# Polychlorinated Dibenzo-*p*-dioxins, Dibenzofurans, and Biphenyls in Fishermen in Finland

***Hannu Kiviranta,[1] Terttu Vartiainen,[1,2] and Jouko Tuomisto[1,2]***

[1]National Public Health Institute, Department of Environmental Health, Kuopio, Finland; [2]University of Kuopio, Kuopio, Finland

**We measured plasma concentrations of polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs), and polychlorinated biphenyls (PCBs) in fishermen from the Finnish Baltic Sea area and fishermen fishing in inland lakes. The concentrations clearly correlated with the frequency of fish meals and consumption of Baltic fatty fish. The body burden of PCDD/Fs reached the median level of 170 pg/g toxic equivalents (I-TEq) in fat for Baltic Sea fishermen, with the maximum being 420 pg/g. Results for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (range = 4.9–110 pg/g fat) showed that lifetime exposure in a population consuming much Baltic fatty fish can reach the levels of exposures seen in Seveso, Italy, in 1976. After we summed the PCB-TEqs, the total median exposure of Baltic Sea fishermen increased to 290 pg/g TEq in fat, and the highest concentration was 880 pg/g. There was a noted individual variation in fishermen's PCDD/F congener patterns, and it was possible to associate this variation with congener patterns of PCDD/Fs in the fish species that the fisherman reported they had consumed. Linear regression models for ln $WHO_{PCDD/F}$-TEq, ln $WHO_{PCB}$-TEq, and ln total WHO-TEq, from the World Health Organization, explained 48%, 60%, and 53% of the variability, respectively. Age was the only significant predictor of ln $WHO_{PCDD/F}$-TEq, whereas age, amount of fish eaten, and place of residence were significant predictors of ln $WHO_{PCB}$-TEq, and ln total WHO-TEq. *Key words:* Baltic Sea, fish consumption, fisherman, modeling TEq, polychlorinated biphenyls, polychlorinated dibenzofurans, polychlorinated dibenzo-*p*-dioxins. *Environ Health Perspect* 110:355–361 (2002). [Online 7 March 2002] *http://ehpnet1.niehs.nih.gov/docs/2002/110p355-361kiviranta/abstract.html***

Polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs) and polychlorinated biphenyls (PCBs) are fat-soluble pollutants, persistent in the environment, and because many of them are resistant to metabolism, they can bioaccumulate. They are present in human food and are considered potential health hazards.

In Finland in the early 1990s, the contributions of different foodstuffs to the PCDD/F intake were estimated (*1*), and fish and fish products were determined to be responsible for 63% of the daily PCDD/F intake. The impact of fish and fish products on the intake of PCDD/Fs was considerably higher in Finland than in many other countries (*2*). A re-evaluation of the PCDD/F daily intake in Finland was conducted in 2000 (*3*). The contribution of fish and fish products to the daily PCDD/F intake had risen to 80%, mainly because of the decrease in the concentrations of these pollutants in cow milk and eggs.

About 75% of the total fish catch in Finland comes from the Baltic Sea, with Baltic herring representing the major catch (*4*). Fatty fishes such as Baltic herring and salmon have been found to be contaminated with PCDD/Fs and PCBs (*5,6*). PCDD/Fs accumulate in herring at the rate 1 pg/g toxic equivalents (I-TEq) per year, wet weight (ww) basis (*6*), so herring used for human consumption carry a body burden of 5–8 pg/g I-TEq on a ww basis. In nonfatty fishes (e.g., pike, pike perch, perch, bream), the concentrations of PCDD/Fs on a ww basis have been below 1 pg/g I-TEq, and concentrations in nonfatty fishes in the Baltic Sea are slightly higher than in the inland lakes (*7–9*).

Individuals consuming fish frequently may be at risk of increasing their body burden levels of PCDD/Fs and PCBs. The risk is especially high in persons eating Baltic fatty fish. One distinct group that has a high consumption of fish is professional fishermen. In Sweden, study groups have found that Baltic Sea fishermen with high consumption of fish can be exposed to high levels of PCDD/Fs and PCBs (*10–13*). In 1998 there were 2,948 registered professional fishermen in the Baltic Sea area in Finland, of whom 1,071 were full-time fishermen. In the inland areas of Finland, there were 1,192 fishermen, of whom 230 were full-time fishermen (*14*).

In this study, we analyzed blood samples from a sample of Finnish Baltic Sea and inland fishermen for PCDD/Fs and PCBs to relate the body burden levels of these environmental contaminants to fish consumption frequencies and to the fish species consumed. We published preliminary PCDD/F-TEq data from this study previously (*15*), and now we provide the complete congener-specific data for PCDD/Fs and PCBs, along with a more detailed description of the study population. In addition, we used regression analyses to identify significant predictors of the variability of toxic equivalents of PCDD/Fs and PCBs.

## Materials and Methods

***Subject selection and data collection.*** Forty-seven male fishermen who had registered at the Employment and Economic Development Centre for southeast Finland volunteered for the study in 1997. These men were living on the northeastern coast of the Gulf of Finland and in the area to the north along the River Kymijoki. The study group subjects were asked to complete a questionnaire about their intake of foods and about the relevant demographic features of their lifestyle (Table 1).

The study group was classified using two different criteria according to information obtained from the questionnaires: the frequency of fish meals consumed and place of residence. Twenty-six fishermen were designated as exposed fishermen because they ate fish at least twice per week. The other fishermen ($n$ = 21) ate fish meals once or less per week. Two groups were assigned based on a place of residence: the coastal group ($n$ = 25) and the Kuusankoski group ($n$ = 22; Figure 1). The average distances of these groups from the coast of the Gulf of Finland were 6 km and 45 km, respectively. The coastal fishermen can be regarded as sea-area fishermen, and the Kuusankoski subjects as inland fishermen. To obtain more information about their fish consumption, we asked the study subjects to rank their preference for different fish species. Seven fish species or group of fish species were available in this ranking: Baltic herring; cultivated rainbow trout; Baltic salmon; imported salmon; vendace; group consisting of pike, pike perch, perch, and bream; and frozen or canned fish.

All subjects signed informed consents, and Ethical Committee of the National Public Health Institute approved the design of the study.

Address correspondence to H. Kiviranta, National Public Health Institute, Department of Environmental Health, P.O. Box 95, FIN-70701 KUOPIO, Finland. Telephone: + 358 9 19127501. Fax: + 358 9 19127518. E-mail: Hannu.Kiviranta@ktl.fi

We thank P. Tiittanen for statistical counseling and T. Korhonen, K. Mehtonen, S. Nyholm, and T. Rissanen for their valuable technical assistance.

This work was financed by the Academy of Finland, project 52876, and the European Union, contract QLK4-1999-01446.

Received 19 June 2001; accepted 27 September 2001.

***Blood sampling and laboratory analysis.*** After subjects fasted for 12 hr, 250 mL of venous blood was drawn from each subject into centrifuge tubes that did not contain anticoagulants or a serum separator. The samples were allowed to clot for at least 40 min, and then were centrifuged for 20 min. The serums were transferred into glass vials and coded; the codes were broken only after the results had been calculated.

