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Exposure and Human PCDD/F and PCB Body Burden in Finland

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Kansanterveyslaitos KTL
Department of Environmental Health,
National Public Health Institute
Kuopio, Finland

Hannu Kiviranta

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A C A D E M I C D I S S E R T A T I O N

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Kansanterveyslaitos (KTL)
Mannerheimintie 166
00300 Helsinki
Puh. vaihde (09) 474 41, telefax (09) 4744 8408

Folkhälsoinstitutet
Mannerheimvägen 166
00300 Helsingfors
Tel. växel (09) 474 41, telefax (09) 4744 8408

National Public Health Institute
Mannerheimintie 166
FIN-00300 Helsinki, Finland
Telephone +358 9 474 41, telefax +358 9 4744 8408

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Supervised by

Professor Terttu Vartiainen, Ph. D.
National Public Health Institute (KTL)
Department of Environmental Health
Kuopio, Finland

Docent Pia Verkasalo, M. D., Ph. D.
National Public Health Institute Institute (KTL)
Department of Environmental Health
Kuopio, Finland

Reviewed by

Docent Matti Viluksela, Ph. D.
National Public Health Institute (KTL)
Department of Environmental Health
Kuopio, Finland

Professor Matti Verta, Ph. D.
Finnish Environment Institute
Helsinki, Finland

Opponent

Professor Gunilla Lindström, Ph.D.
MTM Research Centre
Örebro University
Örebro, Sweden

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ABSTRACT

Polychlorinated dibenzo-p-dioxins, dibenzofurans (PCDD/Fs) and polychlorinated biphenyls (PCBs) are widespread environmental contaminants. Due to their lipophilicity and persistency, they accumulate in the food chain. The most potent of these compounds exert several toxic effects in experimental animals such as immunosuppression, body weight loss, enzyme induction, developmental defects and tumor promotion. In humans, accidental or occupational exposures to high doses of PCDD/Fs and/or PCBs have caused lesions of skin, chloracne, developmental defects, and increased the risk of cancers. Exposure to PCDD/Fs has been associated with mineralisation defects in the first molar teeth, and PCBs are suspected to cause neurobehavioural effects as well as to function as endocrine disrupters.

PCDD/Fs have never been intentionally manufactured, but PCBs have been used in variety of applications e.g. dielectric fluids in transformers, hydraulic systems, and paints. There has been a significant reduction in the PCDD/F and PCB environmental levels due to control measures. Concentrations have declined by as much as 90% in the environment from those of the late 1960s. Today the major sources of PCDD/Fs and PCBs are burning processes (waste incineration and backyard burning), metal industries, contaminated soil and sediments, and landfill sites with contaminated material.

Knowledge about the levels of intake and body burden of PCDD/Fs and PCBs in a population helps to focus efforts to diminish population exposure to these hazardous compounds. The effects of already applied measures to limit the population exposure to these contaminants can be judged by examining the temporal changes in intakes and body burdens. History and current occurrence of these compounds provide a way to assess future development of concentrations. The high PCDD/F and/or PCB exposed group of people would be the best target group to study hazardous effects of PCDD/Fs and PCBs and therefore finding such a group is essential for any epidemiological study.

This study has evaluated the characteristics of average intake of PCDD/Fs and PCBs in Finland. Adipose tissue concentrations of PCDD/Fs and PCBs were measured in the general population, and concentrations in three geographical areas were compared. A survey of Finnish breast milk samples from two locations was conducted. A pilot study of professional fishermen was conducted. PCDD/F and PCB intake assessments, body burdens in the general population, breast milk concentrations, and change with time of these contaminants were compared with the corresponding results from Europe or around the world.

Intake studies revealed that the average adult Finnish intake of PCDD/Fs and PCBs was 1.5 pg WHO-TEq/kg bw/day which is below the suggested tolerable daily intake (TDI) of 2 pg WHO-TEq/kg bw/day according to EU SCF. When comparing to European countries, the intake of PCDD/Fs was similar and the intake of PCBs was slightly lower in Finland. An annual decrease of 6% in the PCDD/F intake during the 1990s has occured. Fish and fish products contributed most (60%-95%) to the intakes in Finland, this being due to the high contribution of Baltic Sea fish contaminated with these substances. It was proposed that in the imminent future, any changes in time in PCDD/F and PCB intakes would mostly be attributable to changes in population food habits and not to changes in the occurrence of these contaminants in foodstuffs. This is not surprising since changes in concentrations in fish take place very slowly and the results suggest that the decline of concentrations in Baltic herring has levelled off during the last

decade. In addition, the contribution of contaminants in other foodstuffs to the intake and concentrations has been small when compared to fish in Finland especially during the last years.

Adipose tissue concentrations of PCDD/Fs (median 24 pg $WHO_{PCDD/F}$-TEq/g) and PCBs (median 17 pg WHO_{PCB}-TEq/g) in the general Finnish population were comparable to European concentrations. After age adjustment, the body burdens declined as one moves from the coastal areas to more inland areas. This decrease was suggested to be due to differences in the consumption of fish species i.e. Baltic herring were consumed more frequently in coastal than in inland areas. Although no numerical estimate from the available data could be made, the population based concentration frequency graph suggested that exposure of Finnish population to these contaminants have been declining during the last decades. Professional fishermen were shown to represent a highly exposed population. Their concentrations were 2 to 4 times higher than in other men of the same age. The $WHO_{PCDD/F}$-TEq concentrations in serum fat of fishermen were at maximum 500 pg/g fat.

A decline similar to other countries was detected in the breast milk concentrations of PCDD/F and PCBs, being annually 5% and 6%, respectively. A difference in concentrations in breast milk was found between the capital and Kuopio area until 1994 but in the most recent study from the year 2000, this difference had disappeared. In the year 2000, the average concentration of $WHO_{PCDD/F}$-TEq was 9.4 pg/g fat, and WHO_{PCB}-TEq 5.9 pg/g fat, both concentrations being close to European levels.

Assessment of congener patterns of PCDD/Fs and PCBs in diet and human samples indicated that dioxins bioaccumulate more efficiently than furans, and lower chlorinated PCBs have a lower bioaccumulation property than higher chlorinated ones. The differences in bioaccumulations suggest that the TEF values alone are not capable in depicting differences of PCDD/F and PCB congeners between different matrices and trophic levels.

Keywords: PCDD/F, dioxin, PCB, dietary intake, Baltic herring, Finnish population, body burden, breast milk, fishermen

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TIIVISTELMÄ

Klooratut dibentso-p-dioksiinit, dibentsofuraanit (PCDD/F) ja klooratut bifenyylit (PCB) ovat kaikkialle levinneitä ympäristömyrkkyjä. Rasvaliukoisuutensa ja pysyvyytensä takia ne kertyvät ravintoverkossa. Kaikkein haitallisimpien niistä on todettu aiheuttavan koe-eläimissä mm. vastustuskyvyn heikkenemistä, painonlaskua, vierasainemetabolian aktivoitumista, kehityshäiriöitä ja kasvaimia. Tapaturmaisesti tai työperäisesti korkeille PCDD/F- ja/tai PCB-pitoisuuksille altistuneilla ihmisillä on raportoitu klooriakne, kehityshäiriöitä sekä kasvanut syöpäriski. Ensimmäisten pysyvien poskihampaiden mineralisaatiohäiriöt on yhdistetty korkeaan PCDD/F altistukseen, ja PCB:eiden on epäilty vaikuttavan haitallisesti keskushermoston kehitykseen ja häiritsevän hormonien toimintaa.

Dioksiineja ja furaaneja ei ole valmistettu tarkoituksellisesti, kun taas PCB:lle on ollut lukuisia eri käyttötarkoituksia, esim. muuntajaöljyinä, hydrauliikkanesteinä ja lisäaineina maaleissa. Päästörajoitukset ovat merkittävästi pienentäneet PCDD/F- ja PCB-yhdisteiden pitoisuuksia ympäristössä. Pitoisuudet ovat laskeneet jopa 90 % 1960-luvun lopulta alkaen. Olemassaolevia lähteitä ovat erilaiset polttoprosessit (jätteenpoltto ja pienpoltto), metalliteollisuus, saastuneet maat ja sedimentit sekä saastuneita materiaaleja sisältävät kaatopaikat.

Tietous yhdisteiden saannista sekä ihmisissä esiintyvistä pitoisuuksista auttaa kohdentamaan toimenpiteitä, joilla tehkkaimmin vähennetään ihmisten altistumista näille haitallisille yhdisteille. Jo toteutettujen päästöjen rajoitusten tehokkuutta voidaan arvioida analysoimalla pitoisuuksien muutosta ajan mukana. Nykyhetken ja historian tunteminen auttaa altistumisen tulevaisuuden ennustamisessa. Jotta altistumisen aiheuttamia terveyshaittoja päästäisiin parhaiten tutkimaan, olisi tärkeätä löytää ryhmä, joka altistuu PCDD/F- ja PCB-yhdisteille selvästi keskimääräistä enemmän.

Tässä tutkimuksessa selvitettiin suomalaisten: a) PCDD/F- ja PCB-saanti ja sen erityispiirteitä, b) keskimääräiset PCDD/F- ja PCB-kudospitoisuudet ja verrattiin pitoisuuksia alueellisesti, c) äidinmaitojen PCDD/F- ja PCB-pitoisuudet kahdella alueella sekä d) ammattikalastajien dioksiini- ja PCB-pitoisuuksia. Pitoisuustuloksia sekä niissä tapahtuneita muutoksia verrattiin vastaaviin tutkimuksiin Euroopassa ja muulla maailmassa.

Suomalaisten aikuisten keskimääräiseksi PCDD/F- ja PCB-päiväsaanniksi saatiin 1,5 pg WHO-TEq/kg rp (ruumiinpainokilo) kohti, joka on alle EU:n elintarvikealan tiedekomitean ehdottaman sallittavan päiväsaannin (TDI), 2 pg WHO-TEq/kg rp. Dioksiinien ja furaanien osalta saanti Suomessa vastaa eurooppalaista saantia, mutta PCB:lle altistutaan Suomessa hiukan vähemmän kuin muulla Euroopassa. Tuloksista määritettiin vuosittainen 6 % alenema PCDD/F saannissa 1990-luvun aikana. Kalan ja kalatuotteiden osuus saannista oli suuri (60 %-95 %), joka johtuu Itämeren kalan runsaasta käytöstä. Itämeren kalan osoitettiin olevan saastunut PCDD/F- ja PCB-yhdisteillä. Tuloksista arvioitiin, että muutokset väestön ruokavaliossa vaikuttavat enemmän altistumiseen, kuin muutokset ravintoaineiden PCDD/F- ja PCB-pitoisuuksissa. Tämä on ilmeistä, sillä muutokset kalojen haitallisten aineiden pitoisuuksissa ovat hitaita.

Kudospitoisuuksissa (PCDD/F mediaani 24 pg $WHO_{PCDD/F}$-TEq/g ja PCB mediaani 17 pg WHO_{PCB}-TEq/g) suomalaisten pitoisuudet vastasivat eurooppalaisia. Kun tulokset ikävakioitiin, pienenivät pitoisuudet siirryttäessä rannikolta sisämaahan. Pitoisuuksien ero johtui kalalajien kulutuseroista eri alueilla niin, että rannikoilla käytetään enemmän silakkaa ja muita Itämeren

kaloja. Väestöstä mitatuista pitoisuuksista pääteltiin myös, että suomalaisten altistuminen PCDD/F:lle ja PCB:lle on pienentynyt viimeisten vuosikymmenten aikana. Tämä oli ilmeistä, sillä vakioaltistuksella ja 7-8 vuoden puoliintumisajalla odotettavissa olevaa aluksi nousevaa ja noin 40 ikävuoden jälkeen tasoittuvaa pitoisuuskäyrää ei löytynyt väestötasolla. Ammattikalastajien osoitettiin altistuvan keskimääräistä väestöä enemmän PCDD/F:lle ja PCB:lle. Heistä mitatut pitoisuudet olivat 2-4 kertaa korkeammat kuin samanikäisillä keskimääräistä väestöä edustavilla miehillä. $WHO_{PCDD/F}$-TEq seerumipitoisuudet olivat enimmillään 500 pg/g rasvaa kohden.

Suomalaisten äidinmaitojen vuosittaiset pitoisuuksien alenemat (PCDD/F:ssa 5 % ja PCB:ssä 6 %) olivat samansuuruiset muiden maiden kanssa ja vastasivat saantiarvioissa määritettyä laskua. Vielä 1994 määritetyissä äidinmaitojen pitoisuuksissa oli pääkaupunkiseudun ja Kuopion välillä ero, mutta uusimmissa, vuonna 2000 määritetyissä äidinmaidoissa eroa ei enää ollut. Vuonna 2000 keskimääräiset pitoisuudet olivat $WHO_{PCDD/F}$-TEq 9,4 pg/g rasvaa kohden ja WHO_{PCB}-TEq 5,9 pg/g, jotka vastasivat eurooppalaisia tasoja.

Tutkituista johdosprofiileista pystyttiin päättelemään, että dioksiinit kertyvät furaaneja tehokkaammin ravinnosta ihmiseen. Pienemmän kloorautumisasteen omaavat PCB-yhdisteet taas kertyivät heikommin ihmisiin verrattuna korkeammin kloorattuihin johdoksiin. Nämä johdosten kertymiserot osoittavat, että nykyään käytössä olevat toksisuusekvivalenttikertoimet (TEF) eivät pysty kuvaamaan eri johdosten eroja eri matriiseissa ja eri ravintoketjun tasoilla.

Avansanat: PCDD/F, dioksiinit, PCB, saanti, silakka, suomalainen, kudospitoisuus, äidinmaito, kalastaja

To my family

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Kuopio, September 2005

Hannu Kiviranta

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ABBREVIATIONS

AHR	aryl hydrocarbon receptor
AMAP	Arctic Monitoring and Assessment Programme
ANOVA	analysis of variance
BMI	body mass index
bw	body weight
cf	condition factor
COT	The UK Committee on Toxicity
EC	European Community
EN ISO/IEC	European Standard/International Organization for Standardization /International Electrotechnical Commission
EU	European Union
FAO	Food and Agriculture Organization
FSA	Food Standards Agency
fw	fresh weight
IARC	International Agency for Research on Cancer
ICES	International Council for the Exploration of the Sea
I-TEF	international toxic equivalency factor according to NATO/CCMS
I-TEQ/I-TEq	toxic equivalent quantity according to I-TEFs
IUPAC	International Union of Pure and Applied Chemistry
JECFA	The Joint FAO/WHO Expert Committee on Food Additives
LOD	limit of determination
LOQ	limit of quantitation
MAFF	Ministry of Agriculture, Fisheries and Food
MBM	market basket method
N-TEQ/N-TEq	Nordic toxic equivalent quantity
OC	organochlorine compounds
PBDE	polybrominated diphenyl ether
PCB	polychlorinated biphenyl
PCB-TEF	toxic equivalency factor for PCBs according to Ahlborg et al. 1994
PCB-TEQ/-TEq	toxic equivalent quantity according to PCB-TEFs
PCDD	polychlorinated dibenzo-*p*-dioxin
PCDF	polychlorinated dibenzofuran
PCDD/F	polychlorinated dibenzo-*p*-dioxin/ polychlorinated dibenzofuran
SCF	Scientific Committee on Food
SD	standard deviation
SSIF	selective study of individual foodstuffs
STS	soft tissue sarcoma
TCDD	2,3,7,8-tetrachlorodibenzo-*p*-dioxin
TDI	tolerable daily intake
TEF	toxic equivalency factor
TEq/TEQ	TCDD-toxic equivalent quantity
TWI	tolerable weekly intake
USEPA	United States Environmental Protection Agency
VIF	variance inflation factor
WHO	World Health Organization
WHO_{PCB}-TEF	TCDD-toxic equivalency factor according to WHO for PCBs
WHO_{PCB}-TEQ/ -TEq	toxic equivalent quantity according to WHO_{PCB}-TEF
$WHO_{PCDD/F}$-TEF	TCDD-toxic equivalency factor according to WHO for PCDD/Fs
$WHO_{PCDD/F}$-TEQ/ -TEq	toxic equivalent quantity according to $WHO_{PCDD/F}$-TEF
NATO/CCMS	North Atlantic Treaty Organization/Committee on the Challenge of Modern Society

LIST OF ORIGINAL PUBLICATIONS

This thesis contains the following original publications:

1. Kiviranta H, Hallikainen A, Ovaskainen M-L, Kumpulainen J, Vartiainen T. 2001. Dietary intakes of polychlorinated dibenzo-*p*-dioxins, dibenzofurans and polychlorinated biphenyls in Finland. *Food Additives and Contaminants* 18 (11), 945-953.
2. Kiviranta H, Ovaskainen M-L, Vartiainen T. 2004. Market basket study on dietary intake of PCDD/Fs, PCBs, and PBDEs in Finland. *Environmental International* 30, 923-932.
3. Kiviranta H, Vartiainen T, Parmanne R, Hallikainen A, Koistinen J. 2003. PCDD/Fs and PCBs in Baltic herring during the 1990s. *Chemosphere* 50, 1201-1216.
4. Kiviranta H, Tuomisto JT, Tuomisto J, Tukiainen E, Vartiainen T. 2005. Polychlorinated dibenzo-*p*-dioxins, dibenzofurans, and biphenyls in the general population in Finland. *Chemosphere* 60, 854-869.
5. Kiviranta H, Purkunen R, Vartiainen T. 1999. Levels and trends of PCDD/Fs and PCBs in human milk in Finland. *Chemosphere* 38 (2), 311-323.
6. Kiviranta H, Vartiainen T, Tuomisto J. 2002. Polychlorinated dibenzo-*p*-dioxins, dibenzofurans, and biphenyls in fishermen in Finland. *Environmental Health Perspectives* 110 (4), 355-361.

CONTENT

CHAPTER 1

GENERAL INTRODUCTION

1. LITERATURE REVIEW

Structure and sources

Polychlorinated dibenzo-*p*-dioxins (PCDDs) and dibenzofurans (PCDFs) have a planar aromatic tricyclic structure with 1-8 chlorine atoms as substituents (Fig 1). It is possible to create 75 different PCDDs and 135 PCDFs, which differ from each other in the number and positions for the chlorine atoms. From the human/biota point of view, 17 PCDD/Fs with lateral (2,3,7,8-) chlorine substitution pattern are considered to be toxicologically important (WHO/IPCS 1989).

Polychlorinated biphenyls (PCBs) have two benzene rings attached to each other, with 1-10 chlorine atoms as substituents (Fig 1). Theoretically it is possible to form 209 different congeners of PCBs, but even the technical mixtures of PCBs have only a fraction of the total possible number of congeners. Some PCBs are called dioxin-like (co-planar/non-*ortho*-) PCBs. Those congeners do not have any or have only one chlorine atom (mono-*ortho*-PCBs) in the *ortho*-position to the carbon-carbon bond between the two benzene rings. A dozen of these congeners are believed to express similar toxicogical effects as PCDD/Fs to humans and biota (van den Berg et al. 1998).

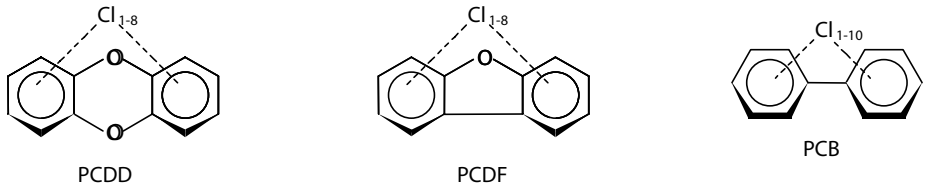

Fig 1. Chemical structures of polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and polychlorinated biphenyls (PCBs).

PCDD/Fs have never been intentionally manufactured. However they do occur as minor impurities in many chlorinated chemicals (e.g. in PCBs, and in chlorinated pesticides as fungicides and herbicides) (WHO/IPCS 1989, Vartiainen et al. 1995, Michalek et al. 1996). Burning processes in the presence of chlorine and with metal catalysts are sources of PCDD/Fs. It has been estimated that municipal solid waste incineration and accidental fires, together with backyard burning, contribute significantly to PCDD/F emissions to land and water in the EU countries (Wenborn et al. 1999). The metal-processing industries e.g. secondary Pb, Cu, and Al

production also contribute to PCDD/F emissions, but not to the same extent as burning processes (Wenborn et al. 1999). Sources of PCDD/Fs in Finland originate partly from air emissions coming from Central Europe, because prevailing winds in Finland are from the southwest direction (Shatalov et al. 2003). Some of the air emissions are domestic in origin and earlier emissions can be attributed to pulp and paper industries using elemental chlorine for pulp bleaching (Wulff et al. 1993, MacDonald et al. 1998). The production and application of a chlorophenol mixture called "Ky-5" which was used as a wood preservative in sawmills from the 1940s until the mid 1980s led to soil and sediment contamination by PCDD/Fs in many sawmill, landfill, and disposal sites as well as in sediments of the Gulf of Finland in the Baltic Sea (Vartiainen et al. 1995, Kitunen and Salkinoja-Salonen 1990, Assmuth and Vartiainen 1994, Isosaari et al. 2002). The prohibition of usage of chlorinated pesticides and chlorophenols, and abandonment of elemental chlorine for pulp bleaching, together with reductions in emissions to air (Quaß et al. 2004) have led to a nearly 90% decrease in PCDD/F emissions since the 1980s in European countries and also in Finland.

PCBs have many useful characteristics, e.g. non-flammability, electrical insulating properties, and stability and they have been used globally in a great variety of applications. So-called closed uses of PCBs included their use as dielectric fluids in transformers, capacitors, and as heat transfer fluids, and in hydraulic systems. Open use has involved the application as pesticide extenders, sealants, carbonless copy papers, industrial oils, paints, adhesives, plastics, flame retardants and controlling of dust on roads (http://europa.eu.int/comm/environment/waste/pcbs/index.htm). At least 1.5 million tonnes of PCBs were produced between 1930s and 1980s under different trade names such as Aroclor, Clophen, and Kanechlor (Bernes 1998). Nowadays PCBs can be found everywhere around the globe including the Arctic (AMAP 2004). The Baltic Sea sediments reveal that the maximum emissions to the area have occured during the late 1960s and early 1970s (Isosaari et al. 2002, Kjeller and Rappe 1995). Current surface sediment concentrations of PCBs are 2-5 times lower than during the periods of maximum concentrations (Isosaari et al. 2002, Kjeller and Rappe 1995, Konat and Kowalewska 2001).

Persistency and toxicity

PCDD/Fs and PCBs are environmentally stable and (in particular 2,3,7,8-chlorine subsituted PCDD/F congeners) biologically persistent (Sinkkonen and Paasivirta 2000). These characteristics together with high lipophilicity; log K_{ow} for PCDD/Fs ranging from 6.1 to 8.2 (Mackay et al. 1992), and for PCBs from 4.9 to 8.2 (Mackay et al. 1991), result in accumulation

of PCDD/Fs and PCBs in food chain (AMAP 2004). The half-lives of 2,3,7,8-chlorine substituted PCDD/Fs in man have been estimated to be on average seven years, with values ranging from few months to decades (Poiger and Schlatter 1986, Flesch-Janus et al. 1996, Liem and Theelen 1997, Geyer et al. 2002), and with the corresponding PCB half-lives in man ranging from months to several years (Chen et al. 1982, Taylor and Lawrence 1992, Ryan et al. 1993).

The toxicity of PCDD/Fs involves the cytosolic aryl hydrocarbon receptor (AHR), which is a ligand-activated transcription factor. Binding of PCDD/Fs to AHR initiates the expression of several genes in a cell (Poellinger 2000) and leads to toxic effects by mechanisms, which are still not fully understood. The most toxic congener of PCDD/Fs is 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), which serves as a reference compound in terms of its affinity to AHR for the other PCDD/Fs, and also for dioxin-like PCBs. The concept of TCDD toxic equivalency factor (TEF) was developed to describe the total toxic equivalent quantity (TEq) of a mixture of PCDD/Fs and/or dioxin-like PCBs (Safe 1990). The TEF concept presupposes that the molecule will bioaccumulate in the food chain, will possess a structural similarity to PCDD/Fs, will bind to AHR, and elicit AHR-mediated responses. The most recent TEFs are based on a consensus statement agreed at a convention organized by the World Health Organization (WHO) in Stockholm in 1997 (van den Berg et al. 1998). Table 1 describes these so-called $WHO_{PCDD/F}$-TEFs and WHO_{PCB}-TEFs together with previously used TEFs (NATO/CCMS 1988, Ahlborg et al. 1994). Equation 1 presents the calculation of TEq in a sample.

(1) $TEq = \sum_{i=1}^{n} (C_i * TEF_i)$ in which C_i is the concentration of a congener with a TEF_i value.

Although there are numerous toxic endpoints of PCDD/Fs and PCBs shown in experimental animals (Pohjanvirta and Tuomisto 1994), only a few of them have been demonstrated in humans. Chloracne is associated with PCDD/Fs in both occupational and accidental exposures to high amounts (Zober et al. 1990, Mocarelli et al. 1991, Ott et al. 1993, Geusau et al. 2001). Cancer is another human endpoint associated with PCDD/Fs, and the International Agency for Research on Cancer (IARC) has concluded, based on experimental animal studies, that TCDD is a human carcinogen (IARC 1997, http://www-cie.iarc.fr/htdocs/monographs/vol69/dioxin.html). IARC has concluded that for other PCDD/F congeners there is inadequate evidence of carcinogenicity to humans. According to IARC, PCBs are probably carcinogenic to humans (http://www-cie.iarc.fr/htdocs/monographs/suppl7/polychlorinatedbiphenyls.htm).

Although epidemiological studies attempting to link PCDD/F exposure (i.e. a mixture of chemicals) to cancer have suffered from simultaneous exposures to other kinds of chemicals e.g.

chlorinated herbicides and fungicides, and limited exposure measurements, it has been estimated that an increased total cancer risk can be associated with high exposures to PCDD/Fs (Fingerhut et al. 1991, Flesch-Janus et al. 1995, Ott and Zober 1996). On the other hand, individual concentration measurements were used in assessing an average and usually low level diet-driven exposure to PCDD/Fs in a Finnish case-control study on soft tissue sarcoma (STS). In this study it was not possible to associate increased risk of STS to increased PCDD/F concentration (Tuomisto et al. 2004).

Table 1.

Toxic equivalency factors (TEFs) according to WHO (van den Berg et al. 1998) ($WHO_{PCDD/F}$-TEFs and WHO_{PCB}-TEFs) together with NATO (NATO/CCMS 1988) TEFs for PCDD/Fs (I-TEF) and PCB-TEFs according to Ahlborg et al. (1994) for PCBs.

Congener	I-TEF	$WHO_{PCDD/F}$-TEF	Congener	PCB-TEF	WHO_{PCB}-TEF
2,3,7,8-TCDD	1	1	PCB 81	-	0.0001
1,2,3,7,8-PeCDD	0.5	1	PCB 77	0.0005	0.0001
1,2,3,4,7,8-HxCDD	0.1	0.1	PCB 126	0.1	0.1
1,2,3,6,7,8-HxCDD	0.1	0.1	PCB 169	0.01	0.01
1,2,3,7,8,9-HxCDD	0.1	0.1	PCB 105	0.0001	0.0001
1,2,3,4,6,7,8-HpCDD	0.01	0.01	PCB 114	0.0005	0.0005
OCDD	0.001	0.0001	PCB 118	0.0001	0.0001
2,3,7,8-TCDF	0.1	0.1	PCB 123	0.0001	0.0001
1,2,3,7,8-PeCDF	0.05	0.05	PCB 156	0.0005	0.0005
2,3,4,7,8-PeCDF	0.5	0.5	PCB 157	0.0005	0.0005
1,2,3,4,7,8-HxCDF	0.1	0.1	PCB 167	0.00001	0.00001
1,2,3,6,7,8-HxCDF	0.1	0.1	PCB 170	0.0001	-
2,3,4,6,7,8-HxCDF	0.1	0.1	PCB 180	0.00001	-
1,2,3,7,8,9-HxCDF	0.1	0.1	PCB 189	0.0001	0.0001
1,2,3,4,6,7,8.HpCDF	0.01	0.01			
1,2,3,4,7,8,9-HpCDF	0.01	0.01			
OCDF	0.001	0.0001			

Developmental toxicity of PCBs (and possibly of PCDFs), have been demonstrated with Yusho and Yu-Cheng accidents, where people were exposed to high concentrations of these contaminants accidentally by consuming contaminated rice-oil (Rogan et al. 1988, Masuda 1996). PCDD/Fs have effects on developmental processes - it was noted that mineralisation defects of the first molar teeth in children were assosiated with high, breast feeding derived exposure to PCDD/Fs, but not to PCBs (Alaluusua et al. 1996, Alaluusua et al. 1999). PCB exposure has been connected to neurotoxic and neurobehavioural effects as well as to alterations of thyroid hormone levels and lower birth weights (Feeley and Brower 2000). It has been claimed that fishermen's wives in Sweden gave birth to lower birth-weight children which was attributed to increased PCB exposure (Rylander et al. 1995, Rylander et al. 1996). PCDD/Fs may also act as endocrine disrupters. A remarkably low boy to girl ratio was found in families of

Seveso where the father had been exposed as a prepubertal boy to high levels of TCDD during the well known massive release of this agent that occurred in that town (Mocarelli 2000).

Guidelines and legislation

Recently a number of authorities have assessed or re-assessed risks of PCDD/Fs and PCBs. In many of the newer risk assessments, the focus has turned away from cancer risk towards developmental risks. The differences in risk assessments originate from uncertainties about dioxin toxicity. The risk assessment is based on animal studies and extrapolation over species to humans also leads to uncertainties and differences between assessments. Risk assessments often describe tolerable daily or tolerable weekly intakes (TDI or TWI, respectively).

WHO re-evaluated the risk assessment of PCDD/Fs and related compounds in a consultation held in 1998. Taking into account laboratory animal results on decreased sperm count, immune suppression, increased genital malformations, neurobehavioural effects, and endometriosis, the consensus meeting ultimately suggested a range of TDI intakes for humans (1-4 pg TEq/kg body weight (bw)) (van Leeuwen and Younes 2002). The upper bound limit should be considered as a maximal TDI while the lower bound limit represents an intake below which the intakes should ultimately decrease.

Based on the rodent studies, The Scientific Committee on Food (EU SCF) of the European Commission assessed a TWI of 14 pg WHO-TEq/kg bw for PCDD/Fs and for dioxin-like PCBs (European Commission 2001). This guideline is in line with the tolerable monthly intake (70 pg WHO-TEq/kg bw) established by the Joint FAO/WHO Expert Committee on Food Additives, JECFA (WHO/FAO 2001). The recommendation for TDI of WHO-TEq of the UK Committee on Toxicity of Chemicals in Food, Consumer Products and Environment (COT) is also in line with EU SCF and JECFA, 2 pg WHO-TEq/kg bw (COT 2001).

The recent re-evaluation by United States Environmental Protection Agency (USEPA) ended up to a TDI range of 0.001 to 0.01 pg WHO-TEq/kg bw (USEPA 2000). In its risk assessment, USEPA has considered cancer risk as the primary risk of PCDD/Fs unlike others mentioned here. The recent re-evaluations of tolerable daily intake are summarized in table 2.

Table 2.

Recent guidelines (in bold) on tolerable intakes (as pg WHO-TEq / kg bw) of PCDD/Fs and PCBs according to risk assessments by different organizations.

Organization	Year	Tolerable daily intake	Tolerable weekly intake	Tolerable monthly intake
WHO	1998	**1-4**	7-28[a]	31-124[a]
EU SCF	2001	2[a]	**14**	62[a]
JECFA	2001	2.3[a]	16[a]	**70**
COT	2001	**2**	14[a]	62[a]
USEPA	2000	**0.001-0.01**	0.007-0.07[a]	0.031-0.31[a]

[a] values calculated by dividing or multiplying by a factor of 7 or 31.

In addition to providing guidelines of intakes of PCDD/Fs and PCBs, in many countries legislative or guideline activities have been undertaken, to limit PCDD/F and PCB emissions, in order to protect humans and the environment from the impact of PCDD/Fs and PCBs (Basler 1994, Farland et al. 1994, Johansson and Ahlborg 1994, Kimura 1994, Newstead and Gemmil 1994, Gilman et al. 1995). The impact of these activities has been reflected in the declining emissions (Quaß et al. 2004).

The Council of the European Union in 2001 in a Council Directive 2001/102/EC decreed the maximum levels of PCDD/Fs in substances and products for animal nutrition (EC 2002) and there is a Council Regulation 2375/2001 setting maximum levels of PCDD/Fs in certain foodstuffs (EC 2001). With these legislative measures, the EU strives to protect its inhabitants from exposure to PCDD/Fs, since marketing of feed and foodstuffs exceeding these maximum levels is not allowed within the EU countries. Only PCDD/Fs were included in these regulations. The Commission reviewed the maximum levels by the end of 2004 and at the beginning of year 2005 made a proposal to add dioxin-like PCBs to the set of compounds. The maximum levels in force for PCDD/Fs as well as the proposed maximum levels for dioxin-like PCBs in foodstuffs are presented in table 3. From the Finnish point of view, Finland (and also Sweden) were granted a derogation from the maximum limit value for fish and fish products (EC 2001). This derogation allows these countries to permit fish, in which the maximum level is exceeded, to be sold, but prohibits the export of such fish to other EU countries. This derogation states that both Finland and Sweden must annually report to the Commission the monitoring results of the levels of PCDD/Fs in fish from the Baltic region and the measures taken to reduce the human exposure to PCDD/Fs from fish. In the proposal given early in 2005, this derogation of Finland and Sweden has been proposed to become a permanent derogation, and also the new EU countries like Estonia, Latvia, and Lithuania would have the same permanent derogation.

Table 3.

Maximum levels in certain foodstuffs, set by the Council of the European Union, of PCDD/Fs as $WHO_{PCDD/F}$-TEqs, and proposed total WHO-TEq (including PCDD/Fs and PCBs) maximum levels.

Products	$WHO_{PCDD/F}$-TEqs	Proposed for total WHO-TEq
Meat and meat products from		
- Ruminants (bovine animals, sheep)	3 pg/g fat	4.5 pg/g fat
- Poultry and farmed game	2 pg/g fat	4 pg/g fat
- Pigs	1 pg/g fat	1.5 pg/g fat
Liver and derived products	6 pg/g fat	12 pg/g fat
Muscle meat of fish and fishery products	4 pg/g fresh weight (fw)	8 pg/g fresh weight (fw)
Milk and milk products, including butter fat	3 pg/g fat	6 pg/g fat
Hen eggs and egg products	3 pg/g fat	6 pg/g fat
Oils and fats		
- Animal fat		
from ruminants	3 pg/g fat	4.5 pg/g fat
from poultry and farmed game	2 pg/g fat	4 pg/g fat
from pigs	1 pg/g fat	1.5 pg/g fat
from mixed animal fat	2 pg/g fat	3 pg/g fat
- Vegetable oil	0.75 pg/g fat	1.5 pg/g fat
- Fish oil intended for human consumption	2 pg/g fat	10 pg/g fat

Human intake of PCDD/Fs and PCBs

Food intake represents the main route of human exposure to PCDD/Fs and PCBs with a contribution of more than 90% of the total exposure and of this dietary exposure, 80% originates from food of animal origin (Dougherty et al. 2000, Parzefall 2002).

It is a challenging task to compare the dietary exposure of populations to PCDD/Fs and PCBs in different countries. PCDD/F and PCB dietary intake assessment studies can include different amounts of food items and food categories analysed for contaminants, and also different methods are used for assessing subjects' food consumption habits. Usually all seventeen 2,3,7,8-chlorine substituted PCDD/F congeners are measured, but with PCBs, the situation is not so clear. In some studies, only non-*ortho*-PCBs have been measured, while others include also mono-*ortho*-PCBs (dioxin-like PCBs) and a set of other PCBs. Since dioxin-like PCBs play an important role in the total TEq in food samples, especially of animal and fish origin (Alcock et al. 1998), they should not be ignored when assessing total intake of these organic pollutants. In addition to these differences, usage of lower bound (where non-detected congeners are designated as nil), medium bound (where non-detected congeners are designated as half of LOQs) or upper bound (where non-detected congeners are designated as LOQs) concentrations of PCDD/Fs and PCBs may have a major impact on the final estimated exposure levels or on assessment of sources of PCDD/Fs and PCBs in a study population.

Table 4 lists the most recent average adult daily intakes of $WHO_{PCDD/F}$-TEqs and WHO_{PCB}-TEqs in various countries along with contributions of different food groups to the PCDD/F or PCB intake.

Daily intakes of $WHO_{PCDD/F}$-TEq (including lower, medium, and upper bound results) in Western Europe ranged between 21 and 97 pg or from 0.3 to 1.45 pg $WHO_{PCDD/F}$-TEq/kg bw. On average in Western Europe, the daily intake was 59 pg or 0.86 pg $WHO_{PCDD/F}$-TEq/kg bw. The difference between lower and upper bound intake estimates can range from 25% to 40% (Becher et al. 1998, FSA 2003). The contribution (from 24% to 44%) of dairy, meat, and egg products on daily intake of PCDD/Fs has been much larger than the contribution from fish (6%-17%) in countries where the per capita consumption of fish is the lowest. Such countries in Europe are Germany, UK, Belgium, and the Netherlands (Welch et al. 2002, EC 2004). In countries, such as Sweden, France, Norway, and Spain, where fish consumption is higher, the contribution of fish and fish products has dominated, ranging from 30% to 43%.

Daily intakes of WHO_{PCB}-TEqs in Western Europe were comparable to PCDD/F intakes, from 35 to 145 pg or 0.4 to 2.1 pg WHO_{PCB}-TEq/kg bw. On average, the daily intake of PCBs was 74 pg or 0.84 pg WHO_{PCB}-TEq/kg bw. There is much less data on PCBs, but the contribution of different food groups to PCB intake seems to be rather similar in central Europe, but in Norway and Sweden, the contribution of fish and fish products to PCB intake was 45% and 51%, respectively.

On average, the total daily intake of $WHO_{PCDD/F}$-TEq and WHO_{PCB}-TEq (1.7 pg WHO-TEq/kg bw) is well below the upper range of WHO guideline on tolerable daily intake, which was the immediate goal of WHO when it set these guidelines (van Leeuwen and Younes 2002). The corresponding guideline by EU SCF (2 pg WHO-TEq/kg bw/day) is quite close to the current average intake in Western European countries (EU SCF). The Netherlands, UK, and Sweden have provided estimates about how large a percentage of their populations is exceeding the EU SCF guideline for daily intake of WHO-TEq. In the Netherlands this value is 8% of the whole population, in UK it is 1.1% of the adult population, whereas in Sweden as much as 12% of the adult population exhibit daily intakes exceeding the EU SCF guideline (Freijer et al. 2001, FSA report 38/03, Lind et al. 2002).

From the USA there are two studies giving quite different, but still comparable to European, estimates for $WHO_{PCDD/F}$-TEq medium bound daily intake, 37 and 108 pg (or 0.53 and 1.73 pg $WHO_{PCDD/F}$-TEq/kg bw) (Schecter et al. 2001, South et al. 2004). The reported daily intake of WHO_{PCB}-TEq (Schecter et al. 2001) corresponded to the lower end of PCB intake in Western Europe. South et al. (2004) have reported daily intake estimates of $WHO_{PCDD/F}$-TEq in

lower, medium, and upper bound concentrations and the upper bound intake was a drastic 2.7 times higher than the lower bound intake estimate. Also the contributions of some of the foods to the intake changed considerably when the upper bound method instead of the lower bound method was used for the intake estimation. The contribution of meat and eggs was in lower bound method 50% and the corresponding contribution of food group "Others" was 27%. When moving to the intake calculated with upper bound method, the respective contributions switched to 27% and 58%.

There are recent intake studies available from China, Korea, Japan, and Taiwan. Daily intakes of $WHO_{PCDD/F}$-TEq (including lower, medium, and upper bound results) ranged from 21 to 82 pg/day or from 0.32 to 1.64 pg $WHO_{PCDD/F}$-TEq/kg bw/day. In Japan, Korea, and Taiwan, the consumption of fish is high and also the contribution of fish to total intake of PCDD/Fs and PCBs was high in these countries, although in the study from Korea the group "Others" contributed by 51% to the daily intake of PCDD/Fs. In China, where the consumption of fish is lower (EC 2004) the dominating source of PCDD/Fs was the food group "meat and eggs".

The lowest intake estimations have been published from New Zealand, where the lower bound intake of PCDD/Fs was 3.8 pg $WHO_{PCDD/F}$-TEq /day (or 0.047 pg $WHO_{PCDD/F}$-TEq/kg bw/day) and the intake of PCBs 7.8 pg WHO_{PCB}-TEq /day (or 0.098 pg WHO_{PCB}-TEq/kg bw/day). The medium bound intake estimates for PCDD/Fs were almost four times higher and for PCBs 1.5 times higher than the lower bound estimates, being 0.18 pg $WHO_{PCDD/F}$-TEq/kg bw/day and 0.15 pg WHO_{PCB}-TEq/kg bw/day, respectively.

Congeners contributing the most to the $WHO_{PCDD/F}$-TEq intake profile have been reported to be 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, 2,3,4,7,8-PeCDF, and 1,2,3,6,7,8-HxCDD (Tsutsumi et al. 2001, Focant et al. 2002, Hsu et al. 2002, Diletti et al. 2004, Fernández et al. 2004). The higher contribution of the group "fish" to the total intake of $WHO_{PCDD/F}$-TEqs increases the contribution of 2,3,7,8-TCDF in the intake profile (Tsutsumi et al. 2001, Focant et al. 2002, Hsu et al. 2002). For WHO_{PCB}-TEqs, the main contribution to the intake has been reported to come from congener PCB 126 (Tsutsumi et al. 2001, Fernández et al. 2004), but studies including all relevant (dioxin-like) PCBs are scarce and sometimes only non-*ortho*-PCBs have been measured.

Due to their higher food intake in relation to the body weight, children are exposed to higher PCDD/F and PCB doses than adults. In this respect, crude estimations of intake of breast-feeding infants have been performed by assuming that an infant weighing 5 kg eats 800 ml of breast milk with 3.5% fat. Using WHO-TEq concentrations in breast milk from the third round of WHO coordinated breast milk studies (Leeuwen and Malisch 2002) from figure 3, the daily

intake of an infant will vary from 32 to over 200 pg WHO-TEq/kg bw. This range is about 20 to 100 times the TDI suggested by EU SCF. In the USA the intake of PCDD/Fs of breastfeeding infants (0-1 years) was about 20 times higher than that of general adult population (see table 4), (Schecter et al. 2001).

Patandin et al. (1999) concluded that breast feeding infants were more exposed to total TEq by a factor of 50, compared to young adults (20-25 years). For children aged 1-5, the factor was 3, for 6-10 years old children the factor was 2 and for children at ages 10-20 it was 1.5; these factors have been verified in the following studies. In Tarragona, Catalonia, Spain the daily intake of PCDD/Fs per bw of children, aged 4-9 years, was about twice (2.1 pg $WHO_{PCDD/F}$-TEq/kg bw/day) the intake of adults (Bocio and Domingo 2005). In Germany, the daily intake of PCDD/Fs of children in the age range 14-47 months was on an average 1.6 pg I-TEq/kg bw/day, ranging from 0.68 to 5.4 pg I-TEq/kg bw/day (Wittsiepe et al. 2001). In the USA, the intake of chilren aged 1-11 years had an intake of PCDD/Fs and PCBs that was about 2.5-3 times higher than the intake of adult population, see table 4 (Schecter et al. 2001, South et al. 2004). In UK, in schoolchildren aged from 4 to 14 years the daily intakes were on average 0.67 and 0.7 pg WHO-TEq/kg bw/day for PCDD/Fs and PCBs, respectively. This was about 1.5 times the intake of the adult UK population. For toddlers in the age range 1.5 to 4 years, the intakes were about twice the adult intakes (FSA 2001). In the Netherlands the intake of PCDD/Fs and PCBs in two year old children was 2.5 times the corresponding intake of the general adult population, and intakes of 10 years old children were 1.4 times the intake of the adult population (Freijer et al. 2001).

Time-trend of human intake of PCDD/Fs and PCBs

Figure 2 illustrates the time-trends of $WHO_{PCDD/F}$-TEq and WHO_{PCB}-TEq intake in the adult UK population between 1982 and 2001, and in the general population of the Netherlands between 1978 and 1999. In twenty years, the intakes of PCDD/Fs and PCBs in UK declined by 90% and 80% (4.8% and 4.3% annually), respectively (FSA 2001), and in the Netherlands by 85 and 88% (4% and 4.2% annually), respectively (Liem and Theelen 1997, Freijer et al. 2001).

The decrease was most dramatic in the period from the end of 1970s till the beginning of 1990s and has been levelling off during the past 9 years in both contaminant groups in the Netherlands and in WHO_{PCB}-TEq in the UK. The decrease of $WHO_{PCDD/F}$-TEq has been steeper in the UK than the decline of WHO_{PCB}-TEq and there has been no obvious levelling off in the decline. As a result of this the contribution of PCBs to the intake has risen over the reporting

period in the UK, representing 36% in 1982 and 55% in 2001. This indicates that restrictions placed on industrial emissions have worked more efficiently for PCDD/Fs than for PCBs in UK, which might be explained by the fact that the sources are more diffuse due to the abundant use of PCBs in many kinds of applications. During the same time period, from 1978 to 1999 in the Netherlands, marker PCB (marker PCBs include congeners PCB 28, 52, 101, 118, 138, 153, and 180) intakes have also declined by 93%, being 83 ng/kg bw/day in 1978, 39 ng/kg bw/day in 1984, 10 ng/kg bw/day in 1994, and 5.6 ng/kg bw/day in 1999 (Bakker et al. 2003). In Germany, the PCDD/F intake has declined by 68% from 1989 to 1999 (from 2.3 to 0.73 pg I-TEq/kg bw/day) (Vieth et al. 2000), which is quite similar to the corresponding trends of PCDD/F intake in UK and the Netherlands. In Sweden, intakes of PCDD/Fs at the beginning of 1990s were three times as high as those measured in 1999 and the intake of PCBs had declined by 75% (Lind et al. 2002).

In Tarragona, Catalonia, Spain the adult intake of PCDD/Fs decreased from 210 pg I-TEq/day in 1998 to 59.6 I-TEq/day (63.8 pg $WHO_{PCDD/F}$-TEq/day) in 2002. This was attributable to a reduction of PCDD/F concentrations in most foodstuffs, because of the decreasing deposits from the atmosphere, and also because dietary habits of the population has changed towards a more healthy diet including more vegetables, fruits, and dairy products (Bocio and Domingo 2005). Between the years 2000 and 2002 in Catalonia, Spain, the intake of children aged 4 to 9 years decreased by 15% annually, from 3.2 to 2.1 pg $WHO_{PCDD/F}$-TEq/kg bw/day (Llobet et al. 2003, Bocio and Domingo 2005) An annual decrease of 11% between 1995 and 1998 in the intake of PCDD/Fs (from 2.6 to 1.6 pg I-TEq/kg bw/day) of children was reported in Germany (Wittsiepe et al. 2001).

Table 4.

Average adult daily intakes of $WHO_{PCDD/F}$-TEqs and WHO_{PCB}-TEqs as pg (pg/kg bw). Contributions of different food groups to the PCDD/F intake (PCB intake). Bolded studies may include lower (0), medium (0.5 * LOQ) and/or upper bound (LOQ) estimations of daily intake.

	Daily intakes, pg (pg/kg bw)			% Contribution of foods from PCDD/Fs (PCBs)				
Country, study period	$WHO_{PCDD/F}$-TEq	WHO_{PCB}-TEq	Method	Dairy	Meat and eggs	Fish	Others[e]	Reference
Norway, 1998	**51 (0.73[c])[d]**	**86 (1.2[c])[d]**	**0**	**13 (20)**	**17 (24)**	**43 (48)**	**27 (8)**	**Becher et al. 1998**
The Netherlands, 1999[a]	45 (0.60)	46 (0.61)	0	24 (30)	26 (29)	9.6 (22)	41 (19)	Freijer et al. 2001
Belgium, 2001	65 (1.00)	68 (1.04)[b]	0	30 (25)	31 (36)	40 (40)	NA	Focant et al. 2002
United Kingdom, 2001	**21 (0.3[c])**	**28[c] (0.4)**	**0**					**FSA report 38/03**
USA, 2002	**20 (0.29[c])**		**0**	**12**	**50**	**11**	**27**	**South et al. 2004**
Japan, 2000	**45 (0.89)**	**68 (1.36)**	**0**	**7.5 (2.1)**	**19 (13)**	**67 (83)**	**6.9 (1.1)**	**Tsutsumi et al. 2001**
Taiwan, 2001	**21 (0.32)**		**0**	**13**	**25**	**63**	**NA**	**Hsu et al. 2002**
New Zealand, 1998	**3.8 (0.047)[d]**	**7.8 (0.098)[d]**	**0**	**1.5 (29)**	**2.5 (27)**	**31 (37)**	**65 (7)**	**Buckland et al. 1998**
Sweden, 1999	57 (0.79)	41 (0.56)	0.5 * LOQ	21 (12)	20 (21)	39 (51)	21 (16)	Lind et al. 2002
Germany, 1996	61 (0.88)[d]		Unknown	31	31	17	21	Malisch 1998
Germany, 1999	51 (0.73)[d]		0.5 * LOQ	39	41	11	9	Vieth et al. 2000
Italy, 1996	45 (0.74)[d]		0.5 * LOQ	26	39	35		SCOOP 2000
Spain, 2000[a]	95 (1.36)		0.5 * LOQ	27	15	30	28	Llobet et al. 2003
Spain, 2002	64 (0.91)		0.5 * LOQ	20	11	34	34	Bocio et al. 2005
USA, 2002	**37 (0.53[c])**		**0.5 * LOQ**	**11**	**34**	**7**	**49**	**South et al. 2004**
USA, 1995	108 (1.73)	38 (0.61)	0.5 * LOQ	31 (24)	36 (51)	5.7 (17)	27 (7.7)	Schecter et al. 2001
China, 2000	72 (1.20)		Unknown	16	56	29	NA	Wu et al. 2002
Korea, 1999	30 (0.51)[d]		Unknown	1	8.7	39	51	Kim et al. 2000
Japan, 2000	**82 (1.64)**	**79 (1.59)**	**0.5 * LOQ**	**6.2 (2.3)**	**12 (12)**	**37 (71)**	**45 (15)**	**Tsutsumi et al. 2001**
Taiwan, 2001	**26 (0.40)**		**0.5 * LOQ**	**15**	**32**	**53**	**NA**	**Hsu et al. 2002**
New Zealand, 1998	**15 (0.18)[d]**	**12 (0.15*)[d]**	**0.5 * LOQ**	**16 (23)**	**39 (42)**	**12 (23)**	**33 (12)**	**Buckland et al. 1998**
Norway, 1998	**85 (1.2[c])[d]**	**106 (1.5[c])[d]**	**LOQ**	**8 (17)**	**10 (31)**	**28 (45)**	**54 (7)**	**Becher et al. 1998**
United Kingdom, 2001	**28 (0.4[c])**	**35[c] (0.5)**	**LOQ**	**44 (20)**	**19 (28)**	**6.0 (31)**	**31 (20)**	**FSA report 38/03**
France, 1999	97 (1.45)[d]		LOQ	33	15	26	26	SCOOP 2000
Italy, 2003	12 (0.20)		LOQ	50	50	NA	NA	Diletti et al. 2004
Spain, 2000-2003	95 (1.36[c])	145 (2.1)	LOQ	14 (14)	58 (37)	9 (15)	18 (34)	Fernández et al. 2004
USA, 2002	**53 (0.76[c])**		**LOQ**	**10**	**27**	**5**	**58**	**South et al. 2004**
Taiwan, 2001	**32 (0.48)**		**LOQ**	**16**	**36**	**47**	**NA**	**Hsu et al. 2002**

[a] including children, [b] only non-*ortho*-PCBs, [c] average weight of 70 kg used, [d] I-TEqs or PCB-TEqs, [e] group Others may include vegetables, fruits, cereals, oils, and ready meals

NA, not analysed

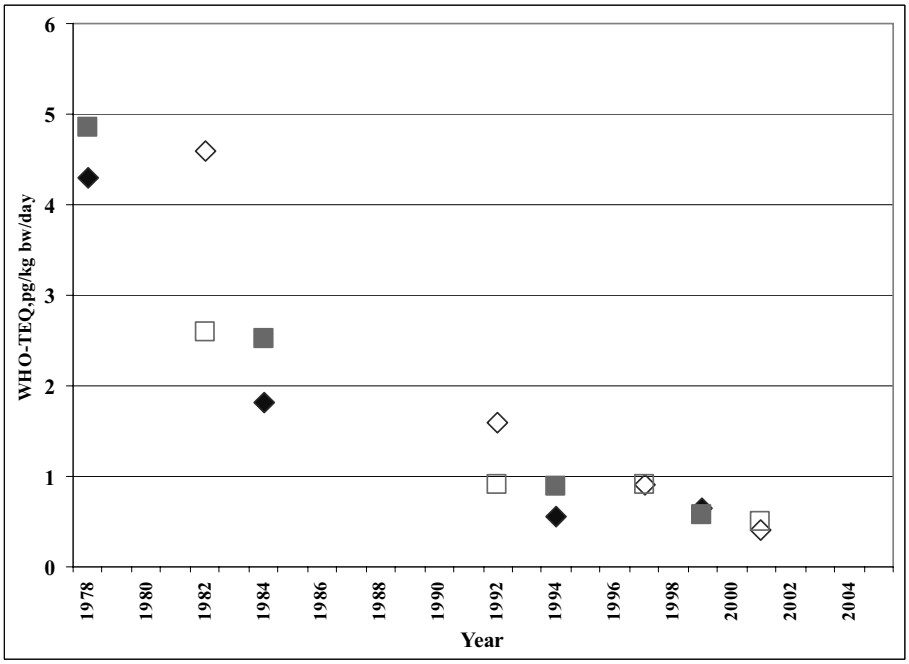

Fig 2. Time-trend of intakes of $WHO_{PCDD/F}$-TEqs (diamonds) and WHO_{PCB}-TEqs (squares) as pg/kg bw/day in the UK between 1982 and 2001 (open diamonds and squares) (MAFF 1984, 1994, 1998, FSA 2001), and in the Netherlands (closed diamonds and squares) (Liem and Theelen 1997, Freijer et al. 2001) between 1978 and 1999.

PCDD/Fs and PCBs in breast milk

Breast milk is a useful bioindicator for assessing and comparing of the exposure of populations to PCDD/Fs and PCBs since its collection is easy and non-invasive. In addition, breast milk has a high content of fat, which makes the analysis easy to perform. It is assumed that the levels of PCDD/Fs and PCBs in breast milk are similar to those in plasma (from fasting blood), serum lipid (from fasting blood), and the adipose tissue of the mother (Norén 1988).

