RESEARCH ARTICLE

Role of dietary patterns for dioxin and PCB exposure

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Dietary patterns were related to intake and blood concentrations of polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/PCDFs), dioxin-like polychlorinated biphenyls (dl-PCBs) and selected non-dioxin-like-PCBs (ndl-PCBs). Intake calculations were based on an extensive food frequency questionnaire and a congener-specific database on concentrations in Norwegian foods. The study (2003) applied a two-step inclusion strategy recruiting representative (n=73) and high consumers (n=111) of seafood and game. Estimated median intakes of sum PCDD/PCDFs and dl-PCBs of the representative and high consumers were 0.78 and 1.25 pg toxic equivalents (TEQ)/kg bw/day, respectively. Estimated median intakes of ndl-PCBs (sum chlorinated biphenyl (CB)-28, 52, 101, 138, 153, 180) were 4.26 and 6.40 ng/kg bw/day. The median blood concentrations of PCDD/PCDFs/dl-PCBs were 28.7 and 35.1 pg TEQ/g lipid, and ndl-PCBs (sum of CB-101, 138, 153 and 180) 252 and 299 ng/g lipid. The Spearman correlations between dietary intake and serum concentration were r=0.34 (p=0.017) for dl-compounds and r=0.37 ($p<0.001$) for ndl-PCBs. Oily fish was the major source of dl-compounds and ndl-PCBs in high and representative consumers. Four dietary patterns were identified by principal component analysis. Two were related to high intakes, one dominated by oily fish ("Ω-3"), the other by fish liver and seagull eggs ("northern coastal"). Only the latter was closely associated with high blood concentrations of dioxins and PCBs.

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1 Introduction

Food is the main source of human exposure to polychlorinated dibenzo-*p*-dioxins/dibenzofurans (PCDD/PCDFs) and polychlorinated biphenyls (PCBs). These persistent environmental contaminants accumulate in the food chain, and may have toxic effects in animals and humans. From a public health point of view, only effects produced at low and background exposure levels are relevant. Such effects are mainly developmental, and include neurodevelopmental, immune, hormonal and metabolic effects. However, the reports of such outcomes are inconsistent [1].

PCDD/PCDFs are commonly referred to as dioxins. They comprise a group of 210 congeners, of which 17 are considered highly toxic. They act *via* activation of the dioxin receptor [2].

The 209 PCB congeners can be divided into two groups, according to their toxicological properties. One group, dioxin-like PCBs (dl-PCBs), consists of 12 congeners that display toxicological properties similar to those of dioxins. The toxicities of the dioxins and dl-PCBs have been ranked relative to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD), the most potent dioxin, and have been assigned

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Abbreviations: bw, body weight; **BMI,** body mass index; **CB,** chlorinated biphenyl; **dl-PCBs,** dioxin-like polychlorinated biphenyls; **FFQ,** food frequency questionnaire; **mo-PCB,** food; **ndl-PCBs,** non-dioxin-like polychlorinated biphenyls; **NFG Study,** Norwegian Fish and Game Study; **no-PCB,** dioxin; **PCA,** principal component analysis; **PCBs,** polychlorinated biphenyls; **PCB_6,** sum of CB-28, 52, 101, 138, 153 and 180; **PCDDs/PCDFs,** polychlorinated dibenzo-*p*-dioxins/furans; **2,3,7,8-TCDD,** 2,3,7,8-tetrachlorodibenzo-*p*-dioxin; **TEF,** 2,3,7,8-TCDD toxicity equivalence factors; **TEQ,** 2,3,7,8-TCDD toxic equivalents; **TWI,** tolerable weekly intake; **WHO,** World Health Organization

2,3,7,8-TCDD toxicity equivalence factors (TEFs) [3, 4]. This allows the total amount of all of the dl-compounds (sum of PCDDs/PCDFs and dl-PCBs) to be expressed as 2,3,7,8-TCDD toxic equivalents (TEQ) (TEQ – the sum of each congener multiplied by its TEF). The remaining 197 PCB congeners are referred to as non-dioxin-like PCBs (ndl-PCBs), and produce their effects *via* different mechanisms [5]. Chlorinated biphenyl (CB)-28, 52, 101, 138, 153 and 180 (PCB_6) together account for ~50% of the ndl-PCBs present in food, and this group is often denoted PCB_6 [5].

Earlier exposure assessments in both Europe and the rest of the world indicate that dietary intake and blood levels of dioxins and dl-PCBs are declining [6–12]. Despite the general decline, the intake of sub-groups may be higher than the tolerable weekly intake (TWI) of 14 pg TEQ/kg body weight (bw)/week set by the EU Scientific Committee on Food [13]. While fish and dairy products are the main contributors to intake of dioxins and PCBs in several European countries, meat is the main source in the United States [14, 15]. For mitigation purposes, it is important to identify which foods or dietary patterns may be connected to the risk of exceeding the TWI. However, most dietary surveys have limitations when it comes to monitoring environmental contaminants, as they usually focus on the general population and regularly eaten food items. The highest concentrations of many environmental contaminants, including dioxins and PCBs, are found in foods, such as seagull eggs, fish liver and fish-liver pâté. Standard diet assessments do not ascertain the intake of these foods in sufficient detail to allow highly precise estimates of exposure. Further, seasonal and regional variations in both food traditions and dioxin and PCB concentrations may contribute to the imprecision of estimated exposure levels of population sub-groups.

The present study is part of the Norwegian Fish and Game Study (NFG study), which was specifically initiated to address these challenges. It was designed to allow in-depth study of dietary habits and patterns. It also enabled us to validate intake estimations through biomonitoring, *e.g.* blood analysis. Identification of dietary patterns by means of principal component analysis (PCA) facilitates the investigation of specific questions about dietary behavior, such as whether food items with relatively high dioxin/PCB concentrations (*e.g.* fish liver, seagull eggs, fish-liver paté used as bread spread, oily fish and shellfish), are consumed together.

The objectives of this study were to: (a) compile a database covering dioxin and PCB levels in Norwegian food, (b) estimate the dietary intake of PCDD/PCDFs and PCBs by a group of Norwegians with a wide range of seafood-consumption levels, (c) measure the blood concentrations of the same congeners and correlate them with the calculated dietary intakes and (d) characterize food groups and patterns by reference to intake and blood concentrations of PCDD/PCDFs and PCBs.

2 Materials and methods

2.1 The NFG study

The aim of the NFG study is to obtain information about the levels of dietary intake of environmental contaminants in the Norwegian population. The main focus is on mercury, cadmium, PCB and dioxins. The objectives of the study are to provide a better description of the population's exposure to these contaminants, to identify any unknown highly exposed groups, and to provide estimates of the exposure of particularly vulnerable groups. The NFG study is a three-stage survey (stages A, B and C). The participants in this study (part C) were recruited from part B of the NFG study [16] (http://www.mattilsynet.no/mattilsynet/multimedia/archive/00016/Fisk_og_vilt__Fish_a_16664a.pdf).

Part A of the NFG study was a national survey of the consumption frequencies relating to specific foods considered to contain potentially high levels of environmental contaminants. Ten thousand people were invited to participate, and the response rate was 60%. One of the main findings of part A was that regional differences are the main explanation for differences in the consumption of fish and game [17]. The study also confirmed that the consumption of food with potentially high levels of contaminants was skewed in the population, and that parts of the Norwegian population might thus be highly exposed to environmental contaminants.

