, 17 had five or more chlorine substitutions. International Union for Pure and Applied Chemistry congeners 118, 101(+90), and 153 were detected in nearly all participants (GM = 0.06 ppb, 0.05 ppb, 0.09 ppb, respectively). *p,p′*-DDE and HCB were detected in 100% and 98% of the samples (GM: *p,p′*-DDE = 0.37 ppb; HCB = 0.03 ppb). Mirex was detected in approximately 46% of the samples (GM = 0.02 ppb). No cases of elevated lead level were observed. One participant had a mercury level marginally higher than the U.S. Environmental Protection Agency's current level of concern (0.50 µg/dL). Although differences in analytic methods and participant ages limit comparability, toxicant levels from the Mohawk youth are lower than those associated with severe food contamination (Yusho and Yu-cheng) but similar to other chronically exposed groups. *Key words:* **adolescents, Iroquois, Native American, persistent organic pollutants, polychlorinated biphenyls, toxicants.** *Environ Health Perspect* **111:954–961(2003). doi:10.1289/ehp.5990 available via *http://dx.doi.org/* [Online 12 February 2003]**

Persistent organic pollutants (POPs) are a group of compounds that includes polychlorinated biphenyls (PCBs), *p,p′*-dichlorodiphenyldichloroethylene (*p,p′*-DDE), hexachlorobenzene (HCB), and mirex. These compounds are lipophilic and bioaccumulate (Matthews and Dedrick 1984). They and/or their metabolites have entered the environment and the food chain and can be detected at some level in many, if not all, human populations (Stehr-Green 1989). A major route of POP intake in humans is consumption of contaminated food (Liem et al. 2000; Patandin et al. 1999). In addition, POPs cross the placenta and are transferred through lactation, resulting in exposure to the fetus and to infants. Some PCBs (i.e., the coplanar congeners) resemble 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (Safe 1994) and produce biologic effects associated with binding to the aryl hydrocarbon (Ah) receptor. In contrast, documented or suspected biologic effects of non-coplanar PCB congeners include disruption of the development and functioning of some endocrine pathways and altered growth, development, and cognitive function in nonhumans and humans (American Council on Science and Health 1997; Brouwer et al. 1999; Carpenter et al. 1998; Schell 1999; Seegal 1996; Swanson et al. 1995).

Previous studies of environmental contamination such as the Exxon Valdez disaster (Palinkas et al. 1992) as well as other work (Akwesasne Task Force on the Environment 1997; Curtis 1992; Grinde and Johansen 1995; Harris and Harper 1997; Hild 1998) have indicated that Native peoples may be differentially exposed to toxicants. They are at particular risk of exposure to environmental contamination because of traditional dietary patterns involving consumption of locally caught fish and riverine species (Sloan and Jock 1990). Also, increased exposure may result from activities involving greater contact with the outdoor environment, such as swimming, wading, hunting, trapping, small-scale farming, and gathering traditional plants for medicines, foods, and other uses (Arquette et al. 2002; Curtis 1992; Sloan and Jock 1990).

The Mohawk Nation, one of the five nations of the Haudenosaunee Confederacy, have long lived, fished, planted, and hunted in the St. Lawrence River valley. The construction of the St. Lawrence Seaway and the St. Lawrence-FDR Power Project in the 1950s has led to substantial industrial development along the St. Lawrence River. The Mohawk Territory of Akwesasne is now adjacent to several industrial complexes, including the General Motors Central Foundry Division, which is a National Priority Superfund Site (Lacetti 1993; U.S. EPA 1984). Also in the vicinity and upriver from Akwesasne are two New York State Superfund sites, the Reynolds Metal Company and Aluminum Company of America aluminum facilities (Fitzgerald et al. 1995, 1998; Lacetti 1993). PCBs and other POPs used during production by all three companies have contaminated the St. Lawrence and its river tributaries (Ecology and Environment, Inc. 1992; Fitzgerald et al. 1995, 1998; RMT 1986; Woodward-Clyde Associates 1991). Some local species of fish, birds, amphibians, and mammals have PCB, *p,p′*-DDE, HCB, and mirex levels that exceed the U.S. Food and Drug administration's tolerance limits for human consumption (Forti et al. 1995; Lacetti 1993; Skinner 1992; Sloan and Jock 1990). In the past, the Mohawk have relied heavily on locally caught fish and game as sources of protein. In this article we describe the levels of several toxicants in a sample of youth from the Akwesasne Mohawk community. Specific toxicants were chosen for analysis because of community concerns about their effects and their documented presence in the local environment.

Materials and Methods

Sample. Present-day Akwesasne is a sovereign nation whose territory lies on both sides of the St. Lawrence River and spans the boundaries of Ontario and Quebec, Canada, and New York State. In 1995, three human health studies were begun under the auspices of the University at Albany's Superfund Basic Research Program (SBRP) and the Akwesasne Task Force on the Environment. Akwesasne is not a federally censused population. Published estimates of the Akwesasne community's population size vary, but recent reports indicate a population of approximately 12,000–13,000 (Akwesasne Task Force on the Environment 1997; Fitzgerald et

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al. 1998; George-Kanentiio 1995; Goran et al. 1995). Residents of the community live within the boundaries of the Akwesasne Mohawk Nation and in neighboring communities that are part of traditional Mohawk territory, including Bombay, Fort Covington, Hogansburg, Massena, and Rooseveltown, New York, and Cornwall, Ontario.

The target population cannot be defined by a municipal boundary because of the dispersed residence pattern of community residents. The official tribal register also cannot serve this purpose because it lists all Nation members, including those living at very large distances from the source of local contamination (e.g., in New York City). To define the target population with risk of exposure to local contaminants, a panel of Akwesasne community representatives was assembled, and they defined the target population as residents of households within the boundaries of the Akwesasne Mohawk Nation and residents of those Mohawk households in neighboring communities, but no more than 10 miles from the Nation border. All Akwesasne households were enumerated using detailed maps and drive-through surveys by Mohawk data collection staff, all of whom are members of the Akwesasne Mohawk community. These households were contacted to solicit participation in the three SBRP studies. Each household was placed on a list from which a random sample of 50 households were selected, and then each selected household was visited to determine the age, sex, and relationships among household members. When the first 50 households were contacted or were unable to be contacted after repeated attempts, another 50 randomly chosen households were selected and the process was repeated.