Since 1987, WHO has coordinated esposure studies on levels of PCDD/Fs and PCBs in breast milk. By adhering to the WHO sampling protocol, the target groups should be homogenous between studies. Selection of breast milk donors in WHO studies has been based on the following criteria: the mother should be primipara, both mother and child should be healthy, the pregnancy should have been normal, the mother should breastfeed only one child

during the sampling, the mother should have lived in the area for at least 5 years, and the mother who is exclusively breastfeeding should be included. In the third round of WHO breast milk studies, the analysis of pooled breast milk samples was also performed in a single laboratory in order to avoid between laboratory variability of the results. Figure 3 depicts the most recent (2000-2002) median $WHO_{PCDD/F}$-TEq and WHO_{PCB}-TEq fat based concentrations in primiparae mothers all over the world measured in the third round of WHO breast milk study and other subsequent studies.

The $WHO_{PCDD/F}$-TEq median concentrations in Western Europe were on average 11.1 pg/g fat and ranged between 6.9 pg/g fat in Ireland and 31.5 pg/g fat in Belgium, but it must be kept in mind that concentrations in Belgium originated from breast milk samples from an area known to be contaminated with PCDD/Fs (Focant et al. 2002).

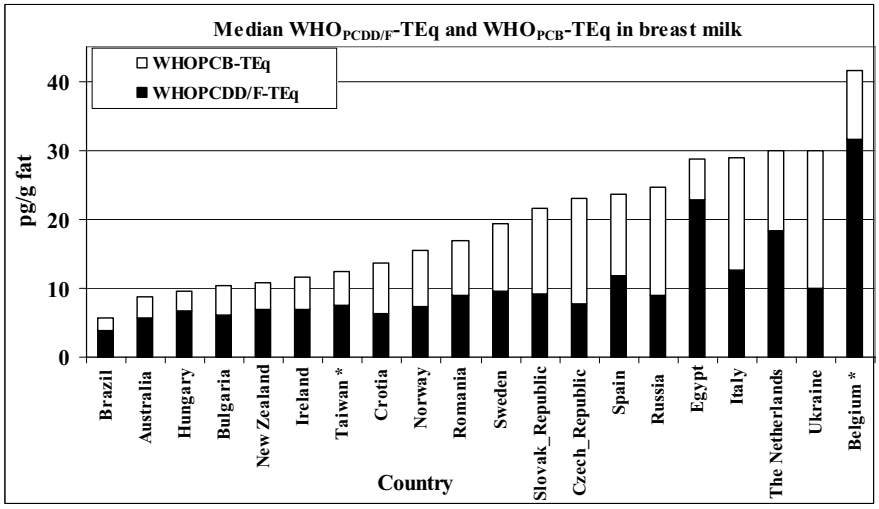

Fig 3. Median $WHO_{PCDD/F}$-TEq and WHO_{PCB}-TEq fat based concentrations in primiparae mothers in different countries measured in the third round of WHO breast milk study (Leeuwen and Malisch 2002) and studies (*) after that from Taiwan (Chao et al. 2004) and from Belgium (Focant et al. 2002).

This PCDD/F contamination of breast milk samples in Belgium was confirmed by WHO_{PCB}-TEq concentrations, which were in Belgium 10 pg/g fat corresponding to an average of WHO_{PCB}-TEq median concentrations in Western Europe, 10.3 pg/g fat (range 4.7 – 16.3 pg/g fat). The contribution of WHO_{PCB}-TEq to the total WHO-TEq in Western Europe was on average 45%.

In Eastern Europe, the $WHO_{PCDD/F}$-TEq median concentrations were lower than those found in Western Europe and ranged from 6.1 pg/g fat in Bulgaria to 10 pg/g fat in Ukraine being on average 8.0 pg/g fat. The contribution of WHO_{PCB}-TEq to the total WHO-TEq was about 10% higher in Eastern than in Western Europe (53%) and was the highest in the Czech Republic, Russia, and Ukraine (65%). This might be due to longer lasting duration of use of PCB in the Eastern European countries.

In Taiwan, Australia, and New Zealand, the median concentrations of both $WHO_{PCDD/F}$-TEq and WHO_{PCB}-TEq were comparable with each other, being on average 6.7 and 4.0 pg/g fat, respectively. The smallest median concentrations of both $WHO_{PCDD/F}$-TEq and WHO_{PCB}-TEq in the third round of WHO breast milk study were measured in Brazil, 3.9 and 1.8 pg/g fat, respectively. The high median concentration of $WHO_{PCDD/F}$-TEq (22.8 pg/g fat) in Egypt may be due to breast milk samples from PCDD/F contaminated locations, since the WHO_{PCB}-TEq concentration was quite comparable to that in other countries, being 6.0 pg/g fat.

The six marker PCB median concentrations in the third round of WHO breast milk study ranged from 16 ng/g fat in Brazil to 502 ng/g fat in the Czech Republic. Between Eastern and Western Europe there was no difference in the marker PCB median concentrations, these being 200 and 195 ng/g fat, respectively.

When comparing breast milk concentrations between studies other than the WHO studies, it must be kept in mind that there might be deviations from WHO protocols in sample collection, for example variation in the time of sampling of the milk and perhaps not all studied mothers have been primiparae. A woman's body burden of lipophilic chemicals, including PCDD/Fs and PCBs, in adipose tissue and breast milk becomes depleted over the duration of lactation. Fürst et al. (1989) reported that concentrations of PCDD/Fs in mothers breast-feeding their second child were 20-30% lower than in primiparae mothers (Fürst et al. 1989). Similar to Fürst et al. percentages of PCDD/F decrease during one breast-feeding period have been reported by Beck et al. (1994). A monthly decrease of 12% for bioaccumulating PCBs and PCDD/Fs was reported in a Swedish study (Dahl et al. 1995). One extreme example of the decrease in concentrations of PCDD/Fs and PCBs in breast milk comes from a case study, in which a mother had breast-fed her twins for 30 months. During this time period, her breast milk concentrations of I-TEQs decreased by 69% and PCB concentrations by 78% on average (Schecter et al. 1998). In addition, the age of studied mothers can vary between studies, and results may not be representative for the whole country in question. A recent example of local differences in concentrations of PCDD/F and PCB TEq is illustrated with results from the Czech

Republic. In the third round of WHO breast milk studies, the median sum of $WHO_{PCDD/F}$-TEq and WHO_{PCB}-TEq was 23 pg/g fat (range 21.8 to 39.2 pg/g fat), but in a more recent study the median TEqs ranged from 28 to 65 pg/g fat depending on the origin of the breast milk samples (Bencko et al. 2004).

In table 5 there are the most recent concentrations of $WHO_{PCDD/F}$-TEq or I-TEq of breast milk from countries, which did not provide samples to the third round of WHO breast milk study. Five of those concentrations originate from the second round of WHO coordinated breast milk studies, and those concentrations ranged from 4.3 pg I-TEq/g fat in Albania to 27.4 pg I-TEq/g fat in Belgium (Liem et al. 1996). In other studies in table 5 the $WHO_{PCDD/F}$-TEq concentration ranged from 3.1 in China to 26.4 pp/g fat in the UK (Schecter et al. 1994, Wearne et al. 1996).

Congeners contributing the most to the $WHO_{PCDD/F}$-TEq in breast milk have been reported to be 2,3,4,7,8-PeCDF, 1,2,3,7,8-PeCDD, 2,3,7,8-TCDD, and 1,2,3,6,7,8-HxCDD, while OCDD contributes the most to the PCDD/F sum concentrations. PCB 126, PCB 156, and PCB 118 are the congeners dominating in the WHO_{PCB}-TEq, while contribution to the sum of PCB congeners are dominated by PCB 153, PCB 138, PCB 180, and PCB 170 (Norén and Meironyté 2000, Focant et al. 2002, Chao et al. 2004, Bencko et al. 2004). These contributions of PCDD/F and PCB congeners are similar to those reported in intake studies for those food groups which contribute most to the intakes. Due to local contamination patterns or due to differences in dietary habits, the contributions might change to some extent.

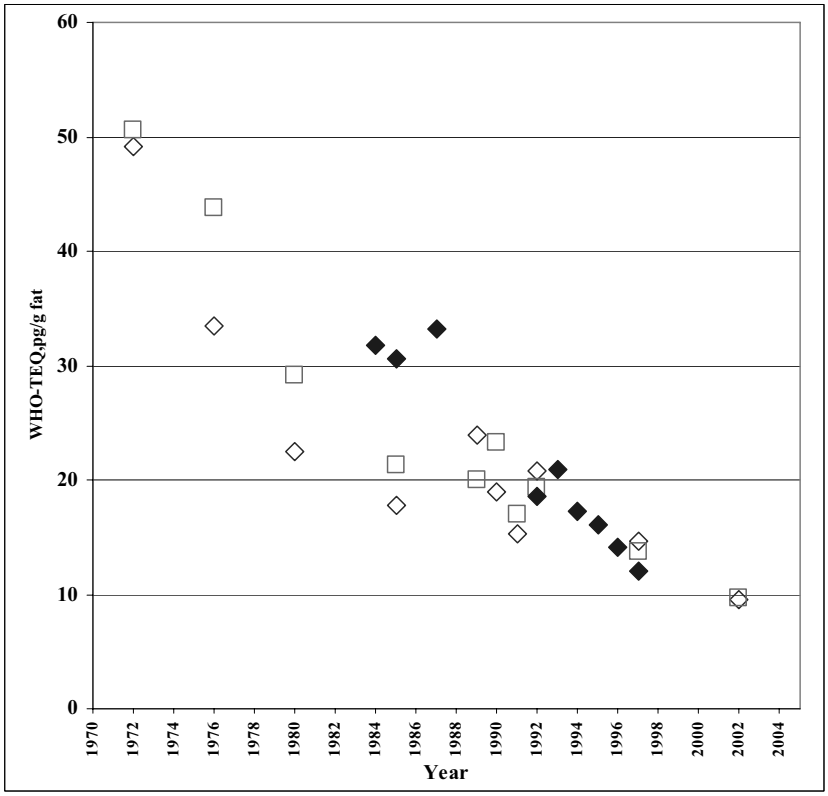

Fig 4. Time-trend of $WHO_{PCDD/F}$-TEqs (open diamonds) and WHO_{PCB}-TEqs (open squares) as pg/g in Swedish breast milk samples (Norén and Meironyté 2000, Leeuwen and Malisch 2002), and of I-TEqs in German (closed diamonds) breast milk samples. (WHO 1989, Jensen and Slorach 1991, Liem et al. 1996 ,EC 1999) between 1976 and 2002.

Time-trend of PCDD/Fs and PCBs in breast milk

Collecting longitudinally breast milk samples, as recommended by WHO, provides a way of assessing, whether control on sources of PCDD/Fs and PCBs has been effective. Similarly to the dietary intake time-trend, the levels of PCDD/Fs and PCBs have been decreasing in breast milk. Figure 4 illustrates the time-trends of $WHO_{PCDD/F}$-TEqs and WHO_{PCB}-TEqs in breast milk samples from Sweden between 1972 and 2002 (Norén and Meironyté 2000, Leeuwen and Malisch 2002), and of I-TEqs from Germany between 1984 and 1997 (WHO 1989, Jensen and Slorach 1991, Liem et al. 1996 ,EC 1999). The decrease of $WHO_{PCDD/F}$-TEq and WHO_{PCB}-TEq concentrations during the last 30 years in Sweden has been

about 80%, being annually 2.7% (Norén and Meironyté 2000). In Germany the total decrease in the given period of I-TEq was 62%, i.e. about 4.8% decline per year.

Table 5 lists the annual declines of I-TEq or $WHO_{PCDD/F}$-TEq from different countries. On average, the annual decline was 4.0% (median 4.1%) ranging from 1% in Ukraine to 6.6% in Brazil. No regional or sampling period differences were observed from those annual declines in table 5.

In Swedish breast milk samples from 1967 to 1997 the concentrations of PCBs initially increased, peaking in 1972 and have been decreasing since that time. The total decrease of PCBs from 1972 was about 70% which was annually 2.8% corresponding to the annual decrease of $WHO_{PCDD/F}$-TEq in the same breast milk samples (Norén and Meironyté 2000). Between the second and third WHO coordinated breast milk studies the concentrations of marker PCBs decreased on average by 3.9% ranging from 0.9% in Russia to 6.4% in Norway (countries participating in both studies were: Croatia, Czech Republic, Hungary, Norway, Russia, Slovakia Republic, Spain, The Netherlands, and Ukraine) (WHO 1996, Leeuwen and Malisch 2002).

Table 5.
Annual decline (%) of concentration of I-TEq or $WHO_{PCDD/F}$-TEq from different countries along with the most recent estimations of concentrations of I-TEq or $WHO_{PCDD/F}$-TEq (pg/g fat) in breast milk in those countries, which did not provide samples to the third round of WHO coordinated breast milk studies.

Country	I-TEq or $WHO_{PCDD/F}$-TEq (year)	Annual decline %	Reference
Albania	4.3[a] (1992)	-	Liem et al. 1996
Austria	11.9[a] (1992)	6 (1986-1992)	WHO 1989, Liem et al. 1996
Belgium	27.4[a] (1992)	4.8 (1986-1992)	WHO 1989, Liem et al. 1996
Brazil		6.6 (1992-2001)	Paumgartten et al. 2000, Leeuwen and Malisch 2002
Canada	16.2[a] (1992)	3 (1981-1992)	Ryan et al. 1993, Liem et al. 1996
China	3.1[b] (1994)	-	Schecter et al. 1994a
Croatia		4.7 (1992-2001)	Liem et al. 1996, Leeuwen and Malisch 2002
Czech Republic		5.9 (1992-2001)	Liem et al. 1996, Leeuwen and Malisch 2002
Denmark	15.2[a] (1992)	2.3 (1986-1992)	Jensen and Slorach 1991, Liem et al. 1996
Hungary		2.8 (1986-2001)	WHO 1989, Leeuwen and Malisch 2002
Japan	18.8[b] (1995)	4.5 (1980-1995)	Jensen and Slorach 1991, Iida et al. 1999
Kazahkstan	22.6[b] (1996)	-	Petreas et al. 1996
Lithuania	16.7[b] (1993)	-	Becher et al. 1995
The Netherlands		5.1 (1985-2001)	Jensen and Slorach 1991, Leeuwen and Malisch 2002
New Zealand		4.1 (1986-2001)	WHO 1989, Leeuwen and Malisch 2002
Norway		4.1 (1986-2001)	WHO 1989, Leeuwen and Malisch 2002
Russia		3.5 (1992-2001)	Liem et al. 1996, Leeuwen and Malisch 2002
Slovakia		1.2 (1992-2001)	Liem et al. 1996, Leeuwen and Malisch 2002
Spain		2.9 (1990-2001)	Gonzalez et al. 1996, Leeuwen and Malisch 2002
UK	26.4[b] (1993)	5.5 (1987-1993)	Wearne et al. 1996
Ukraine		1 (1992-2001)	Liem et al. 1996, Leeuwen and Malisch 2002
USA	18.8[b] (1990)	-	Schecter et al. 1990

[a] I-TEq, [b] $WHO_{PCDD/F}$-TEq

PCDD/Fs and PCBs in adipose tissue and serum

Table 6 lists recent average human adipose tissue and serum fat concentrations of sum of PCDD/Fs, $WHO_{PCDD/F}$-TEq, PCB 126, PCB 153, marker PCBs, and WHO_{PCB}-TEq from different countries. These studies do not include occupationally or accidentally exposed subjects but are based on random sampling of the general population. The high correlation of 2,3,7,8-TCDD and other PCDD/F congeners between serum and adipose tissue of the same individual makes it possible to use both matrices if one wishes to assess the human body burden of PCDD/Fs (Patterson et al. 1988, Schecter et al. 1991). PCBs are included in table 6 irrespective of the claim by Whitcomp et al. (2005) who stated that using serum concentrations of OCs for exposure assessment of young women may result in divergence from the use of adipose tissue concentrations, at least if linear dependency between matrixes is assumed. These workers reported linear correlation coefficient ($r > 0.6$) between lipid adjusted serum and fat concentrations of PCB congeners: 138, 153, 180, 188, 194, and 206.

Human exposure to PCDD/Fs and PCBs starts already before birth during pregnancy, since PCDD/Fs and PCBs are transferred from mother to fetus via the placenta. Placenta PCDD/F concentrations corresponded in the study of Abraham et al. (1998) with the concentrations in breast milk, but concentrations of PCBs in placenta were on average only 30% of the corresponding breast milk concentrations. In a study from Åland, Finland, the concentrations of PCB congeners PCB 118, PCB 138, PCB 153, and PCB 180 were two to three fold lower in cord blood than in venous blood of delivering mothers (Hagmar et al. 1998). A similar result was reported in a Swedish study where the sum concentration of 15 PCB congeners in cord blood plasma was 41% lower than the corresponding concentration in maternal blood plasma (Meironytė Guvenius et al. 2003). Analysis of PCDD/Fs and PCBs from infants and children have rarely been performed due to obvious ethical reasons. From Germany there exist two studies of adipose tissue levels of PCDD/Fs in infants; in the first one, infants aged 3.8-23 months exhibited a concentration range from 2.1 to 22 pg I-TEq/g fat (Beck et al. 1994), and in the second study of 3 stillborns and 17 infants (0.43-44 weeks of age) had PCDD/F concentrations from 1.55 to 29.6 pg I-TEq/g fat, in their adipose tissues (Kreuzer et al. 1997). In Dallas, Texas, the concentrations of $WHO_{PCDD/F}$-TEq in the whole blood in children from 0-14 years ranged between 4.12 and 5.58 pg/g fat and this was about 20% of the concentration measured in the whole blood of adults in the same area (22.3 pg/g fat) (Schecter et al. 2003).

Many studies have reported that concentrations of PCDD/Fs and PCBs increase with the age of the subject (Päpke 1998, Sjödin et al. 2000, Covaci et al. 2002, Costabeber and

Emanuelli 2003, Wicklund Glynn et al. 2003, Harden et al. 2004, Kim et al. 2005). Therefore the concentrations listed in table 6 must be interpreted with caution since the mean age of the subjects has varied between studies. The mean concentration of $WHO_{PCDD/F}$-TEq in Europe was 29.5 pg/g fat in the study populations with a mean age of 52 years. There were three studies from Spain in which the mean age was 50 years and the mean $WHO_{PCDD/F}$-TEq concentration was 20.6 pg/g fat (Wingfors et al. 2000, Bocio et al. 2004). This was a very similar concentration to that measured in Germany (18.8 pg $WHO_{PCDD/F}$-TEq/g fat) five years earlier in a population with a mean age of only 37 years (Päpke 1998). In older populations from Sweden, Belgium, and France (mean age 59.6 years) the $WHO_{PCDD/F}$-TEq concentration was 42 pg/g fat. Concentrations of the sum of PCDD/Fs are available only from five studies in Europe – they have reported a mean concentration of 777 pg/g fat (mean age 53 years).

PCB 153 is one of the most commonly measured PCB congeners in all studies due to its abundance in all kinds of matrices. In recent human adipose and serum fat samples from Europe, the average concentration of PCB 153 was 232 ng/g fat (mean age of the study populations was 53 years) (Table 6). The marker PCB concentrations ranged from 389 to 855 ng/g fat with a mean value of 606 ng/g fat. The WHO_{PCB}-TEq mean concentration is based only on three reported concentrations from Sweden, Belgium, and Spain, and the average concentration was 37.8 pg/g fat (Wingfors et al. 2000, Koppen et al. 2002).

From the USA, there are recent reports of PCDD/F concentrations from the year 2002 with the $WHO_{PCDD/F}$-TEq concentration of 19.3 pg/g fat, and the sum of PCDD/F concentrations of 505 pg/g fat (Schecter et al. 2003).

The mean concentrations in the Far-East, in India, Korea, and Japan, were lower than the concentrations found in Europe, 13.0 pg $WHO_{PCDD/F}$-TEq/g fat, sum of PCDD/Fs 511 pg/g fat, and 13.1 pg WHO_{PCB}-TEq/g fat, but the study populations were also younger than the populations examined in Europe, table 6 (Kumar et al. 2001, Choi et al. 2002, Kim et al. 2005).

The lowest human adipose or serum fat concentrations of PCDD/Fs and PCBs so far reported originate from Australia in 2002 (Harden et al. 2002). The $WHO_{PCDD/F}$-TEq and WHO_{PCB}-TEq concentrations were 6.9 and 4.0 pg/g fat, respectively.

The highly PCB contaminated human adipose or serum fat tissues in Uelen, Russia and Greenland Inuit populations were due to consumption of meat and blubber of marine mammals which themselves had a high PCB body burden (Sandanger et al. 2003, Dewailly et al. 1999).

The main exposure of the general population comes from food, especially food of animal origin, but still no relation of adipose tissue PCDD/F concentrations and daily dietary dioxin

intake or tissue PCB concentrations and alimentary habits have been found in France and Spain, respectively. (Arfi et al. 2001, Costabeber and Emanuelli 2003). On the other hand, it has been shown that consumption of Baltic Sea fish leads to a high contribution of the dioxin congener 2,3,4,7,8-PeCDF (Svensson et al. 1991), and also PCB concentrations in serum have been reported to correlate positively with consumption of fatty fish in the Baltic Sea region (Grimvall et al. 1997, Sjödin et al. 2000, Wicklund Glynn et al. 2003).

As the majority of human exposure to PCDD/Fs originates from fat of animal origin, Welge et al. (1993) postulated that vegetarians should have lower body burdens of PCDD/Fs compared to non-vegetarians. This presumption was not confirmed, since I-TEq concentrations in the blood of both groups were very similar, around 33 pg/g fat. The similarities of PCDD/F concentrations in vegetarians and non-vegetarians were explained by the higher consumption of dairy products by vegetarians, which would compensate for part of the PCDD/F intake originating from meat and fish in non-vegetarians (Welge et al. 1993).

The congeners contributing the most to the sum of PCDD/Fs and $WHO_{PCDD/F}$-TEqs in adipose tissue or serum fat are depicted in figure 5. The contribution of congeners OCDD, 1,2,3,4,6,7,8-HpCDD, 1,2,3,6,7,8-HxCDD, and 2,3,4,7,8-PeCDF to the sum of PCDD/Fs was on an average 92%. There were little differences between depicted areas in these contributions to the sum of PCDD/Fs. Four congeners, 2,3,4,7,8-PeCDF, 1,2,3,7,8-PeCDD, and 1,2,3,6,7,8-HxCDD, and 2,3,7,8-TCDD accounted for on average 87% of the $WHO_{PCDD/F}$-TEq profile in adipose tissue or serum fat samples in the depicted areas in figure 5. The contribution of these four congeners was largest in Sweden and lowest in the USA, 92% and 83%, respectively. The consumption of Baltic fatty fish might explain why congener 2,3,4,7,8-PeCDF contributed more to the $WHO_{PCDD/F}$-TEq profile in the average Swedish population compared to other areas, as it does in Swedish fishermen (Svensson et al. 1991). In Europe, the congener 1,2,3,7,8-PeCDD was the most prevalent congener in $WHO_{PCDD/F}$-TEq profile followed by 2,3,4,7,8-PeCDF, which originates also from milk products in addition to fish products. A quite different profile has been reported from the USA, where the contribution of 2,3,4,7,8-PeCDF to $WHO_{PCDD/F}$-TEq profile was only 15% instead of about 30% in the Europe and Far-East and 40% in Sweden.

Table 6.

Recent adipose tissue and serum fat concentrations of sum of PCDD/Fs, $WHO_{PCDD/F}$-TEq, PCB 126, PCB 153, marker PCBs, and $WHO_{PCDD/F}$-TEq from different countries.

Country, study period	Age of subjects, mean and range	Sum of PCDD/Fs pg/g fat	$WHO_{PCDD/F}$-TEq pg/g fat	PCB 126 pg/g fat	PCB 153 ng/g fat	Marker PCB ng/g fat	WHO_{PCB}-TEq Pg/g fat	Reference
Finland, mid 1990s	30 (19-40)[a]	-	-	-	56	-	-	Hagmar et al. 1998
Sweden, unknown	68	804	32.8	180	300	778	44.3	Wingfors et al. 2000
Sweden, late 1990s	63[b](40-75)	-	-	-	296	675		Wicklund Glynn et al. 2000
Sweden, 1996-1997	63[a] (54-75)	-	-	-	223	-	-	Wicklund Glynn et al. 2003
Sweden, 2001	52[b] (33-79)	-	-	-	241	-	-	Wallin et al. 2003
Latvia, 1993	48[b] (24-79)	-	-	-	403	-	-	Sjödin et al. 2000
Germany, 1996*	37 (18-71)	403	18.8					Päpke 1998
Belgium, 1999	58[a] (50-65)	999	48	102	168	389	23.7	Koppen et al. 2002
Belgium, 2000	47 (19-77)	-	-	-	211	504	-	Covaci et al. 2002
France, 1999*	53 (30-94)	497	45.1	-	-	-	-	Arfi et al. 2001
Spain, unknown	51	1180	33.0	220	300	855	45.5	Wingfors et al. 2000
Spain, 1996-1997	51 (15-87)	-	-	-	121	432	-	Costabeber and Emanuelli 2003
Spain, 2002	58 (19-94)	-	11.1	-	-	-	-	Bocio et al. 2004
Spain, 2003	41 (19-62)	-	17.8	-	-	-	-	Bocio et al. 2004
Uelen/Russia, 2001	37 (20-70)	-	-	1200	744	1410	-	Sandanger et al. 2003
Greenland, 1992-1994	60	-	-	-	1689	4242	-	Dewailly et al. 1999
USA, 2002	Adults	505	19.3	-	-	-	-	Schecter et al. 2003
India, 2000	(20-69)	550	14.4	125	-	-	14.4	Kumar et al. 2001
Korea, 2001*	43 (21-63)	813	12.8	-	64	-	9.6	Kim et al. 2005
Japan, 2000	(40-50)	171	11.9	72	-	-	15.3	Choi et al. 2002
Australia, 2002	(<16->60)	-	6.9	18.6	-	-	4	Harden et al. 2004

[a] only women, [b] only men, * results originally as I-TEqs and here re-calculated as $WHO_{PCDD/F}$-TEqs

In the USA congeners, originating mostly from meat, 1,2,3,7,8-PeCDD, 2,3,7,8-TCDD, and 1,2,3,6,7,8-HxCDD contributed more to the $WHO_{PCDD/F}$-TEq profile than 2,3,4,7,8-PeCDF (Wingfors et al. 2000, Päpke 1998, Koppen et al. 2002, Arfi et al. 2001, Schecter et al. 2003, Kumar et al. 2001, Choi et al. 2002, Kim et al. 2005). Similar patterns to the $WHO_{PCDD/F}$-TEq profile as that found in USA have been reported also from Canada (Schecter et al. 1994b).

Figure 6 depicts the relative contribution of selected PCB congeners to the sum of these particular PCBs and to the WHO_{PCB}-TEq. There were minor differences between the Swedish and the average European profiles of PCBs. Congener PCB 153 was the main contributor in the adipose tissue or serum samples in all areas followed by PCB 138 and PCB 180. The contribution of PCB 153 was clearly more dominant in samples from the Inuit population when compared to other areas (see Fig 6, A, Uelen/Russia). On the other hand, the contributions of PCB 180 and PCB 170 were lower in Inuit population than in Swedish or European populations. These differences in PCB contributions between northern and southern populations might be due to different occurrence of various PCB congeners in the foodstuffs consumed by these populations. Eighty percent or more of the WHO_{PCB}-TEq concentration has been reported to be due to a contribution of congeners PCB 126, PCB 156, and PCB 118 (Fig 6, B). Again the difference between Sweden and Southern Europe was not so evident, but in the Far East, the contribution of PCB 126 dominated the WHO_{PCB}-TEq profile.

Time-trend of PCDD/Fs and PCBs in adipose tissue and serum

Similar declining trends in human adipose and serum fat concentrations as those found in breast milk have been reported from several countries. Figure 7 illustrates the time-trends of PCDD/F-TEqs between 1980 and 2002 from Germany, USA, and Japan (Päpke 1998, Schecter et al. 2003, Choi et al. 2002). The annual decline of PCDD/F- TEq in Germany and Japan has been about 4%, which is very close to the decline in breast milk in these countries. In the USA, the decline of $WHO_{PCDD/F}$-TEq is not so clear with increasing concentrations reported in studies from the years 1996 and 2002. Nevertheless between mid 1990s and the early 2000s the decline of $WHO_{PCDD/F}$-TEq has been about 15% (Schecter et al. 2003).

Declining time-trends of PCB concentrations in adipose tissue or in serum fat in the USA are more clear than the corresponding PCDD/F trends. The concentrations of congener PCB 153 declined from 1985-1989 to 2000-2002 by 61% (about 4% annually) from 90 ng/g

serum fat to 35 ng/g (Sjödin et al. 2004). Another study from the USA has indicated that the concentration of PCB congener 126 declined by about 88% between 1985 and 1995 (Aylward et al. 2002). Also in a study of Swedish men, the decline of serum PCB 153 concentration was on average 34% (3% annually) during the time period 1991-2001 (Wallin et al. 2003), which is similar to $WHO_{PCDD/F}$-TEq decline reported in Swedish breast milk (Norén and Meironyté 2000). In Tarragona, Spain, there was an average 41% reduction of $WHO_{PCDD/F}$-TEq in human plasma samples (10% annually); in the adipose tissue samples the reduction was 70% (18% annually), both of which follow the decline of PCDD/F daily intake in the same district (Bocio et al. 2004).

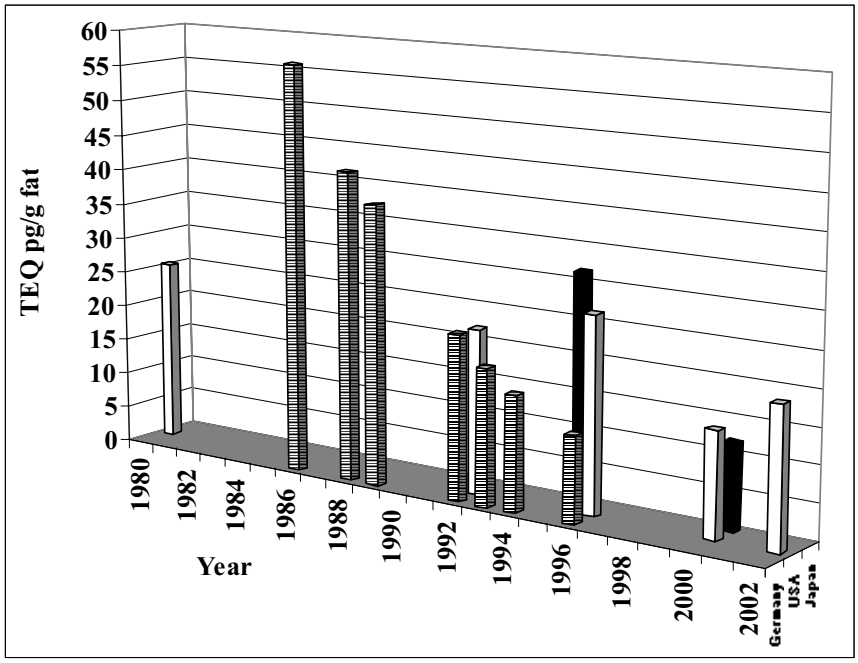

Fig 7. Time-trend in 1980-2002 of human adipose tissue or serum fat PCDD/F-TEq concentrations in Germany (striped bars) (Päpke 1998), in the USA (white bars) (Schecter et al. 2003), and in Japan (black bars) (Choi et al. 2002).

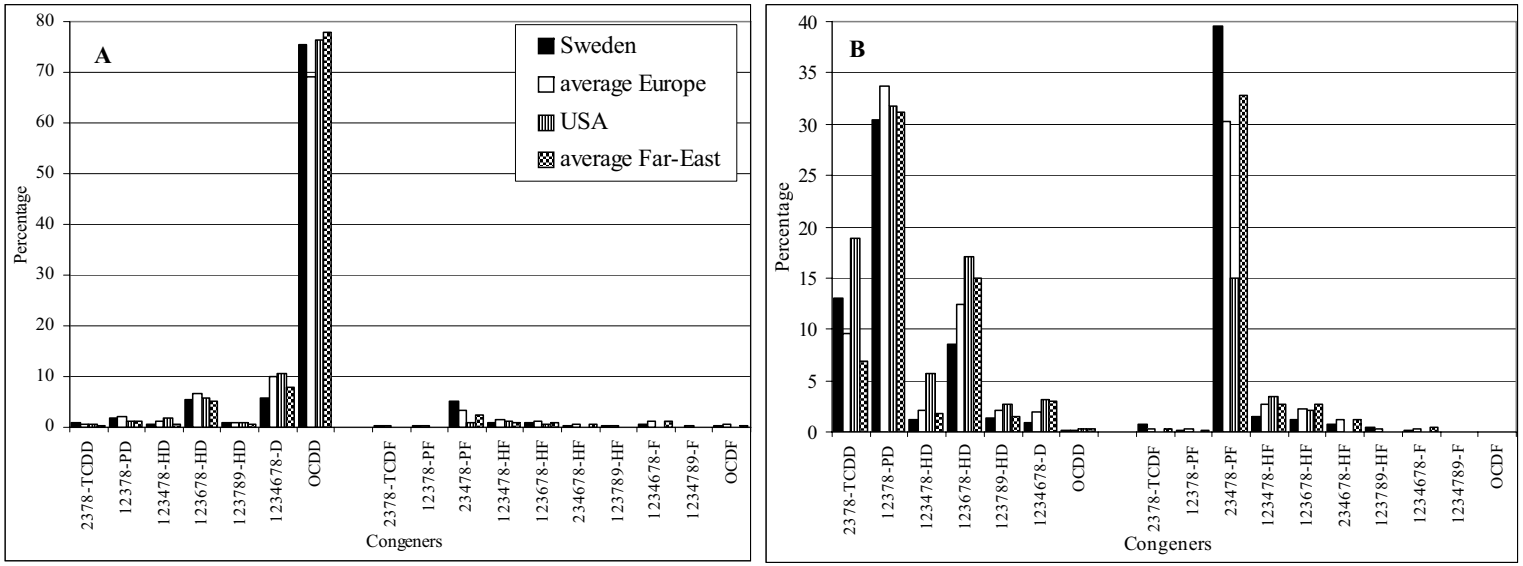

Fig 5. Adipose tissue or serum fat congener profile of (A) PCDD/Fs and (B) $WHO_{PCDD/F}$-TEqs in Sweden, Europe, USA, and Far-East. Calculated from the data by: Wingfors et al. 2000, Päpke 1998, Koppen et al. 2002, Arfi et al. 2001, Schecter et al. 2003, Kumar et al. 2001, Choi et al. 2002, Kim et al. 2005.

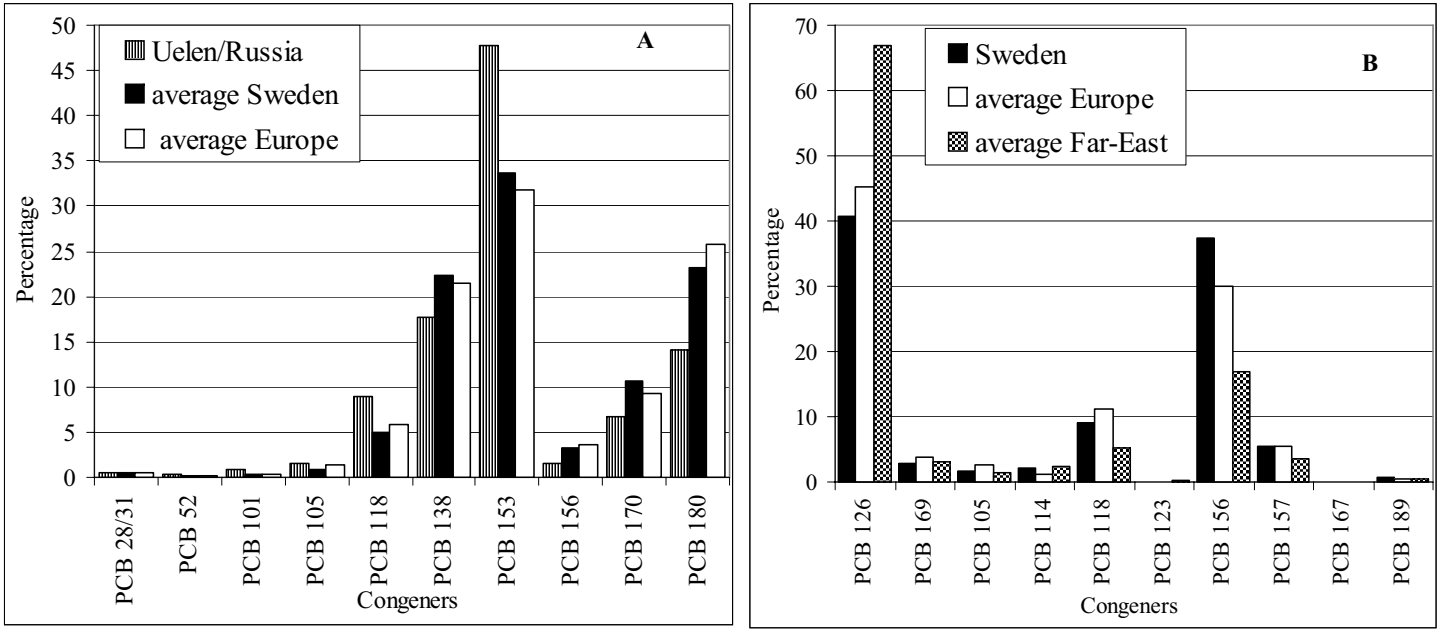

Fig 6. Adipose tissue or serum fat congener profile of (A) certain PCBs and (B) WHO_{PCB}-TEqs in Uelen/Russia, Sweden, Europe, and Far-East. Calculated from the data by: Wingfors et al. 2000, Wicklund Glynn et al. 2000, Wicklund Glynn et al. 2003, Sjödin et al. 2000, Grimvall et al. 1997, Covaci et al. 2002, Koppen et al. 2002, Costabeber and Emanuelli 2003, Sandanger et al. 2003, Kumar et al. 2001, Choi et al. 2002.

2. AIMS OF THE STUDY

The aims of this study were to:

1. Assess the average intake of PCDD/Fs and PCBs of the general population in Finland, with the emphasis on estimating the contribution of different foods to the intake.
2. Analyse the average adipose tissue concentrations of PCDD/Fs and PCBs in the general population in Finland and to determine whether differences in concentrations occur in three geographical areas.
3. Determine the concentrations of PCDD/Fs and PCBs in breast milk in two areas in Finland and to evaluate temporal changes in the concentrations in breast milk.
4. Compare the intake of PCDD/Fs and PCBs and occurrence of these contaminants in human tissues to EU and other countries.
5. Study, if there is a population in Finland, which experience high exposure to PCDD/Fs and PCBs.
6. Study the differences in the PCDD/F and PCB congener profiles between the exposure (diet) and human tissues in order to evaluate the bioaccumulation efficiencies of different congeners.

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CHAPTER 2

DIETARY INTAKES OF POLYCHLORINATED DIBENZO-*P*-DIOXINS, DIBENZOFURANS AND POLYCHLORINATED BIPHENYLS IN FINLAND

H. Kiviranta[1*], A. Hallikainen[2], M.-L. Ovaskainen[3], J. Kumpulainen[4], and T. Vartiainen[1.,5]

[1]Division of Environmental Health, National Public Health Institute, PO Box 95, FIN-70701 Kuopio, Finland

[2]National Food Agency, PO Box 28, FIN-00581 Helsinki, Finland

[3]Department of Nutrition, National Public Health Institute, Mannerheimintie 166, FIN-00300 Helsinki, Finland

[4]Laboratory of Food Chemistry, Agricultural Research Centre of Finland, FIN-31600 Jokioinen, Finland

[5]Department of Environmental Sciences, University of Kuopio, PO Box 1627, FIN-70211 Kuopio, Finland

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1. ABSTRACT

Samples of cow milk, pork, beef, eggs, rainbow trout, flours and vegetables were analyzed for 17 polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/F) and 36 polychlorinated biphenyls (PCB). Daily dietary intake of PCDD/Fs as toxic equivalent (I-TEq) and PCBs (PCB-TEq) was assessed using food consumption data from a 24-hour dietary recall study for 2862 Finnish adults. The calculated intake of PCDD/F was 46 pg I-TEq day^{-1}. The current level was about half of the earlier estimation of intake in Finland made in 1992. The assessed PCB intake was 53 pg PCB-TEq day^{-1}. Thus, the total intake of PCDD/Fs and PCBs was 100 pg TEq day^{-1} (1.3 pg TEq kg^{-1} b.w. day^{-1}), which is within the range of tolerable daily intake (TDI) proposed by the WHO (1-4 pg TEq kg^{-1} b.w. day^{-1}).

2. INTRODUCTION

In spring 1999, polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs) were discovered in Belgian chicken and eggs. Subsequently this contamination event was expanded to cover all foods of animal origin in Belgium. It was subsequently reported that the point introduction of a polychlorinated biphenyl (PCB) containing oil into the production of animal feed in Belgium led to a contamination of part of the food chain of animal origin (Bernard et al. 1999). Public alarm at this incident in Belgium launched or accelerated the pace of national studies into the dioxin content in foods and the intake of dioxins via the food chain in different European countries.

In Finland, the intake of PCDD/Fs was first estimated in 1992 (Hallikainen et al. 1995) using measured concentrations of PCDD/Fs in cow milk, egg, meat, Baltic herring and rainbow trout samples and a 3-day food consumption questionnaire for adults (aged from 25-64 years) in 1992 (Vartiainen et al. 1993). The authors estimated the total PCDD/F daily intake to be 95 pg Nordic toxic-equivalents (N-TEq) or 1.6 pg N-TEq kg^{-1} body weight using 60 kg as average weight of adult population. Fish and fish products accounted for 60% of the daily intake followed by milk and dairy products (31%), eggs (3%) and meat and meat products (1.4%) (Hallikainen and Vartiainen 1997).

This study reports the results of current PCDD/F and PCB concentrations in 1998-2000 for cow milk, pork, beef, eggs, rainbow trout, flours and vegetables, which combined with new food consumption data in 1997 for average adults (National Public Health Institute 1997), allowed estimation of the intake of PCDD/Fs of average adult Finnish population. In addition, for the first time, PCB-TEq intake in Finland was assessed. The contribution of each food and food group was also revised.

3. MATERIALS AND METHODS

Concentrations of PCDD/Fs and PCBs in foods

Representative samples of cow milk, pork, beef, eggs, rainbow trout, flour and vegetables were collected encompassing the Finnish food supply and analysed for PCDD/F and PCB content. Five pooled cow milk samples (three individual samples in each pool) were collected from five dairies around Finland. These dairies represented ~50% of all cow milk production in

Finland. Six pork tenderloin and five beef tenderloin samples were collected from two of the largest slaughterhouse chains in Finland covering all the major production areas and representing ~80% of the total production in Finland. The samples were pooled by production areas (5-24 individual samples in each pool) by weighing equally sized samples from each subsample. Pooled egg samples were collected from five henhouses in South-Western Finland. Each egg pool consisted of eight to nine individual eggs. Two years old rainbow trouts were collected from eight different fish farms in southern Finland. Altogether 40 individual rainbow trout samples were analysed in eight pools (five individual samples in each pool). Domestic leafy vegetables (three different kinds of lettuces and cabbage), fruit vegetables (cucumber, tomato, onion and sweet pepper) and potatoes, 200g of each individual item, were purchased from a supermarket in the province of Kuopio. Also flours (rye and wheat) in 1 kg packages were bought from a supermarket but the origin of the flour remained undetermined. All the samples were collected between 1998 and 2000.

The concentrations of 17 toxic PCDD/Fs (10 PCDF, seven PCDD) congeners of three non-*ortho* (IUPAC 77, 126, 169), eight mono-*ortho* (IUPAC 105, 114, 118, 123, 156, 157, 167, 189), and of 25 other (IUPAC 18, 28, 33, 47, 49, 51, 52, 60, 66, 74, 99, 101, 110, 122, 128, 138, 141, 153, 170, 180, 183, 187, 194, 206, 209) PCB congeners, the total sum of PCDD/Fs (ΣPCDD/F) and PCBs (ΣPCB), and toxic equivalents, I-TEqs (PCB-TEqs, for PCBs) were determined (NATO/CCMS 1988, Ahlborg et al. 1994).

All homogenized samples were spiked with 115 pg ^{13}C-labelled PCDD/F standards (seventeen 2,3,7,8-chlorinated PCDD/F congeners), with 100 pg ^{13}C-labelled non-*ortho* PCB standards (PCB 77, 126, 169), and with 960 pg ^{13}C-labelled PCB standards (PCB 30 [^{12}C-labelled], 80, 101, 105, 138, 153, 156, 180 and 194 Cambridge Isotope Laboratories). Cow milk's fat was extracted with diethyl ether-hexane, fat from eggs with diethyl ether and hexane, and fat from pork, beef and rainbow trout with toluene for 24 h using Soxhlet apparatus. The fat content was determined gravimetrically. All the samples were defatted in a silica gel column and initially purified on activated carbon column (Carbopack C, 60/80 mesh) containing Celite (Merck 2693) to separate PCDD/Fs from PCBs. Both fractions were further cleaned with an activated alumina column (Merck 1097, standardized, activity level II-III). The separated PCB fraction was further fractionated, after having been analyzed for mono- and di-*ortho* PCB congeners on another activated carbon column (without Celite) in order to separate the non-*ortho* PCBs. The quantitation was performed by selective ion recording using a VG 70-250 SE (VG Analytical, UK) mass spectrometer (resolution 10 000) equipped with a HP 6890 gas

chromatograph with fused silica capillary column (DB-DIOXIN, 60 m, 0.25 mm, 0.15 μm). The laboratory reagent and equipment blank samples were treated and analyzed by the same method as the actual samples, one blank for every five samples. In cow milk, pork and beef samples, the limits of determination (LOD) for PCDD/Fs, non-*ortho* PCBs, and other PCBs were 0.1-1, 0.1, and 10 pg g^{-1}, respectively, and in rainbow trout samples 0.01-0.1, 0.01, and 1 pg g^{-1}, respectively. In vegetable and flour samples, LODs were 0.005-0.05, 0.005, and 0.05 pg g^{-1}, respectively. PCDD/F, non-*ortho* PCB, and other PCB LODs for eggs were 0.5-5, 1, and 10 pg g^{-1}, respectively. In the calculations of TEqs, results below the LOD were considered as zero. Concentrations in cow milk, eggs, pork and beef samples were calculated on a fat basis, in other samples on a wet weight (w.w.) basis. Recoveries for internal standards were >60% for all congeners.

The laboratory has participated in several international quality control studies for the analysis of PCDD/Fs, and PCBs. The matrixes in these studies have included cow milk, human milk, human serum and fish. (Yrjänheikki 1991, Rymen 1994, Liem et al. 1996, WHO/EHEC 1996). The laboratory is also an accredited testing laboratory (No T77) in Finland (EN 17025). The scope of accreditation includes PCDD/Fs, PCBs, and non-*ortho* PCBs from milk and tissue samples.

Food consumption data

The food and food group consumption data used in the intake calculations consist of the average consumption figures taken from the 1997 Dietary Survey of Finnish Adults (National Public Health Institute 1998). The method of 24-h dietary recall was applied in this national survey of the adult population from five selected areas, aged 25-64 (n=2862). In the dietary survey, the average consumption (g day^{-1}) of each food item was calculated. The food items which were relevant for intake estimations of PCDD/F and PCB were aggregated into food groups (table 4). The number of individual food items was 54 for milk and milk products, three for eggs, 28 for fish, 45 for meat, 29 for flour, and 43 for potatoes and vegetables.

Estimation of average daily intakes of PCDD/Fs and PCBs

Average daily intakes of PCDD/Fs and PCBs were estimated by multiplying the measured concentrations of PCDD/F and PCB toxic equivalents by the average daily consumption of the respective food. Intakes for PCDD/Fs and PCBs were calculated separately. For Baltic herring, data were used that included 1200 herring samples from the Baltic Sea (data partly published, Vartiainen et al. 1997). The PCB-TEq data for Baltic herring were incomplete

because a major part of the concentrations for congeners IUPAC 114, 123, 157, 167, 170 and 189 was not available. The I-TEq and PCB-TEq used for other fish were estimations based on the unpublished data from Finland.

The intake results were reported as pg toxic equivalents per day (pg TEq day^{-1}) or as pg toxic equivalents kg^{-1} body weight per day (pg TEq kg^{-1} b.w. day^{-1}). The body weight used in these calculations was 76 kg, which was the average body weight of the adult population participating in the 1997 Dietary Survey of Finnish Adults (National Public Health Institute 1998).

4. RESULTS

The mean concentrations of seven toxic PCDDs and 10 PCDFs for foods are presented in table 1 including ∑ PCDD/F and I-TEq results. The results for non-*ortho* (three congeners), mono-*ortho* (eight) and other PCBs (25) are presented in table 2 along with ∑ PCB and PCB-TEq results. The fat contents in the cows' milk, eggs, pork, beef and rainbow trout were 3.2, 9.7, 5.9, 6.6 and 8.3%, respectively.

Table 3 lists the congeners that contributed to TEqs by >5 % and their contribution is presented for the most relevant foods. Although samples of Baltic herring were not analysed here, the contribution of congeners to TEqs in Baltic herrings are added to table 3. The I-TEq contribution pattern in different foods varied extensively for the different foods. In Baltic herrings, rainbow trouts, eggs and cow's milk 2,3,4,7,8-pentachloro dibenzofuran (2,3,4,7,8-PF) was the most dominant congener, while in beef and pork samples 1,2,3,6,7,8-hexachloro dibenzo-*p*-dioxin (1,2,3,6,7,8-HD) was the most dominant congener. The secondary congener contributing most significantly to I-TEq varied greatly for the different foods. It was 2,3,7,8-tetrachloro dibenzofuran (2,3,7,8-TCDF) in rainbow trout samples, 1,2,3,7,8-pentachloro dibenzo-*p*-dioxin (1,2,3,7,8-PD) in Baltic herring, beef and pork samples and 1,2,3,6,7,8-HD in egg and cow milk samples. In PCB-TEq, the situation was different. In all samples, excluding eggs, IUPAC126 was the most dominating congener followed by IUPAC118, which was the most dominating congener in egg samples.

In rainbow trout and egg samples, PCDFs contributed most extensively to ∑ PCDD/F (table 1) and from the individual congeners 2,3,7,8-TCDF and 2,3,4,7,8-PF were dominating in rainbow trout and 1,2,3,4,6,7,8-heptachloro dibenzofuran (1,2,3,4,6,7,8-F) in egg samples. In all other food samples, PCDDs dominated the ∑ PCDD/F and the higher chlorinated PCDDs, 1,2,3,4,6,7,8-heptachloro dibenzo-*p*-dioxin (1,2,3,4,6,7,8-D) and octachloro dibenzo-*p*-dioxin

(OCDD), were the most abundant. In PCBs, the group "other-PCBs" was the most dominating group accounting for >84 % of ∑ PCB in all food groups. With respect to the individual congeners IUPAC 101, 110, 118, 153, 138 and 180 had the strongest impact on ∑ PCBs. In beef and pork samples, congeners IUPAC 51 and 47 also contributed significantly to ∑ PCB.

The daily consumption of different food and food groups, and the concentrations and intakes of PCDD/Fs and PCBs as TEqs in Finland are presented in table 4. In all other food groups excluding eggs, and meat and meat products PCB-TEqs contributed more strongly to total TEq intake than to I-TEq. In herring samples, PCB-TEq was underestimated because in part of the samples all of the PCB congeners were not measured. Therefore the daily intake of PCB-TEq was clearly an underestimation with respect to Baltic herrings.

Table 1. Polychlorinated dibenzo-*p*-dioxins (seven PCDD congeners), dibenzofurans (10 PCDF congeners), sum of PCDD/Fs (∑ PCDD/Fs) and toxic equivalents (I-TEqs) as pg g^{-1} in food samples in Finland, 1998-2000.

Food item	Rainbow trout	Eggs	Beef	Cow milk	Pork	Leafy vegetables[c]	Flour[d]	Potato[a]	Fruit vegetables[e]
n	8	5	5	5	6	4	2	1	4
Analytes	Mean[a] (SD)	Mean[b] (SD)	Mean[b] (SD)	Mean[b] (SD)	Mean[b] (SD)	Mean[a] (SD)	Mean[a] (SD)	Mean[a]	Mean[a] (SD)
PCDD	0.41 (0.16)	6.6 (8.4)	13 (14)	1.6 (1.0)	4.4 (1.6)	0.2 (0.18)	0.5 (0.36)	0. 083	0.003 (0.006)
PCDF	2.7 (0.96)	19 (42)	0.72 (0.52)	0.88 (0.42)	1.0 (1.4)	0.047 (0.088)	0.008 (0.011)	nd	nd
Σ PCDD/F	3.1 (1.1)	26 (40)	14 (14)	2.5 (1.0)	5.4 (1.8)	0.24 (0.25)	0.51 (0.37)	0. 083	0.003 (0.006)
I-TEq	0.74 (0.29)	0.52 (0.44)	0.29 (0.24)	0.12 (0.02)	0.051 (0.023)	0.01 (0.02)	0.00094 (0.00065)	0.00025	0.00003 (0.00006)

nd, <Limit of determination (LOD)
[a] Wet weight (w.w.) basis.
[b] Fat basis.
[c] Three different kinds of lettuces and cabbage.
[d] Rye and wheat.
[e] Cucumber, tomato, onion and sweet pepper.

Table 2. Polychlorinated biphenyls (three non-*ortho*-PCB, eight mono-*ortho*-PCB, and 25 other PCB congeners), sum of PCBs (∑ PCBs) and toxic equivalents (PCB-TEqs) as pg g^{-1} in food samples in Finland, 1998-2000.

Food item	Rainbow trout	Eggs	Beef	Cow milk	Pork	Leafy vegetables[c]	Flour[d]	Potato	Fruit vegetables[e]
N	8	5	5	5	6	4	2	1	4
Analytes	Mean[a] (SD)	Mean[b] (SD)	Mean[b] (SD)	Mean[b] (SD)	Mean[b] (SD)	Mean[a] (SD)	Mean[a] (SD)	Mean[a]	Mean[a] (SD)
Non-*ortho*-PCBs	45 (14)	3.2 (6.3)	4.2 (1.9)	2.1 (1.1)	1.9 (0.78)	1.3 (2.2)	0.17 (0.021)	nd	0.0037 (0.0043)
Mono-*ortho*-PCBs	3 100 (1 100)	730 (430)	530 (250)	410 (110)	60 (27)	34 (50)	1.4 (1.9)	1.6	0.33 (0.28)
Other-PCBs	17 000 (6 200)	5 100 (2 300)	5 400 (2 700)	2 200 (740)	1 800 (230)	390 (550)	38 (14)	23	3.7 (2.5)
Σ PCB	21 000 (7 400)	5 800 (2 300)	5 900 (3 000)	2 600 (840)	1 800 (220)	420 (600)	39 (16)	24	4.1 (2.8)
PCB-TEq	1.5 (0.48)	0.12 (0.12)	0.31 (0.15)	0.22 (0.078)	0.024 (0.012)	0.038 (0.058)	0.00022 (0.00018)	0.00016	0.000036 (0.000031)

nd, <Limit of determination (LOD)
[a] Wet weight (w.w.) basis.
[b] Fat basis.
[c] Three different kinds of lettuces and cabbage.
[d] Rye and wheat.
[e] Cucumber, tomato, onion and sweet pepper.