Part B was a regional survey in 27 selected inland and coastal municipalities with good access to hunting and fishing locations. The selection of municipalities (out of 430) was based on hunting statistics and questionnaires distributed to all municipal food control agencies in Norway [16] (http://www.mattilsynet.no/mattilsynet/multimedia/archive/00016/Fisk_og_vilt__Fish_a_16664a.pdf). Due to the ample supply of seafood and/or game in these municipalities, it was expected that consumption of relevant foods would be higher than in the majority of the population. The goal of the instrument in part B was to identify individuals in the population with high intakes of foods with potentially high levels of heavy metals and persistent organic pollutants. In the year 2000, 10 000 persons aged 18–79 living in the 27 municipalities were selected by random draw and invited to participate in the study. The participants were selected using the Norwegian Population Registry, and 55% responded. The questionnaire used in part B was more detailed as regards both frequencies and types of foods than the one used in part A. The participants answered a simple questionnaire that covered the consumption of different freshwater and saltwater fish species, fish liver, crustaceans, seagull eggs and game. None of the participants were living in areas of known contamination by persistent organic pollutants or heavy metals greater than what could be considered a background level [16] (http://www.mattilsynet.no/mattilsynet/multimedia/archive/00016/Fisk_og_vilt__Fish_a_16664a.pdf). One of the main findings from part B supports the finding from part A that

regional differences are the main explanation for consumption of seafood and game [16] (http://www.mattilsynet.no/mattilsynet/multimedia/archive/00016/Fisk_og_vilt__Fish_a_16664a.pdf).

Part C, the present study, was conducted in 2003, and investigated in-depth a sub-population derived from part B. The general aim of part C is to carry out exposure assessments on people who are high consumers of foods that may contain high levels of environmental contaminants. The exposure assessments are based on food consumption, a database of contaminant levels in Norwegian foods, and the analysis of blood and urine samples. The data collected in part C are used to investigate whether it is possible to establish a dose-response relationship between food intake and levels of mercury, cadmium and PCB/dioxins in the blood. The present study adopted a two-stage inclusion strategy to recruit participants with a wide range of dietary exposure to contaminants. Rough estimates of the individual intakes of these contaminants by the part B participants were calculated on the basis of the concentrations of PCBs, dioxins, mercury and cadmium in the relevant foods. Based on estimated high exposures, 434 subjects were invited to participate in part C as high consumers. A reference group of 267 additional subjects (the representative consumers) was randomly selected from the remaining population and invited to participate. Of the 701 subjects invited in total, 211 (30%) gave informed consent, and 195 (28%) completed data collection. The study group thus consisted of 78 representative-consumer participants and 117 high-consumption participants.

Although the representative consumers ($n = 78$) were drawn at random, they constitute a rather low percentage of the total population invited to participate in the study. However, they were comparable with the participants in part B of the study as regards location (coast/inland), average age, gender, bw, body mass index (BMI), smoking habits and total fish consumption [16] (http://www.mattilsynet.no/mattilsynet/multimedia/archive/00016/Fisk_og_vilt__Fish_a_16664a.pdf). The part B participants were in turn comparable with the participants in part A of the study. Accordingly, we have taken the liberty of calling them representative consumers, although the group is small and may be skewed when it comes to other conditions that were not addressed.

2.2 Data collection

Invitations were sent by mail in the spring of 2003. The participants also submitted contact information for their primary physician when indicating their informed consent. The primary physician was then sent information about processing and postal arrangements, vacutainers for blood sampling, tubes for transportation of blood, serum and urine and envelopes for returning the material. The participants received a package containing the food frequency questionnaire (FFQ), a one-page questionnaire covering demographic data (age, smoking, *etc.*), and a urine collection unit. The FFQ and the demographic questionnaire were completed at home and submitted by means of a pre-paid envelope. The participants themselves made appointments with their physicians, who collected the blood samples, redistributed blood and urine samples into the transportation tubes, and posted these. The biological material was processed, redistributed and frozen within 2 h of arrival by mail. The median transit time at ambient temperature between blood collection and storage in the freezer was one day (mean 1.6 days and 75 percentile 2 days, range 1–11 days). The study protocol was approved by the Regional Committee for Medical Research Ethics and the Norwegian Data Inspectorate (id: S-02138).

2.3 Concentrations in food

An extensive database was compiled. It comprised all available concentrations of dioxins and PCB congeners in Norwegian foods from 2000 to 2006, as shown in Supporting Information Table 2. All food sample analyses were sorted by food type, and for each food item a mean concentration was calculated for each congener on the basis of the lower bound values. (Concentrations lower than the limits of detection were set as zero.) This ''Database of measured concentrations'' comprised from 284 PCB_6 to 361 PCDDs and PCDFs analyses, mainly carried out on composite samples. The food analyses were obtained primarily from the Norwegian Food Safety Authority, which makes annual requests for food sample analyses for surveillance purposes. The food concentration data came from four laboratories: the National Institute of Nutrition and Seafood Research, the Norwegian Institute for Water Research, the Norwegian Institute of Public Health, and the Norwegian Veterinary Institute. A report presenting the main results linked to these data is in press (The Norwegian Food Safety Authority, report 2009). The contamination levels of food items for which no analytical data were available were estimated based on similar foods, with adjustments for fat content.

2.4 Dietary exposure

Dietary exposure was assessed using a 12-page semi-quantitative FFQ consisting of 340 questions adapted to Norwegian food traditions. An FFQ is semi-quantitative when only some of the questions include quantification. Questions like ''How often do you eat?'' relate to frequency, while quantitative questions relate to the amount consumed (*e.g.* ''How many seagull eggs *per* year?''). In this questionnaire, quantitative questions were only included in relation to the number of slices of bread, the amount of butter/margarine used on bread, the number of glasses/cups of various drinks, the number of pieces of fruit, and the number of

seagull eggs *per* year [18]. Our FFQ covered consumption over the last 12 months, and was originally developed for use in the Norwegian Mother and Child Cohort Study (MoBa) [18]. The questionnaires were read optically, and food frequencies were converted into consumption (g/day) by multiplying by standard, gender-specific portion sizes. The questionnaire used in this study has been validated in a pregnancy sub-cohort [19]. (In short, the validation demonstrated that, relative to a dietary reference method and several biological markers, the MoBa FFQ produced a realistic estimate of habitual intake, and is a valid tool for ranking pregnant women according to high and low intakes of energy, nutrients and foods.)

Intakes of PCDD/PCDFs, dl-PCBs and ndl-PCBs were estimated by multiplying food consumption by congener levels in food, using the FoodCalc software (http://www.ibt.ku.dk/jesper/foodcalc).

The following congeners were included in the dietary calculations: all 17 of the 2,3,7,8-substituted (PCDD/PCDFs); non-ortho-substituted PCBs (no-PCBs): CB-77, PCB 81, PCB 126 and PCB 169; mono-ortho-substituted PCBs (mo-PCBs): CB-105, 114, 118, 123, 156, 157, 167 and 189; and ndl-PCBs: PCB_6. The concentrations of dl-compounds in food and blood, as well as estimated dietary intakes, were all expressed in TEQs using World Health Organization (WHO) TEF_{2005} [4].

2.5 Chemical analyses of blood

Concentrations of ndl-PCBs were determined in 119 of the serum samples. Samples were selected for analysis if the available serum volume exceeded 3 mL. Eighty-five samples from the high-consumption group and 34 samples from the representative-consumption group were analyzed.

