

## Dietary exposure and human body burden of dioxins and dioxin-like PCBs in Norway

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### Introduction

It is well established that food, especially fatty food of animal origin, is the dominating source for intake of polychlorinated dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs) and biphenyls (PCBs) in the general population (1). Dietary intake studies have been performed in many countries either by measuring concentrations of these pollutants in individual food groups or market baskets and combining the results with average food consumption data or by performing duplicate diet analyses (2). The objective of this study was to estimate the average dietary intake of PCDDs/PCDFs and PCBs with dioxin-like effects in the Norwegian population by determining the concentrations of these pollutants in basic foodstuffs and using data from Norwegian consumer surveys (3). The dietary intake was then compared with measured concentrations in blood and breast milk.

### Materials and Methods

#### *Samples*

Pooled samples of the various food items, consisting of 20-25 individual samples of seafood and 10-15 individual samples of other foods, were obtained from various parts of Norway through the local Food Control Authorities and through the Department of Quality Control, Directorate of Fisheries.

#### *Determination of PCDDs/PCDFs*

The extraction and sample clean-up was carried out using a multicolonn chromatography system as described by Smith et al. (4) consisting of two columns filled with potassium silicate/silica gel and an activated carbon (AX-21) column. In brief, solid samples were ground with Na<sub>2</sub>SO<sub>4</sub> or milk fat, extracted as described earlier (5), were placed on top of the first column and passed onto the carbon column by cyclohexane/dichloromethane (1:1, v/v). PCDDs, PCDFs and non-ortho PCBs were eluted from the carbon column in the reversed direction with toluene. The crude fraction was further purified by chromatography using two Pasteur pipettes in series filled with acidic silica and basic alumina, respectively. Quantifications using <sup>13</sup>C-

labeled internal standards were performed by GC/high-resolution MS using an AutoSpec MS (EI, selected ion monitoring, resolution 10 000) as described in detail elsewhere (5,6).

#### *Determination of PCBs*

Non-ortho PCBs were either determined in the same extract as the PCDDs/PCDFs or were in some cases isolated using gel chromatography and HPLC on 2-(1-pyrenyl)-ethyl-dimethyl-modified silica gel (Cosmosil PYE, Nacalai Tesque, Japan) as described by Järnberg et al. (7). Other PCBs were determined using ultrasonic extraction with acetone/n-hexane, clean-up by concentrated sulfuric acid and congener specific analysis by capillary GC-ECD (8).

#### *Toxic equivalency factors*

Levels expressed in 2,3,7,8-TCDD toxic equivalents (TE) were calculated by using the International equivalency factors (I-TEFs) for PCDDs/PCDFs (9) and the WHO-TEFs for PCBs (10).

#### *Estimation of intake*

Data on average food consumption per individual were obtained from the Norwegian consumer survey 1992-94 covering all purchased food items for 4033 households. Concentrations of dioxins and PCBs in composite food items were calculated from the relative contributions of the various fatty ingredients and subtracting non-edible parts of the food items.

### **Results and Discussion**

Average concentrations of PCDDs/PCDFs and PCBs in TE per g fresh weight for the 18 different food items or parts of food items analysed are given in Table 1.

Table 1. Average toxic equivalents for PCDDs/PCDFs, non-ortho PCBs and other dioxin-like PCBs in collected food items (pg/g fresh weight, n.a. = not analysed)

Food item	No. of pools	PCDD/Fs	Non-ortho PCBs	Other PCBs	Total TE
Bovine fat	3	0.33	0.52	0.58	1.4
Porcine fat	3	0.2	0.17	0.54	0.91
Pork liver	3	0.25	n.a.	0.03	0.28
Lamb fat	3	0.31	0.34	0.11	0.75
Egg	3	0.19	0.20	0.66	1.0
Margarine	4	1.6	n.a.	n.a.	1.6
Butter	3	0.26	0.62	0.08	0.96
Whole milk	3	0.01	n.a.	0.05	0.01
Herring	4	1.1	0.77	2.0	3.9
Mackerel	3	0.52	0.97	1.1	2.6
Farmed salmon	3	0.95	2.2	1.4	4.6
Haddock liver	1	7.8	59	7.6	75
Cod	2	0.08	n.a.	n.a.	0.08
Cod liver	3	4.8	40	17	61
Cod roe	1	0.2	1.0	2.5	3.7
Saithe liver	1	3.9	15	9.1	28
Plaice	1	0.18	0.38	2.2	2.8
Crab	2	31	10	11	51
Cod liver oil	4	0.67	0.91	2.3	3.9

Due to high fat content and bioaccumulation of organochlorine compounds in the marine food web, particularly high concentrations were found in fish livers and in crabs. For most food items, the PCBs make the major contribution to the total TE.

