National Dioxins Program

Technical Report No. 9
Dioxins in the Australian Population:
Levels in Blood

A consultancy funded by the Australian Government
Department of the Environment and Heritage

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2. Dioxins emissions from Motor Vehicles in Australia
3. Inventory of Dioxin emissions in Australia, 2004
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Foreword

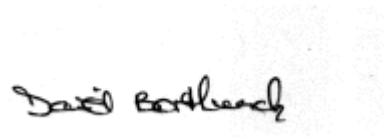
When the Australian Government established the four year National Dioxins Program in 2001, our knowledge about the incidence of dioxins in Australia was very limited.

The aim of the program was to improve this knowledge base so that governments were in a better position to consider appropriate management actions. Starting in mid 2001, a range of studies were undertaken which involved measuring emissions from sources such as bushfires, as well as dioxin levels in the environment, food and population. The findings of these studies were used to shed light on the risk dioxins pose to our health and the environment.

This work has been completed and the findings are now presented in a series of twelve technical reports.

Having good information is essential if there is to be timely and effective action by governments; these studies are a start. Our next step is to foster informed debate on how we should tackle dioxins in Australia, as this is an obligation under the Stockholm Convention on Persistent Organic Pollutants. The Department of the Environment and Heritage will be working closely with other Australian Government, State and Territory agencies to take this step.

Ultimately, the effective management of dioxins will be the shared responsibility of all government jurisdictions with the support of the community and industry.



David Borthwick
Secretary
Department of the Environment and Heritage

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- the Department of Agriculture, Fisheries and Forestry, who assessed the levels of dioxins in agricultural commodities
- Food Standards Australia New Zealand and the Department of Health and Ageing and who assessed the levels of dioxins in foods and assessed the health effects of dioxins
- officers of the Chemical Assessment Section in DEH who assessed the ecological effects of dioxins
- members of the National Dioxins Project Team which included representatives from the State and Territory environment protection agencies, the Australian Health Ministers Conference and the Primary Industries Ministers Council
- members of the National Dioxins Consultative Group which included representatives from industry and agricultural sectors, environment and public health groups and research institutions

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Executive Summary

This study was a component of the National Dioxins Program that was tasked to quantify and assess the concentrations and relative chemical compositions of dioxin-like chemicals in blood serum of the Australian population.

The results of this study provide a measure of the levels of dioxin-like compounds, polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and polychlorinated biphenyls (PCBs), in pooled blood serum collected throughout Australia in 2003. Serum samples were collected using Sullivan and Nicolaides Pathology (SNP), a pathology company based in Brisbane but with an extensive national network. De-identified samples were selected from surplus pathology samples according to stratification criteria that were provided to Sullivan and Nicolaides staff. These stratification criteria were as follows:

Regional Stratification: 5 regions representing the regional and population distribution of Australians

- Southeast urban
- South urban
- Northeast urban
- West urban
- Rural region encompassing all rural regions of Australia

Age Stratification: 5 age groups

- <16 years
- 16-30 years
- 31-45 years
- 46-60 years
- > 60 years

Gender Stratification

- males
- females.

In total 9,090 samples from the 50 strata were collected and pooled to give 96 pools according to the above criteria. An additional 204 samples, representing four pools, were also assessed as part of a pilot study to determine the suitability of the use of surplus pathology samples for this study.

All pooled samples were sent to ERGO- Forschungsgesellschaft mbH, Hamburg, and ten duplicate samples were sent to Health Canada, Ottawa, Canada for inter-laboratory comparison. Both are laboratories accredited for analytical dioxin analysis.

Dioxin-like compounds were detected in all strata. Overall the levels in the Australian population are very low by international standards and comparable with, although lower than, those observed in the New Zealand population (Buckland et al, 2001). It should

be noted that the samples analysed for the New Zealand study were collected in 1996-97, and given that the measured levels of these compounds in humans are continuing to decrease over time, this may account for some of difference. The mean and median levels expressed as upper bound TEQ values for all pooled samples were 10.9 and 8.3 pg TEQ g^{-1} lipid, respectively. For males, and females the mean levels were 10.4 and 11.5 pg TEQ g^{-1} lipid, respectively.

A direct relationship of increasing dioxin- like compound levels with increasing age was observed and could be described by the following equation:

$$\textbf{Levels in blood expressed as pg TEQ } g^{-1} \textbf{ lipid} = 3.3 \exp^{0.0251 age} \qquad (r^2 = 0.87)$$

This relationship was found to hold from approximately 25 years of age until at least the eighth decade and, thus, during these years it is possible to estimate the level of dioxin-like compounds in an individual's blood serum.

No systematic differences were observed in the levels of dioxin-like compounds in samples collected from males and females. However, slightly higher levels of dioxin-like compounds were observed in females in the >60 years age group. This result could not be explained on the basis of differences in the mean age between males and females in this group.

The levels of dioxin-like compounds across the five regions were remarkably similar within each age range. Some general trends were noted and include the following:

- the levels of dioxin-like compounds across all regions and within each age range appear to be very similar
- Despite the similarity in levels, for all strata except the <16 years females, the samples from the Southeast region exhibit slightly higher levels of dioxin-like compounds
- For <16 years females the highest levels of dioxin-like compounds were found in the Rural region.

It should be noted that because de-identified samples were used in this study, determination of regional differences was complicated. The use of such samples did not allow any assessment of the length of time an individual had resided in a particular area prior to their sample being collected or recording of either food intake or possible exposure to environmental contaminants in that region.

In summary, the levels of dioxin-like compounds in the Australian population are low by international standards and are very similar across all regions of Australia within each designated age ranges. The levels of these chemicals increase with age and can be estimated if the age of an individual is known.

Glossary/Abbreviations

ACT	Australian Capital Territory.
ADI	Acceptable daily intake.
Congener	Closely related chemicals derived from the same parent compound.
Dioxins/ Dioxin-like Compounds	Common name when referring to all of the following compounds polychlorinated dibenzo-*p*-dioxins, polychlorinated dibenzofurans and polychlorinated biphenyls.
EnTox	National Research Centre for Environmental Toxicology.
Furan	polychlorinated dibenzofuran.
GC/MS	Gas chromatography/mass spectrometry.
GU	Griffith University.
Homologue	A group of structurally related chemicals that have the same degree of chlorination.
HRGC/HRMS	High resolution gas chromatography/ high resolution mass spectrometry.
I-TEQ	Toxicity equivalencies using NATO-CCMS (1988) toxicity equivalency factors.
IUPAC	International Union of Pure and Applied Chemistry.
LCS	Laboratory control sample.
LOD	Limit of detection, the lowest level at which a chemical can be measured in a sample by the analytical method used.
Lower bound TEQ	Toxic equivalencies (TEQ) for which concentration of a non-detected congener assumed to be equal zero.
ml	Milliliter.
Middle bound TEQ	Toxic equivalencies (TEQ) for which concentration of a non-detected congener assumed to be equal to half the non detect value.
Mono-*ortho* PCBs	includes PCB congener numbers 105, 114, 118, 123, 156, 157, 167, 189.
nd	Non-detect.
NDP	National Dioxin Program.
Non-*ortho* PCBs	Includes PCB congener numbers 77, 81, 126, 169.
NSW	New South Wales.
NT	Northern Territory.

OCDD	Octachlorodibenzo-*p*-dioxin.
OCDF	Octachlorodibenzo-furan.
PCBs	Polychlorinated biphenyls.
PCDDs	Polychlorinated dibenzo-p-dioxins.
PCDFs	Polychlorinated dibenzofurans.
pg g^{-1}	Picogram per gram, 10^{-12} g. Equal to nanogram per kilogram (ng/kg).
Pool	Samples collected within each stratum.
POPs	Persistent organic pollutants.
r^2	Regression coefficient.
Region	Geographical location in Australia.
QC	Quality control.
QA	Quality assurance.
QLD	Queensland.
QHSS	Queensland Health Scientific Services.
SA	South Australia.
SNP	Sullivan and Nicolaides Pathology.
Stratum/strata	Represent samples within defined criteria e.g. age, gender, geographical location.
TEQ	Abbreviate of WHO_{98}-TEQ (this document).
TEF	toxicity equivalency factors.
Upper bound TEQ	Toxic equivalencies (TEQ) for which concentration of a non-detected congener assumed to be equal to the non detect value.
UQ	The University of Queensland.
Vic	Victoria.
WA	Western Australia.
WHO	World Health Organization.
WHO_{98}-TEQ	World Health Organization toxic equivalent: the quantified level of each individual congener multiplied by the corresponding TEF. TEQs of each congener are summed to achieve an overall toxic equivalency for a sample (Van den Berg, 1998). In this document WHO_{98}-TEQ is abbreviated to 'TEQ'.

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1. Introduction

1.1 Background

Dioxin is a general term often used to describe a group of compounds that belong to the larger family of persistent organic pollutants (POPs). These include polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and dioxin-like polychlorinated biphenyls (PCBs). In this report, these three groups of compounds will be referred to as dioxin-like compounds. POPs include some of the most toxic chemicals with respect to adverse health effects on humans and wildlife. Long-term uptake of POPs can have chronic effects in organisms such as carcinogenesis, endocrine disruptive properties, immune system effects and developmental impacts (Van den Berg, 1998). Physico-chemical properties of these compounds result in their extreme persistence in the environment, their ubiquitous distribution from sources to remote areas via long range atmospheric transport and their ability to bioaccumulate and biomagnify in higher trophic organisms. Typically, more than 90% of the POP body burden in humans and other mammals is accumulated via food, in particular seafood, meat and dairy products (U.S. EPA 2000, Liem et al. 2000; Fürst et al. 1992).

1.1.1 Organochlorine contaminants

Persistent organic pollutants (POPs) include chlorinated hydrocarbons such as polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and polychlorinated biphenyls (PCBs). PCDDs and PCDFs are tricyclic aromatic hydrocarbons and belong to the family of chemicals known as the halogenated hydrocarbons. They are comprised of two benzene rings joined via one or two oxygen atoms at adjacent carbons on each of the benzene rings. The base structures of these compounds are shown in Figure 1.1; numbers 1-9 indicate the possible positions of the chlorine atoms. They may have up to eight chlorine atoms attached at carbon atoms 1 to 4 and 6 to 9. Each individual compound is referred to as a congener; there are 75 possible PCDD congeners and 135 possible PCDF congeners depending on the location and/or the number of attached chlorine atoms. The biological activity and toxicity of an individual congener varies with the number and position of the chlorine atoms. It should be noted that the most toxic effects are produced from congeners that have chlorine atoms in the 2,3,7,8 positions. Table 1.1 shows the homologue names, possible congener structure and the number of possible 2,3,7,8 congeners for the dioxin and furan family of compounds.

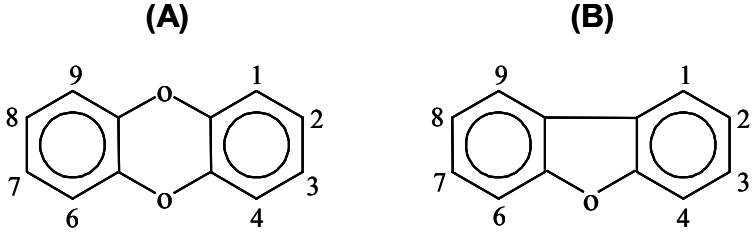

Figure 1.1 The structures of polychlorinated (A) dibenzo-p-dioxins and (B) dibenzofurans.

Table 1.1 Homologues and congeners of PCDDs and PCDFs.

Abbreviation	Homologue name	No. of possible congeners	No. of possible 2,3,7,8-chlorinated congeners
MCDD	Monochlorodibenzo-*p*-dioxin	2	0
DiCDD	Dichlorodibenzo-*p*-dioxin	10	0
TrCDD	Trichlorodibenzo-*p*-dioxin	14	0
TCDD	Tetrachlorodibenzo-*p*-dioxin	22	1
PeCDD	Pentachlorodibenzo-*p*-dioxin	14	1
HxCDD	Hexachlorodibenzo-*p*-dioxin	10	3
HpCDD	Heptachlorodibenzo-*p*-dioxin	2	1
OCDD	Octachlorodibenzo-*p*-dioxin	1	1
MCDF	Monochlorodibenzofuran	4	0
DiCDF	Dichlorodibenzofuran	16	0
TrCDF	Trichlorodibenzofuran	28	0
TCDF	Tetrachlorodibenzofuran	38	1
PeCDF	Pentachlorodibenzofuran	28	2
HxCDF	Hexachlorodibenzofuran	16	4
HpCDF	Heptachlorodibenzofuran	4	2
OCDF	Octachlorodibenzofuran	1	1

1.1.2 Polychlorinated biphenyls

PCBs were compounds that were commercially produced by the chlorination of biphenyl. These compounds were graded and marketed according to their chlorine content. Some exhibit a structural and chemical similarity to the PCDDs and PCDFs and are referred to as dioxin-like PCBs. The base structure of PCBs is shown in Figure 1.2; numbers 2-6 (2'-6') indicate the possible positions of the chlorine atoms at ortho(o), para(p) and meta(m) positions, respectively. As with dioxins, chlorine atoms may attach at any of the positions 2-6 and 2'-6' and there are up to 209 different congeners. The toxicity and the biological activity are also dependent on the number and position of the chlorine atoms. Table 1.2 shows the distribution of PCB congeners derived from the basic biphenyl structure.

Table 1.2 Distribution of PCB congeners.

No of Cl substituents	Cl_1	Cl_2	Cl_3	Cl_4	Cl_5	Cl_6	Cl_7	Cl_8	Cl_9	Cl_{10}
No of congeners	3	12	24	42	46	42	24	12	3	1

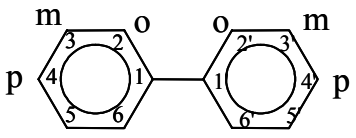

Figure 1.2 The structures of polychlorinated biphenyls (PCBs).

1.1.3 Source of PCDDs and PCDFs

The PCDDs and PCDFs are present as trace contaminants in a number of chemical products and they are formed as by-products during various industrial, chemical and combustion processes. They are formed as undesirable waste products during industrial processes where carbon containing organic material is incinerated in the presence of chlorine. These include the manufacture of chemicals including chlorophenols and herbicides like 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), and in combustion or incineration processes such as waste incinerators and fossil fuel power plants. They also enter the environment from non-industrial and natural sources including domestic wood and waste burning, uncontrolled forest fires, vehicle emissions and tobacco smoke (Gaus et al., 2001).

Since dioxins comprise a large group of compounds with different toxicities, the concept of toxic equivalency factors (TEF) has been developed to facilitate risk assessment of exposure to complex mixtures of these compounds and promote international consistency in addressing dioxin contamination (Van den Berg, 1998, 2000). This concept is based on the evidence that dioxin-like compounds share a common mechanism of action - binding to the Ah-receptor. Of these compounds, 29 are considered by the World Health Organization (WHO) to have significant toxicity (Van den Berg et al., 1998). The toxicity of the different compounds relative to that of 2,3,7,8-TCDD is determined on the basis of toxicological data. Each compound is then assigned a TEF relative to that of 2,3,7,8-TCDD. Hence, the closer the TEF is to 1.0, the more toxic the congener. By multiplying the TEF of the individual compounds by their concentration, toxic equivalencies (TEQ) can be calculated and used for risk characterisation and management purposes. Table 1.3 shows the toxic equivalency factors for PCDDs, PCDFs and dioxin-like PCBs for humans and mammals (Van den Berg et al., 1998).

Table 1.3 TEF values for PCDDs, PCDFs and PCBs.

Congener	TEF value	Congener	TEF value
Dibenzo-p-dioxins		*Non-ortho PCBs*	
2,3,7,8-TCDD	1	PCB#77	0.0001
1,2,3,7,8-PnCDD	1	PCB#81	0.0001
1,2,3,4,7,8-HxCDD	0.1	PCB#126	0.1
1,2,3,6,7,8-HxCDD	0.1	PCB#169	0.01
1,2,3,7,8,9-HxCDD	0.1		
1,2,3,4,6,7,8-HpCDD	0.01		
OCDD	0.0001		
Dibenzofurans		*Mono-ortho PCBs*	
2,3,7,8-TCDF	0.1	PCB#105	0.0001
1,2,3,7,8-PeCDF	0.05	PCB#114	0.0005
2,3,4,7,8-PeCDF	0.5	PCB#118	0.0001
1,2,3,4,7,8-HxCDF	0.1	PCB#123	0.0001
1,2,3,6,7,8-HxCDF	0.1	PCB#156	0.0005
1,2,3,7,8,9-HxCDF	0.1	PCB#157	0.0005
2,3,4,6,7,8-HxCDF	0.1	PCB#167	0.00001
1,2,3,4,6,7,8-HpCDF	0.01	PCB#189	0.0001
1,2,3,4,7,8,9-HpCDF	0.01		
OCDF	0.0001		

1.2 Objectives

The overall objective of this project was to evaluate the concentration of dioxins and dioxin-like PCBs in human blood serum collected from Australia and to compare this with international levels of these compounds. This would establish a sound understanding of contaminant levels in the general Australian population with respect to gender, regional (both urban and rural) and age differences.

Such baseline knowledge, together with environmental exposure studies is fundamental for assessing health and environmental risks that may be associated with the presence of these toxicants. The results of this study will assist in defining priority areas to determine strategies to reduce the exposure of Australians to harmful pollutants.

2. Project Design

The overall study aim for the National Dioxins Program was to obtain between 5,000 and 10,000 blood samples that were stratified according to region, age and gender into 50 discrete replicate strata giving a total of 100 pooled samples for analysis. The study design and stratification approach is described in detail in Section 2.2. To achieve Australia-wide collection of the samples the study relied on a close collaboration with Sullivan and Nicolaides Pathology (SNP), a nationwide pathology laboratory. Specifically, it was proposed to use surplus samples that were collected by the laboratory as part of routine pathology requests. The proposed use of pathology samples introduced the potential for a biased sample population, as by definition, they are collected from individuals who may have some degree of perturbation in their normal physiology. Due to the potential for the introduction of bias, a pilot study was carried out in which the levels of dioxin-like compounds in samples collected for routine pathological assessment were compared with those collected for insurance assessments in order to determine whether there was a detectable difference between the two groups that would preclude the use of this recruitment strategy.

2.1 Pilot Study

The pilot study was carried out prior to the commencement of the main study. Description of the sample collection for the pilot as well as the results and discussion are presented in this section of the report. Details regarding the analytical methodology and quality control procedures from the analytical laboratory are given in Section 3 with the main study results and discussion.

2.1.1 Sample Collection

Two replicate pools from a single region (Brisbane) and a single age group (males 30-45) were collected from SNP using routine pathology samples and those collected for insurance assessments. Each "insurance" sample was matched on the basis of year of birth (±1 year), postcode and collection date (±12 months) with a "pathology" sample. Summary data for the samples included in each pool is given in Table 2.1.

Table 2.1 Summary data for samples used to compare pathology and insurance groups.

Note, that a detailed list of the individual samples and matches is provided in Appendix A.

	Insurance 1	**Pathology 1**	**Insurance 2**	**Pathology 2**
Region	Brisbane	Brisbane	Brisbane	Brisbane
Gender	Male	Male	Male	Male
Age Group (years)	30-45	30-45	30-45	30-45
Age (years) - Mean±SD	37.6±4.3	37.6±4.3	37.1±4.0	37±4.0

Samples were aliquoted by staff at SNP into four pools each consisting of 51 individual one ml samples. The matched samples were designated as "Insurance 1" and "Pathology 1" and "Insurance 2" and "Pathology 2", respectively. Samples were refrozen and transported on dry ice to ERGO- Forschungsgesellschaft mbH (ERGO), Hamburg, Germany laboratory for analysis. Transit time was approximately 48 hours.

2.1.2 Results of Pilot Study

The levels of dioxins, furans and dioxin-like PCBs are given in Table 2.3 and results are expressed as TEQ in Figure 2.1. The full report from ERGO including analytical Methodology is given in Appendix B.

Dioxin-like chemicals were detectable in all of the samples analysed and the levels, expressed as TEQ, ranged from 11 (pathology 2) to 14 (insurance 1) pg TEQ g^{-1} lipid. Considering the non-detectable congeners (mostly higher chlorinated furans) in all the samples about 90% or greater of the TEQ was derived from detected and quantified values. 1,2,3,7,8-pentachlorodibenzodioxin and 3,3',4,4',5-pentachlorobiphenyl were the single most relevant components in the congener profile contributing about 20% and 15%, respectively, to the overall TEQ value. 2,3,7,8-TCDD was detectable in 3 of the 4 samples but was consistently close to the detection limit. Overall the PCDD/PCDF congener profile was dominated by higher chlorinated PCDDs whereas concentrations of higher chlorinated PCDFs (Cl6 or greater) were almost exclusively below the limit of detection in all samples.

Differences between the pools were assessed using the normalised difference (Müller 2000) (see Box 1). For most of the congeners the difference between the pathology and insurance pools in the two comparisons was less than 20% and, thus, may be explained by the normal variations in analytical reproducibility.

Box 1. Normalised Differences

In this report, comparisons between replicate samples or replicated analysis have been made using the mean normalised difference. The normalised difference between two samples is mathematically defined as:

$$\text{normalised difference (\%)} = \frac{|\text{value a} - \text{value b}|}{\frac{(\text{value a} + \text{value b})}{2}} \cdot 100$$

Table 1. below provides a demonstration of the normalised difference (ND) values that would result from a range of differences in sample values.

Examples of normalised differences (ND) that would result from different sample values.

Sample A (pg g^{-1} lipid)	Sample B (pg g^{-1} lipid)	ND %
1.0	1.2	18.2
1.0	1.5	40.0
1.0	2.0	66.7
1.0	3.0	100.0
1.0	10.0	163.6
1.0	100.0	196.0

The normalised difference was compared both within and between groups (Table 2.2). Using the mean of the normalised difference for all detected congeners, the highest normalised difference was apparent in comparisons of the cohorts insurance 1 and insurance 2 (31%) and pathology 1 and insurance 1 (24.9%). The contrast of the means of pathology 1 and pathology 2 with the mean of the cohorts insurance 1 and insurance 2 gave the smaller normalised mean difference of 10.9. As these findings indicate that

there is the possibility of greater variation within the same group (i.e. insurance 1 and insurance 2) than between different groups (insurance and pathology) the authors consider that it is unlikely that sampling bias would preclude the use of pathology samples in the main study. Prior to the decision to utilise SNP for the collection, the use of a minimally biased sampling strategy was considered. This would involve a totally random collection of samples across the general population. When compared to the use of surplus pathology samples, obvious difficulties in using such a method include the following:

- a much more complex procedure
- a greater risk of insufficient response rate and, hence, lower sampling numbers
- greatly increased resources including employment of phlebotomists, container and transport costs
- necessity to consent each participant
- impossible to collect a large number of samples within budget constraints.

Hence, it was concluded that the risk of a sampling bias producing an effect on the results was small compared to the increased expense and risk of obtaining insufficient numbers of samples using a minimally biased sampling strategy.

Table 2.2 Normalised differences for pathology and insurance samples.

The mean pathology versus mean insurance value was calculated by first calculating the mean concentrations and then calculating the normalised difference.

Sample Group	Normalised Difference (%)
pathology 1 v insurance 1	24.9
pathology 2 v insurance 2	17.7
pathology 1 v pathology 1	14.0
insurance 1 v insurance 2	31.0
mean pathology v mean insurance	10.9

Table 2.3 Concentration of dioxin-like chemicals in samples.

Values given are the TEQ expressed in pg g^{-1} lipid. Also included are results of a comparison of the relative concentration in the pathology versus the insurance cohorts for the two matched groups

Congener	pathology 1	insurance 1	pathology 2	insurance 2
2,3,7,8-Tetra-CDD	nd	1.5	1.4	1.1
1,2,3,7,8-Penta-CDD	2.6	3.0	2.0	3.3
1,2,3,4,7,8-Hexa-CDD	2.1	2.2	2.0	2.4
1,2,3,6,7,8-Hexa-CDD	13.0	15	9.5	12
1,2,3,7,8,9-Hexa-CDD	2.9	2.7	2.2	1.4
1,2,3,4,6,7,8-Hepta-CDD	19	26	17	19
OCDD	230	280	210	210
2,3,7,8-Tetra-CDF	nd	nd	nd	nd
1,2,3,7,8-Penta-CDF	0.70	1.1	1.0	0.70
2,3,4,7,8-Penta-CDF	2.5	2.8	2.1	2.6
1,2,3,4,7,8-Hexa-CDF	2.4	2.5	2.7	2.6
1,2,3,6,7,8-Hexa-CDF	1.6	2.0	1.5	1.7
1,2,3,7,8,9-Hexa-CDF	nd	nd	nd	nd
2,3,4,6,7,8-Hexa-CDF	nd	1.8	nd	nd
1,2,3,4,6,7,8-Hepta-CDF	nd	nd	nd	nd
1,2,3,4,7,8,9-Hepta-CDF	nd	nd	nd	nd
OCDF	nd	nd	nd	nd
3,3',4,4'-TCB	nd	nd	nd	nd
3,4,4',5-TCB	nd	nd	nd	nd
3,3',4,4',5-PeCB	24	26	17	17
3,3',4,4',5,5'-HxCB	16	18	15	15
2,3,3',4,4'-PeCB	970	1000	700	670
2,3,4,4',5-PeCB	320	360	290	240
2,3',4,4',5-PeCB	3300	4500	2700	2600
2',3,4,4',5-PeCB	nd	nd	nd	nd
2,3,3',4,4',5-HxCB	2300	2700	3000	2000
2,3,3',4,4',5'-HxCB	630	670	700	490
2,3',4,4',5,5'-HxCB	580	710	560	480
2,3,3',4,4',5,5'-HpCB	280	290	270	230
Total DFs	**280**	**340**	**250**	**260**
Total non-ortho-PCBs	**40**	**44**	**33**	**32**
Total mono-ortho-PCBs	**8500**	**10000**	**8300**	**6600**
TEQ DFs	**7.6**	**9.3**	**7.2**	**8.3**
TEQ PCBs	**4.7**	**5.2**	**4.3**	**3.6**
TEQ	**12**	**14**	**11**	**12**

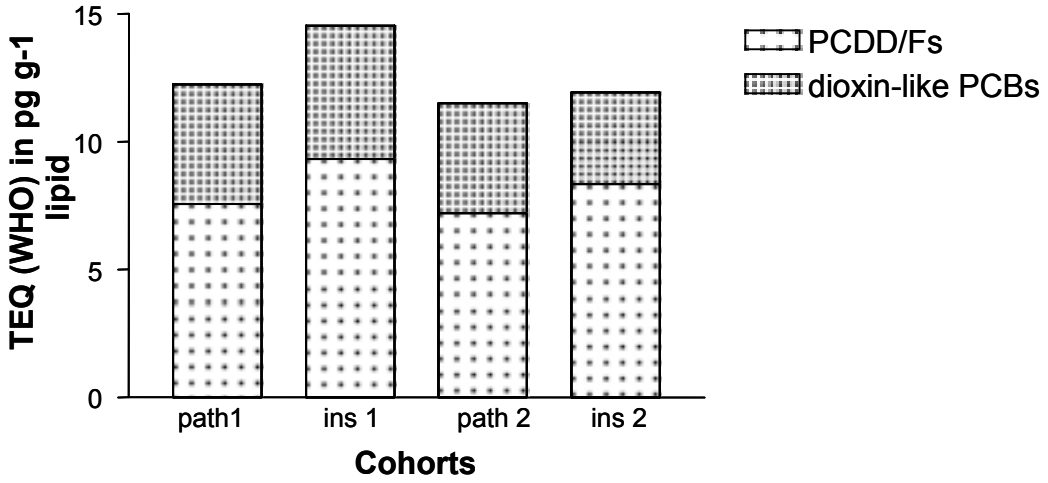

Figure 2.1 Concentration of dioxin-like chemicals in Pilot study for 30-45 age residing in Brisbane metropolitan areas.
For each individual in Pathology 1 and Pathology 2 a matched sample (same age & postcode) was used in pools Insurance 1 and Insurance 2, respectively.

2.2 Sampling Design

Stratification approach for the collection of a representative group of serum samples from the Australian population

The project was designed on the basis of a request to tender from the Australian Government Department of Environment and Heritage Environment (DEH) to assess the levels of dioxin-like compounds in the blood serum of the Australian population. Serum samples were collected by SNP from stored sera that had been collected as part of their routine testing procedures. It should be noted that throughout this report serum specimens are referred to as samples. Samples were obtained according to the stratification criteria outlined below:

Age Stratification

- <16 years
- 16-30 years
- 31-45 years
- 46-60 years
- >60 years

Gender Stratification

- Female
- Male

Geographic Stratification

- Four urban regions:
  - Northeast (including Brisbane, Tweed and Gold Coast and major population centres in Queensland)
  - Southeast (Sydney, Canberra, Wollongong, Newcastle and other major population centres from New South Wales)
  - South (Melbourne, Adelaide, Hobart and other major population centres from Victoria)
  - West (Perth and other major population centres in Western Australia).
- One rural region:
  - Including rural areas from all States and the Northern Territory. Rural areas were defined as those postcodes outside metropolitan or major regional centres.

Table 2.4 shows the 50 different strata from which samples were collected.

Table 2.4 Stratification approach for the collection of a representative group of serum samples from the Australian population.

Samples were collected in strata defined by gender, age and region. Information regarding percent of total population was derived from the Australian Bureau of Statistics.