PCDD/PCDFs and dl-PCBs analysis was carried out on whole-blood samples from participants already analyzed for ndl-PCBs. The 114 participants who had supplied sufficient blood volume (*i.e.* > 40 mL) were divided by dl-compound-intake quartiles (TEQ quartiles), and 50 samples were drawn from this population, 12–13 from each quartile.

2.5.1 Ndl-PCBs in serum

A detailed description of the analytical procedure is given in the online supplement. In brief, serum samples were extracted by means of solid-phase extraction using a highly cross-linked polystyrene-divinylbenzene polymer [12, 20]. An additional clean-up on sulfuric acid-silica columns was performed. The extracts were analyzed using gas chromatography coupled to mass spectrometry, operated in the electron-capture, negative-ionization mode. ^{13}C-labeled internal standards were used for quantification. The limit of detection, which is based on the lowest level of the calibration curve, was about 3 pg/g serum (~0.6 ng/g lipids) for the ndl-PCBs. The lipids were determined enzymatically at the National Hospital of Norway (Oslo, Norway), and the total lipid content of the samples was calculated pursuant to a previously described method [21].

2.5.2 Determination of PCDD/PCDF and dl-PCBs in whole blood

For the whole-blood samples, all 17 of the 2,3,7,8-substituted PCDDs/PCDFs and the 12 dl-PCBs were determined using a clean-up method and capillary gas chromatography/high-resolution mass spectrometry as described previously [22].

In brief, the method involved the following. Before extraction, ^{13}C-UL-labeled internal standards for all congeners were added to the samples. After spiking, the samples were liquid/liquid-extracted by means of *n*-hexane and *n*-hexane/2-propanol. Lipid determination was performed gravimetrically by weighing the dried fat residue after extraction, prior to determination of PCDDs/PCDFs and PCBs.

Following gravimetric lipid determination, clean-up was done on a multi-column system. Measurement took place by means of high-resolution gas chromatography and high-resolution mass spectrometry with VG-AutoSpec or Thermo-Finnigan DFS, using DB-5 or DB-5MS capillary columns. Two isotope masses were measured for each component. Quantification was performed using the isotope dilution technique. The analytical method for blood was successfully tested in various national and international quality control studies and proficiency tests.

2.6 Statistical analysis

Dietary intakes and serum concentrations of dioxins and PCBs were not normally distributed. Both median and mean values of intake and serum concentrations are presented. Correlations were calculated using Spearman's rank. Differences between population sub-groups and dietary pattern groups were tested two-sided using non-parametric tests: Mann-Whitney/Kruskal-Wallis. All *p*-values below 0.05 were considered statistically significant.

Dietary patterns were obtained using explorative PCA as the extraction method, rotated with varimax with Kaiser Normalization for interpretation purposes. PCA reduces the data, constructing new variables (factors) based on linear combinations of the original data. The factors explains as much of the variation in the original variables as possible. New dietary pattern variables (factor scores) identify any underlying dimensions in the data.

A high factor score for a given pattern indicates high intake of the foods that make up that pattern, while a low factor score indicates low intake of those foods. Individuals are given factor scores for each dietary pattern [23, 24].

The statistical analyses were carried out using SPSS (version 14.0), SPSS Chicago, IL, USA.

3 Results

3.1 Dietary intake and blood concentration

3.1.1 Demographic information

Overall, the group had a mean and median age of 54 and 55 years (range 21–80 years), the majority (56%) had a BMI of <25. The subjects were evenly distributed with regard to sex and whether they lived in a coastal or inland municipality. There were slightly more females ($n = 101$) than males ($n = 83$) in the study group, which was reflected in the distribution of the representative and high-consumer groups. The demographic variables are further described in Supporting Information Table 1. Ten of the 195 participants were excluded from analysis of dietary exposure on the basis of unlikely energy intakes (less than 1000 or more than 4000 kcal/day). This is a normal measure, taken to improve the general quality of dietary data [18]. Further, one subject was excluded due to extreme serum levels of ndl-PCBs (2475 ng/g lipid sum CB-101, 138, 153, 180–a value 9.1 times the inter-quartile range of 251). This left 184 participants for the final dietary exposure analysis. Of these, 73 were representative consumers.

3.1.2 Diet

The skewed distribution of estimated dietary intake of dl-compounds and ndl-PCBs in this population indicates that some participants have high intakes, especially in the group of high consumers (Fig. 1). The median dl-compound intake for all 184 participants was 6.9 (range 1.3–99.7) pg TEQ/kg bw/week (data not shown). The respective contributions to total TEQ of PCDDs, PCDFs, no-PCBs and mo-PCBs were 14, 18, 64 and 4%, for both representative and high consumers. Dl-PCBs contributed 68% of total dl-compound (TEQ) intake. The TWI for dioxins and dl-PCBs at 14 pg TEQ/kg bw/week was exceeded by 10% of the representative consumers and 19% of the high consumers. The representative and high consumers, respectively had estimated median dietary intakes of 5.25 and 8.61 pg TEQ/kg bw/week, equivalent to 0.75 and 1.23 pg TEQ/kg bw/day (Table 1).

PCB_6 intake ranged from 0.8 to 75.8 ng/kg bw/day, with a median of 5.2 ng/kg bw/day. The two consumption groups had PCB_6 medians of 4.3 and 6.4 ng/kg bw/day (Table 1). The most abundant ndl-PCB congener was CB-153 (respectively accounting for 35 and 36% of PCB_6 in the two groups).

3.1.3 Blood

Concentrations of dl-compounds in blood were skewed, with a median of 33.1 pg TEQ/g lipid (range 7.9–134.6 pg TEQ/g lipid). The sum of the ndl-PCBs (CB-101, 138, 153 and 180) had a median of 279 ng/g lipid (range 11–1303). The median concentrations in blood were higher for the high-consumer group (Table 1 and Fig. 1). CB-153 was the most abundant ndl-PCB compound in the blood samples (respectively accounting for 37 and 38% in the two selection groups). The next two most abundant ndl-PCB compounds were CB-180 and CB-138 (Table 1).

3.1.4 Demographic variables

The high consumers had significantly higher dietary intakes and blood concentrations of PCDD/PCDFs, dl-PCBs and

