

Longitudinal Assessment of PCBs and Chlorinated Pesticides in Pregnant Women from Western Canada

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Abstract

Background

Maternal exposures to organochlorines prior to pregnancy are considered a risk to neonatal welfare, specifically in relation to neurocognitive functions. There is growing interest in the evaluation of maternal blood testing as a marker for fetal exposure.

Methods

Three hundred and twenty-two women in the second trimester of pregnancy were approached at a prenatal clinic providing genetic counseling information. Subjects who had an indication for genetic amniocentesis based on late maternal age were included. Two hundred and thirty-eight completed an environmental questionnaire. A sample of amniotic fluid was taken for karyotype analysis in 322 women and blood samples during pregnancy (209), at birth (105) and from the umbilical cord (97) and breast milk (47) were also collected. These samples were tested for 29 PCB congeners and organochlorine pesticides.

Results

The concentrations of PCB 153 in these media were relatively low in relation to other studies. Σ PCBs measurements in samples taken during the second trimester of pregnancy, at birth and in the umbilical cord were strongly correlated. Specific measurements of PCB 153 and PCB 180 among those subjects with completed sampling of blood samples from mothers and cord samples were significantly correlated. The concentrations of PCBs and pesticides did not differ in relation to prior spontaneous abortion history. There were no organochlorines present in the amniotic fluid....

Conclusions

Measurement of maternal blood during the second trimester of pregnancy can reliably estimate the fetal exposure to PCBs. This estimate is reliable for Group 2 and 3 PCBs as well as PCB 153 and PCB 180. Measurement of maternal blood during pregnancy provides a reliable estimate of fetal exposure as measured at birth.

Background

Polychlorinated biphenyls (PCBs) are ubiquitously present in the ecosystem and have been reported in the tissues of many animals and human population groups [1]. They are persistent and biomagnify in biota such that humans and predators at the top of the food chain experience the highest concentrations. Banned production of organochlorines in Canada and the United States in the 1970's has had an important effect in reducing exposure to these chemicals but because of the large residual quantities, release from storage and the limited rates of environmental degradation, they are still considered present in the environment.

Maternal exposure to PCBs and organochlorine pesticides is an area of intense research. This is due to the potential long term effects of organochlorine exposure in the fetus and newborn. Potentially severe adverse health effects during pregnancy have included preterm labor and intrauterine growth restriction in association with DDE, but the effects occurred at levels identified during the period 1959-1966[2]. These concentrations are substantially above exposure levels generally observed among current populations [3].

Postnatal adverse events have focused on the known neurotoxic effects of polychlorinated biphenyls [4]. Several studies have implicated exposure to these chemicals with impaired intellectual function in children [5, 6, 7]. Importantly the findings have demonstrated modest but significant effect sizes [7]. The potential mechanisms underlying these findings is unknown but recently dose-response relationships have been found between the concentrations of PCBs among members of the Oswego study and increases in response inhibition errors as well as reduced size of

the splenium of the corpus callosum [8]. Another proposed mechanism of toxicity in the developing brain is the potential for altered thyroid function, a known contributor to impaired intelligence among newborns [9, 10, 11]. There is now evidence the actions of PCBs may reflect a direct effect of the chemical on the action of thyroid hormone in the absence of altered blood concentrations [12].

The severity of the potential adverse central nervous system effects in children and the possibility that early intrauterine susceptibility to organochlorines is a major determinant of impaired intelligence indicates a need for further exposure information. There is little information related to the longitudinal patterns of PCB levels in women during the pregnancy. In some cases there was no correlation of maternal and cord samples [13], although others have reported that there is a strong correlation [14]. The measurement of serum concentrations over the course of the pregnancy and delivery appears not to have been explored in detail previously although the ability to evaluate fetal exposure during pregnancy is an important objective, particularly because of the susceptibility of the developing nervous system.

This study was undertaken to define the concentrations of PCBs and organochlorine pesticides in amniotic fluid, maternal blood collected during the second trimester, and at the time of birth, cord blood and breast milk among a cohort of pregnant women from Calgary, Alberta, Canada. The inter-relationships of the concentrations in these compartments were of interest to determine if maternal blood levels measured during pregnancy could serve as a proxy for fetal exposure. Further, we were particularly interested in the concentrations of PCBs in the Calgary region in relation to other studies that had evaluated neurocognitive development in local children. Finally, there was an interest in evaluating the concentrations of PCB 153 in

light of its proposed use for comparing across environmental epidemiologic studies of PCB toxicity [15].