Table 3. Contibution (%) of single congeners to I-TEqs and PCB-TEqs in different food samples. Only congeners contributing at least 5% to TEqs are shown. The two most dominating congeners are shaded.

Dioxin	Baltic herring	Rainbow trout	Eggs	Beef	Cow milk	Pork
2,3,7,8-TCDF		22				
2,3,4,7,8-PF	68	50	51	12	70	
1,2,3,4,6,7,8-F			16			14
2,3,7,8-TCDD	7	12				
1,2,3,7,8-PD	13	11		19		23
1,2,3,6,7,8-HD			20	47	7	39
1,2,3,4,6,7,8-D				10	5	11
OCDD						9
% of I-TEq	88	96	87	87	82	96
PCB						
IUPAC126	42	67	9	63	68	45
IUPAC169						7
IUPAC105	8		15			
IUPAC118	17	13	49	13	12	22
IUPAC156	18	7	18	8	9	15
IUPAC170				11		5
% of PCB-TEq	85	87	91	95	89	95

5. DISCUSSION

PCDD/F and PCB occurrence data

I-TEq and PCB-TEq concentrations for rainbow trouts, 0.74 pg g^{-1} w.w. and 1.5 pg g^{-1}, respectively, were moderate when one considers that rainbow trout is a fatty fish. Two explanations can be provided for the low concentrations (1) all rainbow trouts were very young (2 years of age) and (2) they were all farmed trouts and they had been fed with artificial fodder. Two-year-old trouts were chosen for the study because this is the age farmed trouts are normally harvested.

A decline of 50% was observed from 1.8 in 1993 (Hallikainen et al. 1995) to 0.74, in the I-TEq concentration of rainbow trout samples in Finland. This current value is now in the same range as the concentration of rainbow trout in Germany (Malisch 1998).

In cows' milk from 1991 (Vartiainen and Hallikainen 1994) to 1998 the decline in concentration of I-TEq was from 0.99 to 0.12 pg I-TEq g^{-1} fat. In Germany and The Netherlands, the concentrations in cow's milk were six to 10 times higher than in Finland, respectively (Liem and Theelen 1997, Malisch 1998). The concentrations may have declined in cows' milk in Finland due to the decrease in deposition of PCDD/Fs onto grassland from the atmosphere.

Similarly to cows' milk, concentration of I-TEq also in eggs has declined during the 1990s from 1.6 to 0.52 pg I-TEq g^{-1} fat. In Germany and The Netherlands, the concentrations in eggs were three to four times higher than in Finland (Liem and Theelen 1997, Malisch 1998).

Table 4. Daily consumption of food and food groups, and concentrations and intakes of polychlorinated dibenzo-*p*-dioxins and furans (PCDD/Fs) and polychlorinated biphenyls (PCBs) as toxic equivalents (TEqs) in Finland.

Food group	Consumption, g day^{-1} (g fat day^{-1})	PCDD/F (pg I-TEq g^{-1})	PCB (pg PCB-TEq g^{-1})	Daily intake, PCDD/F (pg I-TEq)	Daily intake, PCB (pg PCB-TEq)	Daily intake, total (pg TEq)
Milk, high fat	40 (1.3)	0.12 [b]	0.22 [b]	0.16	0.29	0.45
Milk, fat <1.6%	170 (2.6)	0.12 [b]	0.22 [b]	0.32	0.58	0.91
Milk products	51 (16)	0.12 [b]	0.22 [b]	2.0	3.6	5.6
Butter	8 (6.4)	0.12 [b]	0.22 [b]	0.79	1.4	2.2
Butter based mixtures	4 (2.6)	0.12 [b]	0.22 [b]	0.32	0.58	0.91
Milk and dairy products	270 (29)			3.6	6.5	10
Eggs	19 (1.7)	0.52 [b]	0.12 [b]	0.89	0.21	1.1
Herring	3	8.0 [a]	7.9 [a]	24	24	48
Rainbow trout	6.5	0.74 [a]	1.5 [a]	4.8	9.6	14
Other fish	19	0.5 [a]	0.5 [a]	9.5	9.5	19
Fish and fish products	29			38	43	81
Beef	23 (2.1)	0.29 [b]	0.31 [b]	0.62	0.65	1.3
Pork	33 (5.0)	0.051 [b]	0.024 [b]	0.26	0.12	0.38
Other meat, sausages	62 (14)	0.17 [b]	0.17 [b]	2.4	2.3	4.8
Meat and meat products	120 (21)			3.3	3.1	6.4
Flour	160	0.00094 [a]	0.00022 [a]	0.15	0.035	0.19
Potato	110	0.00025 [a]	0.00016 [a]	0.028	0.017	0.045
Leafy vegetables	17	0.01 [a]	0.038 [a]	0.18	0.65	0.82
Fruit vegetables	82	0.00003 [a]	0.000036 [a]	0.0025	0.0029	0.0054
Others	370			0.36	0.70	1.1
Total intake pg TEq day^{-1}				46	53	100

[a] TEqs as pg g^{-1} w.w.
[b] TEqs as pg g^{-1} fat.

I-TEq concentrations in pork have decreased from the 1991 value of 0.29 (Vartiainen and Hallikainen 1994) to 0.051 pg I-TEq g^{-1} fat. The opposite situation was discovered with beef, where the values increased from 0.018 to 0.29 pg I-TEq g^{-1} fat. In Germany and The Netherlands, the concentrations in pork and beef were two to 10 times higher than those found in Finland (Liem and Theelen 1997, Malisch 1998). It was very difficult to detect any time trend in these figures in Finland. Both values are low probably because pigs as well as beef cattle are slaughtered when they are still very young.

The concentrations of I-TEqs and PCB-TEqs in flour, potato and vegetable samples were both virtually negligible and their impact on intake was very small. It is very difficult to compare the concentrations of vegetables between different countries because of the heterogenity of the measured items in each country. The measured concentrations in Finland were very low when compared to Spain or Germany (Malisch 1998, Domingo et al. 1999). Only the I-TEq concentration of leafy vegetables, 0.01 pg g^{-1} w.w., was in the same range as reported in the study from Germany (Malisch 1998).

Intake of PCDD/Fs and PCBs

The intake of PCDD/Fs declined from 95 pg N-TEq (Hallikainen et al. 1995) to 46 pg I-TEq between 1992 and 1999 (N-TEq and I-TEq toxic equivalency factors are almost identical). Two obvious reasons for this decline were found. First, the lower concentrations of I-TEqs in cows' milk and eggs. Second, the consumption of eggs, fish and milk has also diminished compared to the previous dietary survey.

To estimate the impact of new occurrence and new consumption data to the intake of PCDD/Fs, the intake of PCDD/Fs was calculated with new concentrations of PCDD/Fs combined with the old food consumption data. With this kind of procedure, the intake was calculated to be 70 pg I-TEq day^{-1}. It is concluded that the changes in consumption data and concentrations have both affected almost equally the intake of PCDD/Fs.

The intake of PCB-TEqs, 53 pg PCB-TEq day^{-1}, was clearly underestimated in this study because PCB-TEq concentration data for Baltic herring were incomplete. The total intake of TEqs was 100 pg TEq day^{-1} (1.3 pg TEq kg^{-1} b.w. day^{-1}), which is in the range of the tolerable daily intake (TDI), 1-4 pg TEq kg^{-1} b.w. day^{-1}, given by WHO (van den Berg et al. 1998).

The average intake of PCDD/Fs in Europe has been reported to be between 42 and 210 pg I-TEq day^{-1} (Liem and Theelen 1997, Becher et al. 1998, Harrison et al. 1998, Malisch 1998, Domingo et al. 1999, Zanotto et al. 1999). Both the lowest and the highest intakes have been measured in the Mediterranean around Venice, Italy (42 pg I-TEq day^{-1}) and in Spain (210 pg I-TEq day^{-1}) (Domingo et al. 1999, Zanotto et al. 1999). In Western Europe (The Netherlands, Germany, UK), the intake has been calculated to vary between 61 and 90 pg I-TEq day^{-1} (Liem and Theelen 1997, Harrison et al. 1998, Malisch 1998). In the northern parts of Europe in addition to Finland, there is a current estimation available for the daily intake of PCDD/Fs for Norway where it varied between 50.6 and 84.6 pg I-TEq day^{-1} (Becher et al. 1998). The PCDD/F intake in the USA was estimated to be 41 pg TEq day^{-1} (US EPA 2000). In Japan, the estimated

intake of PCDD/Fs was equivalent to European intakes, ~70 pg I-TEq day^{-1} (Yoshida et al. 2000). The lowest intake of PCDD/Fs has been reported from New Zealand, 14.5 pg I-TEq day^{-1} for the average population (Buckland et al. 1998).

The recent PCB-TEq intake estimations are available from UK, USA, Norway and New Zealand. The lowest intake for PCB-TEqs was also found in New Zealand, 12.2 PCB-TEqs day^{-1}, followed by the USA (24 PCB-TEqs), UK (54 PCB-TEqs) and Norway, where the intake was estimated to be between 137 and 190 pg PCB-TEq day^{-1}.

Time trends of PCDD/F intake have been studied in the UK, The Netherlands and Germany (Liem and Theelen 1997, Harrison et al. 1998, Malisch 1998). The trends for the intakes have been declining in all of these studies. In the UK, the intake of PCDD/Fs between 1982 and 1992 decreased from 4.1 to 1.5 pg I-TEq kg^{-1} b.w. day^{-1}. Simultaneously, the daily intake of PCB-TEq also declined from 2.7 to 0.9 pg PCB-TEq kg^{-1} b.w. (Harrison et al. 1998). A decline of almost 40% in the intake of PCDD/Fs was observed in The Netherlands between 1991 and 1997, when the daily intake decreased from 115 to 73 pg I-TEq (Liem and Theelen 1997). In Germany, the time trend results showed that the intake in 1993-96 was about one-half of the intake calculated between 1986 and 1991, from 127 to 61 pg I-TEq day^{-1} (1.82-0.88 pg I-TEq kg^{-1} b.w. day^{-1}) (Malisch 1998).

The contribution of fish in Finland increased from 60 to 82%, indicating that fish is clearly the most important contributor to PCDD/F intake in Finland. In the dietary method used in the FINDIET 1997 survey, the fish consumption can be estimated lower than by other dietary methods and this further emphasizes the impact of fish to the intake. The contribution of milk and dairy products had clearly diminished, from 31 to 8% because of the decreased I-TEq concentration and reduced consumption.

In the Venetian and Norwegian studies, fish and fish products contributed to the PCDD/F intakes as much as in the Finnish study. In Venice, the proportion varied from 42 to 50% and in Norway from 28 to 43% (Becher et al. 1998, Zanotto et al. 1999). In the UK, Germany, The Netherlands, the USA and New Zealand the main source of PCDD/Fs are meat and meat products and milk and dairy products, the proportion of both groups being ~30% (Liem and Theelen 1997, Buckland et al. 1998, Harrison et al. 1998, Malisch 1998, US EPA 2000). The proportion of fish and fish products in these countries varied between 1 and 20%. The impact of vegetables on the intakes of PCDD/Fs have been considered as negligible or insignificant in many studies, but recently in Spain the contribution of vegetables has been estimated as being noteworthy (Domingo et al. 1999). Taking the impact of vegetables into consideration in the intake calculations, Domingo et al. concluded the intake in Catalonia to be 210 pg I-TEq day^{-1}. If

one excluded vegetables from the calculations, then the intake would have been 117 pg I-TEq day^{-1}. Lovett et al. (1997) concluded that the increase in intake of PCBs and PCDD/Fs resulting from eating fruits and vegetables was unlikely to >3% for PCBs and 8% for PCDD/Fs, of the average daily intakes of these contaminants from all food sources.

Table 5 shows the percentage exposure for PCDD/F from the most important food sources, and daily intake of PCDD/Fs in different countries. It reveals that geographically the countries nearest to Finland i.e. Germany and Norway, have the most similar intakes as Finland, 61.3 and 51 pg TEq day^{-1}, respectively. The Western European countries, The Netherlands and UK can be grouped with each other with 73 and 90 pg TEq day^{-1} intakes. The daily intake of PCDD/Fs in Finland was ~3 times higher than the intakes in New Zealand (14.5 pg I-TEq).

In this study, the TEq intakes have been calculated with the concentration results where the non-detected results were considered as zeros. If the intakes of PCDD/Fs and PCBs were calculated with these results replaced by LODs, the PCDD/F intake would be 65 pg I-TEq day^{-1} and PCB intake 54 pg PCB-TEq day^{-1}. This shows the crucial effect of handling the non-detected values when the concentrations of studied substances are very near to the limit of determination (LOD).

6.CONCLUSIONS

In this study, the updated PCDD/F intake calculations revealed ~50% decrease in daily I-TEq intake in the average Finnish population. The main reason for this decrease was the lower concentrations of PCDD/Fs in cows' milk and eggs and also the lower consumption of milk, eggs and fish. This study shows that the contribution of fish to the intake of PCDD/F has become more dominant compared to the previous study in Finland.

The estimated daily intake of PCB-TEq was close to I-TEq intake, but the data for PCBs was not totally comprehensive, thus in the future the PCB occurrence data in foods must be completed in order to obtain a better estimate for PCB-TEq intake. The intake and trend for I-TEq in Finland is very consistent with other European countries. The total intake of PCDD/Fs and PCBs, 1.3 pg TEq kg^{-1} b.w. day^{-1}, is within the range of tolerable daily intake (TDI) provided by the WHO.

Table 5. Percentage exposure of PCDD/F from the most important food sources, and daily intake of PCDD/Fs in different countries.

Country	Milk and dairy products	Meat and meat products	Fish and fish products	Eggs	Others	PCDD/F daily intake (pg I-TEq)	Reference
Spain	16	10	15	2	57	210	Domingo et al. (1999)
USA	29	39	20	3	9	41	US EPA (2000)
Italy (Venice)	29-53	3-11	42-50	2-10	-	15-130	Zanotto et al. (1999)
UK	25	31	8	5	31	90*	Harrison et al. (1998)
Norway	8-13	6-10	28-43	4-7	27-54	51-85	Becher et al. (1998)
The Netherlands	46	23	3	4	24	73	Liem and Theelen (1997)
Germany	31	23	17	8	21	61.3	Malisch (1998)
New Zealand	16	36	12	3	33	15	Buckland et al. (1998)
Finland 1992	31	1.4	60	3	-	95	Hallikainen et al. (1995)
Finland 1999	8	7	82	2	1	46	present study

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CHAPTER 3

MARKET BASKET STUDY ON DIETARY INTAKE OF PCDD/Fs, PCBs, AND PBDEs IN FINLAND

Hannu Kiviranta[a]*, Marja-Leena Ovaskainen[b] , Terttu Vartiainen[a,c]

[a] Department of Environmental Health, National Public Health Institute, PO Box 95, Kuopio 70701, Finland

[b] Department of Nutrition, National Public Health Institute, Mannerheimintie 166, Helsinki 00300, Finland

[c] Department of Environmental Science, University of Kuopio, PO Box 1627, Kuopio 70211, Finland

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1. ABSTRACT

We have measured the concentrations of polychlorinated dibenzo-*p*-dioxins (PCDD/F), polychlorinated biphenyls (PCBs), and polybrominated diphenyl ethers (PBDE) in 10 market baskets consisting of almost 4000 individual food samples representing 228 different food items, and also in the total diet basket. Lower bound concentrations of PCDD/Fs ranged between 0.0057 and 5.6 pg/g fresh weight in the market baskets and the corresponding values for PCBs from 39 to 25,000 pg/g. The fish basket contributed most to the concentrations of dioxins and PCBs, and also to concentrations of PBDEs in which the lower bound range was from 0.82 to 850 pg/g. We also assessed the average daily intakes of these substances by the Finnish adult population. The average daily intake of sum of PCDD/Fs and PCBs as WHO toxic equivalents was assessed to be 115 pg which was 1.5 pg WHO-TEq/kg body weight using an average mean weight of 76 kg for the general population in Finland. The contribution of fish to the intake of PCDD/Fs was between 94% and 72%, depending on whether lower or upper bound concentrations were used. With respect to PCBs, the contribution of fish was 80%. The calculated intake of PBDEs of 44 ng/day was comparable to intake assessments from other countries. Fish also contributed most to the PBDE

intake, but there was some other source of PBDEs that distinguishes the exposure to PBDEs from exposure to PCDD/Fs and PCBs. This additional source seemed to be found in the market basket that included beverages, spices, and sweets.

2. INTRODUCTION

More than 90% of the average human intake of polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans (PCDD/F, dioxins) and polychlorinated biphenyls (PCBs) originates from food, especially food of animal origin (Liem et al., 2000). In a risk assessment of dioxins and dioxin-like PCBs in the diet, the Scientific Committee for Food (SCF) of the European Commission assessed a tolerable weekly intake (TWI) of 14 pg/kg body weight (bw) for these chemicals as toxic equivalents (WHO-TEq), according to the WHO toxic equivalency factor (TEF) scheme (European Commission, 2001; Van den Berg et al., 1998). Exposure estimates, made by SCF, indicated that a proportion of the European population has a dietary intake of dioxins and dioxin-like PCBs which is in excess of the TWI.

In our previous study we concluded that at the end of the 1990s the exposure of the Finnish population to dioxins was only about 50% of their exposure at the beginning of the decade (Kiviranta et al., 2001). In that survey we used the Selective Study of Individual Foodstuffs (SSIF) approach combined with food consumption data from a 24-h dietary recall study for 2862 Finnish adults (National Public Health Institute,1998). The average dioxin intake, using former Ahlborg and NATO TEFs (Ahlborg et al., 1994; NATO/CCMS, 1988), was calculated to be 46 pg TEq/day, and the assessed PCB intake was 53 pg PCB-TEq/day. Thus the total intake of dioxins and dioxin-like PCBs was 100 pg TEq/day. On average, weekly intake of these chemicals in Finland was then assessed to be 9.2 pg TEq/kg bw and this was below the Commission's recommendation.

In this study, the Agricultural Research Centre of Finland composed an average Finnish market basket diet and the National Public Health Institute, Laboratory of Chemistry, analyzed the diet for dioxins and PCBs, and also for polybrominated diphenyl ethers (PBDEs). The compositions and consumption of the market baskets were based on the same Dietary Survey of Finnish Adults (National Public Health Institute,1998) as used in our previous study. Thus we were able to compare intake assessments of dioxins and dioxin-like PCBs between these two methods; Market Basket Method (MBM) and SIFF-method. The intake assessment of PBDEs was conducted for the first time in Finland, with our main interest focussed on the origin and level of adult population exposure to PBDEs.

3. MATERIALS AND METHODS

Food consumption data and composition of the market baskets

The food and food group consumption data used in the composition of the market baskets and in the intake calculations consist of the average consumption figures taken from the 1997 Dietary Survey of Finnish Adults (National Public Health Institute, 1998). In this 24-h recall study the whole adult population, sampled from five major Finnish provinces and cities, aged 25-64, was included (n=2862).

Ten individual market baskets were created: (1) liquid milk products; (2) solid milk products; (3) fish; (4) meat and eggs; (5) fats; (6) cereal products; (7) potato products; (8) vegetables; (9) fruits and berries; (10) beverages, spices, sweets etc. In addition, a total diet basket was created by mixing individual market baskets based on the consumption proportion of each individual market basket in the total average diet. Alcoholic beverages were omitted from the market baskets and also from the total diet basket. Table 1 shows the average daily consumption of market baskets, and the main items within the basket. Market baskets included all food items whose average daily consumption exceeded 0.5 g. A total of 228 different food items was included, with 3,988 individual samples (177 kg in total weight) being combined. In the market basket, the amount of each food item was obtained from the consumption data and the amount of each sample within a food item from market share data obtained from A.C.Nielsen Finland. Sample collection was carried out during a period of April 1997 and June 1999 in order to collect each product at its peak season. Of the samples 39.9% were collected from supermarkets, 38.7% directly from manufacturers, 11.2% from wholesalers, 6.3% from producers, and 3.9% from farmer markets.

Preparation of food samples for analysis of PCDD/Fs, PCBs, and PBDEs

Pooling of food samples into 10 market baskets and to the total diet basket was undertaken in the Agricultural Research Centre of Finland, where measurements of the fat content of market baskets were conducted. Due to the lengthy collection period, the purchased food samples were stored mainly frozen at - 25°C before pooling into their respective market basket.

Table 1.

Market baskets, their average daily consumption, g/d (percentage of total daily consumption), and their main items (percentage in a basket).

Market basket	Consumption g/d (%)	Main items (%)
(1) Liquid milk products	427 (21)	Milk (71)
		Sour milk (15)
		Yogurt (11)
(2) Solid milk products	32.5 (1.6)	Cheese (94)
(3) Fish	27.4 (1.4)	Salmon and rainbow trout (29)
		Tuna and saithe (19)
		Baltic herring (11)
		Vendace (8)
(4) Meat and eggs	132 (6.5)	Processed meat products (31)
		Beef (20)
		Pork (19)
		Poultry (13)
		Eggs (13)
(5) Fats	35.7 (1.8)	Margarines (43)
		Butter (22)
		Butter-oil mixtures (16)
		Vegetable oils (13)
(6) Cereal products	181 (9.0)	Bread (48)
		Flour and other cereals (43)
(7) Potato products	121 (6.0)	Potato (97)
(8) Vegetables	117 (5.8)	Tomato (20)
		Carrot (15)
		Cucumber (12)
		Onion (9)
(9) Fruits and berries	221 (11)	Citrus fruits (32)
		Other fresh fruits (28)
		Whole juices (24)
		Berries (12)
(10) Beverages, spices, sweets	725 (36)	Coffee and tea (80)
		Beverages and juices (14)
		Sugars and honey (3)
Total	2020 (100)	

The fat content of all market baskets, except basket 10, and the total diet basket was determined by diethyl ether extraction after acid hydrolysis. The fat content was determined gravimetrically. All market baskets, with the exception of basket 10 were freeze dried before transporting to the laboratory of chemistry in the National Public Health Institute.

Analysis of PCDD/Fs, PCBs, and PBDEs

The occurrence of 17 PCDD/F (toxic) congeners, of three non-*ortho* (PCB 77, 126, and 169), eight mono-*ortho* (PCB 105, 114, 118, 123, 156, 157, 167, and 189), of 23 (PCB 18, 28, 33, 49, 52, 60, 66, 74, 99, 101, 110, 122, 128, 138, 141, 153, 170, 180, 183, 187, 194, 206, and 209) other PCB congeners, and of five PBDE congeners (PBDE 47, 99, 100, 153, and 154) were

measured. For PCDD/Fs, toxic equivalents (TEq) were calculated with two different sets of toxic equivalency factors (TEF), the NATO factors (NATO/CCMS, 1988) gave I-TEqs, and the factors recommended by WHO in 1998 (Van den Berg et al., 1998) gave $WHO_{PCDD/F}$-TEqs. For PCBs, TEqs were also calculated with two sets of TEFs, factors by Ahlborg et al. (1994) gave PCB-TEqs and factors by WHO gave WHO_{PCB}-TEqs.

Samples were spiked with 16 ^{13}C-labeled PCDD/F standards (2,3,7,8-chlorinated PCDD/F congeners), with three ^{13}C-labeled non-*ortho* PCB standards (PCB 77, 126, and 169), 13 ^{13}C-labeled other PCB standards (PCB 30 [^{12}C-labeled], 80, 101, 105, 118, 138, 153, 156, 157, 170, 180, 194 and 209), and with two ^{13}C-labeled PBDE standards (PBDE 77 and 126). Samples of market baskets 1 to 9 and the total diet basket were extracted with toluene for 24 h using the Soxhlet apparatus. Sample of market basket 10, beverages, spices, sweets etc., was first filtered and the filtrate was then extracted with toluene in a separation funnel. The same toluene was used for extraction of the precipitate of the filtration in a Soxhlet apparatus. All the samples were defatted in a silica gel column containing acidic and neutral layers of silica, and all analytes were eluted with dichloromethane (DCM)/cyclohexane (c-hexane) (1:1). PCDD/Fs were separated from PCBs and PBDEs on activated carbon column (Carbopack C, 60/80 mesh) containing Celite (Merck 2693). The first fraction including PCBs and PBDEs was eluted with DCM:c-hexane (1:1) following a back elution of the second fraction (PCDD/Fs) with toluene. Eluents from both of the fractions were evaporated using nonane as a keeper and then fractions were further cleaned by passing through an activated alumina column (Merck 1097). The PCDD/F fraction was eluted from the alumina column with 20% DCM in *n*-hexane and recovery standards (^{13}C 1,2,3,4-TCDD and ^{13}C 1,2,3,7,8,9-HxCDD) were added to the fraction before DCM and *n*-hexane were replaced by 10-15 µl of nonane. The PCB-PBDE fraction was eluted from the alumina column with 2% DCM in *n*-hexane, and the fraction was further fractionated, after changing the eluent to *n*-hexane and transferring to another activated carbon column (without Celite) in order to separate the non-*ortho* PCBs from other PCBs and PBDEs. DCM (50%) in *n*-hexane was used to elute other PCBs and PBDEs, and non-*ortho* PCBs were back eluted with toluene. Recovery standards, PCB 159 for other PCBs and PBDEs, and ^{13}C PCB 60 for non-*ortho* PCBs were added prior to analysis; for non-*ortho* PCBs toluene was replaced by 10-15 µl of nonane. The quantitation was performed by selective ion recording using a VG 70-250 SE (VG Analytical, UK) mass spectrometer (resolution 10,000) equipped with a HP 6890 gas chromatograph with a fused silica capillary column (DB-DIOXIN, 60 m, 0.25 mm, 0.15 µm). Two microliters were injected into a split-splitless injector at 270°C. The temperature

programs for PCDD/Fs, non-*ortho*-PCBs, mono-*ortho*- and other PCBs , and PBDEs were: start, 140°C (4 min), rate 20°C/min to 180°C (0 min), rate 2°C/min to 270°C (36 min); start, 140°C (4 min), rate 20°C/min to 200°C (0 min), rate 10°C/min to 270°C (12 min); start, 60°C (3 min), rate 20°C/min to 200°C (0 min), rate 4°C/min to 270°C (14 min); start, 100°C (2 min), rate 25°C/min to 240°C (0 min), rate 4°C/min to 300°C (25 min), respectively. Limits of quantitation (LOQ) for PCDD/Fs, non-*ortho* PCBs, mono-*ortho*- and other PCBs, and PBDEs varied between 0.0007 and 0.63, 0.0007 and 0.13, 0.048 and 3.2, 0.035 and 13 pg/g fw, respectively, depending on each individual congener and on the individual market basket. Recoveries for internal standards were more than 50% for all congeners. Fresh weight concentrations were calculated with both lower bound and upper bound methods. In the lower bound method, the results of congeners with concentrations below LOQ were designated as nil, while in the upper bound method they were denoted as the LOQ.

Quality control and assurance

The laboratory reagent and equipment blank samples were treated and analyzed with the same method as the actual samples, one blank for every five samples. Fish oil is used as an internal quality control sample in the laboratory, and the random errors within the laboratory for $WHO_{PCDD/F}$-TEq, WHO_{PCB}-TEq, and sum of PBDEs are 5.7%, 4.6%, and 4.3%, respectively. The laboratory has participated in several international quality control studies for the analysis of PCDD/Fs, and PCBs. The matrices in these studies have included milk, meat, fat and fish samples. (IUPAC, 1995, 1998, 2000; Lindström et al., 2000; Becher et al., 2001; Småstuen Haug et al., 2002). When taking the systematic error obtained from these studies into account, the uncertainty of $WHO_{PCDD/F}$-TEq and WHO_{PCB}-TEq results were 9.2% and 13.1%, respectively. The laboratory of chemistry in the National Public Health Institute is an accredited testing laboratory (No T077) in Finland (current standard: EN ISO/IEC 17025). The scope of accreditation includes PCDD/Fs, non-*ortho* PCBs, mono-*ortho*- and other PCBs, and PBDEs from environmental samples.

Intake calculations

In the intake calculations, the average daily consumption of the food baskets was multiplied with the corresponding concentrations. Daily intakes (pg/day) for PCDD/Fs, PCBs,

and PBDEs were calculated on a fresh weight basis as a sum of the individual baskets and from the total diet basket. Intakes were calculated with both lower bound and upper bound concentrations. When calculating daily intakes per kg body weight (pg/kg bw), the average weight of 76 kg, which represents the average weight of the population participating in the 1997 Dietary Survey (National Public Health Institute, 1998), was used. In that kind of study it can be assumed that even as many as every third subject has underreported the daily food intake, which is a common phenomenon in national dietary surveys (Hirvonen et al., 1997). The underreporters tend to claim a higher consumption of recommended food items, like vegetables, but lower consumption of less favourable food items such as spread fats. Thus, the average food intake estimated by individual dietary recalls is lower than the actual habitual intake.

4. RESULTS

The concentrations, as pg/g fresh weight (fw), of selected sets of PCDD/Fs and PCBs, corresponding toxic equivalents, and concentrations of PBDEs in 10 market baskets and in the total diet basket are presented in Table 2. Both, lower and upper bound concentrations were calculated.

The maximum concentration of the sum of PCDD/Fs was detected in the fish basket 5.6 pg/g fw followed by fats (3.0), and meat and eggs basket (0.55). The lowest concentration occurred in liquid milk products 0.0057 pg/g fw. Also in TEqs, the fish basket (1.8 pg/g of I-TEq or 2.0 pg/g $WHO_{PCDD/F}$-TEq) dominated since its concentration was over 150 or 200 times higher than in the next biggest basket, fats (0.011 pg/g of I-TEq or 0.0088 pg/g $WHO_{PCDD/F}$-TEq). In the baskets, meat and eggs, fats, cereal products, potato products, vegetables, fruit and berries, the most abundant congeners were octachloro dibenzo-*p*-dioxin (OCDD), 1,2,3,4,6,7,8-heptachloro dibenzofuran (1,2,3,4,6,7,8-HpCDF), and 1,2,3,4,6,7,8-heptachloro dibenzo-*p*-dioxin (1,2,3,4,6,7,8-HpCDD). In the two milk product baskets, the most abundant congeners were 1,2,3,6,7,8-hexachloro dibenzo-*p*-dioxin (1,2,3,6,7,8-HxCDD) and 1,2,3,4,6,7,8-HpCDD. In the fish basket, the majority of the PCDD/F congeners could be quantified, with 2,3,4,7,8-pentachloro dibenzofuran (2,3,4,7,8-PeCDF) and 2,3,7,8-tetrachloro dibenzofuran (2,3,7,8-TCDF) being the most abundant congeners. Since the quantified congeners in most of the market baskets were highly chlorinated compounds with low TEF-values, the difference in TEqs between fish and other baskets was larger than the difference in the sum of PCDD/Fs. It also meant that the upper bound TEqs in baskets were from 3 to as much as 1250 times higher than the lower bound TEqs. The congener profiles in the total diet basket in Fig. 1 illustrates the

average Finnish exposure pattern to dioxins. The profile of the sum of PCDD/Fs was dominated by OCDD (60%) and the next abundant congeners were: 2,3,4,7,8-PeCDF, 2,3,7,8-TCDF, 1,2,3,4,6,7,8-HpCDD, and 1,2,3,6,7,8-HxCDD. In the TEq profiles, 2,3,4,7,8-PeCDF accounted for 65% with congeners 2,3,7,8-tetrachloro dibenzo-*p*-dioxin (2,3,7,8-TCDD) and 1,2,3,7,8-pentachloro dibenzo-*p*-dioxin (1,2,3,7,8-PeCDD) being the next abundant congeners.

The fish basket also showed the highest concentrations of PCBs in every subset of congeners. The maximum concentration of the sum of PCBs was 25,000 pg/g fw followed by fats (750), and solid milk products basket (740). The lowest concentration was measured in the potato products basket 39 pg/g. The TEqs in the fish basket (1.6 pg/g of PCB-TEq or 1.5 pg/g WHO_{PCB}-TEq) were 35 times higher than concentrations in the next highest basket, fats (0.046 pg/g of PCB-TEq or 0.043 pg/g WHO_{PCB}-TEq). The majority of PCB congeners exceeded the LOQ values in all baskets. The three and four chlorine substituted PCBs were more abundant than the higher chlorinated congeners in the following baskets; liquid milk products, fats, cereal products, potato products, fruits and berries, and beverages, spices, sweets, but the most abundant PCBs in baskets; solid milk products, fish, meat and eggs, and also in vegetables were penta, hexa and hepta chlorinated congeners: PCB 101, 110, 118, 138, 153, 170, and 180. For the PCBs the TEqs calculated either with upper or lower bound concentrations did not differ from each other. The lower bound TEqs of PCBs were higher than respective PCDD/F TEqs by a factor ranging from 1.5 to 220 in all other baskets except in the fish and the total diet basket. When comparing upper bound TEqs between dioxins and PCBs, only in the baskets with liquid milk products and vegetables did the PCB TEqs exceed the dioxin TEqs. The congener profiles in the total diet basket describe the average Finnish exposure pattern to PCBs, Fig. 2. Congeners PCB 153, 138, 110, 118, 99, 180, 101 dominated the sum of PCBs profile with coverage of 65%. In the TEq profiles, three congeners; PCB 126, 118, and 156 accounted for 82-89% of the profile.

Table 2.

Concentrations of PCDDs, PCDFs, dioxin toxic equivalents, non-*ortho*-PCBs, mono-*ortho*-PCBs, other PCBs, PCB toxic equivalents, PBDEs, and fat percentages of 10 market baskets and total diet basket as pg/g fresh weight.

Analyte group, pg/g	Market baskets										Total diet basket
	(1) Liquid milk products	(2) Solid milk products	(3) Fish	(4) Meat and eggs	(5) Fats	(6) Cereal products	(7) Potato products	(8) Vegetables	(9) Fruits and berries	(10) Beverages, spices, sweets	
Fat%	2.0	21	6.4	11	79	2.1	0.34	0.90	1.3		3.5
PCDD [7]	**0.0043 (2)**	**0.056 (2)**	**0.91 (7)**	**0.50 (2)**	**3.0 (3)**	**0.28 (2)**	**0.031 (1)**	**0.051 (2)**	**0.12 (2)**	**0.00076 (1)**	**0.19 (6)**
	0.018	0.25	0.91	0.54	3.3	0.30	0.038	0.057	0.13	0.011	0.19
PCDF [10]	**0.0014 (1)**	**ND (0)**	**4.7 (7)**	**0.049 (3)**	**ND (0)**	**0.032 (4)**	**ND (0)**	**0.15 (4)**	**0.014 (3)**	**0.0096 (1)**	**0.058 (3)**
	0.019	0.27	4.7	0.17	1.2	0.091	0.022	0.16	0.041	0.022	0.10
Sum of PCDD/Fs	**0.0057**	**0.056**	**5.6**	**0.55**	**3.0**	**0.31**	**0.031**	**0.21**	**0.14**	**0.010**	**0.25**
	0.037	0.52	5.6	0.71	4.5	0.39	0.059	0.22	0.17	0.033	0.29
I-TEq	**0.00093**	**0.0027**	**1.8**	**0.0082**	**0.011**	**0.0043**	**0.000031**	**0.0012**	**0.00083**	**0.00017**	**0.027**
	0.0031	0.042	1.8	0.026	0.19	0.013	0.0033	0.0042	0.0052	0.0021	0.029
$WHO_{PCDD/F}$-TEq	**0.00093**	**0.0027**	**2.0**	**0.0078**	**0.0088**	**0.0041**	**0.0000031**	**0.0010**	**0.00073**	**0.00017**	**0.029**
	0.0036	0.049	2.0	0.029	0.22	0.015	0.0039	0.0047	0.0059	0.0025	0.030
Non-*ortho*-PCB [3]	**0.11 (3)**	**1.1 (3)**	**34 (3)**	**0.59 (3)**	**0.76 (2)**	**0.80 (2)**	**0.080 (2)**	**0.52 (3)**	**0.39 (2)**	**0.0024 (1)**	**0.76 (3)**
	0.11	1.1	34	0.59	0.88	0.81	0.082	0.52	0.39	0.0038	0.76
Mono-*ortho*-PCB [8]	**7.2 (8)**	**89 (8)**	**3600 (8)**	**52 (8)**	**110 (5)**	**24 (6)**	**1.3 (4)**	**14 (8)**	**4.0 (4)**	**1.1 (2)**	**69 (8)**
	7.2	89	3600	52	120	24	1.5	14	4.3	1.6	69
Other PCB [23]	**64 (20)**	**650 (20)**	**22,000 (23)**	**410 (22)**	**640 (13)**	**380 (20)**	**38 (20)**	**250 (23)**	**51 (19)**	**78 (19)**	**540 (22)**
	64	650	22,000	410	680	380	38	250	51	79	540
Sum of PCBs	**71**	**740**	**25,000**	**470**	**750**	**400**	**39**	**260**	**55**	**79**	**610**
	71	740	25,000	470	790	400	40	260	56	80	610
PCB-TEq	**0.0043**	**0.043**	**1.6**	**0.024**	**0.046**	**0.0071**	**0.00076**	**0.0092**	**0.0021**	**0.00046**	**0.030**
	0.0043	0.043	1.6	0.024	0.049	0.0073	0.00085	0.0092	0.0022	0.00056	0.030
WHO_{PCB}-TEq	**0.0040**	**0.039**	**1.5**	**0.021**	**0.043**	**0.0062**	**0.00067**	**0.0068**	**0.0018**	**0.00041**	**0.028**
	0.0040	0.039	1.5	0.021	0.046	0.0064	0.00076	0.0068	0.0019	0.00051	0.028
Sum of PBDEs [5]	**0.82 (1)**	**34 (3)**	**850 (5)**	**13 (4)**	**180 (2)**	**15 (5)**	**1.3 (4)**	**17 (5)**	**3.8 (3)**	**5.4 (3)**	**43 (5)**
	2.0	40	850	15	220	15	1.4	17	4.2	5.5	43

Lower bound concentrations shown in bold and non-bold font represents the corresponding upper bound concentrations. The number of analysed congeners in brackets and the number of congeners exceeding the LOQ concentrations in parenthesis for each analyte group.

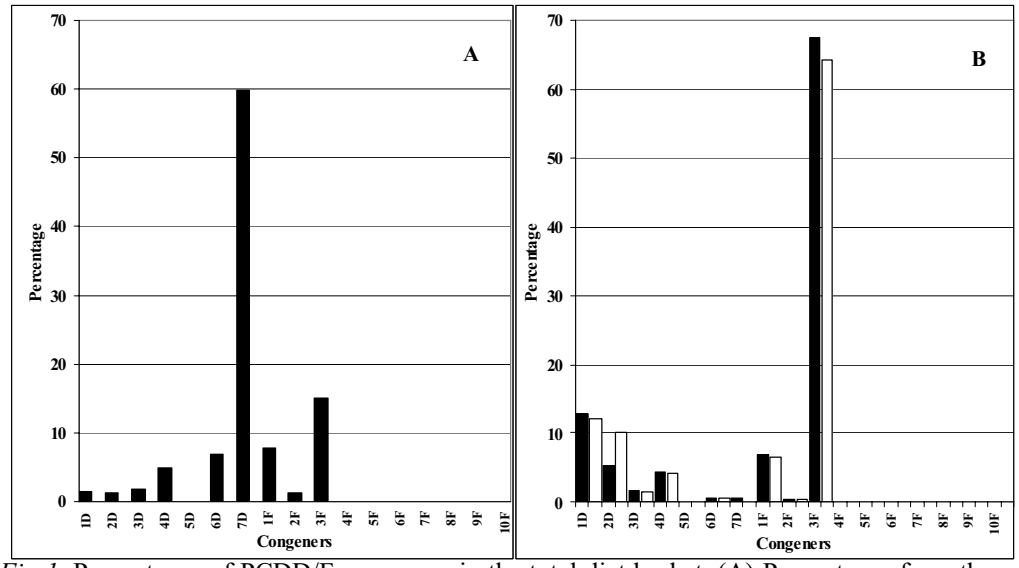

Fig 1. Percentages of PCDD/F congeners in the total diet basket. (A) Percentages from the sum of PCDD/Fs (B) Percentages from toxic equivalents (I-TEq: black bars; $WHO_{PCDD/F}$-TEq: white bars). Congeners: 1D: 2,3,7,8-TCDD; 2D: 1,2,3,7,8-PeCDD; 3D: 1,2,3,4,7,8-HxCDD; 4D: 1,2,3,6,7,8-HxCDD; 5D: 1,2,3,7,8,9-HxCDD; 6D: 1,2,3,4,6,7,8-HpCDD; 7D: OCDD; 1F: 2,3,7,8-TCDF; 2F: 1,2,3,7,8-PeCDF; 3F: 2,3,4,7,8-PeCDF; 4F: 1,2,3,4,7,8-HxCDF; 5F: 1,2,3,6,7,8-HxCDF; 6F: 2,3,4,6,7,8-HxCDF; 7F: 1,2,3,7,8,9-HxCDF; 8F: 1,2,3,4,6,7,8-HpCDF; 9F: 1,2,3,4,7,8,9-HpCDF; 10F: OCDF.

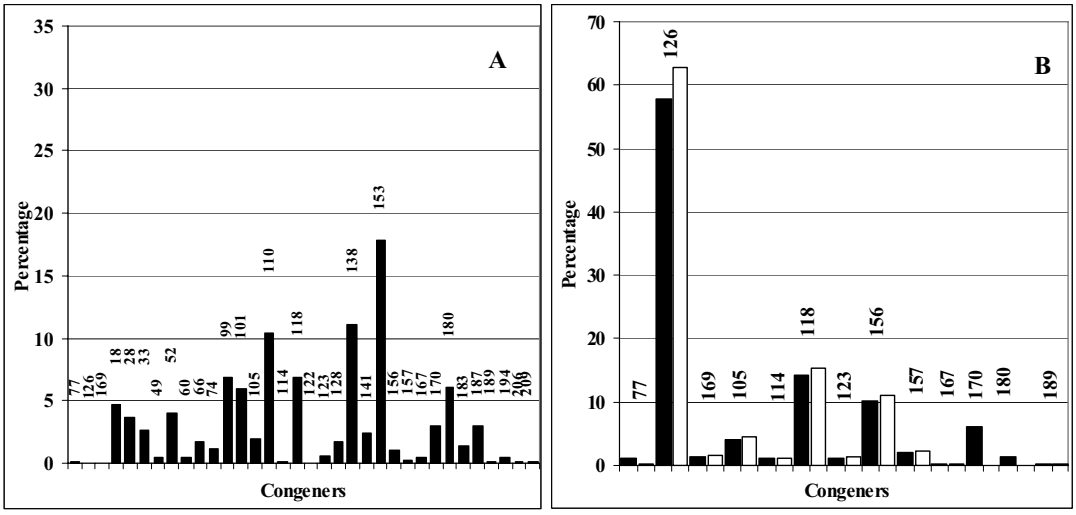

Fig. 2. Percentages of PCB congeners in the total diet basket. (A) Percentages from the sum of PCBs. (B) Percentages from toxic equivalents (PCB-TEq: black bars; WHO_{PCB}-TEq: white bars).

The concentration of the sum of PBDEs ranged from 0.82 to 850 pg/g fw, and similarly to PCDD/Fs and PCBs, the fish basket had the highest concentration of PBDEs. The congener profile of PBDEs in the total diet basket is illustrated in Fig. 3. The main contributor to the profile was PBDE 47, followed by congeners PBDE 99 and 100, which was also the case in the following baskets; fish, potato products, fruits and berries, and beverages, spices, sweets. In all other baskets, the main contributor to the profile was PBDE 99.

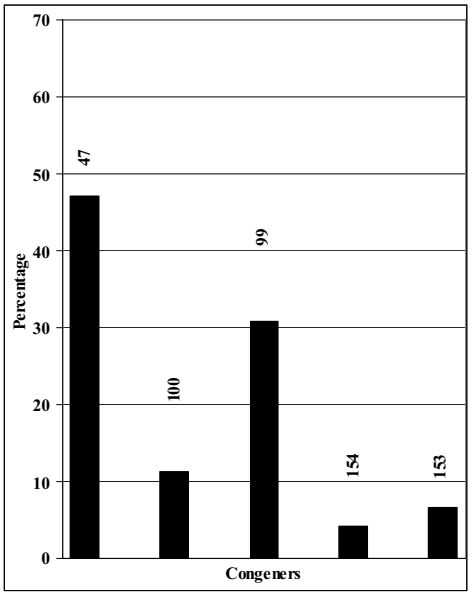

Fig. 3. Percentages of PBDE congeners in the total diet basket.

The average daily intakes of the sum of PCDD/Fs and PCBs, the corresponding toxic equivalents, and of the sum of PBDEs are presented in Table 3. Intakes calculated with lower bound concentrations were higher in the total diet basket than in the sum of the individual baskets. The sum of PCDD/Fs intake was 500 pg/day in the total diet basket this being 9% higher than the intake calculated from the sum of individual baskets (460 pg). In dioxin TEqs, the difference was not so evident in I-TEqs 55 versus 53 pg/day and in $WHO_{PCDD/F}$-TEqs 58 versus 57 pg/day, respectively. The difference in lower bound intakes between the total diet basket and the sum of individual baskets was the largest, 19%, in the sum of PCBs, i.e. either 1200 or 1010 ng/day, respectively. In PCB-TEq and WHO_{PCB}-TEq, the difference was about 9% (60 versus 55 and 56 versus 51 pg/day, respectively). Upper bound sum of PCBs, PCB-TEq, and WHO_{PCB}-TEq intakes in the total diet basket and in the sum of the individual baskets were similar to lower bound intakes. For dioxins, upper bound intakes calculated from the sum of

individual baskets were higher than the corresponding intake from the total diet basket. The difference in the sum of PCDD/Fs was 5%, in I-TEq 17%, and in $WHO_{PCDD/F}$-TEq 25%. The lower and upper bound intakes of the sum of PBDEs were similar, around 44 ng/day.

The contribution of fish to daily intake was overwhelming. In the lower bound intake of $WHO_{PCDD/F}$-TEq, the fish basket accounted for 95% of the daily intake and with WHO_{PCB}-TEq the intake was 80%. When calculating shares in the upper bound intakes, fish accounted for 71% of the dioxin TEq intake while the contribution to the PCB TEqs remained at 80%. In the sum of PCDD/Fs the major contributors to daily intake were fish (30%), fats (24%), meat and eggs (16%), and cereal products (12%). In addition to fish, three other major contributors to the sum of PCBs were cereal products (7%), meat and eggs (6%), and beverages, spices, sweets (6%). Over half (53%) of the sum of PBDEs intake came from the fish basket, followed by fats (17%), beverages, spices, sweets (9%), and cereal products (6%).

5. DISCUSSION

Lower bound I-TEq intake in the sum of individual baskets in this study (53 pg/day) was 15% higher than the corresponding intake in our previous study with the SSIF-method (46 pg/day) (Kiviranta et al. 2001). The corresponding upper bound intake in these two studies were more comparable (68 pg I-TEq/day versus 65 pg/day, respectively). In PCB-TEqs lower and upper bound intakes were comparable between both studies (55 and 53, and 56 and 54 pg PCB-TEq/day, respectively).

In this study, the main contributor, i.e. fish, accounted for 94% of the lower bound dioxin I-TEq intake while in the previous study the contribution of fish was 82%. The contribution of milk products and meat and eggs to the lower bound intake of I-TEqs was 0.93 and 2.0% in this study compared to 8 and 9% in the previous study. Using the upper bound I-TEq values, the contributions of different kind of foodstuffs were alike between the studies (73, 4%, 5%, and 18% for fish, milk products, meat and eggs, and others, respectively in this study versus 59%, 15%, 13%, and 9%, respectively, in the previous study). The contribution of the different kinds of foodstuffs to lower or upper bound PCB-TEq intake between the studies were rather similar and comparable (80%, 6%, 6%, and 8% for fish, milk products, meat and eggs, and others, respectively in this study versus 81%, 12%, 6%, and 1%, respectively in the previous study). The differences in lower bound I-TEq and in percentages of fish, dairy, and meat products of dioxin intake between our two studies are mainly due to the contribution of fish. In this study, the fish basket I-TEq

concentration was 1.8 pg/g, while the corresponding concentration (weighed by fish species consumption) in our previous study would be 1.3 pg/g. In addition, in the dairy and meat products baskets, the pooling of a large amount of different food items into the basket seemed to cause a critical dilution of low chlorinated dioxin analytes (1,2,3,7,8-PeCDD and 2,3,4,7,8-PeCDF) leading to lower I-TEq concentrations in these baskets compared to the corresponding food groups in our previous study. The PCB concentrations were much higher compared to the dioxin concentrations, and therefore there were only minor differences in the concentrations of PCB TEqs and the contributions of different food groups to the daily intake between our two studies.

The total diet basket seems to be the most reliable choice to assess the daily intake of these contaminants. Especially with respect to dioxin intakes, the numerous congeners with concentrations below LOQ in the individual baskets result in an underestimation of the lower bound intakes and an overestimation of the upper bound intakes.

Table 4 provides an overview of the average daily dietary intakes of dioxin- and PCB TEqs of adult populations from a number of countries. In addition, the food groups that contribute most to the intake of dioxins are presented. It is a difficult task to compare the results of intake estimations between countries because there are notable differences in the analytical methods e.g. upper bound versus lower bound concentrations used and set of TEFs utilized. There are differences between studies in collection methods and number of foods analysed, and differences in the means to study food consumpion. The daily intake of dioxins ranged between 29 pg I-TEq in Norway (SCOOP, 2000) and 104 pg $WHO_{PCDD/F}$-TEq in the USA (Schecter et al., 2001), and of PCBs from 31 pg WHO_{PCB}-TEq in Sweden (Lind et al., 2002) to 110 PCB-TEq in Norway. The recent Finnish TEq estimates of daily intakes (46-61 pg in dioxins and 51-60 in PCBs) were within these ranges reported from other countries. The Finnish daily intake of $WHO_{PCDD/F}$-TEq together with WHO_{PCB}-TEq per body weight (bw) was 1.5 pg/kg bw in this study which is at the lower end of the tolerable daily intake (TDI) range set by WHO, 1-4 pg TEq/kg bw (Van Leeuwen and Younes, 2000). None of the reported daily intakes in Table 4 exceeded the WHO TDI upper range value. The TWI of TEqs in Finland was 10.5 pg WHO-TEq/kg bw which is also below the highest recommended TWI value of 14 pg WHO-TEq/kg bw given by EU (EC/SCF 2001). In the future, analyses using distributional information for consumption data are needed in order to assess the percentage of Finns exceeding the TWI.

Depending on the method used in the calculations, in Finland fish accounted for from 63% up to 94 % of the daily intake of dioxins. A rather similar food contribution profile exists in Japan where 71% of the intake comes from fish (Tsutsumi et al., 2001). Fish are also the major

source of dioxin intake in Norway, Sweden, and Italy, but there exist a common trend in central and southern Europe for the dairy and meat/poultry food groups to be the most significant food groups in the intake of dioxins. That was also the case with the USA. The contribution of the food group "other" is very difficult to compare between the countries because of the large differences in food groups analysed in individual studies.

Intake estimations of PBDEs have been reported from Canada (Ryan and Patry, 2001), Sweden (Lind et al., 2002), the United Kingdom (Wijesekera et al., 2002), and the Netherlands (Winter-Sorkina et al., 2003). In Canada, the daily intake was estimated to be 44 ng, which is similar to the results of this study. The food group which contributed most to the daily intake was different in Canada, 70% came from meat products, 6% from dairy products, and only 3% from fish. The corresponding contributions in this study were 4%, 3% and 55%, respectively. In Sweden the daily intake of PBDEs has been assessed to be 31 ng. The contribution of fish to the intake in Sweden was similar to that reported in this study about 58%, and dairy and meat products accounted for about 10% in Sweden. If we exclude cereals, potato products, fruits and vegetables, and beverages from the intake calculations of PBDEs from our study, the daily intake would be 33 ng, which is quite close to the value in the Swedish study, which did not evaluate these food groups. The contribution (9%) of the food group beverages, spices and sweets suggests that in PBDE intake there might be relevant exposure sources other than animal products. In the UK, the average daily lower bound intake was assessed to be 90.5 ng, and it was estimated that this would contribute 73% of the overall daily exposure. The lower bound daily intake of PBDEs in the Netherlands was estimated to be 13 ng, while the medium bound was assessed to be 185 ng (Winter-Sorkina et al., 2003). The difference between the intake estimates in the Netherlands was due to large number of congeners below the detection limits and the relatively high detection limits of some samples.

6. CONCLUSIONS

Two different methods to assess dioxin and PCB intakes, i.e. either by analyzing food baskets (MBM-method) or by analyzing separate food items (SIFF-method), gave quite similar results. This implies that on average, the exposure in Finland is rather stable.

The assessed average daily TEq intake, 1.5 pg/kg bw, was at the lower end of the tolerable daily intake (TDI) range for PCDD/Fs and PCBs set by WHO (1-4 pg WHO-TEq/kg

bw), and the assessed weekly intake, 10.5 pg WHO-TEq/kg bw was below the highest recommended weekly intake value of 14 pg WHO-TEq/kg bw given by EU.

The intake of PBDEs were assessed for the first time in Finland and these were comparable to intakes in Canada and Sweden. The contribution profile of PBDEs suggested that there might be a difference in human exposure sources of PBDEs when compared to dioxins and PCBs.

Table 3.

Average intakes of PCDD/Fs, dioxin toxic equivalents, PCBs, PCB toxic equivalents, and PBDEs from 10 market baskets and total diet basket as pg/day.