When a household was contacted, consent information was explained and eligibility for the three different SBRP studies according to the exclusion criteria was ascertained. To be eligible for this study, the mother/youth pair had to reside in the same household and the youth could not be a twin, have a serious psychologic impairment or problem as determined by a physician or a psychologist, have a serious physical condition as diagnosed by a physician, or have been diagnosed with either fetal alcohol syndrome or fetal alcohol effects. Only one child per household, between 10 and 16.99 years old, was eligible to participate in this study. The oldest eligible child was selected first, but if that child was unwilling to participate, the next oldest was then selected. In 1998, recruitment was expanded because of the small number of age-eligible persons among the households that had been contacted from the randomized lists of households. At this point, all Akwesasne households were included, and volunteers were accepted while all other eligibility criteria were retained. Informed consent was obtained from all participating youths and their parent or guardian, and the study protocols were reviewed and approved by the Institutional Review Board at the University at Albany, State University of New York.

Of the Akwesasne households that met all eligibility requirements, 294 mother/youth pairs participated (138 male and 156 female youth). Of these, 19 dropped out of the study primarily because of an aversion to having their blood drawn, one participant was later found to be ineligible (too young), and three had to be excluded because their blood samples were broken in transit to the lab and they declined to have their blood redrawn. The final sample size was 271 participant pairs, 131 males and 140 females and their mothers.

Interview data collection. Members of the Akwesasne community collected all interview data, and each data collector was trained in measurement techniques specific to this study (anthropometry, interview techniques, and phlebotomy). All data were collected without prior knowledge of the participant's exposure status. Data collection occurred from February 1996 through January 2000.

The youth's mother completed interviews and questionnaires to obtain information about the youth's family background, including breast-feeding history and socioeconomic status, as well as a dietary questionnaire regarding the mother's consumption of locally caught fish and game. However, one participant's mother was deceased and the youth's maternal grandmother, who resided with the youth, completed the questionnaire on breast-feeding history and socioeconomic status, but was unable to complete questionnaires regarding maternal diet during pregnancy.

Laboratory analysis of toxicants. For PCB analysis, two 10-mL and one 5-mL sample of blood were collected from each youth by venipuncture into no-additive (red-top) and ethylenediamine tetracetic acid (EDTA)-additive (lavender-top) glass Vacutainer tubes, respectively. Blood specimens were collected by trained Mohawk staff at each participant's home. The sample was collected within a 5-hr window to minimize the effects of diurnal variation (particularly regarding endocrine assessment). Participants were asked not to eat any locally caught or grown food for 3 days before the collection and not to eat or drink anything after 2200 hr the preceding evening.

After collection, no-additive specimens were allowed to clot for at least 20 min at room temperature and then centrifuged. Aliquots of approximately 5 mL serum were transferred into hexane-washed polytetrafluoroethylene-capped glass vials and stored at –20°C at the Akwesasne laboratory. Aliquots of approximately 1 mL were transferred to plastic Eppendorf vials and stored at –80°C for clinical chemistry analyses. EDTA-additive specimens (for lead and mercury analysis) were stored at 4°C. The blood specimens provided a sufficient volume for at least one PCB/organochlorine (OC) pesticide analysis in addition to a battery of endocrine and clinical chemistry analyses. Universal safety precautions were observed for all human samples processed in the laboratory.

PCB and OC pesticide analyses were conducted at the University at Albany's School of Public Health (SPH) Analytical Laboratory located at the University's East Campus. Assessment of endocrine analytes and clinical chemistries was performed at the Clinical Chemistry and Hematology Laboratory, Wadsworth Center for Laboratories and Research, New York State Department of Health. Lead and mercury analyses were conducted by Le Centre de Toxicologie du Quebec in Sainte-Foy, Quebec, Canada.

A detailed description of the method used for the serum PCB/pesticide analyses is provided elsewhere (DeCaprio et al. 2000). High-resolution, ultratrace, congener-specific analysis was performed by parallel dual-column (splitless injection) gas chromatography (GC) with electron capture detection (ECD). The GC-ECD method quantitates up to 83 individual PCB congeners and 18 congeners as pairs or triplets, in addition to the OC pesticides *p,p′*-DDE, mirex, and HCB (a total of 94 analytical peaks). The analytes include all of the major Aroclor-derived congeners typically present in human samples plus a number of sporadic or rare congeners (DeVoto et al. 1997; McFarland and Clarke 1989; Schecter et al. 1994).

The GC-ECD columns employed were a Hewlett-Packard (Palo Alto, CA) Ultra II 5% phenylmethyloctadecylsilyl-bonded (DB-5) fused silica (25 m, 0.33 µm film thickness, 0.25 mm inner diameter) capillary column and a fused silica Apiezon L (30 m, 0.25 µm film thickness, 0.25 mm inner diameter) column. Instrumentation consisted of two dual-column Hewlett-Packard 5890 GCs equipped with electron capture detectors and autosamplers. Limits of detection for individual congeners, based on a 5-g serum sample, were typically 0.01–0.04 ppb (whole-weight basis). The SPH Analytical Laboratory is accredited by the Clinical Laboratory Evaluation Program of the New York State Department of Health. The quality assurance/quality control program for the laboratory was developed in accordance with recommendations in relevant publications (Brock et al. 1996; Greizerstein et al. 1997; Hess et al. 1995).

Determination of total blood mercury was based on cold-vapor atomic absorption spectrometry. The coefficient of variation for organic mercury was 5.7% and for inorganic mercury was 6.3%. The limit of detection was 0.01 µg/dL for total blood mercury. Blood lead was analyzed by Zeeman-corrected graphite furnace atomic absorption spectrometry.

The coefficient of variation, calculated for "n = 10" different days and analytical runs, ranged from 2.5% to 8.7%. The limit of detection for blood lead was 1.0 µg/dL.

Statistical methods. PCB congeners for which all reported values were below the laboratory method detection limit (MDL) included International Union for Pure and Applied Chemistry (IUPAC) congeners 1, 3, 6, 63, 67, and 185. These congeners were not included in any calculations.

Many of the congeners were detected in only a portion of the sample. For the purpose of describing the distributions of congeners, the undetected values were treated in two ways. Following common practice, we replaced any individual datum of an OC that was below MDL with the midpoint value between zero and the MDL of each compound or congener. This method is called the MDL/2 substitution method. Using this approach, we calculated for each participant one summary variable, Total_PCBs, as the sum of the 94 congeners (in 85 analytical peaks) that were detected.