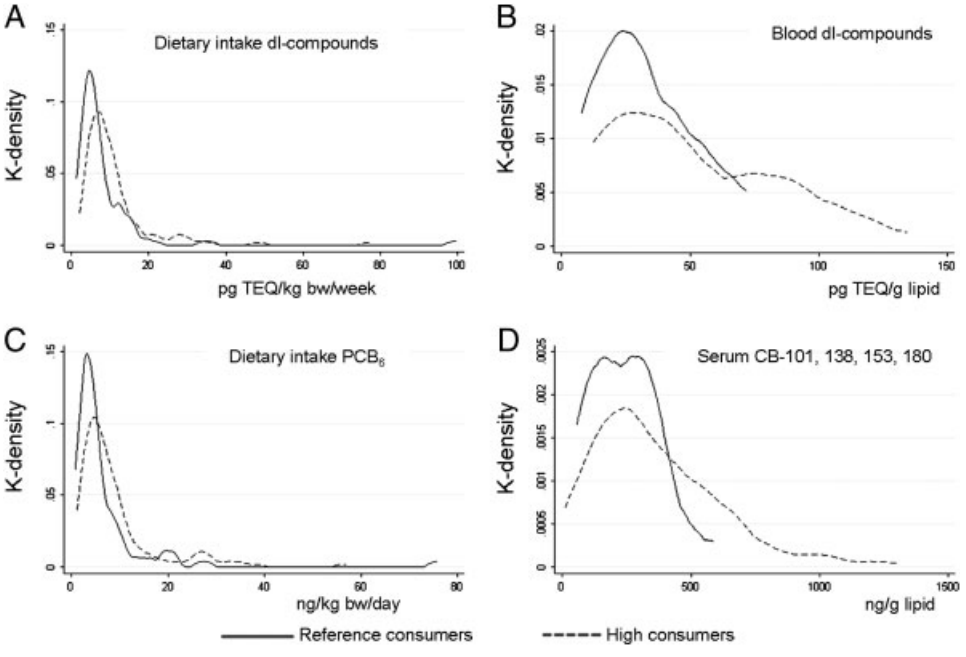

Figure 1. Distribution plot, dietary intakes and blood levels. The *x*-axis shows the dietary intake (A and C) or serum/blood concentration (B and D) of dl-compounds and ndl-compounds. The 3.1.1*y*-axis shows the k-density, which is a measure of frequency.

Table 1. Estimated daily dietary exposure and measured blood concentrations relating to single congeners, sums of TEQ and ndl-PCB, and correlation between blood concentration and dietary intake

PCDD/Fs	Diet[a]				Blood[b]					
	Representative consumers		High consumers		Representative consumers		High consumers		Correlations blood–diet[c]	
	Median	IQR	Median	IQR	Median	IQR	Median	IQR	*r*	*p*
2,3,7,8-TCDD	0.04	0.03–0.06	0.06	0.04–0.08	3.06	1.61–4.22	3.83	2.22–6.31	0.16	0.273
1,2,3,7,8-PeCDD	0.07	0.06–0.09	0.10	0.07–0.14	6.25	4.28–8.07	7.01	4.23–11.0	0.22	0.119
1,2,3,4,7,8-HxCDD[d]	0.00	0.00–0.00	0.00	0.00–0.00	0.10	0.00–0.32	0.25	0.17–0.42	0.09	0.533
1,2,3,6,7,8-HxCDD	0.01	0–0.01	0.01	0.01–0.01	1.48	0.92–1.89	1.65	1.29–2.51	0.29	0.042
1,2,3,7,8,9-HxCDD[d]	0.00	0.00–0.00	0.00	0.00–0.00	0.31	0.23–0.43	0.36	0.23–0.48	0.05	0.753
1,2,3,4,6,7,8-HpCDD[d]	0.00	0.00–0.00	0.00	0.00–0.00	0.22	0.12–0.35	0.33	0.2–0.41	-0.23	0.110
OCDD	0.00	0.00–0.00	0.00	0.00–0.00	0.09	0.07–0.11	0.09	0.07–0.11	-	-
2,3,7,8-TCDF	0.06	0.03–0.08	0.08	0.05–0.13	0.03	0–0.12	0.13	0–0.26	0.29	0.041
1,2,3,7,8-PeCDF[d]	0.00	0.00–0.01	0.00	0.00–0.01	0.00	0.00–0.00	0.00	0.00–0.03	-0.21	0.135
2,3,4,7,8-PeCDF	0.07	0.05–0.09	0.09	0.07–0.13	3.15	1.91–5.22	4.28	2.8–7.9	0.33	0.021
1,2,3,4,7,8-HxCDF	0.01	0.00–0.01	0.01	0.01–0.01	0.50	0.25–0.64	0.55	0.4–0.71	0.26	0.070
1,2,3,6,7,8-HxCDF	0.01	0.01–0.01	0.01	0.01–0.01	0.49	0.28–0.61	0.58	0.39–0.86	0.16	0.263
1,2,3,7,8,9-HxCDF[d]	0.00	0.00–0.00	0.00	0.00–0.00	0.00	0.00–0.00	0.00	0.00–0.00	-0.10	0.506
2,3,4,6,7,8-HxCDF[d]	0.00	0.00–0.00	0.00	0.00–0.01	0.17	0.00–0.24	0.00	0.00–0.3	-0.10	0.507
1,2,3,4,6,7,8-HpCDF[d]	0.00	0.00–0.00	0.00	0.00–0.00	0.05	0.05–0.08	0.05	0.04–0.08	0.12	0.398
1,2,3,4,7,8,9-HpCDF[d]	0.00	0.00–0.00	0.00	0.00–0.00	0.00	0.00–0.00	0.00	0.00–0.00	—	—
OCDF	0.00	0.00–0.00	0.00	0.00–0.00	0.00	0.00–0.00	0.00	0.00–0.00	—	—
dl-PCBs										
CB-77[d]	0.00	0.00–0.00	0.00	0.00–0.00	0.00	0.00–0.00	0.00	0.00–0.00	0.34	0.015
CB-81[d]	0.00	0.00–0.00	0.00	0.00–0.00	0.00	0.00–0.00	0.00	0.00–0.00	0.32	0.023
CB-126	0.44	0.29–0.75	0.71	0.46–1.03	9.23	5.95–13.9	11.7	6.91–28.4	0.48	<0.001
CB-169	0.02	0.01–0.04	0.03	0.02–0.06	2.69	1.72–3.74	4.75	2.61–8.77	0.38	0.007
CB-105	0.01	0–0.01	0.01	0.01–0.02	0.14	0.08–0.26	0.21	0.11–0.43	0.37	0.009
CB-114	0.00	0.00–0.00	0.00	0.00–0.00	0.04	0.02–0.07	0.06	0.04–0.1	0.35	0.013
CB-118	0.02	0.01–0.04	0.03	0.02–0.05	0.92	0.48–1.51	1.20	0.7–2.45	0.28	0.046
CB-123[d]	0.00	0.00–0.00	0.00	0.00–0.00	0.01	0.01–0.02	0.01	0.01–0.03	0.39	0.005
CB-156	0.00	0.00–0.00	0.00	0.00–0.01	0.48	0.27–0.70	0.73	0.41–1.16	0.42	0.002
CB-157	0.00	0.00–0.00	0.00	0.00–0.00	0.10	0.06–0.15	0.16	0.09–0.28	0.39	0.005
CB-167[d]	0.00	0.00–0.00	0.00	0.00–0.00	0.14	0.09–0.26	0.19	0.12–0.35	0.42	0.002
CB-189[d]	0.00	0.00–0.00	0.00	0.00–0.00	0.06	0.03–0.08	0.10	0.05–0.14	0.34	0.015
PCDDs	0.12	0.10–0.18	0.18	0.13–0.24	10.84	8.14–14.4	12.9	8.89–21.3	0.19	0.182
PCDFs	0.15	0.11–0.19	0.20	0.15–0.31	5.00	2.57–7.39	6.27	4.2–9.96	0.28	0.047
PCDD/Fs	0.27	0.21–0.35	0.39	0.28–0.54	15.8	10.7–21.7	19.16	13.1–30.7	0.28	0.051
no-PCBs	0.47	0.3–0.8	0.75	0.48–1.08	11.14	7.92–19.3	15.74	9.52–35.8	0.38	0.007
mo-PCBs	0.03	0.02–0.06	0.05	0.03–0.08	1.88	1.19–3.18	2.45	1.8–4.92	0.37	0.008
dl-PCBs	0.5	0.32–0.86	0.79	0.51–1.15	15.8	9.11–22.6	18.01	11.0–41.1	0.39	0.006
Sum TEQ	0.75	0.52–1.33	1.23	0.82–1.65	28.7	16.7–44.5	35.07	24.8–77.9	0.34	0.017
ndl-PCBs										
CB-28	0.14	0.1–0.21	0.20	0.14–0.29						