Market baskets	Intake, pg/day			Intake, pg/day			Intake, pg/day
	Sum of PCDD/Fs	I-TEq	$WHO_{PCDD/F}$-TEq	Sum of PCBs x 10^3	PCB-TEq	WHO_{PCB}-TEq	Sum of PBDEs x 10^3
(1) Liquid milk products	**2.4 (0.53)**	**0.40 (0.76)**	**0.40 (0.7)**	**30 (3.0)**	**1.8 (3.3)**	**1.7 (3.4)**	**0.35 (0.8)**
	16 (2.6)	1.3 (2.0)	1.5 (2.0)	30 (3.0)	1.8 (3.3)	1.7 (3.4)	0.86 (1.9)
(2) Solid milk products	**1.8 (0.40)**	**0.089 (0.17)**	**0.089 (0.16)**	**24 (2.4)**	**1.4 (2.5)**	**1.3 (2.5)**	**1.1 (2.6)**
	17 (2.8)	1.4 (2.0)	1.6 (2.1)	24 (2.4)	1.4 (2.5)	1.3 (2.5)	1.3 (2.9)
(3) Fish	**150 (33)**	**50 (94)**	**54 (95)**	**690 (68)**	**44 (80)**	**41 (80)**	**23 (55)**
	150 (25)	50 (73)	54 (71)	690 (68)	44 (79)	41 (80)	23 (52)
(4) Meat and eggs	**72 (16)**	**1.1 (2.0)**	**1.0 (1.8)**	**62 (6.1)**	**3.1 (5.7)**	**2.8 (5.6)**	**1.8 (4.2)**
	94 (16)	3.4 (5.0)	3.9 (5.2)	62 (6.1)	3.1 (5.6)	2.8 (5.5)	2.0 (4.5)
(5) Fats	**110 (23)**	**0.41 (0.77)**	**0.31 (0.55)**	**27 (2.6)**	**1.6 (3.0)**	**1.5 (3.0)**	**6.5 (15)**
	160 (26)	6.7 (9.8)	7.7 (10)	28 (2.8)	1.8 (3.2)	1.6 (3.2)	7.9 (18)
(6) Cereal products	**56 (12)**	**0.78 (1.5)**	**0.74 (1.3)**	**73 (7.2)**	**1.3 (2.3)**	**1.1 (2.2)**	**2.8 (6.6)**
	70 (12)	2.3 (3.4)	2.6 (3.5)	73 (7.2)	1.3 (2.4)	1.2 (2.3)	2.8 (6.2)
(7) Potato products	**3.7 (0.81)**	**0.0037 (0.0071)**	**0.00037 (0.00066)**	**4.8 (0.47)**	**0.092 (0.17)**	**0.081 (0.16)**	**0.16 (0.4)**
	7.2 (1.2)	0.40 (0.59)	0.47 (0.62)	4.8 (0.47)	0.10 (0.19)	0.092 (0.18)	0.17 (0.4)
(8) Vegetables	**24 (5.3)**	**0.14 (0.26)**	**0.12 (0.21)**	**31 (3.1)**	**1.1 (1.9)**	**0.80 (1.6)**	**1.9 (4.5)**
	26 (4.2)	0.50 (0.73)	0.55 (0.73)	31 (3.0)	1.1 (1.9)	0.80 (1.6)	1.9 (4.3)
(9) Fruits and berries	**31 (6.7)**	**0.18 (0.35)**	**0.16 (0.29)**	**12 (1.2)**	**0.53 (0.82)**	**0.39 (0.77)**	**0.85 (2.0)**
	38 (6.3)	1.15 (1.7)	1.3 (1.7)	12 (1.2)	0.48 (0.87)	0.42 (0.82)	0.93 (2.1)
(10) Beverages, spices, sweets	**7.5 (1.6)**	**0.12 (0.24)**	**0.12 (0.22)**	**58 (5.7)**	**0.33 (0.6)**	**0.30 (0.59)**	**3.9 (9.2)**
	24 (4.0)	1.5 (2.3)	1.8 (2.4)	58 (5.7)	0.40 (0.73)	0.37 (0.73)	4.0 (8.8)
Sum of baskets	**460**	**53**	**57**	**1010**	**55**	**51**	**43**
	610	68	75	1010	56	51	45
Total diet basket	**500**	**55**	**58**	**1200**	**60**	**56**	**44**
	580	58	60	1200	60	56	44

Intakes with lower bound concentrations are shown in bold and non-bold fonts represent the corresponding upper bound intakes. Percentage of a basket of the intake in parenthesis.

Table 4.
Average daily intakes of dioxin TEqs, PCB TEqs as pg and (pg/kg bw), and contributions of different food groups to the dioxin exposure.

Country, study period	Daily intakes, pg, (pg/kg bw)					% contribution of foods from dioxins					Ref.
	I-TEq	$WHO_{PCDD/F}$-TEq	PCB-TEq	WHO_{PCB}-TEq	Method[a]	Dairy	Meat, poultry	Eggs	Fish	Other[b]	
Finland, 1999	55 (0.72)	58 (0.76)	60 (0.79)	56 (0.74)	0	2	2[c]		94	2	This study
Finland, 1999	58 (0.76)	60 (0.79)	60 (0.79)	56 (0.74)	LOQ	14	5[c]		72	9	This study
Finland, 1999	46 (0.61)		53 (0.70)		0	8	7	2	82	1	Kiviranta et al., 2001
Finland, 1999	61 (1.01)		51 (0.84)		LOQ	16	6	4	63	11	SCOOP, 2000
Japan, 2000		82 (1.64)		79 (1.59)	0.5*LOQ	2	12[c]		71	15	Tsutsumi et al., 2001
Norway, 1997	29		110		LOQ	22	14	12	46	6	SCOOP, 2000
Korea, 1999	30 (0.51)				unknown	1	4	5	39	51	Kim et al., 2000
Belgium, 2001		65			0	30	31		39		Focant et al., 2002
Sweden, 1999		44 (0.62)		31 (0.43)	0.5*LOQ	19	15	1	36	29	Lind et al., 2002
Sweden, 1999	68 (1.06)		63 (0.85)		LOQ	19	31	2	34	14	SCOOP, 2000
Italy, 1996	45 (0.74)				0.5*LOQ	26	32	7	35		SCOOP, 2000
Spain, 2000	78	95			0.5*LOQ	27	13	2	30	28	Llobet et al., 2003
China, 2000		72			unknown	16	35	21	28		Wu et al., 2002
France, 1999	97 (1.45)				LOQ	33	13	2	26	26	SCOOP, 2000
Germany, 1998	51 (0.73)				0.5* LOQ	39	30	11	11	9	SCOOP, 2000
The Netherlands, 1999		45 (0.65)		46 (0.58)	0	24	21	5	10	40	Freijer et al., 2001
The Netherlands, 1991	82		81		LOQ	39	20	4	2	35	SCOOP, 2000
The United Kingdom, 2001		(0.4)		(0.5)	LOQ	44	18	1	6	31	FSA report 38/03
The United Kingdom, 1992	88 (1.26)		57 (0.81)		LOQ	25	20	4	6	45	SCOOP, 2000
USA, 1995	29	104 (1.66)		42 (0.67)	0.5*LOQ	29	30	7	6	28	Schecter et al., 2001

[a]method of denoting concentrations of unquantified congeners in intake calculations: 0=lower bound, 0.5*LOQ=medium bound, LOQ=upper bound
[b]other = e.g. cereals and cereal products, vegetables, fruit, vegetable fats and oils
[c]includes meat, poultry, and eggs

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CHAPTER 4

PCDD/Fs AND PCBs IN BALTIC HERRING DURING THE 1990's

Hannu Kiviranta [a,*], Terttu Vartiainen [a,b], Raimo Parmanne [c], Anja Hallikainen [d], Jaana Koistinen [a]

[a] Department of Environmental Health, National Public Health Institute, P. O. Box 95, FIN-70701 Kuopio, Finland

[b] University of Kuopio, P. O. Box 1627, FIN-70211 Kuopio, Finland

[c] Finnish Game and Fisheries Research Institute, P. O. Box 6, FIN-00721 Helsinki, Finland

[d] National Food Agency, P. O. Box 28, FIN-00581 Helsinki, Finland

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1. ABSTRACT

Baltic herring samples caught from the Baltic Sea during the spring periods of 1993-94 and 1999 were analysed for polychlorinated dibenzo-*p*-dioxins (PCDD), polychlorinated dibenzofurans (PCDF), and polychlorinated biphenyls (PCB). For analyses, 1570 individual herring were combined to 120 pools. Correlations between concentrations of congeners 1,2,3,7,8-PeCDD, 1,2,3,7,8-PeCDF, 2,3,4,7,8-PeCDF, and 2,3,4,6,7,8-HxCDF, and age of herring were the strongest ($r > 0.8$) followed by correlations between PCB congeners PCB 105, 118, 126, 156, 169 and 180 ($r > 0.7$), and age of herring. Due to higher fat percentage in herring in the Gulf of Bothnia the concentrations of PCDD/Fs and PCBs on fresh weight (fw) basis were higher than in herring in the Gulf of Finland. The concentrations of $WHO_{PCDD/F}$-TEQs ranged from 1 to 27 pg/g fw, depending on the age and catchment area of herring, and concentrations of WHO_{PCB}-TEQs reached 32 pg/g fw. Between the two studied time points no clear downward trend in concentrations was observed.

2. INTRODUCTION

Fish and fish products play a significant role in the Finnish dietary intake of polychlorinated dibenzo-*p*-dioxins, polychlorinated dibenzofurans (PCDD/F; dioxins), and

polychlorinated biphenyls (PCB). If one considers the PCDD/F intake, then fish and fish products accounted for 82%, and Baltic herring (*Clupea harengus* L.) alone 52% of the total intake. For PCBs, the contributions were similar although the data were not fully consistent (Kiviranta et al., 2001). In November 2001, the EU Council set maximum levels for PCDD/Fs in foodstuffs (EU, 2001), which will come into force on 1 July 2002. For fish and fishery products, the limit was set at 4 pg toxic equivalents ($WHO_{PCDD/F}$-TEQ) per gram of fresh weight (fw). Finland and Sweden were granted an exception to this value until 2006.

The total catch of Baltic herring by professional fishermen in Finland during the 1990s ranged from 51 000 to 98 000 tonnes of which about 25% was processed for human consumption, with the rest being used as feed for fur animals. The main catchment area was the southern part of the Gulf of Bothnia, the Bothnian Sea, which accounted for about 70% of the catch. About one third of the total catch was caught during the main spawning season in May-June (Finnish Game and Fisheries Research Institute, 2000, 2001).

Baltic herring populations in the Gulf of Finland and in the Gulf of Bothnia have distinctive characteristics which might affect the prevailing levels of PCDD/Fs and PCBs. Fat percentage varies substantially in populations throughout the year being highest in the autumn, and at its minimum in the spring during the main spawning season (Plorinja et al., 1975). The behaviour of the herring population in the Gulf of Finland differs from that of the shoals in the Gulf of Bothnia with respect to migration. Herring in the Gulf of Bothnia have a low migratory behaviour and can therefore be considered to be stationary fish. In the Gulf of Finland, young herring move within a limited area, whereas some of the sexually mature herring migrate over considerable distances as far as the southern Baltic Sea (Parmanne, 1990). The stock of cod (*Gadus morhua* L.), which is the main predator of sprat (*Sprattus sprattus* L.) and herring, has decreased in the Baltic Sea since the 1980's (ICES, 2001). This has led to an increase in numbers of sprat. Sprat compete with herring for food supplies and thus, the growth of herring has been retarded in the Gulf of Finland. This decline is evident when mean weight of herring in age groups is plotted as a function of time (Fig. 1). Another explanation for diminished growth of herring in the Gulf of Finland might be the temporal decline in the water salinity which may affect the amount, composition and availability of zooplankton suitable for herring (Lankov and Raid, 1997). No such major decline in growth of herring has taken place in the Gulf of Bothnia (ICES, 2001), where changes in salinity, zooplankton and in the abundance of cod and sprat have been less extensive than in the Gulf of Finland.

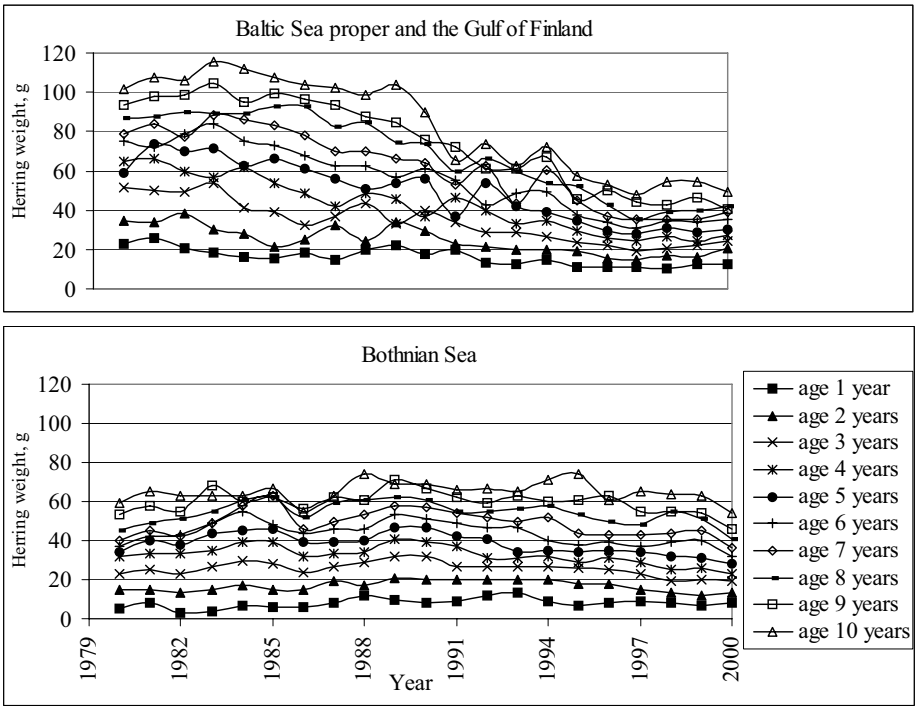

Fig 1. Mean weight of Baltic herring at age groups in Baltic Sea proper and the Gulf of Finland, and in the Bothnian Sea, southern part of the Gulf of Bothnia, from 1980 to 2000. (Data from ICES, 2001)

Prevailing levels of organochlorine pesticides and PCBs in Baltic Sea have been monitored intensively since the late 1960s, and a continuous decline of several organochlorines, including PCBs, in Baltic herring from 1978 to 1995 has been reported (Bignert et al., 1998). Consistent data for PCDD/Fs in Baltic herring is currently missing, but some clues can be derived from studies in which eggs of herring-consuming sea-birds have been studied (Odsjö et al., 1997; Schramm et al., 1997). In these studies, a downward trend in PCDD/F concentrations has been detected in guillemot (*Uria aalge*) eggs from the beginning of the 1970s to 1994, and a similar trend has been reported in herring gull (*Larus argentatus*) eggs from 1988 to 1993. The declining trends in all of the above studies were most intensive during the late 1970s and during the 1980s, but have started to level off at the beginning of the 1990s.

In this study of PCDD/Fs and PCBs in herring, the following main tasks were undertaken: (a) to determine the age correlation of concentrations; (b) to assess possible differences in concentrations between the Gulf of Finland and the Gulf of Bothnia; (c) to evaluate the time trend of concentrations during the 1990s.

3. MATERIALS AND METHODS

Herring sampling and pooling

Altogether 1573 Baltic herring were collected from 11 locations of the Baltic Sea during most vigorous spawning season of the fish (beginning of May to mid-June) in 1993-94 and 1999 (Fig. 2). In 1993-94 herring were caught mainly from the Gulf of Finland and only one catchment area was located in the Gulf of Bothnia. Weight (w) and length (l) of individual fish were measured and a condition factor (Cf) was calculated according to the equation:

$Cf = w\ l^{-3} * 100\ (g\ cm^{-3})$

Otoliths of herring were taken for age determination, n = 1194, in 1993-94 (ICES, 1998). Age was not determined in 1999 from otoliths, instead data (herring weight as function of age) from ICES (Fig. 1) were used for estimation of herring age.

Herring from 1993 to 1994 were pooled into 100 pools. The main determinants for pooling were age (2, 3, 4, 5, 6, 7, 8, 10, 12, 14, and over 15 years old), gender, catchment area (six areas, Fig. 2). Herring caught from the Gulf of Bothnia consisted of older herring (over 8 years), whereas the Gulf of Finland herring represented all age groups. Selective sampling on large herring in age groups 8 to >15 years resulted in non-randomized samples. These samples were collected in order to assess the maximum concentrations of PCDD/Fs and PCBs in Baltic herring and to compare concentrations in the Gulf of Finland and the Gulf of Bothnia. Pooling of herring (20 pools) caught in 1999 was based on two determinants: catchment area (nine areas, Fig. 2) and herring length (below 18.5 cm were "small" and over that "large"). Herring pools from catchment areas 8, 10, and 11 contained small and large herring from the Gulf of Finland (there was double sampling in area 10) and pools from areas 1, 2, 3, 5, and 6 consisted of small and large herring from the Gulf of Bothnia. In one area, Korsnäs, pooling was not done and the results are presented as an average of small and large herring. Numbers of individual herring in all pools varied from 1 to 34.

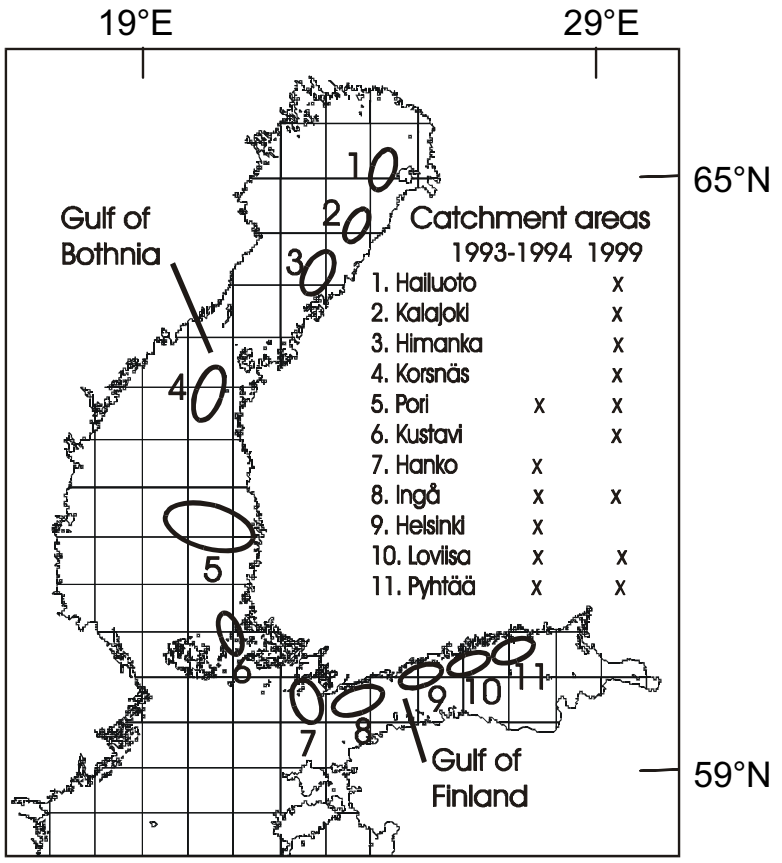

Fig 2. Catchment areas of Baltic herring in two sampling periods 1993-94 and 1999.

Analysis of PCDD/Fs and PCBs

Fats from cleaned (head, fins and gut removed), pooled, freeze-dried, and homogenized herring were Soxhlet extracted using toluene and the fat contents were determined gravimetrically. A previously described method was used for purification of samples and fractionation of PCDD/Fs, PCBs and non-*ortho* PCBs (co-PCB, co-planar PCB) (Kiviranta et al., 1999).

^{13}C-labeled internal PCDD/F standards (16 2,3,7,8-chlorinated PCDD/F congeners) were used for determination of the concentrations of 17 toxic PCDD/Fs. Toxic equivalents (TEQ) for PCDD/Fs were calculated with two different sets of toxic equivalency factors (TEF), the NATO factors (NATO/CCMS, 1988) gave I-TEQs, and the factors recommended by WHO in 1998 (van der Berg et al., 1998) for the $WHO_{PCDD/F}$-TEQ. Limits of determination (LOD) for PCDD/Fs were isomer dependent, and varied between 0.1 and 1 pg/g fat, and between 0.005 and 0.05 pg/g fw. In the calculations of TEQs, concentrations below LODs were considered as zero.

In 1993 co-PCBs (PCB 77, 126, and 169) were determined with corresponding ^{13}C-labeled internal standards. Other ^{13}C-labeled PCBs (PCB 80, 101, 153, 180), and PCB 30 were used to determine 13 other congeners (PCB 8, 18, 28/31, 52, 80, 101, 105, 118, 138, 153, 156,

180 and 181). In 1994 one additional co-PCB (PCB 81) and 21 additional other PCBs (PCB 33, 49, 51, 60, 66, 74, 99, 110, 114, 122, 123, 128, 141, 157, 167, 170, 183, 187, 189, 194, 206) were determined. Two PCBs were excluded from the set of 1993, (PCB 8 and 181). For herring caught in 1999, four additional ^{13}C-labeled internal standards (PCB 105, 138, 156, 194) were used in the analysis of other PCBs. Congeners measured were the same as in 1994, except that PCB 81 was excluded, and two other congeners (PCB 47 and 209) were included in the set of PCBs analysed. TEQs for PCBs were calculated with two different sets of TEFs, factors by Ahlborg et al. (1994) gave PCB-TEQs and factors by WHO gave WHO_{PCB}-TEQs (van der Berg et al., 1998). LODs for co-PCBs and other PCBs were 3 pg/g, and 0.2 ng/g fat, respectively, and 0.15 pg/g, and 0.01 ng/g fw. In the calculations of TEQs results below LODs were considered as zero.

The laboratory reagent and equipment blank samples were treated and analyzed by the same method as the actual samples, one blank for every five samples. Recoveries for internal standards ranged between 60 and 110%.

The laboratory of chemistry in the National Public Health Institute has participated in several international quality control studies for the analysis of PCDD/Fs, and PCBs in fish samples (IUPAC, 1995; IUPAC, 1998; IUPAC, 2000; Lindström et al., 2000; Becher et al., 2001). Since 1996, the laboratory has been an accredited testing laboratory (No. T077) in Finland (current standard: EN ISO/IEC 17025). The scope of accreditation includes PCDD/Fs, PCBs, and co-PCBs from tissue samples.

Statistical Analysis

Statistical analyses were carried out by means of SPSS software (for Windows, release 9.0.1). Before the statistical tests, all results were transformed to a natural logarithm (ln) scale in order to ensure that the concentrations are as normally distributed as possible. Two tailed Pearson's correlation analysis was used to determine if the studied correlations were statistically significant. For comparisons of two groups, the Mann-Whitney U nonparametric test was used to test the statistical significances of the differences of concentrations between groups. One way analysis of variance (ANOVA) or Kruskal-Wallis H test were used to compare the differences of concentrations between multiple groups.

4. RESULTS AND DISCUSSION

No significant sex-related differences in PCDD/F or PCB concentrations were found (either calculated per fresh weight or fat) in herring. Similarly, there were no significant differences in the concentrations between the catchment areas in the Gulf of Finland, or in the Gulf of Bothnia.

PCDD/F and PCB concentrations in 1993-94

Concentrations of PCDD/Fs, marker-, and PCB-congeners with dioxin-like toxicity along with fat percentages, weights, lengths, and Cfs of herring caught in 1993-94 are presented according to age groups in Tables 1 and 2. In age groups 2-7, the fat percentage varied between 0.41 and 4.8, and the differences between ages were not statistically significant. Therefore the proportional differences between age groups in concentrations on a fresh weight and on fat basis were negligible. Weight and length increased significantly in age groups 2-4 years, but in groups older than 4 years no significant differences existed. This implies that the assessment of a herring's age based on its size is very difficult.

All PCDD congeners, and penta-, and hexachlorinated PCDFs (except for 1,2,3,7,8,9-HxCDF) were bioaccumulating in herring in age groups 2-7 (Table 1). Correlations for fresh weight were linear and the strongest correlations ($r > 0.8$) were measured for 1,2,3,7,8-PeCDD, 1,2,3,7,8-PeCDF, 2,3,4,7,8-PeCDF, and 2,3,4,6,7,8-HxCDF. For these congeners, concentration differences between age groups were most evident. Although significant, the correlations between concentrations of 1,2,3,7,8,9-HxCDD, 1,2,3,4,6,7,8-HpCDD, and OCDD and age were poor ($r < 0.5$). The strongest correlation with age was noted for 2,3,4,7,8-PeCDF ($r = 0.92$) followed by both I-TEQ and $WHO_{PCDD/F}$-TEQ ($r = 0.90$). To evaluate herring TEQ in fresh weight according to age, the first-degree equation for the linear line fit was calculated ($y = 0.962 x - 0.77$ for I-TEQ, and $y = 1.08 x - 0.845$ for $WHO_{PCDD/F}$-TEQ). A rule of thumb was created: every year of a herring's life led a rise of one TEQ unit on fresh weight basis.

All PCB congeners (except for PCB 28/31 and 77) showed bioaccumulation, although to a lesser extent than dioxins (Table 2). The strongest correlations with age had congeners PCB 105, 118, 126, 156, 169 and 180 ($r > 0.7$), whereas the marker congeners (PCB 52, 101, 138, and 153) that do not contribute to TEQs had lower correlations ($r < 0.6$). PCB 169 showed the strongest correlation with age ($r = 0.89$) followed by PCB 180 ($r = 0.88$). No rule of thumb for

Table 1.
Medians (range) of fat percentages, weights, lengths, condition factors (Cf), concentrations (**pg/g fw** and pg/g fat) of PCDD/Fs and toxic equivalents of PCDD/Fs in different age groups of Baltic herring, caught in 1993-94 in the Gulf of Finland and in the Gulf of Bothnia.

	Gulf of Finland							Gulf of Bothnia
Age	2	3	4	5	6	7	8-> 15	8-> 15
Amount pools	10	17	17	10	10	10	9	9
Fat%	2.6 (1.3-3.7)[a]	1.8 (0.41-3.3)[a]	1.9 (0.61-4.8)[a]	1.9 (1.1-3.2)[a]	1.7 (0.99-3.5)[a]	1.9 (0.49-3.4)[a]	5.6 (1.8-9.2)*	11 (7.7-14)
Weight	16 (13-16)[a]	19 (14-29)[b]	26 (22-34)[c]	28 (23-29)[c]	30 (23-35)[c]	31 (23-35)[c]	150 (96-250)	160 (58-360)
Length	13.7 (13.2-14.1)[a]	14.6 (13.6-17.3)[b]	16.9 (16.5-17.7)[c]	16.9 (16.7-17.4)[c]	17.5 (16.8-18.3)[c]	17.4 (16.8-18.1)[c]	28.0 (23.7-32.4)	29.0 (20.7-36.4)
Cf	0.58 (0.56-0.69)[a]	0.58 (0.52-0.7)[a]	0.57 (0.48-0.65)[a]	0.55 (0.49-0.61)[a]	0.56 (0.48-0.63)[a]	0.56 (0.46-0.68)[a]	0.71 (0.62-0.77)	0.7 (0.62-0.74)
2378-TCDD	**0.11 (ND-0.15)[a]**	**0.14 (0.051-0.31)[a, b]**	**0.19 (0.11-0.52)[b, c]**	**0.29 (0.15-0.44)[c, d]**	**0.33 (0.25-0.76)[d]**	**0.31 (0.087-0.51)[c,d]**	**0.88 (0.59-1.2)***	**1.7 (0.81-3.5)**
	4.1 (ND-7.0)[a]	8.2 (4.0-20)[b]	10 (4.5-32)[b, c]	15 (8.0-24)[b, c]	17 (11-55)[c]	18 (7.2-21)[c]	17 (9.6-32)	17 (9.1-29)
12378-PeCDD	**0.26 (0.19-0.45)[a]**	**0.45 (0.28-0.79)[b]**	**0.65 (0.47-1.4)[c]**	**1.2 (0.71-1.5)[d]**	**1.4 (1.2-2.5)[d]**	**1.4 (0.77-2.8)[d]**	**4.0 (2.3-6.2)**	**4.9 (1.4-7.5)**
	13 (7.0-17)[a]	24 (13-68)[b]	38 (16-84)[b, c]	64 (28-110)[c, d]	84 (38-180)[d]	82 (41-160)[d]	74 (35-190)	59 (15-61)
123478-HxCDD	**0.013 (ND-0.042)[a]**	**0.022 (ND-0.067)[a]**	**0.013 (ND-0.083)[a, b]**	**0.07 (0.013-0.14)[b]**	**0.095 (0.031-0.14)[b]**	**0.075 (0.049-0.12)[b]**	**0.18 (ND-0.35)**	**0.36 (0.078-0.75)**
	0.57 (ND-1.8)[a]	1.7 (ND-7.7)[a, b]	0.53 (ND-4.4)[a, b, c]	3.9 (0.54-8.1)[b, c]	4.4 (0.88-10)[c]	4.7 (2.0-12)[c]	3.2 (ND-6.7)	3.4 (0.87-6.0)
123678-HxCDD	**0.35 (0.24-0.68)[a]**	**0.51 (0.27-1.1)[a]**	**0.65 (0.38-1.8)[a, b]**	**1.5 (0.53-2.3)[b, c]**	**1.3 (0.47-3.8)[c]**	**1.5 (0.61-2.9)[c]**	**3.1 (1.4-8.2)**	**3.4 (ND-6.7)**
	15 (6.9-25)[a]	30 (11-75)[b]	42 (14-110)[b]	71 (24-170)[b, c]	65 (31-270)[b, c]	93 (28-150)[c]	60 (18-230)	43 (ND-54)
123789-HxCDD	**0.036 (0.019-0.1)[a,b]**	**0.028 (0.006-0.1)[b]**	**0.064 (0.02-0.11)[a, b]**	**0.099 (0.024-0.14)[a]**	**0.10 (ND-0.25)[a]**	**0.065 (0.03-0.13)[a,b]**	**0.029 (ND-0.18)**	**0.17 (ND-0.69)**
	1.6 (0.59-2.1)[a]	1.7 (0.2-7.3)[a]	2.9 (0.76-6.9)[a]	5.7 (1.2-9.6)[a]	5.9 (ND-18)[a]	4.3 (1.4-21)[a]	0.62 (ND-3.2)	1.5 (ND-5.6)
1234678-HpCDD	**0.12 (0.052-0.23)[a]**	**0.15 (0.05-0.88)[a, b]**	**0.17 (0.089-2.8)[a, b]**	**0.28 (0.11-1.2)[a, b]**	**0.25 (0.13-1.2)[a, b]**	**0.48 (0.11-1.2)[b]**	ND	**0.20 (ND-0.92)**
	4.8 (1.8-9.4)[a]	11 (1.9-53)[a, b]	13 (2.4-140)[a, b]	13 (4.0-74)[a, b]	16 (5.6-87)[a, b]	22 (11-140)[b]		1.7 (ND-11)
OCDD	**0.29 (ND-0.98)[a]**	**0.60 (ND-3.6)[a]**	**0.62 (0.18-13)[a]**	**0.81 (0.25-7.2)[a]**	**0.78 (0.29-6.5)[a]**	**1.8 (0.25-6.5)[a]**	**0.62 (0.35-1.1)***	**2.0 (0.50-7.5)**
	17 (ND-31)[a]	34 (ND-230)[a, b]	41 (5.8-640)[a, b]	37 (10-470)[a, b]	38 (14-450)[a, b]	80 (27-450)[b]	9.3 (3.8-50)	16 (3.9-97)
Dioxins	**1.4 (0.66-2.2)[a]**	**2.6 (0.72-5.7)[a, b]**	**2.3 (1.6-18)[b, c]**	**4.6 (1.9-12)[b, c]**	**4.7 (3.1-11)[c]**	**5.4 (1.9-12)[c]**	**8.9 (5.2-16)**	**18 (4.8-24)**
	60 (24-84)[a]	150 (36-420)[b]	170 (45-860)[b, c]	210 (78-800)[b, c]	280 (120-810)[b, c]	320 (120-920)[c]	170 (78-500)	160 (54-240)
2378-TCDF	**1.8 (0.90-2.4)[a]**	**1.3 (0.32-2.5)[a]**	**1.5 (0.27-3.1)[a]**	**1.4 (0.59-2.9)[a]**	**1.3 (0.59-3.1)[a]**	**0.64 (0.26-2.6)[a]**	**4.1 (1.1-6.2)***	**9.6 (6.5-14)**
	69 (37-85)[a]	75 (38-110)[a]	72 (31-100)[a]	80 (43-95)[a]	74 (40-87)[a]	56 (21-95)[a]	72 (54-93)	84 (71-130)
12378-PeCDF	**0.23 (0.18-0.38)[a]**	**0.35 (0.24-0.55)[a]**	**0.62 (0.36-1.0)[b]**	**0.83 (0.62-1.3)[b, c]**	**1.1 (0.90-1.9)[c]**	**0.91 (0.063-1.9)[b, c]**	**1.6 (0.99-4.0)***	**4.9 (0.54-38)**
	10 (6.4-18)[a]	22 (12-57)[b]	32 (19-59)[c]	46 (33-81)[c, d]	68 (37-110)[d]	59 (13-97)[c, d]	27 (18-75)	47 (6.0-340)
23478-PeCDF	**1.5 (1.2-2.0)[a]**	**2.5 (1.6-3.3)[b]**	**3.7 (2.0-5.1)[c]**	**5.5 (4.4-8.1)[d]**	**8.2 (7.3-9.7)[e]**	**8.0 (4.9-15)[e]**	**25 (12-30)***	**36 (12-55)**
	63 (42-94)[a]	120 (81-380)[b]	200 (100-330)[b, c]	320 (190-480)[c, d]	440 (220-850)[d]	490 (260-1010)[d]	460 (190-1400)	280 (130-440)
123478-HxCDF	**0.12 (0.043-0.79)[a, b]**	**0.11 (0.045-0.19)[a]**	**0.25 (0.073-0.62)[a,b]**	**0.28 (0.17-0.53)[a, b]**	**0.41 (0.24-2.0)[a, b]**	**0.37 (0.16-1.2)[a, b]**	**0.36 (ND-0.84)***	**0.99 (ND-3.1)**
	4.7 (1.8-29)[a]	5.9 (2.2-21)[a]	12 (3.7-44)[a, b]	17 (7.6-25)[a, b]	23 (6.6-68)[b]	23 (8.9-60)[b]	5.4 (ND-18)	9.4 (ND-25)
123678-HxCDF	**0.09 (0.057-0.46)[a]**	**0.17 (0.08-0.27)[a, b]**	**0.22 (0.07-0.35)[a,b,c]**	**0.40 (0.095-1.1)[a,b,c]**	**0.50 (0.075-2.6)[c]**	**0.48 (0.034-0.96)[c]**	**1.1 (0.35-2.3)**	**2.0 (0.25-5.0)**
	4.0 (1.8-13)[a]	9.6 (4.4-27)[a, b]	12 (4.9-20)[a, b]	20 (7.6-51)[b]	33 (3.5-88)[b]	34 (2.0-80)[b]	16 (5.9-130)	23 (2.8-40)
234678-HxCDF	**0.17 (0.09-0.25)[a]**	**0.28 (0.13-0.42)[b]**	**0.40 (0.17-0.66)[c]**	**0.65 (0.35-0.98)[d]**	**0.86 (0.69-1.3)[d]**	**0.66 (0.41-1.5)[d]**	**1.3 (0.80-2.5)***	**3.0 (ND-5.8)**
	6.3 (3.3-12)[a]	16 (6.4-38)[b]	19 (12-41)[b, c]	35 (19-50)[c, d]	44 (24-94)[d]	43 (21-96)[d]	23 (12-93)	34 (ND-47)
1234678-HpCDF	**0.21 (0.12-0.45)[a]**	**0.24 (0.11-0.60)[a]**	**0.24 (0.12-0.65)[a]**	**0.31 (0.11-0.63)[a]**	**0.27 (0.14-0.78)[a]**	**0.28 (0.13-0.59)[a]**	ND	ND
	9.7 (3.6-17)[a]	19 (4.8-52)[a]	11 (3.5-48)[a]	15 (6.2-30)[a]	17 (5.9-57)[a]	17 (7.3-93)[a]		
OCDF	**0.07 (ND-0.36)[a]**	**0.091 (ND-0.36)[a]**	**0.055 (ND-0.35)[a]**	**0.057 (ND-0.27)[a]**	**0.082 (ND-0.23)[a]**	**0.15 (ND-0.41)[a]**	ND	ND
	2.6 (ND-11)[a]	5.0 (ND-39)[a]	3.0 (ND-20)[a]	2.9 (ND-17)[a]	4.6 (ND-15)[a]	5.7 (ND-82)[a]		

Table 1 (continued)

	Gulf of Finland							Gulf of Bothnia
Furans	**4.7 (2.8-5.4)** [a]	**5.1 (2.7-7.2)** [a]	**7.0 (3.3-11)** [b]	**9.9 (6.6-14)** [b, c]	**13 (11-20)** [c]	**12 (7.1-20)** [c]	**33 (19-40)***	**57 (21-110)**
	170 (110-220) [a]	280 (160-660) [b]	380 (220-570) [b, c]	560 (340-700) [c, d]	700 (390-1300) [d]	740 (430-1500) [d]	600 (340-1800)	440 (230-970)
Sum of PCDD/Fs	**6.0 (3.7-7.3)** [a]	**7.8 (3.5-11)** [a, b]	**10 (4.9-25)** [b, c]	**15 (8.6-22)** [c, d]	**18 (15-26)** [d]	**19 (9.0-31)** [d]	**43 (24-55)***	**67 (26-130)**
	240 (130-280) [a]	400 (191-950) [b]	570 (270-1200) [b, c]	790 (440-1500) [c, d]	940 (510-1900) [d]	1030 (550-2400) [d]	780 (430-2300)	680 (290-1100)
I-TEQ	**1.4 (0.93-1.5)** [a]	**1.9 (1.1-2.5)** [b]	**2.7 (1.5-3.7)** [c]	**4.3 (3.0-5.7)** [d]	**5.6 (5.0-7.7)** [e]	**5.5 (3.2-10)** [d, e]	**16 (8.6-21)***	**24 (8.3-39)**
	50 (34-72) [a]	110 (59-270) [b]	150 (77-240) [b, c]	240 (130-350) [c, d]	310 (160-570) [d]	330 (180-650) [d]	310 (140-880)	190 (92-310)
$WHO_{PCDD/F}$-TEQ	**1.5 (1.0-1.7)** [a]	**2.2 (1.3-2.8)** [b]	**3.0 (1.7-4.2)** [c]	**4.8 (3.3-6.4)** [d]	**6.3 (5.6-8.9)** [e]	**6.2 (3. 6-11)** [d, e]	**18 (9.8-24)***	**27 (9.0-42)**
	57 (38-79) [a]	120 (65-300) [b]	170 (86-280) [b, c]	270 (150-400) [c, d]	350 (180-650) [d]	370 (200-740) [d]	340 (160-970)	220 (100-340)

Statistical significance of concentration differences between age groups 2-7 (tested with one-way ANOVA, significant level $p < 0.05$) are distinguished by different letter as superscript. Differences between concentrations of age groups 8-> 15 were tested separately from age groups 2-7, and statistically significant differences (tested with Mann-Whitney U test, significant level $p < 0.05$) are denoted here with an asterisk.

ND = concentration below LOD

Concentrations of 1,2,3,7,8,9-HxCDF and 1,2,3,4,7,8,9-HpCDF were all below LOD

Table 2

Medians (range) of concentrations as **ng/g fw** and as ng/g fat of PCBs and as **pg/g fw** and as pg/g fat of co-PCBs and toxic equivalents of PCBs in different age groups of Baltic herring, caught in 1993-1994 in the Gulf of Finland and in the Gulf of Bothnia.

	Gulf of Finland							Gulf of Bothnia
Age	2	3	4	5	6	7	8-> 15	8-> 15
Amount pools	10	17	17	10	10	10	9	9
PCB 28/31	**0.44 (0.023-0.76)[a, b]** 16 (1.3-27) [a, b]	**0.067 (0.017-0.66) [a]** 4.2 (1.0-41) [a]	**0.71 (0.07-1.3) [a, b]** 35 (3.8-68) [a, b, c]	**0.71 (0.048-1.1) [a, b]** 32 (4.5-66) [a, b, c]	**0.63 (0.056-1.7)[a,b]** 30 (3.7-83) [a,b, c]	**0.56 (0.026-1.2)[a,b]** 30 (2.2-42) [a, b, c]	**1.5 (0.55-2.9)*** 28 (22-43)*	**4.5 (2.8-6.1)** 38 (33-54)
PCB 52	**0.68 (0.39-1.2) [a, b]** 30 (18-39) [a]	**0.69 (0.22-1.1) [a]** 37 (16-60) [a]	**1.4 (0.65-2.2) [a, b]** 73 (40-130) [b]	**1.6 (0.74-2.8) [a, b]** 76 (37-130) [b]	**1.3 (0.72-2.9) [a, b]** 72 (53-190) [b]	**1.2 (0.28-2.6) [a, b]** 69 (36-120) [b]	**6.1 (2.3-7.1)*** 97 (77-130)*	**17 (10-21)** 150 (130-180)
PCB 101	**1.3 (1.0-2.0) [a]** 57 (39-80) [a]	**1.9 (0.015-2.4) [a]** 110 (3.7-160) [a, b]	**2.6 (1.5-4.4) [a]** 130 (87-260) [b, c]	**2.6 (1.7-3.9) [a]** 150 (110-220) [b, c]	**2.8 (2.0-5.5) [a]** 160 (110-260) [b, c]	**4.0 (1.3-6.6) [a]** 240 (130-380) [c]	**22 (15-27)*** 360 (270-1100)	**41 (27-57)** 350 (310-470)
PCB 138	**3.1 (2.5-4.9) [a]** 140 (89-190) [a, c]	**4.4 (2.9-6.4) [a]** 270 (130-800) [a, b]	**5.2 (2.6-9.6) [a]** 230 (110-830)[b, c]	**4.1 (2.9-5.7) [a]** 180 (140-510) [a, b]	**5.3 (4.1-9.4) [a]** 280 (160-940) [a, b]	**9.3 (4.4-19) [b]** 610 (250-1300) [b,c]	**36 (20-44)*** 500 (370-2000)*	**83 (48-110)** 780 (600-960)
PCB 153	**0.96 (0.68-1.4) [a]** 39 (25-72) [a]	**1.0 (0.63-1.9) [a]** 65 (31-250) [a, b]	**1.3 (0.59-3.0) [a, b]** 60 (27-270) [a, b]	**0.87 (0.69-1.1) [a]** 41 (32-99) [a]	**1.1 (0.86-2.4) [a, b]** 62 (33-240) [a, b]	**2.0 (0.96-6.2) [b]** 120 (56-390) [b]	**23 (15-57)*** 370 (300-3100)*	**70 (41-87)** 640 (520-740)
PCB 180	**0.28 (0.22-0.32) [a]** 11 (7.2-17) [a]	**0.43 (0.3-0.5) [b]** 23 (14-72)[b]	**0.55 (0.41-0.66)[c]** 28 (13-68) [b]	**0.62 (0.53-0.79) [c, d]** 34 (24-62) [b, c]	**0.75 (0.66-1.0) [d, e]** 43 (28-93) [b, c]	**1.0 (0.5-1.8) [e]** 61 (30-110) [c]	**17 (8.7-31)*** 310 (130-910)	**39 (25-54)** 370 (290-570)
Sum of Marker PCBs	**7.1 (5.2-10) [a]** 290 (200-400) [a]	**9.0 (4.9-11) [a, b]** 490 (280-1200)[b]	**12 (6.9-19) [b, c]** 510 (310-1600) [b, c]	**11 (8.3-14) [a, b]** 560 (390-990) [a, b, c]	**13 (9.3-18) [b, c]** 640 (420-1600) [b, c]	**18 (9.7-36) [c]** 1200 (550-2200) [c]	**120 (64-130)*** 1800 (1200-7200)*	**260 (150-310)** 2200 (1900-2800)
PCB 77	**22 (9.4-34) [a]** 970 (590-1200) [a]	**18 (5.3-26) [a]** 970 (290-1600) [a]	**22 (10-46) [a]** 1100 (540-6700) [a]	**24 (9.3-38) [a]** 1200 (840-1800) [a]	**28 (14-48) [a]** 1200 (1050-2400) [a]	**26 (8.3-39) [a]** 1300 (840-7100) [a]	**78 (9.7-130)*** 1500 (170-2200)	**120 (68-160)** 1030 (760-1400)
PCB 81	NA	NA	NA	NA	NA	NA	**350 (130-550)*** 5800 (2900-9300)	**730 (490-900)** 6400 (5800-7400)
PCB 126	**8.2 (5.8-11) [a]** 340 (270-440) [a]	**11 (6.0-24) [a, b]** 580 (340-1500) [b]	**14 (ND-24)[b, c]** 710 (ND-940) [b]	**17 (13-24)[c, d]** 1000 (680-1500) [b, c]	**22 (18-33)[d]** 1200 (830-2000) [c]	**22 (16-45)[d]** 1300 (700-5050) [c]	**94 (48-160)** 1800 (730-2600)*	**110 (69-140)** 940 (810-1300)
PCB 169	**1.8 (1.2-3.1) [a]** 76 (48-140) [a]	**2.8 (2.1-4.3)[b]** 160 (97-570) [b]	**5.0 (ND-22)[c]** 250 (ND-1300) [b, c]	**7.6 (5.9-11)[c, d]** 440 (270-620)[c, d]	**11 (9.4-13)[d]** 580 (350-1000)[d]	**12 (6.1-20)[d]** 680 (300-3200) [d]	**39 (20-59)** 740 (300-2100)	**45 (30-77)** 410 (330-630)
PCB 123	NA	NA	NA	NA	NA	NA	**3.9 (1.7-7.0)** 64 (33-220)*	**4.0 (2.5-6.7)** 35 (30-55)
PCB 118	**1.1 (0.80-2.8) [a]** 54 (23-88) [a]	**1.8 (0.87-2.5) [a, b]** 100 (58-230) [b]	**1.6 (0.95-5.1) [a, b]** 100 (46-160) [b]	**2.4 (1.3-3.8) [b, c]** 130 (95-160) [b, c]	**2.7 (1.8-4.9) [b, c]** 130 (99-310) [b, c]	**3.3 (1.9-6.3) [c]** 160 (120-580) [c]	**28 (17-44)*** 440 (330-2400)	**40 (27-64)** 350 (300-520)
PCB 114	NA	NA	NA	NA	NA	NA	**0.69 (0.33-1.6)** 13 (6.9-49)*	**0.78 (0.55-1.3)** 7.2 (6.2-11)
PCB 105	**0.46 (0.31-1.2) [a]** 23 (9.0-39) [a]	**0.78 (0.60-1.2) [a, b]** 41 (25-280) [b]	**0.86 (0.4-2.2)[b]** 47 (18-110) [b]	**1.5 (0.94-2.6)[c]** 87 (58-120) [b, c]	**1.7 (1.3-3.4)[c]** 89 (64-150)[c]	**1.5 (1.3-3.3)[c]** 91 (60-270) [c]	**16 (6.9-30)** 280 (130-1030)*	**14 (9.3-25)** 120 (110-210)
PCB 167	NA	NA	NA	NA	NA	NA	**1.3 (0.66-2.4)*** 21 (12-70)	**2.5 (1.4-3.1)** 22 (18-30)
PCB 156	**0.071 (0.051-0.1)[a]** 3.1 (2.0-3.9) [a]	**0.1 (0.073-0.14) [a, b]** 5.8 (3.0-18)[b]	**0.17 (0.092-0.23)[b, c]** 8.6 (3.9-18) [b, c]	**0.27 (0.12-0.41) [c, d]** 17 (5.5-26) [c, d]	**0.31 (0.2-0.46)[d]** 18 (8.0-33) [c, d]	**0.31 (0.15-4.6) [d]** 23 (7.4-200) [d]	**4.7 (2.6-6.9)*** 94 (43-240)	**9.6 (5.4-13)** 87 (70-110)
PCB 157	NA	NA	NA	NA	NA	NA	**1.0 (0.51-1.5)*** 17 (8.7-54)	**1.4 (0.89-2.2)** 13 (11-18)

Table 2 (continued)

	Gulf of Finland							Gulf of Bothnia
PCB 170	NA	NA	NA	NA	NA	NA	**5.1 (2.8-8.9)***	**13 (7.8-17)**
							96 (40-360)	120 (97-190)
PCB 189	NA	NA	NA	NA	NA	NA	**0.44 (0.26-0.84)***	**1.2 (0.67-1.6)**
							9.1 (4.4-16)	12 (8.7-18)
Sum of PCBs[1]	**9.4 (6.9-12)** [a]	**12 (7.1-14)** [a, b]	**15 (9.6-23)** [b, c]	**15 (11-21)** [b, c]	**18 (13-27)** [c, d]	**24 (14-45)** [d]	**280 (140-350)***	**490 (310-630)**
	370 (270-530) [a]	640 (380-1700) [b]	790 (470-1800) [b]	810 (580-1300) [b, c]	890 (600-2100) [b, c]	1600 (840-3100) [c]	4300 (2700-16000)	4300 (3700-5500)
PCB-TEQ[2]	**1.1 (0.77-1.4)** [a]	**1.4 (0.88-2.8)** [a]	**1.9 (0.22-3.0)** [a, b]	**2.3 (1.8-3.3)** [b, c]	**2.9 (2.4-4.5)** [c]	**3.0 (2.2-6.0)** [c]	**17 (9.8-30)***	**25 (16-34)**
	44 (35-59) [a]	79 (46-220) [b]	96 (36-120) [b]	140 (94-190) [b, c]	160 (110-260) [c]	190 (100-640) [c]	320 (200-860)	230 (190-280)
WHO_{PCB}-TEQ[3]	**1.0 (0.77-1.4)** [a]	**1.4 (0.87-2.8)** [a, b]	**1.9 (0.2-3.0)** [a, b]	**2.2 (1.7-3.3)** [b, c]	**2.8 (2.4-4.5)** [c]	**3.0 (2.2-6.0)** [c]	**16 (9.4-29)***	**23 (15-32)**
	43 (35-59) [a]	78 (46-220) [b]	95 (33-120) [b]	140 (93-190) [b, c]	160 (110-260) [c]	190 (100-640) [c]	310 (190-820)	210 (180-270)

Statistical significance of concentration differences in age groups 2-7 (tested with one-way ANOVA, significant level $p < 0.05$) are distinguished by different letter as superscript.
Differences between concentrations of age groups 8-> 15 were tested separately from age groups 2-7, and statistically significant differences (tested with Mann-Whitney U test, significant level $p < 0.05$) are denoted here with an asterisk.
ND = concentration below LOD.
NA = not analysed.
[1] sum of 16 congeners in age groups 2-7 and sum of 36 congeners in age groups 8-> 15.
[2] incomplete PCB-TEq in age groups 2-7.
[3] incomplete WHO_{PCB}-TEq in age groups 2-7.

PCB-TEQ and WHO_{PCB}-TEQ was defined because seven PCB congeners contributing TEQs were not measured in 1993.

The results of the age groups 8 to > 15 years (Tables 1 and 2) were combined because of the selective sampling and the small number of large-sized herring in the pools. The fat percentage of herring in the Gulf of Finland was significantly lower than in the Gulf of Bothnia. There was no significant difference in weight, length, or Cf between the catchment areas. Concentrations in fresh weight in herring in the Gulf of Bothnia were higher than in the Gulf of Finland, partly due to their higher fat content. It was expected that differences in fat percentages would solely explain concentration differences in fresh weight basis. However, the concentrations on a fat basis were not equal. Especially in the marker PCBs, PCB 28/31, 52, 138, and 153 also the fat concentrations in the Gulf of Bothnia were significantly higher than in the Gulf of Finland. The concentrating effect of the lower amount of fat was most evident with congeners which bioaccumulated best, i.e. 1,2,3,7,8-PeCDD, 2,3,4,7,8-PeCDF, and PCB 169. Possible heavier exposure to some of PCDD/F and PCB congeners in the Gulf of Bothnia and/or differences in feeding habits of large herring might explain their higher concentrations also in fat. Herring feed mainly on zooplankton, but the older herring have a diet which also contains crustaceans and small fish, living in the upper trophic level. It is not known whether the large herring in the Gulf of Bothnia feed more frequently on crustaceans and small fish than large herring in the Gulf of Finland. The concentrations in these large herring must be considered as extreme values because of the method of sampling. Total WHO-TEQ in old herring reached value 34 pg/g fw in the Gulf of Finland, and 50 pg/g fw in the Gulf of Bothnia. The contribution of PCDD/Fs and PCBs to the total TEq was equal in both catchment area.

PCDD/F and PCB concentrations in 1999

In 1999 herring were caught in nine locations along the Finnish Baltic Sea coastline (Fig. 2). The concentrations of PCDD/Fs, marker PCBs, and PCB congeners with dioxin-like toxicity are illustrated in Tables 3 and 4, and data on fat percentages, weights, lengths, and Cfs are included in Table 3. The ages of small and large herring in 1999 were obtained from the data of ICES (Fig. 1) using average weights of herring. It must be kept in mind that ages here are crude estimates since the variation of sizes within an age group is considerable wide. Fish from the catchment area number 6, Kustavi, are usually grouped together with fish in the Gulf of Finland in the herring stock assessments (ICES, 2001), but here results of Kustavi were included in the Gulf of Bothnia herring on the basis of their fat percentages (3.6% in small herring and 5.1% in large herring).

Almost all fresh weight and fat concentrations of PCDD/Fs and PCBs in large herring were higher than in small herring in the Gulf of Finland but the differences were not significant. A similar finding was noted also with the 1993-1994 data (Tables 1 and 2), where the concentrations only seldom were significantly different in age groups over 4 years. The situation in the Gulf of Bothnia was different. Excluding those PCDD/Fs and PCBs which showed low bioaccumulation, the concentrations (fresh weight and fat basis) in large herring were significantly higher than in small herring. Some of this clear difference in concentrations between small and large herring in the Gulf of Bothnia can be explained by the greater gap in weights between small and large herring in the Gulf of Bothnia compared to the Gulf of Finland. Another explanation was also: small and large herring in the Gulf of Finland have similar feeding characteristics consuming mainly on zooplankton, but in the Gulf of Bothnia large herring feed relatively more on crustaceans and small fish, and hence are exposed to higher amounts of PCDD/Fs and PCBs.

The greater exposure of large herring to PCDD/Fs and PCBs in the Gulf of Bothnia was supported by the differences of concentrations in small and large herring between the Gulf of Finland and the Gulf of Bothnia. In small herring, the fresh weight concentrations of PCDD/Fs and PCBs (Tables 3 and 4) were almost the same irrespective of whether they were caught in the Gulf of Finland or the Gulf of Bothnia, in spite of lower fat percentage in the Gulf of Finland herring. The lower percentages of fat in small herring from the Gulf of Finland resulted in fat based concentrations, which were twice or even more as high as concentrations in small herring in the Gulf of Bothnia. The concentrations (fresh weight and fat basis) in the small herring

Table 3.
Medians (range) of fat percentages, weights, lengths, condition factors (Cf), concentrations (**pg/g fw** and pg/g fat) of PCDD/Fs and toxic equivalents of PCDD/Fs in small and large Baltic herring, caught in 1999 in the Gulf of Finland and in the Gulf of Bothnia.