A second approach followed the U.S. Environmental Protection Agency (U.S. EPA) recommendation for estimating values for distributions of nondetectable amounts of toxicants in tissue or fluid samples (U.S. EPA 1998). Estimation of parameters of normal populations with nondetectable observations can be considered a problem concerning censored data. This statistical problem has been approached and solved by many authors (Cohen 1950, 1959; Gupta 1952; Hald 1949). We employed the formulae described by Gupta (1952) for populations that are not large because these formulae are more easily computed and are equivalent to Cohen's (Cohen 1950). To estimate parameters of the whole distribution, the percentage of detects and point of censoring must be known, as they were in our sample, and the mean and variance of detected observations were computed. The method relies on Gupta's equation 8, which was solved using code written in Visual Basic (Visual Basic 6.0; Microsoft, Redmond, WA) to create a function for Excel (Excel 2000; Microsoft). Initially, Newton's method for root solving was used to find a numerical solution of Gupta's equation 8, and then, if Newton's method failed to find

Table 1. Summary and specific PCB congener levels within the Akwesasne Mohawk adolescent population (ppb).

				Substitution method											
				MDL/2[a]			U.S. EPA method[b]			Percentiles (no MDL substitution)					
PCBs	Structure	MDL	ROD (%)[c]	Mean	GM	SD	Mean	GM	SD	5	25	50	75	95	Maximum
Summary															
Total PCBs[a]		—	—	1.71	1.62	0.641	—	—	—	1.11	1.29	1.51	1.94	3.20	4.74
$\Sigma PCB_{50\%}$[b,d]		—	—	0.72	0.65	0.372	0.73	0.66	0.366	0.34	0.48	0.63	0.88	1.46	2.70
$\Sigma PCB_{75\%}$[b,e]		—	—	0.57	0.51	0.308	0.57	0.51	0.306	0.25	0.38	0.48	0.69	1.14	2.44
Σ of persistent PCBs[b,f]	—	—	—	0.43	0.38	0.259	0.44	0.39	0.257	0.20	0.29	0.36	0.53	0.87	2.45
PCB IUPAC congener															
118	2,3′,4,4′,5	0.02	99.6	0.07	0.06	0.041	0.07	0.06	0.041	0.03	0.04	0.06	0.09	0.15	0.28
101[+90][g]	2,2′,3,4′,5 + 2,2′4,5,5′	0.02	98.5	0.06	0.05	0.040	0.06	0.05	0.040	0.02	0.03	0.04	0.07	0.14	0.30
153	2,2′,4,4′,5,5′	0.02	97.8	0.10	0.08	0.087	0.10	0.09	0.087	0.04	0.06	0.08	0.13	0.23	0.98
110	2,3,3′,4′,6	0.02	95.2	0.06	0.05	0.040	0.06	0.05	0.040	0.02	0.04	0.05	0.08	0.14	0.34
99	2,2′,4,4′,5	0.02	94.5	0.05	0.04	0.029	0.05	0.04	0.029	—	0.03	0.05	0.06	0.10	0.21
87	2,2′,3,4,5′	0.02	91.5	0.04	0.04	0.024	0.04	0.04	0.024	—	0.03	0.04	0.05	0.10	0.16
138[+163+164][g]	2,2′,3,4,4′,5′ + 2,3,3′,4′,5′,6 + 2,3,3′,4′,5,6	0.02	90.0	0.08	0.06	0.055	0.08	0.07	0.055	—	0.05	0.07	0.10	0.18	0.47
180	2,2′,3,4,4′,5,5′	0.02	89.3	0.05	0.04	0.053	0.05	0.04	0.052	—	0.02	0.04	0.06	0.15	0.39
95	2,2′,3,5′,6	0.02	79.3	0.03	0.02	0.019	0.03	0.03	0.019	—	0.02	0.02	0.03	0.06	0.15
52	2,2′,5,5′	0.02	78.6	0.03	0.03	0.028	0.03	0.03	0.027	—	0.02	0.02	0.04	0.09	0.16
74	2,4,4′,5	0.02	73.4	0.03	0.02	0.038	0.03	0.02	0.037	—	—	0.02	0.03	0.06	0.53
105	2,3,3′,4,4′	0.02	63.5	0.02	0.02	0.017	0.02	0.02	0.016	—	—	0.02	0.03	0.05	0.13
149[+123][g]	2,2′,3,4′,5′,6 + 2,3′,4,4′,5′	0.02	59.0	0.02	0.02	0.019	0.02	0.02	0.018	—	—	0.02	0.03	0.06	0.12
187	2,2′,3,4′,5,5′,6	0.02	57.6	0.02	0.02	0.018	0.02	0.02	0.017	—	—	0.02	0.02	0.05	0.15
70	2,3′,4′,5	0.02	55.4	0.02	0.02	0.017	0.02	0.02	0.016	—	—	0.02	0.03	0.06	0.11
84	2,2′,3,3′,6	0.02	53.1	0.02	0.02	0.010	0.02	0.02	0.009	—	—	0.02	0.02	0.04	0.08
170	2,2′,3,3′,4,4′,5	0.02	45.0	—	—	—	—	—	—	—	—	—	0.02	0.05	0.13
44	2,2′,3,5′	0.02	44.6	—	—	—	—	—	—	—	—	—	0.03	0.06	0.10
66	2,3′,4,4′	0.02	37.6	—	—	—	—	—	—	—	—	—	0.02	0.04	0.08
199	2,2′,3,3′,4,5,5′,6′	0.01	37.3	—	—	—	—	—	—	—	—	—	0.01	0.03	0.13
132	2,2′,3,3′,4,6′	0.02	34.7	—	—	—	—	—	—	—	—	—	0.02	0.03	0.08
28	2,4,4′	0.02	33.6	—	—	—	—	—	—	—	—	—	0.02	0.13	0.25
31	2,4′,5	0.02	31.7	—	—	—	—	—	—	—	—	—	0.02	0.08	0.18
158	2,3,3′,4,4′,6	0.01	31.7	—	—	—	—	—	—	—	—	—	0.01	0.02	0.05
47[+59][g]	2,2′,4,4′ + 2,3,3′,6	0.02	28.4	—	—	—	—	—	—	—	—	—	0.02	0.06	0.12
146	2,2′,3,4′,5,5′	0.02	28.0	—	—	—	—	—	—	—	—	—	0.02	0.04	0.22
183	2,2′,3,4,4′,5′,6	0.01	28.0	—	—	—	—	—	—	—	—	—	0.01	0.02	0.15
92	2,2′,3,5,5′	0.02	27.3	—	—	—	—	—	—	—	—	—	0.02	0.03	0.06
174	2,2′,3,3′,4,5,6′	0.01	26.9	—	—	—	—	—	—	—	—	—	0.01	0.02	0.05
177	2,2′,3,3′,4,5′,6′	0.01	25.5	—	—	—	—	—	—	—	—	—	0.01	0.02	0.06
141	2,2′,3,4,5,5′	0.02	25.5	—	—	—	—	—	—	—	—	—	0.02	0.02	0.06
18	2,2′,5	0.02	25.1	—	—	—	—	—	—	—	—	—	0.02	0.04	0.06
29	2,4,5	0.01	24.7	—	—	—	—	—	—	—	—	—	—	0.02	0.06
33	2,3′,4′	0.02	24.0	—	—	—	—	—	—	—	—	—	—	0.06	0.15
151	2,2′,3,5,5′,6	0.02	22.9	—	—	—	—	—	—	—	—	—	—	0.02	0.05
8	2,4′	0.02	22.5	—	—	—	—	—	—	—	—	—	—	0.05	0.09
97	2,2′,3,4′,5′	0.02	22.1	—	—	—	—	—	—	—	—	—	—	0.03	0.07
130	2,2′,3,3′,4,5′	0.01	21.4	—	—	—	—	—	—	—	—	—	—	0.02	0.06
196	2,2′,3,3′,4,4′,5,6′	0.01	21.4	—	—	—	—	—	—	—	—	—	—	0.03	0.19
56	2,3,3′,4′	0.02	18.8	—	—	—	—	—	—	—	—	—	—	0.02	0.03