We analyzed 17 toxic PCDD/Fs and 36 PCBs from each serum sample using a method described previously (*16*). Proteins from serum were precipitated with ethyl alcohol and ammonium sulfate. Fat was extracted with hexane, and fat content was determined gravimetrically. The analyzing method involved multiple cleanup steps, and finally high resolution mass spectrometry was used for quantification. All the results were reported on a fat basis, and limits of determination (LOD) for PCDD/Fs, non-*ortho*-PCBs, and other PCBs were 0.5–5, 1.5, and 50 pg/g, respectively, depending on the isomer studied. Recoveries for internal standards were more than 60% for all congeners. We calculated toxic equivalents (TEq) for PCDD/Fs and PCBs using the following toxic equivalency factors (TEF): the North Atlantic Treaty Organization (NATO) factors for PCDD/Fs (I-TEq) (*17*), factors by Ahlborg et al. (*18*) for PCBs (PCB-TEq), and factors recommended by the World Health Organization (WHO) in 1998 for both PCDD/Fs and PCBs ($WHO_{PCDD/F}$-TEq and $WHO_{PCB}$-TEq, respectively) (*19*). In the calculations of toxic equivalents, results below the LOD were considered zero. In addition to concentration data of PCDD/Fs and PCBs, we studied the impact of fish species eaten most frequently by comparing congener profiles of individual fisherman with profiles originating from the fish species consumed most.

Our laboratory has participated in several international quality-control studies for the analysis of PCDD/Fs and PCBs. Matrixes in these studies have included cow milk, human milk, human serum, and fish (*20–22*). The laboratory is an accredited testing laboratory (No. T077) in Finland [European Standard/International Organization for Standardization/International Electrotechnical Commission (EN ISO/IEC) 17025]. The scope of accreditation includes PCDD/Fs, PCBs, and non-*ortho*-PCBs from serum samples.

***Statistical analyses.*** We performed statistical analyses with SPSS software (Windows, release 9.0.1; SPSS Inc., Chicago, IL, USA). We used the Mann-Whitney *U* nonparametric test to test the statistical significance of the differences in concentration results. We tested proportional differences in fish consumption frequencies, preferences in fish species consumed, and differences in use of other food items with either the $\chi^2$ test or the Fisher exact test between classified subgroups.

Linear regression models for dependent variables—$WHO_{PCDD/F}$-TEq, $WHO_{PCB}$-TEq, and sum of these (total WHO-TEq)—were established. Predictor variables in the models were age (year), body mass index (BMI, kg/m$^2$), amount of fish eaten (kg/week), and place of residence. Before the regression analyses were done, all the toxic equivalents were transformed to the natural logarithm (ln) scale. The categorical predictor variable "amount of fish eaten" was

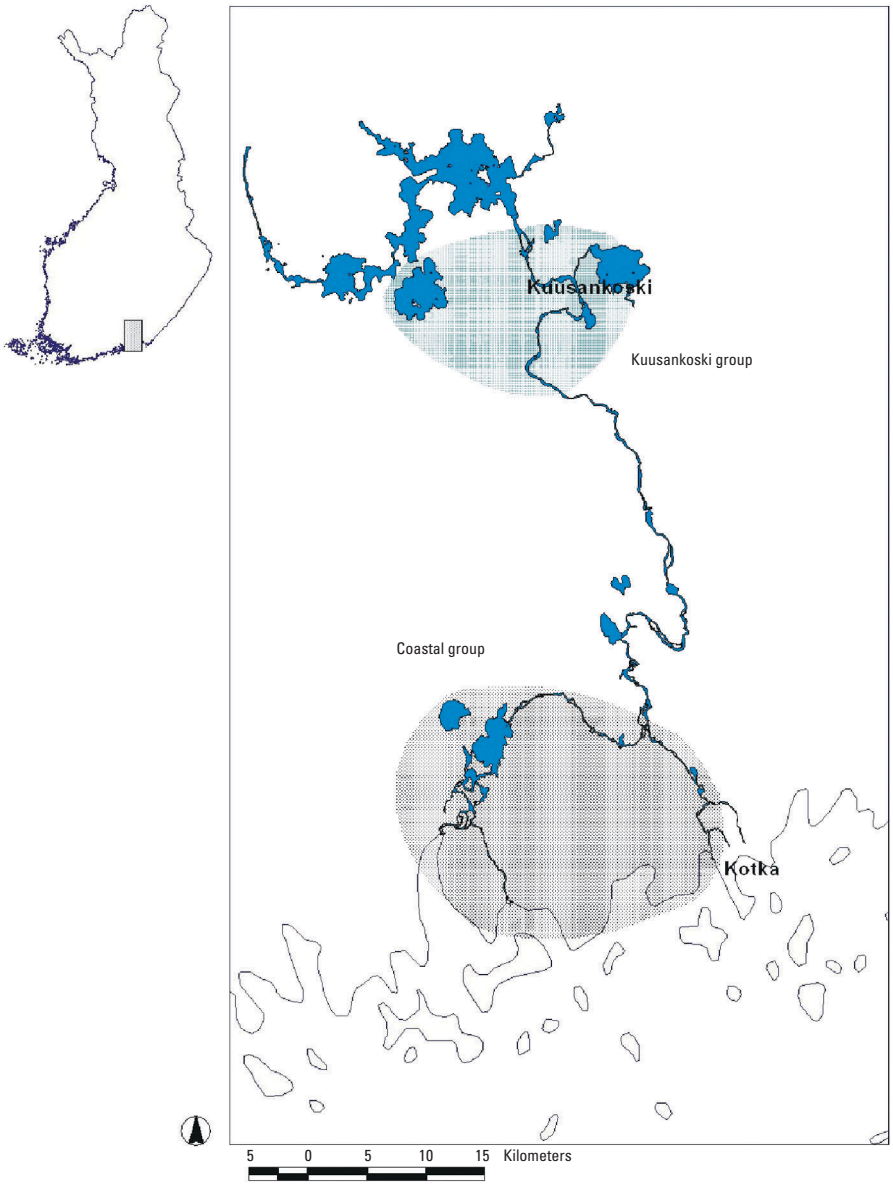


**Figure 1.** Study area showing fishermen subgroups according to place of residence.

**Table 1.** Mean, median, and (range) of age, BMI, and length of time of residence for fishermen and classified fishermen subgroups.

| | | Fish consumption frequency | | Place of residence | |
|---|---|---|---|---|---|
| Characteristics | All subjects *n* = 47 | Exposed fishermen *n* = 26 | Other fishermen *n* = 21 | Coast *n* = 25 | Kuusankoski *n* = 22 |
| Age (years) | 58, 59 (27–77) | 60, 60 (27–77) | 56, 59 (42–73) | 58, 59 (27–76) | 58, 60 (42–77) |
| BMI | 27, 26 (23–36) | 27, 27 (23–35) | 27, 26 (23–36) | 28, 27 (23–36) | 27, 26 (23–33) |
| Tme at present residence (years) | 45, 50 (4–77) | 43, 51 (4–77) | 47, 47 (9–73) | 47, 50 (6–73) | 42, 49 (4–77) |

transformed as a weighted continuous factor, which was also transformed to the natural logarithm scale. In weighting fish amount, the average fish meal portion size, fish consumption frequency, preference in fish species consumption, and average PCDD/F and PCB TEq-concentrations of fish species were used. The predictor variable "place of residence" was used as categorical variable.