Methods

Pregnant women attending a prenatal counseling session at Foothills Hospital in the Calgary Health Region, Calgary, Alberta were approached to participate in this project. Approval of the project was obtained from the University of Calgary Ethics Committee. Three hundred and twenty-three subjects were enrolled after their eligibility to participate was determined. They were required to be seeking genetic counselling for the purpose of age-related indications and not to have another reason for such counselling. Subjects were also required to be 35 years of age at the time of entry

Of those agreeing to participate, two hundred and thirty-eight completed an environmental questionnaire. A sample of amniotic fluid was available from three hundred and twenty-three women. A blood sample at the same time as the amniocentesis during the second trimester of pregnancy was available from 209 women. A blood sample at the time of delivery was made available in 105 women. Cord blood samples were collected from 97 women and a sample of breast milk was collected from 47 women after birth and during the puerperium from home. Clinical information related to the pregnancy and delivery was collected at the three Calgary hospitals and three hospitals outside the Calgary health region. All subjects were given the special tubes and phlebotomy supplies to submit various blood and breast milk samples and contacted on a regular basis for the completion of the sampling. The nursing staff on each delivery suite was oriented to the project to gain maximal compliance with the study.

The blood samples were collected by the regional laboratory in specially prepared glass tubes, centrifuged and the serum transferred to the Centre for Toxicology, University of Calgary for analysis. A similar approach was used for amniotic fluid.

The samples were assayed at the Centre for Toxicology, University of Calgary. They underwent sample clean-up and extraction by solid phase extraction techniques. The PCBs and most pesticides were measured with GC/negative chemical ionization spectrometry (GC/NCIMS). Some pesticides were measured with GC/electron ionization mass spectrometry (GC/EIMS).

In amniotic fluid the PCBs measured were as follows: PCB- 70, 74, 77, 87, 99, 101, 105, 118, 128, 138, 151, 153, 156, 169, 170, 180, 183, 187, 191, 194, 205, 206, 208, 209. The pesticides measured were: Aldrin, p,p'-DDE, p,p'-DDT, dieldrin, endosulfan I and II, endrin, heptachlor, hexachlorobenzene, hexachlorocyclohexanes, α -, β -, γ -, hexachloroethane, methoxychlor, mirex, pentachlorobenzene, 1,2,3-trichlorobenzene, 1,2,4-trichlorobenzene, 1,2,3,4-tetrahalorobenzene. These analytes were also measured in serum and breast milk with some minor changes

The limit of quantification (LOQ) varied among analytes as well as matrices. For example, the LOQ for most PCBs was 0.01 ng/ml in all matrices. The LOQ for most other industrial chemicals was 0.05 ng/ml, however there were exceptional situations. For example, the LOQ for p,p'-DDT was 0.5 ng/ml. In order to evaluate the measurements of Σ PCBs and Group 2 and 3 PCBs, $\frac{1}{2}$ of the level of detection was used in analysis. This may result in some bias in the analysis as only PCB 153, 138, 180 and 170 had detection rates >90% in maternal blood during pregnancy. There were similar patterns of detection rates for the PCBs 153, 138 and 103 in maternal blood at birth and PCBs 153 in cord blood and PCB 118, 153, 138, 187, 183,

156, 180, 170 and 194 in breast milk. The presence of bias in the use of this technique is appreciated. Other approaches such as the use of designation of non-detected samples as zero values was not used because the values are called non-ignorable data that may also result bias if the samples are simply excluded.

Alternative imputation analysis approaches may provide more accurate methods for pregnancy related PCBs but will need to be confirmed in comparison to other studies [16].

The lipid content of serum was calculated by the sum of cholesterol triglycerides and phospholipids. These analytes were measured by enzymatic methods using a chemistry analyzer. The lipid content of milk was measured by a gravimetric method.