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the root, a modified *regula falsi* method was applied. A random sample of values derived from this application was checked against tables in the original publication. The Excel add-in application is available on request from one of the authors (L.A.H.). Following the U.S. EPA recommendation, this method was applied for congeners that were detected in 50% or more of the samples.

Using this estimation method, several variables summarizing commonly detected PCBs were calculated: $Sum_{75\%}$ (the sum of all congeners found in 75% or more of the samples) and $Sum_{50\%}$ (the sum of all congeners found in 50% or more of the samples). An additional summary variable, Persistent_PCBs, was calculated as the sum of the concentrations of persistent congeners found in 50% or more of the samples. Persistent congeners are those known or expected to have long physiologic half-lives in humans due to high lipid solubility and/or low rates of metabolism (Brown 1994). Persistent congeners include IUPAC congeners 74, 99, 105, 118, 138[+163+164], 153, 180, and 187. In contrast, IUPAC congeners 52, 84, 95, 101[+90], 110, and 149[+123] are generally considered to be nonpersistent congeners. Although some classification schemes do include IUPAC congeners 87 and 70 as persistent (Hansen 2001), other data suggest that they should be fairly readily metabolized in humans (Brown 1994). Because of this uncertainty, they were excluded from the persistent congener variable. For congeners detected in fewer than 50% of the sample, no mean or variance is reported but available percentiles and a maximum value are reported following the U.S. EPA recommendation (Table 1).

We log-transformed the $Sum_{50\%}$, $Sum_{75\%}$, Persistent_PCBs, Total_PCBs, and data on any individual congener detected in 50% or more of the samples to normalize their distributions.

All statistical testing was performed using SAS, version 8.1 (SAS Institute 2001) and SPSS, version 10.1.4 (SPSS 2001).

Results

The average age of the sample was 13.2 years; males and females did not differ significantly in age. The average height-for-age percentile using National Center for Health Statistics–growth charts (Epi Info, version 6; Centers for Disease Control and Prevention 2001) was 52%, whereas the average weight-for-age percentile

Table 1. *Continued.*

				Substitution method											
				MDL/2[a]			U.S. EPA method[b]			Percentiles (no MDL substitution)					
PCB IUPAC congener	Structure	MDL	ROD (%)[c]	Mean	GM	SD	Mean	GM	SD	5	25	50	75	95	Maximum
71	2,3′,4′,6	0.02	16.6	—	—	—	—	—	—	—	—	—	—	0.03	0.08
42	2′,2′,3,4′	0.01	15.1	—	—	—	—	—	—	—	—	—	—	0.02	0.03
190	2,3,3′,4,4′,5,6	0.02	14.8	—	—	—	—	—	—	—	—	—	—	0.03	0.10
128	2,2′,3,3′,4,4′	0.02	14.8	—	—	—	—	—	—	—	—	—	—	0.02	0.07
77	3,3′,4,4′	0.02	14.8	—	—	—	—	—	—	—	—	—	—	0.05	0.09
156	2,3,3′,4,4′,5	0.02	14.4	—	—	—	—	—	—	—	—	—	—	0.03	0.14
144	2,2′,3,4,5′,6	0.02	13.7	—	—	—	—	—	—	—	—	—	—	0.02	0.07
49	2,2′,4,5′	0.03	13.3	—	—	—	—	—	—	—	—	—	—	0.07	0.16
40	2,2′,3,3′	0.02	13.3	—	—	—	—	—	—	—	—	—	—	0.02	0.06
24+27	2,3,6 + 2,3′,6	0.02	12.9	—	—	—	—	—	—	—	—	—	—	0.05	0.08
203	2,2′,3,4,4′,5,5′,6	0.02	12.9	—	—	—	—	—	—	—	—	—	—	0.03	0.07
176	2,2′,3,3′,4,6,6′	0.01	11.8	—	—	—	—	—	—	—	—	—	—	0.01	0.08
172	2,2′,3,3′,4,5,5′	0.02	10.7	—	—	—	—	—	—	—	—	—	—	0.03	0.25
15	4,4′	0.03	10.3	—	—	—	—	—	—	—	—	—	—	0.05	0.13
53	2,2′,5,6′	0.02	10.0	—	—	—	—	—	—	—	—	—	—	0.02	0.09
179	2,2′,3,3′,5,6,6′	0.01	9.2	—	—	—	—	—	—	—	—	—	—	0.01	0.06
194	2,2′,3,3′,4,4′,5,5′	0.02	8.9	—	—	—	—	—	—	—	—	—	—	0.02	0.30
17	2,2′,4	0.03	8.9	—	—	—	—	—	—	—	—	—	—	0.03	0.35
32+16	2,4′,6 + 2,2′,3	0.04	8.5	—	—	—	—	—	—	—	—	—	—	0.05	0.10
19	2,2′,6	0.03	8.1	—	—	—	—	—	—	—	—	—	—	0.05	0.16
22	2,3,4′	0.04	7.7	—	—	—	—	—	—	—	—	—	—	0.05	0.08
46	2,2′,3,6′	0.02	7.4	—	—	—	—	—	—	—	—	—	—	0.02	0.08
4[+2][g]	2,2′ + 3	0.02	7.0	—	—	—	—	—	—	—	—	—	—	0.04	0.19
83	2, 2′, 3, 3′, 5	0.02	7.0	—	—	—	—	—	—	—	—	—	—	0.02	0.02
9	2,5	0.02	6.6	—	—	—	—	—	—	—	—	—	—	0.02	0.03
10	2,6	0.02	6.3	—	—	—	—	—	—	—	—	—	—	0.02	0.08
136	2,2′,3,3′,6,6′	0.03	6.3	—	—	—	—	—	—	—	—	—	—	0.03	0.08
134	2,2′,3,3′,5,6	0.01	5.9	—	—	—	—	—	—	—	—	—	—	0.01	0.02
201	2,2′,3,3′,4,5′,6,6′	0.02	5.5	—	—	—	—	—	—	—	—	—	—	0.02	1.08
26	2,3′,5	0.03	4.8	—	—	—	—	—	—	—	—	—	—	—	0.10
7	2,4	0.02	4.8	—	—	—	—	—	—	—	—	—	—	—	0.10
171	2,2′,3,3′,4,4′,6	0.02	4.4	—	—	—	—	—	—	—	—	—	—	—	0.09
25	2,3′,4	0.01	4.1	—	—	—	—	—	—	—	—	—	—	—	0.06
206	2,2′,3,3′,4,4′,5,5′,6	0.02	3.3	—	—	—	—	—	—	—	—	—	—	—	0.09
64	2,3,4′,6	0.02	3.3	—	—	—	—	—	—	—	—	—	—	—	0.03
114	2,3,4,4′,5	0.02	3.3	—	—	—	—	—	—	—	—	—	—	—	0.04
137	2,2′,3,4,4′,5	0.02	3.3	—	—	—	—	—	—	—	—	—	—	—	0.03
195	2,2′,3,3′,4,4′,5,6	0.02	3.0	—	—	—	—	—	—	—	—	—	—	—	0.03
45	2,2′,3,6	0.04	2.6	—	—	—	—	—	—	—	—	—	—	—	0.06
51	2,2′,4,6′	0.05	1.8	—	—	—	—	—	—	—	—	—	—	—	0.11
91	2,2′,3,4′,6	0.03	1.8	—	—	—	—	—	—	—	—	—	—	—	0.04
13	3,4′	0.02	1.5	—	—	—	—	—	—	—	—	—	—	—	0.04
109[+147][g]	2,3,3′,4′,5 + 2,2′,3,4′,5,6	0.03	1.1	—	—	—	—	—	—	—	—	—	—	—	0.06
129	2,2′,3,3′,4,5	0.02	1.1	—	—	—	—	—	—	—	—	—	—	—	0.07
200	2,2′,3,3′,4,5,6,6′	0.02	0.7	—	—	—	—	—	—	—	—	—	—	—	0.15