## Results

***Demographics and fish consumption.*** The average age of the 47 study subjects was 58 years; in the groups classified by fish consumption frequency and place of residence, average ages were almost identical, and the differences were not statistically significant. Also, BMI (27 kg/m$^2$ for all subjects) and time of residence (45 years for all subjects) were very similar between groups, and the differences were not statistically significant (Table 1).

In the group of exposed fishermen, the subjects ate fish at least twice per week; in the other fishermen group, the frequency of fish consumption was once or less per week. When we compared the fish consumption frequency by place of residence (i.e., the coastal group vs. the Kuusankoski group), the $\chi^2$ test did not reach statistically significant difference, ($p < 0.334$). A slightly larger proportion of subjects in the coastal group (15 of 25) ate fish at least twice a week compared with the Kuusankoski group (11 of 22).

Table 2 summarizes the ranked results of the two most favored fish species or group of fish species in classified subgroups of subjects. In the subgroups created according to fish consumption frequency, the proportions of primary and secondary fishes were not statistically significantly different according to Fisher's exact test. For the coastal and Kuusankoski groups, there were statistically significant differences between proportions of fish species in both primary and secondary fishes ($p < 0.003$ and $p < 0.001$, respectively). In the coastal group, Baltic herring or salmon was the primary fish species being consumed by 10 subjects, but no subjects in the Kuusankoski group chose these species as the primary species. For secondary fish species, vendace was the dominant in the Kuusankoski group (14 subjects), whereas no subjects in the coastal group ranked vendace as their primary or secondary fish. No subjects ranked imported salmon or frozen or canned fish as being within the two most favored fish species.

Consumption frequency patterns of milk, milk products, and meat and current and past smoking patterns were very similar among the classified subgroups and were not statistically significantly different (data not shown).

***Serum levels of PCDD/Fs and PCBs.*** Mean levels, median levels, and ranges of 17 toxic PCDD/Fs and TEqs in all 47 subjects and in classified subgroups are summarized in Table 3. The overall median and mean I-TEq concentrations were 120 and 150 pg/g fat, respectively. The four congeners contributing the most to TEq median (mean) concentrations in fat were in ranked order: 1) 2,3,4,7,8-pentachlorodibenzofuran [2,3,4,7,8-PeCDF; 45.5 (50) pg/g I-TEq];

**Table 2.** Ranking frequencies of the two most favored fish species in subgroups of fishermen.

| | Fish consumption frequency | | Place of residence | |
|---|---|---|---|---|
| Ranking of fish species | Exposed fishermen $n = 26$ | Other fishermen $n = 21$ | Coast $n = 25$ | Kuusankoski $n = 22$ |
| Primary fish (*n*) | | | | |
| Baltic herring | 5 | 4 | 9 | 0 |
| Baltic salmon | 1 | 0 | 1 | 0 |
| Cultivated rainbow trout | 4 | 5 | 5 | 4 |
| Pike, pike perch, perch, bream | 16 | 10 | 10 | 16 |
| Vendace | 0 | 2 | 0 | 2 |
| Secondary fish (*n*) | | | | |
| Baltic herring | 5 | 4 | 6 | 3 |
| Baltic salmon | 2 | 2 | 3 | 1 |
| Cultivated rainbow trout | 5 | 4 | 7 | 2 |
| Pike, pike perch, perch, bream | 6 | 5 | 9 | 2 |
| Vendace | 8 | 6 | 0 | 14 |

**Table 3.** Mean, median, and (range) of PCDD and PCDF congeners and TEqs in blood samples for fishermen according to subgroups.[a]

| | | Fish consumption frequency | | Place of residence | |
|---|---|---|---|---|---|
| Congener | All subjects $n = 47$ | Exposed fishermen $n = 26$ | Other fishermen $n = 21$ | Coast $n = 25$ | Kuusankoski $n = 22$ |
| 2,3,7,8-TCDF | 7.4, 5.6 (ND–30) | 8.8, 7.1 (1.1–30) | 5.6, 4.4 (ND–18) | 8.4, 7.0 (0.57–30) | 6.2, 4.3 (ND–24) |
| 1,2,3,7,8-PeCDF | 3.5, 2.6 (ND–33) | 4.0, 3.0 (ND–33) | 3.0, 2.4 (ND–11) | 2.5, 2.9 (ND–8.7) | 4.8, 2.5 (ND–33) |
| 2,3,4,7,8-PeCDF | 100, 91 (22–280) | 120, 120 (39–280)* | 82, 61 (22–260) | 130, 130 (37–280)** | 71, 57 (22–220) |
| 1,2,3,4,7,8-HxCDF | 22, 17 (5.3–84) | 24, 20 (6.0–84) | 18, 16 (5.3–39) | 24, 21 (8.3–69)** | 19, 15 (5.3–84) |
| 1,2,3,6,7,8-HxCDF | 24, 19 (5.2–100) | 27, 21 (7.1–100) | 19, 15 (5.2–42) | 25, 21 (7.1–53) | 22, 15 (5.2–100) |
| 2,3,4,6,7,8-HxCDF | 7.5, 6.3 (1.1–35) | 8.9, 7.1 (1.1–35)* | 5.7, 4.5 (1.9–14) | 8.0, 6.8 (2.8–21) | 7.0, 5.1 (1.1–35) |
| 1,2,3,7,8,9-HxCDF | 1.2, 0.50 (ND–10) | 1.5, 0.52 (ND–10) | 0.88, 0.36 (ND–4.3) | 1.9, 0.99 (ND–10)** | 0.46, 0.28 (ND–3.9) |
| 1,2,3,4,6,7,8-HpCDF | 75, 43 (11–1,100) | 98, 42 (11–1,100) | 47, 44 (17–92) | 100, 52 (14–1,100) | 47, 33 (11–160) |
| 1,2,3,4,7,8,9-HpCDF | 0.23, ND (ND–5.0) | 0.31, ND (ND–5.0) | 0.12, ND (ND–1.4) | ND, ND** | 0.490, ND (ND–5.0) |
| OCDF | 42, ND (ND–1,900) | 74, ND (ND–1,900) | 1.2, ND (ND–11) | 76.5, ND (ND–1,900)** | 1.8, ND (ND–11) |
| 2,3,7,8-TCDD | 19, 13 (2.7–110) | 25, 19 (4.9–110)* | 11, 10 (2.7–32) | 27, 21 (4.1–110)** | 9.5, 7.3 (2.7–27) |
| 1,2,3,7,8-PeCDD | 62, 53 (9.1–180) | 79, 76 (15–180)* | 42, 34 (9.1–140) | 78, 78 (22–180)** | 44, 34 (9.1–150) |
| 1,2,3,4,7,8-HxCDD | 8.3, 7.6 (ND–31) | 8.8, 7.7 (ND–31) | 7.6, 7.2 (ND–23) | 7.5, 7.0 (ND–23) | 9.2, 7.6 (ND–31) |
| 1,2,3,6,7,8-HxCDD | 300, 240 (46–1,700) | 370, 290 (46–1,700)* | 220, 190 (74–640) | 260, 210 (46–650) | 360, 270 (74–1,700) |
| 1,2,3,7,8,9-HxCDD | 73, 46 (ND–320) | 82, 59 (ND–320) | 62, 36 (12–290) | 87, 53 (ND–320) | 56, 39 (12–160) |
| 1,2,3,4,6,7,8-HpCDD | 120, 110 (21–340) | 120, 120 (21–340) | 120, 110 (43–330) | 140, 110 (44–330) | 110, 95 (21–340) |
| OCDD | 800, 610 (230–2,900) | 790, 780 (230–2,900) | 810, 600 (290–2,600) | 830, 630 (310–2,600) | 770, 600 (230–2,900) |
| Sum of toxic congeners | 1,700, 1,400 (580–5,800) | 1,800, 1,600 (580–5,800) | 1,500, 1,100 (630–4,100) | 1,800, 1,500 (580–4,600) | 1,500, 1,200 (630–5,800) |
| I-TEq | 150, 120 (30–420) | 180, 170 (51–420)* | 110, 87 (30–280) | 180, 170 (62–420)** | 120, 92 (30–350) |
| $WHO_{PCDD/F}$-TEq | 180, 150 (34–500) | 220, 210 (58–500)* | 130, 100 (34– 340) | 220, 210 (75–500)** | 140, 110 (34–420) |