Table 1. Continued

PCDD/Fs	Diet[a]				Blood[b]				Correlations blood–diet[c]	
	Representative consumers		High consumers		Representative consumers		High consumers			
	Median	IQR	Median	IQR	Median	IQR	Median	IQR	*r*	*p*
CB-52	0.33	0.2–0.51	0.47	0.31–0.77						
CB-101	0.56	0.33–0.82	0.82	0.51–1.34	4.32	2.86–6.01	3.74	2.43–5.21	-0.05	0.595
CB-138	1.38	0.89–2.14	1.94	1.32–3.04	67.2	37.1–96.1	84.1	52.7–154	0.38	<0.001
CB-153	1.38	0.84–2.41	2.13	1.27–3.25	90.1	47.4–121	108	64.9–188	0.42	<0.001
CB-180	0.37	0.24–0.63	0.55	0.34–0.86	80.5	31.4–106	96.99	56.4–152	0.40	<0.001
Sum of four ndl-PCBs[e]	3.70	2.35–6.22	5.69	3.47–8.57	252	113–329	299	178–539	0.43	<0.001
PCB_6[f]	4.26	2.66–6.91	6.40	3.93–9.73						

Median values and inter-quartile range (IQR) are shown for the representative and high consumers. The correlations are based on diet ($n = 184$) and blood data ($n = 50$ for dl-compounds and $n = 119$ for ndl-PCBs).
a) Dietary intake of dioxin-like compounds shown in pg TEQ/kg bw/day and of ndl-PCBs in ng/kg bw/day.
b) Blood concentrations of dioxins and dl-PCBs in pg TEQ/g lipid, ndl-PCBs in ng/g lipid.
c) Spearman rank $p < 0.05$ is considered statistically significant.
d) Dietary values are > 0, but < 0.01.
e) CB-101, 138, 153 and 180.
f) PCB_6.

ndl-PCBs than the representative consumers (Table 2). Women had lower dietary intakes than men of dl-compounds (6.1 compared with 9.2 pg TEQ/kg bw/week), and ndl-compounds (3.7 compared with 6.2 ng/kg bw/day). This was reflected in the blood concentration measurements for dl-compounds (32.4 compared with 34.9 pg TEQ/g lipid), and ndl-PCBs (240 compared with 329 ng/g lipid). There was an increase in both consumption (TEQ) and serum concentration (TEQ and ndl-PCBs) that correlated with age. BMI did not affect dietary intake or blood concentrations. Both the intakes and blood levels of participants living in coastal municipalities were higher than those of participants living in inland municipalities.

3.1.5 Correlations

The estimated dietary intakes of PCDD/PCDF and PCB congeners and the blood concentrations displayed similar distribution patterns, with the possible exception of 2,3,7,8-TCDF (Fig. 2). The correlations were statistically significant for 18 of the congeners – three PCDD/PCDFs, 12 dl-PCBs and three ndl-PCBs (Table 1). The correlations were also statistically significant for sums of PCDD/PCDFs, dl-PCBs and ndl-PCBs (Table 1).

3.2 Main dietary sources and food patterns

3.2.1 Dietary sources of dioxins and PCBs

The most important sources of dl-compounds and ndl-PCBs for both representative and high consumers were semi-oily and oily fish (Table 3 and Fig. 3). For the representative and high consumers, fish and seafood, respectively contributed 70 and 71% (0.8 and 1.1 pg TEQ/kg bw/day) of total dl-compounds, and 68 and 63% of CB-153 (1.6 and 2.0 ng/kg bw/day). The representative consumers showed lower absolute contributions of dl-compounds and CB-153 from the eleven food groups than the high consumers, although the sources had a similar distribution (Fig. 3). Consumption of food items, such as fish liver and seagull eggs, both featuring high contamination levels, was more frequent in the high consumption group, but hardly affected median dioxin and PCB-intakes. For the 23 participants (17 men and 6 women) who had eaten seagull eggs, this was the major source of no- and mo-PCBs (28 and 60%) and CB-153 (67%), but not PCDD/PCDFs (26 and 4%).

3.2.2 Dietary patterns

In the factor analysis of dietary habits, there were four factors that best represented the data, given the scree plot of eigenvalues, the interpretability of the factor loadings, and an eigenvalue above 1.5 (Fig. 4). Based on quantitative measures and knowledge about local Norwegian dietary

Table 2. Median dietary intake and blood concentrations in population sub-groups

		n	Diet		Blood			
			TEQ[a] pg TEQ/kg bw/day	ndl-PCBs[b] ng/kg bw/day	TEQ[a] pg TEQ/g lipid	n	ndl-PCBs[b] ng/g lipid	n
Participants	All	184	1.0	4.6	33.0	50	279	119
Selection	Repr. cons.	73	0.8	3.7	28.7	16	252	34
	High cons.	111	1.2**	5.7**	35.1**	34	299*	85
Gender	Male	83	1.3	6.2	34.9	20	329	51
	Female	101	0.8**	3.7**	32.4**	30	240*	68
Age group	<40	34	0.8	3.6	17.4	7	108	22
	40–60	76	0.9	4.4	29.5	21	244**	48
	>60	74	1.2*	5.4	67.4*	22	419**	49
BMI group	<25	103	0.9	4.3	30.8	22	264	69
	25–30	62	1.1	5.5	35.4	21	276	39
	>30	18	0.8	3.8	38.4	7	361	11
Municipality	Coastal	85	1.3	5.7	46.9	24	361	55
	Inland	99	0.8**	4.0*	27.3**	26	215**	64

*$p<0.05$ and **$p<0.001$ with non-parametric tests Mann-Whitney/Kruskal-Wallis.
a) Sum of 17 TCDDs/Fs+12 dl-PCBs.
b) PCB_6.

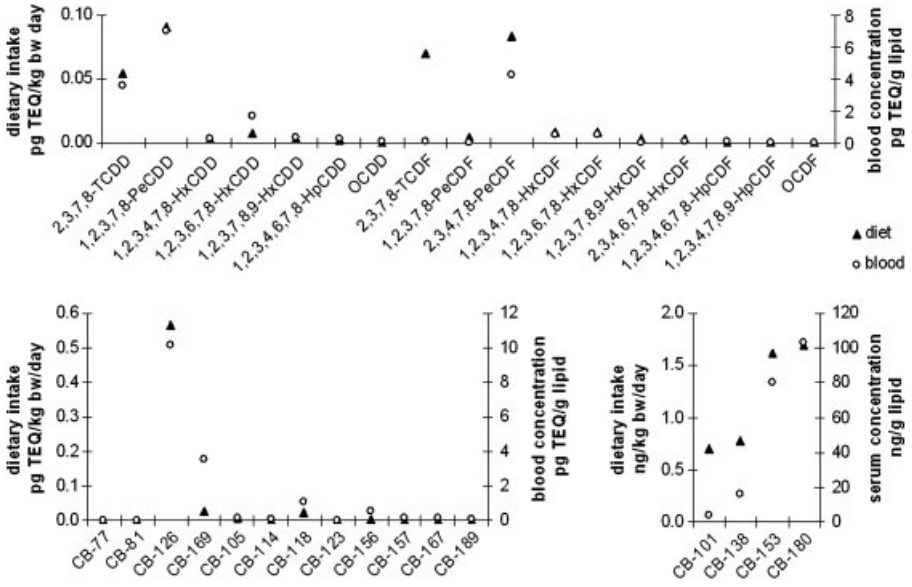

Figure 2. Dietary intake and blood TEQ (n=50) for individual congeners and four ndl-PCBs (n=119) showing the median levels of each congener in the diet and in blood.

