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Association of polychlorinated biphenyls with hypertension in the 1999–2002 National Health and Nutrition Examination Survey

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ABSTRACT

The association of 11 polychlorinated biphenyls (PCBs) with hypertension was investigated using the National Health and Nutrition Examination Survey (NHANES), 1999–2002. The unweighted number of participants assessed for hypertension ranged from 2074 to 2556 depending on the chemical(s) being analyzed. In unadjusted logistic regressions all 11 PCBs were associated with hypertension. After adjustment for age, gender, race, smoking status, body mass index, exercise, total cholesterol, and family history of coronary heart disease, seven of the 11 PCBs (PCBs 126, 74, 118, 99, 138/158, 170, and 187) were significantly associated with hypertension. The strongest adjusted associations with hypertension were found for dioxin-like PCBs 126 and 118. PCB 126 > 59.1 pg/g lipid adjusted had an odds ratio of 2.45 (95% CI 1.48–4.04) compared to PCB 126 ⩽ 26.1 pg/g lipid adjusted. PCB 118 > 27.5 ng/g lipid adjusted had an odds ratio of 2.30 (95% CI 1.29–4.08) compared to PCB 118 ⩽ 12.5 ng/g lipid adjusted. Moreover, participants with one or more elevated PCBs had an odds ratio of 1.84 (95% CI 1.25–2.70) compared to no PCBs elevated in an adjusted logistic regression. The prevalence of one or more elevated PCBs was 22.76% or 32 million of 142 million persons ⩾20 years old in the non-institutionalized US population. We hypothesize that association of seven PCBs with hypertension indicates elevated PCBs are a risk factor for hypertension. What clinicians can do, given the results of this study, is limited unless the appropriate laboratory methods can be made more widely available for testing patients.

1. Introduction

Polychlorinated biphenyls (PCBs) contain mixtures of chlorinated compounds that were manufactured for use as lubricants and coolants in various electrical components. The production of PCBs has been banned in the United States since 1977 due to concerns about environmental persistence and harmful health effects (Cleverly, 2005). PCBs have been linked to a variety of health effects including cancer, hepatic dysfunction, thyroid disease, and neurological effects (ATSDR, 2000). Environmental persistence, transport by man (Fan et al., 2005), air- (Barber et al., 2004) and water-borne movement (von Stackelberg et al., 2002) are the reason exposure to PCBs still occurs three decades after manufacturing ceased. The main source of exposure to PCBs is from dietary intake of contaminated foods that include fish, meat, and dairy products. PCBs enter the food web, either terrestrial or aquatic, and become more concentrated as they move up the food chain to animals and humans.

Several studies have focused on the association between PCB exposure and hypertension. In particular, studies conducted in the early 1980s near the time of banning PCBs suggested a relationship between PCBs and hypertension, although the relationship is unclear. Kreiss et al. (1981) found an association between serum PCBs and high blood pressure in a community-based study of 458 persons, even after adjusting for age, gender, body mass index (BMI), and social class. Akagi and Okumura (1985) studied 59 patients with PCB poisoning and found no association between blood pressure and serum PCB levels. In a study of 106 individuals, serum PCB levels were associated with high blood pressure in an unadjusted analysis. However, after adjustment for age and smoking the association was no longer significant (Stehr-Green et al., 1986a). Studies with less than 150 participants are apparently too small to find a relationship between PCBs and hypertension.

Persons living near one of three waste sites were more likely to have abnormally elevated serum PCB levels (Stehr-Green et al., 1986b). Studying waste sites with persistent organic pollutants (including PCBs) present, Huang et al. (2006) found higher prevalence of hypertension among persons discharged from hospitals near waste sites with persistent organic pollutants present. In the laboratory, a study of female rats has shown increased systolic blood pressure due to PCB 126 exposure (Lind et al., 2004). Exposure to some PCBs has been shown to cause endothelial dysfunction (Toborek et al., 1995; Hennig et al.,

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2002a, b). Endothelial dysfunction is thought to be an important event in the development of atherosclerosis and is associated with hypertension and other diseases (Meyers and Gokce, 2007).