		Northeast Urban	Southeast Urban	South Urban	West Urban	Rural Area	Approx. % of total population
Gender	**Age**	**Urban**	**Urban**	**Urban**	**Urban**	**Rural**	
M	**<16**	2 Pools	2 Pools	2 Pools	2 Pools	2 Pools	**8%**
	16-30	2 Pools	2 Pools	2 Pools	2 Pools	2 Pools	**9%**
	30-45	2 Pools	2 Pools	2 Pools	2 Pools	2 Pools	**10%**
	45-60	2 Pools	2 Pools	2 Pools	2 Pools	2 Pools	**8%**
	>60	2 Pools	2 Pools	2 Pools	2 Pools	2 Pools	**5%**
F	**<16**	2 Pools	2 Pools	2 Pools	2 Pools	2 Pools	**8%**
	16-30	2 Pools	2 Pools	2 Pools	2 Pools	2 Pools	**9%**
	30-45	2 Pools	2 Pools	2 Pools	2 Pools	2 Pools	**10%**
	45-60	2 Pools	2 Pools	2 Pools	2 Pools	2 Pools	**8%**
	>60	2 Pools	2 Pools	2 Pools	2 Pools	2 Pools	**5%**
Approx. % of total population		**25%**	**20%**	**9%**	**7%**	**19%**	**~80%**

It was aimed to collect duplicate samples for each stratum giving a total of 100 samples. Note that the final number of samples analysed was 96 for the main study and 4 for the pilot study. This was due to difficulties encountered in the collection of samples in the <16 years age groups in the south-west and west urban regions. This is explained in more detail in section 3.1. The approximate percent of the population represented by each stratum as determined by Australian Bureau of Statistics data is also shown. Figure 2.2 shows the geographical distribution of the strata.

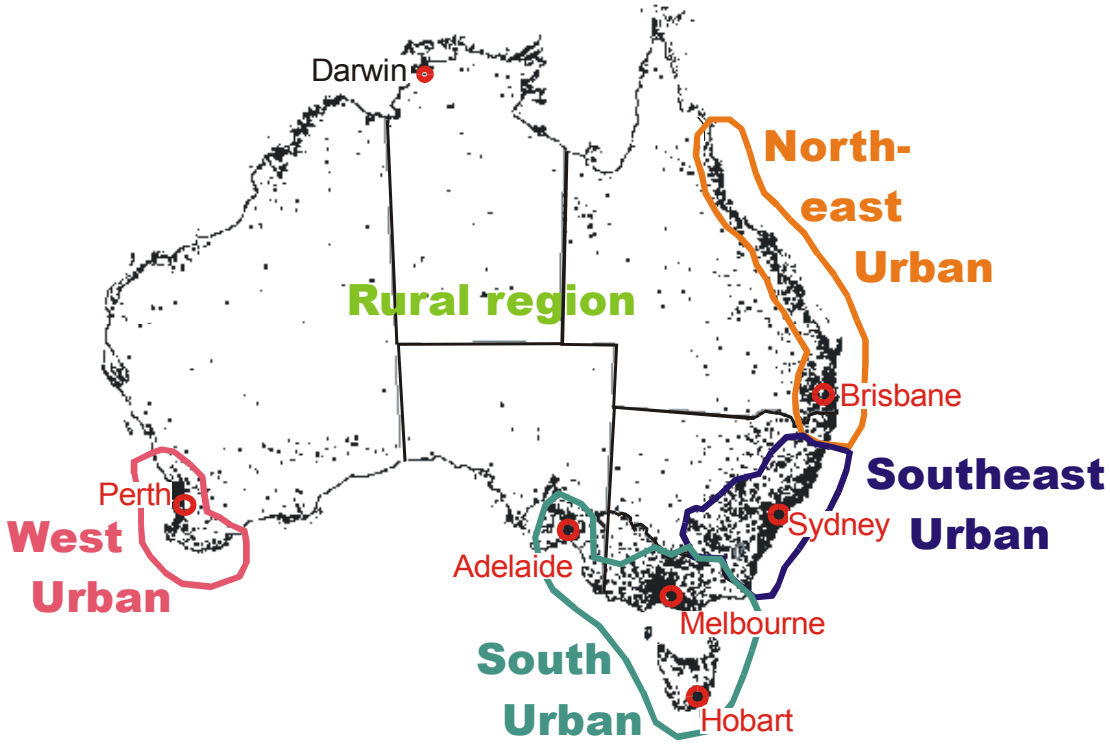

Figure 2.2 The stratification approach indicating the sampling regions of four major population areas of Australia.
The population density of each region is indicated in black (from: Australian Bureau of Statistics, 2002).

In order to obtain the samples from the specified regions, SNP was supplied with a list of postcodes that fell under those regions. This list was compiled using the Official Australian Postcode Map, which was visibly searched and postcodes were allocated as Northeast, South, Southeast, West or Rural.

It was intended that the number of samples collected for each stratum would be 200. From the original project plan it had been calculated that a minimum of 30 samples per pool would be required. 200 samples were not collected in all pools due to difficulties obtaining samples in some of the rural areas particularly in under 16 year age groups and young males. 9,090 individual samples were collected for pooling.

The samples used in this study were obtained from de-identified stored sera that had been collected as part of the routine pathology service. The oldest sample was collected on 27 November 2002 and the most recent sample was collected on 15 April 2003.

Stored sera were collected within each stratum until a maximum of 200 samples were achieved or the sample list was exhausted, for each stratum.

For each sample listed under the five regions, EnTox was provided with the date of birth, postcode, collection date and a laboratory identification number. This sample list was examined for errors in the date of birth, the collection date and the postcodes. Where possible, any samples with incorrect associated data were replaced. All dates of birth were examined to ensure that the year was a sensible 4 digit number. For the date of birth, some samples had the specific date, month and year of birth whereas others had only the year listed. For this reason, it was decided to use only the year of birth to determine the age of the sample donor at the time of collection. Table 2.5 shows the year of birth included in each age range.

Table 2.5 Year of birth included in each age range.

Age Determination
<16 years (born 1987-2002 inclusive)
16-30 years (born 1972-1986 inclusive)
31-45 years (born 1957-1971 inclusive)
46-60 years (born 1942-1956 inclusive)
>60 years of age (born in 1941 or earlier)

Postcodes were examined for two reasons; firstly, to ensure that the postcode listed in the sample list for a certain region actually fell in that region and secondly, to ensure that the postcode was an existent Australian postcode. This was carried out using the Australia Post website Postcode Search and the Official Australian Postcode Map.

Any samples that were identified as having a postcode that was non-existent or a date of birth that was incorrect were removed from the original sample list. Where possible these were replaced with another sample. This was achieved for some but not all strata.

2.2.1 Pooling of Samples

Once the definitive list of samples was obtained, SNP retrieved the samples for each stratum. Each stratum was divided into two pools, with 100 samples in each pool. The 200 samples were divided into two by randomly taking one sample and placing it in Pool 1 and then placing the next one in Pool 2. The 200 samples were not placed in any kind of order by way of date of birth, postcode or collection date prior to random allocation to Pool 1 or 2.

For adult samples, 1ml of each of the 100 samples was used and placed into 100 ml solvent rinsed Schott bottles. For samples in the <16 years old group, the stored volume may have been less than 0.5 ml and so for these samples, the entire sample volume was pooled. Based on the expected variability of the concentration of dioxin-like compounds in blood and the expected minimum volume of 1 ml available for samples obtained from <16 years age groups, a minimum of 30 individual samples was included in each pool. Note that for many of the samples obtained for <16 years age group, the volume available was less than 1 ml and, hence, the minimum number of samples was increased to compensate for the reduced volume (refer section 3.1 and Table 3.1).

2.3 Ethics

The project was submitted to The University of Queensland Medical Research Ethics Committee Approval and approval was obtained on 20 September 2002. The project

was allocated Clearance Number 2002000656. A copy of the Ethics approval is given in Appendix C.

2.4 Sample storage and shipping

Prior to shipping, samples were stored at -30 °C in an alarmed freezer at EnTox. Samples were air freighted frozen on dry ice to either ERGO in Hamburg, Germany or to Dr Jake Ryan, Health Canada Health Products and Food Branch Banting 2203D, Ross Ave Ottawa, Ontario K1A 0L2 CANADA. Samples were received by both laboratories frozen and in good condition.

3. The concentration of dioxin-like chemicals in the blood serum of the Australian population

3.1 Sample Collection

The actual number of samples collected in each stratum is shown in Table 3.1. Collection of samples for the <16 years age groups for males and females in the south and west urban regions was more difficult than in other regions. In addition to this, a smaller volume of samples is often collected in younger children compared to adults and this meant that the volume available was not sufficient for the analysis of two pools. For these reasons only one pool was analysed in each of these strata. It is also important to note that for the <16 years age group, in order to maximise the volume of blood available, the minimum volume used for the older groups was not applied and the entire sample volume was pooled.

Table 3.1 Numbers of Individual Samples collected for each stratum.

Age & Gender	Northeast	Southeast	South	West	Rural
<16yrs Female	200	146	68	24*	166
Male	200	136	66	28*	154
16-30yrs Female	200	200	200	200	198
Male	200	200	200	122	196
31-45yrs Female	198	200	200	200	200
Male	200	200	196	200	200
46-60yrs Female	200	198	200	200	200
Male	200	198	200	200	196
>60yrs Female	200	200	200	200	200
Male	200	200	198	200	202
Totals	**1998**	**1878**	**1728**	**1574**	**1912**
TOTAL					**9090**

* The number of individual samples collected was less than 30 and, hence, were analysed as a single not duplicate pool.

3.1.1 Age of Participants

The average age and the age range for each of the pooled samples is given in Table 3.2. Note that for Northeast females, a record of individual samples in each pool was not made available. For this reason, the calculation of the average age and range was made from all possible samples in that pool.

Human errors during the sample pooling process meant that some samples were incorrectly included in some pools. These are as follows:

For the Northeast region

- males 30-45 (pool 2), included one sample aged 49 years
- males over 60 years (pool 2) contained one sample aged 55 years
- females <16 (pool 2) contained one sample aged 18 years
- females 16-30 years (pool 2) included one sample aged 54 years
- females 30-45 (pool 1), included two samples aged 51 years
- females 45-60 (pool 2) contained one sample aged 16 years

For the Southeast region

- males aged 16-30 (pool 2) included one sample aged 38 years
- females aged 16-30 years (pool 2), included one sample aged 55 years.

For the South region

- females 45-60 (pool 1) included one sample aged 76 years.

For the west region

- all samples were correctly pooled.

For the rural region

- males <16 (pool 2) included one sample aged 49 years
- males 16-30 years (pool 1) included one sample aged 63 and one 67 years
- males 16-30 (pool 2) included one sample aged 47 and one 59 years
- males 30-45 years (pool 2) included one sample aged 49 and one 51 years
- females <16 years (pool 2) included one sample aged 62 years
- females <16 years (pool 1) included one sample aged 63 years.

Despite the pooling errors, the average age for both pools in all strata were remarkably similar and clearly within the accepted age range designated for that group.

Table 3.2 Age of donors in each of the sample pools.

Values indicate the average ages with age range given in parenthesis.

		North-east Urban		South-east Urban		South Urban		West Urban		Rural Area	
Gender	**Age (years)**	**Pool 1**	**Pool 2**	**Pool 1**	**Pool 2**	**Pool 1**	**Pool 2**	**Pool 1**	**Pool 2**	**Pool 1**	**Pool 2**
M	**<16**	10 (1-16)	11 (1-16)	11(2-16)	11 (1-16)	9 (0-16)	N/A	12 (2-16)	N/A	11 (1-16)	11 (0-16)
	16-30	25 (16-31)	25 (17-33)	25 (17-31)	25 (17-31)	26 (16-31)	26 (18-31)	26 (17-31)	24 (16-31)	25 (14-31)	25 (14-33)
	30-45	41 (32-46)	40 (32-46)	40 (32-46)	40 (32-46)	39 (32-46)	39 (32-46)	40 (32-46)	39 (32-46)	40 (32-46)	40 (29-46)
	45-60	55 (47-61)	55 (47-61)	54 (47-61)	55 (47-61)	55 (47-61)	55 (47-61)	55 (47-61)	55 (47-61)	54 (44-61)	55 (47-62)
	>60	75 (62-95)	75 (62-91)	72 (62-85)	73 (62-91)	75 (62-100)	75 (62-95)	74 (62-94)	73 (62-102)	73 (62-87)	73 (62-91)
F	**<16**	12 (1-16)	12 (1-16)	12 (1-16)	12 (2-16)	11 (0-16)	N/A	13 (2-16)	N/A	12 (1-16)	13 (1-16)
	16-30	26 (17-33)	27 (17-31)	26 (17-31)	26 (17-31)	27 (18-31)	27 (17-31)	25 (17-31)	25 (17-31)	26 (17-31)	25 (17-31)
	30-45	40 (32-46)	38 (30-46)	39 (29-46)	40 (32-47)	39 (30-46)	39 (32-46)	39 (32-46)	38 (32-46)	40 (24-46)	40 (32-46)
	45-60	54 (47-61)*		55 (47-61)	54 (47-61)	55 (48-61)	55 (47-61)	53 (47-61)	54 (47-61)	54 (44-61)	54 (45-61)
	>60	76 (62-95)	75 (62-93)	75 (62-93)	74 (62-93)	75 (63-92)	76 (62-95)	75 (59-96)	76 (62-95)	75 (62-94)	75 (60-91)

N/A - not applicable as there was no sample for this pool.

- data to calculate separate mean for each pool is not available.

3.2 Sample Analysis

3.2.1 Analytical Methodology

Analysis of the samples was undertaken by two laboratories. All pooled samples were analysed by ERGO Germany. Analysis of duplicate quality control samples was undertaken by Health Canada. Full details of the analytical methodology are given in Appendix D for Health Canada and Appendix E for ERGO. There is a graphical presentation of the results of quality assurance for human blood samples (QA-pool) in the ERGO Appendix E (pp.12-13.).

3.3 Quality Control and Quality Assurance

3.3.1 Overview

Internal measures:

- regular chemicals and glassware checks (blanks), once a block of 4/6/10 samples
- regular checks of so called instrument blanks (GC/MS)
- regular checks of quality control samples (e.g. blood pools) (GC/MS)
- daily calibration verification tests
- regular GC performance tests (separation, retention windows)
- identification based on definite abundance ratio and retention time criteria, with the use of internal and external standards
- quantification based on the isotope dilution method with the use of internal and external standards
- regular method performance checks by analysing control samples of known PCDD/PCDF concentrations
- daily MS performance checks to control the resolution and sensitivity.

External measures:

- regular participation in inter-laboratory quality control studies
- exchange and control measurements of standards with other qualified laboratories.

3.3.2 Inter-laboratory Calibration

Ten duplicate pooled blood samples representing 10% of the total number of pools were analysed by Health Canada for inter-laboratory calibration. Pooled samples from males and females in the 31-45 year age group representing all regions were sent to both laboratories. Figure 3.1 shows the results obtained from Health Canada compared with those from ERGO. The variation between average values from the inter-laboratory comparisons is about 9% while the average variation between duplicate pools analysed by ERGO pools is approximately 10%. Therefore, the differences between the inter-laboratory comparisons are smaller than the differences observed between the two pools analysed by the same laboratory. Figure 3.1 shows the results from the inter-laboratory comparisons. Considering the similarity of the values between the two laboratories, these differences are acceptable.

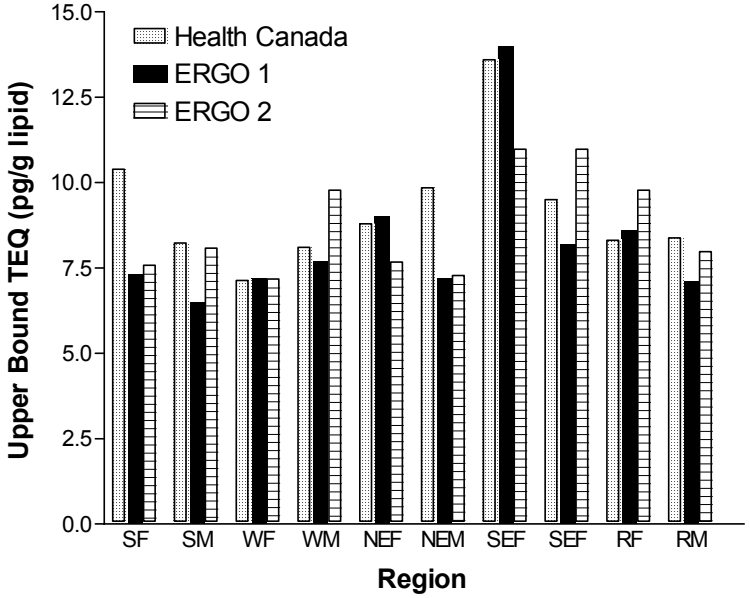

Figure 3.1 Results of inter-laboratory analysis from Health Canada and ERGO.
All samples are from the 31-45 years age group. (S=South, W=West, NE=Northeast, SE=Southeast, R=Rural, M=Male and F=Female)

3.3.3 Analytical Reproducibility

In order to determine the analytical reproducibility of the results obtained by ERGO, four pools were selected for duplicate analysis. These were Southeast females >60 years, Pool I (SEF>60I); Southeast females >60 years, Pool II (SEF>60II) West males 31-45 years, Pool 1 (WM30-45I); Northeast females 46-60 years, Pool 1 (NEF46-60I) and Northeast females 46-60 years Pool II (NEF46-60II). Each duplicate analysis was treated as if they were two different samples. The complete analytical procedure was performed on each and, hence, a separate sample was taken and extracted prior to analysis. The average upper bound TEQ (pg g^{-1} lipid) (± standard deviation) and the TEQ for each analysis for all pools are given in Table 3.3. Full congener profiles are given in Appendix E. These results indicate that the reproducibility of duplicate analyses results from ERGO were acceptable.

Table 3.3 Analytical reproducibility of ERGO data.

Values represent the mean and standard deviation of two separate analyses of each sample from three pools.

Pool Region/Gender/Age	Mean TEQ ± SD (pg g^{-1} lipid)	TEQ Analysis 1 (pg g^{-1} lipid)	TEQ Analysis 2 (pg g^{-1} lipid)
SE F>60I	21.6 ± 2.30	23.3	20.0
SE F>60I	30.4 ± 13.8	40.2	20.8
WM 31-45 II	10.3 ± 4.40	7.2	13.4
NEF46-60I	11.1 ± 1.40	13.1	11.1
NEF46-60II	10.4 ± 0.34	10.2	10.7

3.3.4 Replication Between Pools

Comparisons of TEQ values (pg g^{-1} lipid) from duplicate pools were made using normalised differences. An explanation of the meaning and significance of mean normalised difference is given in Section 2.1.1 Box 1. Normalised differences between upper bound total TEQ (pg g^{-1} lipid) obtained for duplicate pools for all strata ranged from 0-32% and are shown in Table 3.4.

Table 3.4 Normalised difference between TEQs for duplicate pools for all strata.

Region	Age (years)	P 1 (TEQ) pg g^{-1} lipid	P 2 (TEQ) pg g^{-1} lipid	ND (%)
South East Females	<16	6.3	7.3	14.7
	16-30	7.1	8.1	13.2
	31-45	14	11	24.0
	46-60	15	16	6.5
	>60	25	28	11.3
South East Males	<16	8	5.8	31.9
	16-30	5.9	7.6	25.2
	31-45	8.2	11	29.2
	46-60	13	14	7.4
	>60	22	18	20.0
Rural Females	<16	8.3	6.8	19.9
	16-30	6.1	6.5	6.3
	31-45	8.6	9.8	13.0
	46-60	12	12	0.0
	>60	22	27	20.4
Rural Males	<16	6.5	6	8.0
	16-30	5.3	6.6	21.8
	31-45	7.1	8	11.9
	46-60	12	12	0.0
	>60	16	19	17.1
West Females	<16	5.9		N/A
	16-30	4.8	5.3	9.9
	31-45	7.2	7.2	0.0
	46-60	9.7	9.9	2.0
	>60	19	21	10.0
West Males	<16	6.5		N/A
	16-30	4.7	4.9	4.2
	31-45	7.7	9.8	24.0
	46-60	11	10	9.5
	>60	18	19	5.4
South Females	<16	4.6		N/A
	16-30	6	4.9	20.2
	31-45	7.3	7.6	4.0
	46-60	11	10	9.5
	>60	21	19	10.0
South Males	<16	6.1		N/A
	16-30	6	5.4	10.5
	31-45	6.5	8.1	21.9
	46-60	14	12	15.4
	>60	19	20	5.1
North East Females	<16	5.5	5.3	3.7
	16-30	5.8	6.1	5.0
	31-45	9	7.7	15.6
	46-60	14	12	15.4
	>60	25	22	12.8
North East Males	<16	5.9	5.6	5.2
	16-30	5.8	5	14.8
	31-45	7.2	7.3	1.4
	46-60	12	15	22.2
	>60	17	19	11.1

3.4 Levels of dioxin-like chemicals in the serum of the Australian population

3.4.1 Overall Evaluation of PCDDs and PCDFs

Data sheets for all samples analysed are presented in Appendix F. PCDD/PCDFs and PCBs were detectable in all 100 blood samples, 4 pools from the pilot study and 96 pools from the core study. In the data tables shown in Appendix F, the levels of PCDDs and PCDFs in each sample are expressed as upper and lower bound TEQ. In the body of the text all values presented represent upper bound values. An explanation of upper and lower bound values is given in Box 2. Lipid content was measured in all pooled samples and gave an average lipid concentration of 0.60% ± 0.07. The mean and median levels expressed as upper bound TEQ for all pooled samples were 10.9 and 8.3 pg TEQ g^{-1} lipid, respectively. For males and females, the mean levels were 6.5 and 7.2 pg TEQ g^{-1} lipid, respectively. Figure 3.2 shows the average upper bound for total TEQ for all pooled samples.

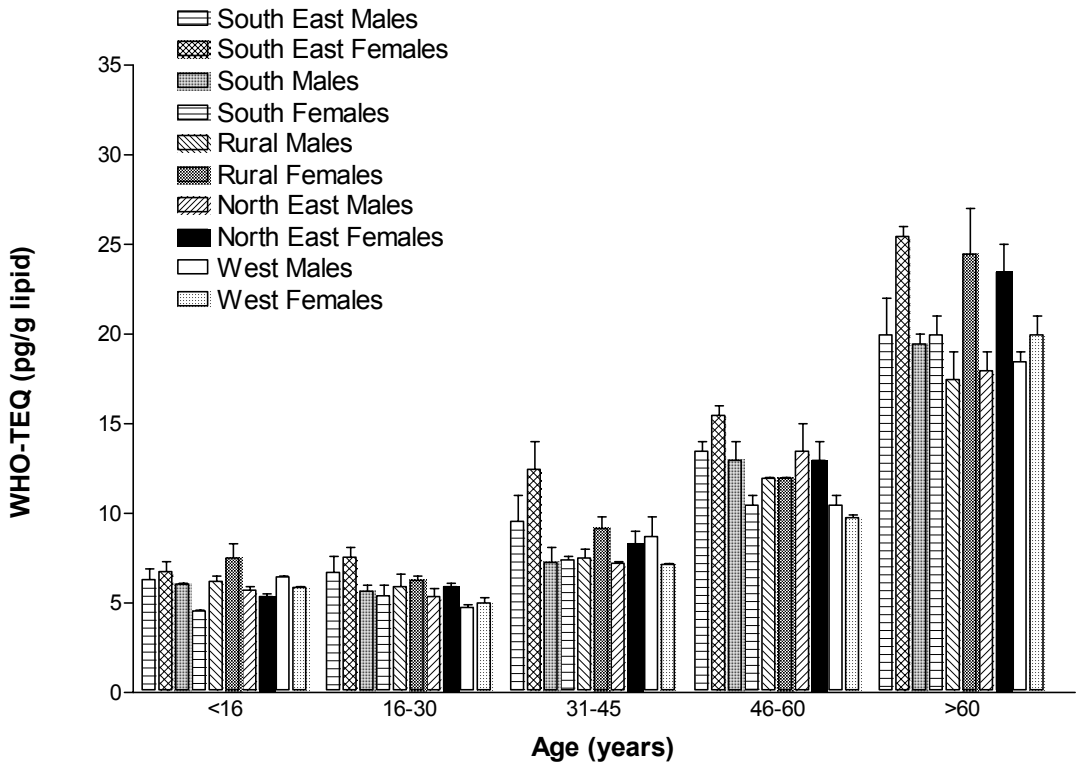

Figure 3.2 Upper bound TEQ in the serum of representative groups of the Australian population.

The levels expressed as TEQ varied by a factor of 6.1 from a minimum of 4.6 pg TEQ g^{-1} lipid detected in South urban females <16 females pool I and II to maximum value of 28 pg TEQ g^{-1} lipid in Southeast urban >60 females pool II. For PCDD/PCDFs, the levels expressed as TEQ varied by a factor of 5.8 from a minimum value of 2.9 pg TEQ g^{-1} lipid detected in Southeast females <16 years pool II to 17 pg TEQ g^{-1} lipid detected in Southeast females >60 pool II.

For PCBs alone, TEQ based on PCB, varied by a factor 9.2 from a minimum value of 1.3 pg TEQ g^{-1} lipid detected in a pool of South region females 16-30 years pool II to a

maximum value of 12 pg TEQ g^{-1} lipid detected in a pool of rural females >60 years pool II.

For PCDD/PCDFs and PCBs the data indicates that on a TEQ basis there is little difference between the upper and lower bound results indicating negligible impact of non-detected congeners. Table 3.5 gives the upper bound for TEQ for all analysed pools from the five age groups and regions for males and females. Note that four strata were analysed as a single rather than duplicate pools because of insufficient volume. These were South urban and west urban <16, males and females.

Table 3.5 Australian serum upper bound TEQ (pg g^{-1} lipid), (PCDD, PCDF and PCB) Pool 1 and 2 by region, gender and age.

		Northeast Urban		Southeast Urban		South Urban		West Urban		Rural Area	
Gender	Age	P 1	P 2	P 1	P 2	P 1	P 2	P 1	P 2	P 1	P 2
M	<16	5.9	5.6	8	5.8	6.1	N/A	6.5	N/A	6.5	6
	Mean (SD)	5.8 (0.2)		6.9 (1.6)		N/A		N/A		6.3 (0.4)	
	16-30	5.8	5	5.9	7.6	6	5.4	4.7	4.9	5.3	6.6
	Mean (SD)	5.4 (0.6)		6.8 (1.2)		5.7 (0.4)		4.8 (0.1)		6.0 (0.9)	
	31-45	7.2	7.3	8.2	11	6.5	8.1	7.7	9.8	7.1	8
	Mean (SD)	7.3 (0.1)		9.6 (2.0)		7.3 (1.1)		8.8 (1.5)		7.6 (0.6)	
	46-60	12	15	13	14	14	12	11	10	12	12
	Mean (SD)	13.5 (2.1)		13.5 (0.7)		13.0 (1.4)		10.5 (0.7)		12.0 (0.0)	
	>60	17	19	22	18	19	20	18	19	16	19
	Mean (SD)	18.0 (1.4)		20.0 (2.8)		19.5 (0.7)		18.5 (0.7)		17.5 (2.1)	
F	<16	5.5	5.3	6.3	7.3	4.6	N/A	5.9	N/A	8.3	6.8
	Mean (SD)	5.4 (0.1)		6.8 (0.7)		N/A		N/A		7.6 (1.1)	
	16-30	5.8	6.1	7.1	8.1	6	4.9	4.8	5.3	6.1	6.5
	Mean (SD)	6.0 (0.2)		7.6 (0.7)		5.5 (0.8)		5.1 (0.4)		6.3 (0.3)	
	31-45	9	7.7	14	11	7.3	7.6	7.2	7.2	8.6	9.8
	Mean (SD)	8.4 (0.9)		12.5 (2.1)		7.5 (0.2)		7.2 (0.0)		9.2 (0.8)	
	46-60	14	12	15	16	11	10	9.7	9.9	12	12
	Mean (SD)	13.0 (1.4)		15.5 (0.7)		10.5 (0.7)		9.8 (0.1)		12.0 (0.0)	
	>60	25	22	25	28	21	19	19	21	22	27
	Mean (SD)	23.5 (2.1)		26.5 (2.1)		20.0 (1.4)		20.0 (1.4)		24.5 (3.5)	

Box 2 Calculation of Upper and Lower Bound TEQ Values

Congeners that record concentrations below the detection limit may be expressed as either *upper* or *lower* bound TEQ values and either of the following methods may be used.

***Upper Bound*: The TEQ level of the congener is calculated using the detection limit**

***Lower Bound*: The TEQ level of the congener is calculated using a zero concentration.**

The greater the number of non-detectable congeners, the greater the difference in the upper and lower bound TEQ values.

3.4.2 Homologue and Congener Profiles and Contributions

Table 3.6 shows the mean, maximum, minimum concentrations as well as the percent contribution of each congener to the overall TEQ. Values are expressed in pg g^{-1} lipid, the percent contribution was calculated by dividing the mean congener concentration by the total congener concentration for PCDD/PCDFs and PCBs separately. For dioxins and furans there is dominance in all samples of the higher chlorinated PCDD, OCDD in the overall congener profile. OCDD contributes an average of approximately 80% to the total of all detected congeners. The dominance of OCDD is consistent with that reported in the New Zealand serum study (Buckland et al, 2001)

Despite the dominance of OCDD, its low toxic equivalency factor (TEF) of 0.0001 means that its contribution to the TEQ is minimal (0.2%). In contrast, other compounds that are present in much lower concentrations have much higher TEFs and therefore have much greater contribution to the total TEQ. These include 2,3,7,8-TCDD and 1,2,3,7,8-Penta CDD.

1,2,3,7,8-pentachlorodibenzodioxin and 3,3',4,4',5-pentachlorobiphenyl were the single most relevant components in the congener profile contributing each approximately 20% to the overall TEQ value. 2,3,7,8-TCDD was not detected in 57 of the 96 pooled samples but was consistently close to the detection limit. Overall the PCDD/PCDF congener profile is dominated by higher chlorinated PCDDs whereas concentrations of higher chlorinated PCDFs (Cl6 or greater) were almost exclusively below the limit of detection in all samples.