Abbreviations: HpCDD, heptachlorodibenzo-*p*-dioxin; HpCDF, heptachlorodibenzofuran; HxCDD, hexachlorodibenzo-*p*-dioxin; HxCDF, hexachlorodibenzofuran; I-TEq, NATO toxic equivalency factors; ND, below limit of determination; OCDD, octachlorodibenzo-*p*-dioxin; OCDF, octachlorodibenzofuran; PeCDD, pentachlorodibenzo-*p*-dioxin; PeCDF, pentachlorodibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; TCDF, tetrachlorodibenzofuran; $WHO_{PCDD/F}$-TEq, WHO toxic equivalency factors for PCDD/Fs.

[a]Concentrations are given in pg/g fat. *Significantly different compared with the other fishermen group ($p < 0.05$ by Mann-Whitney *U*-test). **Significantly different compared with the Kuusankoski place of residence group ($p < 0.05$ by Mann-Whitney *U*-test).

2) 1,2,3,7,8-pentachlorodibenzo-*p*-dioxin [1,2,3,7,8-PeCDD; 26.5 (31) pg/g I-TEq]; 3) 1,2,3,6,7,8- hexachlorodibenzo-*p*-dioxin [1,2,3,6,7,8-HxCDD; 24 (30) pg/g I-TEq]; and 4) 2,3,7,8-tetrachlorodibenzo-*p*-dioxin [2,3,7,8-TCDD; 13 (19) pg/g I-TEq].

More frequent fish consumption produced higher median concentrations for all PCDD/F congeners, and the differences between exposed (median = 170 pg/g) and other fishermen (median = 87 pg/g) I-TEqs were statistically significant ($p < 0.05$). In the exposed fishermen group, 2,3,7,8-TCDD concentrations were as high as 110 pg/g, and I-TEq concentrations reached levels up to 420 pg/g. The coastal-group fishermen were significantly more exposed to dioxins compared with the Kuusankoski group. One distinctive exception to this trend was the concentration of 1,2,3,6,7,8-HxCDD, because concentrations in the Kuusankoski group were higher than in coastal group (270 vs. 210 pg/g fat, respectively).

Sum concentrations of 36 PCB congeners, along with individual congener concentrations and PCB toxic equivalents, are presented in Table 4. Mean and median sum PCB concentrations in all 47 fishermen were 2,100 and 1,400 ng/g fat, respectively, with the maximum value being 8,700 ng/g. The median PCB-TEq level (80 pg/g fat; mean = 110 pg/g fat) was slightly smaller than that in PCDD/Fs, but it did achieve values as high as 460 pg/g fat. The four main congeners accounting for 75% of the median sum PCB concentration were International Union of Pure Applied Chemistry (IUPAC) 138, 153, 170, and 180. The most dominant non-*ortho*-PCB was IUPAC 126, ranging from 35 to 1,500 pg/g fat in all subjects.

More frequent fish consumption produced greater concentrations of all PCB congeners, and PCB-TEq mean and median values were 130 and 120 pg/g fat, respectively. Place of residence produced an even bigger difference between the subgroups than the classification by fish consumption. The median PCB-TEq value in the coastal group (140 pg/g) was over twice that in the Kuusankoski group (65 pg/g), and for IUPAC 153, the difference in concentration between the groups was about 3-fold (800 vs. 280 ng/g fat, respectively).

The ratio between sum concentrations of PCBs and I-TEq in all subjects was about 14,200:1. In subgroups according to fish consumption, the ratio was comparable to the value in all subjects, but in subgroups according to place of residence, the ratio in the coastal group was 16,400:1 (ranging from 8,100:1 to 25,800:1), and the ratio in the Kuusankoski group was 11,300:1 (ranging from 6,000:1 to 14,900:1); this difference

**Table 4.** Mean, median, and (range) of non-*ortho*-PCBs,[a] other PCBs,[b] and TEqs[a] in blood samples for fishermen and the various subgroups.