More recently, Lee et al. (2007) found PCB 118 and PCB 126 were associated with blood pressure $\geqslant$130/85 mmHg in the National Health and Nutrition Examination Survey (NHANES), 1999–2002. Seven other PCBs were not associated with elevated blood pressure. Lee et al. (2007) did not find dose–response relationships for two classes of PCBs: dioxin-like PCBs and non-dioxin-like PCBs. However, their analyses of PCB classes were not restricted to PCBs with significant individual associations. The purpose of our investigation was to determine the association of 11 PCBs with hypertension, the aggregate association of one or more elevated PCBs with hypertension, and the prevalence of elevated PCBs in the adult non-institutionalized US population.

2. Methods

2.1. Sample

Data used for this study were derived from the NHANES, 1999–2002. The NHANES 1999–2002 is a nationally representative sample of the non-institutionalized US population. The NHANES design includes an oversampling of minorities and an ability to make population estimates. More information on the methodology of the NHANES, 1999–2002, including laboratory assessment, can be found at the National Center for Health Statistics (NCHS) website (CDC, 2007). We included participants $\geqslant$20 years old who were part of a one-third, stratified random, subsample of the NHANES, 1999–2002.

2.2. Hypertension

Persons assigned to the hypertension category were defined as those who reported having ever been told by a doctor that they have hypertension or high blood pressure, those on medications for blood pressure (by stating that they are taking medications or on an antihypertensive in the medication inventory part of the exam), and those with systolic blood pressure $\geqslant$140 mmHg or diastolic blood pressure $\geqslant$90 mmHg (average of three measurements). Persons without hypertension were assigned to the normal blood pressure category.

2.3. PCBs

Eleven PCBs were measured in non-fasting serum samples by high-resolution gas chromatography/isotope-dilution high-resolution mass spectrometry. Values are expressed on a lipid adjusted basis (Akins et al., 1989). Serum total cholesterol (TC), non-esterified cholesterol (FC), triglycerides (TG), and phospholipids (PL) were assayed by automated, enzymatic methods and total lipids (TL) were calculated from the equation TL = 1.677*(TC−FC)+FC+TG+PL. The enzymatic 'summation' method and a reference gravimetric method were highly correlated in an evaluation of 30 serum samples ($r^2 = 0.978$). As non-fasting serum samples were used, the quantity of triglycerides could vary based on time since the patient's last meal and the lipid-adjusted PCB inversely affected. The PCBs chosen were detectable at the 95th percentile for both the NHANES, 1999–2000 and the NHANES, 2001–2002 (CDC, 2005), and included both dioxin-like PCBs (coplanar PCBs and mono-ortho-substituted PCBs) and non-dioxin-like PCBs (Table 1). Coplanar PCBs are present in blood in much smaller quantities (by a factor of 1000) than mono-ortho-substituted PCBs or non-dioxin-like PCBs.

Table 1
Polychlorinated biphenyls (PCBs) studied

Coplanar polychlorinated biphenyls
3,3′,4,4′,5,5′-Hexachlorobiphenyl (PCB 169)
3,3′,4,4′,5-Pentachlorobiphenyl (PCB 126)
Mono-ortho-substituted polychlorinated biphenyls
2,4,4′,5-Tetrachlorobiphenyl (PCB 74)
2,3′,4,4′,5-Pentachlorobiphenyl (PCB 118)
2,3,3′,4,4′,5-Hexachlorobiphenyl (PCB 156)
Non-dioxin-like polychlorinated biphenyls
2,2′,4,4′,5-Pentachlorobiphenyl (PCB 99)
2,2′,3,4,4′,5′- and 2,3,3′,4,4′,6-Hexachlorobiphenyl (PCB 138 and 158)
2,2′,4,4′,5,5′-Hexachlorobiphenyl (PCB 153)
2,2′,3,3′,4,4′,5-Heptachlorobiphenyl (PCB 170)
2,2′,3,4,4′,5,5′-Heptachlorobiphenyl (PCB 180)
2,2′,3,4′,5,5′,6-Heptachlorobiphenyl (PCB 187)