	Gulf of Finland	Gulf of Finland		Gulf of Bothnia	
	Small herring 1993-1994	Small herring 1999	Large herring 1999	Small herring 1999	Large herring 1999
Age	5.5 (5-6)	5.5 (4.5-6)[1]	9.3 (9-10)[1]	4.5 (4-5)[1]	7.8 (5.5-9)[1]
Number of pools	20	4	4	5	5
Fat%	1.8 (0.99-3.5)	2.0 (1.5-2.9)**	2.4 (2.1-3.8)***	4.9 (3.6-5.1)	4.9 (4.0-7.2)
Weight	29 (23-35)	32 (27-33) ‡	47 (44-54)	31 (29-33) ‡‡	54 (41-56)
Length	17.2 (16.7-18.3)	17.6 (17.1-17.9) ‡	20.1 (19.8-20.7)	17.4 (17.1-17.5) ‡‡	20.7 (19.7-20.8)
Cf	0.56 (0.48-0.63)	0.58 (0.53-0.6)	0.59 (0.54-0.61)	0.6 (0.56-0.65)	0.6 (0.52-0.63)
2378-TCDD	**0.32 (0.15-0.76)**	**0.3 (0.23-0.38)**	**0.34 (0.29-0.82)**	**0.4 (0.35-0.46)** ‡‡	**0.74 (0.66-1.2)**
	16 (8.0-55)	14 (13-16)**	15 (13-22)	8.2 (6.8-13) ‡‡	17 (9.2-24)
12378-PeCDD	**1.3 (0.71-2.5)**	**1.2 (0.86-1.4)**	**1.5 (1.4-2.6)*****	**1.4 (1.2-1. 5)** ‡‡	**3.9 (2.3-4.0)**
	68 (28-180)	60 (41-66)**	64 (59-73)	29 (24-35) ‡‡	79 (33-86)
123478-HxCDD	**0.082 (0.013-0.14)***	**0.14 (0.13-0.15)** ‡	**0.19 (0.16-0.33)**	**0.18 (0.15-0.2)** ‡‡	**0.31 (0.26-0.54)**
	4.1 (0.54-10)	7.1 (5.3-8.8)**	8.5 (6.7-9.1)	4.0 (2.9-4.7)	7.4 (3.6-11)
123678-HxCDD	**1.4 (0.47-3.8)**	**1.5 (0.89-2.0)**	**2.2 (1.4-3.3)*****	**1.9 (1.5-2.0)** ‡‡	**5.4 (3.3-5.4)**
	66 (24-270)	74 (48-91)**	90 (59-100)	42 (35-47) ‡‡	110 (46-120)
123789-HxCDD	**0.099 (ND-0.25)***	**0.15 (0.14-0.18)**** ‡	**0.21 (0.18-0.58)**	**0.31 (0.16-0.58)**	**0.47 (0.27-1.1)**
	5.8 (0.13-18)	8.2 (5.2-9.2)	9.4 (7.9-15)	6.0 (4.3-14)	9.3 (3.8-23)
1234678-HpCDD	**0.28 (0.11-1.2)***	**0.11 (0.094-0.13)**	**0.14 (0.12-0.2)**	**0.12 (0.11-0.14)** ‡‡	**0.19 (0.15-0.37)**
	14 (4.0-87)*	5.2 (4.1-7.0)**	5.7 (5.3-6.0)	2.5 (2.2-3.8) ‡‡	3.9 (2.6-7.3)
OCDD	**0.78 (0.25-7.2)***	**0.15 (0.086-0.19)**	**0.18 (0.079-0.38)**	**0.1 (0.056-0.33)**	**0.19 (0.099-0.57)**
	38 (10-470)*	7.2 (3.8-11)	6.0 (3.9-15)	2.1 (1.3-9.0)	3.7 (2.5-11)
Dioxins	**4.6 (1.9-12)**	**3.6 (2.5-4.3)**	**4.7 (3.8-7.9)*****	**4.4 (4.1-4.8)** ‡‡	**12 (7.2-13)**
	240 (78-810)	180 (120-200)**	210 (160-220)	93 (79-120) ‡‡	240 (100-260)
2378-TCDF	**1.4 (0.59-3.1)**	**1.7 (0.84-2.5)**	**1.9 (1.7-3.9)**	**4.5 (1.8-5.2)**	**4.2 (2.9-5.8)**
	76 (40-95)	82 (55-86)	81 (80-100)	94 (35-120)	92 (40-110)
12378-PeCDF	**1.0 (0.62-1.9)**	**1.3 (0.89-1.6)**	**1.5 (1.4-3.1)**	**1.7 (1.4-1.9)** ‡‡	**3.0 (2.4-4.9)**
	52 (33-110)	60 (56-64)**	68 (62-82)	37 (27-52)	75 (33-95)
23478-PeCDF	**7.4 (4.4-9.7)***	**10 (7.3-12)**	**13 (11-25)*****	**12 (11-14)** ‡‡	**36 (24-39)**
	390 (190-850)	480 (400-510)**	570 (460-650)	270 (230-360) ‡‡	740 (340-800)
123478-HxCDF	**0.33 (0.17-2.0)**	**0.39 (0.32-0.52)**	**0.51 (0.46-0.93)*****	**0.54 (0.38-0.58)** ‡‡	**0.98 (0.86-1.7)**
	20 (6.6-68)	20 (18-21)**	22 (19-27)	11 (9.3-16) ‡‡	24 (12-33)

Table 3 (continued)

	Gulf of Finland	Gulf of Finland		Gulf of Bothnia	
	Small herring	Small herring	Large herring	Small herring	Large herring
	1993-1994	1999	1999	1999	1999
123678-HxCDF	**0.42 (0.075-2.6)** 23 (3.5-88)	**0.6 (0.44-0.79)** 28 (28-32)**	**0.8 (0.73-1.4)** 35 (31-40)	**0.62 (0.52-0.83)** ‡‡ 13 (12-23) ‡‡	**1.4 (1.2-2.4)** 32 (19-46)
234678-HxCDF	**0.75 (0.35-1.3)*** 41 (19-94)*	**0.49 (0.32-0.56)** 22 (19-26)**	**0.59 (0.52-1.1)** 27 (21-31)	**0.48 (0.4-0.65)** ‡‡ 9.9 (9.3-17) ‡‡	**1.1 (0.96-1.8)** 24 (15-36)
123789-HxCDF	ND	**0.024 (ND-0.034)** 1.1 (ND-1.5)	**0.041 (0.032-0.075)** 1.8 (1.4-2.0)	**0.032 (0-0.055)** ‡‡ 0.65 (0-1.1)	**0.078 (0.045-0.11)** 1.8 (0.63-2.2)
1234678-HpCDF	**0.28 (0.11-0.78)*** 16 (5.9-57)*	**0.1 (0.057-0.16)** 4.7 (2.6-9.0)**	**0.13 (0.074-0.26)** 4.6 (3.2-11)	**0.089 (0.071-0.12)** 2.2 (1.5-2.4)	**0.14 (0.067-0.28)** 3.0 (0.94-5.4)
1234789-HpCDF	ND	**0.006 (ND-0.03)** 0.27 (ND-1.0)	**0.017 (ND-0.019)** 0.74 (ND-0.91)	**0.14 (ND-0.042)** 0.28 (ND-0.86)	**0.029 (ND-0.064)** 0.64 (ND-1.3)
OCDF	**0.066 (0.015-0.27)** 3.5 (1.3-17)	ND	ND	ND	ND
Furans	**12 (6.6-20)** 620 (340-1300)	**15 (10-18)**** 690 (620-730)**	**18 (16-35)***** 820 (690-930)	**20 (19-20)** ‡‡ 410 (380-560) ‡‡	**47 (33-56)** 990 (460-1100)
Sum of PCDD/Fs	**18 (8. 6-26)** 830 (440-1900)	**19 (13-21)**** 880 (740-920)**	**23 (20-43)***** 1030 (870-1100)	**24 (23-25)** ‡‡ 500 (460-670) ‡‡	**58 (40-68)** 1200 (560-1300)
I-TEQ	**5.1 (3.0-7.7)** 260 (130-570)	**6.6 (4.7-7.5)**** 320 (260-330)**	**8.1 (7.1-16)***** 360 (300-410)	**8.0 (7.7-8.6)** ‡‡ 170 (160-230) ‡‡	**23 (15-25)** 460 (210-500)
$WHO_{PCDD/F}$-TEQ	**5.7 (3.3-8.9)** 290 (150-650)	**7.2 (5.1-8.2)**** 350 (280-360)**	**8.8 (7.8-17)***** 400 (340-450)	**8.7 (8.4-9.2)** ‡‡ 180 (170-250) ‡‡	**24 (16-27)** 500 (230-540)

To assess time trend, concentrations of five and six year old herring from the Gulf of Finland in 1993-1994 were added in the table on the basis of the similarity of their weights and lengths with small herring in 1999. Statistical significances of concentration differences (Mann-Whitney U test, significance level $p < 0.05$) between small herring in 1993-1994 and in 1999 are denoted here as (*); between groups small herring in the Gulf of Finland and in the Gulf of Bothnia as (**), and between groups large herring in the Gulf of Finland and in the Gulf of Bothnia as (***). Significances of difference of concentrations (Mann-Whitney U test, $p < 0.05$) between small and large herring in 1999 in the Gulf of Finland are denoted here as (‡); and between small and large herring in the Gulf of Bothnia as (‡‡).
ND = concentration below LOD.
[1] age estimation based on data from ICES 2001.

Table 4.
Medians (range) of concentrations as **ng/g fw** and as ng/g fat of PCBs and as **pg/g fw** and as pg/g fat of co-PCBs and toxic equivalents of PCBs in different age groups of Baltic herring, caught in 1999 in the Gulf of Finland and in the Gulf of Bothnia.

	Gulf of Finland	Gulf of Finland		Gulf of Bothnia	
	Small herring 1993-1994	Small herring 1999	Large herring 1999	Small herring 1999	Large herring 1999
PCB 28/31	**0.63 (0.048-1.7)** 32 (3.7-83)	**0.63 (0.41-0.71)** 29 (24-31)	**0.64 (0.56-1.2)** 29 (23-33)	**0.85 (0.66-1.1)** 17 (14-30)	**1.2 (0.89-1.7)** 20 (18-33)
PCB 52	**1.3 (0.72-2.9)** 73 (37-190)*	**0.9 (0.63-1.1)**** 41 (37-50)**	**1.0 (0.78-2.3)** 44 (38-60)	**1.3 (0.95-1.4)** ‡‡ 25 (19-40) ‡‡	**2.2 (1.7-3.9)** 44 (34-77)
PCB 101	**2.7 (1.7-5.5)*** 150 (110-260)	**4.0 (3.3-5.1)** 220 (140-220) **	**5.6 (4.4-8.8)***** 230 (220-240)	**4.3 (3.9-4.5)** ‡‡ 89 (79-130) ‡‡	**11 (8.5-15)** 220 (120-280)
PCB 138	**4.3 (2.9-9.4)*** 240 (140-940) *	**11 (11-15)** ‡ 650 (380-700) **	**17 (16-27)** 700 (660-860)	**9.9 (8.4-13)** ‡‡ 220 (170-350) ‡‡	**29 (23-36)** 590 (350-710)
PCB 153	**0.97 (0.69-2.4)*** 54 (32-240) *	**15 (14-21)** 870 (530-930) **	**22 (21-35)** 920 (850-1100)	**13 (12-17)** ‡‡ 320 (240-480) ‡‡	**46 (33-53)** 960 (480-1100)
PCB 180	**0.7 (0.53-1.0)*** 37 (24-93) *	**5.0 (4.2-5.5)** ‡ 250 (190-280) **	**6.6 (6.1-13)** 300 (250-340)	**5.1 (4.3-6.8)** 120 (87-170)	**16 (0.91-17)** 340 (23-370)
Sum of Marker PCBs	**11 (8.3-18)*** 600 (390-1600) *	**37 (33-49)** ‡ 2100 (1300-2200) **	**52 (50-87)** 2200 (2060-2600)	**34 (30-43)** ‡‡ 820 (610-1200) ‡‡	**110 (69-130)** 2200 (1200-2500)
PCB 77	**27 (9.3-48)** 1200 (840-2400)	**26 (19-30)** 1300 (820-1600) **	**30 (22-44)** 1200 (1100-1300)	**33 (20-39)** ‡‡ 720 (400-930)	**47 (37-63)** 1000 (520-1300)
PCB 126	**20 (13-33)** 1040 (680-2000)	**23 (18-29)** 1200 (820-1300) **	**30 (25-48)***** 1200 (1200-1300)	**26 (19-31)** ‡‡ 570 (370-740)	**54 (37-74)** 1300 (520-1500)
PCB 169	**9.7 (5.9-13)** 480 (270-1000)	**9.7 (8.8-12)** ‡ 510 (370-580) **	**15 (13-23)***** 590 (550-820)	**10 (8.9-12)** ‡‡ 230 (180-300) ‡‡	**34 (22-39)** 680 (310-850)
PCB 123	NA	**0.62 (0.6-0.94)** 37 (21-41) **	**0.95 (0.85-1.3)** 37 (34-49)	**0.5 (0.4-0.62)** ‡‡ 11 (8.1-17) ‡‡	**1.2 (1.1-2.0)** 27 (15-40)
PCB 118	**2.5 (1.3-4.9)*** 130 (95-310) *	**6.5 (5.7-11)** 380 (210-440) **	**9.4 (8.7-14)** 370 (360-480)	**5.0 (4.1-6.4)** ‡‡ 110 (84-180) ‡‡	**14 (11-22)** 290 (170-420)
PCB 114	NA	**0.12 (0.1-0.18)** 6.4 (4.2-8.0) **	**0.17 (0.14-0.26)** 6.9 (5.9-8.3)	**0.096 (0.079-0.12)** ‡‡ 2.1 (1.6-3.3) ‡‡	**0.25 (0.21-0.4)** 5.3 (3.2-7.8)
PCB 105	**1.7 (0.94-3.4)** 88 (58-140)	**2.0 (1.8-3.0)** 120 (64-130) **	**3.2 (2.4-3.8)** 120 (100-150)	**1.5 (1.4-1.9)** ‡‡ 35 (27-51) ‡‡	**4.3 (3.1-5.8)** 88 (51-110)
PCB 167	NA	**0.24 (0.22-0.3)** ‡ 13 (8.6-14) **†	**0.4 (0.35-0.58)** 16 (15-18)	**0.26 (0.23-0.33)** ‡‡ 6.3 (4.7-7.9) ‡‡	**0.71 (0.46-0.9)** 14 (6.5-18)

Table 4 (continued)

	Gulf of Finland	Gulf of Finland		Gulf of Bothnia	
	Small herring 1993-1994	Small herring 1999	Large herring 1999	Small herring 1999	Large herring 1999
PCB 156	**0.29 (0.12-0.45)*** 17 (5.5-33) *	**1.0 (0.89-1.3)** 59 (33-59) **	**1.5 (1.2-2.1)** 57 (52-74)	**0.89 (0.72-1.0)** ‡‡ 19 (15-28) ‡‡	**2.7 (2.0-3.2)** 55 (29-63)
PCB 157	NA	**0.28 (0.19-0.4)** 17 (6.6-17) **	**0.35 (0.32-0.45)***** 14 (12-17)	**0.19 (0.13-0.24)** ‡‡ 3.8 (2.7-6.1) ‡‡	**0.51 (0.38-0.73)** 10 (7.2-14)
PCB 170	NA	**2.9 (2.5-3.4)** 150 (110-160) **	**3.2 (3.0-7.5)***** 140 (130-200)	**2.9 (2.4-4.1)** ‡‡ 79 (49-84) ‡‡	**7.9 (5.7-10)** 160 (110-200)
PCB 189	NA	**0.12 (0.096-0.15)** 5.7 (5.1-6.4) **	**0.14 (0.11-0.3)** 6.6 (4.7-8.0)	**0.11 (0.08-0.16)** ‡‡ 2.4 (1.6-4.3) ‡‡	**0.32 (0.26-0.44)** 6.8 (3.7-8.7)
Sum of PCBs[1]	**16 (11-27)** 870 (580-2100)	**69 (60-91)** ‡ 3900 (2400-4000) **	**94 (92-160)** 4040 (3800-4600)	**71 (54-77)** ‡‡ 1500 (1100-2100) ‡‡	**180 (130-140)** 3700 (2100-4600)
PCB-TEQ[1]	**2.7 (1.7-4.5)** 150 (94-260)	**4.4 (3.6-5.8)** 240 (150-260) **	**5.7 (5.4-9.3)** 250 (230-270)	**4.5 (3.9-4.7)** ‡‡ 93 (78-130) ‡‡	**11 (8.0-14)** 220 (110-280)
WHO_{PCB}-TEQ[1]	**2.7 (1.7-4.5)** 140 (93-260)	**4.0 (3.3-5.4)** 220 (140-240) **	**5.4 (5.0-8.4)** 230 (220-250)	**4.1 (3.5-4.4)** ‡‡ 83 (68-120) ‡‡	**9.5 (7.1-13)** 210 (99-250)

To assess time trend, concentrations of five and six year old herring from the Gulf of Finland in 1993-94 are added in the table on the basis of the similarity of their weights and lengths with small herring in 1999. Statistical significances of concentration differences (Mann-Whitney U test, significance level $p < 0.05$) between small herring in 1993-1994 and in 1999 are denoted here as (*); between groups small herring in the Gulf of Finland and in the Gulf of Bothnia as (**), and between groups large herring in the Gulf of Finland and in the Gulf of Bothnia as (***). Significances of difference of results (Mann-Whitney U test, $p < 0.05$) between small and large herring in 1999 in the Gulf of Finland are denoted here as (‡); and between small and large herring in the Gulf of Bothnia as (‡‡).

[1] Statistical significance between small herring in 1993-1994 and 1999 in the Gulf of Finland not tested because of the different number of congeners measured.

indicated that these fish probably feed on zooplankton in both sea regions, and exposure to PCDD/Fs and PCBs was equal or slightly greater in the Gulf of Finland. In large herring most of the fresh weight and fat concentrations of PCDD/Fs and PCBs (Tables 3 and 4) in the Gulf of Bothnia were greater than in the Gulf of Finland. This may be due to the different feeding habits of large herring in the Gulf of Bothnia.

The median value of total WHO-TEQ in small herring from the Gulf of Finland was 11 pg/g fw, and in large herring it increased to 14 pg/g. In the Gulf of Bothnia, median values in small and large herring were 13, and 34 pg/g fw, respectively. PCBs accounted for 37% of the toxic equivalents in the Gulf of Finland, but their contribution decreased to 30% in the Gulf of Bothnia.

In the catchment area Korsnäs, from where two samples of mixed herring were analysed, the concentration of $WHO_{PCDD/F}$-TEQ was 15 pg/g fw with the concentration of WHO_{PCB}-TEQ being 5.8 pg/g fw. These values corresponded well with the average of small and large herring in the Gulf of Bothnia.

Comparability of the results with other studies

Concentrations of PCDD/Fs and PCBs in this study were not compared to previously published studies because the methodology of sample collection and sample preparation were dissimilar. For example, Bignert et al. (1998) have collected most of their material in the autumn when the fat percentages in herring are very different from the values obtained in spring. In addition they skinned the herring prior to analysis, thus their herring samples were so different from ours that the comparison of results would be misleading. In this study we have analysed herring in the form they are sold for human consumption.

Time-trends of PCDD/Fs and PCBs

To obtain time-trends of PCDD/Fs and PCBs in herring, concentrations of age groups 5 and 6 were extracted from the data in 1993-1994 and are shown separately in the Tables 3 and 4. The basis for selection of these age groups was similarity of weights and lengths between these groups and small herring in 1999. Time-trend assessment was possible only with small herring in the Gulf of Finland.

PCDD/F concentrations (Table 3) present in the small herring in the Gulf of Finland in 1993-1994 and 1999 indicated that concentrations have not been decreasing during this time period. Since age was not determined by otoliths of herring in 1999, there is a possibility that small herring in 1999 were older than those assessed in Table 3. In Fig. 1 it can be observed that the size of herring in the Gulf of Finland has declined between 1993 and 1999, and this shrinkage as a function of time might obscure the possible downward trend in herring exposure to PCDD/Fs. Since no downward trend was evident, the rule of thumb of TEQs, based on 1993-1994 results, could be tested with the 1999 results. With the reservation that the age estimation of 1999 herring was only indicative, the measured I-TEQ and $WHO_{PCDD/F}$-TEQ values in the Gulf of Finland, 6.6 and 7.2 pg/g fw, respectively, corresponded quite well with the values estimated with the rule of thumb, 4.9 and 5.6 pg/g. Also the actual I-TEQ and $WHO_{PCDD/F}$-TEQ values determined, i. e. 8.1 and 8.8 pg/g fw, respectively for large herring in the Gulf of Finland corresponded quite well with estimated values, 8.8 and 9.9 pg/g, respectively. This suggested that the rule of thumb is still applicable when assessing TEQ concentrations in the Gulf of Finland.

Differences in PCB concentrations (Table 4) of the small herring in the Gulf of Finland between 1993-1994 and 1999 gave contradictory information about time trends of PCBs. The results for the purely co-planar PCBs (PCB 77, 126 and 169) suggested, as with the PCDD/Fs, that

there has not been any decreasing trend during this time period. For some of the other PCBs, the time-trend of concentrations even seemed to be increasing. These results do not agree with earlier results about the PCB time-trends in Baltic Sea fauna reported by other study groups (Odsjö et al., 1997; Bignert et al., 1998). The fact that PCB analytic methods have advanced from 1993-1994 to 1999, for example there is now an increased number of standards (allowing at least one internal standard per each chlorination degree of PCBs), suggests that some of the results of other PCBs in 1993-1994 herring might be systematically too low.

If time-trends of PCDD/Fs and PCBs are evaluated through contributions to total TEQs there exist two possible scenarios. Either exposure to PCDD/Fs has increased or exposure to PCBs has continued to decrease as stated by Bignert et al. (1998), because the contribution of PCBs to total TEQ have dropped from 50% in 1993-1994 to 30-37% in 1999.

Congener profiles of PCDD/Fs and PCBs

In Fig. 3 median percentage profiles of PCDD/F congeners from the sum of PCDD/Fs and from toxic equivalents are shown. The dominating congener in both profiles was 2,3,4,7,8-PeCDF followed by 2,3,7,8-TCDF, 1,2,3,7,8-PeCDF, 1,2,3,7,8-PeCDD, 1,2,3,6,7,8-HxCDD and OCDD in the sum of PCDD/Fs profile, and 2,3,7,8-TCDD and 1,2,3,7,8-PeCDD in the toxic equivalent profiles. Similar profiles of Baltic herring, and herring caught from the North Sea have been reported by Rappe et al. (1989). The PCDD/F profiles in Baltic herring were similar to those measured from seafood, in particular anchovy and mackerel, in the Adriatic Sea (Bayarri et al., 2001).

Percentages of PCB congeners from sum of PCBs and from toxic equivalents in herring caught in 1999 are shown in Fig. 4. Congener profiles were consistent irrespective of the herring's age, sampling area or time. The dominant congeners in the profiles from sum of PCBs were PCB 153, 138, 118, 180, 101, 110. In profiles of toxic equivalent, the dominant congeners were PCB 126, 118, and 156. The PCB profile of anchovy, reported by Bayarri et al. (2001), greatly resembled the herring profiles found here, although the set of measured PCBs was not fully consistent.

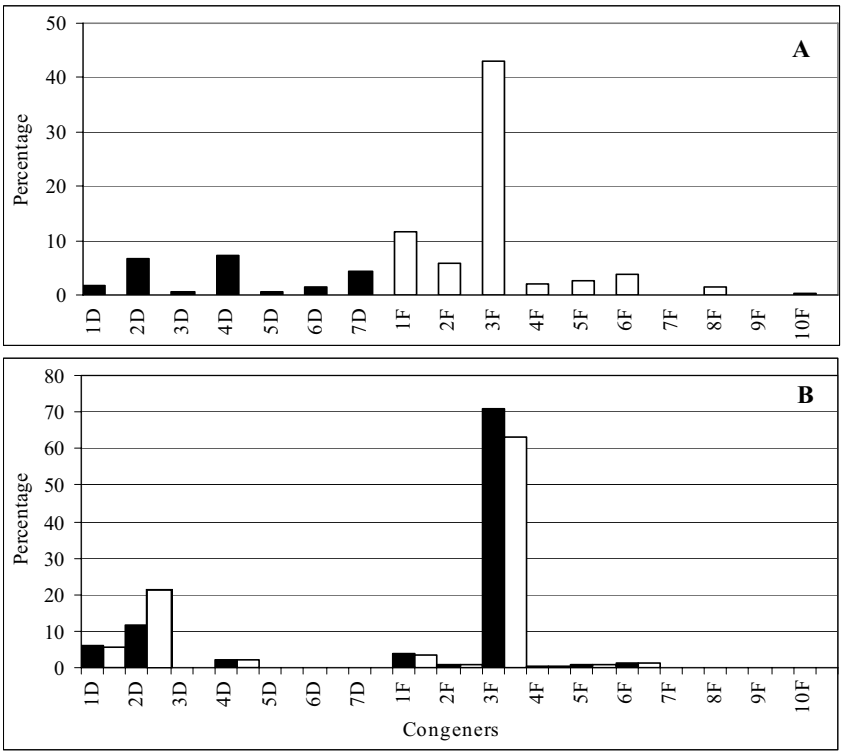

Fig 3. Median percentages of PCDD/F congeners in Baltic herring in the 1990's. (A) Percentages from sum of PCDDs (black bars) and PCDFs (white bars), and (B) percentages from toxic equivalents (I-TEQ: black bars; $WHO_{PCDD/F}$-TEQ: white bars).
Congeners: 1D: 2,3,7,8-TCDD; 2D: 1,2,3,7,8-PeCDD; 3D: 1,2,3,4,7,8-HxCDD; 4D: 1,2,3,6,7,8-HxCDD; 5D: 1,2,3,7,8,9-HxCDD; 6D: 1,2,3,4,6,7,8-HpCDD; 7D: OCDD; 1F: 2,3,7,8-TCDF; 2F: 1,2,3,7,8-PeCDF; 3F: 2,3,4,7,8-PeCDF; 4F: 1,2,3,4,7,8-HxCDF; 5F: 1,2,3,6,7,8-HxCDF; 6F: 2,3,4,6,7,8-HxCDF; 7F: 1,2,3,7,8,9-HxCDF; 8F: 1,2,3,4,6,7,8-HpCDF; 9F: 1,2,3,4,7,8,9-HpCDF; 10F: OCDF.

The source of exposure of herring to PCDD/Fs and PCBs, air-zooplankton versus sediments-zooplankton or sediments-crustacean, remained obscure. PCDD/F congeners in zooplankton, and in sediments in open sea areas and in areas without additional sources both originate from air deposits (Rappe et al., 1989; Kjeller and Rappe, 1995), and because of that it is impossible to assess the origin of PCDD/Fs in zooplankton. In the eastern Gulf of Finland, there are many major point sources of PCDD/Fs, especially 1,2,3,4,6,7,8-HpCDF and OCDF (Verta et al., 1999). Herring caught in this particular area did not have higher concentrations of PCDD/Fs, and the profiles of those herring did not express increased percentages of 1,2,3,4,6,7,8-HpCDF or

OCDF. However this result does not exclude exposure via sediment, since those two major congeners in this point source do not bioaccumulate in herring.

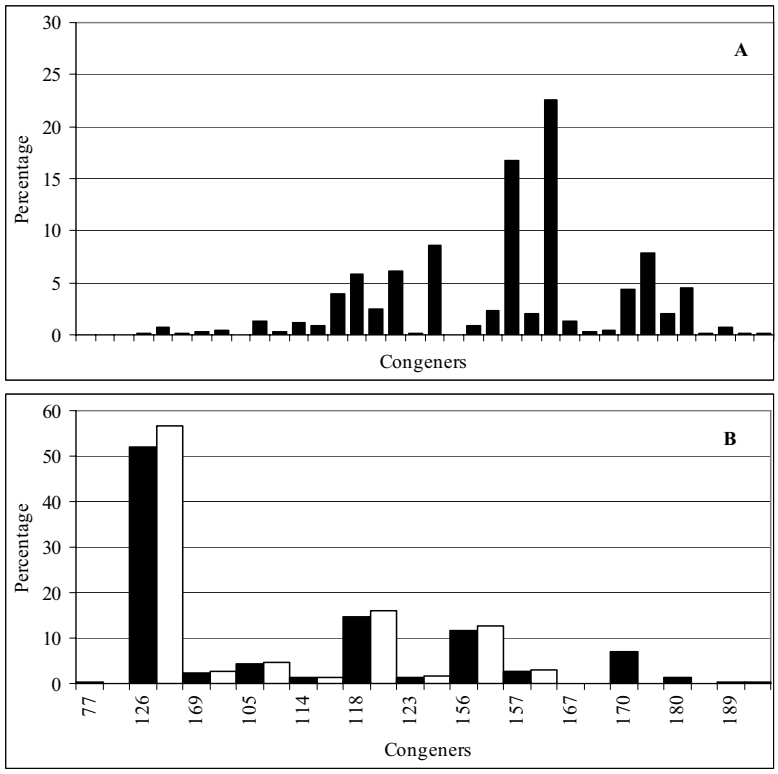

Fig. 4. Median percentages of PCB congeners in Baltic herring in 1999. (A) Percentages from sum of PCBs, and (B) percentages from toxic equivalents (PCB-TEQ: black bars; WHO_{PCB}-TEQ: white bars). Congeners in A, left to right: PCB 77, 126, 169, 18, 28/31, 33, 47, 49, 51, 52, 60, 66, 74, 99, 101, 105, 110, 114, 118, 122, 123, 128, 138, 141, 153, 156, 157, 167, 170, 180, 183, 187, 189, 194, 206, and 209.

5. CONCLUSIONS AND HUMAN EXPOSURE

Concentrations in herring measured in 1993-1994 in the Gulf of Finland showed a clear age dependency of PCDD/Fs. A rule of thumb was that there was one unit increase of TEQ concentration for every year of a herring's life. This was found to be valid also with herring sampled in 1999. For PCBs no such a rule could be produced because of the missing data in 1993-1994 of congeners contributing to toxic equivalents. The higher fat percentage in herring and hence higher concentrations on a fresh weight basis in the Gulf of Bothnia limits the use of the rule of thumb to the herring caught from the Gulf of Finland. In small herring, the differences in fat percentage were the main reason for the differences in concentrations on a fresh weight basis.

With large herring it was concluded that the exposure source of PCDD/Fs and PCBs in the Gulf of Bothnia differs from exposure in the Gulf of Finland. Based on two time points, 1993-1994 and 1999, the concentrations of PCDD/Fs and PCBs in herring in the Gulf of Finland did not reveal any clear decline.

On average the consumption of herring in the Finnish population varies between 800 to 1100 g per year as filleted weight (Finnish Game and Fisheries Research Institute, 2000; Kiviranta et al., 2001), but the assessment of the exposure of Finns to PCDD/Fs and PCBs via herring is quite difficult. There is no reliable information about the size or age distribution of consumed herring, and data on concentrations of harmful substances in herring in seasons other than spring are missing. Also proper age or size correlation data on PCDD/Fs and PCBs from the Gulf of Bothnia is currently missing. The age of herring for human consumption usually is 3-6 years or older. Hence, according to the rule of thumb created in this study, concentrations in a major fraction of the herring used by Finns as food, will exceed the limit value of 4 pg $WHO_{PCDD/F}$-TEQ/g set by EU.

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CHAPTER 5

POLYCHLORINATED DIBENZO-*P*-DIOXINS, DIBENZOFURANS, AND BIPHENYLS IN THE GENERAL POPULATION IN FINLAND

Hannu Kiviranta[a*], Jouni T. Tuomisto[a], Jouko Tuomisto[a,b], Erkki Tukiainen[c], and Terttu Vartiainen[a,d]

[a] Department of Environmental Health, National Public Health Institute, P.O.Box 95, FI-70701 Kuopio, Finland

[b] University of Kuopio, Department of Public Health and General Practice, P.O.Box 1627, FI-70211 Kuopio, Finland

[c] Helsinki University Hospital, Department of Plastic Surgery, P.O.Box 266, FI-00029 HUS, Finland

[d] University of Kuopio, Department of Environmental Sciences, P.O.Box 1627, FI-70211 Kuopio, Finland

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1. ABSTRACT

We measured adipose tissue concentrations of polychlorinated dibenzo-*p*-dioxins, dibenzofurans (PCDD/Fs), and polychlorinated biphenyls (PCBs) in 420 general Finns living in southern Finland. The mean (median) concentrations of $WHO_{PCDD/F}$-TEQ and WHO_{PCB}-TEQ were 29.0 (24.1) and 20.7 (16.7) pg g^{-1} fat, respectively. The concentrations clearly correlated with age. Expressing the concentrations as a function of subject's ages revealed that the exposure of Finns has declined over the last 30 years. A downward gradient was found in the concentrations from the Baltic Sea coast to inland areas in Finland, and this was assessed to be due to consumption of the Baltic Sea fish, especially Baltic herring. Linear regression models for natural logarithm $WHO_{PCDD/F}$-TEq, natural logarithm WHO_{PCB}-TEq, and natural logarithm WHO_{total}-TEq, explained

70%, 69%, and 72% of the variability, respectively. Age, lactation, place of residence, and fish consumption frequencies were significant predictors in the models.

2. INTRODUCTION

The objective of this study was to determine the occurrence of polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs), and polychlorinated biphenyls (PCBs) in the general adult Finnish population. The source of PCDD/Fs and PCBs is food, especially food of animal origin. In Finland, most of human exposure can be traced to consumption of fish, especially fatty Baltic Sea fish (Kiviranta et al., 2001). Therefore in this study we paid special attention to the fish consumption habits of our subjects.

A decreasing gradient of PCDD/F and PCB concentrations from the Baltic Sea coast to inland areas has been described in mothers' milk samples, which were collected during the late 1980s (Vartiainen et al., 1997). The purpose of this study was to investigate if a similar decreasing concentration gradient could be seen in the average population body burdens. An exposure model would be a valuable tool in epidemiological studies to assess the exposure to PCDD/Fs and PCBs. The development of such a model would mean that the exposure of the population, even at the individual level, could be assessed, without actually measuring concentrations with expensive and time-consuming methods. We assessed linear regression models for toxic equivalents of PCDD/Fs ($WHO_{PCDD/F}$-TEQ), PCBs (WHO_{PCB}-TEQ), and the sum of these two parameters (WHO_{total}-TEQ). The models were also validated with concentration results of a reference population comparable to the original study population.

3. MATERIALS AND METHODS

Subjects

PCDD/Fs and PCBs were determined from appendicitis patients who were chosen as controls in our case-control study of soft tissue sarcoma in 1997-1999 (Tuomisto et al., 2004). The concentrations were measured from a total of 420 subjects. The place of residence of all but three subjects, was southern Finland. They were operated in university, central, district or municipality hospitals in Espoo, Helsinki, Hyvinkää, Joensuu, Jyväskylä, Kotka, Kuopio, Lahti, Lappeenranta, Pori, Seinäjoki, Tampere, Turku, and Vaasa. The concentration data obtained from these subjects were used to depict concentrations in men and women of different ages and age groups, in

different classified subgroups of subjects, and to assess the average concentrations of PCDD/Fs and PCBs in the Finnish population. Concentration data served also as source for composing models to assess the exposure of Finns to the analytes of interest. The subjects were asked to complete a questionnaire about their intake of foods and about relevant demographic features and their lifestyle.

The study subjects were classified by two different criteria: the age of the subjects (≤ 46 years and > 46 years) and the place of residence (coastal area [Kotka, Pori, Seinäjoki, Turku, Vaasa], capital area [Espoo, Helsinki, Hyvinkää], and inland area [Joensuu, Jyväskylä, Kuopio, Lahti, Lappeenranta, Tampere]). Table 1 depicts the age, the body mass index (BMI), the number of children and duration of lactation in women, and the fish consumption statistics for all subjects as well as in the classified subgroups. Detailed questions about subjects' fish consumption habits including favoured fish species were asked.

The exposure models obtained from the concentration data of appendicitis patients were validated with the data obtained from the soft tissue sarcoma case patients (n = 148) (Tuomisto et al., 2004). The dioxin concentrations in cases and controls did not differ from each other as was described in the previous sarcoma study.

Informed consent was obtained from all patients in writing before the operation. The study was approved by the Ethics Committees of the National Public Health Institute and the hospitals involved.

Exposure assessment

The concentrations of the 17 toxic PCDD/F congeners (Table 2) and of the 36 PCB congeners (Table 3) were measured from fat of a subcutaneous tissue sample (0.3-1.5 g of fat) which was obtained during an appendectomy or sarcoma operation. The toxic equivalents ($WHO_{PCDD/F}$-TEQ and WHO_{PCB}-TEQ) were calculated with the sets of toxic equivalency factors (TEF), recommended by WHO in 1998 (Van den Berg et al., 1998).

Fat from tissue sample was extracted with toluene for 18-24 hours using the Soxhlet apparatus. The fat content was determined gravimetrically after changing the solvent to hexane using nonane as a keeper. Fat sample was spiked with a set of ^{13}C-labeled internal standards: sixteen 2,3,7,8-chlorinated PCDD/F congeners, three non-*ortho* PCBs (PCB 77, 126, 169), and nine other PCBs (PCB 30 [^{12}C-labeled], 80, 101, 105, 138, 153, 156, 180, 194).

The sample was defatted in a silica gel column containing acidic and neutral layers of silica, and all analytes were eluted with dichloromethane (DCM):cyclohexane (c-hexane) (1:1). PCDD/Fs were separated from PCBs on activated carbon column (Carbopack C, 60/80 mesh) containing Celite (Merck 2693). The first fraction including PCBs was eluted with DCM:c-hexane (1:1) following a back elution of the second fraction (PCDD/Fs) with toluene. Eluents from both of the fractions were evaporated using nonane as a keeper and then fractions in *n*-hexane were further cleaned by passing them through an activated alumina column (Merck 1097). The PCDD/F fraction was eluted from the alumina column with 20% DCM in *n*-hexane and recovery standards (^{13}C 1,2,3,4-TCDD and ^{13}C 1,2,3,7,8,9-HxCDD) were added to the fraction before DCM and *n*-hexane were replaced by 10-15 µl of nonane. The PCB fraction was eluted from the alumina column with 2% DCM in *n*-hexane, and the fraction, after changing the eluent to *n*-hexane, was transferred to another activated carbon column (without Celite) in order to separate the non-*ortho* PCBs from other PCBs. DCM (50%) in *n*-hexane was used to elute other PCBs while non-*ortho* PCBs were back eluted with toluene. Recovery standards, PCB 159 for other PCBs and ^{13}C PCB 60 for non-*ortho* PCBs were added prior to analysis; the solvent for other PCBs (DCM:*n*-hexane, 1:1) was replaced by 300 µl of *n*-hexane, for non-*ortho* PCBs toluene was replaced by 10-15 µl of nonane. The quantitation was performed by selective ion recording mode using a VG 70-250 SE (VG Analytical, UK) mass spectrometer (resolution 10,000) equipped with a HP 6890 gas chromatograph with a fused silica capillary column (DB-DIOXIN, 60 m, 0.25 mm, 0.15 µm). Two µl were injected into a split-splitless injector at 270°C. The temperature programs for PCDD/Fs, non-*ortho*-PCBs, and other PCBs were:

start, 140°C (4 min), rate 20°C min^{-1} to 180°C (0 min), rate 2°C min^{-1} to 270°C (36 min);

start, 140°C (4 min), rate 20°C min^{-1} to 200°C (0 min), rate 10°C min^{-1} to 270°C (12 min);

start, 60°C (3 min), rate 20°C min^{-1} to 200°C (0 min), rate 4°C min^{-1} to 270°C (14 min); respectively.

Limits of quantitation (LOQ) for PCDD/Fs and non-*ortho* PCBs varied between 0.1-5 and 1-5 pg g^{-1} fat, respectively, and for other PCBs between 0.02-0.1 ng g^{-1} fat, depending on each individual congener. Recoveries for internal standards were more than 50% for all congeners. Concentrations were calculated with lower bound method in which the results of congeners with concentrations below the LOQ were designated as nil.

Table 1. Descriptive statistics of the study subjects.

		Age subgroups		Area		
	All subjects n = 420	≤ 46 years n = 225	> 46 years n = 195	Coastal n = 147	Capital n = 104	Inland n = 166
Age (years)	44,**44**,*15*,(13-81)	32,**32**,*8.3*,(13-46)	58,**55**,*8.0*,(46-81)	42,**38**,*16*,(13-73)**	42,**42**,*15*,(16-78)	47,**49**,*15*,(17-81)
BMI (kg m^{-2})	26,**25**,*3.8*,(18-39)	25,**24**,*3.7*,(18-39)*	27,**26**,*3.5*,(20-39)	25,**25**,*3.6*,(18-39)	25,**25**,*3.4*,(19-39)	26,**26**,*4.0*,(19-39)
Female (%)	51	50	53	47	51	57
No. of children of women	1.8, **2.0**, (0-7)	1.2, **1.0**, (0-4)*	2.2, **2.0**, (0-7)	1.4, **1.5**, (0-4)**	1.7, **2.0**, (0-5)	2.1, **2.0**, (0-7)
Total lactation, months	8.2, **5.0**, *11*, (0-72)	7.2, **0**, *13*, (0-72)	8.9, **6.0**, *10*, (0-72)	7.8, **4.0**, *12*, (0-72)	7.9, **5.0**, *11*, (0-45)	8.6, **5.0**, *12*, (0-72)
Questinnaire returned (%)	77	74	81	84	74	72
Fish consumption, times per month						
All fish	4.9,**4.0**, (0.5-24)	4.6,**4.0**, (0.5-24)	5.3,**4.0**, (0.5-24)	5.0,**4.0**, (0.5-24)	5.2,**4.0**, (0.5-24)	4.7,**4.0**, (0.5-16)
Baltic herring	1.2,**0.5**, (0.5-16)	0.9,**0.5**, (0-16)*	1.5,**0.5**, (0-16)	1.5,**0.5**, (0-16)	1.0,**0.5**, (0-4)	1.1,**0.5**, (0-8)
Farmed trout or salmon	2.0,**2.0**, (0.5-16)	1.7,**2.0**, (0-16)*	2.3,**2.0**, (0-16)	2.0,**2.0**, (0-16)	2.2,**2.0**, (0-16)	1.8,**2.0**, (0-8)
Other fish[a]	2.2,**1.5**, (0.5-15)	2.4,**1.5**, (0.5-15)	2.1,**0.5**, (0.5-15)	2.1,**1.5**, (0.5-12)	2.4,**1.5**, (0.5-15)	2.3,**1.5**, (0.5-15)

Mean, **median**, SD and (range) of age, body mass index (BMI), and total lactation. Mean, **median**, and (range) of number of chidren of women. The consumption frequency, mean, **median** and (range), of different types of fish per month.

*Significantly different compared with the > 46 years group ($p < 0.05$ by independent samples T-test)

** Significant difference between groups ($p < 0.05$ by one-way ANOVA)

[a] Consists of pike, perch, burbot, pike perch, vendace, whitefish, bream, roach, frozen fish, canned fish and shrimps

Table 2.

Mean, **median**, *SD*, and (range) of PCDD/F concentrations and $WHO_{PCDD/F}$-TEQs as pg g^{-1} fat in adipose tissue samples (n = 420) from general population in Finland.

Congener	Mean, **median**, *SD*, (range)	% of sum of congeners	% of $WHO_{PCDD/F}$-TEQ
2,3,7,8-TCDD	2.55, **2.02**, *1.84*, (0.157–16.4)	0.650	9.03
1,2,3,7,8-PeCDD	7.61, **6.21**, *5.42*, (0.986–43.2)	1.89	26.2
1,2,3,4,7,8-HxCDD	2.73, **2.42**, *1.58*, (nq–12.1)	0.684	1.04
1,2,3,6,7,8-HxCDD	44.3, **40.2**, *23.7*, (5.10–148)	11.3	16.9
1,2,3,7,8,9-HxCDD	4.17, **3.72**, *2.35*, (nq–13.8)	1.04	1.66
1,2,3,4,6,7,8-HpCDD	40.8, **33.1**, *29.3*, (nq–222)	9.57	1.67
OCDD	263, **227**, *169*, (37.7–1730)	62.8	0.113
2,3,7,8-TCDF	1.07, **0.747**, *1.24*, (nq–18.3)	0.274	0.379
1,2,3,7,8-PeCDF	0.551, **0.380**, *0.625*, (nq–5.91)	0.138	0.094
2,3,4,7,8-PeCDF	24.2, **18.2**, *20.2*, (2.30–165)	5.98	38.7
1,2,3,4,7,8-HxCDF	4.34, **3.71**, *2.66*, (0.774–19.7)	1.11	1.67
1,2,3,6,7,8-HxCDF	4.00, **3.35**, *2.61*, (0.663–22.8)	1.01	1.47
2,3,4,6,7,8-HxCDF	1.53, **1.22**, *1.11*, (nq–6.93)	0.380	0.580
1,2,3,7,8,9-HxCDF	0.052, **nq**, *0.110*, (nq–0.658)	0.014	0.022
1,2,3,4,6,7,8-HpCDF	10.6, **7.97**, *10.7*, (nq–148)	2.79	0.486
1,2,3,4,7,8,9-HpCDF	0.057, **nq**, *0.180*, (nq–1.88)	0.014	0.002
OCDF	1.26, **nq**, *6.76*, (nq–81.9)	0.373	0.0008
Sum of PCDD/Fs	413, **364**, *230*, (78.0–2080)		
$WHO_{PCDD/F}$-TEQ	29.0, **24.1**, *19.7*, (3.64–153)		

Percentages of congeners of the sum of PCDD/Fs and of $WHO_{PCDD/F}$-TEQ are also shown.
Abbreviations: HpCDD, heptachlorodibenzo-*p*-dioxin; HpCDF, heptachlorodibenzofuran; HxCDD, hexachlorodibenzo-*p*-dioxin; HxCDF, hexachlorodibenzofuran; nq, below limit of quantitation; 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.

Quality control and assurance

Fat samples were analyzed during and after the collection period 1997-1999. All analytical work was performed blind such that the chemistry laboratory knew only the code of the sample. The laboratory reagent and equipment blank samples were treated and analyzed with the same method as the actual samples, one blank for every eight to ten samples. Quality assurance of analysis was performed in two separate ways: a) two preformulated pools of human fat with different concentrations of PCDD/Fs [10.6 (n = 35) and 40.2 (n = 33) pg g^{-1} ($WHO_{PCDD/F}$-TEQ in fat)] and PCBs [4.72 and 24.2 pg g^{-1} (WHO_{PCB}-TEQ), respectively] were always run with each lot of samples and b) 36 individual fat samples with $WHO_{PCDD/F}$-TEQs ranging from 6.9 to 116 pg g^{-1} and WHO_{PCB}-TEQs from 4.6 to 95 pg g^{-1} were analyzed in duplicate. The coefficients of variation (CV) for $WHO_{PCDD/F}$-TEQ in preformulated pools were 5.1% and 5.7%, respectively and for

WHO_{PCB}-TEQ 12% and 9.0%, respectively. In duplicate analysis the CV was 6.2% for $WHO_{PCDD/F}$-TEQ and 18% for WHO_{PCB}-TEQ.

The laboratory has successfully participated in several international quality control studies for the analysis of PCDD/Fs, and PCBs. Matrices in these studies have included cow milk, human milk and human serum. (Yrjänheikki, 1991; Rymen, 1994; WHO, 1996; Lindström et al., 2000). The laboratory of chemistry in the National Public Health Institute is an accredited testing laboratory (No T077) in Finland (EN ISO/IEC 17025). The scope of accreditation includes PCDD/Fs, non-*ortho* PCBs, and other PCBs from human tissue samples.

Statistical Analysis

Statistical analyses were carried out by means of SPSS software (for Windows, release 10.1.3). Before the statistical tests, all concentrations were transformed to a natural logarithm (ln) scale in order to ensure the normal distribution of concentrations. For comparisons of two groups either the Mann-Whitney U nonparametric test or the independent samples T-test was used to test the statistical significances of the differences of concentrations/variables between two groups. One way analysis of variance (ANOVA) or Kruskal-Wallis H test were used to compare the differences of concentrations/variables between multiple groups and age as covariate was added to analysis of variance because there was a suspected dependence of concentrations and age. The differences with $p < 0.05$ were considered to be statistically significant.

Linear regression models for dependent variables: ln $WHO_{PCDD/F}$-TEQ, ln WHO_{PCB}-TEQ, and their sum; ln WHO_{total}-TEQ, were established. Continuous predictor variables in the models were: age (year), BMI (kg m^{-2}), lactation (months), and fish consumption frequencies (times per month). Binary variables in the models were: living in the capital area (no/yes) and living in the inland area (no/yes).

4. RESULTS

Demographics and fish consumption

The average age of all study subjects was 44 years; in the groups classified by age the average ages were 32 (32 median) and 58 (55 median) years, respectively. The average age of the inland area group was higher than in the other groups (Table 1). There was no age difference between men and women.

The average BMI of all study subjects was 26 (25 median) kg m^{-2} with the BMI being significantly higher in the older population. There was no geographical difference in the BMI. With respect to all study subjects, men had significantly higher BMI than women, this being attributable to the higher BMI in younger men than women. Also in two places of residence, coastal and capital area, men had higher BMI than women.

The number of children borne by women was on average 1.8 (2.0 median), and the number of children was significantly higher in the older population than the younger. There was a significant difference in number of children born in the different places of residence with most being born in the inland area. Lactation lasted on average for 8.2 months (5.0 median) and there were no significant differences in lactation between age subgroups or places of residence.

About two thirds (66%) of our subjects consumed fish once a week or more. The fish consumption was on average four to five times per month and the difference between the subgroups of different ages and places of residence was not significant. Baltic herring and farmed trout or salmon were consumed significantly more often in the age subgroup > 46 years than in subgroup ≤ 46 years, and there was a trend that the fish group which consisted of other fish, was consumed more by the younger population. Thus younger subjects consumed significantly more frozen fish products and shrimps, which were included in the other fish group. Baltic herring was consumed more in the coastal area than in the other areas, but this trend was not statistically significant (Table 1). Only in the capital area was the monthly consumption of farmed trout or salmon significantly higher in women than in men. Otherwise there was no difference in fish consumption between genders.

Table 3.

Mean, **median**, *SD*, and (range) of PCB concentrations and WHO_{PCB}-TEQs in adipose tissue samples (n = 420) from general population in Finland.

Congener	Mean, **median**, *SD*, (range)	% of sum of congeners	% of WHO_{PCB}-TEQ
Non-*ortho*-PCBs [a]			
PCB 77	16.5, **10.9**, *30.6*, (nq–505)	0.0040	0.009
PCB 126	75.2, **52.7**, *75.0*, (nq–817)	0.015	35.1
PCB 169	67.4, **54.5**, *51.7*, (nq–399)	0.013	3.33
Other PCBs [b]			
PCB 18	0.441, **0.278**, *0.546*, (nq–4.35)	0.133	
PCB 28/31	4.61, **2.75**, *6.92*, (nq–99.5)	1.08	
PCB 33	0.267, **0.134**, *0.444*, (nq–5.02)	0.076	
PCB 47	0.455, **0.386**, *0.348*, (nq–0.412)	0.123	
PCB 49	0.172, **0.125**, *0.199*, (nq–2.72)	0.050	
PCB 51	0.021, **0.014**, *0.033*, (nq–0.429)	0.0067	
PCB 52	0.784, **0.606**, *0.779*, (0.053–8.54)	0.209	
PCB 60	0.674, **0.445**, *1.23*, (0.022–21.8)	0.137	
PCB 66	1.89, **1.25**, *3.76*, (0.240–70.8)	0.393	
PCB 74	8.63, **6.87**, *7.77*, (1.55–115)	1.77	
PCB 99	10.4, **7.71**, *13.4*, (0.283–216)	2.02	
PCB 101	1.40, **1.06**, *2.02*, (nq–36.2)	0.316	
PCB 105	4.21, **3.31**, *3.54*, (0.525–28.7)	0.814	2.04
PCB 110	0.534, **0.344**, *0.875*, (nq–12.6)	0.130	
PCB 114	1.00, **0.800**, *0.754*, (0.114–4.88)	0.195	2.46
PCB 118	19.9, **15.2**, *16.9*, (2.14–134)	3.84	9.57
PCB 122	0.001, **nq**, *0.006*, (nq-0.086)	0.0004	
PCB 123	0.835, **0.518**, *1.75*, (0.027–33.1)	0.150	0.355
PCB 128	1.20, **0.986**, *0.949*, (0.082–7.98)	0.254	
PCB 138	74.7, **62.7**, *52.9*, (9.08–461)	14.9	
PCB 141	0.273, **0.172**, *0.394*, (nq–5.44)	0.068	
PCB 153	135, **116**, *94.9*, (16.8–958)	26.8	
PCB 156	16.2, **13.7**, *11.9*, (0.293–82.3)	3.14	40.2
PCB 157	2.27, **1.93**, *1.63*, (0.204–10.9)	0.441	5.64
PCB 167	2.29, **1.80**, *1.85*, (0.213–11.6)	0.437	0.109
PCB 170	53.9, **48.3**, *35.2*, (5.80–313)	10.8	
PCB 180	106, **94.9**, *74.8*, (11.3–833)	20.9	
PCB 183	10.8, **8.64**, *7.57*, (1.38–64.9)	2.22	
PCB 187	23.0, **19.9**, *16.2*, (2.17–142)	4.56	
PCB 189	2.17, **1.91**, *1.43*, (0.187–9.60)	0.431	1.13
PCB 194	15.3, **13.8**, *10.4*, (1.23–81.9)	3.01	
PCB 206	2.03, **1.77**, *1.45*, (0.175–9.04)	0.401	
PCB 209	0.716, **0.468**, *0.711*, (0.029–6.35)	0.146	
Sum of marker PCBs[b]	343, **294**, *235*, (43.4–2,360)		
Sum of PCBs[b]	502, **437**, *338*, (63.2–3,240)		
WHO_{PCB}-TEQ[a]	20.7, **16.7**, *15.7*, (2.46–129)		

Percentages of congeners of the sum of congeners and of WHO_{PCB}-TEQ are also shown.
Abbreviations: nq, below limit of quantitation; PCB, polychlorinated biphenyl; WHO_{PCB}-TEQ, WHO toxic equivalency factors for PCBs.
[a] = concentrations are given in pg g^{-1} fat.
[b] = concentrations are given in ng g^{-1} fat.

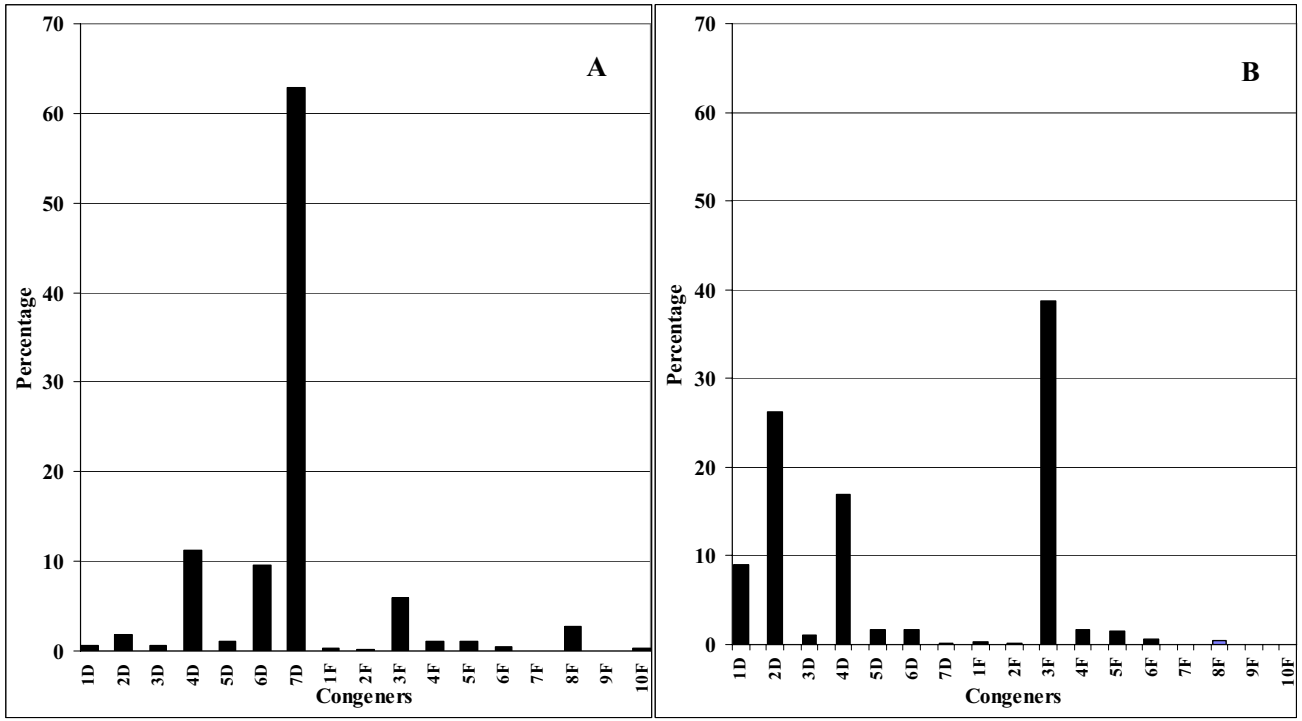

Fig. 1. Percentages of PCDD/F congeners in tissue samples of average Finnish. A: percentages from sum of PCDD/Fs, and B: percentages from $WHO_{PCDD/F}$-TEQ. Congeners: (1D) 2,3,7,8-TCDD; (2D) 1,2,3,7,8-PeCDD; (3D) 1,2,3,4,7,8-HxCDD; (4D) 1,2,3,6,7,8-HxCDD; (5D) 1,2,3,7,8,9-HxCDD; (6D) 1,2,3,4,6,7,8-HpCDD; (7D) OCDD; (1F) 2,3,7,8-TCDF; (2F) 1,2,3,7,8-PeCDF; (3F) 2,3,4,7,8-PeCDF; (4F) 1,2,3,4,7,8-HxCDF; (5F) 1,2,3,6,7,8-HxCDF; (6F) 2,3,4,6,7,8-HxCDF; (7F) 1,2,3,7,8,9-HxCDF; (8F) 1,2,3,4,6,7,8-HpCDF; (9F) 1,2,3,4,7,8,9-HpCDF and (10F) OCDF.