preferences, these four factors were defined as: (i) the ''northern coastal'' pattern, characterized by consumption of lean fish, fish liver, roe, seagull eggs but not poultry or cereals/fruits/vegetables (Table 4), (ii) the ''ω-3'' pattern, characterized by consumption of salmon, semi-oily fish, fish-liver paté and eggs, (iii) the ''shellfish'' pattern, characterized by consumption of oily fish except for salmon, crab, shrimp and bivalves and cereals/fruits/vegetables and (iv) the ''meat'' pattern, characterized by consumption of ruminants, offal, pork, vegetable margarine/oils, seagull eggs, dairy products, sugar and miscellaneous and non-lean fish. The ''ω-3'' pattern was positively correlated with the ''shellfish'' pattern ($r = 0.173$, $p = 0.019$), while the ''shellfish'' pattern was negatively correlated with the ''northern coastal'' pattern ($r = -0.214$, $p = 0.004$) (two-sided Spearman's rank).

The pattern scores were divided into quintiles. The pattern in question is always most prominent in the upper quintile (Q5). The Q5s of the different patterns are not mutually exclusive. Rather, they are useful in describing and comparing the patterns in relation to type/amount of food items and blood concentrations of dioxins and PCBs. The

Table 3. Median food consumption for representative (rep) and high (high) consumers (g/day), and the median contribution by the 11 food groups to the dl-compounds and CB-153

Food groups	Intake in grams		PCDDs[a)]		PCDFs[a)]		no-PCB[a)]		mo-PCB[a)]		TEF[a)]		CB-153[b)]	
	Rep median and 95 P	High median and 95 P	Rep	High	Rep	High	Rep	High	Rep	High	Rep	High	Rep	High
Dairy products	447	1,148	387	1261	0.04	0.05	0.02	0.02	0.05	0.05	0.00	0.00	0.11	0.12	0.15	0.18
Eggs	15	58	16	51	0.00	0.00	0.01	0.01	0.01	0.01	0.00	0.00	0.02	0.02	0.05	0.05
Meat	82	134	68	113	0.00	0.01	0.01	0.01	0.02	0.02	0.00	0.00	0.04	0.04	0.07	0.07
Vegetable margarine/oils	27	77	26	65	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01
Lean fish	19	63	28	78	0.00	0.00	0.00	0.00	0.01	0.01	0.00	0.00	0.01	0.01	0.01	0.03
Semi-oily and oily fish	25	74	30	130	0.05	0.07	0.09	0.12	0.25	0.32	0.02	0.02	0.42	0.53	0.75	1.26
Fish liver, roe, fish-liver paté	2	16	3	17	0.00	0.00	0.00	0.00	0.00	0.02	0.00	0.00	0.00	0.02	0.01	0.07
Shellfish	5	18	5	16	0.01	0.01	0.01	0.01	0.00	0.00	0.00	0.00	0.02	0.02	0.01	0.01
Fish oils	0	7	0	7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Seagull eggs	0	1	0	3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Other	2687	4513	2692	4121	0.01	0.02	0.01	0.01	0.06	0.07	0.00	0.00	0.08	0.10	0.02	0.02

a) pg TEQ/kg bw/day.
b) ng/kg bw/day.

food items with the highest factor loadings (>0.7) were fish liver and lean fish in the ''northern coastal'' pattern, salmon and semi-oily fish in the ''ω-3'' pattern, and crab, oily fish other than salmon, shrimp and bivalves in the ''shellfish'' pattern (Table 4).

Dietary intakes of dl-compounds and ndl-PCB differed between the four patterns (Table 5). The highest median intakes were found in the Q5 of the ''northern coastal'' and ''ω-3'' patterns: 1.8 pg TEQ/kg bw in both patterns for dl-compounds and 8.8 and 8.0 ng/kg bw, respectively for ndl-PCBs ($\sum$CB-101, 138, 153, 180). The dietary intake of dl-compounds and ndl-PCBs generally increased in each quintile in all of the patterns except for the ''meat'' pattern, in which intake tended to form a u-shaped curve (data not shown). The median high intake in the ''northern coastal'' pattern was reflected in the blood concentrations of the compounds. However, this was not observed in the ''ω-3'' pattern (79 compared with 30 pg TEQ/g lipid and 542 compared with 275 ng/g lipid for dl-compounds and ndl-PCBs, respectively), as shown in Table 5. Interestingly, in the ''northern coastal'' pattern the estimated dietary intake of dioxins and PCBs was closely related to the serum concentrations of the same compounds in all quintiles (Fig. 5).

Inclusion in the upper quintile of the ''northern coastal'' and ''shellfish'' patterns was more frequent among participants from coastal municipalities ($n = 26$ compared with $n = 11$ and $n = 25$ compared with $n = 12$). The ''ω-3'' pattern was more frequent in inland municipalities ($n = 13$ compared with $n = 24$), while the ''meat'' pattern was about equally distributed between the coastal and inland municipalities ($n = 17$ compared with $n = 20$). More men than women tended to have a high score in the ''ω-3'' (89%), ''meat'' (81%) and ''northern coastal'' (76%) patterns, while the ''shellfish'' pattern was equally distributed between the sexes. The highest median ages in the upper quintiles were found in the ''northern coastal'' and the ''ω-3'' patterns (61 and 60 years), while the lowest age was found in the ''meat'' pattern (50 years).

4 Discussion

This is the first study investigating both estimated dietary exposure of adult Norwegians to PCDD/PCDFs and PCBs and blood concentrations of these. The inclusion approach adopted in the study ensured a wide range of exposures and, as expected, both dietary intakes and blood levels were higher for the high consumers than the representative consumers. The main source of dioxins and PCBs was oily and semi-oily fish. The correlation coefficients between the dietary intakes and blood levels relating to the individual congeners covered a wide range. Low intake of certain congeners was associated with low correlation coefficients. The strongest correlations were found for PCBs (CB-126, -189, -156, -153 and -180, all with $r > 0.40$ and $p < 0.01$). The

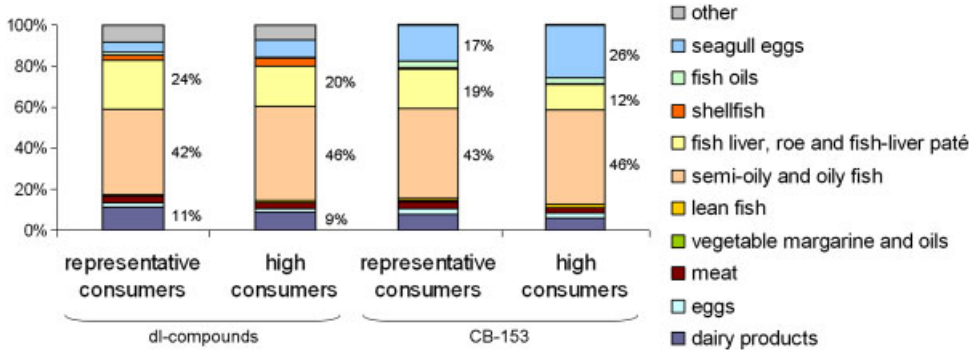

Figure 3. Dietary sources of dl-compounds and CB-153. Mean percentage contributions by the 11 different food groups are shown for the high consumers ($n = 111$) and representative consumers ($n = 73$).