[a]Values below the detection limit have been replaced by the value midway between the detection limit and zero (MDL/2 method). [b]Values below the MDL were calculated following the U.S. EPA recommended method for estimating nondetected values as described in "Materials and Methods." [c]Rate of detection. [d]Congeners with ≥ 75% detection rate; IUPAC congeners: 52, 74, 95, 99, 101[+90], 110, 118, 138[+163+164], 153, 180. [e]Congeners with ≥ 50% detection rate; IUPAC congeners: 52, 70, 74, 84, 87, 95, 99, 101[+90], 105, 110, 118, 149[+123], 138[+163+164], 153, 180, 187. [f]Sum of IUPAC congeners: 74, 99, 105, 118, 138[+163+164], 153, 180, 187. [g]Brackets indicate "minor" congener based on Aroclor concentration (Hansen 1998).

was 78% and 73% in males and females respectively. The average body mass index was 24.2, and was higher in males than females.

All summary measures of POPs and congener-specific PCB levels (found in 50% or more of the samples) are reported (Tables 1 and 2) to facilitate comparison with other studies. Ten chromatographic peaks, containing 13 congeners, were detected in 75% or more of the individuals sampled and an additional six peaks, containing seven congeners were detected in 50 to 75% of the sample. Of the 20 congeners detected in 50% or more of the samples, 17 were highly chlorinated (i.e., five or more chlorine substitutions). Five of the 20 are mono-*ortho* substituted and 11 are di-*ortho* substituted. Eight congeners have substitutions at both *para* positions (i.e., 4,4′-substituted) and may be considered highly persistent. Of the other toxicants (Table 2), only *p,p′*-DDE was detected in 100% of the sample, whereas HCB was detected nearly as often. Lead levels were low; no participant had a level at or above 10 µg/dL, the current level of concern in the United States. All participants but one had a mercury level below 0.5 µg/dL, which is below the blood levels reportedly associated with health effects in humans (ATSDR 2000).

Breast-feeding was an important influence on PCB levels in the sample even though lactation had ceased many years before the blood sampling (Table 3). With sexes combined, breast-fed youth had on average 1.3 times the level of Total_PCBs, $Sum_{75\%}$, $Sum_{50\%}$, Persistent_PCBs, mirex, and *p,p′*-DDE compared with non-breast-fed youth. HCB, lead, and mercury were similar in both groups. Of the congeners found in 50% or more of the samples, several were present at significantly higher levels in breast-fed individuals: IUPAC congeners 74, 87, 99, 105, 118, 153, 138[+163+164], 180, and 187.

Estimation and substitution method. We explored the effect of using the U.S. EPA recommended method for the estimation of toxicant distributions to impute values of nondetectable observations. For each congener observed in at least 50% of the samples, we compared the mean value of the distribution constructed with the U.S. EPA recommended method of estimation for nondetected observations to the mean value using the MDL/2 method of substitution. The correlation between the means of the congeners calculated in these two ways were high ($r > 0.999$), but the paired difference of log-transformed means equaled 0.025 (2.5%), a highly significant difference ($t = 14.77$, $p < 0.001$). The values estimated by the U.S. EPA recommended method for non-detected observations were, on average, 1.46 times (range, 1.26–1.67) the value of half the MDL of each congener. The ratio was correlated with the number of nondetected observations ($r = 0.78$, $p = 0.0003$) such that as the percentage of detected values increased, the difference between the two estimation algorithms increased also. This result is a direct function of the formulae used for estimation. We did not examine the influence of the U.S. EPA recommended estimation method on Total_PCBs. The estimation method should be applied only to distributions involving compounds detected in more than 50% of the samples, and 69 of the congeners included in the Total_PCBs summary variable did not meet this criterion.