Statistical analysis

The data from the clinical charts, analysis of chemical results and the environmental questionnaires were assembled in SPSS. Only normally distributed data were evaluated using parametric analysis (paired t-test and Pearson Correlation). Parametric analysis was undertaken wherever possible. The individual analytes were not normally distributed and were normalized by logarithmic function and adjusted to account for non-detection of samples. All PCB concentrations were highly skewed. Samples were corrected for non-detection ($0.5 \times$ level of quantification), lipid adjusted and log transformed for a normal distribution. Because the relationships of maternal blood to cord blood are key in this study and the fetal blood is lower in lipid content, PCBs levels were evaluated as ng/ml and as ng/g lipid. Specific analysis was done on PCB 153 because of the relatively high concentrations observed. Samples also were aggregated by measuring Σ PCBs as well as Σ PCBs in Group 2 and Group 3 of those proposed by Wolff et al. [17]. For this study, Group 2 included PCBs 74, 118, 156,

138 and 170. Group 3 included PCBs 99, 153, 180 and 183. Data concerning the concentrations of HCB and DDE were treated in a similar fashion for non-detection and normalization.

To explore changes in the concentrations in pregnancy, paired t-tests were undertaken between all possible groupings of samples on the normalized data. Although there were sufficient pairings to evaluate differences in the pair groups, it is important to note these comparisons do not exactly reflect the same cohort over the four periods of study because of missing data. In order to evaluate the correlation of the results, and bearing in mind the desire to study the maternal blood during pregnancy and at birth as a potential marker of fetal exposure, the non-lipid adjusted samples were normalized for comparison purposes. The non-detection samples were adjusted to reflect 0.5 the level of quantification. To evaluate the potential association of exposures to spontaneous abortion, the concentrations were assessed in relation to clinical history by one way analysis of variance

Results

Demographics

The subjects were all from the Calgary Health Region, located in southern Alberta, Canada. A total of 322 subjects were entered into the study. From this group, 308 provided samples of amniotic fluid and the balance decided not to have the amniocentesis undertaken. There were 209 blood samples collected during the second trimester around the time of the amniocentesis. There were 105 blood samples analyzed from the mothers at the time of giving birth. There were 97 samples of cord blood and 47 samples of breast milk collected.

There were 203 pairs of amniotic fluid and maternal blood during the time of amniocentesis and 85 pairs of samples that were obtained from maternal blood at birth

and cord samples. There were 23 subjects in which all samples were collected in all tissues compartments.

The demographic and reproductive histories of the subjects are presented in Table 1. Consistent with the inclusion criteria, the age of the subjects was 39.0+/- 0.1 (Mean+/- SEM). During the previous year, 15.1% of the subjects identified themselves as smokers and 8% continued to smoke during the pregnancy. Most of these smokers reported smoking less than 20 cigarettes per day (Table1).

A specific diet was reported by 66 subjects including weight loss (13.4%), sodium free (3.4%), fat free (8.4%), diabetic (0.4%), weight gain(0.4%) and vegetarian (1.7%). Eighty five individuals reported a significant weight loss on the preceding year and the average amount was reported to be 7.3 pounds.

Prior medical history was significant for the regular use of prescription medication (27%), Anesthetic exposure(18%), and infertility (12%). There was an infrequent rate of prior cancer (2.1%), skin disease (2.9 %) and liver disease (1.7%).

The most common chemical exposure in the previous year was to paint (50.5%) and solvents (32.8%). There was a reported exposure of this cohort to pesticides in 22%, herbicides 22%, fungicides 5.9% and dry cleaning chemicals 6.5%.

Of the cohort, there were only 36 primigravidas. Sixteen percent reported one induced abortion and 6% had had 2 or more. There were 21% of the subjects with one, 6.3% with two and 6% with three or more spontaneous abortions. Two percent reported an ectopic pregnancy.

Of the pregnancies under review, there were 291 births, of which there was a complication rate of pregnancy of pregnancy induced hypertension in 8.2%, premature labor in 3.4%, gestational diabetes in 5.2% a birth weight less than 2500 g in 4.8% and a Caesarian section rate of 27%.

The sex ratio of the cohort of infants was 1.13 (male/female). Karyotype indicated two cases of Klinefelter syndrome and 2 cases of trisomy that proceeded to delivery. Other anomalies resulted in pregnancy termination.

Amniotic Fluid

None of the PCBs or pesticides were measurable in the amniotic fluid.

Total PCBs and Pesticides

The concentrations of PCB 153, Σ PCBs, Group 2 and Group 3 PCBs and the pesticides DDE and HCB (lipid adjusted) are presented in Table 2 A. For Σ PCB samples a paired t-test of normalized data demonstrated significant differences between all groups. Notably the samples taken during pregnancy were significantly higher than those from mothers at birth ($t=3.6$, $p<0.001$) and were significantly lower than the cord blood ($t=-2.14$, $p<0.035$). At birth Σ PCB samples were also lower than the cord blood Σ PCBs ($t=-5.59$, $p<0.001$). Σ PCBs from maternal blood during pregnancy, at birth and cord blood were all found to be significantly higher than breast milk Σ PCBs ($p<0.001$). In relation to the specific congener PCB 153, the concentrations in cord blood were not different from maternal blood during pregnancy and at birth although they were all significantly higher than breast milk.