2.4. Control variables

Control variables for adjusted logistic regressions were: age, gender, race, smoking status, BMI, exercise, total cholesterol, and family history of coronary heart disease. Race was classified as Non-Hispanic White, Non-Hispanic Black, and Hispanic and other race. Smoking status was categorized as current smoker or current non-smoker. BMI was derived from height and weight measurements (kg/m^2) collected in the NHANES physical examination. Regular versus sedentary physical activity was defined as moderate or vigorous activity in the last 30 days, based on participant self-report. Regular activity was characterized by answers to the questions: (1) "Over the past 30 days, did you do any vigorous activities for at least 10 min that caused heavy sweating, or large increases in breathing or heart rate? Some examples are running, lap swimming, aerobics classes or fast bicycling." (2) "Over the past 30 days did you do moderate activities for at least 10 min that cause only light sweating or a slight to moderate increase in breathing or heart rate? Some examples are brisk walking, bicycling for pleasure, golf, and dancing." If a person had not engaged in vigorous or moderate activity over the past 30 days they were classified as sedentary. Total cholesterol was measured enzymatically in serum in a series of coupled reactions that hydrolyze cholesteryl esters and oxidize the 3-OH group of cholesterol (CDC, 2007). Family history of coronary heart disease in an individual's parents was defined as having a mother or father with myocardial infarction or angina before age 50. BMI and total cholesterol were used as control variables because these chemicals are lipophilic.

2.5. Analysis

Receiver operating characteristic (ROC) curve analyses were used to determine cut-points for the logistic regressions. The ROC curve analyses took into consideration the weights associated with each participant. The lower cut-point had the fewest false positive and false negative values for predicting hypertension. When this value was below the (maximum) limit of detection, we used the (maximum) limit of detection as the lower cut-point. The upper cut-point was the value that produced 95% specificity for predicting hypertension. These cut-points were then used in unadjusted and adjusted logistic regressions to determine the association of individual PCBs and hypertension. For ROC curve analyses, values below the sample-specific limit of detection were set to the limit of detection divided by the square root of 2. For logistic regressions values below the (maximum) limit of detection were assigned to the lowest category. Values above the upper cut-point were assigned to the highest category for logistic regressions.

In addition to testing the association of the 11 PCBs individually, we also tested the association of one or more elevated PCBs with hypertension. PCBs 118, 126, 74, 99, 138/158, 170, and 187 were used in this analysis. No PCBs elevated was compared to one or more PCBs elevated. Demographics were determined for persons with and without one or more elevated PCBs.

We used SUDAAN software, for most analyses, to allow us to make appropriate estimates from the complex sample design used in the NHANES (Research Triangle Institute, 2005). Our analysis incorporated both the stratification and clustering aspects of the sampling design. The proper weighting procedures include adjustments for non-response and poststratification. Since minorities were oversampled and a complex sampling design was employed, sampling weights provided by the NHANES for the persistent organic pollutants subsample were used to compute population estimates based on weighted parameter estimates and standard errors (CDC, 2007). Preliminary ROC curve analyses were performed using MedCalc software taking into consideration the weighting of the data. The unweighted number of participants assessed for hypertension ranged from 2252 to 2556 depending on the chemical being analyzed. When seven chemicals were evaluated together to determine one or more PCBs elevated (the PCBs with significant separate adjusted logistic regressions) the unweighted number of participants was 2074. The proportion of each sample above the upper cut-point ranged from 8.33% to 11.72% depending on the PCB analyzed. For the analysis of one or more elevated PCBs, 77.24% had no PCBs elevated and 22.76% had one or more PCBs elevated.

