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Polychlorinated naphthalenes in human adipose tissue from New York, USA

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Polychlorinated naphthalenes have been measured in human adipose tissues from the USA for the first time.

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ABSTRACT

Polychlorinated naphthalenes (PCNs) are persistent, bioaccumulative, and toxic contaminants. Prior to this study, the occurrence of PCNs in human adipose tissues from the USA has not been analyzed. Here, we have measured concentrations of PCNs in human adipose tissue samples collected in New York City during 2003–2005. Concentrations of PCNs were in the range of 61–2500 pg/g lipid wt. in males and 21–910 pg/g lipid wt. in females. PCN congeners 52/60 (1,2,3,5,7/1,2,4,6,7) and 66/67 (1,2,3,4,6,7/1,2,3,5,6,7) were predominant, collectively accounting for 66% of the total PCN concentrations. Concentrations of PCNs in human adipose tissues were 2–3 orders of magnitude lower than the previously reported concentrations of polychlorinated biphenyls (PCBs) and polybrominated diphenyl ethers (PBDEs). Concentrations of PCNs were not correlated with PCB concentrations. The contribution of PCNs to dioxin-like toxic equivalents (TEQs) in human adipose tissues was estimated to be <1% of the polychlorinated dibenzo-*p*-dioxin/dibenzofuran (PCDD/F)-TEQs.

1. Introduction

Polychlorinated naphthalenes (PCNs) are ubiquitous environmental contaminants. PCNs are planar molecules, with physical and chemical properties similar to those of polychlorinated biphenyls (PCBs); the two types of compounds share numerous applications (*e.g.*, dielectric fluids, engine-oil additives, cable insulation, and wood preservatives). In addition to commercial formulations (*e.g.*, Halowaxes in the US), PCNs are formed in solid waste combustion processes and chlor-alkali processes, and they are present as byproducts in technical PCB mixtures. PCNs, like PCBs, are persistent, lipophilic, and bioaccumulative in the food web. PCN congeners have been detected in air, water, sediment, and biota from many global locations including the Arctic and the Antarctic (Falandysz, 1998; Hayward, 1998; Helm et al., 2006).

In North America, PCNs have been measured in air, water, and sediments, and in biota such as fish and birds, especially in the Great Lakes region (Helm et al., 2006). Elevated PCN concentrations have been detected in sediments (Helm et al., 2008; Kannan et al., 2001a; Marvin et al., 2002), mussels (Hanari et al., 2004), and fish (Hanari et al., 2004; Helm et al., 2008; Kannan et al., 2000) collected from the Great Lakes region. In addition, selective accumulation of penta- (CN 52/60) and hexa-CN (CN 66/67) congeners has been found in eggs of fish-eating birds from the Great Lakes (Kannan et al., 2001b). High concentrations of PCNs have been reported in fish collected near a former chlor-alkali plant in Georgia, USA (Kannan et al., 1998). Consumption of contaminated fish is considered to be an important route of human exposures to PCNs.

The toxic effects of PCNs closely resemble those of dioxins (for details, see Hayward, 1998). *In vitro* bioassays for aryl hydrocarbon receptor (AhR)-dependent reporter gene activation or enzyme induction have shown dioxin-like responses for several PCN congeners. The relative potencies (REPs) of PCN congeners to 2,3,7,8-tetrachlorinated dibenzo-*p*-dioxin (TCDD) have been reported (Behnisch et al., 2003; Blankenship et al., 2000; Villeneuve et al., 2000). In certain environmental and biological matrices, the contribution of PCNs to dioxin-like total toxic equivalents (TEQs) was greater than the contribution from PCBs or polychlorinated dibenzo-*p*-dioxins/dibenzofurans (PCDD/Fs) (Helm et al., 2002, 2008; Ishaq et al., 2000; Kannan et al., 1998, 2000), while in fish-eating water birds, PCNs contributed 2–3% of the total TEQs (Kannan et al., 2001b).