Table 2. Lead, mercury, and non-PCB OC levels in adolescent Akwesasne Mohawk youth.

						Percentiles (no MDL substitution)					
	ROD (%)[a]	MDL	Mean[b]	GM[b]	SD	5	25	50	75	95	Maximum
p,p′-DDE (ppb)	100.0	0.02	0.431	0.368	0.3447	0.19	0.26	0.35	0.48	0.91	3.08
HCB (ppb)[a]	97.8	0.02	0.037	0.034	0.0194	0.02	0.03	0.03	0.04	0.06	0.19
Mirex (ppb)[a]	45.80	0.02	0.036	0.017	0.0924	—	—	—	0.04	0.09	1.17
Blood lead (µg/dL)[a]	70.90	0.10	1.309	0.706	0.9699	—	—	1.40	1.90	2.90	4.80
Mercury (µg/dL)[a]	93.60	0.02	0.118	0.090	0.0968	—	0.05	0.09	0.16	0.30	0.58

[a]Rate of detection. [b]Values below the MDL were calculated following the U.S. EPA recommended method for estimating nondetected values, as described in "Materials and Methods."

Table 3. Summary and specific PCB congener levels in adolescent Akwesasne Mohawk youth: breast-fed versus non-breast-fed (ppb).

	Not breast-fed ($n = 145$)[a]				Breast-fed ($n = 124$)[a]				Breast-fed/ not breast-fed		
	GM	Mean	Max	SD	GM	Mean	Max	SD	GM ratio	*t*-Value	*p*-Value
Total PCBs[b]	1.53	1.61	3.79	0.548	1.74	1.84	4.74	0.719	1.13	3.367	≤ 0.001
$\Sigma PCB_{50\%}$[c,d]	0.59	0.64	1.66	0.272	0.76	0.84	2.70	0.430	1.28	4.890	≤ 0.001
$\Sigma PCB_{75\%}$[c,e]	0.45	0.49	1.43	0.220	0.60	0.67	2.44	0.362	1.32	5.119	≤ 0.001
Σ Persistent PCBs[c,f]	0.33	0.36	1.44	0.180	0.46	0.52	2.45	0.306	1.39	6.022	≤ 0.001
p,p′-DDE[b]	0.31	0.34	1.23	0.162	0.45	0.54	3.08	0.456	1.45	6.118	≤ 0.001
HCB[c]	0.03	0.04	0.19	0.021	0.04	0.04	0.13	0.017	1.07	1.384	0.168
Mirex[c]	0.02	0.03	0.38	0.040	0.02	0.05	1.17	0.129	1.26	1.895	0.059
PCB IUPAC congener											
52	0.03	0.03	0.16	0.026	0.03	0.04	0.14	0.029	1.06	0.774	0.439
70	0.02	0.02	0.11	0.015	0.02	0.03	0.08	0.018	1.12	1.832	0.068
74	0.02	0.02	0.09	0.011	0.03	0.04	0.53	0.053	1.44	5.512	≤ 0.001
84	0.02	0.02	0.05	0.007	0.02	0.02	0.08	0.010	1.05	1.159	0.247
87	0.03	0.04	0.13	0.020	0.04	0.05	0.16	0.027	1.16	2.345	0.020
101[+90][g]	0.05	0.05	0.20	0.033	0.05	0.06	0.30	0.047	1.13	1.662	0.098
95	0.02	0.03	0.08	0.014	0.03	0.03	0.15	0.023	1.12	1.782	0.076
99	0.04	0.04	0.13	0.021	0.05	0.06	0.21	0.034	1.29	4.120	≤ 0.001
105	0.02	0.02	0.06	0.011	0.02	0.03	0.13	0.019	1.14	2.309	0.022
110	0.05	0.06	0.16	0.032	0.06	0.07	0.34	0.047	1.15	1.916	0.056
118	0.06	0.06	0.16	0.030	0.07	0.08	0.28	0.049	1.24	3.459	0.001
149[+123][g]	0.02	0.02	0.08	0.015	0.02	0.03	0.12	0.021	1.07	0.967	0.334
153	0.07	0.08	0.39	0.053	0.11	0.13	0.98	0.110	1.48	5.700	≤ 0.001
138[+163+164][g]	0.06	0.07	0.34	0.041	0.08	0.10	0.47	0.065	1.35	3.809	≤ 0.001
180	0.03	0.04	0.29	0.043	0.05	0.07	0.39	0.060	1.52	4.900	≤ 0.001
187	0.02	0.02	0.15	0.016	0.02	0.03	0.14	0.017	1.25	3.983	≤ 0.001
Blood lead (µg/dL)[a,c]	0.70	1.30	4.80	0.987	0.72	1.33	4.40	0.955	1.03	0.165	0.869
Mercury (µg/dL)[a,c]	0.09	0.12	0.58	0.098	0.09	0.12	0.05	0.095	0.96	0.469	0.639

Max, maximum level detected in sample.
[a]The *n* for blood lead and mercury is 142 for non-breast-fed and 121 for breast-fed. [b]Values below the detection limit have been replaced by the value midway between the detection limit and zero (MDL/2 method). [c]Values below the MDL were calculated following the U.S. EPA recommended method for estimating nondetected values as described in "Materials and Methods." [d]Congeners with ≥ 50% detection rate; IUPAC congeners 52, 70, 74, 84, 87, 95, 99, 101[+90], 105, 110, 118, 149[+123], 138[+163 = 164], 153, 180, 187. [e]Congeners with ≥ 75% detection rate; IUPAC congeners 52, 74, 95, 99, 101[+90], 110, 118, 138[+163 = 164], 153, 180. [f]Sum of IUPAC congeners 74, 99, 105, 118, 138[+163+164], 153, 180, 187. [g]Brackets indicate "minor" congener based on Aroclor concentration (Hansen 1998).

To determine the influence of the MDL/2 method of substitution on Total_PCBs, we calculated the contribution to this total by adding half the MDL for each of the 69 congeners that were not detected in 50% or more of the samples and found that it equaled 0.57 ppb of Total_PCBs. In other words, if each nondetectable value had been substituted with zero instead of half the MDL, the geometric mean (GM) of the summary variable, Total_PCBs, would have averaged 1.14 ppb instead of 1.71 ppb as we report here.