Table 3.6 Mean, media, maximum and minimum concentrations for all congeners from all pooled samples.

		Concentration in pg/g lipid based				Contribution to TEQ (%)
Congener	No. of positive detects	Mean	Median	Minimum	Maximum	Mean (Range)
2,3,7,8-Tetra-CDD	41	0.9	0.77	0.4	2.3	9.1 (3.8 - 19)
1,2,3,7,8-Penta-CDD	90	2.1	1.8	0.6	5.4	19 (10.9 - 27)
1,2,3,4,7,8-Hexa-CDD	60	2.0	2.0	0.6	6.3	2.0 (0.85 - 3.6)
1,2,3,6,7,8-Hexa-CDD	96	13	10	3.1	38	11 (4.3 - 19)
1,2,3,7,8,9-Hexa-CDD	85	2.4	1.9	0.7	6.6	2.3 (0.59 - 3.5)
1,2,3,4,6,7,8-Hepta-CDD	96	24	20	9.2	66	2.3(1.3 - 4.2)
OCDD	96	250	220	106	670	0.2 (0.13 - 0.44)
2,3,7,8-Tetra-CDF	10	0.5	0.5	0.3	1.0	0.6 (0.17 - 1.4)
1,2,3,7,8-Penta-CDF	16	0.6	0.5	0.3	3.0	0.4 (0.096 - 0.97)
2,3,4,7,8-Penta-CDF	96	1.8	1.5	0.6	4.4	8.1 (5.0 - 11.2)
1,2,3,4,7,8-Hexa-CDF	79	1.6	1.4	0.6	4.1	1.5 (0.88 - 2.4)
1,2,3,6,7,8-Hexa-CDF	85	1.4	1.3	0.5	3.1	1.4 (0.79 - 2.3)
1,2,3,7,8,9-Hexa-CDF	0	n.c.	n.c.	n.c.	n.c.	n.c.
2,3,4,6,7,8-Hexa-CDF	9	1.3	1.0	0.9	3.6	1.6 (0.45 - 3.8)
1,2,3,4,6,7,8-Hepta-CDF	95	3.0	2.6	0.9	8.2	0.4 (0.059 - 1.1)
1,2,3,4,7,8,9-Hepta-CDF	0	n.c.	n.c.	n.c.	n.c.	n.c.
OCDF	16	3.2	3.0	1.0	15	0.0038 (0.00051 - 0.017)
3,3',4,4'-TCB (77)	0	n.c.	n.c.	n.c.	n.c.	n.c.
3,4,4',5-TCB (81)	33	1.3	1.0	1.0	4.3	0.0016 (0.00049 - 0.0036)
3,3',4,4',5-PeCB (126)	59	19	14	7.0	56	18 (8.8 - 46)
3,3',4,4',5,5'-HxCB (169)	96	13	11	3.4	32	1.2 (0.59 - 2.0)
2,3,3',4,4'-PeCB (105)	96	760	560	140	2700	0.06 (0.21 - 1.2)
2,3,4,4',5-PeCB (114)	96	240	150	46	1050	1.0 (0.39 - 2.1)
2,3',4,4',5-PeCB (118)	96	4500	3200	800	15000	3.6 (1.4 - 7.1)
2',3,4,4',5-PeCB (123)	46	106	94	26	330	0.1 (0.027 - 0.55)
2,3,3',4,4',5-HxCB (156)	96	2200	1700	370	7300	9.2 (3.5 - 16)
2,3,3',4,4',5'-HxCB (157)	96	490	450	97	1500	2.0 (0.90 - 3.4)
2,3',4,4',5,5'-HxCB (167)	92	700	450	120	2300	0.1 (0.02 - 0.11)
2,3,3',4,4',5,5'-HpCB (189)	95	230	190	37	590	0.2 (0.059 - 0.40)
Sum PCDD		**290**	**260**	**120**	**780**	
Sum PCDF		**19**	**17**	**11**	**40**	
Sum PCDD/ PCDF		**300**	**270**	**128**	**790**	
Sum non-ortho PCB		**27**	**26**	**3.4**	**82**	
Sum mono-ortho PCB		**9100**	**6300**	**1800**	**31000**	

if a congener was not detected in some samples, the non-detect value was used
if a congener was not analysed, no value was used.
if a congener was not detected in any samples then no value was calculated (n.c.)

3.5 Evaluation of factors that affect the concentration of dioxins in humans

Exposure to organic pollutants and subsequent effects on total body burden can be influenced by a range of factors. In this study, as outlined in the previous section, samples were selected according to three key parameters. These were (i) age of the donor (ii) region where the sample donor resided at the time of collection (iii) gender of the donor. These are dealt with in the following sections. It should be noted that because de-identified samples were used, there was no means by which the length of residence in a particular area could be verified. This means that sample donors may have only resided in the area from which the sample was collected for a short period of time. There was no means by which this could be controlled for in the present study. In addition, the study design did not allow assessment of whether individuals within a

particular pool had been excessively exposed to organic pollutants through either diet or occupation.

3.5.1 Relationship between age and the levels of dioxin-like chemicals in the Australian population

Figures 3.4-3.8 inclusive indicate the effects of age on the levels of PCDD/PCDFs and PCBs in male and female donors from each of the five regions. For all regions, the TEQ increase with age from the 16-30 years age group onwards. The data shown in each figure represents average upper bound TEQ values for two pooled samples obtained for males and females from each of the five regions over the five age groups.

For upper bound TEQ, there is a clear trend of increasing levels of dioxin-like chemicals from the younger strata (6.3 ± 0.4 pg g^{-1} lipid) to the older strata (22.7 ± 0.91 pg g^{-1} lipid) in samples from all regions (Figures 3.4-3.8). Notably, this increase in concentration only applies from the second youngest age group onward whereas no difference was observable between the concentrations in the <16 year old groups (6.3 ± 0.4) and the 16-30 year old groups (6.1 ± 0.3). The relationship between the age of an individual and the levels of dioxin-like chemicals (expressed as pg TEQ g^{-1} lipid) in their blood serum can be described using an exponential equation from approximately the middle of the third decade onwards to at least the eighth decade high age from the equation:

Levels in blood expressed as pg TEQ g^{-1} lipid = 3.3 $\exp^{0.0251age}$ (r^2 = 0.87)

During this period, the above equation can be used to adjust the concentration of dioxin-like chemicals for a given age. Figure 3.3 indicates the relationship between age and the level, expressed as TEQ, of dioxin-like chemicals in blood serum in the Australian population. Table 3.7 shows examples of the expected concentration in Australian blood serum for selected ages, levels were calculated using the formula above.

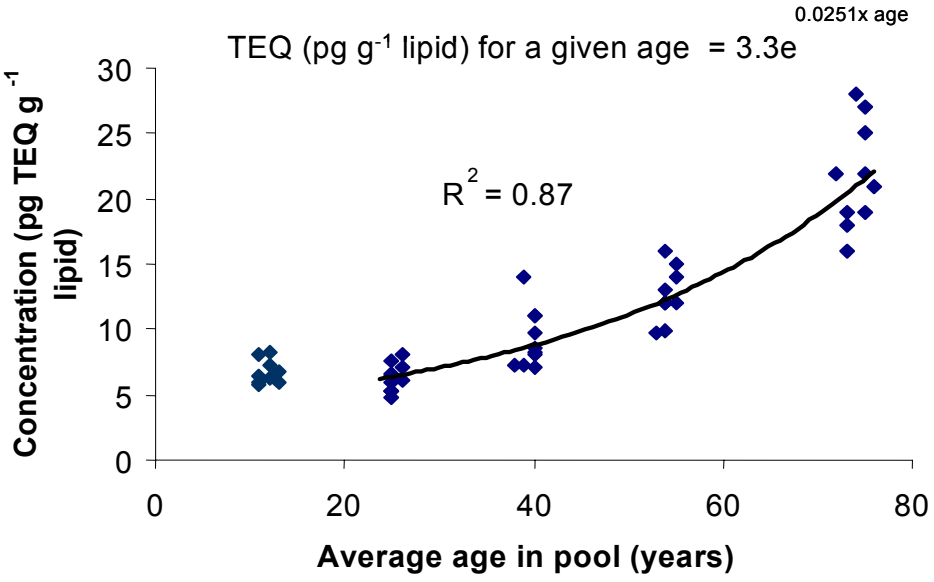

Figure 3.3 Relationship between age and the level of dioxin-like chemicals in the blood.

Table 3.7 Examples of the expected levels of dioxin-like chemicals in the blood serum of Australians over the age range 25-85 years.

Age (Years)	Levels of dioxin (pg TEQ g^{-1} lipid based)
25	6.2
35	7.9
45	10.2
55	13.1
65	16.9
75	21.7
85	27.9

Notably this equation does not appear to hold for persons below the age of 25 since the data suggest that the concentrations were relatively stable between the age groups representing the <16 year old and the 16-30 year old population. The reason for the flattening of the curve is not known but may be due in part to placental transfer during pregnancy and neonatal loading during breastfeeding. Note also that a smaller number of samples was analysed in the <16 years age group compared to the 16-30 years age group and this may also have contributed to the flattening of the curve. Comparison of the <16 years age group with the New Zealand serum study (Buckland et. al., 2001) is difficult because the youngest age group assessed in that study was 15-24 years.

Many studies have found that with increasing age of an individual there is a corresponding increase in the concentrations of dioxin-like chemicals in the blood serum (Buckland et al. 2001). Reasons for such an increase include but may not be limited to:

- continuous accumulation (i.e. steady state is not attained over a life time)
- decrease in historical contamination (i.e. older people were exposed at much higher levels in the 1950s and 1960s when regulations governing the control of industrial emissions were not existing)
- potential differences in metabolism and body fat.

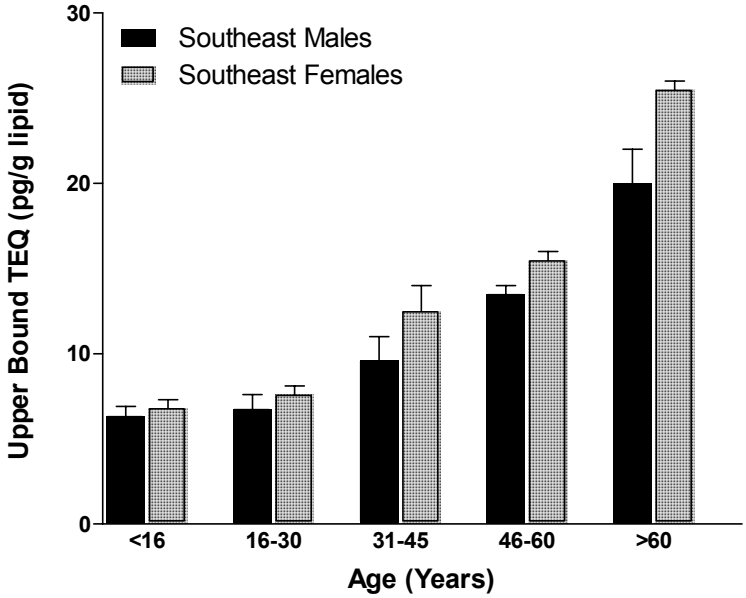

Figure 3.4 Levels of PCDDs, PCDFs and PCBs (total TEQ) in the serum of the Southeast group of the Australian population.

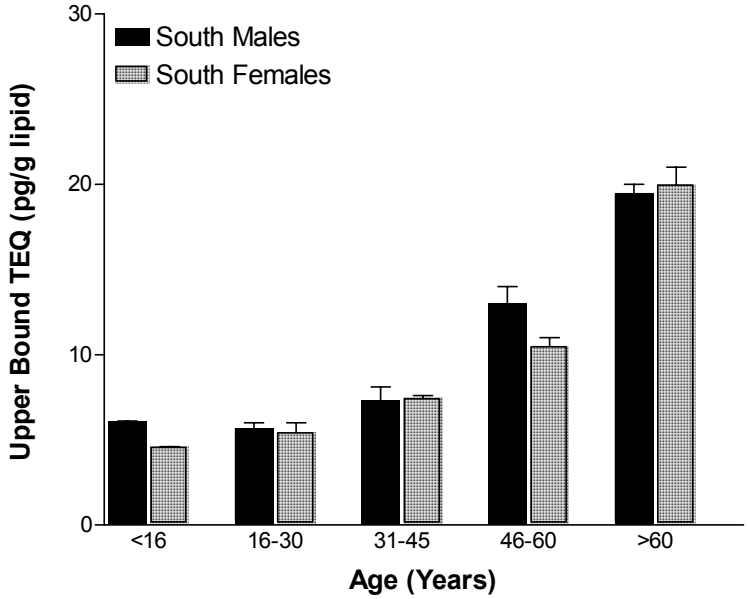

Figure 3.5 Levels of PCDDs, PCDFs and PCBs (total TEQ) in the serum of the South group of the Australian population.

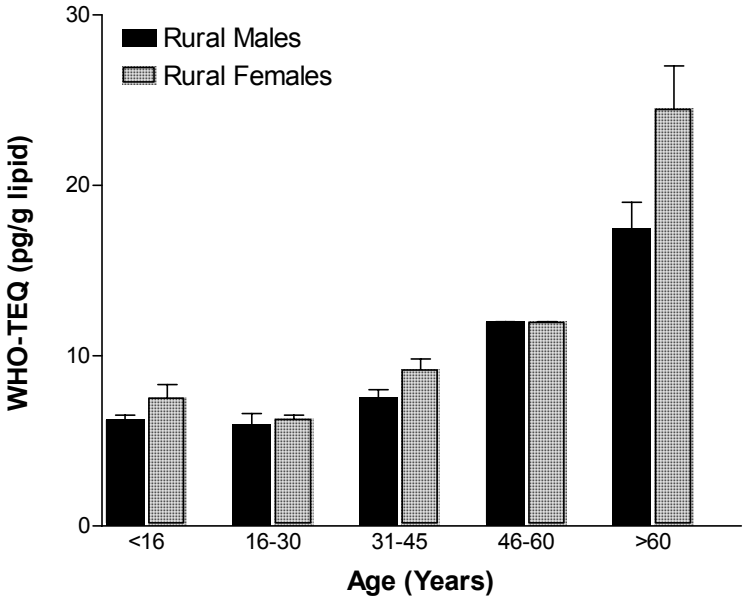

Figure 3.6 Levels of PCDDs, PCDFs and PCBs (total TEQ) in the serum of the Rural group of the Australian population.

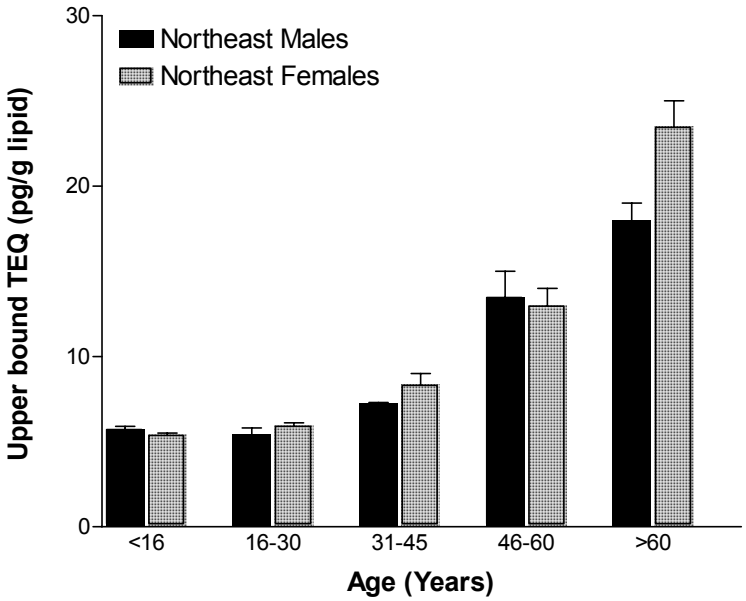

Figure 3.7 Levels of PCDDs, PCDFs and PCBs (total TEQ) in the serum of the Northeast group of the Australian population.

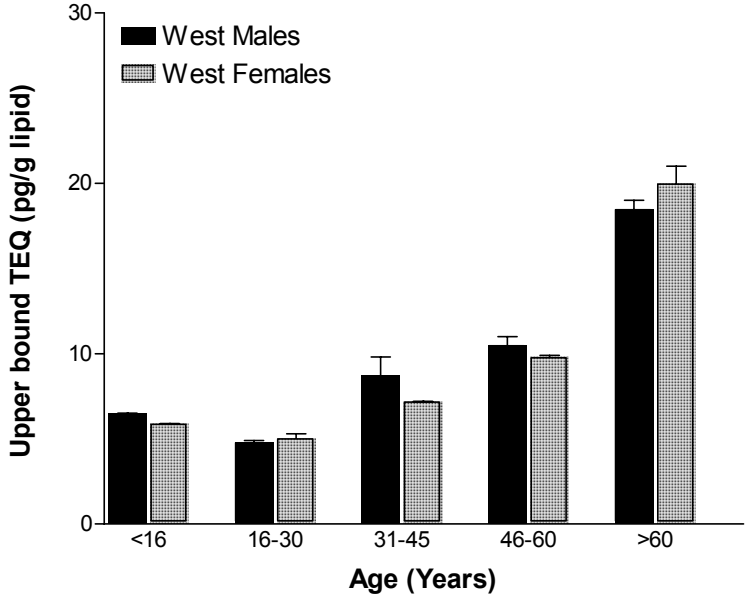

Figure 3.8 Levels of PCDDs, PCDFs and PCBs (total TEQ) in the serum of the West group of the Australian population.

3.5.2 Regional differences in the levels of dioxin-like chemicals in the Australian population

Figures 3.9-3.13 show the TEQ for males and females across each of the five regions and for each of the five age groups. Data shown represent average upper bound TEQ values for two pooled samples obtained for males (A) and females (B) from each age group over the five regions; Southeast (SE), South (S), Rural (R), Northeast urban (N) and West urban (W).

It should be noted that assessment of regional differences is complicated by the fact that the collection of de-identified samples did not enable assessment of where donors have lived during their lifetime and particularly during the last five years. Hence, an individual donor may only have resided in the area that their sample was collected for a short period of time prior to the collection date. One of the advantages of pooling is that any such anomalies are smoothed out such that he data are representative of the true average. This means that it is unlikely that any one individual will have a profound effect on the overall result and it is also likely that the samples will represent a range of resident periods in the area. Little difference was observed in the levels of dioxin-like chemicals across the five regions. Some general trends were noted and these include the following:

- the levels of dioxin-like chemicals across all regions and age groups appear to be very similar
- despite the similarity in levels, for all strata except the <16 years females, the samples from the southeast region exhibit slightly higher levels of dioxin-like chemicals. This seems to be more so for males than for females. The reason for this is not known
- For <16 years females the highest levels of dioxin-like chemicals were found in the rural region.

Regional differences in the levels of dioxin-like chemicals in blood serum were not expected to be observed. The reason for this is that throughout Australia, the food that is consumed is derived from similar sources and hence exposure of individuals through diet is very similar. An accurate assessment or regional differences would require completion of questionnaire and also the determination of an individual's residential patterns over the previous decade. It was not the primary intent of this study to assess regional differences and to have designed a study that were to specifically address this issue would have been far more complex and expensive than the current approach.

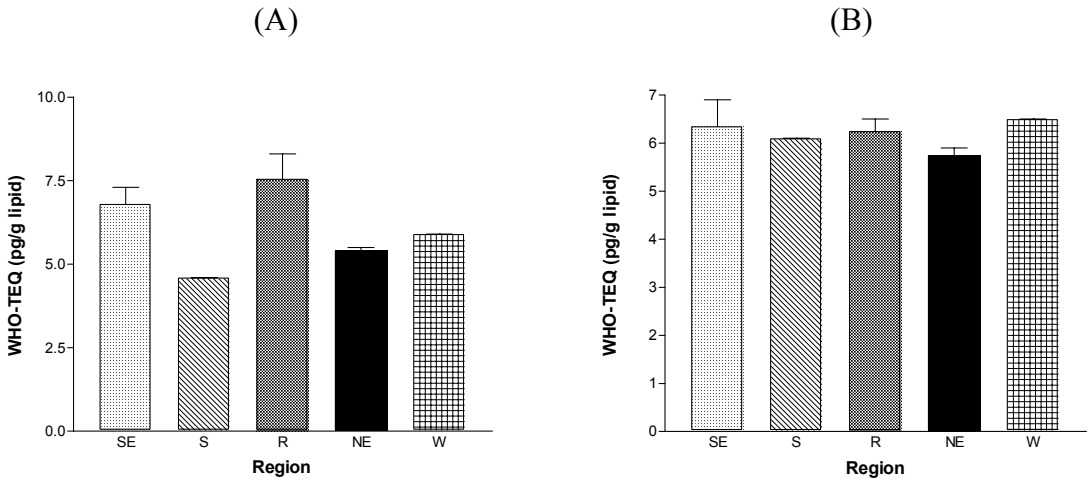

Figure 3.9 Levels of PCDDs, PCDFs and PCBs (total TEQ) in the serum of the < 16 age group of the Australian population.

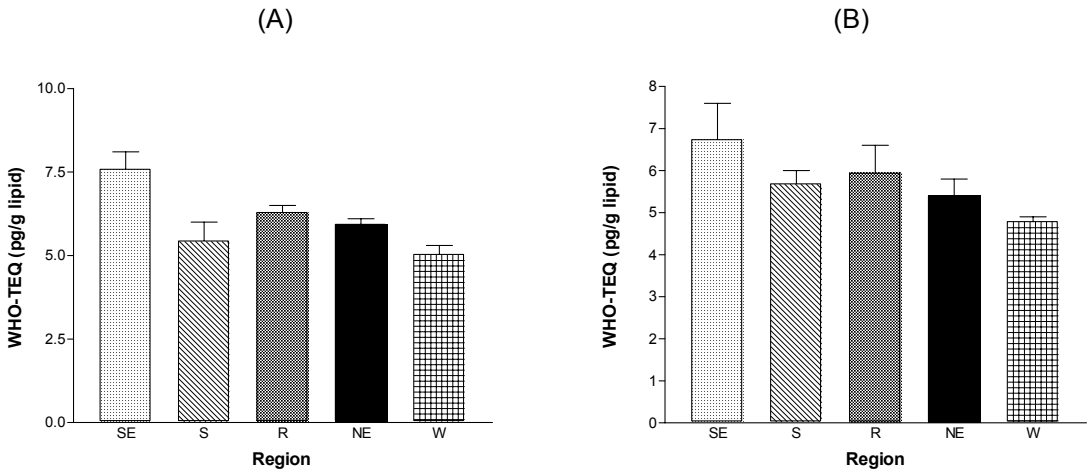

Figure 3.10 Levels of PCDDs, PCDFs and PCBs (total TEQ) in the serum of the 16-30 age group of the Australian population.

(A) (B)

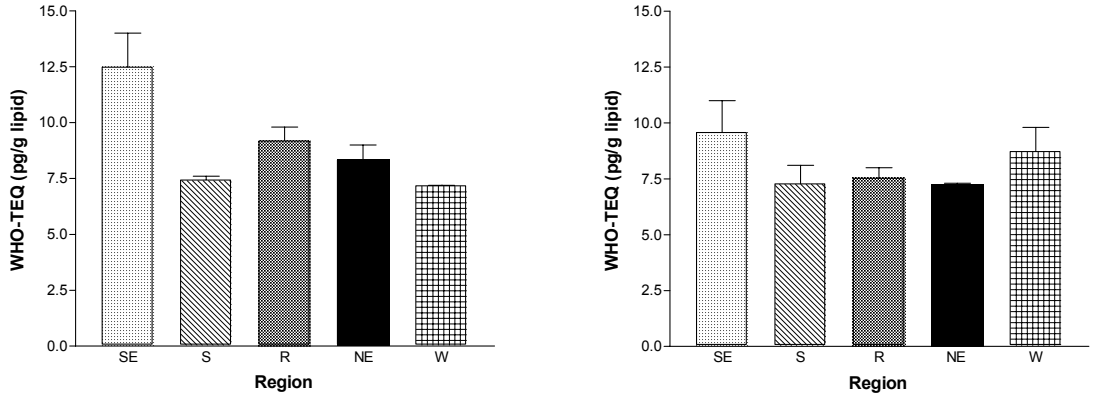

Figure 3.11 Levels of PCDDs, PCDFs and PCBs (total TEQ) in the serum of the 31-45 age group of the Australian population.

(A) (B)

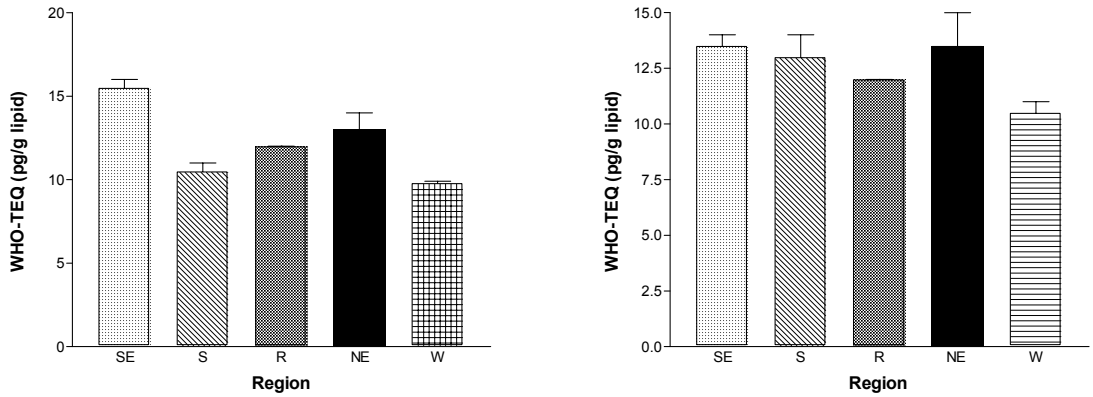

Figure 3.12 Levels of PCDDs, PCDFs and PCBs (total TEQ) in the serum of the 46-60 age group of the Australian population.

(A) (B)

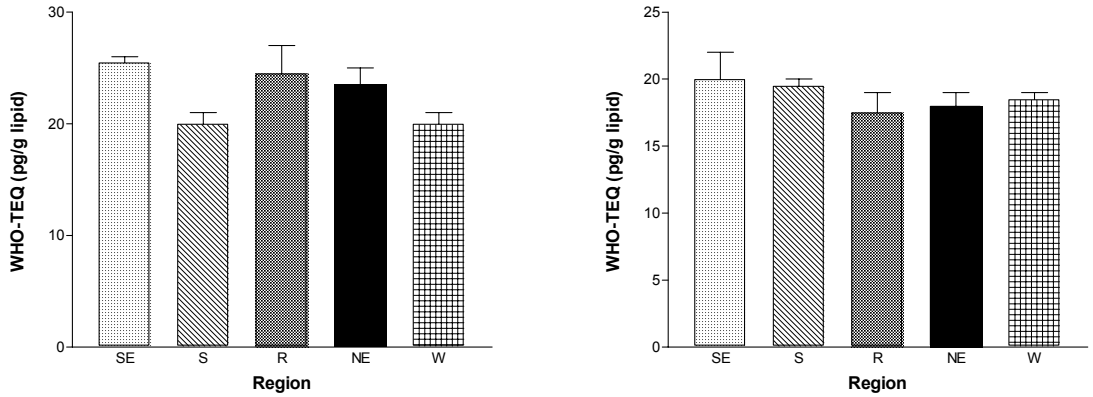

Figure 3.13 Levels of PCDDs, PCDFs and PCBs (total TEQ) in the serum of the > 60 age group of the Australian population.

3.5.3 Effects of gender on the levels dioxin-like chemicals in the Australian population

Figure 3.14 shows a summary of the effects of gender on the TEQ across all regions and for each age group. Data shown represent average upper bound TEQ values based on PCDD/PCDF for pooled samples obtained for males and females from the five age groups across all regions. For total TEQ, the data do not show any systematic difference between males and females. For all age groups, except the >60 years age group there are no differences between males and females. For the >60 years age group, the total TEQ appears to be slightly higher for females than males. This difference cannot be explained by differences in the average age, as these were remarkably similar for males and females (Refer Table 3.2). A similar result has been found in other studies (e.g. Buckland et. al., 2001) and it was postulated that elimination rates might be slower in older women than in men.

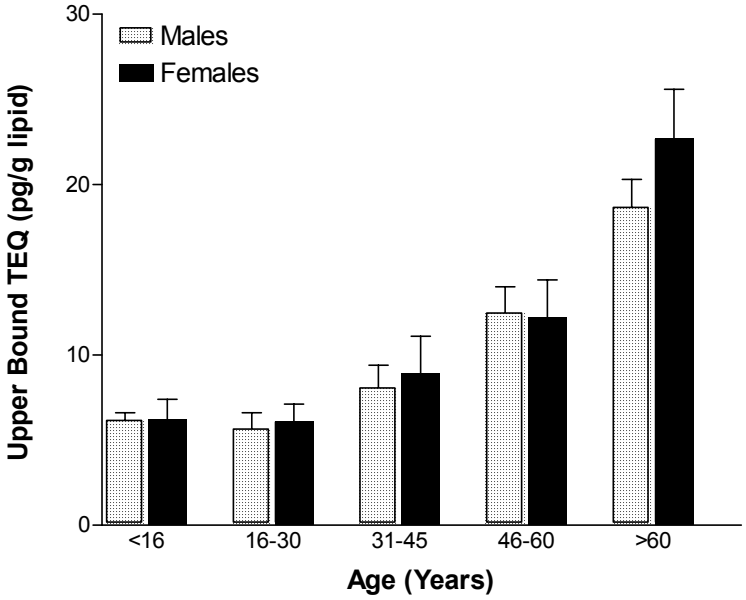

Figure 3.14 Levels of PCDDs, PCDFs and PCBs (total TEQ) in the serum of the Australian population.