Information regarding exposure to PCNs in humans is as yet scanty. Although PCNs have been detected in human adipose tissue, blood, and breast milk samples from Sweden (Lundén and Norén, 1998; Weistrand et al., 1997; Weistrand and Norén, 1998), Canada (Williams et al., 1993), and Germany (Witt and Niessen, 2000),

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those investigations were conducted in the late 1980s to the early 1990s, and the PCN isomers identified were few in number. In the USA, only one study has reported PCNs in human milk, collected from Los Angeles in 1987; in that study, individual PCN isomers were not identified due to the lack of authentic standard available at that time (Hayward et al., 1989).

In the present study, concentrations of individual tri- to octa-CN congeners were measured in human adipose tissue samples collected in New York City. Profiles of PCN congeners, gender- and age-dependent accumulation, and the relationship between previously reported concentrations of PCBs and polybrominated diphenyl ethers (PBDEs) (Johnson-Restrepo et al., 2005) were examined. To our knowledge, this is the first study to determine PCN concentrations in human adipose tissue from the USA.

2. Materials and methods

2.1. Sample collection

Institutional Review Board approval was obtained from the New York State Department of Health for the analysis of human samples. Adipose tissue samples were collected from liposuction procedures conducted on patients (male; $n = 14$, female; $n = 29$) in New York City during October 2003 through November 2005. The samples were devoid of personal identifiers. The only demographic identifiers were age (male; 21–51, female; 17–49 years old), gender, ethnicity (Caucasian; $n = 22$, African American; $n = 10$, Hispanic; $n = 9$, Asian; $n = 2$), and date of collection. All of the samples were stored in pre-cleaned glass bottles at −20 °C until analysis.

2.2. Chemical analysis

PCNs in human adipose tissues were analyzed following the method described elsewhere (Kannan et al., 1998), with some modifications. Two grams of each adipose tissue sample were ground with anhydrous sodium sulfate and were Soxhlet-extracted with dichloromethane (DCM) and hexane (3:1) for 16 h. The extract was rotary-evaporated, and an aliquot was taken for the determination of lipid content by gravimetry. $^{13}C_{12}$-labeled PCDD/Fs were spiked into the remaining extract as internal standards. Lipid in the extract was removed by gel permeation chromatography (GPC) packed Bio-Bead SX-3 (Bio-Rad Laboratories, Hercules, CA, USA), and then passed through a multilayer silica (silica, 40% sulfuric acid–silica, silica, 10% $AgNO_3$–silica, and silica) gel column. The column was cleaned with hexane prior to transfer of the sample. PCNs in the sample were eluted with 15% DCM in hexane and then passed through a glass column packed with silica gel-impregnated carbon (Wako Pure Chemical Industries, Osaka, Japan). After non-planar compounds had been eluted with 15% DCM in hexane, PCNs were eluted with toluene. The toluene fraction was rotary-evaporated and concentrated for gas chromatographic (GC) – high-resolution mass spectrometric (HRMS) analysis.

Identification and quantification were performed using a GC (Thermo Trace Ultra)–HRMS (Thermo DFS, Dreieich, Germany) with a resolving power of >10,000.

Table 1
Concentrations (pg/g lipid wt.) of PCNs in human adipose tissues from New York.