Discussion

High sensitivity, congener-specific PCB analysis has become an important tool for investigating the sources, time frame, and intensity of exposure to these ubiquitous contaminants in human populations. PCB analysis of blood specimens from children and youth is a particular challenge, because of the typically low (i.e., < 100 ppt) levels of individual congeners present. For example, whereas almost 50 congeners were recently reported at a 50% or higher detection rate in a cohort of Akwesasne Mohawk adults (DeCaprio et al. 2000), only one-third as many were noted in Akwesasne Mohawk youth in the present study. These lower levels may be a function of a shorter potential bioaccumulation period in children compared with adults and also a decline in consumption of local fish and wildlife in the community since the mid-1980s (Fitzgerald et al. 1995, 1999). Despite these limitations, the Mohawk youth PCB database provides a unique opportunity to understand the toxicokinetics and potential health effects of these contaminants within this age group.

Serum levels of individual PCB congeners reflect their persistence in tissue storage depots such as adipose tissue because of differences in lipid solubility and toxicokinetics. Metabolic rates for individual congeners are highly dependent on chlorine substitution pattern, with *para*,*para*′- (i.e., 4,4′-) substitution generally associated with slow metabolism and high persistence (Brown 1994). Congeners most frequently detected at high levels in serum include IUPAC congeners 118, 138 [+163+164], 153, and 180, which are penta- or higher chlorinated and *para*-substituted. These congeners have physiologic half-lives in humans that may range up to a decade or more, and their serum levels reflect cumulative exposure over many years (Hansen 1998). In contrast, congeners with at least one *para* ring position unsubstituted, particularly those with *meta*,*para*-unsubstituted ring positions and lower chlorination levels, are more rapidly metabolized (Safe 1994). Their presence in serum is good evidence for recent exposure or, alternatively, genotypic or phenotypic factors contributing to lower rates of metabolism. PCB IUPAC congeners 52, 95, 101[+90], and 110 fall into this category.

In the present study, patterns and levels of serum PCB congeners in Mohawk youth were consistent with a combination of cumulative and recent PCB exposure. The relatively high detection rates of the shorter-lived congeners (52, 84, 95, 101[+90], 110, and 149[+123]) suggest a continuing source of exposure. The possible source of recent exposures in Mohawk youth is not known but may be related, in part, to ambient exposure to PCB-contaminated environmental media (i.e., air, soil, sediment)

Table 4. Comparison of PCB results (ppb) with other studies of similarly aged samples.

Study (year)	Age range			*n*	Toxicant level	Statistic compared	Mohawk study Toxicant level[a]	Mohawk study *n*
Karmaus et al. (2001) [sample used in Osius et al. (1999)]	7–10 years	Σ of 8 PCB congeners[b]	BF	44	0.98	GM	0.44	124
			NBF	293	0.64		0.32	145
		HCB	BF	44	0.42		0.04	124
			NBF	293	0.29		0.03	145
		p,p′-DDE[c]	BF	44	0.60		0.45	124
			NBF	293	0.40		0.31	145
		Σ of 8 PCB congeners[b]	M	192	0.64		0.39	131
			F	145	0.53		0.36	140
		HCB	M	192	0.31		0.04	131
			F	145	0.29		0.03	140
		p,p′-DDE[c]	M	192	0.55		0.39	131
			F	145	0.56		0.35	140
Osius et al. (1999) [sample used in Osius et al. (1999)]	7–10 years	Σ of 7 PCB congeners[d]		320	0.89	GM	0.32	271
		Σ of 3 PCB congeners[e]		298	0.71		0.22	271
		118		319	0.05		0.06	271
		138		320	0.24		0.07	271
		153		320	0.31		0.08	271
		170		314	0.07		0.02	271
		180		320	0.15		0.04	271
		183		300	0.04		0.01	271
		187		320	0.04		0.02	271
Mazhitova et al. (1998)	7.5–15 years	Σ of 7 PCB congeners[f]		12	0.89	Median	0.34	271
		105		12	0.06		0.02	271
		101		12	0.14		0.04	271
		118		12	0.15		0.06	271
		138		12	0.19		0.07	271
		153		12	0.27		0.08	271
		156		12	0.03		0.01	271
		180		12	0.09		0.04	271
Staessen et al. (2001) [sample used in Nawrot et al. (2002)]	17 years	Σ of Marker PCBs[g]	CG[h]	100	0.44	GM	0.22	271
			SG1[h]	42	0.55		0.22	271
			SG2[h]	58	0.44		0.22	271
Nawrot et al. (2002) [sample used in Staessen et al. (2001)]	17 years	Σ of Marker PCBs[g]	M	80	0.62	GM	0.23	131
			F	120	0.38		0.21	140

Abbreviations: BF, breast-fed; F, Females; M, Males; NBF, non-breast-fed.
[a]Values below the MDL were calculated following the U.S. EPA recommended method for estimating nondetected values as described in "Materials and Methods," [b]Σ of IUPAC congeners 101, 118, 138, 153, 170, 180, 183, 187. Karmaus et al. (2001) report OC concentrations in whole blood controlling for covariates in their model. Converted from whole blood in μg/L: divided values given by 0.55, which assumes 45% hematocrit. [c]Values below the detection limit have been replaced by the value midway between the detection limit and zero. [d]Σ of IUPAC congeners 118, 138, 153, 170, 180, 183, 187. Converted from whole blood in μg/L: divided values given by 0.55, which assumes 45% hematocrit. [e]Σ of IUPAC congeners 138, 153, 180. [f]Σ of IUPAC congeners 101, 118, 153, 105, 138, 156, 180. Converted into ppb by dividing reported level by 200. [g]Σ of marker PCBs: IUPAC congeners 138, 153, 180. [h]CG: Control Group, SG1: Study group from Wilrijik, SG2: Study group from Hoboken. Adjusted for sex, body mass index, weeks breast-feeding, parental social class, dietary fat intake. Values converted from nmol/L to ppb by multiplying by 0.372.

associated with past discharges from local industries adjacent to the Akwesasne community (Chiarenzelli et al. 2001; Ramil et al. 2002). The elevated serum levels of the persistent congeners 118, 138[+163+164], 153, and 180 are typical of cumulative, nonoccupational exposure scenarios. For the present cohort, past exposure may include prenatal transfer and postnatal uptake via breast-feeding and/or fish consumption. The higher total serum PCB and higher levels of individual congeners 74, 99, 118, 138[+163+164], 153, 180, and 187 in participants who were breast-fed indicate breast-feeding as a potentially significant source of persistent congeners in the present study. Similar findings have been reported in other studies (Karmaus et al. 2001; Patandin et al. 1997). These elevations are also consistent with previous analyses of PCB content in breast milk of Mohawk mothers (Fitzgerald et al. 1998) and other maternal cohorts with suspected PCB exposure (Bush et al. 1984; Duarte-Davidson et al. 1994; Greizerstein et al. 1999). Finally, the high prevalence of persistent congeners 118 and 138 in conjunction with relatively low levels of 199, 203, and 206 is consistent with the known discharge of Aroclor 1248 as the major contaminant to the river.