| Congener IUPAC no. | All subjects *n* = 47 | Fish consumption frequency: Exposed fishermen *n* = 26 | Fish consumption frequency: Other fishermen *n* = 21 | Place of residence: Coast *n* = 25 | Place of residence: Kuusankoski *n* = 22 |
|---|---|---|---|---|---|
| Non-*ortho*-PCBs | | | | | |
| 77 | 63, 55 (ND–190) | 77, 68 (13–190)* | 45, 37 (ND–100) | 79, 68 (28–190)** | 44, 30 (ND–150) |
| 126 | 300, 230 (35–1,500) | 360, 260 (49–1,500)* | 240, 150 (35–950) | 430, 360 (61–1,500)** | 160, 150 (35–330) |
| 169 | 160, 130 (50–490) | 190, 180 (72–490)* | 130, 100 (50–280) | 190, 190 (67–490) | 130, 110 (50–300) |
| Other PCBs | | | | | |
| 18 | 0.88, 0.53 (ND–3.7) | 0.94, 0.52 (ND–3.7) | 0.82, 0.53 (ND–3.3) | 0.54, 0.23 (ND–2.4)** | 1.3, 0.90 (ND–3.7) |
| 28/31 | 13, 9.5 (0.24–94) | 15, 10 (0.54–94) | 9.9, 4.4 (0.24–36) | 17, 13 (0.24–94) | 8.0, 5.8 (0.54–33) |
| 33 | 1.0, 0.13 (ND–4.4) | 1.1, 0.43 (ND–4.4) | 0.92, 0.045 (ND–4.1) | 0.72, ND (ND–3.5) | 1.4, 0.76 (ND–4.4) |
| 47 | 1.1, 0.90 (ND–6.8) | 1.4, 1.1 (ND–6.8) | 0.85, 0.78 (0.11–2.3) | 1.3, 1.2 (ND–6.8) | 0.91, 0.78 (0.19–2.3) |
| 49 | 0.66, 0.53 (ND–2.0) | 0.71, 0.56 (ND–2.0) | 0.60, 0.48 (ND–2.0) | 0.43, 0.41 (ND–1.3)** | 0.93, 0.82 (0.063–2.0) |
| 51 | 0.063, 0.034 (ND–0.23) | 0.068, 0.043 (ND–0.22) | 0.056, 0.028 (ND–0.23) | 0.035, ND (ND–0.17)** | 0.095, 0.078 (ND–0.23) |
| 52 | 2.2, 1.6 (ND–14) | 2.5, 1.9 (0.69–14)* | 1.9, 1.3 (ND–11) | 2.3, 1.6 (ND–14) | 2.1, 1.7 (0.67–11) |
| 60 | 2.8, 1.5 (0.20–35) | 3.7, 1.6 (0.54–35) | 1.6, 1.0 (0.20–5.8) | 4.2, 2.8 (0.33–35)** | 1.2, 0.93 (0.20–3.6) |
| 66 | 16, 5.1 (0.54–200) | 22, 6.8 (1.6–200) | 8.2, 3.5 (0.54–35) | 27, 19 (2.1–200)** | 3.4, 2.6 (0.54–11) |
| 74 | 55, 36 (4.7–460) | 72, 41 (9.4–460) | 34, 23 (4.7–110) | 87, 56 (17–460)** | 18, 15 (4.7–51) |
| 99 | 59, 34 (6.0–290) | 74, 53 (8.2–290)* | 40, 28 (6.0–140) | 90, 82 (22–290)** | 24, 21 (6.0–57) |
| 101 | 4.7, 3.7 (0.27–26) | 5.8, 4.7 (0.27–26)* | 3.5, 3.1 (0.42–13) | 6.4, 5.2 (0.87–26)** | 2.9, 3.0 (0.27–6.5) |
| 105 | 31, 22 (2.7–150) | 38, 27 (4.2–150)* | 21, 11 (2.7–84) | 47, 39 (5.7–150)** | 12, 10 (2.7–23) |
| 110 | 3.0, 2.5 (0.21–13) | 3.6, 2.9 (0.40–13)* | 2.3, 1.7 (0.21–8.0) | 4.0, 3.8 (0.94–13)** | 1.9, 1.9 (0.21–3.4) |
| 114 | 5.7, 4.0 (0.81–22) | 6.9, 6.0 (1.3–22)* | 4.2, 2.6 (0.81–11) | 8.4, 8.1 (1.9–22)** | 2.6, 2.4 (0.81–5.9) |
| 118 | 150, 110 (16–730) | 180, 140 (24–730)* | 110, 59 (16–410) | 220, 180 (45–730)** | 66, 58 (16–120) |
| 122 | ND, ND | ND, ND | ND, ND | ND, ND | ND, ND |
| 123 | 6.2, 4.1 (0.56–25) | 7.6, 5.5 (0.70–25)* | 4.5, 2.6 (0.56–17) | 9.5, 8.7 (1.0–25)** | 2.5, 2.4 (0.56–5.1) |
| 128 | 4.5, 2.4 (ND–20) | 5.8, 4.6 (ND–20)* | 2.9, 1.1 (ND–14) | 7.9, 6.9 (1.1–20)** | 0.61, 0.22 (ND–3.3) |
| 138 | 320, 210 (41–1,600) | 400, 340 (77–1,600)* | 220, 180 (41–660) | 450, 400 (140–1,600)** | 160, 150 (41–420) |
| 141 | 1.4, 0.97 (ND–6.3) | 1.7, 1.2 (ND–6.3)* | 0.90, 0.69 (ND–5.5) | 1.9, 1.4 (ND–6.3)** | 0.73, 0.69 (ND–1.6) |
| 153 | 600, 380 (87–2,600) | 740, 590 (180–2,600)* | 430, 290 (87–1,400) | 860, 800 (240–2,600)** | 310, 280 (87–840) |
| 156 | 58, 50 (14–230) | 70, 63 (22–230)* | 43, 40 (14–89) | 72, 71 (23–230)** | 42, 39 (14–120) |
| 157 | 11, 7.9 (2.0–45) | 13, 11 (3.2–45)* | 8.0, 6.5 (2.0–22) | 15, 15 (4.1–45)** | 6.0, 6.0 (2.0–16) |
| 167 | 17, 14 (2.4–81) | 20, 18 (4.1–81)* | 12, 9.4 (2.4–35) | 24, 21 (6.2–81)** | 9.0, 8.8 (2.4–21) |
| 170 | 190, 160 (48–670) | 220, 200 (79–670)* | 140, 130 (48–270) | 220, 200 (87–670)** | 140, 130 (48–390) |
| 180 | 370, 300 (84–1,200) | 440, 370 (130–1,200)* | 280, 230 (84–620) | 470, 460 (190–1,200)** | 260, 230 (84–750) |
| 183 | 37, 25 (4.5–150) | 45, 35 (11–150)* | 26, 21 (4.5–80) | 49, 46 (15–150)** | 22, 20 (4.5–54) |
| 187 | 83, 65 (15–340) | 100, 100 (29–340)* | 57, 48 (15–130) | 110, 110 (38–340)** | 57, 48 (15–160) |
| 189 | 7.2, 6.4 (1.8–24) | 8.5, 7.7 (2.6–24)* | 5.5, 4.3 (1.8–11) | 8.8, 9.1 (3.8–24)** | 5.3, 4.3 (1.8–14) |
| 194 | 44, 41 (12–140) | 51, 47 (18–140)* | 34, 30 (12–57) | 52, 50 (22–140)** | 34, 29 (12–88) |
| 206 | 7.4, 6.0 (1.8–22) | 8.7, 7.9 (3.2–22)* | 5.8, 5.1 (1.8–12) | 9.7, 9.4 (4.1–22)** | 4.8, 4.7 (1.8–10) |
| 209 | 3.6, 3.4 (0.95–9.0) | 4.1, 3.8 (0.95–9.0)* | 3.0, 2.6 (1.2–6.5) | 4.2, 4.0 (1.5–9.0)** | 2.9, 2.9 (0.95–5.7) |
| Sum of PCBs | 2,100, 1,400 (360–8,700) | 2,600, 2,200 (680–8,700)* | 1,500, 1,200 (360–4,200) | 2,900, 2,700 (950–8,700)** | 1,200, 1,200 (360–3,100) |
| PCB-TEq | 110, 80 (21–460) | 130, 120 (30–460)* | 81, 68 (21–230) | 140, 140 (45–460)** | 66, 65 (21–150) |
| $WHO_{PCB}$-TEq | 89, 66 (17–400) | 110, 96 (22–400)* | 67, 52 (17–200) | 120, 110 (34–400)** | 51, 50 (17–110) |

ND, below limit of determination.
[a]Concentrations are given in pg/g fat. [b]Concentrations are given in ng/g fat *Significantly different compared with the other fishermen group ($p < 0.05$ by Mann-Whitney *U*-test). **Significantly different compared with the Kuusankoski place of residence group ($p < 0.05$ by Mann-Whitney *U*-test).

was statistically significant ($p < 0.001$). A similar difference was observed when the proportion of PCB-TEq was calculated from the total TEq. In the coastal group, PCB-TEq contributed 44% of the total TEq (320 pg/g fat), whereas in the Kuusankoski group, PCB-TEq accounted for 35% of the total TEq (186 pg/g fat). In both groups classified by fish consumption, the contribution of PCB-TEq to total TEq was 42%.