	Male ($n = 14$)				Female ($n = 29$)			
	Mean ± SD	Median	(Range)	Detection rate (%)	Mean ± SD	Median	(Range)	Detection rate (%)
Age[a]	33 ± 8	34	(21–51)		32 ± 7	32	(17–49)	
Lipid (%)	69 ± 19	72	(24–95)		71 ± 11	71	(45–98)	
T_3CNs								
1,3,6-/1,3,5- (20/19)	7.1 ± 11	<5.0	(<5.0–37)	42.9	<5.0	<5.0	(<5.0–15)	10.3
1,3,7-/1,4,6- (21/24)	33 ± 49	11	(<5.0–160)	57.1	<5.0	<5.0	(<5.0–35)	20.7
T_4CNs								
1,3,5,7- (42)	5.7 ± 7.3	<5.0	(<5.0–22)	50.0	<5.0	<5.0	(<5.0–34)	27.6
1,2,4,6-/1,2,4,7-/1,2,5,7- (33/34/37)	18 ± 17	14	(<5.0–56)	78.6	7.1 ± 11	<5.0	(<5.0–33)	37.9
1,4,5,7- (47)	5.8 ± 7.6	<5.0	(<5.0–25)	50.0	<5.0	<5.0	<5.0	0
1,2,5,6-/1,3,6,8- (36/45)	21 ± 28	12	(<5.0–110)	85.7	7.2 ± 11	<5.0	(<5.0–38)	48.3
1,2,3,5-/1,3,5,8- (28/43)	26 ± 33	17	(<5.0–120)	71.4	8.7 ± 13	<5.0	(<5.0–52)	44.8
1,2,3,7-/1,2,3,4-/1,2,6,7- (30/27/39)	18 ± 26	11	(<5.0–100)	64.3	<5.0	<5.0	(<5.0–30)	34.5
2,3,6,7-/1,2,4,8- (48/35)	11 ± 19	7.6	(<5.0–74)	64.3	<5.0	<5.0	(<5.0–19)	37.9
1,2,5,8-/1,2,6,8- (38/40)	7.2 ± 9.8	<5.0	(<5.0–33)	50.0	<5.0	<5.0	(<5.0–15)	3.4
1,4,5,8- (46)	16 ± 24	10	(<5.0–92)	64.3	<5.0	<5.0	(<5.0–33)	37.9
P_5CNs								
1,2,3,5,7-/1,2,4,6,7- (52/60)	210 ± 270	120	(38–1100)	100	120 ± 86	89	(11–320)	100
1,2,4,6,8- (61)	5.6 ± 8.1	<5.0	(<5.0–23)	42.9	7.1 ± 12	<5.0	(<5.0–37)	37.9
1,2,3,4,6-/1,2,3,5,6- (50/51)	22 ± 25	17	(<5.0–73)	64.3	13 ± 16	8.6	(<5.0–77)	69.0
1,2,4,5,6- (57)	<5.0	<5.0	(<5.0–32)	28.6	<5.0	<5.0	(<5.0–18)	13.8
1,2,4,7,8- (62)	8.0 ± 17	<5.0	(<5.0–60)	35.7	6.0 ± 10	<5.0	(<5.0–35)	34.5
1,2,3,5,8-/1,2,3,6,8- (53/55)	9.5 ± 18	<5.1	(<5.0–63)	35.7	<5.0	<5.0	(<5.0–46)	20.7
1,2,4,5,8- (59)	<5.0	<5.0	(<5.0–17)	21.4	5.4 ± 9.2	<5.0	(<5.0–34)	37.9
1,2,3,4,5- (49)	10 ± 14	8.3	(<5.0–50)	64.3	5.0 ± 8.0	<5.0	(<5.0–39)	48.3
H_6CNs								
1,2,3,4,6,7-/1,2,3,5,6,7- (66/67)	100 ± 77	76	(23–280)	100	47 ± 32	42	(10–150)	100
1,2,3,5,7,8- (69)	25 ± 41	15	(<5.0–160)	85.7	11 ± 15	5.6	(<5.0–49)	55.2
1,2,4,5,6,8-/1,2,4,5,7,8- (71/72)	11 ± 14	7.2	(<5.0–39)	57.1	<5.0	<5.0	(<5.0–28)	20.7
1,2,3,4,5,6- (63)	14 ± 18	7.8	(<5.0–50)	57.1	<5.0	<5.0	(<5.0–31)	31.0
Σ PCNs	590 ± 620	410	(61–2500)[c], *		270 ± 220	190	(21–910)	
TEQs[b]	0.089 ± 0.071	0.068	(0.019–0.26)[c], **		0.042 ± 0.029	0.035	(0.0082–0.13)	

Concentration of H_7CN and O_8CN congeners was below LOQ in all the samples.
Concentration below LOQ was treated as zero for arithmetic mean and median values.
SD = standard deviation.
Numbers in parentheses represent IUPAC No.
[a] Year.
[b] TEQs were calculated using DR-CALUX-REPs reported by Behnisch et al. (2003).
[c] Concentrations in males were significantly higher than those in females. $^{*}p < 0.05$, $^{**}p < 0.01$.