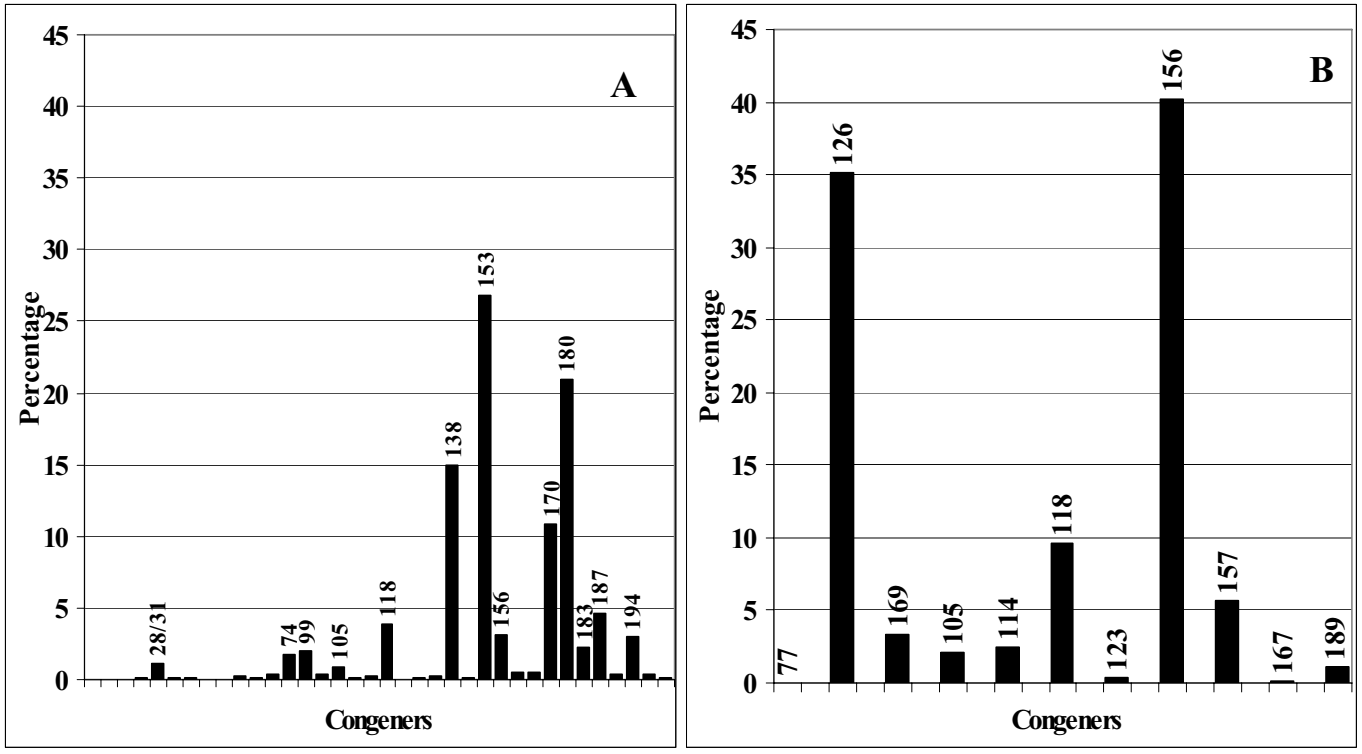

Fig. 2. Percentages of PCB congeners in tissue samples of the general Finnish population. (A) percentages from sum of PCBs, and (B) percentages from WHO_{PCB}-TEQ.

Concentrations of PCDD/Fs and PCBs

The mean and median concentrations, standard deviations, and ranges of PCDD/Fs and PCBs along with sums of congeners and TEQs in all subjects are summarized in Tables 2 and 3. The mean and median $WHO_{PCDD/F}$-TEQ concentrations were 29.0 and 24.1 pg g^{-1} fat, respectively, and the congeners contributing most to the $WHO_{PCDD/F}$-TEQ were in ranked order; 2,3,4,7,8-PeCDF, 1,2,3,7,8-PeCDD, 1,2,3,6,7,8-HxCDD, and 2,3,7,8-TCDD (sum contribution 91%). On the other hand, the congeners contributing the most to the sum of congener's mean and median concentrations (413 and 364 pg g^{-1} fat) were OCDD, 1,2,3,4,6,7,8-HpCDD, 1,2,3,6,7,8-HxCDD, and 2,3,4,7,8-PeCDF (Fig. 1). The mean and median WHO_{PCB}-TEQ concentrations were 20.7 and 16.7 pg g^{-1} fat, respectively, and the mean and median PCB sum concentrations 502 and 437 ng g^{-1} fat, respectively. In Fig. 2 the contributions of PCB congeners to sum of PCBs and to the WHO_{PCB}-TEQ are depicted. Congeners PCB 153, 180, 138, and 170 were the most abundant congeners of the sum of PCBs, while PCB 156, 126, 118, and 157 dominated the congener profiles of WHO_{PCB}-TEQ (91%).

The age dependence of TEQ concentrations is illustrated in Figs. 3 and 4. The correlation of age and $WHO_{PCDD/F}$-TEQ was r = 0.71, for age and WHO_{PCB}-TEQ r = 0.67. The increase of $WHO_{PCDD/F}$-TEQ concentration by age was best explained by the exponential function: $y = 5.4071\ e^{0.0338\ x}$. With respect to WHO_{PCB}-TEQ, the function was $y = 3.3201\ e^{0.0363\ x}$. The concentrations of men and women did not differ from each other in all subjects ($p < 0.49$ for $WHO_{PCDD/F}$-TEQ, and $p < 0.07$ for WHO_{PCB}-TEQ). In the age groups between 36 and 65 years, there was a pattern that the concentrations were lower in women than men, but only in the age group 36-40 years was this difference statistically significant.

Concentrations, standard deviations, and ranges of the most abundant PCDD/Fs and PCBs along with sums of congeners and TEqs in the different subgroups are summarized in Tables 4 and 5. The $WHO_{PCDD/F}$-TEQ in the age group < 46 years was 17.2 pg g^{-1} fat (15.7 median) which was significantly lower than the corresponding concentration in the older population, 42.7 pg g^{-1} fat (39.5 median). The concentrations of all selected congeners were significantly lower in younger people. The covariate analysis of variance indicated that the age difference between the places of residence (Table 1) did affect the concentrations to the extent that the comparison between areas was not feasible. This was also the case with selected PCB congeners. Also the PCB concentrations were significantly lower in the younger population than in the older, e.g. the average WHO_{PCB}-TEQ was 11.8 pg g^{-1} fat (10.3 median) in the younger population and 30.9 pg (26.6 median) in the older population.

In order to compare the concentrations between places of residence, we adjusted for age for every congener's concentration to the entire study population. In this way, age adjusted mean and median concentrations of the most abundant PCDD/Fs and PCBs along with sums of congeners and TEQs in different areas are summarized in Table 6. In the age adjusted concentrations of PCDD/Fs, congeners 2,3,4,7,8-PeCDF, 2,3,7,8-TCDD, and 1,2,3,7,8-PeCDD showed the highest decreasing gradient of concentrations from the coastal area to inland area, while for the congeners 1,2,3,4,6,7,8-HpCDD and OCDD there was hardly any appreciable decreasing gradient in their concentrations. All selected PCB age adjusted concentrations showed a decreasing gradient, being highest in the coastal area and lowest in inland area.

Regression models of PCDD/Fs and PCBs

The summary of the regression analyses conducted to determine predictors of the variance of natural logarithms of $WHO_{PCDD/F}$-TEQ, WHO_{PCB}-TEQ, and WHO_{total}-TEQ is shown in Table 7. The models explained 70%, 69%, and 72% of the variance of the dependents, respectively. Age, lactation, living in the inland area, and Baltic herring and farmed trout/salmon consumption frequency predictors were significant regression predictors in all models. Age was the most important predictor with a contribution of at least 64% in all models. 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 the models.

To validate the obtained regression models, we used the $WHO_{PCDD/F}$-TEQ, WHO_{PCB}-TEQ, and WHO_{total}-TEQ data from soft tissue sarcoma case patients. Useful data was obtained from 102 cases out of 148. In Fig. 5 the measured concentrations are shown with a ln scale as a function of the modelled concentrations. The correlations coefficients between modelled and measured concentrations for ln $WHO_{PCDD/F}$-TEQ, ln WHO_{PCB}-TEQ, and ln WHO_{total}-TEQ were 0.81, 0.74, and 0.80, respectively.

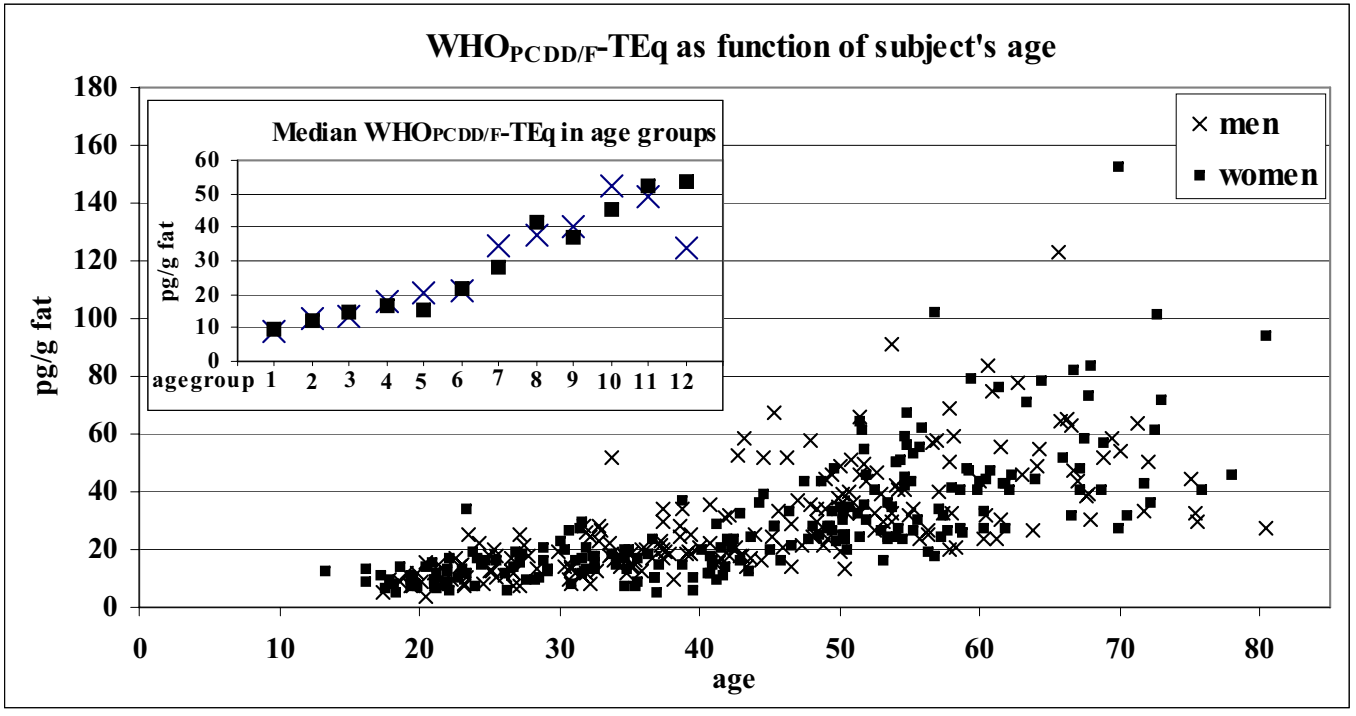

Fig. 3. $WHO_{PCDD/F}$-TEQ concentrations in Finnish tissue samples as a function of the age of the subject (n = 420). In the inset median concentrations in age groups: (1) 16-20; (2) 21-25; (3) 26-30; (4) 31-35; (5) 36-40; (6) 41-45; (7) 46-50; (8) 51-55; (9) 56-60; (10) 61-65; (11) 66-70 and (12) 71-80 years.

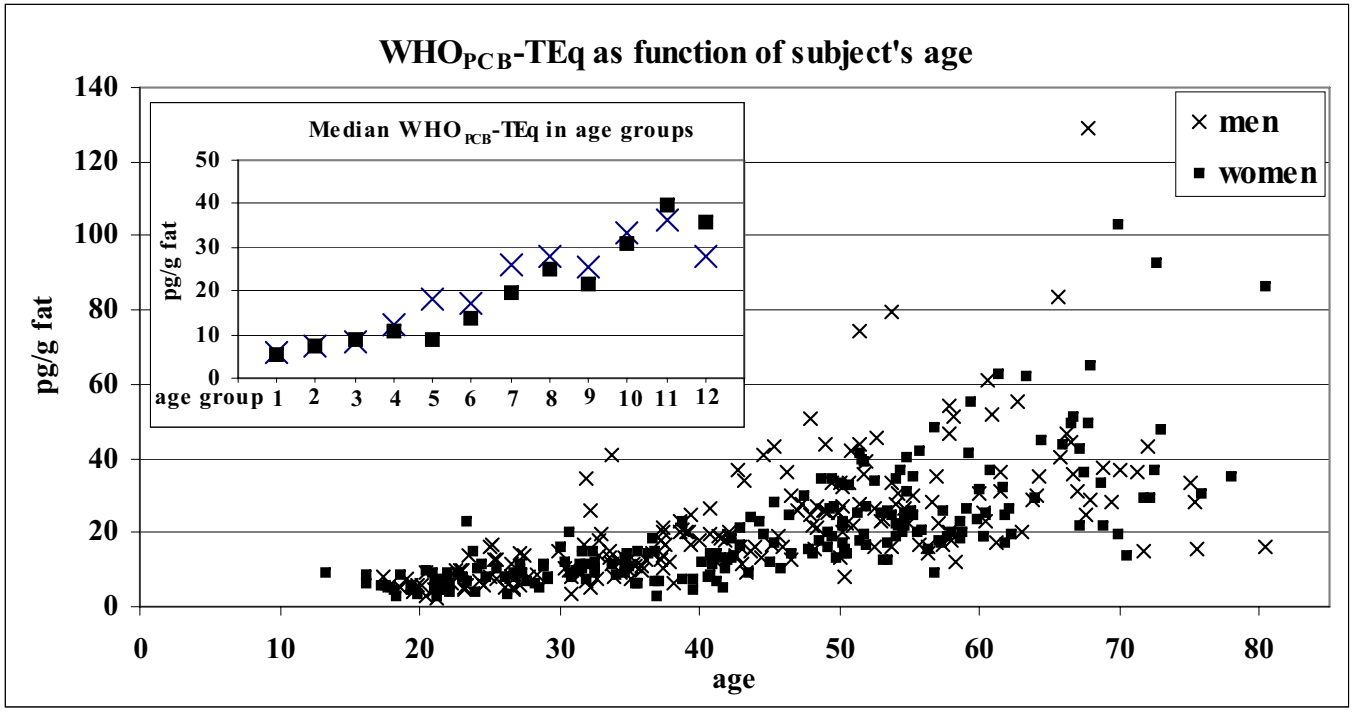

Fig. 4. WHO_{PCB}-TEQ concentrations in Finnish tissue samples as a function of the age of subject (n = 420). In the inset: median concentrations in age groups: (1) 16-20; (2) 21-25; (3) 26-30; (4) 31-35; (5) 36-40; (6) 41-45; (7) 46-50; (8) 51-55; (9) 56-60; (10) 61-65; (11) 66-70; (12) 71-80 years.

5. DISCUSSION

Demographics and fish consumption

With respect to their BMI values and fish consumption habits, the subjects in this study represented the general Finnish population. The mean and median BMI were similar to BMIs measured in the recent Finnish adult health study (Helakorpi et al., 2003) in which the proportion of subjects with BMI exceeding 25 kg m^{-2} was 50%. Also the higher BMI in young men versus women was reported in that study (Helakorpi et al., 2003). The proportions of subjects who had consumed fish at least once in the previous week was similar in both studies, 66% in this study and 72% in the Finnish adult health study. Although there were no statistical significances between the differences in fish species consumed in places of residence, it is likely that there was greater consumption of Baltic herring in the coastal area due to the proximity of this fish source.

Concentrations of PCDD/Fs and PCBs

Adipose tissue PCDD/F and PCB concentrations measured in this study showed that average exposure in Finland to these contaminants was similar to those recently reported in Belgium, France, Germany, Spain, Sweden, and US (Päpke, 1998; Wicklund Glynn et al., 2000; Wingfors et al., 2000; Arfi et al., 2001; Covaci et al., 2002; Koppen et al., 2002; Costabeber and Emanuelli, 2003; Schecter et al. 2003; Wicklund Glynn et al., 2003;) (Table 8). In India (Kumar et al., 2001) and Japan (Choi et al., 2002) the reported concentrations were slightly lower than those detected in Finland. However in the general Inuit populations in Greenland and Uelen/Russia, due to their consumption of meat and blubber of marine mammals, the PCB body burdens were about ten times higher than in the general population in Finland (Dewailly et al., 1999; Sandanger et al., 2003). The exposure of professional fishermen in Finland, especially Baltic Sea fishermen, to PCDD/Fs and PCBs was about four times as high as the general population (Kiviranta et al., 2002). PCB concentrations measured in fishermen in Latvia and Sweden were also higher than in the general population in Finland (Sjödin et al., 2000), but somewhat lower than in Finnish Baltic Sea fishermen.

Similar PCDD/F congener profiles as in this study have been reported in other studies in the 1990s (Päpke, 1998; Arfi et al., 2001; Kumar et al., 2001; Choi et al., 2002). In US and Canada the OCDD/F profile differs from the one in Finland. In $WHO_{PCDD/F}$-TEQ profile the contributions of 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, and 1,2,3,6,7,8-HxCDD to profile exceed the contribution of 2,3,4,7,8-PeCDF, which is the main contributor in Finnish profile (Schecter et al., 1994; Schecter et

al., 2003). Although the comparison of PCB congener profiles between studies is difficult due to the different numbers of measured congeners, the dominant congeners reported in most studies are the same (PCB 153, 180, 138, and 170), as in this study.

The concentrations of PCDD/Fs increased with ages of the subjects. If the half-life of congeners in humans is in the order of 7-8 years, then at a constant exposure this would mean that the body burden of a person would increase until about 40 years of age, and then achieve a steady state. At the population level, we did not detect this kind of upward convex curve probably indicating a previous higher exposure. The decreasing time trend of PCDD/F and PCB concentrations in human samples have been frequently reported (Päpke, 1998; Kiviranta et al., 1999; Norén and Meironyte, 2000; He et al., 2001; Choi et al., 2002). The higher TEQ concentrations in men than in women in the age groups between 36 and 50 years may be explained by two obvious reasons. First, men in the age range of interest had higher BMI, which might correlate with higher intake of dietary fat, and hence higher exposure to PCDD/F and PCBs than in women. Second, breast-feeding results in a decreased body burden of PCDD/Fs and PCBs in women (Abraham et al., 1998; Päpke, 1998; Kiviranta et al., 1999). The decreasing gradient of PCDD/F and PCB concentrations from the coast of the Baltic Sea to inland area in Finland was reported in Finnish mother's milk samples collected in 1987 (Vartiainen et al., 1997). In order to investigate if this decreasing gradient would be present also in the general population concentrations, we classified the subjects according to place of residence. Coastal and inland groups were obvious groups based on the earlier mother's milk study. Although the capital area is located on the coastline of the Gulf of Finland in the Baltic Sea, we classified it as an intermediate group between coastal and inland areas. This was based on the fact that there has been a considerable internal emigration from inland areas to the capital area over the last 50 years. Therefore the population in the capital area is a mixture of coastal and inland area populations with respect to their PCDD/F and PCB exposure. When concentrations of PCDD/Fs and PCBs were age adjusted, this gradient of decreasing concentrations from coast to inland area was seen for all selected PCB congeners and also for certain PCDD/F congeners. This can be explained by the fact that most of the average exposure of Finns to PCDD/Fs and PCBs originates from fish, especially from Baltic herring (Kiviranta et al. 2003), and in this study Baltic herring was consumed most frequently in the coastal area, although not statistically significantly. Also the concentrations of PCDD/Fs and PCBs are higher in the Baltic Sea in most of the fish species when compared to the same fish species in inland areas, also resulting in higher population exposure to these contaminants in the coastal area. The failure to detect any clear decreasing gradient of exposure for 1,2,3,4,6,7,8-HpCDD and OCDD from the coastal

Table 4.

Mean, **median**, *SD*, and (range) concentrations of selected PCDD/Fs and $WHO_{PCDD/F}$-TEQs as pg g^{-1} fat in adipose tissue samples in age and regional subgroups of general population in Finland.

Congener	Age subgroups		Area		
	< 46 years n = 225	> 46 years n = 195	Coastal n = 147	Capital n = 104	Inland n = 166
2,3,7,8-TCDD	1.59, **1.37**, *0.926*, (0.157–6.56)*	3.66, **3.23**, *2.00*, (0.794–16.4)	2.64, **1.95**, *2.13*, (0.551–16.4)**	2.39, **2.12**, *1.52*, (0.478–8.85)	2.57, **2.04**, *1.76*, (0.157–8.98)
1,2,3,7,8-PeCDD	4.44, **4.05**, *2.45*, (0.986–17.6)*	11.3, **9.92**, *5.60*, (3.72–43.2)	7.87, **5.75**, *6.37*, (1.38–43.2)**	6.73, **5.70**, *4.39*, (1.56–25.6)	7.98, **6.90**, *5.09*, (0.986–27.0)
1,2,3,6,7,8-HxCDD	30.5, **28.1**, *16.2*, (5.10–113)*	60.1, **56.2**, *20.9*, (24.3–148)	44.9, **39.7**, *26.3*, (5.10–148)**	40.9, **36.8**, *22.4*, (8.48–134)	45.9, **45.9**, *22.0*, (5.37–109)
1,2,3,4,6,7,8-HpCDD	30.2, **26.7**, *18.6*, (nq–107)*	53.0, **47.5**, *34.2*, (8.08–222)	38.1, **30.4**, *25.6*, (5.35–140)**	40.5, **34.6**, *30.6*, (3.37–206)	43.4, **35.1**, *31.6*, (nq–222)
OCDD	217, **180**, *124*, (37.7–950)*	316, **261**, *197*, (68.7–1730)	257, **212**, *161*, (57.9–1090)**	254, **222**, *157*, (37.7–1030)	275, **237**, *184*, (67.8–1730)
2,3,4,7,8-PeCDF	12.9, **10.5**, *8.77*, (2.30–60.9)*	37.2, **33.0**, *21.7*, (8.09–165)	27.5, **19.0**, *24.3*, (2.30–165)**	21.8, **16.2**, *17.2*, (2.86–106)	22.9, **18.6**, *17.6*, (2.32–112)
Sum of PCDD/Fs	321, **290**, *165*, (78.0–1270)*	519, **456**, *249*, (163–2080)	408, **371**, *221*, (103–1450)**	392, **341**, *220*, (78.0–1420)	430, **380**, *244*, (109–2080)
$WHO_{PCDD/F}$-TEQ	17.2, **15.7**, *9.26*, (3.64–67.7)*	42.7, **39.5**, *19.8*, (13.4–153)	31.0, **22.9**, *23.1*, (4.92–153)**	26.2, **22.7**, *17.1*, (5.77–102)	29.2, **25.3**, *18.0*, (3.64–102)

Abbreviations: HpCDD, heptachlorodibenzo-*p*-dioxm; HxCDD, hexachlorodibenzo-*p*-dioxin; nq, below limit of quantitation; OCDD, octachlorodibenzo-*p*-dioxin; PeCDD, pentachlorodibenzo-*p*-dioxin; PeCDF, pentachlorodibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; $WHO_{PCDD/F}$-TEQ, WHO toxic equivalency factors for PCDD/Fs.

*Significantly different compared with the > 46 years group ($p < 0.05$ by independent samples T-test)

** Age as a covariate significantly effects the concentration in different areas (covariate analysis of variance), and therefore areas are not comparable.

Table 5.

Mean, **median**, *SD*, and (range) concentrations of selected PCBs and WHO_{PCB}-TEQs in adipose tissue samples in age and regional subgroups of general population in Finland.

Congener	Age subgroups		Area		
	< 46 years n = 225	> 46 years n = 195	Coastal n = 147	Capital n = 104	Inland n = 166
Non-*ortho*-PCBs [a]					
PCB 126	40.9, **35.2**, *27.7*, (nq–170)*	115, **93.8**, *91.3*, (15.3–817)	81.3, **52.8**, *88.5*, (12.2–817)**	72.5, **51.1**, *66.1*, (11.9–454)	72.0, **56.1**, *67.5*, (nq–541)
PCB 169	39.0, **32.1**, *25.8*, (nq–169)*	100, **86.2**, *54.8*, (36.4–399)	75.2, **55.1**, *63.9*, (5.63–399)**	63.6, **55.2**, *45.4*, (8.04–259)	63.4, **54.3**, *42.5*, (nq–295)
Other PCBs [b]					
PCB 118	11.8, **9.98**, *8.13*, (2.14–49.0)*	29.3, **25.4**, *19.4*, (4.19–134)	21.6, **15.4**, *18.8*, (3.24–100)**	20.2, **15.3**, *18.2*, (2.69–134)	18.4, **14.3**, *14.3*, (2.14–106)
PCB 138	47.5, **42.7**, *25.8*, (9.08–167)*	106, **92.7**, *58.5*, (10.3–461)	81.6, **58.3**, *67.1*, (9.08–461)**	71.7, **62.9**, *45.9*, (9.89–244)	71.0, **64.8**, *41.8*, (11.2–251)
PCB 153	85.9, **78.1**, *46.9*, (16.8–290)*	192, **165**, *104*, (30.2–958)	146, **107**, *123*, (16.8–958)**	131, **114**, *82.0*, (20.2–433)	129, **122**, *70.8*, (19.1–423)
PCB 156	9.51, **7.86**, *6.13*, (0.293–43.0)*	24.0, **21.6**, *12.3*, (0.795–82.3)	17.6, **13.4**, *14.9*, (1.78–82.3)**	15.1, **12.6**, *10.5*, (0.795–47.0)	15.9, **14.5**, *9.51*, (0.293–49.3)
PCB 157	1.34, **1.06**, *0.866*, (0.204–5.71)*	3.33, **2.94**, *1.66*, (0.530–10.9)	2.48, **1.91**, *2.06*, (0.243–10.9)**	2.12, **1.81**, *1.40*, (0.315–6.04)	2.19, **2.10**, *1.32*, (0.204–6.54)
PCB 170	34.5, **29.5**, *21.3*, (5.80–132)*	76.2, **68.6**, *34.8*, (12.2–313)	56.1, **45.7**, *42.8*, (5.80–313)**	50.8, **44.1**, *31.1*, (7.32–146)	54.1, **51.1**, *30.0*, (7.49–157)
PCB 180	65.7, **55.8**, *40.3*, (11.3–254)*	152, **135**, *78.6*, (23.8–833)	111, **88.7**, *96.3*, (11.3–833)**	101, **86.8**, *62.7*, (14.2–277)	105, **100**, *58.9*, (13.5–337)
Sum of marker PCBs [b]	216, **190**, *118*, (43.4–744)*	488, **423**, *251*, (83.8–2360)	368, **280**, *302*, (43.4–2360)**	331, **296**, *204*, (48.4–1110)	329, **307**, *180*, (53.0–1080)
Sum of PCBs [b]	317, **282**, *175*, (63.2–1110)*	716, **622**, *354*, (124–3240)	540, **414**, *433*, (63.2–3240)**	482, **437**, *294*, (73.5–1590)	485, **460**, *260*, (79.2–1530)
WHO_{PCB}-TEQ [a]	11.8, **10.3**, *6.93*, (2.46–43.1)*	30.9, **26.6**, *16.8*, (7.89–129)	22.4, **16.3**, *19.3*, (2.89–129)**	19.7, **16.4**, *14.2*, (3.00–92.8)	19.9, **17.4**, *12.9*, (2.46–86.5)

Abbreviations: nq, below limit of quantitation; PCB, polychlorinated biphenyl; WHO_{PCB}-TEQ, WHO toxic equivalency factors for PCBs.

*Significantly different compared with the > 46 years group ($p < 0.05$ by independent samples T-test)

** Age as a covariate significantly effects the concentration in different areas (covariate analysis of variance), and therefore areas are not comparable.

[a] = concentrations are given in pg g^{-1} fat.

[b] = concentrations are given in ng g^{-1} fat

Table 6.

Mean and **median** age adjusted concentrations of selected PCDD/Fs, $WHO_{PCDD/F}$-TEQs, PCBs, and WHO_{PCB}-TEQs in adipose tissue samples in regional subgroups of average population in Finland.

Congener	Area			Congener	Area		
	Coastal n = 147	Capital n = 104	Inland n = 166		Coastal n = 147	Capital n = 104	Inland n = 166
PCDD/Fs[a]				Non-*ortho*-PCBs [a]			
2,3,7,8-TCDD	2.77, **2.55**	2.55, **2.34**	2.34, **2.04**	PCB 126	82.5, **71.8**	80.1, **73.5**	65.5, **57.7**
1,2,3,7,8-PeCDD	8.30, **7.55**	7.22, **6.71**	7.24, **6.69**	PCB 169	80.0, **72.5**	69.4, **66.9**	58.5, **54.4**
1,2,3,6,7,8-HxCDD	47.7, **44.3**	43.6, **40.2**	42.6, **40.6**				
1,2,3,4,6,7,8-HpCDD	39.7, **36.0**	44.0, **38.4**	40.7, **34.9**	Other PCBs [b]			
OCDD	277, **248**	268, **230**	262, **233**	PCB 118	23.0, **20.7**	21.1, **18.8**	16.7, **15.2**
				PCB 138	86.5, **81.8**	74.3, **68.3**	64.9, **62.8**
2,3,4,7,8-PeCDF	28.8, **26.5**	24.1, **22.9**	20.4, **18.7**	PCB 153	155, **141**	137, **129**	118, **112**
				PCB 156	18.6, **16.6**	15.7, **15.1**	14.5, **13.7**
Sum of toxic congeners	435, **406**	417, **373**	407, **362**	PCB 157	2.64, **2.43**	2.23, **2.13**	1.99, **1.86**
				PCB 170	60.5, **54.4**	53.2, **52.2**	49.5, **47.9**
$WHO_{PCDD/F}$-TEQ	32.6, **30.1**	28.5, **27.2**	26.4, **24.4**	PCB 180	120, **105**	106, **104**	96.2, **90.4**
				Sum of marker PCBs[b]	392, **359**	345, **326**	302, **288**
				Sum of PCBs[b]	574, **524**	502, **476**	445, **425**
				WHO_{PCB}-TEQ[a]	23.4, **21.6**	21.1, **19.5**	18.1, **17.4**

Abbreviations: HpCDD, heptachlorodibenzo-*p*-dioxin; HxCDD, hexachlorodibenzo-*p*-dioxin; nq, below limit of quantitation; OCDD, octachlorodibenzo-*p*-dioxin; PeCDD, pentachlorodibenzo-*p*-dioxin; PeCDF, pentachlorodibenzofuran; PCB, polychlorinated biphenyl; TCDD, tetrachlorodibenzo-*p*-dioxin; WHO_{PCB}-TEQ, WHO toxic equivalency factors for PCBs; $WHO_{PCDD/F}$-TEQ, WHO toxic equivalency factors for PCDD/Fs.

[a] = concentrations are given in pg g^{-1} fat.

[b] = concentrations are given in ng g^{-1} fat.detect any clear decreasing gradient

Table 7.
Predictors of the variance of natural logarithms of $WHO_{PCDD/F}$-TEQ, WHO_{PCB}-TEQ, and total WHO-TEQ for population in Finland.

Predictor variable	Parameter estimate	SE	*p*-Value
Dependent variable: ln $WHO_{PCDD/F}$-TEQ			
Constant	1.73	0.15	< 0.0001
Age (years)	0.0348	0.002	< 0.0001
BMI (kg m^{-2})	- 0.00354	0.006	< 0.54
Lactation (months)	- 0.0106	0.002	< 0.0001
Living in the capital area (no/yes)	- 0.089	0.053	< 0.093
Living in the inland area (no/yes)	- 0.116	0.048	< 0.015
Consumption of baltic herring (times per month)	0.037	0.014	< 0.009
Consumption of farmed trout or salmon (times per month)	0.0374	0.012	< 0.002
Consumption of other fish (times per month)	0.0087	0.008	< 0.25
ln $WHO_{PCDD/F}$-TEQ model percentage $r^2 = 0.70$			
Dependent variable: ln WHO_{PCB}-TEQ			
Constant	1.19	0.16	< 0.0001
Age (years)	0.037	0.002	< 0.0001
BMI (kg m^{-2})	- 0.0017	0.006	< 0.79
Lactation (months)	- 0.0101	0.002	< 0.0001
Living in the capital area (no/yes)	- 0.0196	0.059	< 0.074
Living in the inland area (no/yes)	- 0.145	0.053	< 0.007
Consumption of baltic herring (times per month)	0.0377	0.016	< 0.017
Consumption of farmed trout or salmon (times per month)	0.0504	0.013	< 0.0001
Consumption of other fish (times per month)	- 0.0000987	0.008	< 0.99
ln WHO_{PCB}-TEQ model percentage $r^2 = 0.69$			
Dependent variable: ln WHO_{total}-TEQ			
Constant	2.19	0.15	< 0.0001
Age (years)	0.0357	0.002	< 0.0001
BMI (kg m^{-2})	- 0.00304	0.006	< 0.60
Lactation (months)	- 0.0104	0.002	< 0.0001
Living in the capital area (no/yes)	- 0.0573	0.053	< 0.28
Living in the inland area (no/yes)	- 0.126	0.047	< 0.008
Consumption of baltic herring (times per month)	0.0368	0.014	< 0.009
Consumption of farmed trout or salmon (times per month)	0.0422	0.012	< 0.001
Consumption of other fish (times per month)	- 0.00492	0.020	< 0.51
ln WHO_{total}-TEQ model percentage $r^2 = 0.72$			

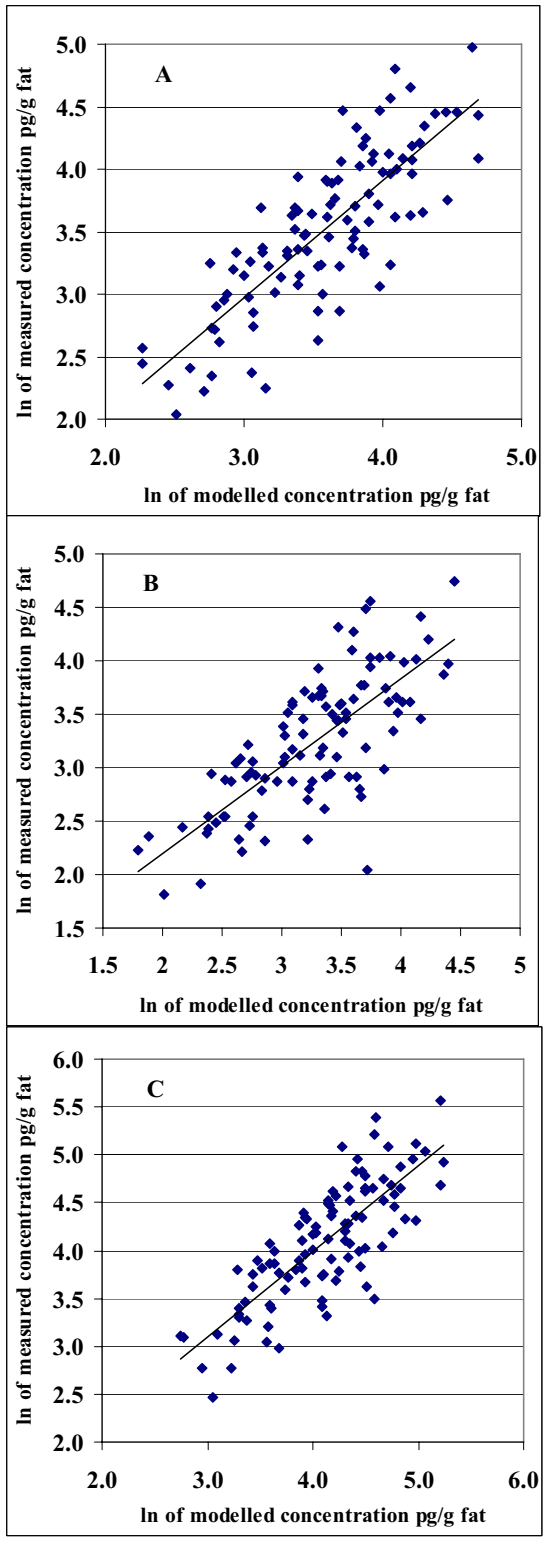

Fig. 5.
Measured concentrations as functions of modelled concentrations: (A) ln $WHO_{PCDD/F}$-TEQ; (B) ln WHO_{PCB}-TEQ; and (C) ln WHO_{total}-TEQ.

Table 8.
Adipose and serum fat concentrations of sum of PCDD/Fs, $WHO_{PCDD/F}$-TEQ, PCB 126, PCB 153, PCB 180, markes PCBs, and WHO_{PCB}-TEQ in various countries during 1990s.

Adipose tissue studies:									
Origin	Finland	Spain	Japan	Belgium	India	France	Sweden	Greenland	Germany
Year of sampling	1997-1099	1996-1997	2000	2000	2000	1999	Unknown	1992-1994	1996
Number of samples	420	123	10	20	21	16	28	26	139
Mean age of subjects	44 (13-81)	51 (15-87)	40-50	47 (19-77)	20-69	53 (30-94)	68	60	37 (18-71)
Population	Men/women	Men/women	Men/women	Men/women	Men/women	Men/women	Men/women	Men/women	Men/women
Sum of PCDD/Fs[a]	413		171		550	490	804		403
$WHO_{PCDD/F}$-TEQ[a]	29.0		11.9		14.4	35.6	32.8		16.1
PCB126[a]	75.2		72		125		180		
$PCB153_b$	135	121		211			300	1689	
PCB180[b]	106	134		105			200	1147	
marker PCBs[b]	343	432					778	4242	
WHO_{PCB}-TEQ[a]	20.7		15.3		14.4				
Reference	this study	Costabeber and Emanuelli (2003)	Choi et al. (2002)	Covaci et al. (2002)	Kumar et al. (2001)	Arfi et al. (2001)	Wingfors et al. (2000)	Dewailly et al. (1999)	Päpke (1998)

Serum/blood studies:									
Origin	Finland	Sweden	Uelen/Russia	Belgium	Sweden	Latvia	Sweden	Åland/Finland	US
Year of sampling	1997	1996-1997	2001	1999	1991	1993	late 1990s	mid 1990s	2002
Number of samples	47	205	50	200	43	67	120	30	249
Age of subjects	58 (27-77)	63 (50-74)	37 (20-70)	58 (50-65)	42 (23-69)	48 (24-79)	63 (40-75)	30 (19-40)	adults
Population	Fishermen	Women	Men/women	Women	Fishermen	Fishermen	Men	Women	Men/women
Sum of PCDD/Fs[a]	1700			999					505
$WHO_{PCDD/F}$-TEQ[a]	180			48					19.3
PCB126[a]	300		1200	102					
PCB153[b]	600	223	744	168	360	403	296	56	
PCB180[b]	370	152	220	104	233	194	218	32	
marker PCBs[b]	1460		1410	389			675		
WHO_{PCB}-TEQ[a]	89			23.7					
Reference	Kiviranta et al. (2002)	Wicklund Glynn et al. (2003)	Sandanger et al. (2003)	Koppen et al. (2002)	Sjödin et al. (2000)	Sjödin et al. (2000)	Wicklund Glynn et al. (2000)	Hagmar et al. (1998)	Schecter et al. (2003)

[a] = concentrations are given in pg g^{-1} fat.
[b] = concentrations are given in ng g^{-1} fat

area to the inland area is also in line with the previous consideration that fish are not the source of these congeners (Kiviranta et al., 2003). However fish are the source of 2,3,4,7,8-PeCDF, 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, and also 1,2,3,6,7,8-HxCDD. For those congeners, the decreasing gradient of concentrations from coast to inland area was most clearly evident.

Regression models of PCDD/Fs and PCBs

In the regression models the goal was to develop a suitable model to assess a population body burden of PCDD/Fs and PCBs in order to avoid measuring those concentrations with expensive and time-consuming methods. According to previous and this present study, the predictor variables were chosen according to the criteria that these variables would contribute most to the Finnish body burdens of PCDD/Fs and PCBs, and that they would be easy to enquire from a questionnaire. Age, weight, length, lactation, place of residence, and fish consumption frequencies fulfilled those criteria. The analysis showed that age was the most significant predictor in all three models, but also lactation, place of residence, and frequency of consumption of Baltic herring and farmed trout or salmon were significant variables in the models. The correlations between modelled and measured concentrations for TEQs were quite satisfactory. On the individual basis, the models failed most often to assess the concentrations of older people. Changes in food habits and also changes in PCDD/F and PCB concentrations in food items throughout the years are most evident in older subjects, resulting in more scattering between the modelled and measured concentrations.

6. CONCLUSION

We found that the body burdens of PCDD/Fs and PCBs in Finland were at the same levels as other countries in Europe, and there existed a downward trend in concentrations from coastal to inland areas. This decreasing trend in concentrations was most likely a result from consumption of more contaminated fish from the Baltic Sea in the coastal area compared to the inland area. The age dependence of concentrations was shown to be strong. Concentrations as a function of age also revealed that the exposure of Finns to PCDD/Fs and PCBs has declined over the last 30 years. By asking subjects age, height, weight, place of residence, and fish consumption frequencies it would be possible to obtain an estimate of their PCDD/F and PCB TEQ body burdens.

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CHAPTER 6

LEVELS AND TRENDS OF PCDD/Fs AND PCBs IN HUMAN MILK IN FINLAND

Hannu Kiviranta[1], Raija Purkunen[1], and Terttu Vartiainen*[1.,2]

[1]Division of Environmental Health, National Public Health Institute, P.O.Box 95, FIN-70701 Kuopio, Finland

[2]Department of Environmental Sciences, University of Kuopio, P.O.Box 1627, FIN-70211 Kuopio, Finland

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1. ABSTRACT

Word Health Organization, WHO/EURO, has coordinated two rounds of follow-up studies on levels of PCDDs, PCDFs, and PCBs in human milk which were analyzed as two pooled samples from each participating country, one from urban and the other one from rural area. Finland has taken part to both of those studies and we are now reporting results of all the - second round randomly sampled human milk samples (84 samples) from Southern (20) and Eastern (64) Finland. The levels of PCDD/Fs and PCBs in human milk in Southern Finland were considerably higher than in Eastern Finland. The level of PCDD/Fs in human milk in Southern Finland was the same as in the Central Europe but the level in Eastern Finland was similar to levels in Norway and eastern parts of Europe. The concentrations of PCDD/Fs and PCBs showed a significant decrease from 1987 to 1994. Declining of PCDD/Fs and PCBs was 36 and 49 % in primiparae mothers= milk, respectively. This decrease in concentrations of PCDD/F and PCB was slightly greater in Eastern than in Southern Finland.

2. INTRODUCTION

Polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/F) are globally distributed toxic chemicals in the environment and were found in human milk in the 1980s [1,2]. Food is the main source of PCDD/Fs and polychlorinated biphenyls (PCB) in humans [3]. In Finland, meat, milk and milk products were quite clean of PCDD/Fs but sometimes eggs contained

PCDD/Fs, because the feed of poultry had contained fish products [4]. The Baltic Sea is highly contaminated with PCDD/Fs and PCBs causing also contamination of Baltic herring and salmon [5]. PCB concentrations in Finnish food have been reported to be low, except for Baltic fish, on average 32 μg/kg in beef, 11 μg/kg in pork, 9 μg/kg in chicken, 29 μg/kg in eggs, and 0.21 pg/kg in milk containing 1.9% fat [6]. The total intake of PCDD/Fs as international toxic equivalent (I-TEq) in Finland was assumed to be about 94 pg/day per person [7] and total PCB intake 1.64 μg/day per person [6].

The decrease of concentrations of PCDD/Fs between 1986 and 1993 in human blood and milk has been reported from Germany and the Netherlands [8,9]. Concentrations of PCBs have also been diminishing but not as clearly as concentrations of PCDD/Fs [9]. The studies conclude that measures taken to reduce the PCDD/F and PCB emissions to the environment have resulted in a reduction of human body burdens of these compounds.

WHO/EURO has coordinated two rounds of follow-up studies on levels of PCDDs, PCDFs and PCBs in human milk. Finland participated in both of them [10,11]. The objectives in this study are to describe the concentrations of PCDD/Fs and PCBs in human milk in two areas in Finland between 1992 and 1994, and to evaluate the time trend of the concentrations of PCDD/Fs and PCBs between 1987 and 1994.

3. METHODS

Sample Collection

The study was part of a follow-up study coordinated by WHO/EURO on levels of PCDDs, PCDFs and PCBs in human milk. In Finland, we carried out a larger population-based study in two geographic areas, in Helsinki, the capital, and in Kuopio and its surroundings, which is located approximately 400 km north-east from Helsinki. All consecutive women giving birth were recruited from one of the maternity clinics in Helsinki and from the maternity clinic of Kuopio University Hospital between March 1992 and August 1994, in WHO/EURO study between April 1992 and August 1993. The study population who collected and returned the milk sample constituted a total of 84 mothers, 20 (24 % of the total number) in Helsinki and 64 (76 %, respectively) in Kuopio. In the urban and rural areas 14 and 28 mothers were primiparae, respectively. In the rural area, human milk sample from a mother nursing her thirteenth child was found, and in the urban area a sample from a mother nursing her fourth child. The study population is described in Table 1.

Table 1.
Number of mothers, ages of mothers and fat contents of human milk in urban (N=20) and rural (N=64) areas of Finland between 1992-1994. Numbers in the panel indicate the number of child mother was nursing. 1./(WHO) indicates that mother was primipara between 20-30 years and she had lived in the community at least the five last years (the pooled sample of these mothers was included in the WHO/EURO study).

mother/parity	1.	1./(WHO)	2.	3.	4.	6	13.
Urban area, Helsinki							
Number of mothers							
	14	10	3	2	1	-	-
Age of mothers							
mean ± s.d.	27.9 ± 4.6	27.0 ± 3.1	25.3 ± 0.6	32.5	34		
range	19-36	23-32	25-26	31-34			
Fat content of human milk							
mean ± s.d.	3.75 ± 1.52	3.91 ± 1.03	4.62 ± 1.06	3.64	3.31	-	-
range	0.63 - 6.61	1.82 - 5.13	3.71 - 5.78	2.73 - 4.55	-	-	-
Rural area, Kuopio and surroundings							
Number of mothers							
	28	23	19	11	4	1	1
Age of mothers							
mean ± s.d.	27.0 ± 4.7	25.4 ± 2.6	29.5 ± 4.7	33.5 ± 3.3	32.0 ± 0.8	32	42
range	18-39	18-30	23-44	27-38	31-33		
Fat content of human milk							
mean ± s.d.	3.88 ± 0.99	3.99 ± 1.05	4.36 ± 1.61	4.01 ± 1.31	4.18 ± 1.05	5.13	3.57
range	1.58 - 5.93	1.58 - 5.93	1.36 - 7.74	1.12 - 5.94	2.72 - 5.21	-	-

Determination of PCDDs, PCDFs, and PCBs

The concentrations of 17 toxic PCDD/Fs, of three non-*ortho* (IUPAC 77, 126, and 169) PCB congeners, of five mono-*ortho* (IUPAC 105, 114, 118, 156 and 157) PCB congeners, and of 28 di-*ortho* (IUPAC 18, 28, 33, 47, 49, 51, 52, 60, 66, 74, 99, 101, 110, 122, 123, 128, 138, 141, 153, 167, 170, 180, 183, 187, 189, 194, 206 and 209) PCB congeners, the total sum of PCDD/F (∑PCDD/F) and PCB (∑PCB) congeners, and toxic equivalents, I-TEqs (TEqs, for PCBs) of them were determined from human milk samples. About 40 ml of each human milk sample, equivalent to 1.2 g fat, was spiked with 115 pg of ^{13}C-labeled PCDD and PCDF standards (seventeen 2,3,7,8-chlorinated PCDD/F congeners), with 100 pg of ^{13}C-labeled non-*ortho* PCB standards (PCB 77, 126, and 169), and with 9600 pg of ^{13}C-labeled PCB standards (PCB 30 [^{12}C-labeled], 80, 101, 153, 180, Cambridge Isotope Laboratories). Milk fat was extracted with diethyl ether/hexane and the fat content determined. The extract was defatted in a silica gel column and initially purified on activated carbon column (Carbopack C, 60/80 mesh) containing Celite (Merck

2693) to separate PCDD/Fs from PCBs and both fractions further cleaned with an activated alumina column (Merck 1097, standardized, activity level II-III). The separated PCB fraction was further purified, after having analyzed for mono- and di-*ortho* PCB congeners, on another activated carbon column (without Celite) and the non-*ortho* PCBs were also analyzed with high resolution mass spectrometer equipped with a fused silica capillary column (DB-DIOXIN, 60 m, 0.25 mm, 0.15 μm). The quantitation was performed by selective ion recording using a VG 70-250 SE (VG Analytical, UK) mass spectrometer (resolution 10,000). The levels of 17 most toxic PCDD/Fs were expressed in TCDD toxic equivalents (I-TEq) calculated by using the international toxic equivalency factors [12]. Toxic equivalency factors used for PCBs were 0.1 for PCB 126, 0.01 for PCB 169, 0.0005 for PCBs 77, 114, 156, and 157, 0.0001 for PCBs 105, 118, 123, 170 and 189, and 0.00001 for PCB 167 [13]. The laboratory reagent and equipment blank samples were treated and analyzed by the same method as the actual samples, one blank for every five samples. Detection limits for the different PCDD/F congeners were 0.1 - 1.0 pg/g in fat and for the different PCB congeners 1-10 pg/g fat. Recoveries for internal standards were more than 60% for all congeners. The laboratory has participated successfully in international quality control studies for the analysis of PCDDs and PCDFs in cow milk samples organized by EU/BCR-project in 1993 [14, 15]. Laboratory is also an accredited testing laboratory (No T77) in Finland (SFS-EN 45001 and ISO/IEC Guide 25). The scope of accreditation includes PCDD/Fs, PCBs, and non-*ortho* PCBs from human milk. Statistical analysis was carried out by means of SPSS (for Windows, version 6.1.3). Mann-Whitney U nonparametric test was used to test the statistical significance of results.

4. RESULTS

Fat, PCDD/F, and PCB contents of human milk

Fat content in human milk of primiparae was on average 3.75% in the urban and 3.88% in the rural area (Table 1). In this study data are given on fat basis, as PCDD/Fs and PCBs are conventionally reported. PCDD/F concentrations as I-TEqs were between 4.90 pg/g fat (thirth child in rural area) and 34.4 pg/g fat (primipara, in urban area), and ∑PCDD/F concentrations were between 51.6 pg/g fat (second child in rural area) and 559 pg/g fat (primipara in urban area) (Tables 2a and 2b). ∑PCB concentrations ranged from 52.6 (second child in rural area) to 464 ng/g fat (primipara in urban area), and TEqs were between 2.37 (second child in rural area) and 32.8 pg/g fat (primipara in urban area) (Tables 3a and 3b). The average ∑PCDD/F and

∑PCB concentrations of all primiparae were in the urban area 381 pg/g and 296 ng/g fat, respectively, and in the rural area 217 pg/g and 198 ng/g fat, respectively. The average I-TEqs and TEqs of all primiparae were in the urban area 19.9 pg/g and 18.5 pg/g fat, respectively, and in the rural area 13.6 pg/g and 11.6 pg/g fat, respectively.