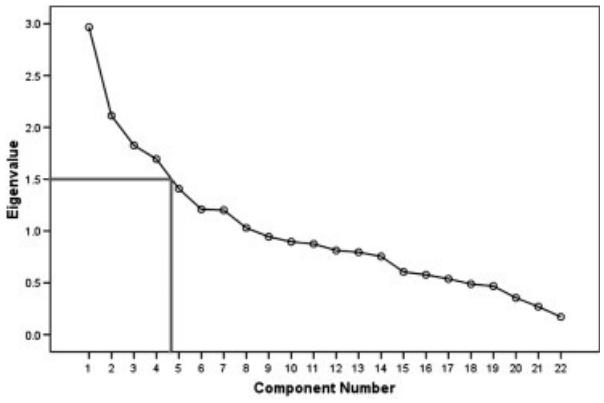

Figure 4. Scree plot showing the dietary patterns and their eigenvalues.

correlation coefficients for the sums of dl-compounds and ndl-PCBs were 0.34 and 0.43,respectively. These correlations all fall into a range that is considered a moderate but acceptable measure of association for use in comparing dietary intake with blood concentration [25]. They are also stronger than the correlations found between dietary intakes and blood concentrations of PBDEs in the same study [26]. In relation to PCA, we identified four characteristic dietary patterns. Interestingly, there were major differences in dietary exposure to and blood concentrations of dioxins and PCBs, connected to differing dietary patterns. Even more striking was the difference in the blood concentrations of the participants in the upper quintiles of the ''northern coastal'' (fish liver, fish roe, lean fish and seagull eggs), and ''ω-3'' (salmon, semi-oily fish, fish-liver patés and other oily fish) patterns, the latter concentration being less than half of the former, despite similar intake estimates. The advantage of factor analysis is the knowledge it provides about how foods are consumed in combination [24]. To the best of our knowledge, this is the first time this method has been used in a dietary assessment of PCDDPCD/Fs and PCBs.

The median dietary intake of dl-compounds of the representative consumers (0.75 pg TEQ/kg bw/day) was a little lower than the intakes reported in recent studies of the general populations of Sweden [27], Finland [28], Catalonia [8], Holland [29], the US [15] and the UK [30] (1.3, 1.5, 1.1, 1.2, 2.2–2.4 (women–men), and 0.9 pg TEQ/kg bw/day, respectively). These reported intake levels, with the possible exception of that relating to the UK, are more in line with the 1.25 pg TEQ/kg bw/day found in the high-consumer group in the present study. However, the intake estimates referred to above were bound to be higher than the estimates calculated in the present study, because they were produced using the 1998 WHO TEF values, which were higher than the 2005 WHO TEF values used in the present study. The intakes in the present study were ~16% higher when the 1998 TEFs were used [31]. Analyses of breast milk samples from primiparus women reflect dietary exposure. Results from the third round of the WHO breast milk study, sampled between 2001 and 2003, indicated that exposure in Norway was in the middle of the concentration range for dl-compounds and ndl-PCBs calculated for the 26 participating countries [32]. Direct comparison of dietary exposure reported in different studies is generally complicated. One major reason is that the levels of dioxins and PCBs in foodstuffs are declining. For example, it has been suggested that a 68% reduction occurred in Spain between 2000 and 2008 [8]. Accordingly, the overall dietary exposure may be over-estimated if food concentrations are used that are a few years old. In our study, we deemed it reasonable to include food samples analyzed between 2000 and 2006, since the FFQs and blood samples were collected in 2003, in the middle of this time span.

The present intake estimate relating to dl-compounds, when calculated using TEF_{2005}, shows that 10% of representative consumers and 19% of high consumers exceeded the EU TWI of 14 pg TEQ/kg bw/week for dl-compounds. When TEF_{1998} is used, the percentages are higher: 16 and 26%, respectively. However, the TWI was exceeded only moderately. The three participants with the highest intakes only exceeded by between three and seven times.

The median serum concentrations were 28.7 and 35.1 pg TEQ/g lipid (TEF_{2005}) for the representative and high-consumer groups, respectively. These Figs. are relatively high in comparison with concentrations detailed in a review of background exposure, in which the range of medians was 16–43.8 pg TEQ/g lipid, calculated using $TEFs_{1998}$ [33]. Our findings may be due to the relatively high-average age (54 years) of the participants in our study. The well-documented bio-accumulative effect associated with higher age is a

Table 4. Rotated factor loadings of the eight dietary factors identified by principal component analysis of the 184 participants

Interpreted dietary pattern	Food name	Factor loading[a)]	Cumulative %[b)]
(1) Northern coastal	Lean fish	0.73	10
	Fish liver	0.72	
	Roe	0.60	
	Seagull eggs	0.46	
	Poultry	−0.46	
	Cereals, vegetables, fruit, berries, nuts and seeds	−0.33	
(2) ω-3	Salmon	0.90	20
	Semi-oily fish	0.72	
	Fish-liver paté	0.60	
	Eggs	0.36	
(3) Shellfish	Oily fish except salmon	0.75	29
	Crab	0.72	
	Shrimp and bivalves	0.71	
	Cereals, vegetables, fruit, berries, nuts and seeds	0.33	
(4) Meat	Ruminants	0.61	36
	Offal	0.58	
	Pork	0.56	
	Vegetable margarine and oils	0.50	
	Seagull eggs	0.46	
	Dairy products	0.44	
	Sugar and miscellaneous	0.37	
	Lean fish	−0.31	

a) Factor loadings are the correlation coefficients (r) between the original variables (food consumption) and the extracted factors.
b) Cumulative explained variance of initial eigenvalues. Food groups are sorted by size of loading coefficient. Food groups with factor loadings between 0.3 and −0.3 are not listed.

Table 5. Median dietary intake and blood concentrations of dl-compounds and ndl-PCBs among participants in the highest and lowest quintiles (Q1 and Q5) of each dietary pattern

	Q	dl-Compounds Diet[a)]	Blood[b)]	ndl-PCBs Diet[c)]	Serum[d)]
Northern coastal	1	0.8	28.7	3.5	178
	5	1.8	78.9	8.8	542
ω-3	1	0.7	30.1	3.0	240
	5	1.8	29.8	8.0	275
Shellfish	1	0.8	34.7	3.8	326
	5	1.4	37.0	7.2	321
Meat	1	1.1	63.5	5.6	359
	5	1.4	24.8	6.4	276

a) pg TEQ/kg bw/day.
b) pq TEQ/g lipid.
c) ng/kg bw/day.
d) ng/g lipid. The ndl-PCBs CB-101, 138, 153 and 180.

consequence of the long half-life of dioxins and PCB [34, 35]. Moreover, the relatively high blood levels observed may also reflect the fact that concentration levels in food were higher several decades ago than today. Since 1991, a 50–60% decrease has been observed in the concentrations of PCDD/PCDFs and PCBs in human milk in both Norway and several other countries [6–12, 33, 36, 37].