3.6 Comparison with International Data

3.6.1 A comparison of Australian levels of PCDDs and PCDFs with other countries

A comparison of the Australian levels of PCDD/PCDFs with those from other countries is depicted in Figure 3.15. Data can be found in tabular form in Appendix G. Table 3.8 shows the mean, upper bound, TEQs for PCDD/PCDFs for all combinations of age groups.

Table 3.8 Mean TEQ PCDD/PCDF, PCB and PCDD/PCDF/PCB for males and females all age groups.

Average Australian (all regions) TEQ PCDD/Fs (pg/g lipid), upper bound)

age	male	female	male and females combined
<16	4.3	3.5	3.9
16-30	3.7	3.7	3.7
31-45	5.3	5.7	5.5
46-60	7.8	7.8	7.8
>60	11.2	14.6	12.9
<16 and 16-30	4	3.6	3.8
<16, 16-30 and 31-45	4.4	4.3	4.4
<16, 16-30, 31-45 and 46-60	5.3	5.3	5.3
ALL AGES COMBINED	6.5	7.2	6.9
16-30 and 31-45	4.5	4.7	4.6
16-30, 31-45 and 46-60	5.6	5.7	5.7
16-30, 31-45, 46-60 and >60	7	7.9	7.5
31-45 and 46-60	6.6	6.7	6.7
31-45, 46-60 and >60	8.1	9.4	8.8
46-60 and >60	9.5	11.2	10.4

Average Australian (all regions) TEQ PCBs (pg/g lipid), upper bound)

age	male	female	male and females combined
<16	2	2.7	2.4
16-30	2	2.4	2.2
31-45	2.7	3.2	3
46-60	4.7	4.4	4.6
>60	7.3	8.6	8
<16 and 16-30	2	2.5	2.3
<16, 16-30 and 31-45	2.2	2.8	2.5
<16, 16-30, 31-45 and 46-60	2.9	3.2	3.1
ALL AGES COMBINED	3.8	4.3	4.1
16-30 and 31-45	2.3	2.8	2.6
16-30, 31-45 and 46-60	3.1	3.3	3.2
16-30, 31-45, 46-60 and >60	4.2	4.6	4.4
31-45 and 46-60	3.7	3.8	3.8
31-45, 46-60 and >60	4.9	5.4	5.2
46-60 and >60	6	6.5	6.3

Average Australian (all regions) TEQ PCDD/Fs and PCBs (pg/g lipid), upper bound

age	male	female	male and females combined
<16	6.3	6.2	6.3
16-30	5.7	6.1	5.9
31-45	8	8.9	8.5
46-60	12.5	12.2	12.4
>60	18.5	23.2	20.9
<16 and 16-30	6	6.1	6.1
<16, 16-30 and 31-45	6.6	7.1	6.9
<16, 16-30, 31-45 and 46-60	8.2	8.5	8.4
ALL AGES COMBINED	10.3	11.5	11
16-30 and 31-45	6.8	7.5	7.2
16-30, 31-45 and 46-60	8.7	9	8.9
16-30, 31-45, 46-60 and >60	11.2	12.5	11.9
31-45 and 46-60	10.3	10.5	10.5
31-45, 46-60 and >60	13	14.8	14
46-60 and >60	15.5	17.7	16.5

Values were calculated by adding the male and female PCDD/F and PCB values, not by taking and average of the PCDD/F and PCB male and females

North American studies

Tepper et al (1997) investigated PCDD/PCDF levels in individual samples from community residents, low exposure potential workers and high exposure potential workers from a pulp and paper mill in the United States. All participants were males. The serum levels of PCDDs and PCDFs were measured in sixteen community residents or controls with a median total I-TEQ (does not include PCBs) of 19.1 pg g^{-1} (lipid adjusted). Median PCDD/PCDF levels for the control group (734.9 pg g^{-1} lipid adjusted) were found to display 'no appreciable differences' to the other two exposure groups. Variations in I-TEQ were attributed neither to exposure group nor exposure duration, although I-TEQ was positively correlated with age (p=0.001) (Tepper et al. 1997).

Mean TEQs of pre-delivery blood and postpartum blood obtained from five females residing in upstate New York between 1995 and 1996 were 12.1 pg g^{-1} and 10.0 pg g^{-1} respectively. The authors did not state which TEFs were used in this study. The age of the donors was not specified. Results from individual samples in the same study indicated that total PCDD, PCDF and PCDD/PCDF levels were consistently higher in the pre-delivery blood (465, 26.0 and 491pg g^{-1} lipid based) than postpartum blood (301, 23.5 and 324.5 pg g^{-1} lipid based) (Schecter et al. 1998). This study proposed that levels of dioxin were decreasing in the general population within the United States indicating that levels of contamination were declining as TEQ were slightly less than earlier studies.

The results of these studies are higher for males and similar for females compared to those found in our current study where the mean TEQ PCDD/F for males was 6.5 pg g-1 and for females 7.2 pg g^{-1} lipid based. The mean PCDD/PCDF level for males and females, all ages, was 269 and 354 pg g^{-1} lipid based, respectively. The sum concentrations of PCDD and PCDF for males were 250 and 19 pg g^{-1} lipid, respectively, and for females, 336 and 18 pg g^{-1} lipid, respectively. Note that comparison of results from different studies is difficult because the levels of dioxins and furans in the general population appear to be falling. Therefore, temporal effects must be considered when such comparisons are undertaken. (Päpke, et al. 1996, Wittsiepe et al. 2000)

European Studies

In a study of the dioxin levels in exposed and unexposed workers in three sawmills in Finland, a mean I-TEQ (does not include PCBs) level of 50.4 pg g^{-1} lipid-adjusted was found in the unexposed groups from all three sawmills (Kontsas et al. 1998). Participants of this study consisted of males, aged 31 to 52 years old and the mean levels of PCDDs, PCDFs and PCDD/PCDF in the eighteen member unexposed group were 790.1, 102.7 and 893.7pg g^{-1} (lipid adjusted). In comparison, the mean TEQ PCDD/F for Australian males aged 31-60 years is 6.6 pg TEQ g^{-1} (lipid based) and the mean concentrations of PCDD, PCDF and PCDD/PCDF in the 31-60 year group for Australian males were 254, 18 and 272 pg g^{-1} lipid, respectively.

A study in Mataro, Spain by Gonzalez et al. (2000) assessed the levels of PCDDs and PCDFs in 198 subjects, of both genders, aged 18 to 69 years, 97 of whom were unexposed. In 1995, the pooled blood samples had an I-TEQ of 13.4pg g^{-1} fat pre-installation of a municipal solid waste incinerator. In 1997, when the waste incinerator was in operation, the I-TEQ increased to 16.7 pg g^{-1} fat. Despite this the authors note

that the small increase in dioxin blood levels would appear unlikely to have resulted from the commencement of the incinerator as both exposed and unexposed populations had an increased dioxin level. Total PCDD concentration was shown to increase with age in both genders, and as in the current Australian study, was slightly higher in females than in males. In 1995, the sum PCDDs, PCDFs and PCDD/PCDFs in pg g^{-1} fat was 610.3, 27.9 and 640.3, respectively. The authors did not supply the corresponding data for 1997.

Another Spanish study by Jimenez et al. (1996) evaluated the background serum levels of PCDDs and PCDFs in eleven unexposed people living in Madrid in 1993. The donors were aged between 19 and 55 years, with no known occupational exposure to dioxins and related compounds. Average levels were found to be 515.29 pg g^{-1} for PCDDs and 66.73 pg g^{-1} for total PCDFs on a lipid weight basis. Calculated I-TEQ values were 8.78 pg g^{-1} for PCDDs and 6.96 for PCDFs on a lipid weight basis. Total PCDD/PCDF I-TEQ was found to be associated with age (correlation coefficient 0.79, $p<0.01$).

A third study in Tarragona, Spain by Schuhmacher et al. (1999) determined the concentrations of PCDDs and PCDFs in individual plasma samples of 20 'non-occupationally' exposed subjects. Subjects lived in the vicinity of a new hazardous waste incinerator and industrial region with participants aged between 28 and 62 years. There were seven female and 13 male participants. Schuhmacher et al. (1999) cites incinerators of municipal and hazardous waste, as possible sources for human exposure to dioxins through inhalation of emissions. General population exposure is however primarily attributed to ingestion via food. Although the authors do not supply the individual levels of PCDDs and PCDFs the mean I-TEQ PCDD/PCDF value given was 27.0 pg g^{-1} lipid. Results indicate that I-TEQ were higher in women (27.7 pg g^{-1} lipid) than in men (25.2 pg I-TEQ g^{-1} lipid), however, the difference was not statistically significant. A significant correlation ($r=0.565$, $p<0.01$) between the age of the subjects and the levels of PCDD/PCDF in plasma could be observed but no significant differences were found in relation to the specific residential area (urban or industrial).

These studies carried out in Spain all found TEQs for PCDDs and PCDFs to be higher than those found in Australia where PCDD/PCDF TEQ for males all ages was 6.5 pg g^{-1} lipid and for females was 7.2 pg g^{-1} lipid.

Covaci et al. (2001) analysed concentrations of PCDDs and PCDFs in 47 pooled human serum samples from 200 women aged between 50 and 65 years living in two areas of Flanders, Belgium in 1999. TEQ were obtained using two methods, which included the use of a CALUX® bioassay and gas chromatograph analysis. The TEQ values for PCDD, PCDF and PCDD/PCDF from rural and suburban areas were 25.6, 23.2 and 48.6 pg g^{-1} fat, respectively. However, levels of individual congeners and sum values were not specified. Levels in Flemish women (49 pg TEQ g^{-1}) were found to be higher when compared to other countries, relative to other industrialized and neighboring countries and considerably higher when compared to current Australian data which had a mean of 11.1 pg TEQ g^{-1} (lipid based) for the females aged 46-60 and greater than 60 years. No statistical difference in individual PCDD/PCDF concentrations between the rural and suburban areas was found nor was there any statistical difference found between TEQ for the two regions.

In Norway, Johansen et al. (1996) determined the concentrations of PCDDs and PCDFs in whole blood samples from 24 crab consumers and 10 referents. Subjects were males aged 40-54 years of age. The sum PCDDs in pg g^{-1} fat for the referent group was 562.8, sum PCDFs was 72.8 and sum PCDD/ PCDFs was 631.1. The Nordic-TEQs for PCDDs, PCDFs and PCDD/PCDFs were 9.7, 11.4 and 21.4 pg g^{-1} fat, respectively. The mean total TEQPCDD/F for the Australian males in the age group 31-60 years was 6.6 pg TEQ g^{-1} (lipid based) and the sum PCDDs, PCDFs and PCDD/PCDFs were 254, 18 and 272 pg g^{-1} lipid. The authors state that they found no correlation between the level of PCDDs and PCDFs and age.

Wittsiepe et al. (2000) analysed 744 individual whole blood samples collected from Germany in 1989-1998. The gender of the sample donors is not indicated. The mean levels found were 43.7 pg I-TEQ g^{-1} (lipid basis) in 1989, 20.7 pg I-TEQ g^{-1} (lipid basis) in 1997/98 and 35.6 pg g-1 I-TEQ g^{-1} (lipid basis) for the total sample from 1989-1998. The average ages for 1989, 1997/98 and 1989-1998 were 43.6, 44.2 and 43.1 years, respectively. In comparison, the total PCDD/PCDF TEQ for the age range 31-45 years male and female combined, of the current study was 5.5 pg TEQ g^{-1} (lipid base). The authors state that each one-year subset of the collective and the entire collective, shows a positive correlation of the PCDD/PCDF blood levels with age for most of the congeners, the sum values and the calculated toxicity equivalents.

Menzel et al. (1998) reported on the levels of dioxin body burden in exposed and unexposed workers in Germany. For the individual samples of the 16 unexposed workers the median dioxin body burden was 18.5 pg I-TEQ g^{-1} blood fat. The levels for exposed workers were greater than unexposed. The authors did not supply the levels of the individual congeners or the sum totals. The result of this study was similar those of other German studies such as Wittsiepe et al. (2000) in 1997/98, Päpke et al. (1996) and Wuthe et al. (1996).

In another Germany study, Päpke et al. (1996) collected blood samples in 1994 from 134 subjects. The mean total PCDD was 462.8 pg g^{-1}, total PCDFs was 46.0 pg g^{-1} and total PCDD/PCDF 508.8 pg g^{-1}. The I-TEQ was 19.1 pg g^{-1}.

A third study from Germany, Wuthe et al. (1996) measured the blood fat concentrations of PCDDs and PCDFs in pooled samples from children and in individual samples from adults. The sample of children consisted of 142 boys and 144 girls with a mean age of 10 years and a range of 9-12 years. The adult study population consisted of 15 volunteers. The mean sum PCDDs in pg g^{-1} lipid base was 511.2, for sum PCDFs 46.2 and sum PCDD/PCDFs 573.1. The I-TEQ was 18.4 pg g^{-1}. The children were from three different areas in Southern Germany, an urban industrial area, an industrial area within a rural setting and a rural area. The results were available for children who had been a resident in the area for a minimum of 2 years and for those two-year residents who also were born in Germany. The mean sum PCDD in pg g^{-1} lipid based for the three regions, for two-year resident children born in Germany were 290.1, 230.6 and 303.1, respectively. For sum PCDFs this was 28.0, 30.0 and 30.7. For the sum PCDD/PCDF the results were 318.1, 260.6 and 333.8. The I-TEQ PCDD/PCDF was 8.2, 9.0 and 10.1 for the three regions. In the current Australian study the mean TEQ PCDD/F for children aged less than 16 years was 3.9 pg TEQ g^{-1} (lipid based). The sum concentrations of PCDD, PCDF and PCDD/PCDF for children less than 16 years in Australia were 165, 17 and 182 pg g^{-1} lipid, respectively.

Asian Studies

The studies from Asia show that levels of PCDD/PCDFs in human sera compared to the Australian data are higher in Japan and lower in China.

Kumagai S et al. (2002) studied the concentrations of PCDDs and PCDFs in exposed and unexposed workers in Japan. The average age of controls was 42.1 years. The mean serum TEQ of PCDD/PCDFs in the controls was 20.3 pg TEQ g^{-1} lipid. The mean levels of sum PCDDs, PCDFs and PCDD/PCDFs were 361.3, 39.3 and 400.6 pg g^{-1} lipid, respectively. This is in comparison to the Australian levels of sum PCDDs, PCDFs and PCDD/PCDFs for 31-45 year olds, male and females, at 259, 17 and 276 pg g^{-1} lipid.

From a survey of the state of dioxins in human blood conducted by the Environment Agency of Japan in fiscal year 1998 (Udea et al., 1999), mean TEQ (WHO TEF) for PCDDs and PCDFs was 11 pg TEQ g^{-1} fat with a range of 0.91-33 pg TEQ g^{-1} fat. A total of 253 subjects (112 women and 131 men) were included in the study. 234 subjects resided in "normal environmental regions" and nineteen resided in the vicinity of waste incineration facilities. There were no significant differences detected between the regions. The Australian mean TEQPCDD/F for males and females all ages was 6.9 pg TEQ g^{-1} lipid.

Schecter et al. (1996) reported on the concentrations of PCDD and PCDFs in the general population in a city in the Jiangxi province of China. Two pools of human blood were analysed, one from participants 15 to 19 years of age and the other one from participants 35 to 70 years of age. The total dioxin (PCDD/PCDF) TEQ in pg g^{-1}, lipid was 4.8 for 15 to 19 year old group and 6.4 for the 35 to 70 year old group. This is similar to 3.7 pg TEQ g^{-1} (lipid based) for 16-30 years in Australia but lower than 8.7 pg TEQ g^{-1} (lipid based) which was found for the age group 31 years to greater than 60 years. The sum PCDDs, PCDFs and PCDD/PCDFs levels in pg g^{-1} fat were126, 21.7 and 148 for the younger age group and 149.5, 28.2 and 178 for the older age group. The authors noted, that China, in comparison with more industrialised countries had low background levels of dioxins and furans and that this was most likely a result of lower levels of chemical use and environmental contamination. The sum PCDDs, PCDFs and PCDD/PCDFs for the Australian 16-30 years group were 184, 15 and 199 pg g^{-1} lipid and for the 31 to greater than 60 years group, 363, 20 and 383 pg g^{-1} lipid.

New Zealand studies

In New Zealand, Buckland et al (2001), 1,834 samples were analysed for levels of PCDDs and PCDFs. Samples were collected in 1996-97 and were obtained from people aged 15 years and older, Maori and non-Maori, male and female. The samples were pooled according to age, ethnicity, gender and geographic location to obtain 60 pools. The mean PCDD/PCDF concentration across the New Zealand population aged 15 years and older was 12.8 pg TEQ g^{-1} lipid. The weighted mean of sum PCDD/PCDFs excluding limit of detection values was 440 pg g^{-1} lipid basis. The levels of PCDD and PCDF congeners increased from 6.69 pg TEQ g^{-1} lipid for the 15-24 age group to 20.7 pg TEQ g^{-1} for the 65+ age group. In comparison to the Australian data where the mean TEQ for 16-30 year olds was 3.7 and for >60 years olds it was 13 pg TEQ g^{-1} (lipid based).

In another New Zealand study, Hannah et al (1994) reported the levels of PCDDs and PCDFs in blood from 28 unexposed subjects, both male and female. Subjects were in the age range 20-60 years old. The mean TE in pg g^{-1} lipid for all ages and genders was 11.5 compared to 7.5 for male and females combined ages 16 to greater than 60 in the Australian study. The sum PCDDs, PCDFs and PCDD/PCDFs in pg g^{-1} lipid were 841.7, 24.9 and 866.6, respectively. This corresponds with results from Buckland et al.

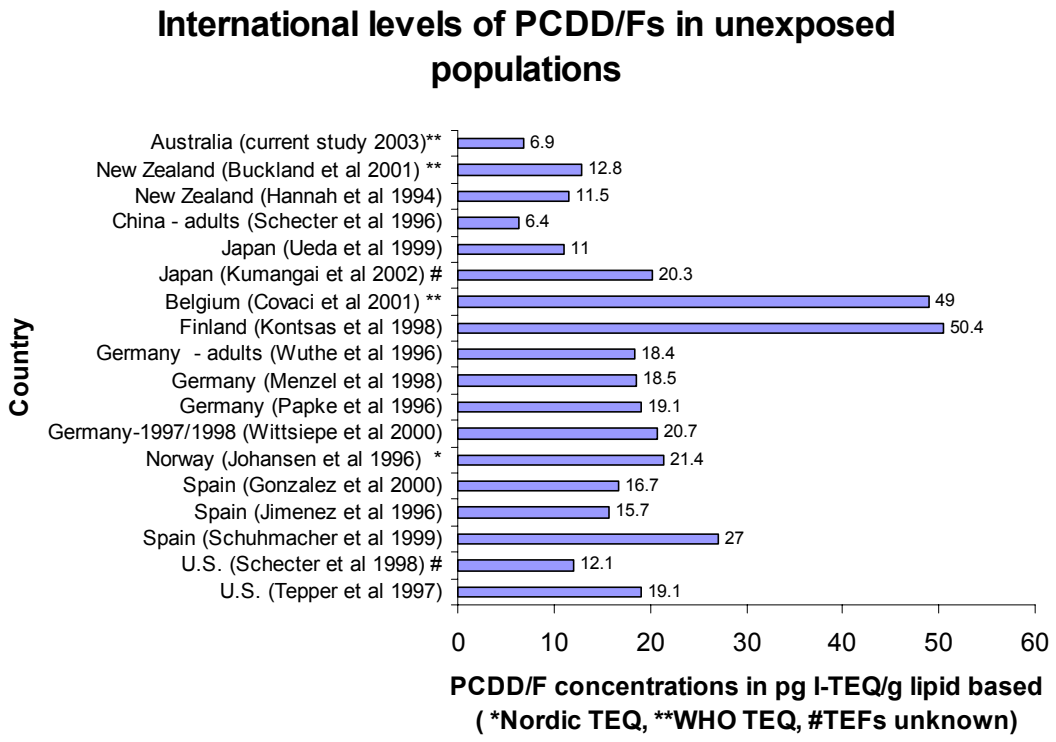

Figure 3.15 International comparison of the concentration (pg I-TEQ g^{-1} lipid based in blood serum) of PCDD/PCDFs.

3.6.2 A comparison of Australian levels of PCBs with other countries

Comparisons of the results of the current study with international data obtained over recent decades should be undertaken with caution. The reasons for this include the following: firstly, it is difficult to make comparisons between the current study data and international data for the levels of PCBs, due to the inclusion of varying PCB congeners chosen for analysis in international studies; and secondly, many authors have not provided TEQ values or the raw data to make these calculations, making direct comparisons with the Australian data difficult. A comparison of the concentration of PCB 126 and 169 in Australia compared to other countries is depicted in Figure 3.16. Data can be found in tabular form in Appendix G. Table 3.8 has mean, upper bound, TEQs for PCBs for all combination of ages. In addition, comparisons of studies that have used samples that have been collected over different time periods are complicated because the levels of PCBs in human populations are known to have fallen over the last decade and continue to fall. Consequently, studies in which the samples were collected five to ten years apart can be expected to report quite different concentration levels as a consequence of this temporal effect. (Malisch and van Leeuwen 2003)

North American studies

Kang, Tepper & Patterson (1997) investigated PCB levels in the serum of sixteen community residents from the United States. The study group also included low exposure potential workers and high exposure potential workers from a pulp and paper mill. All participants were male. The mean concentrations of PCB 126 and 169 were 18 pg g^{-1} and 27 pg g^{-1}, respectively, which was similar to previous results from pooled samples of the general population and in fishermen from Quebec (Kang, Tepper & Patterson 1997). The TEQ for PCB 126 was 1.8 pg TEQ g^{-1} (lipid based) and for PCB 169 it was 0.27 TEQ g^{-1} (lipid based). This is similar to the levels in the current study for males in the Australian population where PCB 126 was 1.58 pg TEQ g^{-1} lipid based and PCB 169 was 0.15 pg TEQ g^{-1} lipid based. Age, body mass index, and consumption of locally caught fish were deemed significant predictors for coplanar PCB (PCB 77, 126 and 169 in this study) levels in human serum.

Greizerstein et al. (1999) determined the levels of PCB congeners in the serum of seven lactating women in New York State, U.S.A. The mean age of the donors was 31 years. The sum of the congeners present above the limit of detection was used to estimate the total PCB concentration that was in the range of 2.6 to 5.8 ng g^{-1} of serum. Of the specific congeners contributing the greatest to the total PCBs in serum was the 'dioxin like' PCB 2,3',4,4',5-penta PCB (IUPAC #118). The average concentration of this congener, normalised for lipid content, was 28.5 ng g^{-1}, whereas in Australian females aged 31-45 years the average concentration of PCB 118 was 3.6 ng g^{-1}. The authors did not report TEQ.

In a study by Schecter, Kassis & Päpke (1998) the pre-delivery and post delivery whole blood of five women living in upstate New York was analysed in the time interval between 1995 and 1996. The mean coplanar PCB concentration (which included PCB 77, 126 and 169) was 35.4 pg g^{-1} (lipid) for pre-delivery blood and 27.6 pg g^{-1} (lipid) for post delivery blood. Mean TEQ (coplanar PCBs only) levels were 2.26 pg g^{-1} for pre-delivery blood and 1.70 pg g^{-1} for postpartum blood. The authors did not state which TEFs were used. The concentration for PCB 126 was 21.7 pg g^{-1} lipid for pre-delivery blood and 16.3 pg g^{-1} lipid for post delivery blood. For PCB 169 the concentration was 9 pg g^{-1} lipid for pre-delivery blood and 6.7 pg g^{-1} lipid for post delivery blood.

Shadel et al. (2001) determined the levels of four PCB congeners (IUPAC #77, 81, 126 and 169) in a group of 150 men and women in Missouri, U.S.A. Subjects had no documented exposure to PCBs. The concentration of PCB 126 was 10.8 pg g^{-1} lipid and for PCB 169 was 15.7 pg g^{-1} lipid. The TEQ for PCB 126 was 1.08 pg TEQ g^{-1} lipid based and for PCB 169 it was 0.16 pg TEQ g^{-1} lipid based. Age was significantly related to these PCBs. The corresponding TEQ values for the Australian population were 1.8 pg and 0.13 pg TEQ g^{-1} (lipid based), respectively. (PCB 126 TEQ calculated from current study using the non-detect value and the maximum value where applicable.)

European studies

In a Finnish study by Kontsas et al. (1998), levels of PCBs in exposed and non-exposed workers in three sawmills were analysed. In this study all participants were male and aged 31 to 52 years. The total sample size for the unexposed group was 18 and the mean levels of 3,3',4,4',5-pentaCB (IUPAC #126) was 69.4 pg g^{-1} lipid-adjusted. The

mean level in plasma at three different sawmills was 82.8 pg g^{-1} lipid-adjusted for 3,3'4,4',5,5'-hexaCB (IUPAC #169). The mean I-TEQs for non-exposed workers was 11.1 and ranged from 8.3 to 14 pg g^{-1} lipid-adjusted for three sawmills. The mean TEQ PCB 126 and TEQ PCB 169 for the Australian study were 1.42 and 0.16 pg TEQ g^{-1} (lipid based) for males aged 31-60 years.

A study in Madrid, Spain by Jimenez et al. (1996) evaluated the background serum levels of non-ortho PCBs in eleven unexposed people during the year 1993. The age of the donors ranged between 19 and 55 years and the mean level found for non-ortho PCBs was 85.47 pg g^{-1} (lipid weight basis). Calculated I-TEQ values were 7.03 pg I-TEQ g^{-1} (lipid based) for coplanar PCBs (PCB 126 and PCB 169). The mean values of PCB 126 and PCB 169 in pg g^{-1} fat weight were 55.21 and 30.26, respectively. In comparison the mean TEQ for coplanar PCBs (126 and 169) for the Australian population was 1.98 pg g^{-1} (lipid based).

Another Spanish study, conducted in Mataro, by Gonzalez et al. (2000) assessed the levels of PCBs in 198 subjects of both genders aged 18 to 69 years. A total of ninety-seven of these subjects were classified as unexposed to pollution from a municipal solid waste incinerator. In 1995, the mean PCB concentration (138 + 153 + 180) was 1.76 ug L^{-1} in the pooled blood samples. In 1997, two years after a municipal solid waste incinerator started functioning, it was 1.94 ug L^{-1}. The authors note that blood levels of PCBs did not vary by place of residence, that is, near or far from the incinerator.

Pauwels et al. (2000) reported the levels of PCBs in 96 serum samples collected from infertile women in Belgium from 1996 to 1998. PCB 28, 52 and 101 were less than the detection limit. The level of four PCBs that were above the detection limit (sum of 118, 138, 153 and 180) in serum was 259 ng g^{-1} (lipid based). All concentrations expressed in this study were on a lipid weight basis and the authors do not provide TEQ values or age of the sample donors.

Covaci et al. (2001) analysed concentrations of PCBs in 47 pooled human serum samples from 200 women between 50 and 65 years living in two areas of Flanders, Belgium in 1999. The women sampled were chosen based on criteria that deemed them not at risk from occupational exposure to PCBs and dioxins and residing in one of two sites, which included a rural and a suburban area of Antwerp. The TEQ value for PCBs was 25.8 pg g^{-1}, fat weight basis and ranged from 20.3 to 32.4 pg g^{-1}. The total PCB concentration (sum of 27 congeners) was 550.6 ng g^{-1} fat and indicator PCB (including IUPAC #28, 52, 101, 138, 153 and 180) was 365.4 ng g^{-1} fat. The mean total TEQ PCB for Australian women aged 46 + years was 6.4 pg TEQ g^{-1} (lipid based) which is less than the level for Belgium women.

Johansen et al. (1996) determined the concentrations of 19 PCB congeners in whole blood samples from 24 crab consumers and 10 referents in a contaminated fjord area in Norway. The subjects included in this study were all males aged between 40-54 years. The sum of PCBs for the referents was 1,344.2 ng g^{-1} fat. The levels of PCB 126 and PCB 169 were 93.4 pg g^{-1} lipid and 70.1 pg g^{-1} lipid, respectively. The TEQ for PCBs taken from a figure is around 45 pg TEQ g^{-1} fat compared to 3.7 pg TEQ g^{-1} (lipid based) for Australian males aged 31-60 years.

Päpke et al. (1996) analysed 104 blood samples in 1994 for PCB 77, 126 and 169 where the mean age of the participants was 38.5 years. The mean concentrations for the three congeners were 16.1, 80.3 and 101.8 pg g^{-1}, respectively. The authors did not provide

TEQ values and also note that the levels of PCB 77 may be affected by an outside contamination.

In Germany, Wuthe et al. (1996) measured the blood fat concentrations of PCBs in pooled samples from children and in individual samples from adults. The sample of children consisted of 142 boys and 144 girls with a mean age of 10 years and in a range of 9-12 years. The adult study population consisted of 15 volunteers. For the adults, the mean values of PCB 126 and 169 were 67.3 and 116.2 ng g^{-1} lipid based, respectively. The children were from three different areas in Southern Germany, an urban industrial area, an industrial area within rural setting and a rural area. Results were available for children who had been a resident in the area for a minimum of 2 years and for those who also were born in Germany. The mean sum value of PCB 126 in for the three regions, for children born in Germany were, 44.8, 45.2 and 41.9 ng g^{-1} lipid based, respectively. For PCB 169 levels were 29.4, 30.3 and 34.0 ng g^{-1} lipid based, respectively. The authors did not calculate TEQ values.

Asian studies

Iida et al. (1999), reported mean TEQ, using WHO-TEF, for coplanar PCBs was 4.9 pg g^{-1} (lipid basis). The study comprised of 50 "normal" Japanese women, approximately 20 years of age, without children. The samples were collected between June 1993 and 1994. The levels of PCB 126 and 169 were 46 and 23 pg g^{-1} lipid, respectively. Australian females aged 16-30 years had a mean non-ortho PCB concentration of 15 pg g^{-1} lipid based and a mean total PCB TEQ of 2.4 pg TEQ g^{-1} (lipid based).