Figure 2 illustrates the impact of fish species consumed on the congener profile of an individual fisherman, the congener profiles of three fish species (Baltic herring/salmon, pike, and bream) and three fishermen. All three fishermen reported that they consumed solely or mostly the respective fish species.

Table 5 summarizes the regression analyses conducted to determine predictors of the variance of natural logarithms of $WHO_{PCDD/F}$-TEq, $WHO_{PCB}$-TEq, and total WHO-TEq. Age was the only significant regression predictor of ln $WHO_{PCDD/F}$-TEq, and the whole model explained 48% of the variance of ln $WHO_{PCDD/F}$-TEq. Age and the amount of fish consumed were the most important predictors, with contributions of 22.5% and 19.3%, respectively. Place of residence, age, and amount of fish consumed were significant regression predictors of both ln $WHO_{PCB}$-TEq and ln total WHO-TEq. For PCBs, the most important predictor was place of residence, with a 35.4% contribution, followed by age, with a 17.7% contribution. The most important predictors of variance for ln total WHO-TEq were the same as those for ln $WHO_{PCDD/F}$-TEq—age and amount of fish consumed—with the contributions being 21.5% and 23.6%, respectively.

In each of these three models, the normal distribution of residuals was verified with normal probability plots. Variance inflation factors (VIF) showed no multicollinearity between predictors in any of these three models.

## Discussion

Because the median age and distributions of ages among classified subgroups were so similar, we did not adjust the concentrations of PCDD/Fs and PCBs for age. The mean time of residence at the current address in the subgroups was also so long that each person would have adopted the local exposure pattern to PCDD/Fs and PCBs via their living habits. All persons with time of residence ≤ 9 years had been living in the same area earlier only at a different address.

Results of this study clearly associated higher body burden of PCDD/Fs and PCBs with higher intake of fish. Consuming fish at least twice a week resulted in plasma

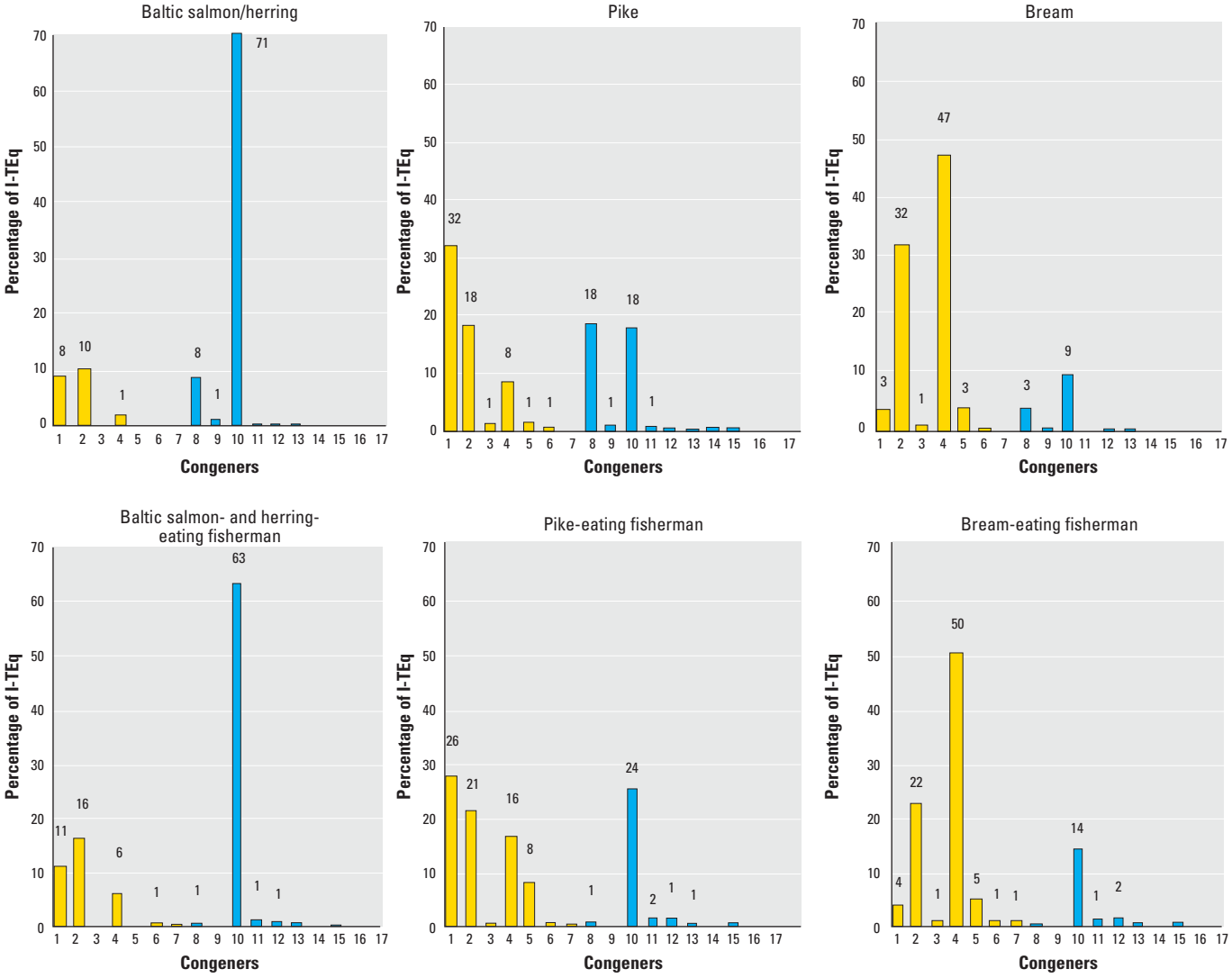


**Figure 2.** Congener I-TEq profiles of individual fishermen and profiles of fish species that each fisherman reported he prefers to consume. Congeners: 1: 2,3,7,8-TCDD; 2: 1,2,3,7,8-PeCDD; 3: 1,2,3,4,7,8-HxCDD; 4: 1,2,3,6,7,8-HxCDD; 5: 1,2,3,7,8,9-HxCDD; 6: 1,2,3,4,6,7,8-HpCDD; 7: OCDD; 8: 2,3,7,8-TCDF; 9: 1,2,3,7,8-PeCDF; 10: 2,3,4,7,8-PeCDF; 11: 1,2,3,4,7,8-HxCDF; 12: 1,2,3,6,7,8-HxCDF; 13: 2,3,4,6,7,8-HxCDF; 14: 1,2,3,7,8,9-HxCDF; 15: 1,2,3,4,6,7,8-HpCDF; 16: 1,2,3,4,7,8,9-HpCDF; 17: OCDF.

concentrations of PCDD/Fs over five times those found in a corresponding nonfisherman population in Finland (*15*). Fishermen who reported eating fish once a week or less also had elevated blood levels of PCDD/Fs and PCBs. Between the exposed fishermen and other fisherman subgroups, there was no difference in the species of fish consumed; therefore, the difference between these groups must be assumed to derive solely from the frequency of fish consumption. When the fishermen were grouped according to place of residence, the frequency of fish consumption did not have a critical effect on concentrations of PCDD/Fs and PCBs, although subjects in the coastal group ate fish more frequently than subjects in the Kuusankoski group. The species of fish consumed had a more critical effect because subjects in the coastal group ate fatty Baltic fish species more frequently than did subjects in the Kuusankoski group. Also, the consumption of rainbow trout by the coastal group was more frequent than by the Kuusankoski group, and one must bear in mind that in the Baltic sea, fishes in the class "pike" also have a higher content of PCDD/Fs and PCBs in their tissues compared with inland lake "pikes" (*7,8*).