The European Commission is considering establishing maximum levels for ndl-PCBs in food based on PCB_6, but very few dietary intake data have been published. [5]. Similar to dl-compounds, the median PCB_6 intake was higher in the high-consumer group than in the representative group (6.4 compared with 4.3 ng/kg bw/day). This is comparable with a study from the Netherlands reporting an average intake of PCB_7 (PCB_6+CB-118) of 5.6 ng/kg bw/day [29]. For the general adult population in Europe, the European Food Safety Authority estimated an average intake of 10–45 ng/kg bw for total ndl-PCB [5]. Assuming that PCB_6 constitutes

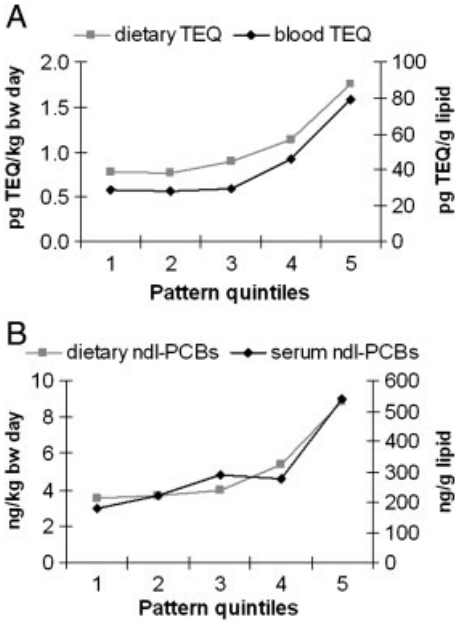

Figure 5. ''Northern coastal'' pattern scores divided into quintiles. The lines show the median dietary intakes (gray) and median blood concentrations in each quintile.

~50% (5–22.5 ng/kg bw/day) of total ndl-PCBs [38], the median intake estimates for our two groups fall into the lower part of this range. No internationally accepted maximum intake level has yet been defined for ndl-PCBs. However, the level of 10 ng/kg bw/day has been suggested as a temporary guideline [38, 39]. Twelve percent of the representative consumers and 23% of the high consumers moderately exceeded this guideline level. There was an overlap between the groups who exceeded the guideline level for ndl-PCBs and TWI for dl-compounds [31].

The median serum concentration of CB-153, which is a major constituent of ndl-PCBs, was higher in the high-consumer group (108.3 ng/g lipid) than in the representative-consumer group (90.1 ng/g lipid). The blood concentrations of CB-153 in the present study are in line with previously reported levels in the cohorts studying neurodevelopment [40].

The statistically significant correlations between the intakes of PCDD/PCDFs, mo-PCBs, no-PCBs, dl-compounds and ndl-PCBs indicate that the dietary sources are similar. The correlation between dietary intake and blood concentrations indicates that the FFQ was a reasonable tool for estimating dietary exposure to contaminants, both in relation to food eaten on a daily basis and in relation to food eaten rarely or on a seasonal basis.

Our results show that seafood is the major source of PCDD/PCDFs and PCBs in the Norwegian diet, contributing ~70% of total intake. This may be caused by relatively high-fish consumption (62 g/day on average for representative consumers) [26]. In a market–basket study from Sweden, where food contamination is comparable with Norway but fish consumption is lower, fish contributed 32% to dl-compounds (TEQ) and 57% to ndl-PCBs [27]. In a market–basket study from Finland, fish contributed as much as 80% to both TEQ and ndl-PCBs [28]. The reason was the high-contamination level in fish from the Baltic Sea, rather than high fish consumption. In a study from Catalonia, Spain in which fish consumption (68 g/day) was comparable with the level in our study, fish and seafood contributed 58% of the sum of PCDDs/PCDFs and dl-PCBs intakes, while meat and meat products accounted for only 6.2% [8]. In a study from the Netherlands, the respective contributions to TEQ and ndl-PCB were 23 and 27% for meat products, 27 and 17% for dairy products and 16 and 26% for fish [29]. Meat is also the major contributor in the USA and Canada [14, 15, 41].

In this study, the ndl-PCB intake followed the dl-compound intake. The results in this study imply that Norwegians with a prominent ''northern coastal'' or ''ω-3'' dietary pattern tend to have a relatively high intake of dl-compounds, although it does not necessarily exceed the TWI. However, the degree of adherence to a ''northern coastal'' pattern was closely associated with the blood concentrations relating to dl-compounds and ndl-PCBs. This was not observed for the ''ω-3'' pattern. This finding may be explained by higher concentrations in fish liver and seagull eggs in the years prior to the present investigation, or by an underestimation of the exposure from these foods. It could also be that the degree of adherence to the ''northern coastal'' pattern is so dominant that other food items are less important with regard to dietary dioxin and PCB exposure. The median ages in the Q5 of both these patterns were about the same (61 and 57 years), and the highest among the four patterns. The difference in serum concentrations is thus probably not explained by this difference in age.

Our results suggest that it may be advisable to emphasize a further reduction in levels of PCDD/PCDFs and PCBs in food wherever possible, *e.g.* for farmed fish. This could be combined with more efficient communication of existing dietary guidelines relating to food items like seagull eggs, fish liver and fish-liver patés, which seem to be of major importance with regard to blood concentrations.

The strengths of this study are an extensive database of contaminant concentrations in food and the use of a comprehensive FFQ, plasma biomarkers and factor analysis to describe dietary intake. Further, the study population represents both men and women, in all age groups, and both inland and coastal municipalities.

However, the limitations of this study should be kept in mind. The concentrations of contaminants recorded in the database vary due to seasonal and regional differences in concentration levels. While most food items were based on sufficient underlying concentration data, some were based on a small number of samples. Other items had to be estimated, a process that introduced further uncertainties, in addition to the ones generally related to food consumption

data [25]. In this study, the number of participants was quite small, and relatively few blood concentrations were measured. Even though the participants were selected to represent high and representative consumers, the latter may not be representative at all, due to the sample size and response rate. However, for variables like fish intake, age, education and smoking habits, the representative consumers corresponded to the general population. The FFQ contained a number of questions on fish consumption, which may have resulted in over-reporting. However, this appears unlikely when the reported levels of fish intake are compared with earlier Norwegian food surveys [42]. The present FFQ has been validated, although the validation related to pregnant women and not this particular population. This may not necessarily be a problem, given that correlations are generally weaker in pregnant than in non-pregnant populations, and validation is thus more complicated in pregnant populations [43].

In conclusion, the present study shows a wide range of dietary intake estimates and blood concentrations of dioxins and PCBs. The median serum levels of ndl-PCBs are in line with other studies, but are relatively high for dl-compounds. A statistically significant correlation was observed between the estimated dietary intake of PCDD/PCDFs and PCBs and their respective serum levels. The main source of PCDD/PCDFs and PCBs was oily and semi-oily fish. This was confirmed when food habits were examined by means of dietary pattern analysis, through the ''ω-3'' pattern. The ''ω-3'' (oily fish) and the ''northern coastal'' (fish liver and seagull eggs) dietary patterns featured the highest dioxin and PCB intakes, but only the ''northern coastal'' pattern was associated with a higher blood concentration.

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