From a survey of "The state of dioxins in human blood" conducted by the Environment Agency of Japan in fiscal year 1998 (Udea et al., 1999), mean TEQ (WHO TEF) for coplanar PCBs was 7.3 pg TEQ g^{-1} fat with a range of 0.33-32 pg TEQ g^{-1} fat. A total of 253 subjects (112 women and 131 men) were included in the study. 234 subjects resided in "normal environmental regions" and nineteen resided in the vicinity of waste incineration facilities. There were no significant differences in PCB levels detected between the regions.

New Zealand studies

In New Zealand, Buckland et al. (2001) had 1,834 samples analysed for levels of PCBs. Samples were obtained from people aged 15 years and older, Maori and non-Maori, male and female. The samples were pooled according to age, ethnicity, gender and geographic location to obtain 60 pools. The weighted mean levels of PCB 126 and 169 were 30 and 20 pg g^{-1} lipid, respectively. The total PCB TEQ (including ½ LODs) was 6.86 pg TEQ g^{-1} lipid based.

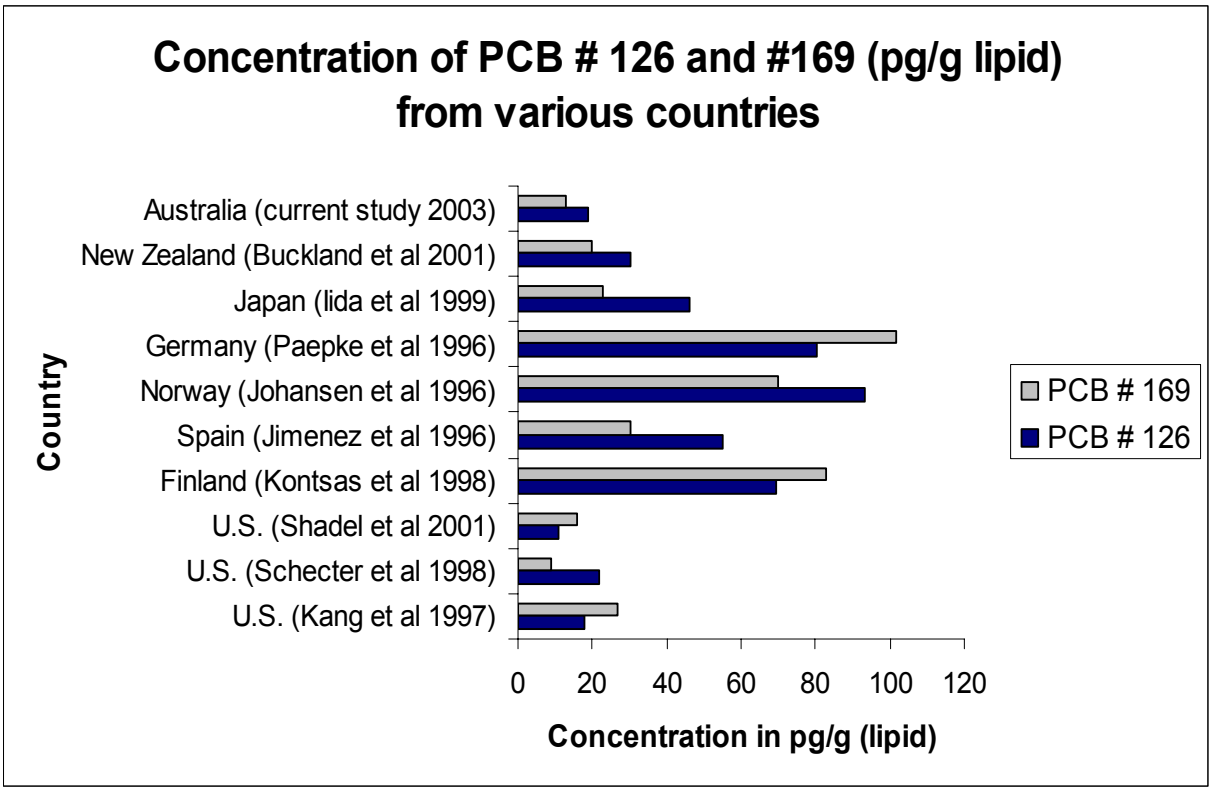

Figure 3.16 International comparison of the concentration (pg g^{-1} lipid in blood serum) of PCB 169 and 126.

4. Summary of Findings

The results of this study provide a measure of the levels of polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and polychlorinated biphenyls (PCBs) in pooled blood serum collected throughout Australia in 2003. Serum samples were collected throughout Australia using Sullivan and Nicolaides Pathology, a pathology company based in Brisbane but with an extensive national network. De-identified samples were selected from surplus pathology samples according to stratification criteria that were provided to Sullivan and Nicolaides staff. These stratification criteria were as follows:

Regional Stratification: 5 regions representing the regional and population distribution of Australians

- Southeast urban
- South urban
- Northeast urban
- West urban
- Rural region encompassing all rural regions of Australia

Age Stratification: 5 age groups

- <16 years
- 16-30 years
- 31-45 years
- 46-60 years
- >60 years

Gender Stratification

- males
- females.

In total 9,090 samples from the 50 strata were collected and pooled to give 96 pools according to the above criteria. An additional 204 samples representing four pools were also assessed as part of a pilot study to determine the suitability of the use of surplus pathology samples for this study.

All pooled samples were sent to ERGO- Forschungsgesellschaft mbH, Hamburg, and 10 duplicate samples were sent to Health Canada, Ottawa, Canada for interlaboratory comparison. Both are laboratories accredited for analytical dioxin analysis.

Dioxin-like chemicals were detected in all strata. Overall the levels in the Australian population are very low by international standards and comparable with, although lower than, those observed in the New Zealand population (Buckland et al, 2001). The mean and median levels expressed as TEQ for all pooled samples were 10.9 and 8.3 pg TEQ g^{-1} lipid, respectively. For males, and females the mean levels were 10.4 and 11.5 pg TEQ g^{-1} lipid, respectively.

No systematic differences were observed in the levels of dioxin-like chemicals in samples collected from males and females. However, slightly higher levels of dioxin-like chemicals were observed in females in the >60 years age group. This result could not be explained on the basis of differences in the mean age between males and females in this group.

The levels of dioxin-like chemicals across the five regions were remarkably similar within each age range. Some general trends were noted and include the following:

- the levels of dioxin-like chemicals across all regions and within each age range appear to be very similar
- Despite the similarity in levels, for all strata except the <16 years females, the samples from the Southeast region exhibit slightly higher levels of dioxin-like chemicals
- For <16 years females the highest levels of dioxin-like chemicals were found in the rural region.

It should be noted that because de-identified samples were used in this study, determination of regional differences was complicated. The use of such samples did not allow any assessment of the length of time an individual had resided in a particular area prior to their sample being collected or recording of either food intake or possible exposure to environmental contaminants in that region.

In contrast to the minimal differences observed in both gender and region a direct relationship of increasing dioxin level with increasing age was observed and could be described by the following equation:

$$\textbf{Levels in blood expressed as pg TEQ } g^{-1} \textbf{ lipid} = 3.3 \exp^{0.0251 age} \qquad (r^2 = 0.87)$$

This relationship was found to hold from approximately 25 years of age until at least the eighth decade and thus during these years it is possible to estimate the level of dioxin-like chemicals in an individual's blood serum.

In summary, the levels of dioxin-like chemicals in the Australian population are low by international standards and are very similar across all regions of Australia within each designated age ranges. The levels of these chemicals increase with age and can be estimated if the age of an individual is known.

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Appendix A Demographic data from samples collected for pilot study

Path ID No.	Age	Collection Date	Ins ID No.	Age	Collection Date	Postcode
SA1	41	03.06.2002	HA1	41	22.08.2002	4101
SA2	36	04.06.2002	HA2	35	31.05.2002	4011
SA3	38	04.06.2002	HA3	38	01.07.2002	4069
SA4	33	04.06.2002	HA4	34	20.12.2001	4124
SA5	33	04.06.2002	HA5	33	21.06.2002	4169
SA6	40	04.06.2002	HA6	41	11.02.2002	4163
SA7	32	04.06.2002	HA7	32	29.01.2002	4031
SA8	40	05.06.2002	HA8	39	07.06.2002	4209
SA9	44	06.06.2002	HA9	44	01.07.2002	4035
SA10	42	06.06.2002	HA10	43	04.03.2002	4207
SA11	42	06.06.2002	HA11	42	06.11.2002	4152
SA12	45	07.06.2002	HA12	45	25.10.2002	4000
SA13	32	11.06.2002	HA13	31	05.12.2001	4103
SA14	32	11.06.2002	HA14	31	27.04.2002	4105
SA15	44	12.06.2002	HA15	44	22.11.2002	4069
SA16	34	12.06.2002	HA16	34	27.09.2002	4064
SA17	37	12.06.2002	HA17	38	16.11.2001	4000
SA18	36	13.06.2002	HA18	36	29.10.2002	4173
SA19	32	13.06.2002	HA19	32	19.06.2002	4161
SA20	43	13.06.2002	HA20	43	31.12.2001	4169
SA21	43	13.06.2002	HA21	42	17.04.2002	4157
SA22	43	14.06.2002	HA22	43	16.05.2002	4113
SA23	34	14.06.2002	HA23	34	17.01.2002	4069
SA24	40	15.06.2002	HA24	41	12.04.2002	4070
SA25	44	17.06.2002	HA25	44	11.02.2002	4053
SA26	35	17.06.2002	HA26	36	30.01.2002	4160
SA27	41	18.06.2002	HA27	41	08.12.2001	4171
SA28	40	18.06.2002	HA28	40	20.03.2002	4178
SA29	33	19.06.2002	HA29	34	05.07.2002	4074
SA30	36	19.06.2002	HA30	36	08.01.2002	4000
SA31	33	19.06.2002	HA31	33	23.11.2002	4178
SA32	32	19.06.2002	HA32	32	25.10.2002	4122
SA33	38	20.06.2002	HA33	38	23.06.2002	4121
SA34	44	20.06.2002	HA34	43	06.08.2002	4105
SA35	42	20.06.2002	HA35	41	12.07.2002	4030
SA36	34	21.06.2002	HA36	35	16.11.2002	4035
SA37	41	21.06.2002	HA37	41	05.11.2002	4061
SA38	33	21.06.2002	HA38	34	21.06.2002	4101
SA39	32	24.06.2002	HA39	31	16.05.2002	4000

SA40	36	24.06.2002	HA40	36	30.08.2002	4121
SA41	36	24.06.2002	HA41	36	16.11.2002	4069
SA42	30	24.06.2002	HA42	30	29.10.2002	4152
SA43	39	24.06.2002	HA43	39	12.08.2002	4122
SA44	43	25.06.2002	HA44	43	31.03.2002	4109
SA45	37	25.06.2002	HA45	37	18.11.2002	4104
SA46	36	25.06.2002	HA46	36	01.12.2001	4122
SA47	37	25.06.2002	HA47	37	09.05.2002	4105
SA48	37	26.06.2002	HA48	37	26.03.2002	4064
SA49	37	26.06.2002	HA49	37	31.10.2002	4053
SA50	44	26.06.2002	HA50	43	24.08.2002	4121
SA51	32	26.06.2002	HA51	33	26.08.2002	4053
SB1	38	27.06.2002	HB1	38	29.01.2002	4030
SB2	31	27.06.2002	HB2	32	17.05.2002	4011
SB3	43	27.06.2002	HB3	43	10.07.2002	4178
SB4	32	27.06.2002	HB4	33	05.02.2002	4171
SB5	32	28.06.2002	HB5	31	05.03.2002	4059
SB6	40	28.06.2002	HB6	39	24.07.2002	4066
SB7	40	01.07.2002	HB7	39	11.09.2002	4074
SB8	39	01.07.2002	HB8	40	30.10.2002	4006
SB9	40	01.07.2002	HB9	41	14.01.2002	4105
SB10	31	02.07.2002	HB10	31	19.11.2002	4121
SB11	33	02.07.2002	HB11	33	13.03.2002	4064
SB12	39	03.07.2002	HB12	40	23.04.2002	4101
SB13	31	03.07.2002	HB13	30	20.12.2001	4178
SB14	39	03.07.2002	HB14	39	20.11.2002	4020
SB15	31	05.07.2002	HB15	31	20.08.2002	4122
SB16	40	06.07.2002	HB16	41	10.09.2002	4115
SB17	37	08.07.2002	HB17	37	07.08.2002	4032
SB18	36	09.07.2002	HB18	36	06.11.2002	4157
SB19	36	10.07.2002	HB19	36	09.09.2002	4110
SB20	40	10.07.2002	HB20	40	28.02.2002	4159
SB21	33	10.07.2002	HB21	33	29.10.2002	4121
SB22	39	10.07.2002	HB22	40	13.09.2002	4051
SB23	35	13.07.2002	HB23	36	23.04.2002	4103
SB24	36	15.07.2002	HB24	35	03.10.2002	4179
SB25	35	15.07.2002	HB25	35	15.10.2002	4065
SB26	39	17.07.2002	HB26	40	30.08.2002	4051
SB27	44	17.07.2002	HB27	44	22.06.2002	4170
SB28	37	18.07.2002	HB28	37	16.04.2002	4207
SB29	38	18.07.2002	HB29	37	22.05.2002	4064
SB30	40	18.07.2002	HB30	41	13.06.2002	4116
SB31	33	18.07.2002	HB31	32	25.10.2002	4035
SB32	43	19.07.2002	HB32	43	20.08.2002	4017

SB33	37	18.07.2002	HB33	37	30.10.2002	4101
SB34	44	19.07.2002	HB34	44	10.01.2001	4069
SB35	34	22.07.2002	HB35	34	02.01.2002	4171
SB36	45	22.07.2002	HB36	44	04.11.2002	4113
SB37	41	22.07.2002	HB37	40	18.02.2002	4179
SB38	34	22.07.2002	HB38	34	22.05.2002	4034
SB39	39	23.07.2002	HB39	38	26.06.2002	4005
SB40	37	23.07.2002	HB40	37	24.10.2002	4171
SB41	33	24.07.2002	HB41	33	25.01.2002	4069
SB42	35	25.07.2002	HB42	34	04.03.2002	4103
SB43	37	26.07.2002	HB43	36	05.03.2002	4113
SB44	34	26.07.2002	HB44	34	15.05.2002	4064
SB45	32	26.07.2002	HB45	31	03.12.2001	4060
SB46	32	26.07.2002	HB46	33	06.11.2001	4065
SB47	37	26.07.2002	HB47	37	30.07.2002	4055
SB48	43	29.07.2002	HB48	42	03.09.2002	4160
SB49	33	29.07.2002	HB49	33	24.09.2002	4207
SB50	43	30.07.2002	HB50	43	16.11.2002	4161
SB51	42	31.07.2002	HB51	42	19.06.2002	4101

Appendix B Results of Pilot Study

ERGO Forschungsgesellschaft mbH, Geierstr.1, 22305 Hamburg, Germany

University of Queensland
Attn. Dr. Jochen Müller
39 Kessels Road
Coopers Plains Qld 4108
Australia

Your sign, your information from	Our sign (Normal.dot)	Name (direct dial)	Date
	Report 2002-0953th.doc	Olaf Päpke (-27)	20.12.2002

University of Queensland
Attn. Dr. Jochen Müller
39 Kessels Road
Coopers Plains Qld 4108
Australia

Dear Dr. Müller,
enclosed please find our Report 2002-0953th.doc. The report was already sent by e-mail.
If you have any further questions, please do not hesitate to contact us.

Best regards

ERGO Forschungsgesellschaft mbH

Olaf Päpke
board member

Thomas Herrmann (Dipl.-Ing.)
manager analytical service

Enclosures

Investigation on polychlorinated Dibenzodioxins and –furans and PCBs in human blood

Client:

University of Queensland
39 Kessels Road
Coopers Plains Qld 4108
Australia

Hamburg, December 20th, 2002

Olaf Päpke Thomas Herrmann (Dipl.-Ing)

1 Order

The order was given in writing on by the client mentioned above.

The order has the following internal project code: A-1033-02-400.

2 Sampling and shipment

The sampling was done resp. organized by the customer. The samples were sent in frozen state by a courier service. The samples arrived frozen in the ERGO laboratory and were stored at –18°C until the beginning of the analyses.

3 Description of sample

Sample code	Client code	Matrix	Receipt of sample	Date of the test performance
H-02-12-0114	Group 1 "HEALTHY"	human blood	06.12.2002	06.12.2002 – 19.12.2002
H-02-12-0115	Group 2 "HEALTHY"	human blood	06.12.2002	06.12.2002 – 19.12.2002
H-02-12-0116	Group 3 "Sick"	human blood	06.12.2002	06.12.2002 – 19.12.2002
H-02-12-0117	Group 4 "Sick"	human blood	06.12.2002	06.12.2002 – 19.12.2002

4 Analytical method

In the following the analytical procedures for the analysis of human blood is shown. We would like to mention, that the measurements are done *by high resolution mass spectrometry (HRMS)*, which guarantees high specificity and high sensitivity.

Prior the extraction following ^{13}C-UL-labeled internal standards are added to the sample:

Internal standards (^{13}C-UL), PCDDs/PCDFs			
PCDDs		**PCDFs**	
2,3,7,8	-Tetra-CDD	2,3,7,8	-Tetra-CDF
1,2,3,7,8	-Penta-CDD	1,2,3,7,8 2,3,4,7,8	-Penta-CDF -Penta-CDF
1,2,3,4,7,8 1,2,3,6,7,8 1,2,3,7,8,9	-Hexa-CDD -Hexa-CDD -Hexa-CDD	1,2,3,4,7,8 1,2,3,6,7,8 1,2,3,7,8,9 2,3,4,6,7,8	-Hexa-CDF -Hexa-CDF -Hexa-CDF -Hexa-CDF
1,2,3,4,6,7,8	-Hepta-CDD	1,2,3,4,6,7,8 1,2,3,4,7,8,9	-Hepta-CDF -Hepta-CDF
1,2,3,4,6,7,8,9	-Octa-CDD	1,2,3,4,6,7,8,9	-Octa-CDF

	Internal standards (^{13}C-UL), PCBs		
	Compound		**IUPAC Code**
Non-ortho PCBs	3,3´,4,4´	-Tetra-CB	PCB 77
	3,4,4´,5	-Tetra-CB	PCB 81
	3,3´,4,4´,5	-Penta-CB	PCB 126
	3,3´,4,4´,5,5´	-Hexa-CB	PCB 169
Mono-ortho PCBs	2,3,3´,4,4´	-Penta-CB	PCB 105
	2,3,4,4´,5	-Penta-CB	PCB 114
	2,3´,4,4´,5	-Penta-CB	PCB 118
	2´,3,4,4´,5	-Penta-CB	PCB 123
	2,3,3´,4,4´,5	-Hexa-CB	PCB 156
	2,3,3´,4,4´,5´	-Hexa-CB	PCB 157
	2,3´,4,4´,5,5´	-Hexa-CB	PCB 167
	2,3,3´,4,4´,5,5´	-Hepta-CB	PCB 189

After spiking, the samples are extracted with appropriate solvents for ultratrace-analyses (e.g. nanograde) by using a solid/lipid extraction.

After performing the gravimetric lipid determination, the clean up is done on a multicolumn system (involving carbon-on-glasfibre or carbon-on-celite for PCDDs/PCDFs and certain PCBs). The measurement is done by means of high resolution gaschromatography and high resolution mass spectrometry (HRGC/HRMS) with VG-AutoSpec and/or Finnigan MAT 95 XL using DB-5 capillary columns.

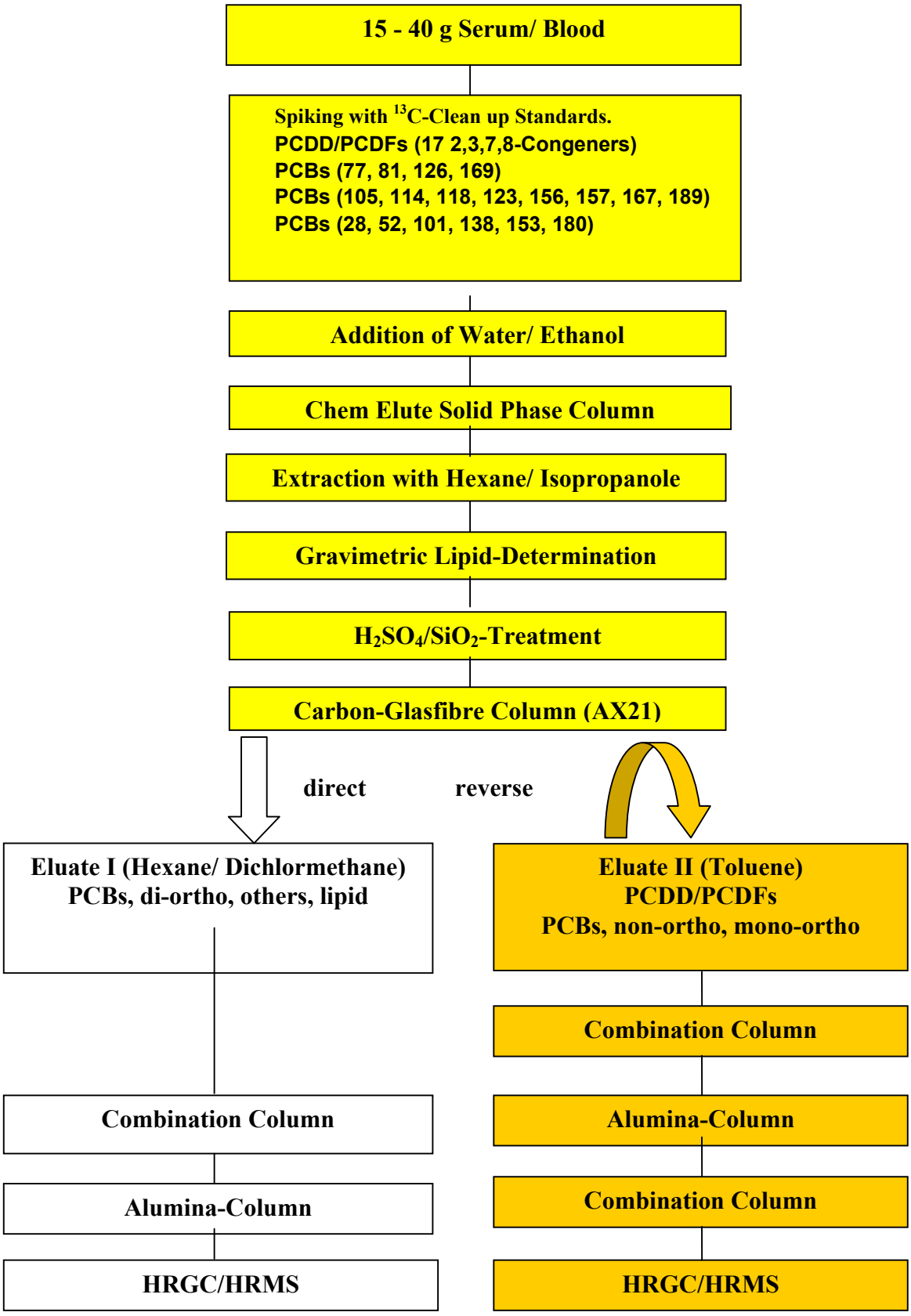
For each component 2 isotope masses are measured. The quantification is carried out by the use of internal/external standard mixtures (isotope dilution method). Following PCDDs/PCDFs and PCBs are determined and reported.

PCDDs/PCDFs			
PCDDs		**PCDFs**	
2,3,7,8	-Tetra-CDD	2,3,7,8	-Tetra-CDF
1,2,3,7,8	-Penta-CDD	1,2,3,7,8 2,3,4,7,8	-Penta-CDF -Penta-CDF
1,2,3,4,7,8 1,2,3,6,7,8 1,2,3,7,8,9	-Hexa-CDD -Hexa-CDD -Hexa-CDD	1,2,3,4,7,8 1,2,3,6,7,8 1,2,3,7,8,9 2,3,4,6,7,8	-Hexa-CDF -Hexa-CDF -Hexa-CDF -Hexa-CDF
1,2,3,4,6,7,8	-Hepta-CDD	1,2,3,4,6,7,8 1,2,3,4,7,8,9	-Hepta-CDF -Hepta-CDF
1,2,3,4,6,7,8,9	-Octa-CDD	1,2,3,4,6,7,8,9	-Octa-CDF

	PCBs		
	Compound		**IUPAC Code**
Non-ortho PCBs	3,3´,4,4´	-Tetra-CB	PCB 77
	3,4,4´,5	-Tetra-CB	PCB 81
	3,3´,4,4´,5	-Penta-CB	PCB 126
	3,3´,4,4´,5,5´	-Hexa-CB	PCB 169
Mono-ortho PCBs	2,3,3´,4,4´	-Penta-CB	PCB 105
	2,3,4,4´,5	-Penta-CB	PCB 114
	2,3´,4,4´,5	-Penta-CB	PCB 118
	2´,3,4,4´,5	-Penta-CB	PCB 123
	2,3,3´,4,4´,5	-Hexa-CB	PCB 156
	2,3,3´,4,4´,5´	-Hexa-CB	PCB 157
	2,3´,4,4´,5,5´	-Hexa-CB	PCB 167
	2,3,3´,4,4´,5,5´	-Hepta-CB	PCB 189

In addition to the single results, calculations of the toxicity equivalents (TEQ) according to the NATO/CCMS and the WHO-system are carried out.

Analytical Procedure for PCDD/PCDFs (and PCBs) in Serum/Blood



5 General information about PCDDs/PCDFs and PCBs

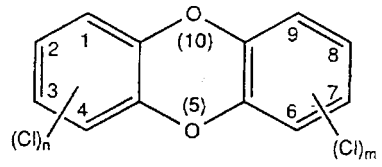
Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are unwanted by-products in a variety of industrial and thermal processes. However, their levels in the environment increased significantly with the beginning of the industrial chlorine industry in this century. Because of their many sources, PCDDs and PCDFs are ubiquitously distributed. The degree of chlorination of the tricyclic components varies between 1 and 8 atoms per molecule. The overall number of dioxins and furans is 75 and 135, respectively.

In humans, only the isomeres with 2,3,7,8-substitution are found, totaling seven dioxins and 10 furans. Humans may become contaminated with PCDD/PCDF through environmental (background), occupational, or accidental exposure.

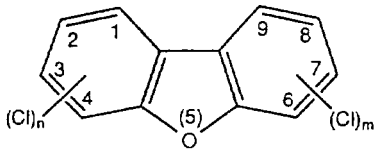
It is generally agreed that for the normal population, food represents the main route of environmental exposure to PCDD/s/PCDFs. Usually more than 90% of the total daily intake of these contaminants derives from food.

In contrast, exposure via other routes, such as inhalation and ingestion of particles from air, ingestion of contaminated soil, and dermal absorption, normally contributes less than 10% of daily intake. Because humans are the high end of the food chain, it becomes obvious that human tissue may contain relatively high amounts of xenobiotics such as PCDDs/PCDFs. Because of the lipophilic nature of these two classes of environmental contaminants, foodstuffs of animal origin are of special importance.

The following figure shows the general structure of polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs):

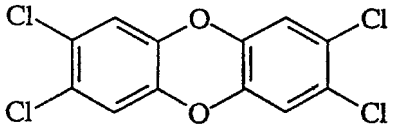


Polychlorinated dibenzo-p-dioxins (PCDDs)



Polychlorinated dibenzofurans (PCDFs)

The following figure shows the formula of 2,3,7,8-Tetrachlorodibenzo-p-dioxin, the most toxic compound of PCDDs/PCDFs.



2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN

Certain PCBs were found to have “dioxin-like” properties and were given a TEF (toxic equivalent factor) by WHO as well:

International TEFs for human beings and mammals

PCDDs (dioxins)	TEF
2,3,7,8-TCDD ('TCDD')	1
1,2,3,7,8-PeCDD	1
1,2,3,4,7,8-HxCDD	0.1
1,2,3,7,8,9-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
OCDD	0.0001
PCDFs (furans)	
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
1,2,3,4,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
OCDF	0.0001

PCBs with no chlorine at *ortho* positions ('coplanar' PCBs)	TEF
3,3',4,4'-TCB	0.0001
3,4,4',5-TCB	0.0001
3,3',4,4',5-PeCB	0.1
3,3',4,4',5,5'-HxCB	0.01
PCBs with one chlorine atom at *ortho* position	
2,3,3',4,4'-PeCB	0.0001
2,3,4,4',5-PeCB	0.0005
2,3',4,4',5-PeCB	0.0001
2',3,4,4',5-PeCB	0.0001
2,3,3',4,4',5-HxCB	0.0005
2,3,3',4,4',5'-HxCB	0.0005
2,3',4,4',5,5'-HxCB	0.00001
2,3,3',4,4',5,5'-HpCB	0.0001

T = tetra (4 chlorine atoms)
Pe = penta (5 chlorine atoms)
Hx = hexa (6 chlorine atoms)
Hp = hepta (7 chlorine atoms)
O = octa (8 chlorine atoms)

Source : *Persistent Organic Pollutants*, Monitor 16, 2000, Swedish Environmental Protection Agency

6 Results

The detailed results of all 2,3,7,8-substituted PCDDs/PCDFs and PCBs are shown on the data sheets (please see following pages). The sheets also present the detection limit (in case of "n.d.") and the individual Toxic Equivalent Factor (according to WHO) which is used for the calculation of the *single* individual Toxic Equivalents. The *total* of these values is the TEQ value, which is used for the quantitative evaluation of the overall PCDD/PCDF resp. PCB-contamination of a sample.