This increase in the concentrations of PCBs in the cord blood samples was potentially a consequence of the relatively low concentrations of lipid in the fetal circulation. The levels of non-lipid adjusted levels of PCBs are shown in Table 2 B. Paired t-tests indicated a significant reduction in the PCBs in cord blood compared to maternal blood during pregnancy ($t= 2.905$, $p<0.01$) and at birth ($t=19.0$, $p<0.001$).

The lipid concentrations of the tissue compartments were as follows (mean +/- S dev.): maternal blood during pregnancy - 0.71+/-0.13, at birth - 0.74+/-0.16, cord blood- 0.21+/-0.06 and breast milk - 2.83+/-1.33 g/100 ml plasma.

Groups 2 and 3

Among lipid adjusted samples, Group 2 PCB cord blood samples were higher than maternal blood during pregnancy (T=-10.9, p<0.01) and at birth (T=-11.02, p<0.01) (Table 2 A). All samples (maternal blood during pregnancy, at birth and cord blood) were found to be higher than the concentrations found in breast milk. In regard to Group 3 PCBs, the cord blood samples were found to be higher than samples taken of mothers during pregnancy (T=-2.05, p<0.05). Samples of blood during pregnancy, at birth and in cord blood were higher than breast milk (during pregnancy T=7.87, p<0.01; at birth, T=5.51, p<0.01 and cord blood, T=4.93, p<0.01).

The pattern of PCBs among non-lipid adjusted groups is shown in Table 2 B. There was no difference in the maternal samples during pregnancy compared to the cord blood samples. There were significantly higher levels of both Groups in maternal blood during pregnancy and at birth compared to cord blood, consistent with the Σ PCB level pattern (p<0.001)

The concentrations of pesticides are presented in Table 2. There were no differences in lipid adjusted HCB concentrations of maternal blood during pregnancy and at birth when compared to cord blood (n.s.) There were significantly lower concentrations of lipid adjusted HCB in breast milk when compared to all other groups (p<0.001). The same pattern of result was observed for lipid adjusted DDE samples.

Without lipid adjustment there was a significant difference in HCB in maternal blood during pregnancy and at birth compared to cord blood ($p < 0.001$) (Table 2). HCB during pregnancy and at birth were significantly less than breast milk samples without lipid adjustment ($p < 0.001$). The same pattern was observed for non-lipid adjusted DDE samples.

PCB Correlations

The relationships of the Σ PCBs are closely correlated in all of the tissues as indicated in Table 3. There are several notable relationships with very high correlation coefficients. Non-lipid adjusted, normalized Σ PCBs during pregnancy were found to highly correlate with levels at birth ($r = 0.770$, $p < 0.01$) and in cord blood ($r = 0.498$, $p < 0.01$) but not with breast milk (n.s.). Σ PCBs taken at birth also correlated well with cord blood ($p < 0.001$) but not with breast milk PCBs.

There were significant correlations between Group 2 and 3 PCBs (Table 4). The most significant correlations were between groups but within tissue compartments. Group 2 PCBs of maternal blood during pregnancy correlated most strongly with Group 3 PCBs of maternal blood during pregnancy ($r = 0.755$, $p < 0.01$) but also strongly correlated with Group 2 PCBs at birth ($r = 0.628$, $p < 0.01$) and Group 3 PCBs at birth ($r = 0.717$, $p < 0.001$); Group 3 PCBs of maternal blood during pregnancy also correlated with Group 2 and 3 at birth and Group 2 and 3 cord blood (all $p < 0.001$). The Group 2 PCBs at birth correlated well with Group 2 and 3 cord blood cord ($r = 0.603$, $p < 0.001$ and $r = 0.783$, $p < 0.001$ respectively); Group 3 PCBs at birth also correlated with Group 2 and 3 cord blood (all $p < 0.001$). There was no correlation between breast milk and the other values.

