Levels of Polychlorinated Dibenzo-p-dioxins (PCDD), Polychlorinated Dibenzo furans (PCDF) and Dioxin-Like PCB in Adipose Tissue of Turkish Men

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Introduction

The Stockholm Convention is a global treaty to protect human health and the environment from persistent organic pollutants (POP). The Convention addresses the production, use, import, release of by-products, stockpile management and disposal of an initial 12 POPs, they so called ''Dirty Dozen''. POPs are chemicals that remain intact in the environment for long periods, become widely distributed geographically, accumulate in the fatty tissue of living organisms and are toxic to human and wildlife. Polychlorinated dibenzo-p-dioxins (PCDD), polychlorinated dibenzofurans (PCDF) and polychlorinated biphenyles (PCB) are 3 of the 12 UNEP internationally recognized POP and also they are most important, toxic members of POPs. They are lipophilic unwanted by-products in a variety of industrial and thermal processes and contaminants found in a variety of environmental media, including air, soil, sediment and biota. The most important sources of PCDD/F are incineration of mixed waste in too low temperatures, metal smelting and refining, chlorine bleaching of pulp [1].

With respect to exposure assessment, it has typically been assumed that the vast majority of the general public is exposed to PCDD/F. These are lipophilic compounds with various half-life within human bodies, most of them being as seven to ten years [2] and they are bio-accumulate in human primarily via the diet specifically ingestion of animal origins [3,4]. In human, a wide variety of health effects have been linked to high exposure to dioxins and dioxin-like compounds, including growth retardation of the fetus and infants, developmental defects, reproductive effects and chloracne, hormonal dysfunctions, mood alterations, reduced mental performance, diabetes, changes in white blood cells, dental defects, endometriosis[5,6,7,8]. The effect that has caused the greatest public concern is cancer[8,9] . Dioxin was shown to be rather promoting agent: it promotes the growth and transformation of already initiated cancer cells. It has also been proposed that TCDD will induce active oxygen radicals, which may secondarily cause genetic damage[4,10].

The aim of our study is to evaluate the individual human background PCDD/F and dioxin-like PCB contamination of a sample of the men population living in Ankara, Turkey. There is almost no data about the levels of PCDD/F and dioxin-like PCB in human, environment and wild life in Turkey.

Materials and methods

Subjects

Between June 2003 and September 2005, 45 human adipose tissue samples were taken during surgical operations from the Ankara University, School of Medicine from different donors which had been living in Ankara area for at least 5 years. All subjects participated in the study voluntarily and all were male. Adipose tissue samples were obtained during an appendectomy or sarcoma operation and kept frozen at -80 °C in glass containers until analysis. The age of subjects ranged from 21 to 45 years (mean age 34.8±?6.5). All subjects acknowledged their participation by signing an informed consent form. All the subjects were mixed food consumers.

Extraction and Cleanup

1.5 to 2 g of adipose tissue were ground with Isolute HM-N, spiked with ^{13}C labelled PCDD/F and PCB standards and extracted by use of pressurized liquid extraction (ASE 200, Dionex GmbH, Idstein, Germany)

with n-hexane: acetone 3:1 as extraction solvent. The extract was evaporated to dryness for gravimetrical determination of the lipid content. The residue was resolved in 2 to 3 ml of n-hexane.

Instrumentation

The PCDD/F and PCB analysis was performed with a high resolution mass spectrometer Finnigan MAT 95S (Thermo Electron GmbH, Bremen, Germany) coupled with an Agilent GC 6890 (Agilent Technologies, Palo Alto, CA, USA). Chromatographic separation was achieved by splitless injection (cold injection system CIS4, Gerstel GmbH, Mülheim, Germany) of 1 µl on a Restek Rtx-2330 column with a length of 60 m, ID 0.25 mm, ft 0.1 µm (Restek GmbH, Sulzbach, Germany). The GC oven was programmed as follows: 90°C initial hold for 1.5 min, increase at a rate of 20°C/min to 170°C, hold for 7 min., followed by an increase of 3°C/min to 260°C and a final hold at 260°C for 10 min.
The MS was operated in SIM mode at a resolution of 10000 and the two most intense ions of the molecular ion cluster were monitored for the unlabelled and labelled isomers.

Results and Discussion

Data on exposure to POP, except for PCDD/F and PCB compounds, for people residing in Ankara had been reported already[11]. In our study, human adipose tissue was collected from men living in Ankara, which is an industrialized city in central Turkey, far from the sea and large lakes. Nevertheless, all subjects reported having a mixed diet including meat and fish. Sources of PCDD/F and dioxin-like PCBs for people living in Ankara could be considered the results of careless disposal practices, accidents, or leakage from various industrial facilities and from chemical waste disposal sites. Although limited studies conducted in Turkey have reported the presence of PCB compounds in human, the environment, and foodstuffs, Cok et al. reported, in the first study aiming to investigate PCB in human, that the indicator PCB was present in both milk and adipose tissues of women[11,12]. The levels of PCDD/F and dioxin-like PCB in human in Turkey were also reported by Cok et al. recently[13]. In this study, they reported the levels of PCDD/F and dioxin-like PCB in adipose tissue samples of 23 males, and TEQs were estimated using WHO-TEFs[14].

This is a follow up study of a previously extended study. It has been aimed to obtain more effective and explanatory results about PCDD/F by increasing the number of subject.

Because of the limited data about PCB and PCDD/F contaminations in human in Turkey , the results of the this study is very important to provide baseline data on the concentrations of Dioxin and Furan contaminants in Turkey.