The results are valid for the analyzed samples only.

7 Final Remarks

It is not allowed to duplicate the report in parts without written permission by ERGO Forschungsgesellschaft mbH.

The samples are stored – on dependence of the test parameters - not longer than three months after the date of the report.

PCDD/PCDF and PCB in Serum

Values in pg/g (ppt), lipid based, upper bound

Analysis-No. H-02-12-0114 Code GROUP 1 "HEALTHY"

		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD	1,5	1,000	1,490	
	1.2.3.7.8-Penta-CDD	3,0	1,000	3,045	
	1.2.3.4.7.8-Hexa-CDD	2,2	0,100	0,215	
	1.2.3.6.7.8-Hexa-CDD	15,1	0,100	1,513	
	1.2.3.7.8.9-Hexa-CDD	2,7	0,100	0,270	
	1.2.3.4.6.7.8-Hepta-CDD	25,9	0,010	0,259	
	OCDD	284,4	0,0001	0,028	
PCDF	2.3.7.8-Tetra-CDF	n.d.	0,100	0,176	(1,8)
	1.2.3.7.8-Penta-CDF	1,1	0,050	0,054	
	2.3.4.7.8-Penta-CDF	2,8	0,500	1,416	
	1.2.3.4.7.8-Hexa-CDF	2,5	0,100	0,248	
	1.2.3.6.7.8-Hexa-CDF	2,0	0,100	0,203	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	0,148	(1,5)
	2.3.4.6.7.8-Hexa-CDF	1,8	0,100	0,184	
	1.2.3.4.6.7.8-Hepta-CDF	n.d.	0,010	0,045	(4,5)
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	0,034	(3,4)
	OCDF	n.d.	0,0001	0,001	(12)
non-ortho PCB	3,3',4,4'-TCB (77)	n.d.	0,0001	0,0061	(61)
	3,4,4',5-TCB (81)	n.d.	0,0001	0,0002	(2)
	3,3',4,4',5-PeCB (126)	26	0,1000	2,5849	
	3,3',4,4',5,5'-HxCB (169)	18	0,0100	0,1798	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	1004	0,0001	0,100	
	2,3,4,4',5-PeCB (114)	359	0,0005	0,179	
	2,3',4,4',5-PeCB (118)	4505	0,0001	0,451	
	2',3,4,4',5-PeCB (123)	n.d.	0,0001	0,010	(101)
	2,3,3',4,4',5-HxCB (156)	2690	0,0005	1,345	
	2,3,3',4,4',5'-HxCB (157)	670	0,0005	0,335	
	2,3',4,4',5,5'-HxCB (167)	712	0,00001	0,007	
	2,3,3',4,4',5,5'-HpCB (189)	290	0,0001	0,029	
	Total PCDD/PCDF	345,1			
	Total non-ortho-PCB	44			
	Total mono-ortho-PCB	10229			
	TEQ (WHO) based on PCDD/F			9,330	
	TEQ (WHO) based on PCB			5,227	
	TEQ (WHO)			**14,558**	

TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

n.d. = not detected, limit of detection (LOD) in ( ), n.a. = not analysed, Values with < contribute with 50%

(M) = maximum value, contains possible outside contamination

Small differences on totals result from computerroundings

PCDD/PCDF and PCB in Serum

Values in pg/g (ppt), lipid based, upper bound

Analysis-No. H-02-12-0115 Code GROUP 2 "HEALTHY"

		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD #	1,1	1,000	1,100	
	1.2.3.7.8-Penta-CDD	3,3	1,000	3,258	
	1.2.3.4.7.8-Hexa-CDD	2,4	0,100	0,235	
	1.2.3.6.7.8-Hexa-CDD	12,1	0,100	1,212	
	1.2.3.7.8.9-Hexa-CDD	1,4	0,100	0,142	
	1.2.3.4.6.7.8-Hepta-CDD	18,9	0,010	0,189	
	OCDD	213,5	0,0001	0,021	
PCDF	2.3.7.8-Tetra-CDF	n.d.	0,100	0,155	(1,6)
	1.2.3.7.8-Penta-CDF	0,7	0,050	0,037	
	2.3.4.7.8-Penta-CDF	2,6	0,500	1,298	
	1.2.3.4.7.8-Hexa-CDF	2,6	0,100	0,259	
	1.2.3.6.7.8-Hexa-CDF	1,7	0,100	0,169	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	0,089	(0,9)
	2.3.4.6.7.8-Hexa-CDF	n.d.	0,100	0,104	(1,0)
	1.2.3.4.6.7.8-Hepta-CDF	n.d.	0,010	0,040	(4,0)
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	0,026	(2,6)
	OCDF	n.d.	0,0001	0,001	(11)
non-ortho PCB	3,3',4,4'-TCB (77)	n.d.	0,0001	0,0054	(54)
	3,4,4',5-TCB (81)	n.d.	0,0001	0,0002	(2)
	3,3',4,4',5-PeCB (126)	17	0,1000	1,7253	
	3,3',4,4',5,5'-HxCB (169)	15	0,0100	0,1506	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	671	0,0001	0,067	
	2,3,4,4',5-PeCB (114)	238	0,0005	0,119	
	2,3',4,4',5-PeCB (118)	2558	0,0001	0,256	
	2',3,4,4',5-PeCB (123)	n.d.	0,0001	0,010	(98)
	2,3,3',4,4',5-HxCB (156)	1977	0,0005	0,989	
	2,3,3',4,4',5'-HxCB (157)	488	0,0005	0,244	
	2,3',4,4',5,5'-HxCB (167)	478	0,00001	0,005	
	2,3,3',4,4',5,5'-HpCB (189)	234	0,0001	0,023	
	Total PCDD/PCDF	260,2			
	Total non-ortho-PCB	32			
	Total mono-ortho-PCB	6644			
	TEQ (WHO) based on PCDD/F			8,337	
	TEQ (WHO) based on PCB			3,594	
	TEQ (WHO)			**11,931**	

TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

n.d. = not detected, limit of detection (LOD) in ( ), n.a. = not analysed, Values with < contribute with 50%

(M) = maximum value, contains possible outside contamination

= preliminary results, data doesn't fulfill quality control criteria

Small differences on totals result from computerroundings

PCDD/PCDF and PCB in Serum

Values in pg/g (ppt), lipid based, upper bound

Analysis-No. H-02-12-0116 Code GROUP 1 "SICK"

		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD	n.d.	1,000	0,951	(1,0)
	1.2.3.7.8-Penta-CDD	2,6	1,000	2,550	
	1.2.3.4.7.8-Hexa-CDD	2,1	0,100	0,208	
	1.2.3.6.7.8-Hexa-CDD	12,6	0,100	1,260	
	1.2.3.7.8.9-Hexa-CDD	2,9	0,100	0,292	
	1.2.3.4.6.7.8-Hepta-CDD	19,2	0,010	0,192	
	OCDD	229,5	0,0001	0,023	
PCDF	2.3.7.8-Tetra-CDF	n.d.	0,100	0,174	(1,7)
	1.2.3.7.8-Penta-CDF	0,7	0,050	0,037	
	2.3.4.7.8-Penta-CDF	2,5	0,500	1,240	
	1.2.3.4.7.8-Hexa-CDF	2,4	0,100	0,239	
	1.2.3.6.7.8-Hexa-CDF	1,6	0,100	0,164	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	0,063	(0,6)
	2.3.4.6.7.8-Hexa-CDF	n.d.	0,100	0,117	(1,2)
	1.2.3.4.6.7.8-Hepta-CDF	n.d.	0,010	0,045	(4,5)
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	0,012	(1,2)
	OCDF	n.d.	0,0001	0,001	(12)
non-ortho PCB	3,3',4,4'-TCB (77)	n.d.	0,0001	0,0060	(60)
	3,4,4',5-TCB (81)	n.d.	0,0001	0,0002	(2)
	3,3',4,4',5-PeCB (126)	24	0,1000	2,3705	
	3,3',4,4',5,5'-HxCB (169)	16	0,0100	0,1608	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	974	0,0001	0,097	
	2,3,4,4',5-PeCB (114)	320	0,0005	0,160	
	2,3',4,4',5-PeCB (118)	3356	0,0001	0,336	
	2',3,4,4',5-PeCB (123)	n.d.	0,0001	0,015	(148)
	2,3,3',4,4',5-HxCB (156)	2373	0,0005	1,186	
	2,3,3',4,4',5'-HxCB (157)	631	0,0005	0,316	
	2,3',4,4',5,5'-HxCB (167)	577	0,00001	0,006	
	2,3,3',4,4',5,5'-HpCB (189)	277	0,0001	0,028	
	Total PCDD/PCDF	276,1			
	Total non-ortho-PCB	40			
	Total mono-ortho-PCB	8508			
	TEQ (WHO) based on PCDD/F			7,568	
	TEQ (WHO) based on PCB			4,681	
	TEQ (WHO)			**12,249**	

TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

n.d. = not detected, limit of detection (LOD) in ( ), n.a. = not analysed, Values with < contribute with 50%

(M) = maximum value, contains possible outside contamination

Small differences on totals result from computerroundings

PCDD/PCDF and PCB in Serum

Values in pg/g (ppt), lipid based, upper bound

Analysis-No. H-02-12-0117 Code GROUP 2 "SICK"

		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD	1,4	1,000	1,377	
	1.2.3.7.8-Penta-CDD	2,0	1,000	2,040	
	1.2.3.4.7.8-Hexa-CDD	2,0	0,100	0,196	
	1.2.3.6.7.8-Hexa-CDD	9,5	0,100	0,950	
	1.2.3.7.8.9-Hexa-CDD	2,2	0,100	0,216	
	1.2.3.4.6.7.8-Hepta-CDD	17,1	0,010	0,171	
	OCDD	210,4	0,0001	0,021	
PCDF	2.3.7.8-Tetra-CDF	n.d.	0,100	0,149	(1,5)
	1.2.3.7.8-Penta-CDF	1,0	0,050	0,048	
	2.3.4.7.8-Penta-CDF	2,1	0,500	1,047	
	1.2.3.4.7.8-Hexa-CDF	2,7	0,100	0,272	
	1.2.3.6.7.8-Hexa-CDF	1,5	0,100	0,146	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	0,412	(4,1)
	2.3.4.6.7.8-Hexa-CDF	n.d.	0,100	0,100	(1,0)
	1.2.3.4.6.7.8-Hepta-CDF	n.d.	0,010	0,038	(3,8)
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	0,021	(2,1)
	OCDF	n.d.	0,0001	0,001	(10)
non-ortho PCB	3,3',4,4'-TCB (77)	n.d.	0,0001	0,0052	(52)
	3,4,4',5-TCB (81)	n.d.	0,0001	0,0002	(2)
	3,3',4,4',5-PeCB (126)	17	0,1000	1,7431	
	3,3',4,4',5,5'-HxCB (169)	15	0,0100	0,1550	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	702	0,0001	0,070	
	2,3,4,4',5-PeCB (114)	289	0,0005	0,145	
	2,3',4,4',5-PeCB (118)	2740	0,0001	0,274	
	2',3,4,4',5-PeCB (123)	n.d.	0,0001	0,012	(116)
	2,3,3',4,4',5-HxCB (156)	3036	0,0005	1,518	
	2,3,3',4,4',5'-HxCB (157)	698	0,0005	0,349	
	2,3',4,4',5,5'-HxCB (167)	559	0,00001	0,006	
	2,3,3',4,4',5,5'-HpCB (189)	270	0,0001	0,027	
	Total PCDD/PCDF	251,8			
	Total non-ortho-PCB	33			
	Total mono-ortho-PCB	8295			
	TEQ (WHO) based on PCDD/F			7,206	
	TEQ (WHO) based on PCB			4,304	
	TEQ (WHO)			**11,510**	

TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

n.d. = not detected, limit of detection (LOD) in ( ), n.a. = not analysed, Values with < contribute with 50%

(M) = maximum value, contains possible outside contamination

Small differences on totals result from computerroundings

Appendix C Ethics Approval

OFFICE OF RESEARCH AND POSTGRADUATE STUDIES

Jan Mazzer

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Friday, 20 September 2002

Dr Fiona Harden
National Research Centre for Environmental Toxicology

Dear Dr Harden

Concerning: Ethical clearance for project:- *Determination of the levels of dioxins in the Australian population by analysis of blood serum*

Clearance No: 2002000656

The Medical Research Ethics Committee has approved your project.

Please note that:-

(i) The Clearance number should be quoted on the protocol coversheet when applying to a granting agency and in any correspondence relating to ethical clearance;

(ii) Clearance will normally be for the duration of the project unless otherwise stated in the institutional clearance;

(iii) Adverse reaction to treatment by subjects, injury or any other incident affecting the welfare and/or health of subjects attributable to the research should be promptly reported to the Head of Department and the Behavioural and Social Sciences Ethical Review Committee.

(iv) Amendments to any part of the approved protocol, documents or questionnaires attached to this clearance are to be submitted to the Behavioural and Social Sciences Ethical Review Committee for approval.

2002000656

(v) Advisers on 'Integrity in Research'
As part of the University's commitment to the institutional statement, *Code of conduct for the Ethical Practice of Research (1990)*, and the NH&MRC's *National Statement on Ethical Conduct in Research Involving Humans (1999)*, designated positions have been appointed as advisers on integrity in research. The Chairperson of each ethics committee acts in an advisory capacity to provide confidential advice on such matters as misconduct in research, the rights and duties of postgraduate supervisors, and procedures for dealing with allegations on research misconduct within the University. The contact number for the Chairperson of each ethics committee can be obtained from the Ethics Officer.

(vi) The Committee reserves the right to visit the research site and materials at any time during the project.

(vii) It is the Committees expectation whenever possible, this work should result in publication and the Committee would require details to be submitted for our records.

Staff and students are also encouraged to contact either the Ethics Officer (3365 3924), or Chairperson on other issues concerning the conduct of experimentation/research (e.g. involvement of children, informed consent) prior to commencement of the project and throughout the course of the study.

Yours sincerely

Michael Tse
Ethics Officer

Encs.
cc: Head of School, National Research Centre for Environmental Toxicology

Institutional Approval Form For Experiments On Humans Including Behavioural Research

Chief Investigator: Dr Fiona Harden

Project Title: Determination of the levels of dioxins in the Australian population by analysis of blood serum

Supervisor: None

Co-Investigator(s): Dr Jochen Mueller

Department(s): National Research Centre for Environmental Toxicology

Project Number: 2002000656

Granting Agency/Degree: Environment Australia

Duration: 12-15 months

Comments:

Name of responsible Committee:-
Medical Research Ethics Committee
This project complies with the provisions contained in the *National Statement on Ethical Conduct in Research Involving Humans* and complies with the regulations governing experimentation on humans.

Name of Ethics Committee representative:-
Dr Peter Nixon
Chairperson
Medical Research Ethics Committee

Date [illegible] Signature [illegible]

Appendix D Analytical Methodology

1) Summary of Analytical Protocol for determination of dioxin-like compound (n=29) in human plasma

Health Canada, October 20003

Surrogate addition

To each sample is added solutions of carbon-13 labelled surrogate standards of all analytes to be determined. These surrogates consist of all 7 dioxins and 10 furans with 2,3,7,8-substitution, and the 12 PCBs (4 non *ortho* and 8 mono *ortho*) classified as dioxin-like by the World Health Organization (WHO). Amounts vary from 50 pg for the dioxins up to 2.5 ng for the mono *ortho* PCBs.

Extraction

For each volume of plasma, an equal volume of saturated aqueous ammonium sulfate and an equal volume of ethanol are added followed by three volumes of hexane. The mixture is then homogenized with a mechanical homogenizer and, after separation of phases, the hexane portion is withdrawn. The extraction, mixing, and separation are repeated with 1.5 volumes of hexane.

Lipid determination

The combined hexane extracts are filtered, washed with water to remove residual acetone, dried over a little anhydrous sodium sulfate, and evaporated to dryness on a rotary evaporator under vacuum. The residue is weighed over a period of time until constant weight is obtained (12-24 hrs). This weight is used to calculate the lipid content of the sample.

Lipid removal

The lipid residue containing the dioxin like compounds is reconstituted in 200 mL hexane and defatted by shaking in a separatory funnel with 20 mL portions of concentrated sulfuric acid. The acid portion is withdrawn and discarded and the acid treatment is repeated up to 10 times until the acid portions are clear and pale yellow. The hexane extract is washed with water, dilute aqueous base, again with water, dried and concentrated to a small volume (circa 1-2 mL) in preparation for column purification.

Extract purification

a) Acid silicate and Florisil columns: The hexane extract is added to a small silicate column containing strong acid to remove traces of interfering compounds. The eluent from this column goes directly onto a heat activated (150 °C) and not water deactivated Florisil column (1.5 g). Two fractions are collected: 1) about 40 mL of hexane (discarding the first 3.5 mL containing polar lipids) consisting of most of the PCBs including all 8 mono *ortho* congeners, and 2) 60 mL of dichloromethane containing dioxins, furans, and the four non *ortho* PCBs. Fraction 1 (most PCBs and organochlorines (OCs)) only is evaporated passively to 50 uL by weight in steps. 20 uL are taken and made up to 40 uL with recovery standard prior to injection on high resolution GC-MS.

b) Carbon column: Fraction 2 (dioxins, furans and non ortho PCBs) only from the Florisil column is further purified on a Carbopack C carbon column washing with hexane and eluting in the forward direction with toluene. The extract is taken to dryness in steps and reconstituted in 5 uL of toluene containing recovery standards prior to MS.

Measurement by gas chromatography (GC)-mass spectrometry (MS)

A) The GC is a Agilent 6890 containing for both fractions a DB-5 MS (Supelco) bonded phase capillary column of 30 m length, 0.25 mm id, and 0.25 μm thickness with retention gap. Injection of 1 μL is by the on column method at 80 °C with a fast ramp to 300 °C. The GC column is programmed in steps up to 250 °C.

B) The MS is a Micromass Auto Spec Ultima operating in the positive electron impact (EI) mode at 40 eV ionization energy, source temperature of 250 °C, and mass resolution (10% valley) of 10 K. Detectability for 2,3,7,8-TCDD after injection on GC and using up to 14 ions in the selected ion mode (SIM) is at least 100 femtograms (0.1 picograms). Identification of each analyte is governed by its gas chromatographic retention time (with 1.2 seconds of the standard), correct amu ion ratio (within 15% of standard) and a signal to noise ratio of at least 3:1.

C) PCB fraction (F1): Ions include the tetra, penta, hexa, and hepta homologues and these are monitored in groups including a lock and dummy mass. Switching between groups is governed by the elution characteristics from the GC as established from a calibration standard. For each analyte two molecular masses are taken for the carbon-12 channel and two for the carbon-13 channel.

D) dioxin/furan/non ortho PCB fraction (F2): Similar conditions as for the PCB fraction are used for this fraction.

E) Quantification: A standard curve is established consisting of an eight-point concentration level of carbon 12 analytes with constant concentration of carbon 13 isotopomers. Concentrations in the sample are calculated from the standard curve using the isotope dilution internal standard method comprising relative response factors (RRFs), concentration changes, and amounts of whole weight and lipid in the unknown sample. Results are expressed in ng/kg (parts per trillion; ppt) on both a whole and lipid basis. Recoveries of the carbon-13 surrogates added at the beginning are calculated using the recovery standards they added just prior to GC-MS.

Quality Control Measures

Each sample batch contains a laboratory blank to gauge the amount of analyte from the laboratory processing. This amount is subtracted from the total amount in the unknown sample prior to calculation of concentration. A reference or repeat sample is also analysed in every batch in ensure the analytical process is under control and results compare to previous work and other laboratories. Detection limits for dioxin-like compounds in human blood depend on the sample size, its lipid content, and contribution from the laboratory blank. Typically for a 50 mL sample of human blood the limit of detection (LOD) would be about 1 ppt for 2,3,7,8-TCDD.

Health Canada has participated successfully in all of the interlaboratory studies on dioxin-like compounds in foods organized by the Norwegian Institute of Health as well as the Canadian national check sample programs for PCBs.

Appendix E Analytical Reproducibility

- Double analysis – Presentation of single data
- Quality assurance – Pool data
- Lower bound calculation
- Upper bound calculation
- Comparison lower/upper bound
- WHO qualified-laboratories for blood
- Quality assurance Measures – PCDD/PCDF and PCB-determinations
- Quality assurance by participation in interlaboratory quality control studies
- Quality assessment – WHO-coordinated study
- List of WHO accepted laboratories for analysis of PCDDs/PCDFs in blood

Double analysis – Presentation of single data

	PCDD/PCDF and PCB in Human Serum				
	Values in pg/g (ppt), lipid based				
	Analysis-No. H-03-07-0400	Analysis I	Name: Not provided Code: B5F I Date of birth: Not provided Age: Not provided		
		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD	2,1	1,000	2,078	
	1.2.3.7.8-Penta-CDD	3,4	1,000	3,424	
	1.2.3.4.7.8-Hexa-CDD	3,6	0,100	0,363	
	1.2.3.6.7.8-Hexa-CDD	26,4	0,100	2,637	
	1.2.3.7.8.9-Hexa-CDD	5,3	0,100	0,534	
	1.2.3.4.6.7.8-Hepta-CDD	46,8	0,010	0,468	
	OCDD	369,9	0,0001	0,037	
PCDF	2.3.7.8-Tetra-CDF	0,6	0,100	0,058	
	1.2.3.7.8-Penta-CDF	0,7	0,050	0,037	
	2.3.4.7.8-Penta-CDF	4,2	0,500	2,116	
	1.2.3.4.7.8-Hexa-CDF	4,4	0,100	0,440	
	1.2.3.6.7.8-Hexa-CDF	2,9	0,100	0,289	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	-	(6,7)
	2.3.4.6.7.8-Hexa-CDF	3,3	0,100	0,327	
	1.2.3.4.6.7.8-Hepta-CDF	3,0	0,010	0,030	
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	-	(1,0)
	OCDF	1,7	0,0001	0,0002	
non-ortho PCB	3,3',4,4'-TCB (77)	n.a.	0,0001	-	
	3,4,4',5-TCB (81)	n.a.	0,0001	-	
	3,3',4,4',5-PeCB (126)	56	0,1000	5,595	(M)
	3,3',4,4',5,5'-HxCB (169)	24	0,0100	0,243	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	2018	0,0001	0,202	
	2,3,4,4',5-PeCB (114)	600	0,0005	0,300	
	2,3',4,4',5-PeCB (118)	12817	0,0001	1,282	
	2',3,4,4',5-PeCB (123)	333	0,0001	0,033	
	2,3,3',4,4',5-HxCB (156)	4504	0,0005	2,252	
	2,3,3',4,4',5'-HxCB (157)	1011	0,0005	0,506	
	2,3',4,4',5,5'-HxCB (167)	1769	0,00001	0,018	
	2,3,3',4,4',5,5'-HpCB (189)	373	0,0001	0,037	
	Total PCDD/PCDF	478,3			
	Total non-ortho-PCB	80			
	Total mono-ortho-PCB	23425			
	TEQ (WHO) based on PCDD/F			12,837	
	TEQ (WHO) based on PCB			10,467	
	TEQ (WHO)			**23,304**	

TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

n.d. = not detected, limit of detection (LOD) in ( ), n.a. = not analysed, Values with < contribute with 50%

(M) = maximum value, contains possible outside contamination

Small differences on totals result from computerroundings

PCDD/PCDF and PCB in Human Serum

Values in pg/g (ppt), lipid based

Analysis-No. H-03-07-0400 Analysis II

Name: Not provided
Code: B5F I
Date of birth: Not provided
Age: Not provided

		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD	2,2	1,000	2,231	
	1.2.3.7.8-Penta-CDD	4,7	1,000	4,665	
	1.2.3.4.7.8-Hexa-CDD	4,6	0,100	0,456	
	1.2.3.6.7.8-Hexa-CDD	31,5	0,100	3,146	
	1.2.3.7.8.9-Hexa-CDD	5,9	0,100	0,590	
	1.2.3.4.6.7.8-Hepta-CDD	45,3	0,010	0,453	
	OCDD	424,5	0,0001	0,042	
PCDF	2.3.7.8-Tetra-CDF	n.d.	0,100	-	(1,0)
	1.2.3.7.8-Penta-CDF	n.d.	0,050	-	(1,0)
	2.3.4.7.8-Penta-CDF	4,5	0,500	2,249	
	1.2.3.4.7.8-Hexa-CDF	3,7	0,100	0,372	
	1.2.3.6.7.8-Hexa-CDF	2,9	0,100	0,290	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	-	(6,0)
	2.3.4.6.7.8-Hexa-CDF	n.d.	0,100	-	(1,5)
	1.2.3.4.6.7.8-Hepta-CDF	1,5	0,010	0,015	
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	-	(1,1)
	OCDF	n.d.	0,0001	-	(2,0)
non-ortho PCB	3,3',4,4'-TCB (77)	n.d.	0,0001	-	(58)
	3,4,4',5-TCB (81)	n.d.	0,0001	-	(4)
	3,3',4,4',5-PeCB (126)	n.d.	0,1000	-	(58)
	3,3',4,4',5,5'-HxCB (169)	21	0,0100	0,214	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	2385	0,0001	0,239	
	2,3,4,4',5-PeCB (114)	728	0,0005	0,364	
	2,3',4,4',5-PeCB (118)	14966	0,0001	1,497	
	2',3,4,4',5-PeCB (123)	302	0,0001	0,030	
	2,3,3',4,4',5-HxCB (156)	5089	0,0005	2,544	
	2,3,3',4,4',5'-HxCB (157)	1161	0,0005	0,581	
	2,3',4,4',5,5'-HxCB (167)	1897	0,00001	0,019	
	2,3,3',4,4',5,5'-HpCB (189)	432	0,0001	0,043	
	Total PCDD/PCDF	531,2			
	Total non-ortho-PCB	21			
	Total mono-ortho-PCB	26960			
	TEQ (WHO) based on PCDD/F			14,510	
	TEQ (WHO) based on PCB			5,530	
	TEQ (WHO)			**20,040**	

TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

n.d. = not detected, limit of detection (LOD) in ( ), n.a. = not analysed, Values with < contribute with 50%

(M) = maximum value, contains possible outside contamination

Small differences on totals result from computerroundings

PCDD/PCDF and PCB in Human Serum

Values in pg/g (ppt), lipid based

Analysis-No. H-03-07-0405 Analysis I

Name: Not provided
Code: B5F II
Date of birth: Not provided
Age: Not provided

		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD	2,2	1,000	2,196	
	1.2.3.7.8-Penta-CDD	4,4	1,000	4,351	
	1.2.3.4.7.8-Hexa-CDD	4,3	0,100	0,426	
	1.2.3.6.7.8-Hexa-CDD	29,3	0,100	2,930	
	1.2.3.7.8.9-Hexa-CDD	4,6	0,100	0,456	
	1.2.3.4.6.7.8-Hepta-CDD	46,3	0,010	0,463	
	OCDD	421,9	0,0001	0,042	
PCDF	2.3.7.8-Tetra-CDF	n.d.	0,100	-	(0,5)
	1.2.3.7.8-Penta-CDF	2,0	0,050	0,099	
	2.3.4.7.8-Penta-CDF	4,1	0,500	2,065	
	1.2.3.4.7.8-Hexa-CDF	4,7	0,100	0,465	
	1.2.3.6.7.8-Hexa-CDF	3,2	0,100	0,323	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	-	(5,9)
	2.3.4.6.7.8-Hexa-CDF	3,6	0,100	0,361	
	1.2.3.4.6.7.8-Hepta-CDF	3,0	0,010	0,030	
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	-	(1,0)
	OCDF	n.d.	0,0001	-	(1,7)
non-ortho PCB	3,3',4,4'-TCB (77)	n.a.	0,0001	-	
	3,4,4',5-TCB (81)	n.a.	0,0001	-	
	3,3',4,4',5-PeCB (126)	181*	0,1000	18,110	(M)
	3,3',4,4',5,5'-HxCB (169)	58*	0,0100	0,582	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	2054	0,0001	0,205	
	2,3,4,4',5-PeCB (114)	671	0,0005	0,335	
	2,3',4,4',5-PeCB (118)	12072	0,0001	1,207	
	2',3,4,4',5-PeCB (123)	305	0,0001	0,031	
	2,3,3',4,4',5-HxCB (156)	8972*	0,0005	4,486	
	2,3,3',4,4',5'-HxCB (157)	1640*	0,0005	0,820	
	2,3',4,4',5,5'-HxCB (167)	3844*	0,00001	0,038	
	2,3,3',4,4',5,5'-HpCB (189)	2402*	0,0001	0,240	
	Total PCDD/PCDF	533,5			
	Total non-ortho-PCB	239,3			
	Total mono-ortho-PCB	31960,1			
	TEQ (WHO) based on PCDD/F			14,208	
	TEQ (WHO) based on PCB			26,055	
	TEQ (WHO)			**40,263**	

TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

n.d. = not detected, limit of detection (LOD) in ( ), n.a. = not analysed, Values with < contribute with 50%