To explore more rigorously the relationships of PCBs between compartments PCB 153 and PCB 180 were evaluated among all subjects with completed collection of maternal and fetal blood samples. These PCBs were selected because of the relatively high rate of detection of analytes. Cord blood PCB 153 was significantly correlated with maternal blood during pregnancy ($r=0.691$, $p<0.001$) and maternal blood at birth ($r=0.905$, $p<0.001$). Cord blood PCB 180 was also significantly correlated with maternal blood during pregnancy ($r=0.511$, $p<0.001$) and maternal blood at birth ($r=0.830$, $p<0.001$).

The correlations between the tissue compartments and HCB and DDE are presented in Table 5. Specifically, there were significant correlations for both HCB and DDE between both the samples during pregnancy and at birth when compared to cord blood (all $p<0.001$).

Clinical Variables and Concentrations of PCBs and Pesticides

There were no differences in the concentrations of PCBs or pesticides among women reporting one or two prior spontaneous abortions compared to those individuals who had had no spontaneous abortion. There were no significant differences in the concentrations of PCBs and pesticides among the subjects reporting medical conditions with the exception that those individuals reporting infertility had significantly higher concentrations of HCB. There were no differences in these concentrations among women reporting exposure to herbicides, pesticides, fungicides or insecticides compared to those reporting no such exposure in the previous year. Similarly, there was no significant association of a particular diet (weight loss, diabetic, salt free, low fat) with the measurement of PCB and pesticides in this cohort of subjects. Although there was a dose related increase in the mean concentrations of

Σ PCBs in maternal blood during pregnancy in relation to fish consumption, the relationship was not significant.

Discussion

The results of this study are reassuring and support the observations that indicate levels of PCBs tend to be relatively low in western Canada compared to other studies of previous years among pregnant women in different countries.

Specifically, using PCB 153 as a marker as recommended [18], it would appear the women in Calgary are at the lower range of values among studies that evaluated neurocognitive outcomes of children. The meta analysis of ten studies indicated a range of the 25th to the 75th percentiles of PCB 153 to range from approximately 20 to 800 ng/g lipid [18]. In this study, the arithmetic mean concentrations of PCB 153 were 29.3, 25.7 and 16.7 ng/g lipid in the maternal blood during pregnancy, at birth and cord blood respectively, indicating a relatively low exposure level in relation to the previous reports.

There is evidence that the maternal serum during the second trimester of pregnancy can be considered a biomarker for the exposure of the fetus *in utero* as determined by cord blood levels. It should be noted however that lipid correction increases the cord blood concentrations of Σ PCBs owing to the relatively low concentrations of fetal blood lipid concentrations and a better evaluation of the maternal - fetal gradient is represented as wet weight (ng/ml). When analyzed in this way there appears to be a gradient of approximately 4.5 to 5.5 in favour of the mother. It is not known what significance the relatively high lipid adjusted levels have for the fetus. The solubility of PCBs in the lipid fraction of blood could theoretically be a factor associated in the bioavailability of the chemicals in the brain and one mechanism for increased susceptibility of the fetal neural tissue to PCB-induced

toxicity although this is speculative. Because of the interest in PCB 153 as a marker, it should be noted that the elevation of the cord concentrations was not present in the same manner as for Σ PCBs

The concentrations of this study are consistent with other reports of the concentrations of PCB 153, HCB and DDE in the cord samples. Specifically, there is close agreement with the findings in southern Quebec, Canada and New Bedford, USA for all three chemicals measured on a wet weight basis [19]. Additionally the combination of PCB 138, 153 and 180 in the New Bedford study was approximately twice the concentrations of the same PCBs in the current study (mean, median and standard deviation: 0.84, 0.6, 1.14 ng/ml respectively), and agreed more closely with the Σ PCBs which contained PCBs 74, 118, 138, 153, 187, 183, 156, 180, 170. These differences may reflect a greater exposure in New Bedford than in southern Alberta [19].

The differences in cord concentrations from other reports of higher levels may be a reflection of the declining concentrations in recent years since the chemicals were banned [20, 21, 22, 23]. Decreases in tissue concentrations have been reported in Northern Canada between 1994 and 2001 [24]. The lower concentrations may also reflect the findings of a population study of healthy women attending a genetics counseling service that was unselected for dietary intake of foods containing PCBs [25].

Absence of these chemicals in the amniotic fluid is confirmed in these studies although the findings are not unexpected because of the low lipid concentration, an assessment of the amniocytes may have isolated analytes [26, 27]. In this study the cells were not available owing the need for karyotyping.