The ratio between sum concentrations of PCBs and I-TEq in the coastal group was statistically significantly different from the corresponding ratio in the Kuusankoski group. This could be a result of the relatively more severe contamination of Baltic fish by PCBs than of fish in inland lakes. Furthermore, this ratio between the sum concentrations of PCB and I-TEq varied significantly within groups, from 8,100:1 to 25,800:1 in the coastal group and from 6,000:1 to 14,900:1 in the Kuusankoski group. Because the correlation between PCB congener IUPAC 153 and the sum concentrations of PCBs was almost 1, the use of IUPAC 153 as an indicator of dioxin TEqs can produce misleading results.

When we compared I-TEq congener patterns, we discovered individual differences. Because the role of fish is profound with respect to the fishermen's intake of PCDD/Fs, and because there were no statistically significant differences in other food consumption habits or smoking habits between the classified subgroups, we hypothesized that these differences in the I-TEq congener patterns were caused by consumption of different fish species. If a fisherman reported that he was consuming mainly one kind of fish species, it was often possible to detect a similar I-TEq congener profile in his fasting blood sample. Figure 2 shows that only the congener 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF) was missing from the fishermen's profiles. This is a result of rapid metabolism of this congener in humans. Almost half the fishermen in the Kuusankoski group fish from a lake famous for its bream catches. Examination of the I-TEq congener pattern reveals that 1,2,3,6,7,8-HxCDD is the main congener in bream, which might explain why the 1,2,3,6,7,8-HxCDD concentrations in the Kuusankoski group were higher than in coastal group, in contrast to the general trend. It was not possible to discern a similar effect when studying PCB congener patterns (i.e., the consumption of a certain fish species by one individual fisherman was not reflected in his blood PCB congener profile).

PCDD/F concentrations (in all subjects, 120 pg/g I-TEq in fat) assayed in this study are comparable to body burdens found in Swedish Baltic fishermen of the same age (*12*). Therefore, fishermen in Finland and all around the Baltic Sea area can accumulate via their diet dioxin body burdens that are comparable to the concentrations found in Seveso, Italy, after the accidental release of 2,3,7,8-TCDD. In our study, 2,3,7,8-TCDD concentrations rose up to 110 pg/g fat, which is at the same level found in Seveso B zone (*23*). The PCDD/F concentrations found in this study were somewhat higher than those found in Canada among the Inuits (39.6–56.7 pg/g I-TEq in fat) (*24,25*). The PCDD/F concentrations in frequent consumers of fish from the Great Lakes in the United States (*26*) also showed considerably lower levels (13.9–19.6 pg/g I-TEq in fat) than those found in the present study.

In this study, the median value for 36 PCB congeners was 1,400 ng/g fat, ranging up to 8,700 ng/g in the coastal area in those fishermen eating fish at least twice a week. In Swedish studies, the range of PCBs has been from 1,600 to 5,300 ng/g fat, but in those studies the number of congeners is not comparable to those in our study (*12,13*). The values for one of the main congeners of PCBs, IUPAC 153, are about the same in the Swedish studies (280–1,700 ng/g fat) as in our study (87–2,600 ng/g fat). In our study, the lower end of the PCB range comes from the inland lake fishermen; therefore, it would be better to compare the coastal group results from our study with the Swedish results. The range of IUPAC 153 in the coastal group from our study was from 240 to 2,600 ng/g fat, which is almost identical to concentrations measured in Sweden. The dominant congener in PCB-TEq is IUPAC 126. In our study, the concentrations of IUPAC 126 were slightly lower (median = 230 pg/g fat for all subjects and 360 pg/g fat for the coastal group) than those found in Sweden (from 560 to 1,050 pg/g fat) (*12*). In contrast to PCDD/Fs, the PCB concentration levels in Canada seem to be somewhat higher than those in our study. Ryan et al. (*25*) reported the sum PCB concentration for 11 congeners to be 6,000 ng/g fat and the concentration for IUPAC 126 to be 619 pg/g fat. The mean concentration of 20 PCBs in adult Inuits living in Nunavik was reported to be 4,000 ng/g, ranging up to 9,870 ng/g, and levels of IUPAC 153 ranged from 240 to 3,070 ng/g fat (*24*).

We used only four predictor variables in the linear regression analyses of ln $WHO_{PCDD/F}$-TEq, ln $WHO_{PCB}$-TEq, and ln total WHO-TEq. Using more variables with these 47 subjects would have increased the predictability of the models, but it would have reduced the model's generalization and limited the model's use with other Finnish fishermen samples. Age was the only significant predictor in all three models. The amount of fish consumed was the second dominating predictor of variance of ln

**Table 5.** Predictors of the variance of natural logarithms of $WHO_{PCDD/F}$-TEq, $WHO_{PCB}$-TEq, and total WHO-TEq for Finnish fishermen.

| Predictor variable | Parameter estimate | SE | *p*-Value |
|---|---|---|---|
| Dependent variable: ln $WHO_{PCDD/F}$-TEq | | | |
| Constant | 2.4 | 0.67 | < 0.001 |
| Age | 0.028 | 0.007 | < 0.0001 |
| BMI | 0.034 | 0.021 | < 0.12 |
| Amount of fish consumed | 0.12 | 0.064 | < 0.062 |
| Place of residence | 0.26 | 0.18 | < 0.14 |
| ln $WHO_{PCDD/F}$-TEq model percentage $r^2$ = 0.48 | | | |
| Dependent variable: ln $WHO_{PCB}$-TEq | | | |
| Constant | 1.9 | 0.65 | < 0.005 |
| Age | 0.027 | 0.006 | < 0.0001 |
| BMI | 0.021 | 0.02 | < 0.31 |
| Amount of fish consumed | 0.15 | 0.062 | < 0.02 |
| Place of residence | 0.53 | 0.17 | < 0.003 |
| ln $WHO_{PCB}$-TEq model percentage $r^2$=0.60 | | | |
| Dependent variable: ln total WHO-TEq | | | |
| Constant | 2.9 | 0.65 | < 0.0001 |
| Age | 0.027 | 0.006 | < 0.0001 |
| BMI | 0.030 | 0.021 | < 0.16 |
| Amount of fish consumed | 0.13 | 0.062 | < 0.041 |
| Place of residence | 0.35 | 0.17 | < 0.05 |
| ln total WHO-TEq model percentage $r^2$ = 0.53 | | | |

$WHO_{PCDD/F}$-Teq, in contrast to the predictor of variance of ln $WHO_{PCB}$-TEq, which was place of residence. This might be caused by differences in dioxin congener profiles among fish species, because fish species eaten was taken into account when weighted fish amounts were calculated. We detected no difference in PCB profiles among fish species similar to that seen in dioxin profiles. This might explain why place of residence, not consumption of fish, was the second dominating predictor of variance of ln $WHO_{PCB}$-TEq.