(M) = maximum value, contains possible outside contamination

Small differences on totals result from computerroundings

* Because of quality criteria not considered

PCDD/PCDF and PCB in Human Serum

Values in pg/g (ppt), lipid based

Analysis-No. H-03-07-0405 Analysis II

Name: Not provided
Code: B5F II
Date of birth: Not provided
Age: Not provided

		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD	2,3	1,000	2,268	
	1.2.3.7.8-Penta-CDD	4,9	1,000	4,936	
	1.2.3.4.7.8-Hexa-CDD	4,7	0,100	0,470	
	1.2.3.6.7.8-Hexa-CDD	34,1	0,100	3,409	
	1.2.3.7.8.9-Hexa-CDD	6,8	0,100	0,683	
	1.2.3.4.6.7.8-Hepta-CDD	43,7	0,010	0,437	
	OCDD	448,4	0,0001	0,045	
PCDF	2.3.7.8-Tetra-CDF	n.d.	0,100	-	(1,1)
	1.2.3.7.8-Penta-CDF	n.d.	0,050	-	(1,0)
	2.3.4.7.8-Penta-CDF	4,6	0,500	2,282	
	1.2.3.4.7.8-Hexa-CDF	3,4	0,100	0,342	
	1.2.3.6.7.8-Hexa-CDF	2,6	0,100	0,261	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	-	(7,4)
	2.3.4.6.7.8-Hexa-CDF	n.d.	0,100	-	(1,3)
	1.2.3.4.6.7.8-Hepta-CDF	1,5	0,010	0,015	
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	-	(1,1)
	OCDF	n.d.	0,0001	-	(2,3)
non-ortho PCB	3,3',4,4'-TCB (77)	n.d.	0,0001	-	(62)
	3,4,4',5-TCB (81)	n.d.	0,0001	-	(4)
	3,3',4,4',5-PeCB (126)	n.d.	0,1000	-	(52)
	3,3',4,4',5,5'-HxCB (169)	20	0,0100	0,196	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	2280	0,0001	0,228	
	2,3,4,4',5-PeCB (114)	843	0,0005	0,421	
	2,3',4,4',5-PeCB (118)	14281	0,0001	1,428	
	2',3,4,4',5-PeCB (123)	293	0,0001	0,029	
	2,3,3',4,4',5-HxCB (156)	5259	0,0005	2,630	
	2,3,3',4,4',5'-HxCB (157)	1244	0,0005	0,622	
	2,3',4,4',5,5'-HxCB (167)	1826	0,00001	0,018	
	2,3,3',4,4',5,5'-HpCB (189)	416	0,0001	0,042	
	Total PCDD/PCDF	557,0			
	Total non-ortho-PCB	20			
	Total mono-ortho-PCB	26442			
	TEQ (WHO) based on PCDD/F			15,148	
	TEQ (WHO) based on PCB			5,614	
	TEQ (WHO)			**20,762**	

TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

n.d. = not detected, limit of detection (LOD) in ( ), n.a. = not analysed, Values with < contribute with 50%

(M) = maximum value, contains possible outside contamination

Small differences on totals result from computerroundings

PCDD/PCDF and PCB in Human Serum

Values in pg/g (ppt), lipid based

Analysis-No. H-03-07-0453 Analysis I

Name: Not provided
Code: R3M II
Date of birth: Not Provided
Age: Not provided

		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD	n.d.	1,000	-	(1,3)
	1.2.3.7.8-Penta-CDD	1,8	1,000	1,770	
	1.2.3.4.7.8-Hexa-CDD	1,2	0,100	0,123	
	1.2.3.6.7.8-Hexa-CDD	10,2	0,100	1,024	
	1.2.3.7.8.9-Hexa-CDD	1,4	0,100	0,143	
	1.2.3.4.6.7.8-Hepta-CDD	23,2	0,010	0,232	
	OCDD	277,1	0,0001	0,028	
PCDF	2.3.7.8-Tetra-CDF	n.d.	0,100	-	(0,4)
	1.2.3.7.8-Penta-CDF	n.d.	0,050	-	(0,3)
	2.3.4.7.8-Penta-CDF	1,8	0,500	0,923	
	1.2.3.4.7.8-Hexa-CDF	1,7	0,100	0,167	
	1.2.3.6.7.8-Hexa-CDF	1,4	0,100	0,138	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	-	(4,9)
	2.3.4.6.7.8-Hexa-CDF	n.d.	0,100	-	(1,9)
	1.2.3.4.6.7.8-Hepta-CDF	3,0	0,010	0,030	
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	-	(1,0)
	OCDF	n.d.	0,0001	-	(1,5)
non-ortho PCB	3,3',4,4'-TCB (77)	n.d.	0,0001	-	(31)
	3,4,4',5-TCB (81)	n.d.	0,0001	-	(1)
	3,3',4,4',5-PeCB (126)	11	0,1000	1,132	
	3,3',4,4',5,5'-HxCB (169)	15	0,0100	0,152	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	391	0,0001	0,039	
	2,3,4,4',5-PeCB (114)	116	0,0005	0,058	
	2,3',4,4',5-PeCB (118)	2065	0,0001	0,206	
	2',3,4,4',5-PeCB (123)	n.d.	0,0001	-	(32)
	2,3,3',4,4',5-HxCB (156)	1710	0,0005	0,855	
	2,3,3',4,4',5'-HxCB (157)	361	0,0005	0,180	
	2,3',4,4',5,5'-HxCB (167)	394	0,00001	0,004	
	2,3,3',4,4',5,5'-HpCB (189)	284	0,0001	0,028	
	Total PCDD/PCDF	322,9			
	Total non-ortho-PCB	27			
	Total mono-ortho-PCB	5320			
	TEQ (WHO) based on PCDD/F			4,577	
	TEQ (WHO) based on PCB			2,656	
	TEQ (WHO)			**7,233**	

TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

n.d. = not detected, limit of detection (LOD) in ( ), n.a. = not analysed, Values with < contribute with 50%

(M) = maximum value, contains possible outside contamination

Small differences on totals result from computerroundings

PCDD/PCDF and PCB in Human Serum

Values in pg/g (ppt), lipid based

Analysis-No. H-03-07-0453 Analysis II

Name: Not provided
Code: R3M II
Date of birth: Not Provided
Age: Not provided

		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD	1,5	1,000	1,550	
	1.2.3.7.8-Penta-CDD	2,1	1,000	2,068	
	1.2.3.4.7.8-Hexa-CDD	n.d.	0,100	-	(1,4)
	1.2.3.6.7.8-Hexa-CDD	10,2	0,100	1,023	
	1.2.3.7.8.9-Hexa-CDD	2,1	0,100	0,207	
	1.2.3.4.6.7.8-Hepta-CDD	23,1	0,010	0,231	
	OCDD	253,9	0,0001	0,025	
PCDF	2.3.7.8-Tetra-CDF	n.d.	0,100	-	(1,3)
	1.2.3.7.8-Penta-CDF	n.d.	0,050	-	(0,8)
	2.3.4.7.8-Penta-CDF	2,0	0,500	1,024	
	1.2.3.4.7.8-Hexa-CDF	2,0	0,100	0,197	
	1.2.3.6.7.8-Hexa-CDF	1,4	0,100	0,139	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	-	(2,3)
	2.3.4.6.7.8-Hexa-CDF	n.d.	0,100	-	(2,1)
	1.2.3.4.6.7.8-Hepta-CDF	3,2	0,010	0,032	
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	-	(1,3)
	OCDF	n.d.	0,0001	-	(2,5)
non-ortho PCB	3,3',4,4'-TCB (77)	n.d.	0,0001	-	(65)
	3,4,4',5-TCB (81)	n.d.	0,0001	-	(10)
	3,3',4,4',5-PeCB (126)	53*	0,1000	5,345	
	3,3',4,4',5,5'-HxCB (169)	17	0,0100	0,168	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	439	0,0001	0,044	
	2,3,4,4',5-PeCB (114)	121	0,0005	0,061	
	2,3',4,4',5-PeCB (118)	2443	0,0001	0,244	
	2',3,4,4',5-PeCB (123)	n.d.	0,0001	-	(67)
	2,3,3',4,4',5-HxCB (156)	1752	0,0005	0,876	
	2,3,3',4,4',5'-HxCB (157)	362	0,0005	0,181	
	2,3',4,4',5,5'-HxCB (167)	467	0,00001	0,005	
	2,3,3',4,4',5,5'-HpCB (189)	269	0,0001	0,027	
	Total PCDD/PCDF	301,6			
	Total non-ortho-PCB	70			
	Total mono-ortho-PCB	5855			
	TEQ (WHO) based on PCDD/F			6,496	
	TEQ (WHO) based on PCB			6,950	
	TEQ (WHO)			**13,446**	

TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

n.d. = not detected, limit of detection (LOD) in ( ), n.a. = not analysed, Values with < contribute with 50%

(M) = maximum value, contains possible outside contamination

Small differences on totals result from computerroundings

* Because of quality criteria not considered

PCDD/PCDF and PCB in Human Serum

Values in pg/g (ppt), lipid based

Analysis-No. H-03-07-0479 Analysis I

Name: Not provided
Code: Y4F I
Date of birth: Not Provided
Age: Not provide

		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD	1,1	1,000	1,085	
	1.2.3.7.8-Penta-CDD	2,9	1,000	2,855	
	1.2.3.4.7.8-Hexa-CDD	2,6	0,100	0,256	
	1.2.3.6.7.8-Hexa-CDD	17,5	0,100	1,746	
	1.2.3.7.8.9-Hexa-CDD	3,4	0,100	0,338	
	1.2.3.4.6.7.8-Hepta-CDD	31,1	0,010	0,311	
	OCDD	364,9	0,0001	0,036	
PCDF	2.3.7.8-Tetra-CDF	n.d.	0,100	-	(0,4)
	1.2.3.7.8-Penta-CDF	n.d.	0,050	-	(0,3)
	2.3.4.7.8-Penta-CDF	2,5	0,500	1,237	
	1.2.3.4.7.8-Hexa-CDF	1,9	0,100	0,192	
	1.2.3.6.7.8-Hexa-CDF	1,4	0,100	0,142	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	-	(3,7)
	2.3.4.6.7.8-Hexa-CDF	n.d.	0,100	-	(1,8)
	1.2.3.4.6.7.8-Hepta-CDF	1,5	0,010	0,015	
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	-	(1,0)
	OCDF	n.d.	0,0001	-	(1,4)
non-ortho PCB	3,3',4,4'-TCB (77)	n.d.	0,0001	-	(27)
	3,4,4',5-TCB (81)	n.d.	0,0001	-	(2)
	3,3',4,4',5-PeCB (126)	17	0,1000	1,726	
	3,3',4,4',5,5'-HxCB (169)	15	0,0100	0,149	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	1024	0,0001	0,102	
	2,3,4,4',5-PeCB (114)	387	0,0005	0,194	
	2,3',4,4',5-PeCB (118)	6151	0,0001	0,615	
	2',3,4,4',5-PeCB (123)	112	0,0001	0,011	
	2,3,3',4,4',5-HxCB (156)	3358	0,0005	1,679	
	2,3,3',4,4',5'-HxCB (157)	712	0,0005	0,356	
	2,3',4,4',5,5'-HxCB (167)	930	0,00001	0,009	
	2,3,3',4,4',5,5'-HpCB (189)	283	0,0001	0,028	
	Total PCDD/PCDF	430,7			
	Total non-ortho-PCB	32			
	Total mono-ortho-PCB	12959			
	TEQ (WHO) based on PCDD/F			8,214	
	TEQ (WHO) based on PCB			4,871	
	TEQ (WHO)			**13,085**	

TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

n.d. = not detected, limit of detection (LOD) in ( ), n.a. = not analysed, Values with < contribute with 50%

(M) = maximum value, contains possible outside contamination

Small differences on totals result from computerroundings

PCDD/PCDF and PCB in Human Serum

Values in pg/g (ppt), lipid based

Analysis-No. H-03-07-0479 Analysis II

Name: Not provided
Code: Y4F I
Date of birth: Not Provided
Age: Not provide

		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD	1,3	1,000	1,283	
	1.2.3.7.8-Penta-CDD	3,0	1,000	3,044	
	1.2.3.4.7.8-Hexa-CDD	2,6	0,100	0,260	
	1.2.3.6.7.8-Hexa-CDD	15,4	0,100	1,537	
	1.2.3.7.8.9-Hexa-CDD	3,6	0,100	0,365	
	1.2.3.4.6.7.8-Hepta-CDD	28,9	0,010	0,289	
	OCDD	323,7	0,0001	0,032	
PCDF	2.3.7.8-Tetra-CDF	n.d.	0,100	-	(0,8)
	1.2.3.7.8-Penta-CDF	n.d.	0,050	-	(0,5)
	2.3.4.7.8-Penta-CDF	2,0	0,500	1,012	
	1.2.3.4.7.8-Hexa-CDF	2,0	0,100	0,200	
	1.2.3.6.7.8-Hexa-CDF	1,6	0,100	0,162	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	-	(2,9)
	2.3.4.6.7.8-Hexa-CDF	n.d.	0,100	-	(2,0)
	1.2.3.4.6.7.8-Hepta-CDF	1,8	0,010	0,018	
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	-	(1,1)
	OCDF	n.d.	0,0001	-	(2,1)
non-ortho PCB	3,3',4,4'-TCB (77)	n.d.	0,0001	-	(38)
	3,4,4',5-TCB (81)	n.d.	0,0001	-	(2)
	3,3',4,4',5-PeCB (126)	n.d.	0,1000	-	(25)
	3,3',4,4',5,5'-HxCB (169)	14	0,0100	0,137	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	1064	0,0001	0,106	
	2,3,4,4',5-PeCB (114)	392	0,0005	0,196	
	2,3',4,4',5-PeCB (118)	6464	0,0001	0,646	
	2',3,4,4',5-PeCB (123)	79	0,0001	0,008	
	2,3,3',4,4',5-HxCB (156)	2982	0,0005	1,491	
	2,3,3',4,4',5'-HxCB (157)	662	0,0005	0,331	
	2,3',4,4',5,5'-HxCB (167)	847	0,00001	0,008	
	2,3,3',4,4',5,5'-HpCB (189)	249	0,0001	0,025	
	Total PCDD/PCDF	385,9			
	Total non-ortho-PCB	14			
	Total mono-ortho-PCB	12738			
	TEQ (WHO) based on PCDD/F			8,202	
	TEQ (WHO) based on PCB			2,949	
	TEQ (WHO)			**11,151**	

TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

n.d. = not detected, limit of detection (LOD) in ( ), n.a. = not analysed, Values with < contribute with 50%

(M) = maximum value, contains possible outside contamination

Small differences on totals result from computerroundings

PCDD/PCDF and PCB in Human Serum

Values in pg/g (ppt), lipid based

Analysis-No. H-03-07-0484 Analysis I

Name: Not provided
Code: Y4F II
Date of birth: Not Provided
Age: Not provided

		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD	1,0	1,000	1,004	
	1.2.3.7.8-Penta-CDD	2,7	1,000	2,679	
	1.2.3.4.7.8-Hexa-CDD	2,0	0,100	0,201	
	1.2.3.6.7.8-Hexa-CDD	17,0	0,100	1,705	
	1.2.3.7.8.9-Hexa-CDD	2,9	0,100	0,291	
	1.2.3.4.6.7.8-Hepta-CDD	27,6	0,010	0,276	
	OCDD	282,4	0,0001	0,028	
PCDF	2.3.7.8-Tetra-CDF	n.d.	0,100	-	(0,4)
	1.2.3.7.8-Penta-CDF	n.d.	0,050	-	(0,4)
	2.3.4.7.8-Penta-CDF	1,9	0,500	0,964	
	1.2.3.4.7.8-Hexa-CDF	1,5	0,100	0,151	
	1.2.3.6.7.8-Hexa-CDF	1,5	0,100	0,150	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	-	(5,0)
	2.3.4.6.7.8-Hexa-CDF	n.d.	0,100	-	(1,6)
	1.2.3.4.6.7.8-Hepta-CDF	1,5	0,010	0,015	
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	-	(1,1)
	OCDF	n.d.	0,0001	-	(2,1)
non-ortho PCB	3,3',4,4'-TCB (77)	n.d.	0,0001	-	(24)
	3,4,4',5-TCB (81)	n.d.	0,0001	-	(1)
	3,3',4,4',5-PeCB (126)	n.d.	0,1000	-	(23)
	3,3',4,4',5,5'-HxCB (169)	14	0,0100	0,143	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	869	0,0001	0,087	
	2,3,4,4',5-PeCB (114)	324	0,0005	0,162	
	2,3',4,4',5-PeCB (118)	5564	0,0001	0,556	
	2',3,4,4',5-PeCB (123)	48	0,0001	0,005	
	2,3,3',4,4',5-HxCB (156)	2881	0,0005	1,441	
	2,3,3',4,4',5'-HxCB (157)	578	0,0005	0,289	
	2,3',4,4',5,5'-HxCB (167)	802	0,00001	0,008	
	2,3,3',4,4',5,5'-HpCB (189)	272	0,0001	0,027	
	Total PCDD/PCDF	342,1			
	Total non-ortho-PCB	14			
	Total mono-ortho-PCB	11338			
	TEQ (WHO) based on PCDD/F			7,463	
	TEQ (WHO) based on PCB			2,717	
	TEQ (WHO)			**10,180**	

TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

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(M) = maximum value, contains possible outside contamination

Small differences on totals result from computerroundings

PCDD/PCDF and PCB in Human Serum

Values in pg/g (ppt), lipid based

Analysis-No. H-03-07-0484 Analysis II

Name: Not provided
Code: Y4F II
Date of birth: Not Provided
Age: Not provided

		Concentration	TEF (WHO)	TEQ (WHO)	LOD
PCDD	2.3.7.8-Tetra-CDD	1,2	1,000	1,173	
	1.2.3.7.8-Penta-CDD	2,8	1,000	2,754	
	1.2.3.4.7.8-Hexa-CDD	2,6	0,100	0,256	
	1.2.3.6.7.8-Hexa-CDD	18,2	0,100	1,817	
	1.2.3.7.8.9-Hexa-CDD	3,3	0,100	0,326	
	1.2.3.4.6.7.8-Hepta-CDD	26,8	0,010	0,268	
	OCDD	273,5	0,0001	0,027	
PCDF	2.3.7.8-Tetra-CDF	n.d.	0,100	-	(1,3)
	1.2.3.7.8-Penta-CDF	n.d.	0,050	-	(0,6)
	2.3.4.7.8-Penta-CDF	2,0	0,500	0,988	
	1.2.3.4.7.8-Hexa-CDF	2,3	0,100	0,231	
	1.2.3.6.7.8-Hexa-CDF	1,7	0,100	0,174	
	1.2.3.7.8.9-Hexa-CDF	n.d.	0,100	-	(2,3)
	2.3.4.6.7.8-Hexa-CDF	n.d.	0,100	-	(2,0)
	1.2.3.4.6.7.8-Hepta-CDF	1,8	0,010	0,018	
	1.2.3.4.7.8.9-Hepta-CDF	n.d.	0,010	-	(1,1)
	OCDF	n.d.	0,0001	-	(2,1)
non-ortho PCB	3,3',4,4'-TCB (77)	n.d.	0,0001	-	(34)
	3,4,4',5-TCB (81)	n.d.	0,0001	-	(2)
	3,3',4,4',5-PeCB (126)	n.d.	0,1000	-	(23)
	3,3',4,4',5,5'-HxCB (169)	16	0,0100	0,157	
mono-ortho PCB	2,3,3',4,4'-PeCB (105)	926	0,0001	0,093	
	2,3,4,4',5-PeCB (114)	330	0,0005	0,165	
	2,3',4,4',5-PeCB (118)	5727	0,0001	0,573	
	2',3,4,4',5-PeCB (123)	72	0,0001	0,007	
	2,3,3',4,4',5-HxCB (156)	2640	0,0005	1,320	
	2,3,3',4,4',5'-HxCB (157)	573	0,0005	0,287	
	2,3',4,4',5,5'-HxCB (167)	822	0,00001	0,008	
	2,3,3',4,4',5,5'-HpCB (189)	247	0,0001	0,025	
	Total PCDD/PCDF	336,0			
	Total non-ortho-PCB	16			
	Total mono-ortho-PCB	11337			
	TEQ (WHO) based on PCDD/F			8,031	
	TEQ (WHO) based on PCB			2,634	
	TEQ (WHO)			**10,665**	

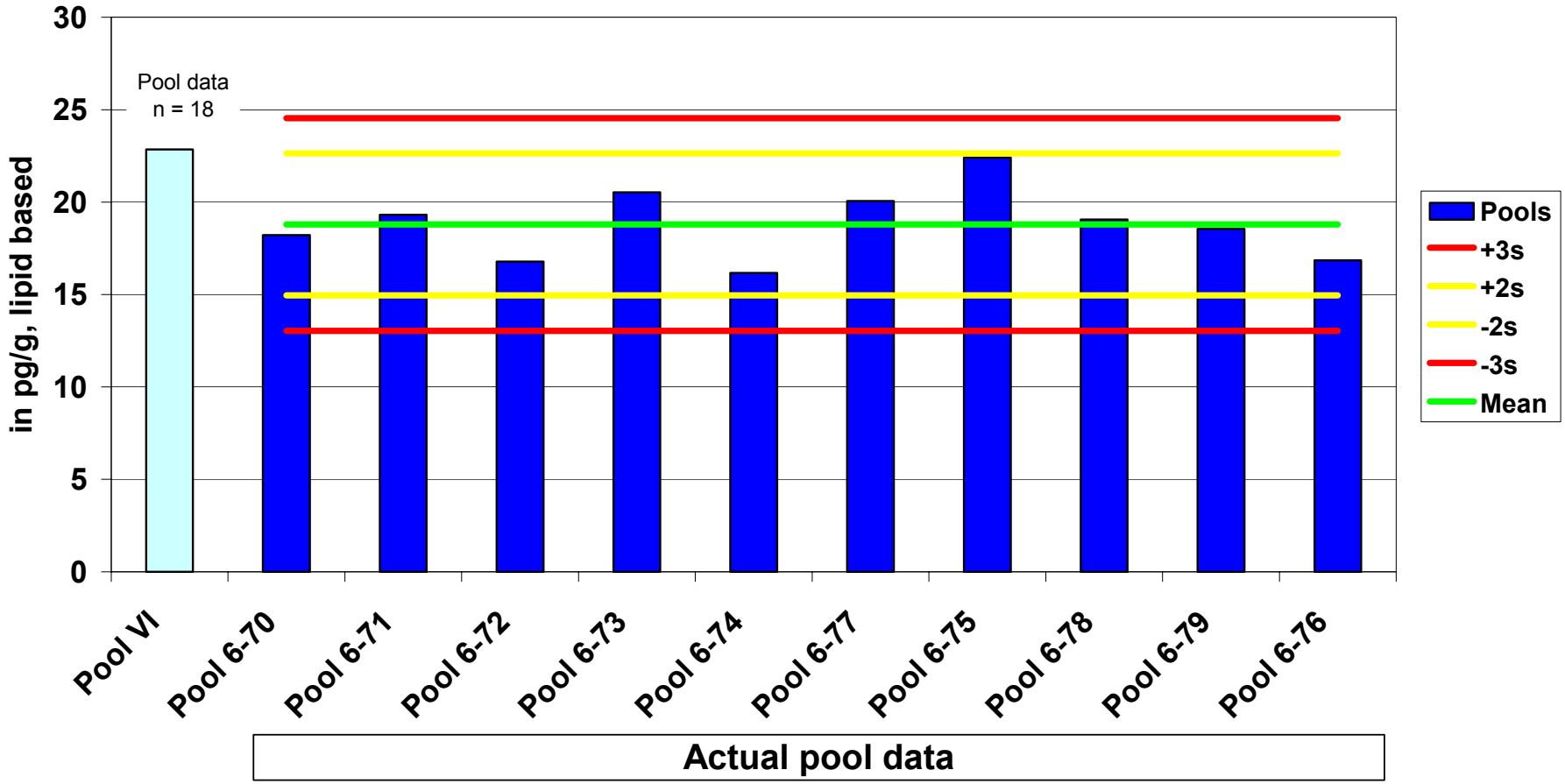
TEQ, TEF (WHO) = Toxic equivalent / -faktor by WHO for humans & mammals

n.d. = not detected, limit of detection (LOD) in ( ), n.a. = not analysed, Values with < contribute with 50%

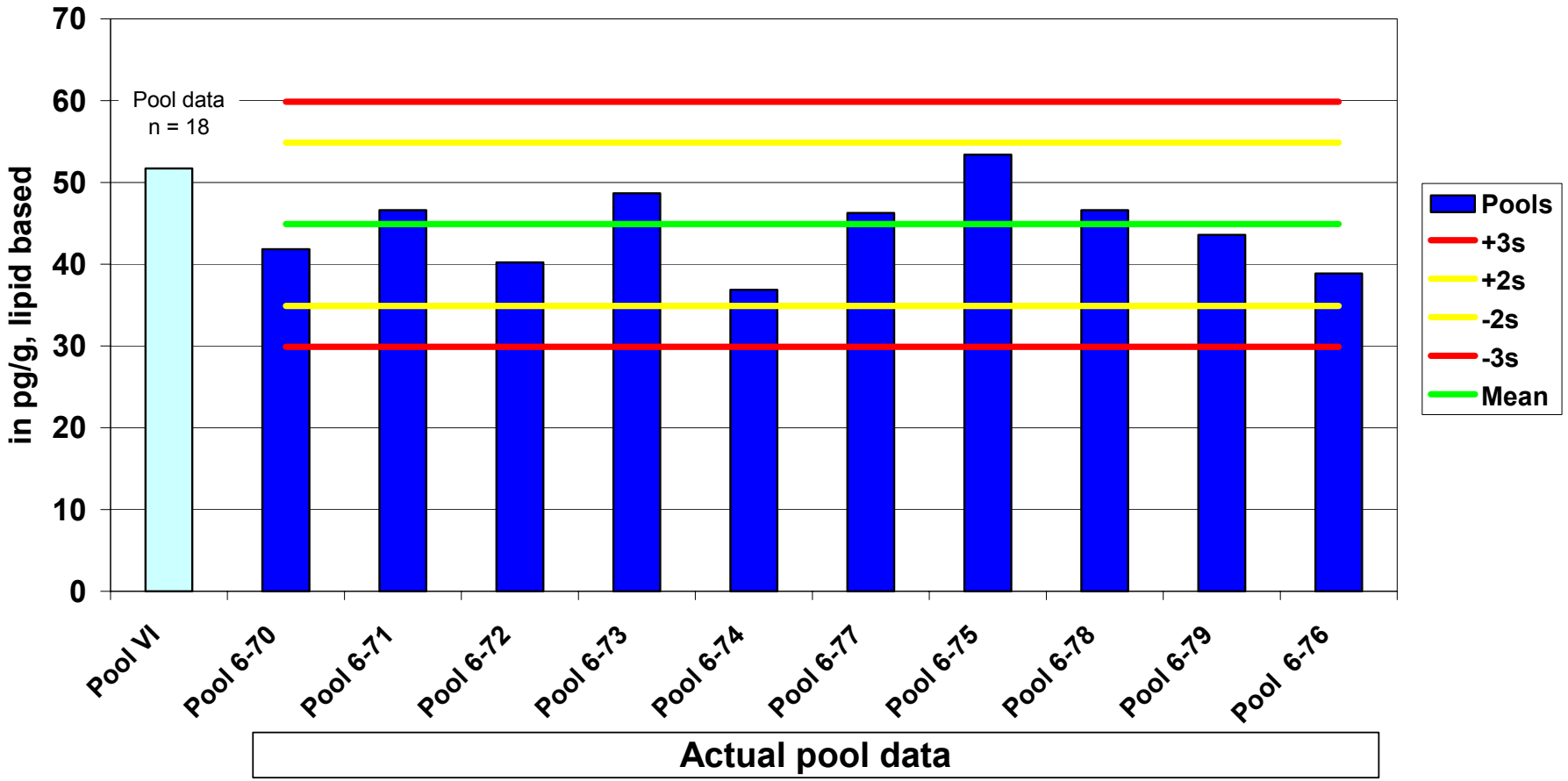
(M) = maximum value, contains possible outside contamination

Small differences on totals result from computerroundings

Quality Assurance for Human blood samples (QA-Pool)
TEQ-WHO including PCDDs/PCDFs only

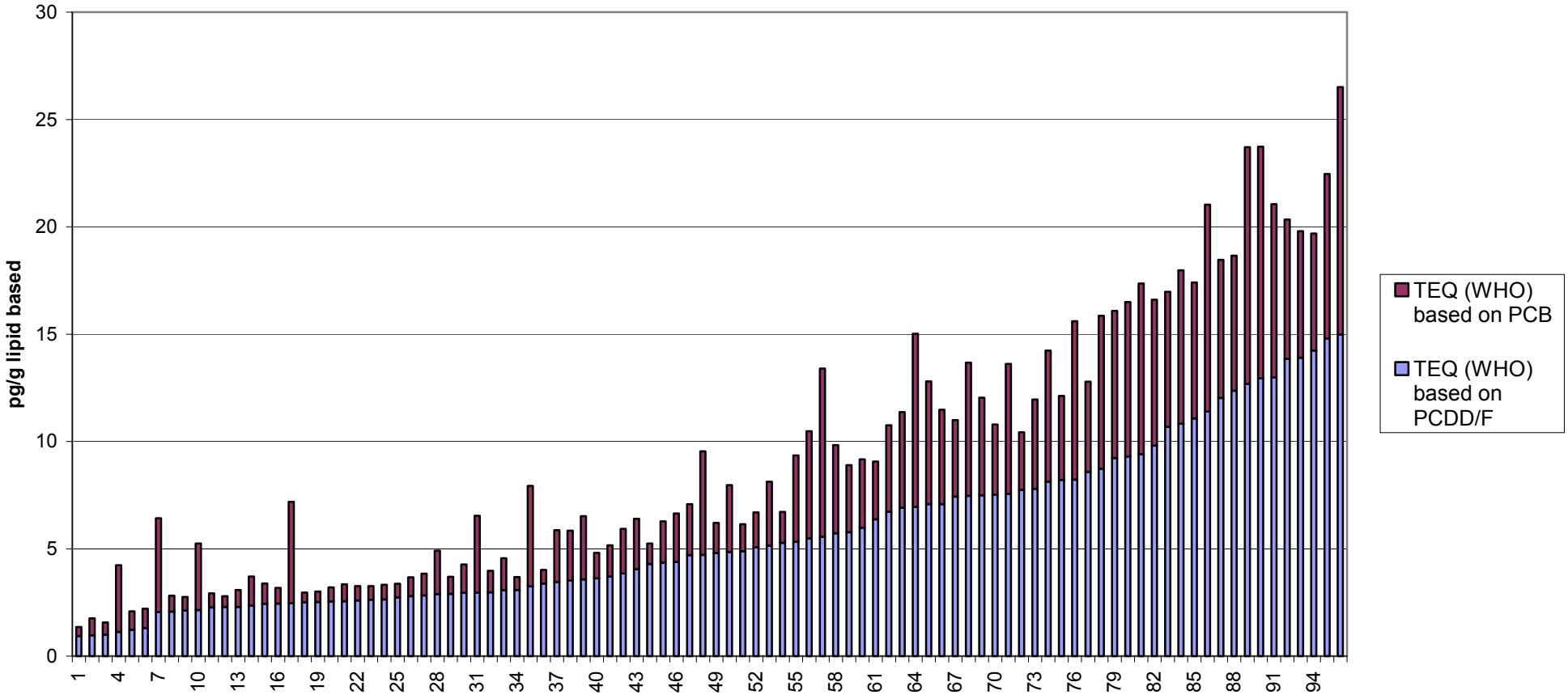


Quality Assurance for Human blood samples (QA-Pool)
TEQ-WHO including PCDDs/PCDFs and WHO-PCBs