3. Results

Demographic characteristics of the sample are shown in Table 2. The proportion of persons that have elevated PCBs (as shown in Table 3) varied by age, gender, and race-ethnicity. Older participants were much more likely to have elevated PCBs ($p<0.01$). Women were more likely than men to have elevated PCBs ($p<0.01$), and Non-Hispanic Blacks were more likely to have

Table 2
Demographics of persons having and not having elevated polychlorinated biphenyls (PCBs)[a]

	Normal	Elevated PCBs
Age (yr) (%)		
20–44	93.9	6.1
45–64	70.6	29.4
⩾65	36.1	63.9
Gender (%)		
Male	81.1	18.9
Female	73.7	26.3
Race-ethnicity (%)		
Non-Hispanic White	76.8	23.2
Non-Hispanic Black	68.8	31.2
Hispanic and other race	83.6	16.4
Body mass index (kg/m^2)		
<25	77.55	22.45
25–29.9	77.88	22.12
⩾30	76.07	23.93

[a] Elevated PCBs being defined as PCB 126 > 59.1 pg/g lipid adjusted, PCB 74 > 22.7 ng/g lipid adjusted, PCB 118 > 27.5 ng/g lipid adjusted, PCB 99 > 17.1 ng/g lipid adjusted, PCB 138 and 158 > 66.0 ng/g lipid adjusted, PCB 170 > 27.1 ng/g lipid adjusted, or PCB 187 > 21.7 ng/g lipid adjusted.

elevated PCBs than Non-Hispanic Whites or Hispanics and other race ($p<0.01$). There was little difference in the proportion with elevated PCBs attributable to BMI.

Results of unadjusted and adjusted logistic regressions for 11 PCBs are shown in Table 3. Unadjusted logistic regressions were significant in each case indicating strong unadjusted associations between each PCB tested and hypertension. Adjusted logistic regressions were significant for PCBs 126, 74, 118, 99, 138/158, 170, and 187. The strongest adjusted associations with hypertension were found for dioxin-like PCBs 126 and 118. PCB 126 > 59.1 pg/g lipid adjusted had an odds ratio of 2.45 (95% CI 1.48–4.04) compared to PCB 126 ⩽ 26.1 pg/g lipid adjusted. PCB 118 > 27.5 ng/g lipid adjusted had an odds ratio of 2.30 (95% CI 1.29–4.08) compared to PCB 118 ⩽ 12.5 ng/g lipid adjusted.

One or more elevated PCBs had an adjusted odds ratio of 1.84 (95% CI 1.25–2.70) compared to no PCBs elevated in an adjusted logistic regression. The prevalence of one or more elevated PCB was 22.76% or 32 million of 142 million persons ⩾20 years old in the non-institutionalized US population.

Table 3
Unadjusted and adjusted association of polychlorinated biphenyls and hypertension

	Unadjusted		Adjusted[a]	
	Odds ratio	95% CI	Odds ratio	95% CI
PCB 169 pg/g lipid adjusted				
⩽27.0	1.00	–	1.00	–
27.1–46.4	2.91	2.27–3.75	1.06	0.77–1.46
>46.4	4.24	2.94–6.11	1.31	0.82–2.08
PCB 126 pg/g lipid adjusted				
⩽26.1	1.00	–	1.00	–
26.2–59.1	2.25	1.85–2.74	1.13	0.92–1.39
>59.1	7.90	4.95–12.60	2.45	1.48–4.04
PCB 74 ng/g lipid adjusted				
⩽12.4	1.00	–	1.00	–
12.5–22.7	3.46	2.64–4.52	1.13	0.77–1.66
>22.7	7.62	5.53–10.48	1.65	1.05–2.61
PCB 118 ng/g lipid adjusted				
⩽12.5	1.00	–	1.00	–
12.6–27.5	3.43	2.70–4.35	1.36	0.99–1.89
>27.5	8.67	5.71–13.15	2.30	1.29–4.08
PCB 156 ng/g lipid adjusted				
⩽12.5	1.00	–	1.00	–
12.6–15.4	3.02	1.89–4.83	1.41	0.90–2.20
>15.4	3.48	2.25–5.38	1.31	0.81–2.11
PCB 99 ng/g lipid adjusted				
&⩽12.5	1.00	–	1.00	–
12.6–17.1	2.73	1.81–4.12	1.05	0.71–1.55
>17.1	4.85	3.44–6.85	1.49	1.04–2.15
PCB 138 and 158 ng/g lipid adjusted				
⩽41.1	1.00	–	1.00	–
41.2–66.0	3.77	2.55–5.57	1.34	0.83–2.18
>66.0	5.49	3.76–8.02	1.75	1.12–2.73
PCB 153 ng/g lipid adjusted				
⩽55.6	1.00	–	1.00	–
55.7–96.0	3.15	2.22–4.48	1.03	0.68–1.58
>96.0	5.38	3.59–8.07	1.45	0.90–2.33
PCB 170 ng/g lipid adjusted				
⩽17.2	1.00	–	1.00	–
17.3–27.1	2.83	1.99–4.02	0.89	0.59–1.35
27.1	5.36	3.91–7.34	1.52	1.02–2.26
PCB 180 ng/g lipid adjusted				
⩽28.2	1.00	–	1.00	–
28.3–71.2	3.13	2.47–3.97	0.93	0.65–1.32
71.2	6.90	5.16–9.23	1.38	0.89–2.14
PCB 187 ng/g lipid adjusted				
⩽12.4	1.00	–	1.00	–
12.5–21.7	2.91	2.18–3.89	1.00	0.69–1.46
>21.7	5.72	4.08–8.03	1.56	1.05–2.31