Table 2a.
PCDD/F concentrations and I-TEqs (mean ± standard deviation and range as pg/g fat) in the mother's milk from the urban area in Finland, in 1992-94. Asterisks indicate a statistically significant difference to the rural area (*p<0.01, **p<0.005, ***p<0.001). 1,2,3,7,8,9-Cl_6DF and 1,2,3,4,7,8,9-Cl_7DF were below the detection limits. Other conditions as in Table 1.

congener	1.	1./(WHO)	2.	3.	4.
2,3,7,8-Cl_4DF	1.93 ± 0.74***	1.83 ± 0.62***	1.34 ± 0.2	2.64	1.47
	0.99 - 3.31	0.99 - 2.79	1.22 - 1.57	2.39 - 2.89	
2,3,7,8-Cl_4DD	2.66 ± 1.46	2.48 ± 1.24	1.88 ± 0.61	2.87	1.03
	1.11 - 5.81	1.35 - 5.2	1.37 - 2.54	2.72 - 3.02	
1,2,3,7,8-Cl_5DF	0.79 ± 0.44*	0.73 ± 0.47	0.61 ± 0.2	0.99	0.88
	<0.1 - 1.39	<0.1 - 1.27	0.48 - 0.83	0.89 - 1.09	
2,3,4,7,8-Cl_5DF	16.3 ± 7.0*	17.0 ± 6.13**	7.95 ± 3.25	15.2	7.08
	5.2 - 27.7	7.9 - 24.8	5.15 - 11.5	12.2 - 18.2	
1,2,3,7,8-Cl_5DD	6.22 ± 2.16*	6.61 ± 1.67**	3.9 ± 1.15	5.56	2.01
	2.23 - 9.71	3.95 - 8.64	2.57 - 4.57	5.31 - 5.8	
1,2,3,4,7,8-Cl_6DF	4.91 ± 1.55	5.13 ± 1.0	2.81 ± 0.81	3.81	2.41
	2.45 - 8.15	4.01 - 6.75	2.0 - 3.63	3.14 - 4.48	
1,2,3,6,7,8-Cl_6DF	3.44 ± 1.54	3.34 ± 1.42	2.37 ± 0.67	3.26	2.07
	1.34 - 6.59	1.34 - 6.3	1.93 - 3.14	2.63 - 3.88	
2,3,4,6,7,8-Cl_6DF	1.88 ± 0.88	1.91 ± 0.91	1.19 ± 0.49	1.95	1.21
	0.91 - 4.0	1.0 - 4.0	0.89 - 1.75	1.59 - 2.31	
1,2,3,4,7,8-Cl_6DD	2.69 ± 1.33	2.68 ± 1.34	1.68 ± 0.34	2.21	1.09
	1.22 - 5.91	1.33 - 5.91	1.32 - 2.0	2.02 - 2.4	
1,2,3,6,7,8-Cl_6DD	33.2 ± 8.94	34.3 ± 6.34**	22.8 ± 4.49	26.9	11.7
	15.2 - 49.9	26.7 - 46.9	19.9 - 27.9	26.4 - 27.4	
1,2,3,7,8,9-Cl_6DD	3.03 ± 3.0	2.61 ± 3.12	2.98 ± 0.63	4.3	1.72
	0.96 - 9.68	0.95 - 9.68	2.29 - 3.53	3.92 - 4.68	
1,2,3,4,6,7,8-Cl_7DDF	9.79 ± 7.86	7.83 ± 7.43	11.8 ± 2.84	17.9	16.6
	1.35 ± 25.8	1.35 - 25.8	9.6 - 15.0	16.4 - 19.5	
1,2,3,4,6,7,8-Cl_7DD	51.7 ± 25.8***	53.8 ± 25.0***	24.9 ± 10.9	47.5	21.6
	15.1 - 110	27.5 - 110	13.6 - 35.4	40.1 - 54.9	
OCDF	11.7 ± 9.2***	12.9 ± 10.6**	9.03 ± 4.22	9.87	11.5
	<1 - 29.9	<1 - 29.9	5.92 - 13.8	9.41 - 10.3	
OCDD	230 ± 80.9***	251 ± 63.4***	102 ± 56.2	177	127
	58.0 - 349	177 - 349	66.4 - 167	161 - 194	
3 PCDD/F	381 ± 120***	404 ± 94.9***	197 ± 83.4	323	210
	128 - 559	267 - 559	140 - 293	292 - 353	
I-TEq	19.9 ± 7.42*	20.4 ± 6.01**	11.8 ± 2.95	18.7	8.31
	7.7 - 34.4	12.3 - 29.0	8.92 - 14.8	16.6 - 20.7	

Table 2b.
PCDD/F concentrations and I-TEqs (mean + standard deviation and range as pg/g fat) in the mother's milk from the surroundings of the rural area in Finland,in 1992-94. 1,2,3,7,8,9-Cl_6DF ans 1,2,3,4,7,8,9-Cl_7DF were below the detection limits. Other conditions as in Table 1.

congener	1.	1./(WHO)	2.	3.	4.	6.	13.
2,3,7,8-Cl_4DF	0.49 ± 0.44 <0.1 - 1.8	0.43 ± 0.44 <0.1 - 1.8	0.5 ± 0.33 <0.1 - 1.30	0.74 ± 0.42 0.25 - 1.81	0.67 ± 0.25 0.4 - 0.95	1.44	0.9
2,3,7,8-Cl_4DD	1.71 ± 0.68 0.4 - 3.03	1.7 ± 0.69 0.4 - 3.03	1.03 ± 0.33 0.24 - 1.5	1.61 ± 0.52 0.91 - 2.42	1.08 ± 0.2 0.87 - 1.34	0.88	0.99
1,2,3,7,8-Cl_5DF	0.33 ± 0.42 <0.1 - 1.39	0.33 ± 0.45 <0.1 - 1.4	0.15 ± 0.23 <0.1 - 0.92	0.23 ± 0.21 <0.1 - 0.54	0.2 ± 0.16 <0.1 - 0.38	0.3	0.2
2,3,4,7,8-Cl_5DF	10.1 ± 4.65 3.24 -25.7	9.51 ± 2.85 3.25 - 15.5	7.27 ± 2.74 1.23 - 12.6	9.32 ± 3.18 4.05 - 15.5	7.01 ± 1.4 5.03 - 8.2	5.05	3.66
1,2,3,7,8-Cl_5DD	4.36 ± 1.56 1.67 - 7.09	4.25 ± 1.31 1.67 - 6.35	3.0 ± 1.05 0.47 - 4.91	3.56 ± 1.05 1.31 - 5.52	2.94 ± 0.52 2.25 - 3.5	1.35	2.36
1,2,3,4,7,8-Cl_6DF	3.94 ± 1.85 0.79 - 7.42	4.12 ± 1.73 1.66 - 7.42	2.1 ± 1.0 <0.1 - 3.77	2.31 ± 1.07 0.48 - 4.29	2.16 ± 0.58 1.55 - 2.85	0.6	1.39
1,2,3,6,7,8-Cl_6DF	2.8 ± 1.15 0.8 - 6.27	2.78 ± 0.85 0.95 - 4.21	1.66 ± 0.77 <0.1 - 3.02	2.04 ± 0.89 0.61 - 3.9	1.47 ± 0.3 1.3 - 1.91	0.62	1.05
2,3,4,6,7,8-Cl_6DF	1.28 ± 0.92 <0.1 - 2.98	1.28 ± 0.9 <0.1 - 2.71	0.64 ± 0.41 <0.1 - 1.37	0.91 ± 0.46 0.29 - 1.88	0.74 ± 0.37 0.21 - 1.06	0.43	0.48
1,2,3,4,7,8-Cl_6DD	1.87 ± 0.95 <0.1 - 3.76	1.94 ± 0.94 <0.1 - 3.76	1.04 ± 0.65 <0.1 - 2.2	1.27 ± 0.54 <0.1 - 2.11	0.76 ± 0.66 <0.1 - 1.39	<0.1	0.72
1,2,3,6,7,8-Cl_6DD	26.9 ± 8.16 10.3 - 47.9	26.3 ± 5.49 17.5 - 39.4	23.4 ± 7.69 5.63- 37.6	26.7 ± 8.61 9.62 - 43.6	17.0 ± 2.46 13.4 ± 19.0	4.24	13.4
1,2,3,7,8,9-Cl_6DD	2.19 ± 1.63 <0.1 - 5.51	2.09 ± 1.45 <0.1 - 5.02	2.1 ± 1.5 <0.1 - 4.09	2.4 ± 2.24 <0.1 - 7.22	2.01 ± 1.07 0.48 - 2.9	<0.1	1.94
1,2,3,4,6,7,8-Cl_7DF	5.99 ± 4.09 1.3 ± 19.2	6.3 ± 4.25 2.52 - 19.2	4.19 ± 3.37 <0.1 - 11.5	5.64 ± 4.18 0.49 - 13.1	5.48 ± 1.33 4.25 - 7.28	1.32	7.29
1,2,3,4,6,7,8-Cl_7DD	28.3 ± 11.3 9.35 - 62.8	28.6 ± 12.0 9.35 - 62.8	22.6 ± 9.98 8.04 - 47.6	26.6 ± 9.87 10.0 - 40.9	27.6 ± 6.29 18.8 - 32.9	7.55	14.9
OCDF	<1 ± 1.47 <1 - 7.49	<1 ± 1.6 <1 - 7.49	<1	<1	<1 ± 1.31 <1 - 2.62	<1	<1
OCDD	126 ± 55.7 62.7 - 310	129 ± 58.3 62.7 - 310	114 ± 43.3 36.0 - 223	126 ± 61.9 43.5 - 259	120 ± 31.1 82.4 - 157	47.4	62.3
3 PCDD/F	217 ± 76.5 118 - 455	219 ± 77.3 122 - 455	184 ± 59.8 51.6 - 305	210 ± 84.4 72.5 - 390	191 ± 35.1 145 - 225	71.3	112
I-TEq	13.6 ± 4.57 6.06 - 26.0	13.0 ± 3.21 6.06 - 18.4	9.7 ± 3.06 1.77 - 14.1	12.1 ± 3.48 4.9 - 17.7	9.0 ± 1.33 7.03 - 9.81	4.97	6.28

Table 3a.
PCB concentrations and TEqs (mean ± standard deviation and range, ng/g fat) of non- and mono-*ortho* PCB congeners and of the di-*ortho* congeners which showed the highest concentrations of PCBs in the mother's milk from the urban area in Finland, in 1992-94. Asteriks indicate significant difference to the rural area (*p<0.01, **p<0.005, ***p<0.001). Other conditions as in Table 1.

congener	1.	1./(WHO)	2.	3.	4.
PCB 77[a]	47.4 ± 38.8***	44.1 ± 42.3***	32.3 ± 14.6	77.0	25.5
	<1 - 134	<1 - 134	20.9 - 48.9	73.1 - 81.0	
PCB 126[a]	89.7 ± 41.7***	90.1 ± 36.0***	50.1 ± 5.62	100	31.9
	29.8 - 173	47.4 - 155	45.7 - 56.4	86.5 - 113	
PCB 169[a]	38.5 ± 13.0***	40.6 ± 11.2***	24.4 ± 9.47	33.7	15.5
	13.1 - 58.5	24.3 - 58.5	14.0 - 32.5	32.6 - 34.9	
PCB 105[b]	3.75 ± 1.88	3.9 ± 1.68	1.78 ± 0.21	4.64	1.21
	0.86 - 6.9	1.94 - 5.9	1.58 - 2.0	3.51 - 5.78	
PCB 114[b]	0.61 ± 0.28	0.64 ± 0.24	0.38 ± 0.06	0.64	0.19
	0.17 - 1.09	0.36 - 0.98	0.32 - 0.43	0.48 - 0.79	
PCB 118[b]	17.7 ± 9.59	18.1 ± 8.81	9.91 ± 1.57	20.1	6.34
	4.74 - 34.9	8.27 - 32.2	8.14 - 11.2	14.9 - 25.3	
PCB 156[b]	7.83 ± 2.66	8.14 ± 1.99	5.21 ± 2.03	7.04	2.41
	2.35 - 12.6	5.23 - 11.0	2.93 - 6.8	6.65 - 7.43	
PCB 157[b]	1.32 ± 0.46**	1.41 ± 0.33***	0.76 ± 0.25	1.14	0.37
	0.38 - 2.08	0.9 - 1.86	0.48 - 0.91	1.02 - 1.27	
PCB 28	2.39 ± 1.77	2.58 ± 1.7	1.46 ± 2.53	0.36	1.99
	0.2 - 5.9	0.43 - 5.9	<0.001 - 4.38	<0.001- 0.71	
PCB 74	11.2 ± 5.67***	11.6 ± 4.32***	6.61 ± 1.84	15.0	2.57
	2.18 - 23.0	7.51 - 20.2	4.68 - 8.34	14.6 - 15.4	
PCB 99	11.1 ± 5.36***	12.0 ± 5.16***	6.06 ± 1.89	10.7	3.32
	2.54 - 22.9	5.95 - 22.9	4.7 - 8.21	8.06 - 13.4	
PCB 153	92.3 ± 33.7**	96.1 ± 27.0***	54.0 ± 19.8	77.0	34.0
	24.3 - 148	60.3 - 137	32.0 - 70.3	65.3 - 88.7	
PCB 138	56.8 ± 22.9***	59.7 ± 19.4***	32.6 ± 10.1	50.4	18.7
	14.2 ± 92.5	36.5 - 85.5	23.6 - 43.5	39.9 - 61.0	
PCB 180	39.2 ± 11.8	42.6 ± 8.0**	22.4 ± 10.7	29.2	14.5
	9.85 - 60.5	29.9 - 60.5	10.5 - 31.2	29.2 - 29.2	
PCB 170	19.5 ± 5.92	20.6 ± 4.33	12.5 ± 5.8	15.8	7.15
	5.31 - 30.0	14.8 - 30.0	6.27 - 17.8	15.7 - 16.0	
PCB 187	12.3 ± 4.25	13.4 ± 3.3**	5.8 ± 2.54	9.18	4.83
	2.46 - 19.9	8.84 - 19.9	2.88 - 7.48	8.64 - 9.72	
PCB 183	6.05 ± 2.28**	6.64 ± 1.85***	2.99 ± 1.17	4.41	2.23
	1.14 - 10.6	4.39 - 10.6	1.78 - 4.12	3.5 - 5.32	
PCB 194	3.49 ± 1.07*	3.5 ± 0.82**	2.71 ± 1.45	3.62	2.28
	1.16 - 5.3	2.31 - 4.69	1.19 - 4.08	3.35 - 3.89	
3 PCB	296 ± 108**	312 ± 85.3***	170 ± 48.6	275	106
	73.8 - 464	200 - 442	115 - 207	256 - 295	
TEq[c]	18.5 ± 7.48**	18.9 ± 6.13**	10.9 ± 2.33	18.9	6.34
	5.68 - 32.8	10.8 - 29.4	8.47 - 13.1	16.6 - 21.2	

[a]Non-*ortho*-substituted PCBs, given as pg/g fat
[b]Mono-*ortho*-substituted PCBs
[c] given as pg/g fat

Table 3b.
PCB concentrations and TEqs (mean + standard deviation and range, ng/g fat) of non- and mono-*ortho* PCB congeners and of the di-*ortho* congeners which showed the highest concentrations of PCBs in the mother's milk from the surroundings of the rural area from Finland, in 1992-94. Conditions as in Table 1 and 3a.

congener	1.	1./(WHO)	2.	3.	4.	6.	13.
PCB 77[a]	1.74 ± 2.41	1.79 ± 2.47	1.62 ± 3.33	3.87 ± 5.59	5.27 ± 2.77	<1	2.73
	<1 - 9.44	<1 - 9.44	<1 - 12.1	<1 - 17.6	2.15 - 8.68		
PCB 126[a]	42.9 ± 18.6	40.6 ± 16.1	29.6 ± 14.9	43.0 ± 15.4	38.0 ± 4.95	34.4	31.6
	10.5 - 87.4	10.5 - 70.8	<1 - 56.2	18.5 - 67.6	31.6 - 43.4		
PCB 169[a]	23.7 ± 18.5	22.0 ± 17.9	16.8 ± 8.48	19.7 ± 6.57	13.5 ± 4.03	11.2	8.48
	5.64 - 98.0	5.64 - 98.0	<1 - 37.9	8.01 - 29.4	8.16 - 17.1		
PCB 105[b]	2.35 ± 1.12	2.37 ± 1.1	1.51 ± 0.59	1.8 ± 0.7	1.48 ± 0.2	1.36	1.45
	0.47 - 5.27	0.47 - 5.27	0.45 - 2.61	1.06 - 3.34	1.25 - 1.72		
PCB 114[b]	0.5 ± 0.23	0.49 ± 0.2	0.28 ± 0.1	0.37 ± 0.11	0.24 ± 0.05	0.16	0.15
	0.14 - 1.08	0.14 - 0.93	0.05 - 0.47	0.16 - 0.59	0.18 - 0.31		
PCB 118[b]	11.1 ± 4.59	11.2 ± 4.49	6.88 ± 2.48	8.34 ± 2.76	6.87 ± 0.89	5.63	5.36
	3.14 - 22.3	3.14 - 22.3	1.25 - 10.4	4.0 - 13.3	6.04 - 7.88		
PCB 156[b]	6.46 ± 3.64	6.03 ± 3.0	5.65 ± 3.11	6.11 ± 3.42	3.87 ± 1.02	1.34	2.71
	1.81 - 17.8	1.81 - 13.8	1.08 - 13.0	1.88 - 13.9	2.85 - 5.26		
PCB 157[b]	0.84 ± 0.46	0.77 ± 0.31	0.67 ± 0.29	0.8 ± 0.35	0.51 ± 0.11	0.24	0.37
	0.25 - 2.57	0.25 - 1.43	0.13 - 1.11	0.26 - 1.54	0.43 - 0.68		
PCB 28	4.04 ± 4.1	4.23 ± 4.36	2.0 ± 1.91	2.2 ± 2.8	2.05 ± 0.5	<0.001	1.95
	0.21 - 23.1	0.25 - 23.1	<0.001 - 6.61	<0.001 - 8.2	1.53 - 2.7		
PCB 74	5.84 ± 2.24	5.82 ± 1.94	3.91 ± 1.44	4.22 ± 1.88	3.46 ± 0.41	1.54	1.79
	2.06 - 10.5	2.41 - 8.95	0.39 - 5.98	1.52 - 8.72	2.99 - 3.98		
PCB 99	5.62 ± 2.42	5.91 ± 2.34	3.99 ± 1.76	3.51 ± 1.01	3.02 ± 1.15	2.3	3.09
	1.84 - 10.8	2.19 - 10.8	0.72 - 8.08	1.87 - 5.42	1.99 - 4.65		
PCB 153	57.7 ± 24.9	55.3 ± 22.4	55.8 ± 26.9	49.5 ± 19.9	36.9 ± 9.68	15.9	26.6
	18.2 - 110	22.4 - 110	10.6 - 115	23.0 - 83.1	25.0 - 48.0		
PCB 138	32.4 ± 12.7	31.6 ± 12.2	32.0 ± 15.2	26.9 ± 10.2	23.0 ± 7.53	9.22	20.0
	9.04 ± 59.5	12.7 - 59.5	5.21 - 68.4	11.7 - 44.4	12.4 - 28.5		
PCB 180	31.9 ± 17.5	29.2 ± 15.2	38.8 ± 26.9	34.5 ± 12.6	21.5 ± 4.66	12.4	15.2
	10.8 - 78.5	10.8 - 68.2	9.18 - 116	19.1 - 63.3	17.7 - 28.1		
PCB 170	17.2 ± 9.93	15.9 ± 8.98	20.1 ± 13.8	18.1 ± 8.03	10.9 ± 2.15	5.42	8.12
	5.92 - 42.7	5.92 - 42.6	4.5 - 59.7	9.14 - 37.9	9.47 - 14.1		
PCB 187	9.26 ± 4.43	8.66 ± 3.79	9.26 ± 4.16	8.87 ± 2.32	5.61 ± 1.7	4.63	4.16
	2.85 - 21.0	2.85 - 17.0	3.39 - 22.3	7.01 - 13.5	3.86 - 7.75		
PCB 183	3.91 ± 1.7	3.77 ± 1.49	4.8 ± 2.15	3.8 ± 0.93	2.87 ± 0.5	2.02	2.83
	1.62 - 8.73	1.62 - 7.58	1.72 - 9.44	2.24 - 5.34	2.34 - 3.51		
PCB 194	2.52 ± 1.55	2.24 ± 1.3	3.44 ± 2.61	3.21 ± 1.1	2.01 ± 0.67	1.66	2.01
	0.78 - 7.04	0.78 - 5.83	0.66 - 10.5	2.15 - 5.76	1.48 - 2.9		
3 PCB	198 ± 80.8	190 ± 71.9	194 ± 94.5	177 ± 63.3	129 ± 25.8	66.6	100
	75.7 - 371	80.4 - 352	52.6 - 409	91.9 - 301	103 - 162		
TEq[c]	11.6 ± 5.03	11.0 ± 4.16	9.37 ± 3.77	11.1 ± 4.06	8.24 ± 1.27	5.72	6.41
	3.86 - 26.3	3.86 - 20.3	2.37 - 17.9	4.54 - 19.1	7.24 - 10.1		

[a]Non-*ortho*-substituted PCBs, given as pg/g fat.
[b]Mono-*ortho*-substituted PCBs
[c] given as pg/g fat

Geographical Aspects

When expressed as ∑PCDD/F, primiparae human milk concentrations were significantly higher in the urban area than in the rural area resulting from the fact that concentrations of the congeners which contribute mostly to ∑PCDD/F were statistically significantly higher in the urban area than in the rural area. The concentrations of the congeners which contribute mostly to I-TEqs were only moderately higher in urban area than rural area. Consequently, the PCDD/F I-TEqs concentrations were higher in the urban than in rural area in primiparae but statistically less significantly than ∑PCDD/F (Table 2). ∑PCB and TEq concentrations were significantly higher among urban than rural primiparae (see Table 3).

Correlation between PCDD/F and PCB concentrations

The correlations between I-TEq and ∑PCB concentrations among primiparae mother milk samples in the urban and rural area are shown in Fig. 1. The linear regression correlation coefficient (R) for the primiparae mother milk was 0.87 (0.91 in the urban and 0.76 in the rural area). R value for all milk samples was 0.82 (0.93 in the urban and 0.70 in the rural area).

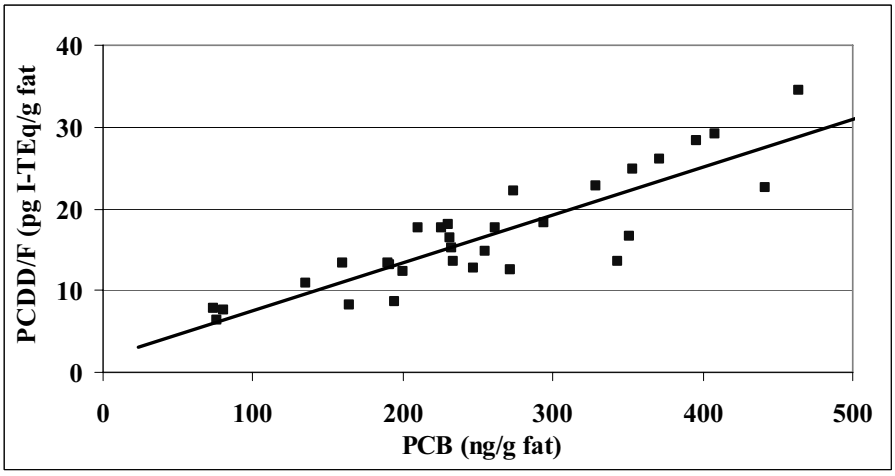

Fig. 1. Correlation between ΣPCB and PCDD/F (as I-TEq) concentrations in human milk. Primiparae of all milk samples included.

Time trends of PCDD/F and PCB concentrations

The concentrations of PCDD/Fs and PCBs (primiparae mothers) in this study are compared in Figure 2. with those found in 1987 in Finland [16]. Average decreases in

primiparae concentrations of PCDD/Fs and PCBs between 1987 and 1994 were 36 and 49%, respectively. Decreases of PCDD/Fs in urban and rural areas were 27 and 45%, respectively, and of PCBs 47 and 52%, respectively. The decreases of toxic equivalents of PCDD/F in urban and rural areas were 24 and 32%, respectively, and the corresponding values for PCB toxic equivalents were 50 and 56%, respectively. The changes of congeners 1,2,3,7,8-Cl_5DF, 1,2,3,4,7,8-Cl_6DF, OCDF and PCB 77 are excluded from the examination above due to low levels of congeners or analytical differences between years 1987 and 1994.

5. DISCUSSION

Ages of the mothers and milk fat contents did not differ statistically significantly from each other when comparing urban and rural primiparae mothers in this study. Furthermore, ages and milk fat contents in this study did not differ statistically from those in our previous study [16]. This makes it possible to compare the concentrations and time trend of concentrations of PCDD/Fs and PCBs in primiparae mother milk of urban and rural areas between 1987 and 1994.

The difference between urban and rural concentrations of PCDD/Fs and PCBs in primiparae mother milk remained when comparing current results with 1987 results. In fact, the differences in concentrations between areas have expanded. When the rural concentrations of 3PCDD/F, I-TEq, 3PCB and TEq were 90, 76, 80, and 73%, respectively, of the concentrations in urban area in 1987, the current percentages are 57, 68, 67 and 63, respectively.

Like in 1987, the correlation between I-TEq and 3PCB concentrations in 1994 was statistically significant (linear correlation coefficient $R = 0.82$; $p < 0.0001$) in the whole material and especially significant ($R = 0.93$; $p < 0.0001$) when examining the primiparae mothers in the urban area. These high correlation values give further confidence to the hypothesis that the sources of PCDD/Fs and PCBs are the same in Finland.

The well documented [1,2,16,17] decrease of PCDD/F and PCB concentrations in human milk with the increasing number of children was observed also in this study, though the concentrations of mothers having their third child exceeded the concentrations of mothers having their second child. This was most probably because of the low numbers of mothers nursing their third child, in this study. The lowest PCDD/F and PCB levels were found in Eastern Finland in milk of the mother who was breast-feeding her sixth child, 4.97 pg I-TEq/g fat and 5.72 pg TEq/g fat.

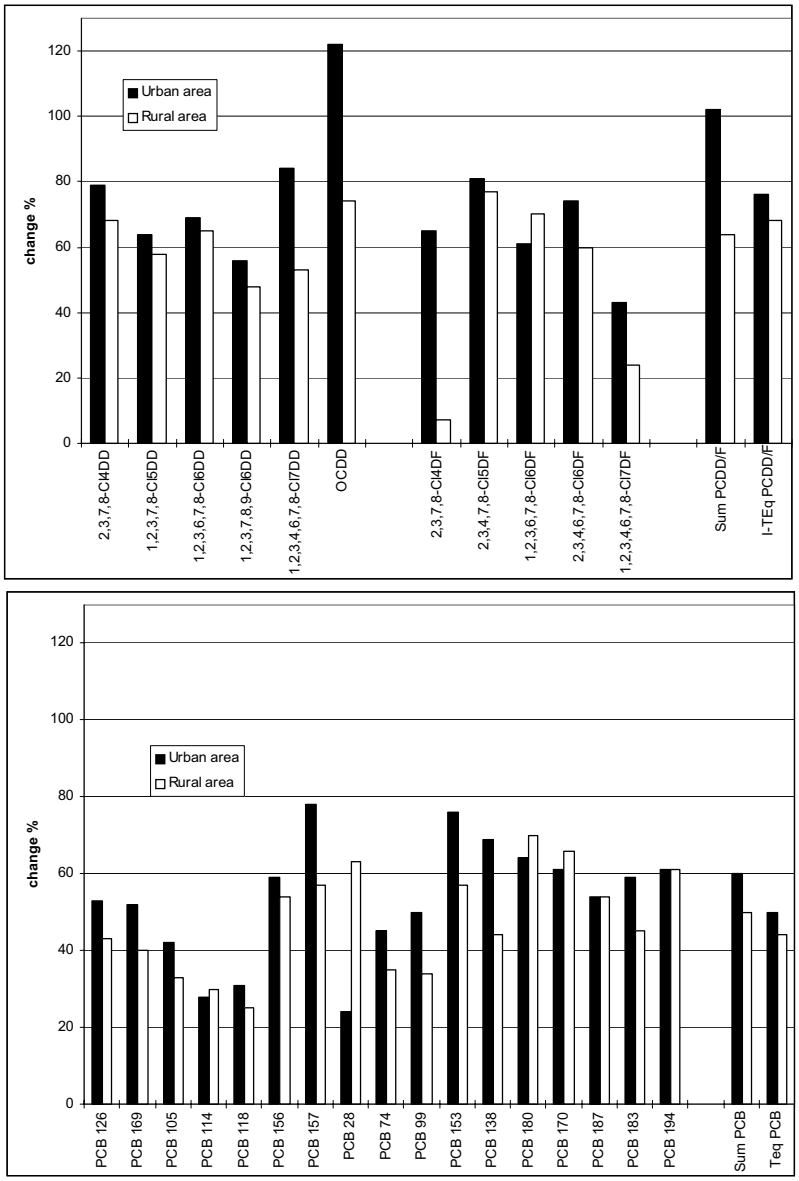

Fig. 2. PCDD/F and PCB concentrations for Finnish primiparae human milk samples in 1994 in percents when compared to 1987 concentrations.

WHO/EURO analyzed two pooled Finnish human milk samples in the second round of WHO-coordinated exposure study [11]. Milk samples were the same as in this study marked as 1./(WHO). Pooling for WHO study was performed by us on volume basis. The average I-TEqs analyzed in this study (20.4 pg/g fat for urban area and 13.0 pg/g fat for rural area) are in good agreement with those values measured in the WHO study (21.5 pg/g fat for urban area and 12.0 pg/g fat for rural area) [11]. Also the sums of marker PCBs (IUPAC 28, 52, 101, 138, 153 and 180) as ng/g fat in this study (192 and 127, for urban and rural area, respectively) are similar to those values measured in the WHO/EURO study (189 and 134, for urban and rural area,

respectively). However the concentrations of non-*ortho* PCBs in this study differ considerably from those measured from the pooled samples in the second round of WHO-coordinated exposure study [11].

Primiparae mother milk I-TEq values of Helsinki (19.9 pg I-TEq/g fat) were similar to those measured in Europe (Belgium, Germany, The Netherlands and Spain), while values of Kuopio area (13.6 pg I-TEq/g fat) were similar to values measured in Norway, Austria and eastern parts of Europe.

The decrease of concentrations of PCDD/Fs between 1986 and 1993 in human blood and milk has been reported from Germany and the Netherlands [8,9]. There is a specific uncertainty about the declining of concentrations of PCBs in human milk in the Central Europe [9] though the duplicate diet study showed a significant decline in the dietary exposure to PCBs in the period 1978-1994 [18]. In this study, the decrease of PCDD/Fs and PCBs in primiparae mothers= milk was found to be 36% and 49%, respectively, when compared to the concentrations of the primiparae mothers= milk in 1987 [16]. The declining of PCDD/Fs and PCBs seems to be greater in rural (45 and 52%, respectively) than in urban (27 and 47%, respectively) area. In the WHO/EURO study there was a value 2.2% reported for the annual percentual decrease of dioxin levels (in pg I-TEq/g fat) in Finland, in between 1988-1993 [11]. That annual decrease was based on the results of two different laboratories [10,11]. The estimation of 5-6% for annual declining of PCDD/Fs in this study is based on the results of the same laboratory with similar methods. The differences between annual decrease can be explained with the differences of analytical methods. In Europe the declining of PCDD/Fs and PCBs in humans have been connected to measures taken to reduce PCDD/F and PCB emissions from industry [8,9]. There is not data available for the time trends of PCDD/Fs and PCBs in Finnish food between 1987-1994 and therefore the cause of the possible decrease of concentrations of PCDD/Fs and PCBs in human milk is unclear. In Finland, only one small municipal incinerator is functioning in comparison with the hundreds in Central Europe, but the prevailing winds may also carry their emissions towards Finland. Since 40% of the total SO_2 deposit in Finland comes via the air from Central Europe, it may be assumed that also a major proportion of the total PCDD/F load is carried by the wind from other parts of Europe. This proportion most probably has decreased.

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CHAPTER 7

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

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1. ABSTRACT

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.

2. INTRODUCTION

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 analysed 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.

3. 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 southeastern 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 the approved the design of the study.

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 analysed 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 analysis

We performed statiatical analysis 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 χ^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 to 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 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.

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	
	All subjects	exposed fishermen	other fishermen	coast	Kuusankoski
Characteristics	n = 47	n = 26	n = 21	n = 25	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)
Time at present residence (years)	45, 50 (4-77)	43, 51 (4-77)	47, 47 (9-73)	47, 50 (6-73)	42, 49 (4-77)

4. 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 χ^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).

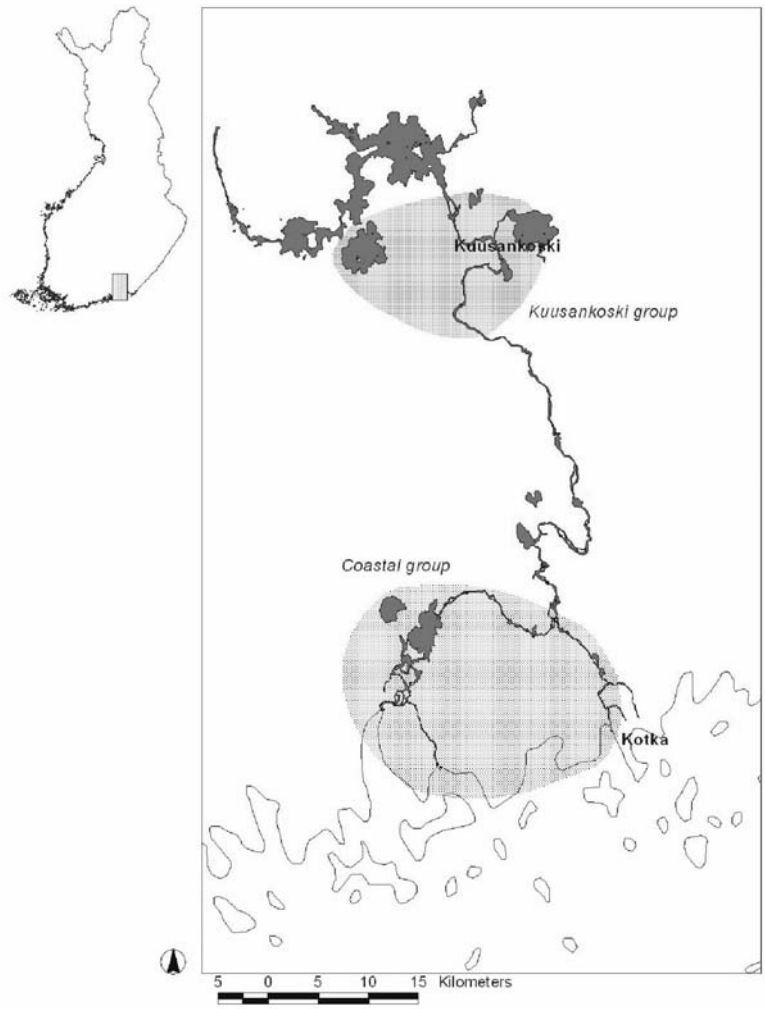

Fig 1. Study area showing fishermen subgroups according to place of residence.

Table 2 summarizes the ranked results of the two most favoured 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 favoured 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]; 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 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 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.

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	
	All subjects	Exposed fishermen	Other fishermen	Coast	Kuusankoski
Congener	n = 47	n = 26	n = 21	n = 25	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; WHOPCDD/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).

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.

		Fish consumption frequency		Place of residence	
Congener IUPAC no.	All subjects n = 47	Exposed fishermen n = 26	Other fishermen n = 21	Coast n = 25	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).

5. 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 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 lakes "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

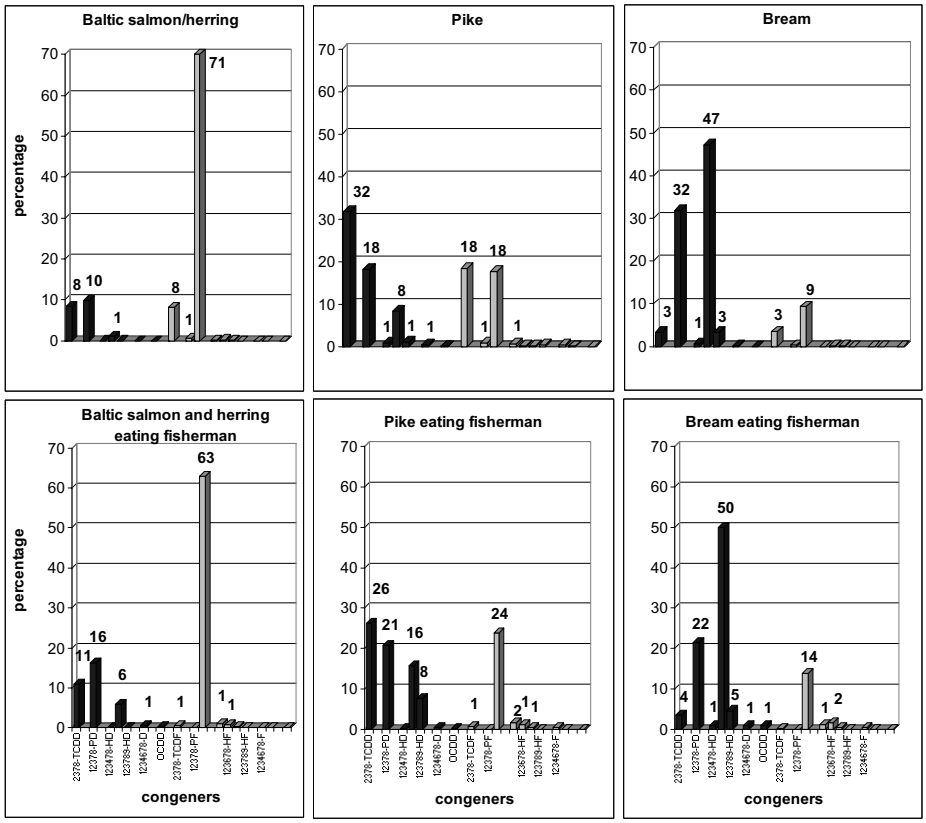

*Fig 2.*Congener I-TEq profiles of individual fishermen and profiles of fish species that each fisherman reported he prefers to consume. Congeners: 1: 2,3,7,8-TCDD; 2: 1,2,3,7,8-PeCDD; 3: 1,2,3,4,7,8-HxCDD; 4: 1,2,3,6,7,8-HxCDD; 5: 1,2,3,7,8,9-HxCDD; 6: 1,2,3,4,6,7,8-HpCDD; 7: OCDD; 8: 2,3,7,8-TCDF; 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.

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 of the fishermen in the Kuusankoski group fish from a lake famous for its bream catches. Examination of 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 I-TEq/g 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 $WHO_{PCDD/F}$-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.

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 $WHO_{PCDD/F}$-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			

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CHAPTER 8

GENERAL DISCUSSION

1. GENERAL ADULT INTAKE OF PCDD/Fs AND PCBs

On average, the Finnish adult daily intake of PCDD/Fs was 60 pg or 0.79 pg WHO-TEq/kg bw, being very similar to other European intakes. The calculation methods selected had a major impact in assessments of the intake of PCDD/Fs. If we chose to use SIFF method and lower bound concentrations of congeners in foodstuffs and I-TEFs for the calculations of TEqs, then the daily intake was 46 pg I-TEq or 0.61 pg I-TEq/kg bw. The use MBM-method and upper bound concentrations and $WHO_{PCDD/F}$-TEFs resulted in intake assessments of 75 pg $WHO_{PCDD/F}$-TEq or 0.99 pg $WHO_{PCDD/F}$-TEq/kg bw. When using upper bound concentrations of PCDD/Fs in foodstuffs and the same set of TEFs, then the PCDD/F intake would increase by 22-29%, when compared to the use of lower bound concentrations.

With respect to PCB TEqs, the difference between lower and upper bound intake assessments was less than 2%, irrespective of the TEF set used. The intake of PCBs was similar to PCDD/F intake, being on average 0.74 pg TEq/kg bw/day. This was somewhat less than has been assessed elsewhere in Europe.

Total adult exposure in Finland to PCDD/Fs and PCBs via the diet was hence assessed to be on average 116 pg/day or 1.5 pg TEq/kg bw/day, for a person weighing 76 kg. This intake as well as the maximum, upper bound, daily intake of PCDD/Fs and PCBs (1.8 pg WHO-TEq/kg bw/day), was below the EU SCF TDI of 2 pg WHO-TEq/kg bw/day (European Commission 2001), though the margin was quite narrow, and it can be assumed that a considerable portion of the Finnish population exceed this TDI. It was not possible to assess the proportion of the Finnish population exceeding EU SCF's TDI in this study since that would require data on individual based intake assessments including all age-classes.

Finland is one of those countries where the consumption of fish is high compared to many other European countries (Welch et al. 2002), about 15 kg/person/year (http://www.rktl.fi/www/uploads/pdf/taskutilasto2004.pdf). Nevertheless, high consumption of fish together with the fact that the origin of a considerable part of the fish originate from the Baltic Sea which is contaminated with PCDD/Fs and PCBs explain the high contribution of fish and fish products to the intake of PCDD/Fs and PCBs. Depending on the calculation method (lower- or upper bound) the contribution of fish accounted for 60% to 95% of the PCDD/F

intake. The contribution of Baltic herring alone to the intake was 50%. The lower or upper bound contributions from milk products were 1% to 15%, respectively, and the contribution of meat and eggs was from 2% to 17%, respectively. Fish also contributed the main part to the PCB intake, accounting for 80%.

A similar decrease in PCDD/F intake, as in other countries worldwide, could be detected when comparing Finnish intake assessments from the beginning of 1990s to the assessments at the end of 1990s. Hallikainen and Vartiainen (1997) reported the daily intake of PCDD/Fs to be 95 pg N-TEq which was comparable to lower bound I-TEq estimate of 46 pg I-TEq/day obtained with the SSIF-method in this study. Hence, we detected an annual decrease of 6%, which was close to other reported decreases in the intake of PCDD/Fs (see CHAPTER 1, Fig. 2). The decline of dietary intakes of PCDD/Fs and PCBs in Finland was probably affected by two equally important reasons. First the concentrations in foodstuffs, especially in cow's milk and eggs, had been declining. Secondly, the consumption habits of Finnish people have been changed to favour less fatty products, especially with regards to milk, cheese, and meat products. This changing of diets to consumption of less fatty foods has continued since the above studies (Helakorpi et al. 2004). In the future, dietary habit changes may be more crucial in reducing the PCDD/F and PCB intakes rather than changes in concentrations of PCDD/Fs and PCBs actually present in the foodstuffs. A decrease in the concentrations of PCDD/Fs and PCBs in fish products would be the most effective way to diminish Finnish exposure to these contaminants. However, the concentrations in Baltic herring, which can be used as a proxy to domestic wild fish, have not been decreasing during the last decade, according to this study and a more recent report (Hallikainen et al. 2004, Isosaari et al. 2005). The control measures taken by the EU Commission to decrease the PCDD/F and PCB concentrations in feedstuffs (EC 2002) will probably decrease the concentrations in cultivated fish and eggs, but a similar dramatic decrease as occured during the 1990s cannot be anticipated.

Since fish are the main source of PCDD/Fs and PCBs in Finland one way to reduce the exposure to these contaminants is to advice people to avoid eating fish. However this is not recommended in Finland. Instead there is a recommendation given by the National Nutrition Council and the National Food Agency (http://www.elintarvikevirasto.fi/english/ -> Press releases 2004) to consume fish at least twice a week but alternating the fish species in the diet (Annex 1). There are specific recommendations to eat large Baltic herring or wild salmon only once or twice a month in order to avoid excessive exposure to PCDD/Fs and PCBs. This also applies to consumption of pike in order to avoid exposure to mercury. In their global assessment

of organic contaminants in farmed salmon, Hites et al. (2004) and later Foran et al. (2005) concluded that in order to diminish the risk of cancer, farmed salmon should not be eaten more than once per month. This risk assessment by Hites et al. was challenged by Tuomisto et al. (2004) who conducted a risk-benefit analysis for eating farmed salmon. It was concluded that by following the recommendations of Hites et al., the number of cancer deaths would decrease by 40 cases per year, but the number of cardiac deaths would increase by over 5000 cases per year in Europe. This would be due to a decrease in the intakes of omega-3 fatty acids. In addition to healthy fatty acids, fish contain several vitamins, minerals, and are also a protein-rich food source. Fish are a particularly good source of vitamin D for the elderly population in Finland. Instead of losing the beneficial health effects by not using fish (because it is contaminated) we should further diminish the European and global releases of PCDD/Fs and PCBs in order to lower the concentrations of these contaminants in the marine environment. One way to decrease the concentrations of PCDD/Fs and PCBs in the Baltic fish for human consumption would be more efficient exploitation of herring stocks and a transition to utilizing smaller herring in the manufacture of prepared fish dishes.

2. ADIPOSE TISSUE CONCENTRATIONS OF PCDD/Fs AND PCBs IN THE GENERAL POPULATION

Since the concentrations of PCDD/Fs and PCBs in this study correlated with the age of the subject, an adjustment according to age had to be done before any comparisons between countries, studies or time periods could be made. Also the sample collection period should be the same, due to the decreases occurring in exposure to and the adipose tissue concentrations of PCDD/Fs and PCBs. When taking the age and sample collection period into account, the concentrations of PCDD/Fs and PCBs in Finland were very similar to concentrations reported in other European countries, as can be seen from figures 1 and 2 depicting TEq concentrations in six, and PCB 153 concentrations in four European countries, respectively.

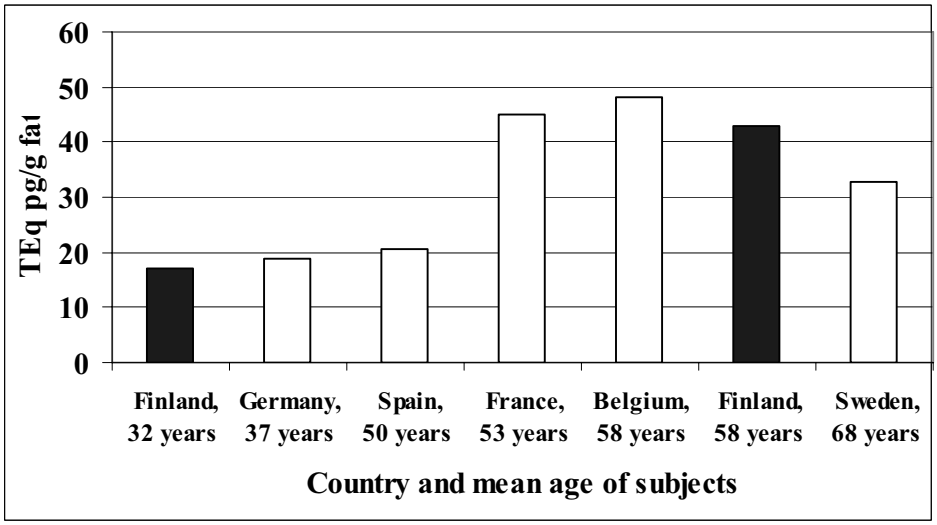

Fig 1. Mean TEq concentrations in six European countries at the end of 1990s and at the beginning of 2000 (mean age of studied population). Data from: CHAPTER 5 (Table 4), Päpke 1998, Wingfors et al. 2000, Bocio et al. 2004, Arfi et al. 2001, Koppen et al. 2002.

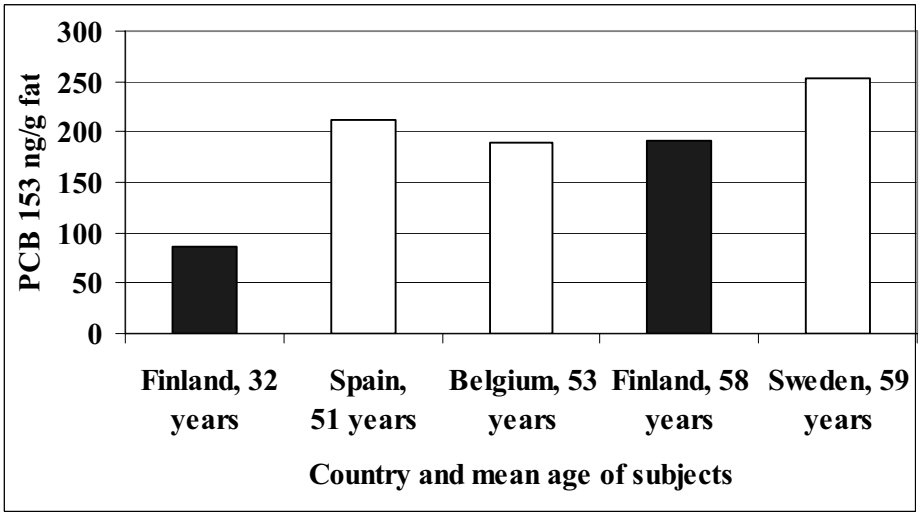

Fig 2. Mean PCB-congener PCB 153 concentrations in different countries in Europe at the end of 1990s and at the beginning of 2000 countries (mean age of studied population). Data from: CHAPTER 5 (Table 5), Costabeber and Emanuelli 2003, Wingfors et al. 2000, Koppen et al. 2002, Covaci et al. 2002, Wicklund Glynn et al. 2000, Wicklund Glynn et al. 2003, Wallin et al. 2003.

A decrease in the concentrations of PCDD/Fs and PCBs from coastal to inland areas, similar to breast milk samples in 1994, was detected in adipose tissue samples of the general population in Finland. With the older population, the decrease was more pronounced towards inland areas when compared with the younger population. This was consistent with the results from breast milk samples in 2000 (Leeuwen and Malisch 2002) failing to confirm any differences in concentrations between Helsinki area and Kuopio. Also the food frequency questionnaire from the general population revealed that younger population's fish consumption around Finland was already quite uniform with respect to frequency of fish and fish species consumed. Among the older population, Baltic herring (as well as other Baltic fish species) was consumed more in coastal areas when compared to inland areas, and this is the explaination for the more pronounced decline in adipose tissue concentrations between areas.

It was not possible to determine the annual decrease in the exposure of Finnish population to PCDD/Fs and PCBs from the adipose tissue data available. Nevertheless, at the population level the PCDD/F concentrations did not follow the upward convex curve, reaching steady state at about the age of 40 years, which would be the case if the exposure had been stable. This indicates that the exposure has decreased during recent decades.

3. PCDD/Fs AND PCBs IN BREAST MILK

The estimated annual declines in PCDD/F and PCB TEqs in Finnish breast milk samples during the time period between 1987 and 1994 were 4% and 8%, respectively. This was in line with the reported decreases in breast milk samples worldwide and was also comparable to the annual decrease in PCDD/F intake (6%) in Finland reported in the present study. The paper of the third round of WHO breast milk studies described the most recent analysis of pooled breast milk concentrations from the year 2000 (Leeuwen and Malisch 2002). The average Finnish $WHO_{PCDD/F}$-TEq concentration in breast milk was 9.4 pg/g fat which is 85% of the average concentration in Western Europe. The concentration of WHO_{PCB}-TEq, 5.9 pg/g fat, was at the lower end of WHO_{PCB}-TEq concentrations in Western Europe, representing 57% of the average Western European concentrations. The time-trend between 1987 and 2000 in Finland (see Fig 3) suggested an annual decrease of 5% and 6% of $WHO_{PCDD/F}$-TEq and WHO_{PCB}-TEq, respectively.

A decrease in breast milk PCDD/F and PCB concentrations from the capital area to inland (Kuopio) area was found in the 1994 study confirming an earlier study in 1987 (Vartiainen et al. 1997). By the year 2000, this regional difference no longer existed. One

explanation might be that in 1987 and still at the beginning of 1990s, the fish consumed in capital area included more Baltic herring or other Baltic fish than fish consumed in the Kuopio area, resulting in higher concentrations of PCDD/Fs and PCBs in breast milk of mothers in the capital area. Since the mid 1990s, the fish consumption habits in the two areas have converged. This change depicts the increasing consumption of cultivated salmon imported from Norway (in 1992 the imported amount of Norwegian cultivated salmon was 0.1 million kg, while in 2000 it was already 6.6 million kg (Finnish custom statistics 2004, received by phone from the Finnish Game and Fisheries Research Institute)). It is assumed that young women prefer to use cultivated salmon fillet or frozen ready fish meals instead of Baltic herring or other fish species.

For the year 2000 the intakes of exclusively breast feeding infants were calculated to be 53 and 33 pg/kg bw/day of $WHO_{PCDD/F}$-TEq and WHO_{PCB}-TEq, respectively, presuming an infant weight of 5 kg and a daily consumption of 800 ml of breast milk with 3.5% fat. Thus the total exposure of an infant would exceed the TDI proposed by EU SCF, by a factor of 40. This is at the lower end when compared to the corresponding values in other countries. Since the TDI suggestion is based on lifelong exposure, it is probable that this relatively short period of high exposure to PCDD/Fs and should not cause hazardous health effects to infants. Instead, because of the many beneficial health effects of breast milk, breast feeding has been encouraged by the WHO (WHO 2000).

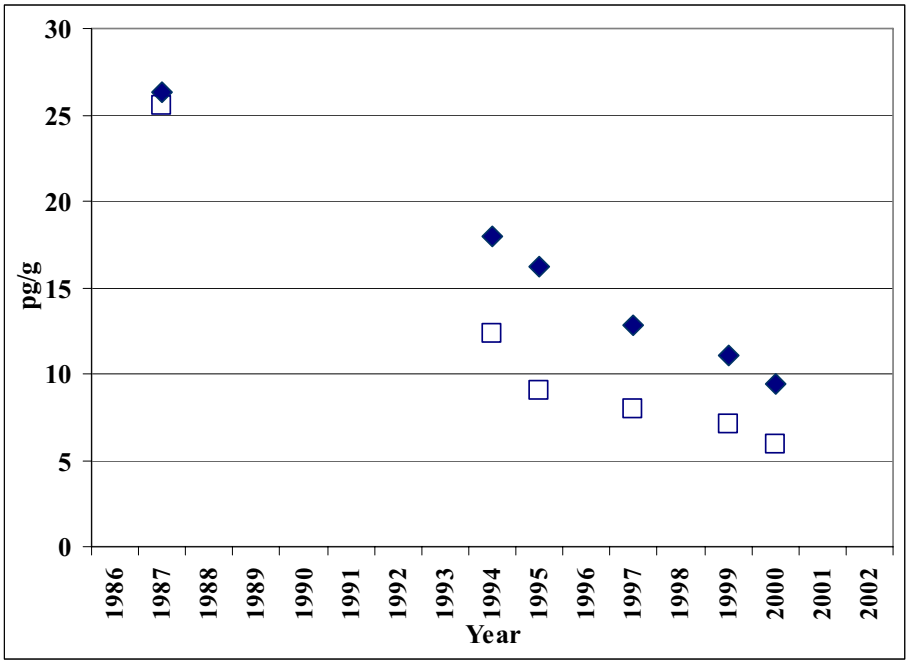

Fig 3. Time-trend of $WHO_{PCDD/F}$-TEqs (diamonds) and WHO_{PCB}-TEqs (open squares) as pg/g fat in breast milk in Finland between 1987 and 2000. Results from 1987 and 1994 from this study, results from 1995 are unpublished data, results from 1997 partly published by Hölttä et al. (2001), results from 1999 published by Alaluusua et al. (2002), and results from 2000 published by Leeuwen and Malisch (2002).

4. PCDD/Fs AND PCBs IN A SAMPLE OF FISHERMEN

A pilot study of 47 professional fishermen, population anticipated to be highly exposed to these environmental pollutants, was conducted, because the previous intake studies of PCDD/Fs and PCBs had indicated that fish are the major source of these agents in Finland. Of the studied fishermen 55% consumed fish at least once a week, compared to 40% of Finnish males in general. The fishermen preferred to consume wild fish more often than the general population. Fishermen's serum fat concentrations of PCDD/Fs and PCBs were 2 to 4 times higher than those of the general population men of the same age (Fig. 4). The concentrations were higher in the Baltic Sea fishermen compared to inland lake fishermen. This was attributable to the fact that they ate more Baltic wild fish, which are more contaminated with PCDD/Fs and PCBs in comparison with inland lake fish (Hallikainen et al. 2004). High exposures of fishermen

populations have been reported from Sweden, with respect to Swedish Baltic Sea fishermen, and from North America, with respect to sport anglers fishing in the Great Lakes (Svensson et al. 1991, Svensson et al. 1995, Sjödin et al. 2000, Cole et al. 1997, Anderson et al. 1998, He et al. 2001).