In conclusion, we found that in Finland, fish consumption can cause elevated levels of PCDD/Fs and PCBs. Especially high levels of these contaminants can result from consumption of fatty Baltic fish. It was possible to determine the type of fish species that an individual fisherman consumed most from his blood I-TEq congener pattern.

## References and Notes


1. Hallikainen A, Mustaniemi A, Vartiainen T. Dioxin Intake from Food [in Finnish; summary in English]. Helsinki, Finland:National Food Administration, 1995;1–17.
2. Hallikainen A, Vartiainen T. Food control surveys of polychlorinated dibenzo-*p*-dioxins and dibenzofurans and intake estimates. Food Addit Contam 14(4):355–366 (1997).
3. Kiviranta H, Hallikainen A, Ovaskainen M-L, Kumpulainen J, Vartiainen T. Dietary intakes of polychlorinated dibenzo-p-dioxins, dibenzofurans and polychlorinated biphenyls in Finland. Food Addit Contam 18:945–953 (2001).
4. Finnish Game and Fisheries Research Institute. Finnish Fisheries Statistics 2000. Available: http://www.rktl.fi/tilasto/taskutilasto.pdf [cited 11 January 2001].
5. Rappe C, Bergqvist PA, Kjeller LO. Levels, trends, and patterns of PCDDs and PCDFs in Scandinavian environmental samples. Chemosphere 18:651–658 (1989).
6. Vartiainen T, Parmanne R, Hallikainen A. Ympäristömyrkkyjen kertyminen silakkaan [in Finnish]. Ympäristö ja terveys-lehti 7–8:18–22 (1997).
7. Korhonen M. Dioksiinit rannikon kaloissa [in Finnish]. Ympäristö 7:21 (1997).
8. Korhonen M. Dioksiinit sisävesien kaloissa [in Finnish]. Ympäristö 7:22 (1998).
9. Kiviranta H, Korhonen M, Hallikainen A, Vartiainen T. Kalojen dioksiinien ja PCB:iden kulkeutuminen ihmiseen [in Finnish]. Ympäristö ja terveys-lehti 3:65–69 (2000).
10. Svensson B-G, Nilsson A, Hansson M, Rappe C, Åkersson B, Skerfving S. Exposure to dioxins and dibenzofurans through the consumption of fish. N Engl J Med 324(1):8–12 (1991).
11. Asplund L, Svensson B-G, Nilsson A, Eriksson U, Jansson B, Jensen S, Wideqvist U, Skerfving S. Polychlorinated biphenyls, 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane (*p,p'*-DDT) and 1,1-dichloro-2,2-bis(*p*-chlorophenyl)-ethylene (*p,p'*-DDE) in human plasma related to fish consumption. Arch Environ Health 49(6):477–486 (1994).
12. Svensson B-G, Nilsson A, Jonsson E, Schütz A, Åkersson B, Hagmar L. Fish consumption and exposure to persistent organochlorine compounds, mercury, selenium and methylamines among Swedish fishermen. Scand J Work Environ Health 21:96–105 (1995).
13. Sjödin A, Hagmar L, Klasson-Wehler E, Björk J, Bergman Å. Influence of the consumption of fatty Baltic Sea fish on plasma levels of halogenated environmental contaminants in Latvian and Swedish men. Environ Health Perspect 108:1035–1041 (2000).
14. Ministry of Agriculture and Forestry. Fisheries. Available: http://www.mmm.fi/english/fisheries/occupational/ [cited 11 January 2001].
15. Kiviranta H, Vartiainen T, Verta M, Tuomisto JT, Tuomisto T. High fish-specific dioxin concentrations in Finland. Lancet 355:1883–1885 (2000).
16. Kiviranta H, Purkunen R, Vartiainen T. Levels and trends of PCDD/Fs and PCBs in human milk in Finland. Chemosphere 38(2):311–323 (1999).
17. NATO/CCMS. International Toxicity Equivalency Factors (I-TEF)- Method of Risk Assessment for Complex Mixtures of Dioxins and Related Compounds. Report 176. Washington, DC:North Atlantic Treaty Organization/ Committee on the Challenge of Modern Society, 1988.
18. Ahlborg UG, Becking GC, Birnbaum LS, Brouwer A, Derks HJGM, Feeley M, Golor G, Hanberg A, Larsen JC, Liem AKD. Toxic equivalency factors for dioxin-like PCBs. Chemosphere 28:1049–1067 (1994).
19. Van den Berg M, Birnbaum L, Bosveld ATC, Brunström B, Cook P, Feeley M, Giesy JP, Hanberg A, Hasegawa R, Kennedy SW, et al. Toxic equivalency factors (TEFs) for PCBs PCDDs, PCDFs for humans and wildlife. Environ Health Perspect 106:775–792 (1998).
20. Rymen T. History of the BCR work on dioxins. Fresenius J Anal Chem 348:9–22 (1994).
21. WHO/ECEH. Quality Assessment of PCBs, PCDD and PCDF Analysis: Third Round of WHO-Coordinated Study. Environmental Health in Europe 2. Bilthoven-Copenhagen-Nancy-Rome:WHO, European Centre for Environment and Health, 1996.
22. Yrjänheikki EJ. Levels of PCBs, PCDDs and PCDFs in Human Milk and Blood: Second Round of Quality Control Studies. Copenhagen:FADL (published on behalf of the WHO Regional Office for Europe, Environment and Health in Europe Series No. 37 ), 1991.
23. Needham LL, Gerthoux PM, Patterson DG Jr, Brambilla P, Turner WE, Beretta C, Pirkle LJ, Colombo L, Sampson EJ, Tramacere PL, et al. Serum dioxin levels in Seveso, Italy, population in 1976. Teratog Carcinog Mutagen 17:225–240 (1997/1998).
24. Ayotte P, Dewailly É, Ryan JJ, Bruneau S, Lebel G. PCBs and dioxin-like compounds in plasma of adult Inuit living in Nunavik (arctic Québec). Chemosphere 34(5-7):1459–1468 (1997).
25. Ryan JJ, Dewailly É, Gilman A, Laliberté C, Ayotte P, Rodrigue J. Dioxin-like compounds in fishing people from the lower north shore of the St. Lawrence Rver, Québec, Canada. Arch Environ Health 52:309–316 (1997).
26. Falk C, Hanrahan L, Anderson HA, Kanarek MS, Draheim L, Needham L, Patterson D Jr, the Great Lakes Consortium. Body burden levels of dioxin, furans, and PCBs among frequent consumers of Great Lakes sport fish. Environ Res Sect A 80:19–25 (1999).