[a] Adjusted for age, gender, race-ethnicity, smoking status, body mass index, exercise, total cholesterol, and family history of heart attack.

4. Discussion

PCB 126 is a coplanar PCB and PCB 118 is a mono-ortho-substituted PCB. Both are dioxin-like, suggesting the strong associations of PCB 126 and PCB 118 with hypertension is due to the dioxin-like character of these PCBs. However, some coplanar and mono-ortho-substituted PCBs were not associated with hypertension, indicating the specific arrangement of chlorines on the molecule is more important than the classification scheme would suggest.

A limitation of our study is that it is a cross-sectional investigation. We cannot determine the cause of hypertension in our sample, though the laboratory animal study by Lind et al. (2004) did show that PCB 126 caused increased blood pressure in rats. A strength of our study is that it is nationally representative. We show that having elevated PCBs is associated with hypertension after controlling for age, gender, race, smoking status, BMI, exercise, total cholesterol, and family history of coronary heart disease. We hypothesize that association of seven PCBs with hypertension indicates elevated PCBs are a risk factor for hypertension. This suggests the importance of cleaning up superfund sites that are known to be highly contaminated with PCBs, and indicates that a great number of people, 32 million, are still exposed to PCBs. Future research could examine if elevated serum PCB levels influence the effectiveness of hypertension medications. What clinicians can do, given the results of this study, is limited unless the appropriate laboratory methods can be made more widely available for testing patients. Appropriate tests need to be developed which are correlated with serum PCB 126 and PCB 118 as measured in this study. Persons 45 years old or older that do not have hypertension could be screened for elevated PCBs. Elevated PCBs could then be considered a possible additional risk factor for hypertension.

In the context of the obesity epidemic which increases the risk of hypertension, the results of our study raise the question as to how we should perceive body fat gain. As repeatedly demonstrated (Chevrier et al., 2000; Imbeault et al., 2002; Pelletier et al., 2003), body weight loss results in an accentuation of the decrease in thyroid hormones, skeletal muscle oxidative enzymes, and energy expenditure, in association with the weight loss-induced increase in PCB blood concentrations. Our study gives a conceptual extension to these observations suggesting that obesity exerts a protective role regarding some body pollutants by increasing their dilution space (thus reducing their blood and tissue concentrations) and consequently by attenuating their detrimental health-related effects. In fact, the proportion of elevated PCBs among those with BMI $\geqslant 30\,kg/m^2$ was not much greater than those with BMI $< 25\,kg/m^2$ (Table 2).

Further research on dietary sources of PCBs and health advisories on PCBs in foods should be pursued. PCB exposure is clearly a long-term problem that must be addressed at several levels.

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