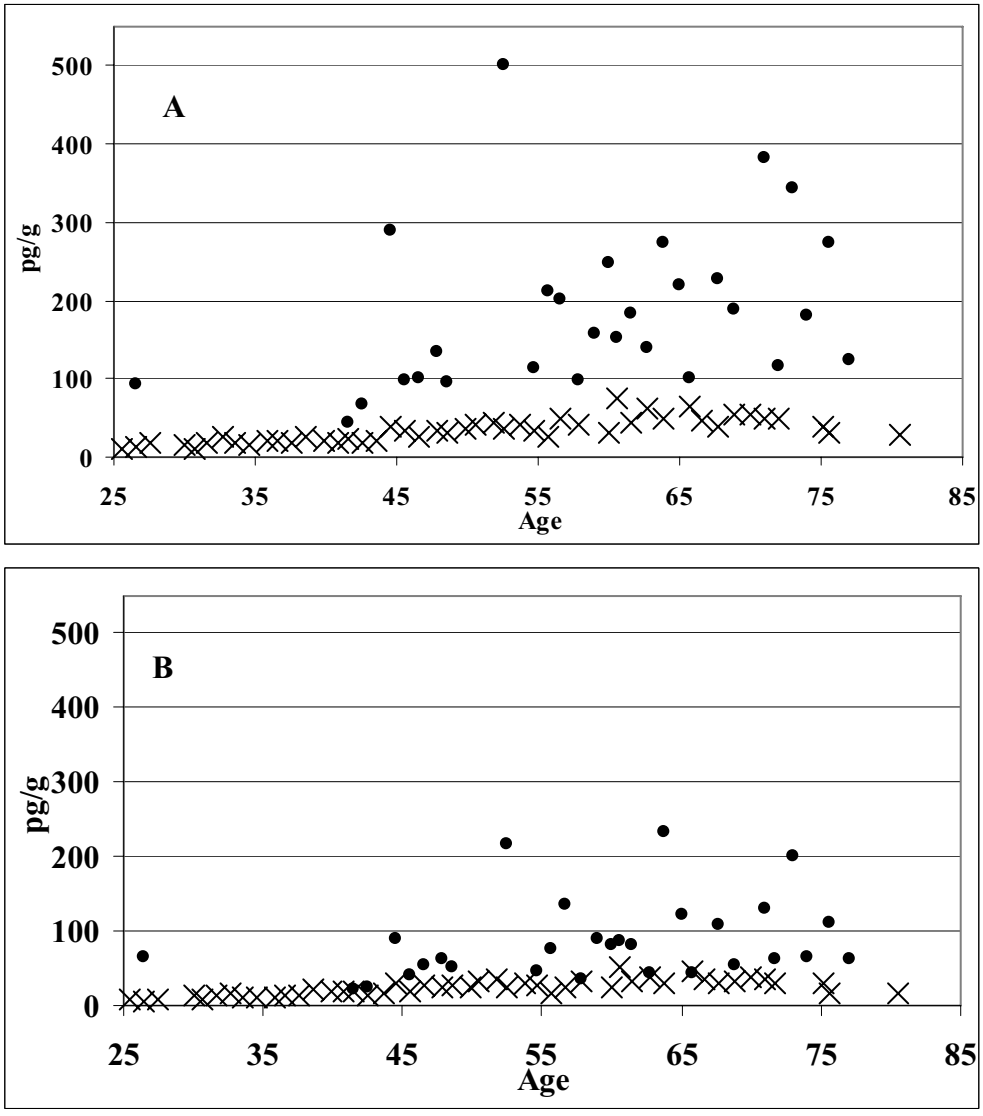

Fig 4. Median year class (CHAPTERS 5 and 7) concentrations of A: $WHO_{PCDD/F}$-TEq, and B: WHO_{PCB}-TEq in fishermen (dots) and in general population men of the same age in Finland (crosses).

5. CONGENER OCCURRENCE AND ACCUMULATION

Figures 5 to 8 depict the congener profiles of PCDD/Fs and PCBs as percentages of the total concentrations and as percentages of the WHO-TEqs in market baskets representing the exposure of a general population in Finland. In addition, profiles of PCDD/F and PCB in deposition between 1997 and 2004 are illustrated in figures 5 to 8 (unpublished data from joint research between The Finnish Environment Institute and KTL). Deposition of PCDD/Fs and PCBs will contribute to the exposure of population directly via non-animal origin foodstuffs, cereals, vegetables, fruits and berries etc. in which the profiles of PCDD/Fs and PCBs were quite similar to corresponding deposition profiles. Higher chlorinated PCDD/Fs dominated the concentration profiles, while in PCBs the lower chlorinated congeners expressed relatively high contribution to the profile. Different bioaccumulation properties of PCDD/F and PCB congeners in food chains resulted in differences in congener profiles in animal origin foodstuffs when compared to non-animal origin foodstuffs. Tetra and penta chlorinated PCDD/Fs expressed higher accumulation efficiency than the hepta and octa chlorinated congeners. Especially in many fish species these lower chlorinated PCDD/Fs: 2,3,4,7,8-PeCDF, 2,3,7,8-TCDF, 1,2,3,7,8-PeCDD, and 2,3,7,8-TCDD bioaccumulate efficiently. The total diet $WHO_{PCDD/F}$-TEq profile resembled very much the corresponding profile in Baltic herring, salmon, and rainbow trout. The contribution of the fish basket to the total basket was the largest due to the high concentrations of contaminants in fish. OCDD dominated the concentration based profile due to OCDD load from non-animal origin foodstuffs and also because the occurrence of OCDD in pork meat, poultry, and eggs was abundant. With PCBs, due to their different bioaccumulation potencies, the congener abundance pattern changed from lower chlorinated congeners towards higher chlorinated compounds. The most abundant congeners in the total diet were PCB 153, 138, 118, and 180.

Comparison of the average Finnish exposure profiles of PCDD/Fs with the corresponding profiles in adipose tissue or serum samples in different subgroups of Finnish population (Fig 9 and 10) revealed that dioxins bioaccumulate more efficiently from the food into humans than furans do. Especially the higher chlorinated dioxin congeners (hexa to octa substituted) made a higher contribution to the PCDD/F profile in human samples than in the total diet. On the contrary, the contribution of all ten furans diminished when moving from the total diet to human samples. The stronger contributions of dioxin than furan congeners in PCDD/F profiles as one moved from the total diet to the general population was in accordance with the reported half-lives of PCDD/Fs in humans (Flesch-Janys et al. 1996, Liem and Theelen 1997). In almost all chlorination patterns, dioxin congeners have been reported to have longer half-lives than the corresponding furan

congeners. One exception does exist, the half-life of 2,3,4,7,8-PeCDF have been reported to be 9.9 or 19.6 years by Liem and Theelen (1997) and Flesch-Janys et al. (1996), respectively, while the corresponding half-life of dioxin congener 1,2,3,7,8-PeCDD were 8.6 or 15.7 years, respectively. On the basis of half-life of 2,3,4,7,8-PeCDF, one would have expected it to make a considerable addition to the contribution of this congener in the profile in general population.

The contributions of the different PCDD/F congeners in profiles in young women (breast milk samples) and general population (adipose tissue samples) were very similar. Only with congener 2,3,4,7,8-PeCDF was the contribution slightly lower within young women. This might be due to their lower consumption of fish as compared to the general population. It was expected that the contribution of 2,3,4,7,8-PeCDF would be the highest among fishermen, but this proved not to be the case (Fig 9 and 10). It transpired that our sample of fishermen was not optimal for the purpose of comparing average contributions of different PCDD/F congeners between fishermen and other population subgroups. There were only 46 fishermen in the study (CHAPTER 7) and half of them were inland lake fishermen, mostly fishing from the same lake. By chance, a relatively large proportion of the inland lake fishermen preferred bream over other fish species and therefore congeners contributing the most in bream, 1,2,3,7,8-PeCDD and 1,2,3,6,7,8-HxCDD, also contributed relatively strongly in average fishermen profile (Fig 9 and 10). The crucial effect of fish species mainly consumed on congener profiles in an individual was reported in CHAPTER 7. Some of the individual fishermen in the study reported that they consumed exclusively or at least mostly one species of fish. The congener profiles of those individual fishermen were very similar to the profiles measured in the fish in the corresponding areas. We have further, still unpublished, data from professional Baltic Sea fishermen, and in that data the contribution of 2,3,4,7,8-PeCDF exceeds the corresponding contribution in other population subgroups.

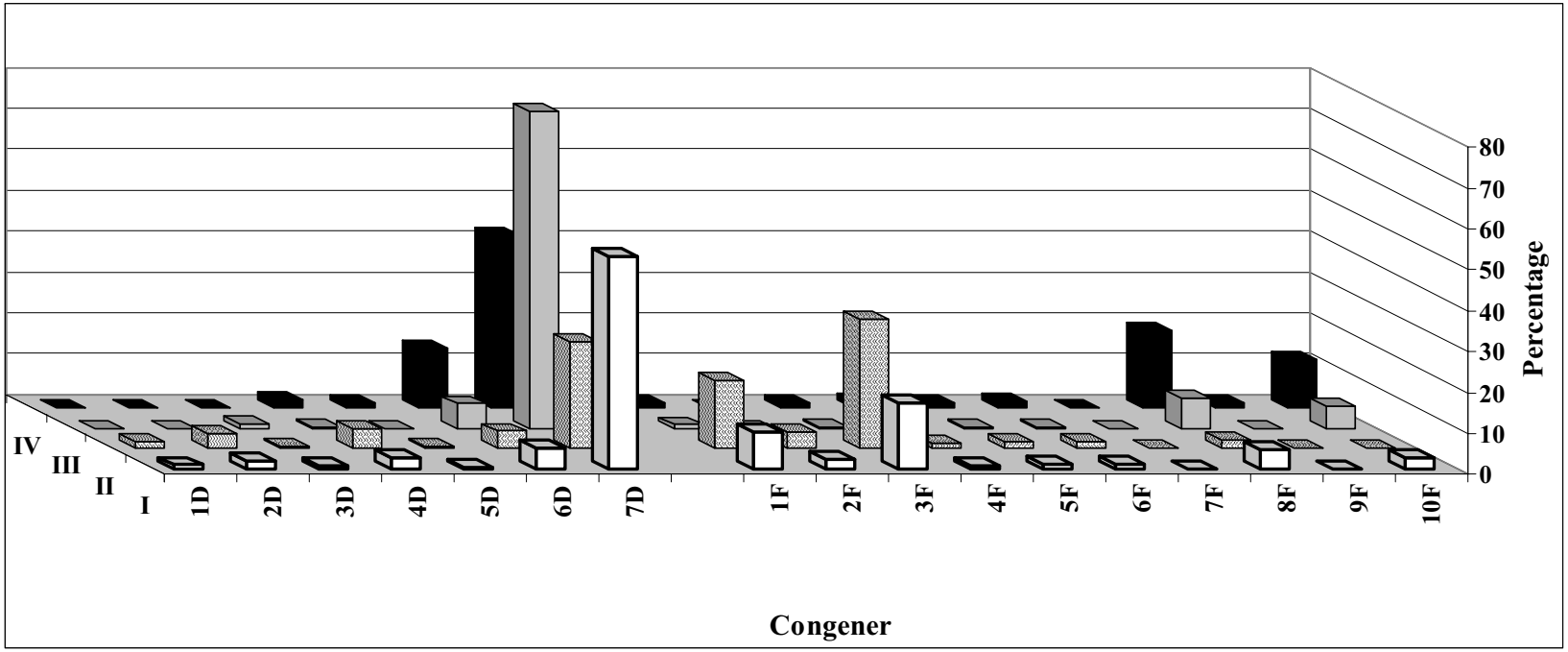

Fig 5. Congener profiles of PCDD/Fs as concentration basis in deposition and in Finnish diet. Profiles: I=Total diet, II=Animal origin market baskets, III=Non-animal origin market baskets, IV=Average deposition profile in Finland during 1997-2004. Congeners: 1D: 2,3,7,8-TCDD; 2D: 1,2,3,7,8-PeCDD; 3D: 1,2,3,4,7,8-HxCDD; 4D: 1,2,3,6,7,8-HxCDD; 5D: 1,2,3,7,8,9-HxCDD; 6D: 1,2,3,4,6,7,8-HpCDD; 7D: OCDD; 1F: 2,3,7,8-TCDF; 2F: 1,2,3,7,8-PeCDF; 3F: 2,3,4,7,8-PeCDF; 4F: 1,2,3,4,7,8-HxCDF; 5F: 1,2,3,6,7,8-HxCDF; 6F: 2,3,4,6,7,8-HxCDF; 7F: 1,2,3,7,8,9-HxCDF; 8F: 1,2,3,4,6,7,8-HpCDF; 9F: 1,2,3,4,7,8,9-HpCDF; 10F: OCDF. (Market basket data from CHAPTER 3, deposition data from joint research between The Finnish Environment Institute and KTL).

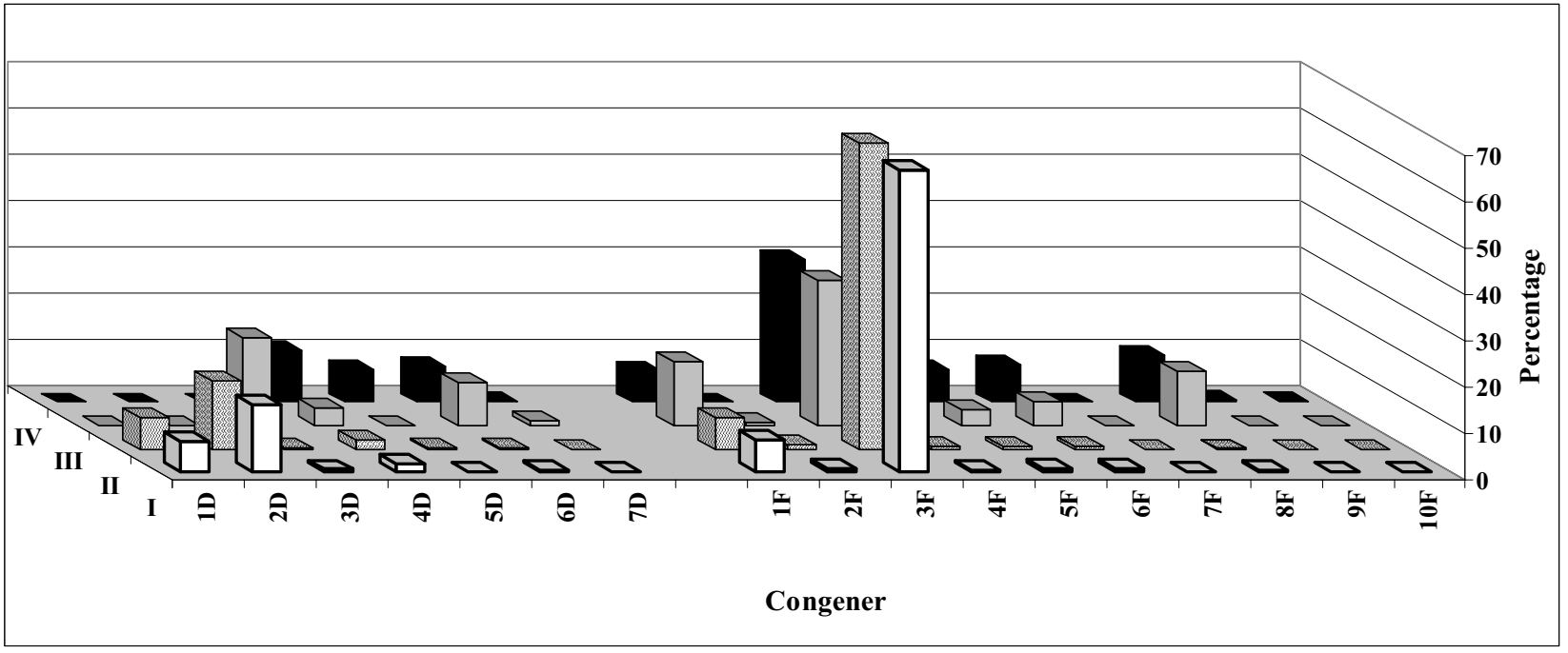

Fig 6. Congener profiles of PCDD/Fs as $WHO_{PCDD/F}$-TEq basis in deposition and in Finnish diet.. Profiles: I=Total diet, II=Animal origin market baskets, III=Non-animal origin market baskets, IV=Average deposition profile in Finland during 1997-2004. Congeners: 1D: 2,3,7,8-TCDD; 2D: 1,2,3,7,8-PeCDD; 3D: 1,2,3,4,7,8-HxCDD; 4D: 1,2,3,6,7,8-HxCDD; 5D: 1,2,3,7,8,9-HxCDD; 6D: 1,2,3,4,6,7,8-HpCDD; 7D: OCDD; 1F: 2,3,7,8-TCDF; 2F: 1,2,3,7,8-PeCDF; 3F: 2,3,4,7,8-PeCDF; 4F: 1,2,3,4,7,8-HxCDF; 5F: 1,2,3,6,7,8-HxCDF; 6F: 2,3,4,6,7,8-HxCDF; 7F: 1,2,3,7,8,9-HxCDF; 8F: 1,2,3,4,6,7,8-HpCDF; 9F: 1,2,3,4,7,8,9-HpCDF; 10F: OCDF. (Market basket data from CHAPTER 3, deposition data from joint research between The Finnish Environment Institute and KTL).

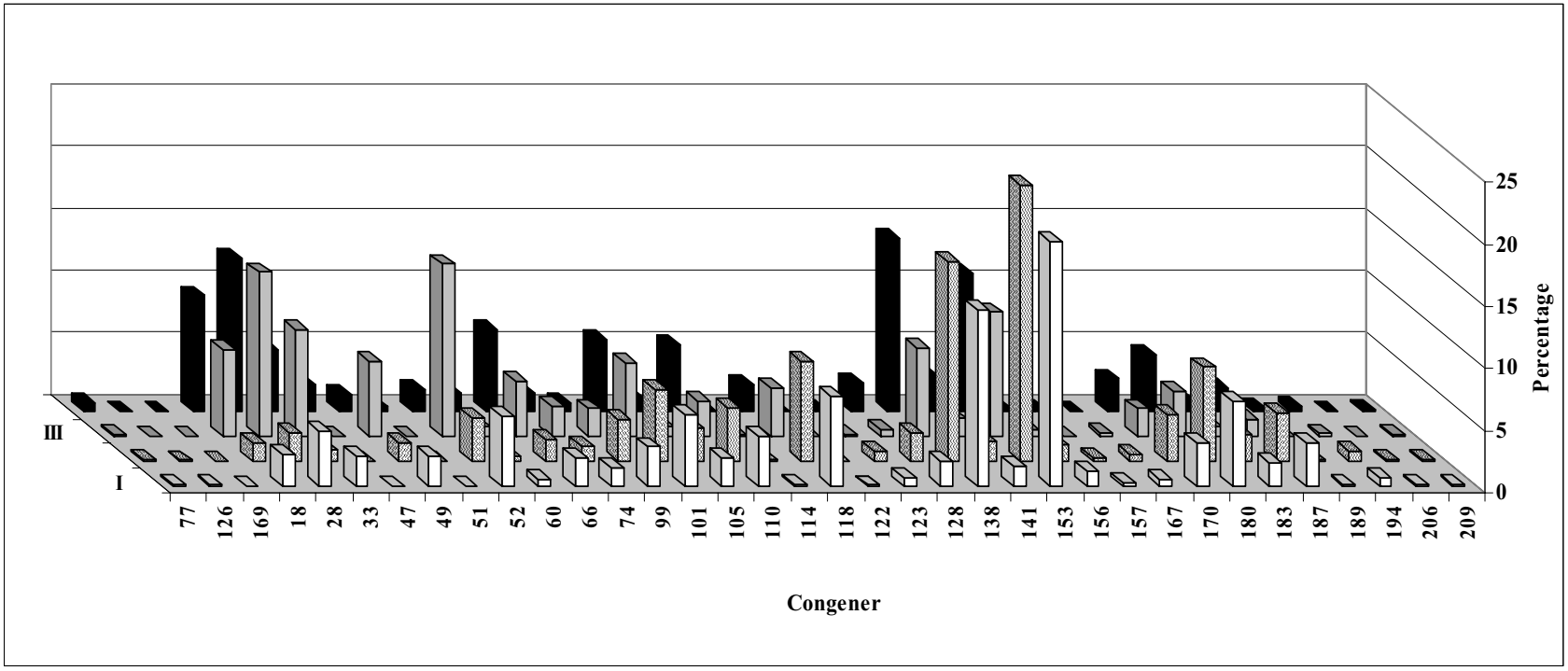

Fig 7. Congener profiles of PCBs as concentration basis in deposition and in Finnish diet. Profiles: I=Total diet, II=Animal origin market baskets, III=Non-animal origin market baskets, IV=Average deposition profile in Finland during 1997-2004. (Market basket data from CHAPTER 3, deposition data from joint research between The Finnish Environment Institute and KTL).

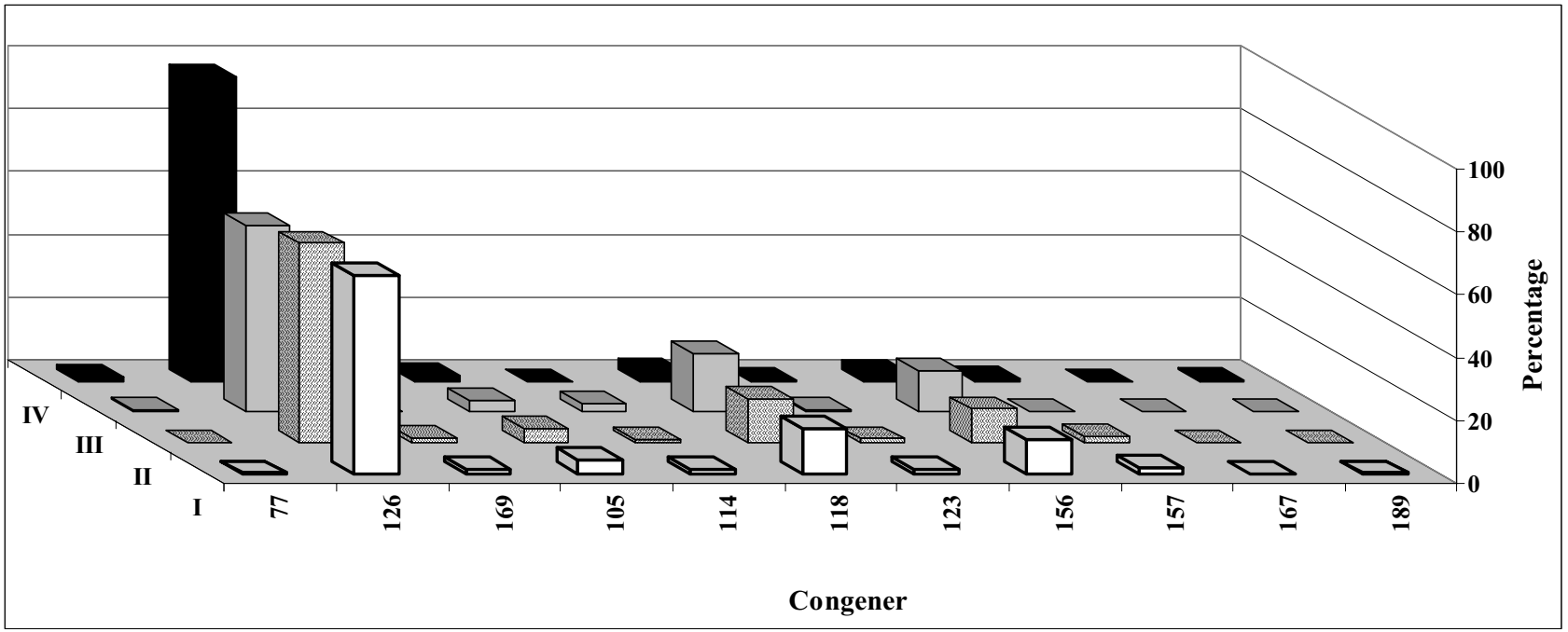

Fig 8. Congener profiles of PCBs as WHO_{PCB}-TEq basis in deposition and in Finnish diet. Profiles: I=Total diet, II=Animal origin market baskets, III=Non-animal origin market baskets, IV=Average deposition profile in Finland during 1997-2004. (Market basket data from CHAPTER 3, deposition data from joint research between The Finnish Environment Institute and KTL).

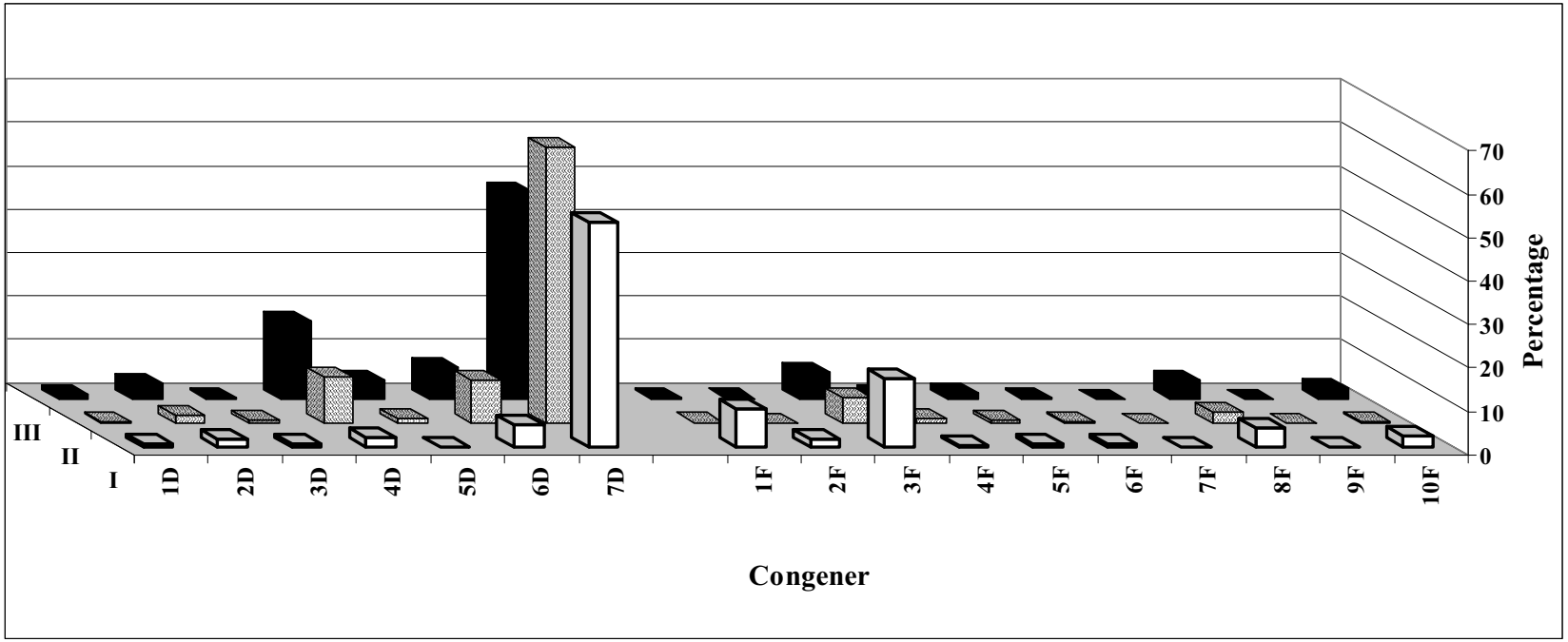

Fig 9. Average congener profiles of PCDD/Fs as concentration basis in the average Finnish diet and in different human samples. Profiles: I=Total diet, II=General Finnish population, and III=Inland lake and Baltic Sea fishermen. Congeners: 1D: 2,3,7,8-TCDD; 2D: 1,2,3,7,8-PeCDD; 3D: 1,2,3,4,7,8-HxCDD; 4D: 1,2,3,6,7,8-HxCDD; 5D: 1,2,3,7,8,9-HxCDD; 6D: 1,2,3,4,6,7,8-HpCDD; 7D: OCDD; 1F: 2,3,7,8-TCDF; 2F: 1,2,3,7,8-PeCDF; 3F: 2,3,4,7,8-PeCDF; 4F: 1,2,3,4,7,8-HxCDF; 5F: 1,2,3,6,7,8-HxCDF; 6F: 2,3,4,6,7,8-HxCDF; 7F: 1,2,3,7,8,9-HxCDF; 8F: 1,2,3,4,6,7,8-HpCDF; 9F: 1,2,3,4,7,8,9-HpCDF; 10F: OCDF.

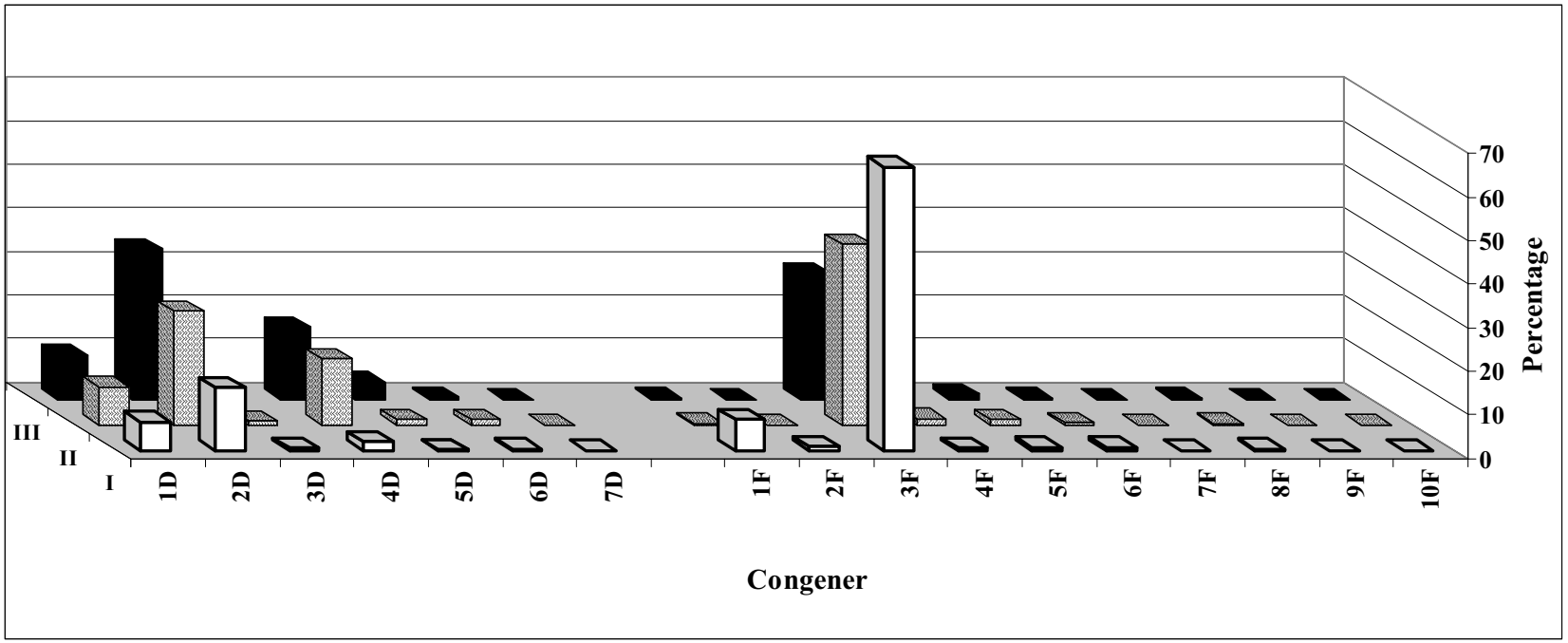

Fig 10. Average congener profiles of PCDD/Fs as $WHO_{PCDD/F}$-TEq basis in the average Finnish diet and in different human samples. Profiles: I=Total diet, II=General Finnish population, and III=Inland lake and Baltic Sea fishermen. Congeners: 1D: 2,3,7,8-TCDD; 2D: 1,2,3,7,8-PeCDD; 3D: 1,2,3,4,7,8-HxCDD; 4D: 1,2,3,6,7,8-HxCDD; 5D: 1,2,3,7,8,9-HxCDD; 6D: 1,2,3,4,6,7,8-HpCDD; 7D: OCDD; 1F: 2,3,7,8-TCDF; 2F: 1,2,3,7,8-PeCDF; 3F: 2,3,4,7,8-PeCDF; 4F: 1,2,3,4,7,8-HxCDF; 5F: 1,2,3,6,7,8-HxCDF; 6F: 2,3,4,6,7,8-HxCDF; 7F: 1,2,3,7,8,9-HxCDF; 8F: 1,2,3,4,6,7,8-HpCDF; 9F: 1,2,3,4,7,8,9-HpCDF; 10F: OCDF.

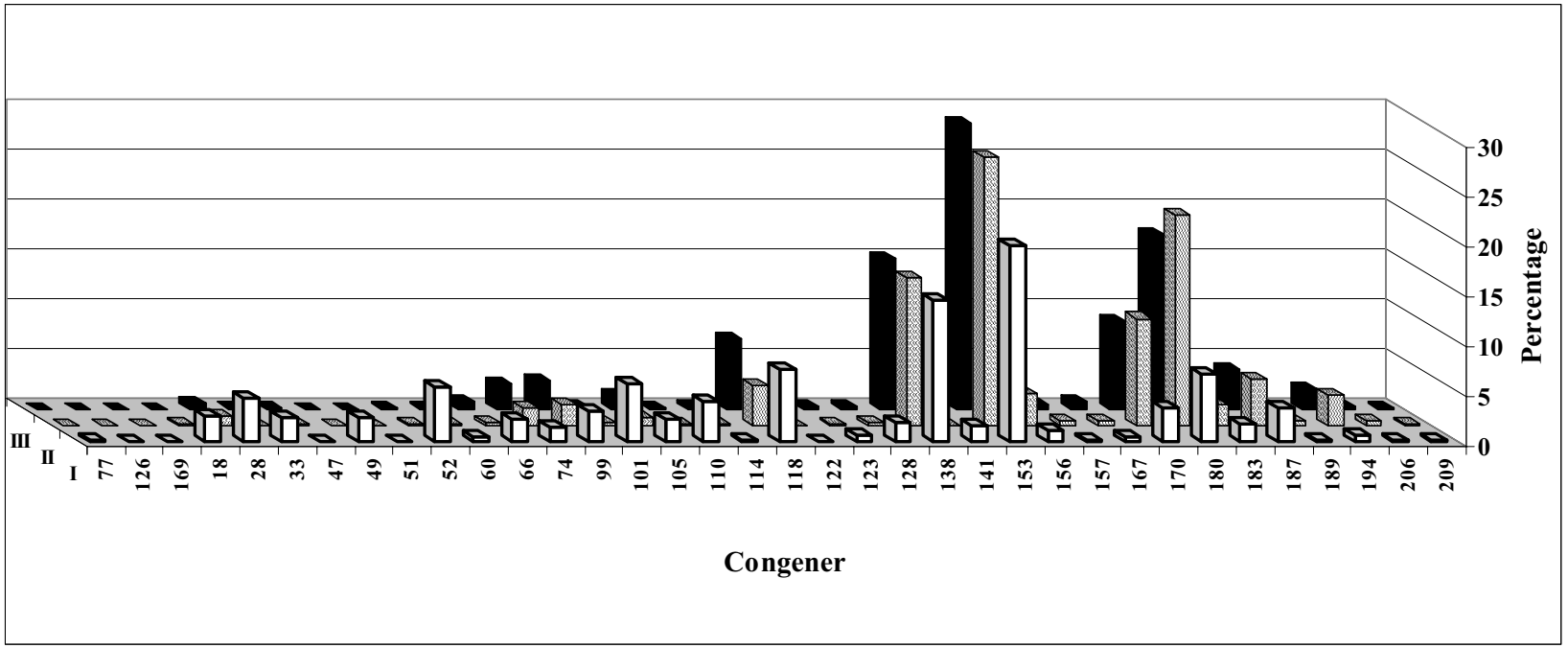

Fig 11. Average congener profiles of PCBs as concentration basis in the average Finnish diet and in different human samples. Profiles: I=Total diet, II=General Finnish population, and III=Inland lake and Baltic Sea fishermen.

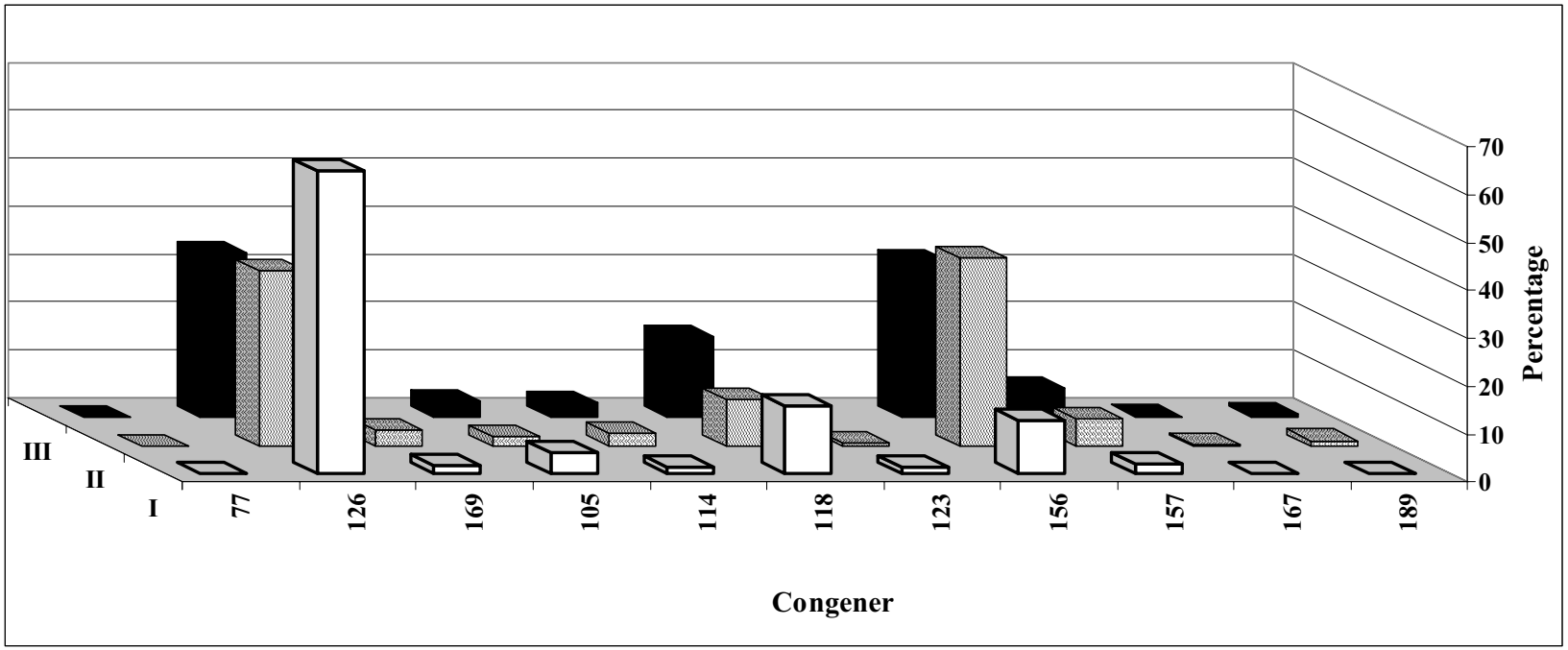

Fig 12. Average congener profiles of PCBs as WHO_{PCB}-TEq basis in the average Finnish diet and in different human samples. Profiles: I=Total diet, II=General Finnish population, and III=Inland lake and Baltic Sea fishermen.

According to changes in PCB congener contributions between the total diet/exposure and human samples, it is obvious that congeners with five or less chlorine substituents are not bioaccumulating in humans from food to the extent seen with the higher chlorinated PCBs (Fig 11 and 12). All tri, tetra, and penta chlorinated PCB congeners, including non-*ortho*-PCB 126, and two hexachlorinated congeners (PCB 128, and 141) expressed a weaker contribution to the congener profile in human samples than would have been predicted from their presence in the total average Finnish diet (Fig 11 and 12). Those congeners showing the highest bioaccumulation tendency included PCBs 180, 170, 194, 156, 153 and 138.

Changes in PCDD/F and PCB profiles attributable to the deposition or other non-animal origin sources via the food-chain into humans raises the question of the applicability of the current TEF scheme in assessing the risk to human health of these contaminants in different matrices. The differences in accumulation efficiencies should be taken into account when assessing TEF by e.g. using matrix specific TEFs.

The dominating congeners in adipose tissue samples of the Finnish general population were almost the same as those reported for the Swedish population with two exceptions (see CHAPTER 1, Fig. 5 and 6). In Finnish people, the contribution of 1,2,3,6,7,8-HxCDD exceeded the contribution of 2,3,7,8-TCDD in the $WHO_{PCDD/F}$-TEq profile, while the reverse was true for the Swedes (Fig 13). In WHO_{PCB}-TEq, the proportion of PCB 156 in the Finnish population was slightly higher than the contribution of PCB 126, while the reverse was true for Swedes as well as inhabitants of other countries (Fig 14). The predominance of the congener 2,3,4,7,8-PeCDF in $WHO_{PCDD/F}$-TEq profile in the general population in Finland can be traced to the consumption of Baltic Sea fish, similar to Sweden. The relatively lower contribution of congeners 2,3,7,8-TCDD and 1,2,3,7,8-PeCDD might reflect the fact that these foods are relatively less contaminated with PCDD/Fs and PCBs in Finland, when compared to other European countries and the USA.

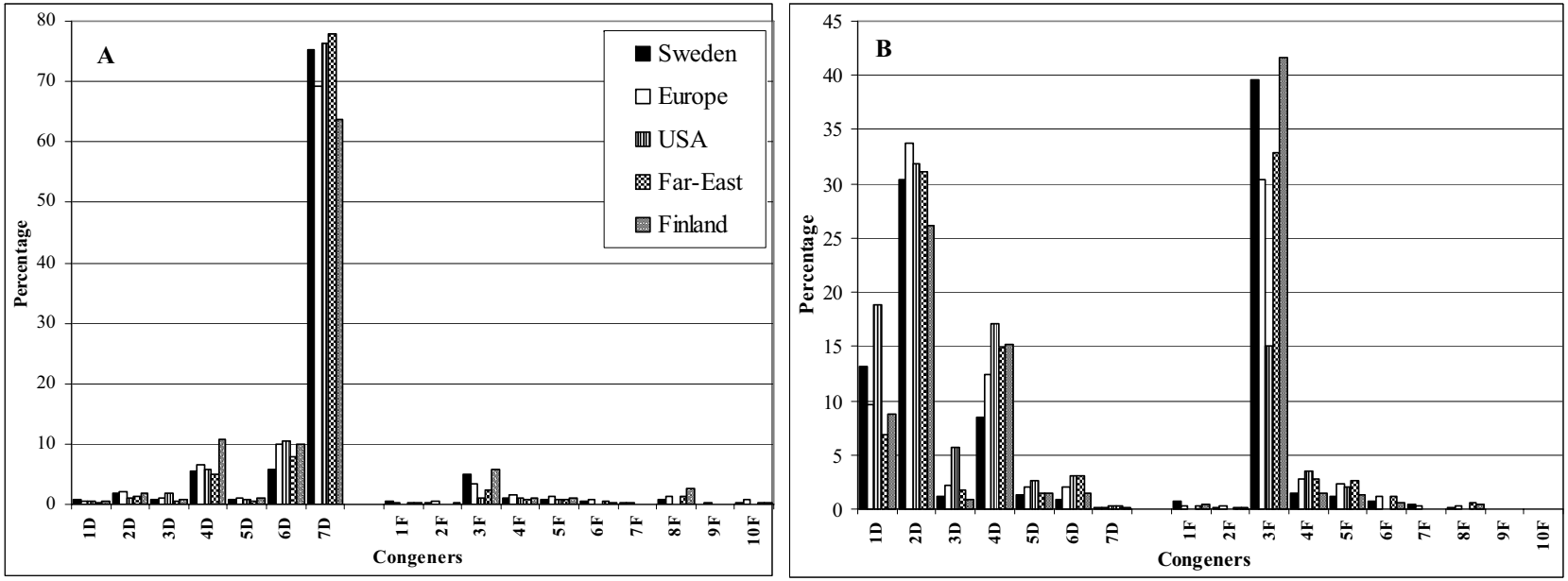

Fig 13. Adipose tissue or serum fat congener profile of (A) sum of PCDD/Fs and (B) $WHO_{PCDD/F}$-TEqs in Sweden, Europe, USA, Far-East, and Finland. (Wingfors et al. 2000, Päpke 1998, Koppen et al. 2002, Arfi et al. 2001, Schecter et al. 2003, Kumar et al. 2001, Choi et al. 2002, Kim et al. 2005, this study). Congeners: 1D: 2,3,7,8-TCDD; 2D: 1,2,3,7,8-PeCDD; 3D: 1,2,3,4,7,8-HxCDD; 4D: 1,2,3,6,7,8-HxCDD; 5D: 1,2,3,7,8,9-HxCDD; 6D: 1,2,3,4,6,7,8-HpCDD; 7D: OCDD; 1F: 2,3,7,8-TCDF; 2F: 1,2,3,7,8-PeCDF; 3F: 2,3,4,7,8-PeCDF; 4F: 1,2,3,4,7,8-HxCDF; 5F: 1,2,3,6,7,8-HxCDF; 6F: 2,3,4,6,7,8-HxCDF; 7F: 1,2,3,7,8,9-HxCDF; 8F: 1,2,3,4,6,7,8-HpCDF; 9F: 1,2,3,4,7,8,9-HpCDF; 10F: OCDF.

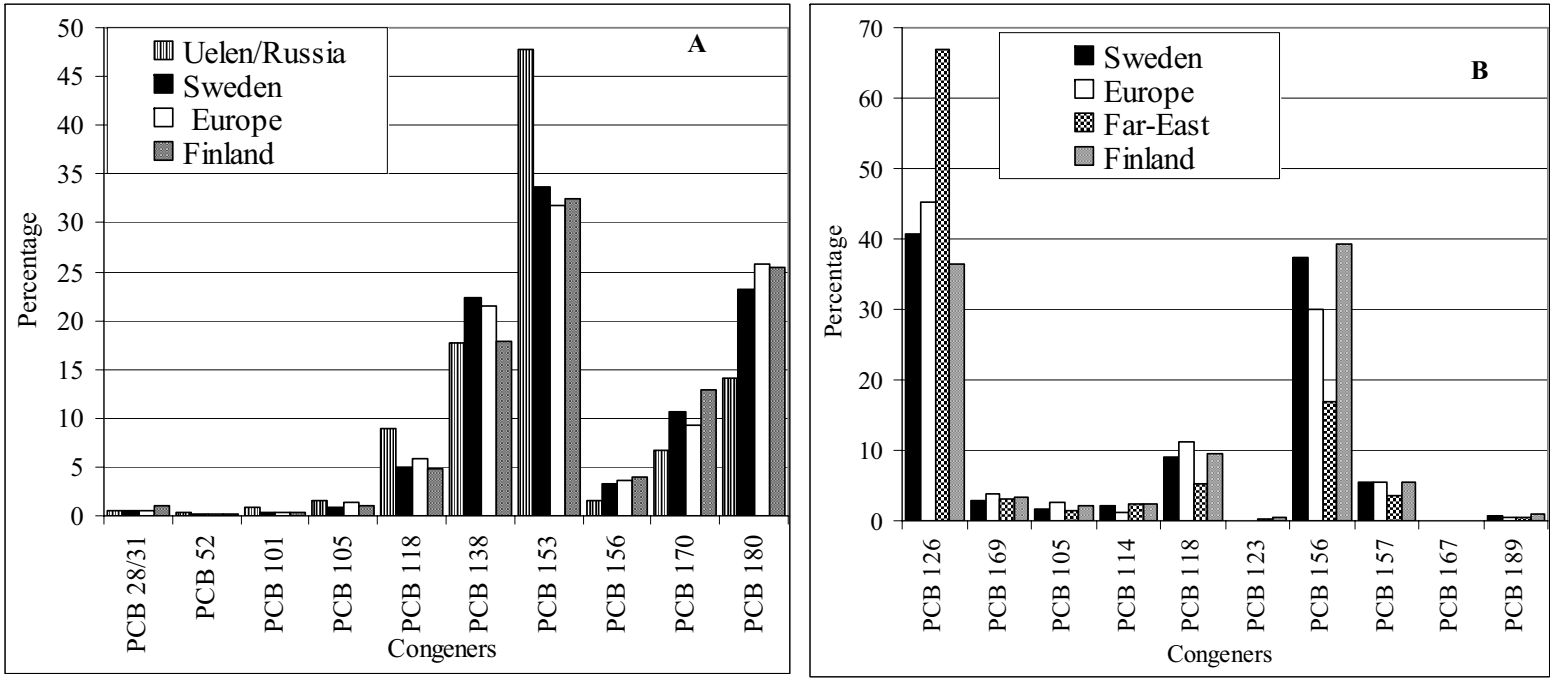

Fig 14. Adipose tissue or serum fat congener profile of (A) certain PCBs and (B) WHO_{PCB}-TEqs in Uelen/Russia, Sweden, Europe, Far-East, and Finland (Wingfors et al. 2000, Wicklund Glynn et al. 2000, Wicklund Glynn et al. 2003, Sjödin et al. 2000, Grimvall et al. 1997, Covaci et al. 2002, Koppen et al. 2002, Costabeber and Emanuelli 2003, Sandanger et al. 2003, Kumar et al. 2001, Choi et al. 2002, this study).

6. CONCLUSIONS

Based on the results of the present studies, the following conclusions can be drawn about the exposure and human PCDD/F and PCB body burden in Finland:

1. *Assessment of the average daily intake of PCDD/Fs and PCBs*

- On average, PCDD/Fs and PCBs contributed equally to the total daily adult intake of these contaminants. The total average intake was below the EU SCF suggested tolerable daily intake (2 pg WHO-TEQ/kg bw). With respect to the WHO suggested TDIs, the average exposure of the Finnish population was well below the TDI on a provisional basis (4 pg WHO-TEQ/kg bw), but the suggested ultimate goal of TDI (1 pg WHO-TEQ/kg bw) was exceeded.
- The average daily adult intake of $WHO_{PCDD/F}$-TEq was very similar to that in other European countries, while WHO_{PCB}-TEq intake was somewhat lower.
- A significant part of PCDD/Fs and PCBs in the Finnish diet originated from fish and fish products.
- A decline in the intake of PCDD/Fs during 1990s was detected, which is similar to that seen in other countries throughout the world. The main causes of the diminishing concentrations are likely to be (1) lowering concentrations in foodstuffs, but not in fish and (2) changes in population dietary habits.

2. *Assessment of the average body burden of PCDD/Fs and PCBs*

- Adipose tissue concentrations of PCDD/Fs and PCBs in Finnish people were comparable to the concentrations found in other countries.
- There was a decrease in concentrations of PCDD/Fs and PCBs from coastal area to the inland area, and this was thought to be due to differences in fish consumption.
- The body burdens of PCDD/Fs and PCBs in the general Finnish population did not follow the upward convex curve with increasing concentrations until 40 years of age to be expected on toxicokinetic basis at constant intake. This was concluded to be due to decreasing exposure of the general population to these contaminants.

3. *Assessment of the PCDD/Fs and PCBs concentrations in breast milk*

- A decline in breast milk concentrations of PCDD/Fs and PCBs was detected between 1987 and 1994 being annually 4% and 8%, respectively. This decrease continued also after taking into account the more recent breast milk concentrations from the years 1995 to 2000, and was similar to that found in other countries.

- Concentrations of the contaminants in breast milk samples in 2000 were somewhat lower than those in other European countries.
- The concentrations in breast milk declined as one moved away from the capital area to a more inland (Kuopio) area at least till the mid 1990s. This gradient was concluded to be due to consumption of different fish species in these two locations.

4. *High exposure fishermen population*

- Professional fishermen were found to be a population highly exposed to PCDD/Fs and PCBs, with the concentrations of both compound groups being 2 to 4 times higher compared to non-fishermen of the same age.
- The source of this high exposure was concluded to be more frequent use of wild fish, especially Baltic fatty fish by professional fishermen.
- PCDD/F congener profiles of many individual fishermen closely resembled the congener profile of the fish species that the fisherman mostly consumed as food.

5. *Congener occurrence and accumulation*

- Dioxin congeners, especially higher chlorinated compounds, expressed more efficient accumulation potency from diet to human adipose tissue than furan congeners. The accumulation efficiency of the furan congener 2,3,4,7,8-PeCDF was not as high as one would expect it to be on the basis of reported half-life for that congener.
- Lower chlorinated PCB congeners expressed lower bioaccumulation potencies than higher chlorinated congeners.
- When comparing the average Finnish adipose tissue congener pattern to the corresponding patterns in other countries, it was evident that the Finnish pattern most resembled the Swedish pattern.

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ANNEX I

Dietary advice on fish consumption dated April 28, 2004

Fish is recommended food and consumption of fish should be increased. Fish contain healthy fatty acids, several vitamins and minerals and a lot of protein. Fish are a particularly good source of n-3 fatty acids and vitamin D. The useful fatty acids contained in fish have been shown to reduce the risk of cardiovascular diseases.

The National Nutrition Council recommends that
- fish should be eaten at least twice a week
- different fish species should be varied in the diet.

EXCEPTIONS TO DIETARY ADVICE ON FISH CONSUMPTION

Despite the favourable nutritional qualities of fish, salmon and herring caught in the Baltic Sea, particularly in the Gulf of Bothnia and the Gulf of Finland, may subject consumers to higher than normal levels of dioxins and PCB compounds which are harmful to health. Also, higher than normal levels of methyl-mercury can be derived from predatory fish caught in inland waters, particularly pike, but also from pike caught in the sea. The older the fish, the more contaminants will have been accumulated in it. For these reasons, the following special recommendations have been issued to children, young people and people at fertile age.

Large Baltic herring and wild-caught salmon
Large herring, more than 17 cm in length (whole fish), can be eaten once or twice a month and as an alternative to large herring salmon caught in the Baltic Sea can be eaten once or twice a month.

Pike and predatory fish from inland waters
Pike caught in the sea or inland waters can be eaten once or twice a month.

In addition to these recommendations
- consumers who eat fish from inland waters on an almost daily basis should also reduce their consumption of the following predatory fish that accumulate mercury: large perches, pike perches and burbots
- pregnant women and nursing mothers should not eat pike due to the mercury risk

FISH CONTAMINANTS AND DIETARY ADVICE

The purpose of the dietary advice is to ensure safe consumption of fish. The advice concerns dioxins, PCB compounds, mercury and cesium-137 contained in fish. The safety assessments are based on a portion size of 100 g of fish. If the portion eaten is smaller, fish can be eaten more often. Herring as well as salmon caught from the Baltic Sea and predatory fish from inland waters can be eaten from time to time. In summer, for example, they can be eaten in larger amounts, as long as the total annual consumption is balanced and restricted.

Part (up to one third) of the dioxins and PCB compounds accumulated in fish can be removed by skinning the fish before preparing it as food. The exceptions to dietary advice do not apply to small herring, less than 17 cm long (whole fish). Filleted herring are usually large, more than 17 cm in length.

The dioxin and PCB levels in fish from inland waters are normally low, and the mercury levels in other lake fish are lower than in pike. The mercury and cesium-137 levels in fish vary from one lake to the other.

Farmed fish contain only low levels of dioxin and PCB compounds, thanks to the control of fish feed quality.