In Norway, fish and fish products constitute the largest contribution to the intake of PCDDs/PCDFs and PCBs (Table 2). In most food groups, the PCBs contribute more to the dioxin-related toxicity than the PCDDs/PCDFs.

Table 2. Calculated average intake per week of different food groups given in pg TE. (Concentration of non-detected congeners in food items: Low = 0; High = detection limit)

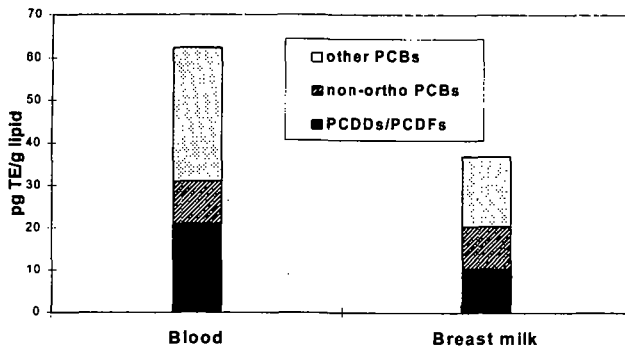
Food group	Intake of PCDD/ PCDF		Intake of PCBs		Total intake	
	Low	High	Low	High	Low	High
Fish	151	163	289	338	437	498
Milk	46	46	122	123	168	169
Meat	35	36	109	116	144	151
Fats	94	316	20	20	113	337
Eggs	25	25	37	116	63	138
Cod liver oil	3	6	28	30	31	36
Total	354	592	605	743	956	1329

The average weekly intake of 960 to 1330 pg TE per week is for an adult person about half of the tolerable weekly intake of 35 pg TE/kg body weight established by a Nordic expert group (11). It must, however, be born in mind that the intake estimation is based on average consumption data. High level intake of food with high concentrations of PCDDs/PCDFs and PCBs, e.g. sea food from contaminated areas, can have a large impact on the intake of toxic equivalents.

Due to the slow elimination in humans, PCDDs/PCDFs and PCBs accumulate in body fat and their concentrations on lipid basis in adipose tissue, breast milk and blood reflect a long term exposure. As part of a WHO program, PCDDs/PCDFs and PCBs have been measured in breast milk from 30 mothers (5). Blood levels of the same pollutants have been determined in men in connection with a study on exposure through crab consumption (6). Total TE in blood of 10 men (age 41-55, not exposed to contaminated crabs) and in breast milk of 30 primiparous mothers (age 17-34) were 62 and 37 pg TE/g lipid, respectively (Figure 1). Dioxin-like PCBs were found to contribute 2 to 3 times more to the total TE in both breast milk and blood than the PCDDs/PCDFs.

Assuming that the whole dose of PCDDs/PCDFs and PCBs is evenly distributed in the body fat compartment, the mean body burdens can be calculated from the percentage of body fat obtained from the body mass index and age according to an empirical equation developed by Deurenberg et al. (12). The mean body burdens were 15.7 and 10.3 ng/kg b.w. for the groups of men and women, respectively. The lower body burden found for the mothers may be explained by the fact that exposure to organochlorine compounds have declined during recent years and that the young women have not reached a steady state with respect to intake and excretion of PCDDs/PCDFs and PCBs.

Figure 1. Concentrations of PCDDs/PCDFs, non-ortho PCBs and other PCBs (in pg TE/g lipid) for blood from Norwegian men and breast milk from primiparous mothers.



If we further assume a steady state and a first order kinetic for excretion of the organochlorine pollutants with a half-life of 5 to 8 years, a weekly intake of TE may be calculated from the concentrations in blood and breast milk and the percentage of body fat (5). This gives a weekly intake of 26-41 pg TE/kg b.w. for the men and 17-27 pg TE/kg b.w. the young women. Given the uncertainty in the assumptions, a good agreement is obtained with the intake of 14-19 pg TE/kg b.w. (Table 2) for a person whose body weight is 70 kg.

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