In this study PCDD/F and dioxin-like PCB (non-ortho PCBs -PCB #77, 81,126,169- and mono-ortho PCB -PCB #105,114,118,123,156, 157,167, 189) which had not been analyzed in Turkey, have been studied on the male adipose tissue.
TEQs were estimated using WHO-TEFs (Table 1) [14,15]. The $WHO_{PCDD/F}$-TEQ concentrations ranged between 2.8 and 17.2 pg WHO-TEQ/g fat (4.2 and 31.5 WHO-TEQ/g fat, respectively, including dioxin-like PCB). The mean concentrations of WHO-PCDD/F-TEQ and ΣTEQ (WHO 2005, Human) were 7.1 and 11.1 pg/g on a lipid basis , respectively.

The mean level of the sum of all dioxin-like PCB found in the samples was 9792 pg/g lipid. Mono-ortho-PCB congeners accounted for greater than %99 of the concentrations of dioxin-like PCB in men. The compound found at the highest concentration was PCB 118, followed by the congeners 156 and 105.

When results of studies on human adipose tissue from various countries are considered, determined **$WHO_{PCDD/F}$-TEQ** values were found in this study respectively lower than that of the some other countries.

This study put forward the exposure to PCDD/F and dioxin-like PCB of Turkish population. In order to provide a better understanding of exposure to these compounds studies on wider population is needed and daily exposure levels from different sources should be determined. The support of the results of this study with PCDD and PCDF results will help to evaluate the toxicological implications (for instance, the effects on fertility) on human resulted from toxic POP in various conditions.

Table 1 : Concentrations of PCDD/Fs and dioxin-like PCBs in Turkish male adipose tissue (pg/g lipid)

Isomers	TEF	n	Conc.(pg/g) Lipid Mean±SD	Min	Max	%<DL*
Dioxin Congeners						
2,3,7,8-Tetrachlorobenzo-p-dioxin	1	45	1.3	n.d	5.20	91.3
1,2,3,7,8-Pentachlorodibenzo-p-dioxin	1	45	2.42	n.d	6.1	93.2
1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	0.1	45	1.3	n.d	5.6	68.5
1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	0.1	45	3.5	n.d	8.7	97.3
1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	0.1	45	0.5	n.d	8.5	48.2
1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	0.01	45	5.6	2.6	23.9	100
1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	0.0003	45	37.6	11.7	131.0	100
Furan Congeners						
2,3,7,8-Tetrachlorodibenzofuran	0.1	45	1.06	n.d	3.0	75
1,2,3,7,8-Pentachlorodibenzofuran	0.03	45	0.47	n.d	1.5	75
2,3,4,7,8-Pentachlorodibenzofuran	0.3	45	7.1	2.4	19.0.2	97.3
1,2,3,4,7,8-Hexachlorodibenzofuran	0.1	45	2.5	n.d	7.5	97.3
1,2,3,6,7,8-Hexachlorodibenzofuran	0.1	45	2.4	n.d	6.8	97.3
1,2,3,7,8,9-Hexachlorodibenzofuran	0.1	45	0.1	n.d	0.7	17.4
2,3,4,6,7,8-Hexachlorodibenzofuran	0.1	45	3.3	1.2	8.6	100
1,2,3,4,6,7,8-Heptachlorodibenzofuran	0.01	45	5.3	1.2	47.9	100
1,2,3,4,7,8,9-Heptachlorodibenzofuran	0.01	45	0.4	n.d	0.9	8.8
1,2,3,4,6,7,8,9-Octachlorodibenzofuran	0.0003	45	13.1	0.5	369	100
Non-ortho PCBs:						
3,3',4,4'-Tetrachlorobiphenyl (PCB #77)	0.0001	45	23.1±16.0	4,5	72.5	100
3,4,4',5-Tetrachlorobiphenyl (PCB #81)	0.0003	45	5.26±5.6	n.d	39.4	95.6
3,3',4,4',5-Pentachlorobiphenyl (PCB #126)	0.1	45	24.7±16.4	7.0	39.4	100
3,3',4,4',5,5'-Hexachlorobiphenyl (PCB #169)	0.03	45	27.0±14.5	9.2	91.8	100
Mono-ortho PCBs:						
2,3,3',4,4'-Pentachlorobiphenyl (PCB #105)	0.00003	45	1160±890	294	7270	100
2,3,4,4',5-Pentachlorobiphenyl (PCB #114)	0.00003	45	252.5±156	61.8	1360	100
2,3',4,4',5-Pentachlorobiphenyl (PCB #118)	0.00003	45	4330±3456	802	28182	100
2',3,4,4',5-Pentachlorobiphenyl (PCB #123)	0.00003	45	51.3±55	n.d	381	95.6
2,3,3',4,4',5-Hexachlorobiphenyl (PCB #156)	0.00003	45	2271±1289	321	12095	100
2,3,3',4,4',5'-Hexachlorobiphenyl (PCB #157)	0.00003	45	425±334	81	2446	100
2,3',4,4',5,5'-Hexachlorobiphenyl (PCB #167)	0.00003	45	609±433	99	4303	100
2,3,3',4,4',5,5'-Heptachlorobiphenyl (PCB #189)	0.00003	45	613±196	49.2	1618	100
WHO $_{PCDD/F}$-TEQ			7.1±3.5			
ΣTEQ (WHO 2005, Human)			11.1±5.5			

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