Some comparisons between the present data and published studies of blood PCB levels in children and youth can be made. These comparisons are complicated by differences in analytic methodologies, the choice of individual congeners measured, the estimation method for nondetected observations, the metric used to calculate total PCB levels, and a lack of uniformity in reporting results by breast-feeding status, age, and individual congeners. We examined all relevant studies published from January 1995 to the present that involved persons 17 years or younger. In general, levels of Total_PCBs for the Mohawk youth are somewhat lower than those reported for cohorts of similar age (Table 4). For example, Osius et al. (1999) reported total PCB levels as the sum of either three or seven persistent congeners in whole blood from a large cohort of German 7–10-year-olds. Assuming a hematocrit of 45% (DeKoning and Karmaus 2000), their data convert to total serum PCB levels of 0.89 and 0.71 ppb, respectively, compared with values of 0.32 and 0.22 ppb in the present study. Comparable differences in total serum PCB levels calculated as the sum of eight persistent congeners were reported by Karmaus et al. (2001) in a subset of the Osius et al. (1999) cohort. Their data indicated PCB levels of 0.98 and 0.64 ppb in breast-fed and non-breast-fed individuals, respectively, compared with values of 0.44 and 0.32 ppb in the present study. Similar differences are apparent for *p,p′*-DDE between the cohorts, whereas serum HCB levels are 10-fold higher in the German children compared with the Mohawk youth. Because the body burden of HCB is derived from a number of environmental and industrial sources, its level tends to be highly variable among study populations.

Generally higher total PCB levels (based on various summation procedures) have also been reported in a small cohort of hospitalized 7.5–15-year-olds in the Aral Sea region (Mazhitova et al. 1998) and in a group of 17-year-old Belgian adolescents (Nawrot et al. 2002; Staessen et al. 2001). Differences in age, potential exposure sources, and overall intensity of exposure between these cohorts likely account for some or all of the observed differences in total serum PCB levels. For example, in the German children, uptake via contaminated vegetables and general ambient exposure associated with regional industry predominates (Osius et al. 1999), whereas exposure among Belgian adolescents is attributed primarily to the presence of hazardous waste incinerators and other combustion sources in the vicinity (Nawrot et al. 2002; Staessen et al. 2001). Within the Akwesasne Mohawk community, contaminated fish was an important source of exposure. Levels of PCBs in Mohawk youth reflect the decline in local fish consumption in the 1980s following well-publicized advisories recommending local fish not be consumed (Fitzgerald et al. 1995, 1999).

In addition to comparing total serum PCB levels, important conclusions can be drawn through comparison of individual PCB congener distributions among the cohorts. For example, the relative levels of each congener reported for the exposed samples near Antwerp (Nawrot et al. 2002; Staessen et al. 2001) are almost identical to the pattern seen in the Mohawk youth, with persistent congeners 118, 138, and 153 predominating. Interestingly, the relative contribution of the less persistent PCB 101 to total body burden is also comparable between these cohorts. These data suggest similar primary sources (i.e., Aroclor mixtures), time frame of exposure, and exposure pathways for the two groups. Larger differences are present between the Mohawk youth and German children. Although PCBs 138 and 153 dominate the serum pattern in both groups, PCBs 118 and 180 are more and less significant contributors, respectively, in the Mohawks compared with the German cohort. In addition, the lack of detection of PCB 101 in German children in comparison with its significant presence in the Mohawk youth is noteworthy and may reflect the presence of continuing PCB exposure in the latter group.

Our examination of the effects of different methods of estimating values for nondetectable levels suggests that reported levels of PCBs may be significantly affected by this choice. The computationally simpler method of substituting nondetected values with the mid-point between zero and the MDL (the MDL/2 method) underestimates PCB burden by a small amount in comparison with the values obtained by using the method recommended by the U.S. EPA. The difference in estimated values is small in absolute terms, but statistically significant. For congeners detected in 50% or more of individuals in our sample, the U.S. EPA method produces a value that averages 1.5 times the mean estimated by the MDL/2 method. When PCB levels are low, this difference is small, but it is compounded when summary measures of PCBs are computed. It may be worthwhile to examine this potential bias in reporting summaries of PCB levels and consider it when making comparisons across different studies.

The levels of mercury and lead were low. No participants had blood lead levels above the U.S. Centers for Disease Control and Prevention's action level of 10 µg/dL (Centers for Disease Control and Prevention 2000), and all but one had blood mercury levels below that associated with health effects in humans (0.50 µg/dL), according to the U.S. EPA (1997a, 1997b). The lower level of mercury seen here may be a function of the decline in consumption of local fish in the community because of the advisories that went into effect in the mid 1980s. The low level of lead may be related to a substantial phase of new construction following the ban on the use of lead in paint, and the predominantly rural character of the community.

The interpretation of these results is limited by incomplete knowledge of the target population and the number of potential participants in and out of the study. Although bias in sample selection is always a possibility without randomization, the participants had no knowledge of their PCB level or actual exposure to motivate them to participate or to refuse.

Finally, the effect of contamination in this community is not fully indexed by the toxicant burdens reported here. Typical models of exposure risk do not fit all communities, and Native American communities are potentially affected differentially by environmental contamination in comparison with surrounding communities. Recent reports have suggested that more holistic models of risk assessment are needed to incorporate the social, cultural, and spiritual implications of risk management on the health of a community exposed to toxicants (Arquette et al. 2002), because avoiding toxicants may involve costs not apparent to the dominant culture that have far-reaching health effects themselves. For example, the cessation of fishing that followed advisories against consumption of local fish at Akwesasne has altered traditional subsistence patterns, having a profound effect on the preservation of indigenous Mohawk culture. Avoidance of foods and activities that may expose people to PCBs means that traditional activities are not performed and social bonds forged between generations through the transfer

of culture are not created. Additionally, the community has lost a primary source of protein and other nutrients such as iron, calcium, zinc, and essential omega-3 fatty acids due to the avoidance of contaminated foods, further exacerbating chronic, diet-related health problems in the community, such as diabetes and cardiovascular disease (Akwesasne Task Force on the Environment 1997; Arquette et al. 2002; Tarbell and Arquette 2000).

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