There were a number of important findings from this cohort that again indicate the relatively low concentrations did not exercise a measurable biological effect of the prior health of the women. For example, there were no differences in the concentrations of PCBs or pesticides among women reporting prior spontaneous abortion. The sample size may be too small to detect a significant increase. The higher concentrations of HCB during pregnancy among those reporting infertility may simply reflect the lack of prior pregnancy-related loss of body burden and is not interpreted as a causal function of infertility. There were no significant differences in the concentrations of PCBs and pesticides among the subjects reporting current or previous serious medical conditions or exposure to pesticides, fungicides, herbicides or other common chemicals such as dry cleaning fluid in the previous year. Similarly, there was no significant association of diet with the measurement of Σ PCB and pesticides in this cohort of subjects. Although there was an increase in the mean concentrations of Σ PCBs in relation to daily fish consumption, the relationship of diet was not significant.

Conclusions

In summary, it would appear that pregnant subjects from Calgary Alberta are exposed to PCBs and pesticides as are most individuals, and the extent of exposure appears to be low in relation to published studies. There is no evidence of exposure to pesticides or PCBs in the amniotic fluid. Measurement of maternal blood during the second trimester of pregnancy reliably estimates the fetal exposure to PCBs and organochlorines. A maternal-fetal gradient of 4 – 5 : 1 is best observed as wet weight (ng/ml), however the notable increase in cord Σ PCBs on a lipid adjusted method suggest the latter measure may have a greater biological relevance. The evaluation of

subjects who had complete blood sampling indicated that the cord blood PCB 153 and PCB 180 are reliably predicted by a measure of maternal blood during the second trimester of pregnancy.

Competing interests

The authors declare they have no competing interests.

Authors Contributions

JJ was original co-investigator and undertook patient recruitment, clinical data acquisition and data analysis in relation to the analysis of human tissue samples, and drafted the article. SC was principal investigator of the original proposal, undertook sample measurement and analysis and participated in revising the draft for consideration; RH participated in data analysis and revisions of the article; HH participated in data analysis and revisions of the article. All authors read and approved the final manuscript.

List of Abbreviations

The following abbreviations were used in this manuscript:

DPreg:	maternal blood taken during the first trimester of pregnancy
ABrth:	Maternal blood taken at the time of birth
Crd	Blood taken from the cord at birth
BMk:	Breast milk
DDE:	Dichlorodiphenyldichloroethylene
HCb:	Hexachlorobenzene
DDT:	Dichlorodiphenyltrichloroethane

PCBs:	Polychlorinated biphenyls
PCB 70	2,3',4',5-Tetrachlorobiphenyl
PCB 74	2,4,4',5-Tetrachlorobiphenyl
PCB 77	3,3',4,4'-Tetrachlorobiphenyl
PCB 87	2,2'3,4,5'-Pentachlorobiphenyl
PCB 99	2,2',4,4'5-Pentachlorobiphenyl
PCB 101	2,2'4,5,5'-Pentachlorobiphenyl
PCB 105	2,3,3',4,4'-Pentachlorobiphenyl
PCB 118	2,3',4,4',5-Pentachlorobiphenyl
PCB 128	2,2',3,3',4,4'-Hexachlorobiphenyl
PCB 138	2,2',3,4,4',5'-Hexachlorobiphenyl
PCB 151	2,2',3,5,5'6-Hexachlorobiphenyl
PCB 153	2,2',4,4',5,5'-Hexachlorobiphenyl
PCB 156	2,3,3'4,4',5-Hexachlorobiphenyl
PCB 169	3,3'4,4',5,5'-Hexachlorobiphenyl
PCB 170	2,2',3,3',4,4',5-Heptachlorobiphenyl
PCB 180	2,2',3,4,4',5,5'-Heptachlorbiphenyl
PCB 183	2,2'3,4,4'5',6-Heptachlorbiphenyl
PCB 187	2,2',3,4',5,5',6-Heptachlorbiphenyl
PCB 191	2,3,3',4,4',5',6-Heptachlorbiphenyl
PCB 194	2,2',3,3',4,4'5,5'-Octachlorbiphenyl
PCB 205	2,3,3',4,4',5,5',6-Octachlorobiphenyl
PCB 206	2,2',3,3',4,4',5,5',6-Nonachlorbiphenyl
PCB 208	2,2',3,3',4,5,5',6,6'-Nonachlorobiphenyl
PCB 209	Decachlorobiphenyl

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