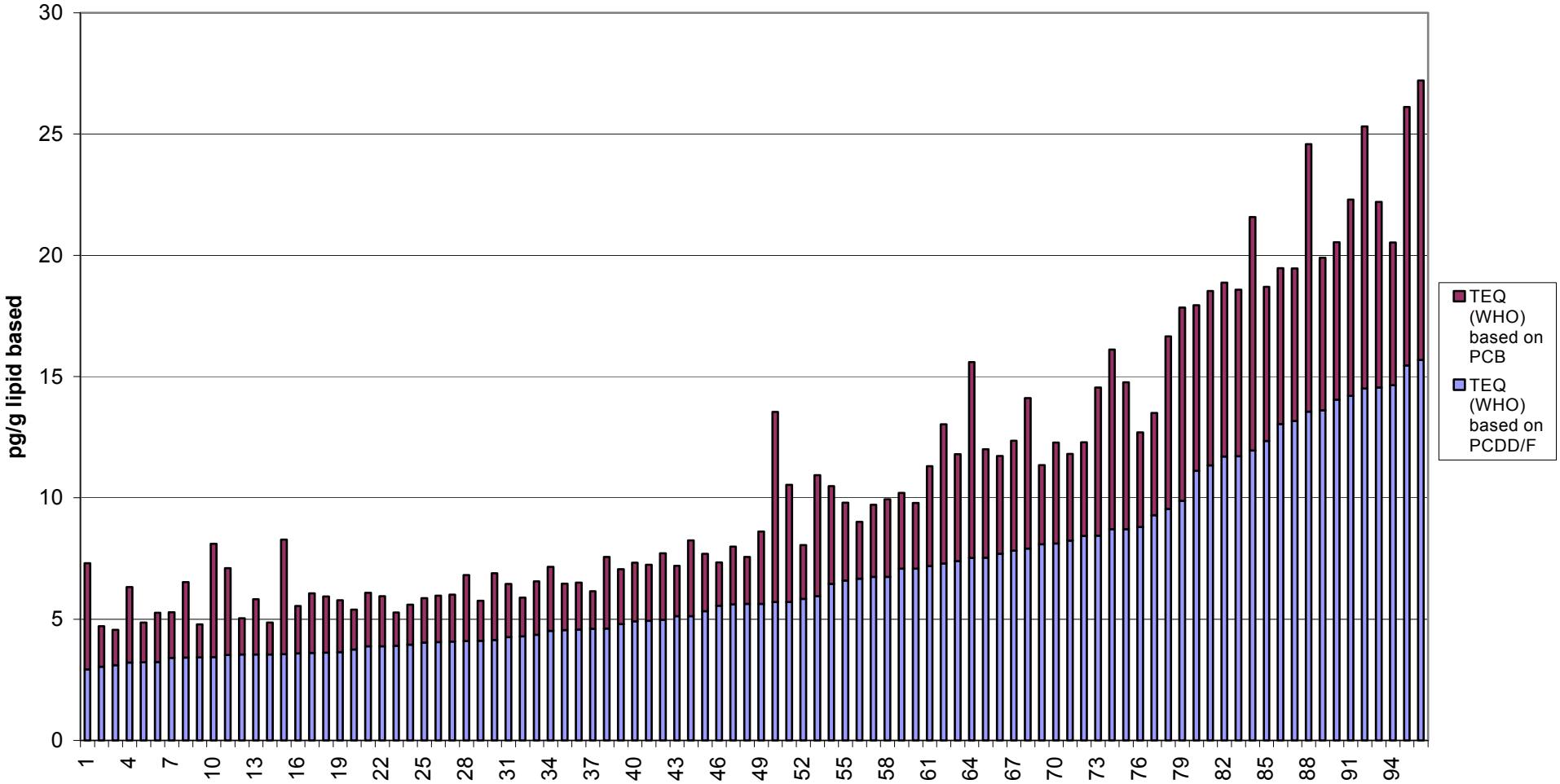
PCDD/Fs and PCBs in Australian Serum

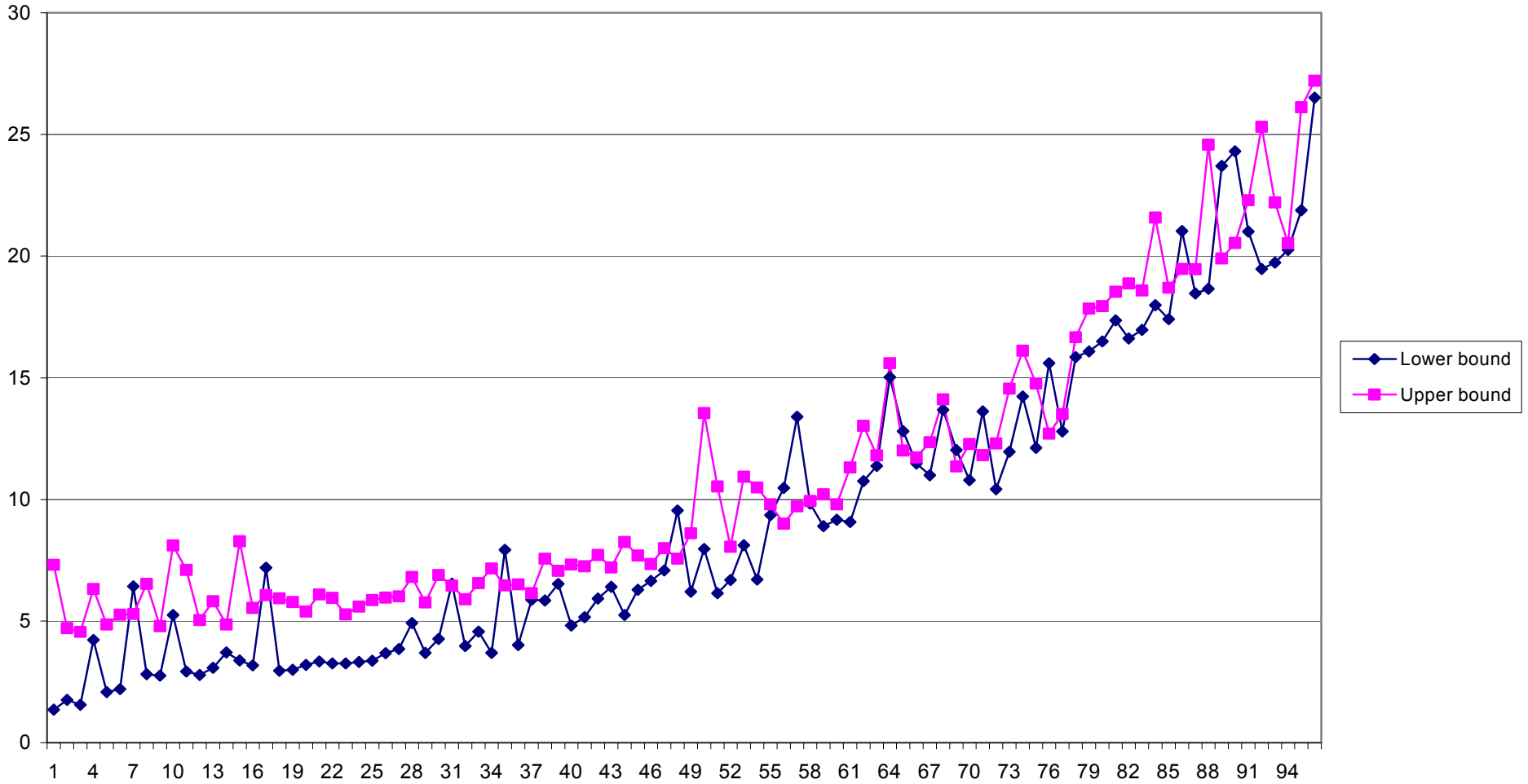
- **lower** bound calculation -



PCDD/Fs and PCBs in Australian Serum

- **upper** bound calculation -




Comparison Lower-/upperbound calculation
30
25
20
15
10
5
0
1 4 7 10 13 16 19 22 25 28 31 34 37 40 43 46 49 52 55 58 61 64 67 70 73 76 79 82 85 88 91 94
Lower bound
Upper bound

WHO QUALIFIED
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*** R.D. Stephens, C. Rappe, D.G. Hayward, M. Nygren, J. Startin A. Esbøll, J. Carlé and E.J. Yrjänheikki;** *World Health Organization International Intercalibration Study on Dioxins and Furans in Human Milk and Blood; Anal. Chem. 1992, 64, 3109-3117*

Quality Assurance Measures - PCDD/PCDF Determinations

Internal measures

- regular chemicals and glassware checks (blanks), once a block of 4/6/10 samples
- regular checks of so called instrument blanks (GC/MS)
- regular checks of quality control samples (e.g. blood pools) (GC/MS)
- daily calibration verification tests
- regular GC performance tests (separation, retention windows)
- identification based on definite abundance ratio and retention time criteria, with the use of internal and external standards
- quantitation based on the isotope dilution method with the use of internal and external standards
- regular method performance checks by analysing control samples of known PCDD/PCDF concentrations
- daily MS performance checks to control the resolution and sensitivity

External measures

- regular participation in interlaboratory quality control studies
- exchange and control measurements of standards with other qualified laboratories

Quality assurance by participation in interlaboratory quality control studies			
Components	**Matrix**	**Year**	**Organized by**
PCBs HCB HCH DDE PCP	Blood Blood Blood Blood Urine	2002	Deutsche Gesellschaft für Arbeitsmedizin e.V. Erlangen/Nürnberg
PCBs	Sludge	2003	Environment Authorities Hamburg
PCDDs/PCDFs PCBs (WHO)	Turkey, Salomon, Cheese	2003	folkehelseinstituttet Norwegian Institute of Public Health Our laboratory code: 81 Homepage: http://www.fhi.no/ publ/diverse/food2003.ht ml#TopOfPage
PCDDs/PCDFs PCB (WHO)	Cod Liver Oil	2002	Man Technology Environment Research Centre Örebro University Sweden ERGO was the reference laboratory.
PCDDs/PCDFs PCB (WHO)	Bleaching earth (used for purification of oil, fats and waxes)	2002	Safety & Environmental Assurance Centre Unilever Colworth Laboratory Sharnbrook MK44 1LQ UK **"FEDIOL Ring Trial"**
PCBs Organochloride Pesticides	Mussel Salmon	2002	Quasimeme Project Office FRS Marine Laboratory Aberdeen United Kingdom
PAH	Mussel	2002	Quasimeme Project Office FRS Marine Laboratory Aberdeen United Kingdom
Sulfurdioxide Nitricdioxide Total Organic Carbon	Air	2002	Hessisches Landesamt für Umwelt und Geologie Kassel (Environment Authorities, Hessen, Germany)
PCDDs/PCDFs PCBs (WHO)	Cod Liver Oil	2002	FAPAS Central Sience Laboratory York United Kingdom Our laboratory code: 32
PCP	Wood	2002	Landesgesundheitsamt Baden-Württemberg (Head Office Baden-Würtemberg, Germany)

Quality assurance by participation in interlaboratory quality control studies			
Components	**Matrix**	**Year**	**Organized by**
PCDDs/PCDFs PCBs (WHO)	Animal Feeding Stuff	2002	C.A.R.T. Centre for Analysis of Residues in Traces - University of Liege, Belgium Our laboratory code: 1
PCBs HCB DDE	Blood	2002	Landesgesundheitsamt Baden-Württemberg (Head Office Baden-Würtemberg, Germany)
PCDDs/PCDFs PCBs (WHO)	Tuna, pork, egg yolk	2002	folkehelseinstituttet Norwegian Institute of Public Health Our laboratory code: 18 Homepage: http://www.fhi.no/publ/diverse/food2002.html#TopOfPage
PCBs	Sludge	2002	Environment Authorities Hamburg
PCBs HCB HCH DDE DDT PCP	Blood Blood Blood Blood Blood Urine	2002	Deutsche Gesellschaft für Arbeitsmedizin e.V. Erlangen/Nürnberg
PCBs Organochloride Pesticides	mussle, fish	2002	Quasimeme Project Office FRS Marine Laboratory Aberdeen United Kingdom
Sulphate	Air	2001	KEMA Nederland B.V. Arnhem
Pb Cd Cr Co Cu Ni	Dust	2001	Hessisches Landesamt für Umwelt und Geologie Kassel (Environment Authorities, Hessen, Germany)
PAH	Air	2001	BIA (Berufsgenossenschaftliches Institut für Arbeitssicherheit)
PCBs	Sludge	2001	Environment Authorities Hamburg
PCDDs/PCDFs	Sludge	2001	Environment Authorities Hamburg
PCBs HCB HCH DDE DDT PCP	Blood Blood Blood Blood Blood Urine	2001	Deutsche Gesellschaft für Arbeitsmedizin e.V. Erlangen/Nürnberg

Quality assurance by participation in interlaboratory quality control studies			
Components	**Matrix**	**Year**	**Organized by**
PCDDs/PCDFs PCBs (WHO)	Beef, Human Milk, Fish Liver	2001	folkehelseinstituttet Norwegian Institute of Public Health Our laboratory code: 29 Homepage: http://www.fhi.no/publ/diverse/food2001.html#TopOfPage
PCDDs/PCDFs	various meat	2000	Federal Agency for Meat Research, Germany
PCDDs/PCDFs	Chicken, butter, fish	2000	folkehelseinstituttet Norwegian Institute of Public Health Our laboratory code: 23 Homepage: http://www.fhi.no/ publ/diverse/food2000.html#TopOfPage
PCBs	Sludge	2000	Environment Authorities, Hamburg
TRGS 410: PCBs HCH HCB PCP PCP	 Blood Blood Blood Blood Urine	2000	Deutsche Gesellschaft für Arbeitsmedizin e.V. Erlangen/Nürnberg Reference laboratory since 1994
PAH	Air	2000	BIA (Berufsgenossenschaftliches Institut für Arbeitssicherheit)
Sulfurdioxide Nitricdioxide Benzene	Air	2000	Environment Authorities Nordrhein-Westfalen Essen
Pb Cd Cr Co Cu Ni	Dust	2000	Hessisches Landesamt für Umwelt und Geologie Kassel (Environment Authorities, Hessen, Germany)
PCDDs/PCDFs	Sludge	2000	Environment Authorities Hamburg
PCBs HCB HCH DDE	Blood	2000	Deutsche Gesellschaft für Arbeitsmedizin e.V. Erlangen/Nürnberg
PCBs HCB DDE	Blood	2000	Landesgesundheitsamt Baden-Württemberg (Head Office Baden-Würtemberg, Germany)
PCP/HCH	Air/ Material	1999	German Engineer Association (VDI) working group "Measurements of PCP/HCH"
PCBs	Sludge	1999	Environment Authorities, Hamburg
PCP/HCH	Air/ Material	1998	German Engineer Association (VDI) working group "Measurements of PCP/HCH"

Quality assurance by participation in interlaboratory quality control studies			
Components	**Matrix**	**Year**	**Organized by**
PCBs	Sludge	1998	Environment Authorities, Hamburg
TRGS 410: PCBs HCH HCB PCP PCP	 Blood Blood Blood Blood Urine	1998	Deutsche Gesellschaft für Arbeitsmedizin e.V. Erlangen/Nürnberg
PCBs DDE HCB PCP	Blood Blood Blood Urine	1998	Landesgesundheitsamt Baden-Württemberg (Head Office Baden-Würtemberg, Germany)
Cyclophosphamid Ifosfamid	Urine	1998	Institut für Arbeits-, Sozial- und Umweltmedizin, München
PAH (EPA)	Soil and Waste	1998	Environment Authorities, Hamburg
PCDDs/PCDFs	Sludge	1998	Environment Authorities, Hamburg
Pyretroide	Airborn Particle	1998	Institut für Arbeits-, Sozial- und Umweltmedizin, Erlangen
Aldehydes	Air	1998	BIA (Berufsgenossenschaftliches Institut für Arbeitssicherheit)
PCBs	Sludge	1997	Environment Authorities, Hamburg
PCDDs/PCDFs	Sludge	1997	Environment Authorities, Hamburg
LHBC Benzene Toluene Xylene	Drinking water	1997	Environment Authorities, Hamburg
Various Solvents	Air	1997	BIA (Berufsgenossenschaftliches Institut für Arbeitssicherheit)
TRGS 410: PCBs HCH HCB PCP PCP	 Serum " " " Urine	1997	Deutsche Gesellschaft für Arbeitsmedizin e.V. Erlangen/Nürnberg
PCP/HCH	Air/ Material	1997	German Engineer Association (VDI) working group "Measurements of PCP/HCH"
Sulfurdioxide Nitricdioxide Benzene	Air	1997	Environment Authorities Nordrhein-Westfalen Essen
Pesticides	Textile	1997	Hanse Control, Hamburg
PCBs DDE HCB PCP	Blood " " Urine	1997	Landesgesundheitsamt Baden-Württemberg (Head Office Baden-Würtemberg, Germany)
HCH DDT, DDD, DDE Chlorobenzenes PCBs PAK (EPA)	Sediment from the river Elbe	1997	Landesamt für Umweltschutz Sachsen-Anhalt, ARGE-Elbe (Environment Authorities, Sachsen-Anhalt, Germany)

Quality assurance by participation in interlaboratory quality control studies			
Components	**Matrix**	**Year**	**Organized by**
PCBs	Sludge	1996	Environment Authorities, Hamburg
TRGS 410: PCBs HCH HCB PCP PCP	 Serum " " " Urine	1996	Deutsche Gesellschaft für Arbeitsmedizin e.V. Erlangen/Nürnberg Reference laboratory since 1994
PCDDs/PCDFs	Human blood	1996	Regional and State Office for Veterinary Investigations Bund/Länder AG DIOXINE
PCDDs/PCDFs	Cow's milk	1996	Regional and State Office for Veterinary Investigations Bund/Länder AG DIOXINE
PCDDs/PCDFs	Liver	1996	Ökometric, Bayreuth, Germany
PCDDs/PCDFs	Sludge	1996	Environment Authorities, Hamburg
HCH DDT, DDD, DDE PCBs PAH (EPA)	Sediment from the river Elbe	1996	Landesamt für Umweltschutz Sachsen-Anhalt, ARGE-Elbe (Environment Authorities, Sachsen-Anhalt, Germany)
PAH	Soil	1995	Environment Authorities, Hamburg
PCBs	Sludge	1995	Environment Authorities, Hamburg
PCBs, 29 Components	Food	1995	IUPAC
TRGS 410: PCBs HCH HCB PCP PCP	 Serum " " " Urine	1995	Deutsche Gesellschaft für Arbeitsmedizin e.V. Erlangen/Nürnberg Reference laboratory since 1994
PCP/HCH	Air/ Material	1995	German Engineer Association (VDI) working group "Measurements of PCP/HCH"
PCDDs/PCDFs	Sludge	1995	Environment Authorities, Hamburg
PCDDs/PCDFs	Sludge	1995	Environment Authorities, Hamburg
TRGS 410: PCBs HCH HCB PCP PCP	 Serum " " " Urine	1994	Deutsche Gesellschaft für Arbeitsmedizin e.V. Erlangen/Nürnberg Reference laboratory since 1994

Quality assurance by participation in interlaboratory quality control studies			
Components	**Matrix**	**Year**	**Organized by**
PCP/HCH	Air/ Material	1994	German Engineer Association (VDI) working group "Measurements of PCP/HCH"
PCDDs/PCDFs	Sludge	1994	Regional Authorities Pfalz, Institute for Agricultural Investigations Speyer
PCDDs/PCDFs	Compost	1994	Environment Ministery, Baden-Württemberg, University of Tübingen
Blei Cadmium Chrom Cobalt Kupfer Nickel	Dust contaminants derived from Flue gas	1994	Hessisches Landesamt für Umwelt und Geologie Kassel (Environment Authorities, Hessen, Germany)
PCBs	Sludge	1993	Federal Environment Agency (UBA)
PCBs	Sludge	1993	Environment Authorities, Hamburg
Aldehydes/ Ketones	Air	1993	Commission of the European Communities; Community Bureau of Reference (BCR)
Benzene Toluene Xylene	Air	1993	Commission of the European Communities; Community Bureau of Reference (BCR)
Dichlormethane Trichlorethane Tetrachlorethene	Air	1993	Commission of the European Communities; Community Bureau of Reference (BCR)
PCP/HCH	Air/ Material	1993	German Engineer Association (VDI) working group "Measurements of PCP/HCH"
PCDDs/PCDFs	Sludge	1993	Environment Ministery, Baden-Württemberg, University of Tübingen
PCDDs/PCDFs	Sludge	1993	Federal Environment Agency (UBA)
PCDDs/PCDFs	Soil	1993	Environment Ministery, Baden-Württemberg, University of Tübingen
Various Solvents	Air	1997	BIA (Berufsgenossenschaftliches Institut für Arbeitssicherheit)
PCBs	Indoor Air	1992	Federal Health Agency (BGA now BfR)
Aldehydes/ Ketones	Air	1992	Commission of the European Communities; Community Bureau of Reference (BCR)

Quality assurance by participation in interlaboratory quality control studies			
Components	**Matrix**	**Year**	**Organized by**
LBHC	Water	1992	Environment Authorities, Hamburg
PCDDs/PCDFs	Human blood	1992	WHO / Copenhagen
PCDDs/PCDFs	Cow's milk	1992	WHO / Copenhagen
PCDDs/PCDFs	Soil	1992	Environment Ministery, Baden-Württemberg, University of Tübingen
Mercury	Air	1992	German Engineer Association (VDI) / Landesanstalt für Immissionsschutz Nordrhein-Westfalen
PAH	Water Sediment	1991	Senatsverwaltung für Stadtentwicklung und Umweltschutz Berlin
PCDDs/PCDFs	Human blood	1991	Regional Office für Chemical Investigations, Nordrhein-Westfalen, Germany
PCDDs/PCDFs	Human blood	1990	WHO / Copenhagen
PCDDs/PCDFs	Cow's milk	1990	RIVM, Bilthoven, NL
PCDDs/PCDFs	Air	1990	Environment Authorities, Hamburg
PCDDs/PCDFs	Coffee filters	1988	Federal Health Office (BGA now BfR)
PCDDs/PCDFs	Fly ash	1986	German Engineer Association (VDI), Federal Environment Agency (UBA)
PCDDs/PCDFs	Waste-oil	1985	German Chemist Association

Table 15. Coefficient of variation (CV) weighted by TEFs from different laboratories: PCDDs and PCDFs in blood

Laboratory	CVrepeat	CVrepro	CVcombined
1	28.5	145.2	148.0
2	41.9	97.4	106.0
3	14.5	38.1	40.8
15	10.1	47.6	48.7
17	17.3	216.0	216.7
18	21.2	46.4	51.0
20	10.6	547.7	547.8
22 ◄ **ERGO**	29.7	53.6	61.3
23	36.3	133.0	137.9
25	19.8	57.5	60.8
34	46.4	94.1	105.0

Table 16. Coefficient of variation (CV) weighted by TEFs from different laboratories: PCDDs and PCDFs in cow's milk

Laboratory	CVrepeat	CVrepro	CVcombined
1	20.4	41.9	46.6
3	14.7	68.7	70.3
6	20.0	55.2	38.7
10	14.8	87.2	88.5
12	13.2	49.3	51.0
19	37.3	82.9	90.8
21	61.2	1181.7	1183.3
22 ◄ **ERGO**	15.0	28.6	32.3
23	36.2	176.4	180.1
28	28.8	202.9	205.0
30	60.1	83.0	102.4
31	26.1	110.6	113.7
32	17.0	28.7	33.4
33	14.9	67.7	69.3
34	24.7	64.4	68.9
35	13.0	31.5	34.1

List of WHO accepted laboratories for analysis of PCDDs and PCDFs in blood*

(List in alphabetical order of countries)

Organisation	**Responsible Person**
ERGO Forschungsgesellschaft mbH Hamburg, Germany	O. Päpke
Gesellschaft für Arbeitsplatz- und Umweltanalytik mbH Münster-Roxel, Germany	J. Theisen
Institut für Hygiene Ruhr-Universität Bochum, Germany	J. Wittsiepe
Department of Environmental and Toxicological Chemistry University of Amsterdam, Netherlands	K. Olie
National Institute of Public Health and Environmental Protection (RIVM) Bilthoven, Netherlands	D. Liem
Norwegian Institute for Air Research Kjeller, Norway	M. Oehme
U.S. Centers for Disease Control Toxicology Branch Atlanta, GA, USA	L. Needham

Appendix F Levels of PCDD/PCDF/PCBs in the serum of the Australian population

South, Females PCDD/PCDFs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1 and 2	Pool 2 - not applicable	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.493		0.558	0.550	0.566	0.597	0.639	0.632	0.638	0.644
2,3,7,8-Tetra-CDD	n.d.(0.4)		n.d.(0.7)	n.d.(0.6)	n.d.(0.6)	n.d.(0.7)	0.76	n.d.(0.6)	1.7	1.1
1,2,3,7,8-Penta-CDD	0.99		1.2	0.99	1.9	1.9	2.8	2.1	4.9	4.0
1,2,3,4,7,8-Hexa-CDD	n.d.(0.8)		1.4	n.d.(1)	2.1	2.7	3.1	2.4	6.3	5.5
1,2,3,6,7,8-Hexa-CDD	4.9		9	6.1	12	9.9	17	15	32	30
1,2,3,7,8,9-Hexa-CDD	1.3		2.1	1.5	1.8	2.6	3.1	2.4	6.4	6.6
1,2,3,4,6,7,8-Hepta-CDD	13		20	20	26	25	43	37	61	66
OCDD	160		225	198	279	286	503	364	671	650
2,3,7,8-Tetra-CDF	n.d.(0.3)		n.d.(0.5)	n.d.(0.5)	n.d.(0.5)	n.d.(0.5)	n.d.(0.4)	n.d.(0.4)	n.d.(0.5)	0.43
1,2,3,7,8-Penta-CDF	n.d.(0.3)		n.d.(0.6)	n.d.(0.6)	n.d.(0.6)	n.d.(0.9)	n.d.(0.5)	n.d.(0.6)	n.d.(0.6)	n.d.(0.5)
2,3,4,7,8-Penta-CDF	0.73		1.0	0.66	0.97	1.1	1.7	1.8	3.3	3.5
1,2,3,4,7,8-Hexa-CDF	0.60		0.9	0.95	n.d.(1)	n.d.(1)	1.8	1.7	2.5	2.6
1,2,3,6,7,8-Hexa-CDF	0.69		1.1	0.73	0.96	1.3	1.4	1.5	2.4	2.4
1,2,3,7,8,9-Hexa-CDF	n.d.(1)		n.d.(14)	n.d.(1)	n.d.(3)	n.d.(2)	n.d.(3)	n.d.(5)	n.d.(6)	n.d.(6)
2,3,4,6,7,8-Hexa-CDF	n.d.(2)		n.d.(2)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)
1,2,3,4,6,7,8-Hepta-CDF	3.0		2.9	2.7	2.6	2.2	n.d.(0.9)	1.7	1.4	2.1
1,2,3,4,7,8,9-Hepta-CDF	n.d.(1)		n.d.(2)	n.d.(2)	n.d.(2)	n.d.(2)	n.d.(2)	n.d.(2)	n.d.(2)	n.d.(1)
OCDF	1.00		n.d.(3)	n.d.(3)	n.d.(3)	n.d.(3)	n.d.(3)	n.d.(3)	n.d.(3)	n.d.(2)
Total PCDD/PCDF	187		263	232	327	333	578	430	792	774
Lower bound										
TEQ based on PCDD/PCDF	2.3		3.4	2.5	4.3	4.4	7.5	5.8	14	12
TEQ based on PCB	0.50		0.6	0.45	0.95	1.9	3.3	3.1	5.9	6.3
TEQ	2.8		4	3.0	5.3	6.3	11	8.9	20	19
Upper bound										
TEQ based on PCDD/PCDF	3.1		4.1	3.5	5.6	5.6	8.1	7.1	15	13
TEQ based on PCB	1.4		1.9	1.3	1.8	1.9	3.3	3.1	5.9	6.3
TEQ	4.6		6.0	4.9	7.3	7.6	11	10	21	19
Ratio upper / lower bound	163		150	164	140	120	105	115	104	104

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()

n.a. = not analysed

M = Maximum data

South, Females PCBs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1 and 2	Pool 2 - not applicable	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.493		0.558	0.550	0.566	0.597	0.639	0.632	0.638	0.644
3,3',4,4'-TCB (77)	n.d.(33)		n.d.(29)	n.d.(30)	n.d.(30)	n.d.(28)	n.d.(26)	n.d.(27)	n.d.(26)	n.d.(26)
3,4,4',5-TCB (81)	1.2		n.d.	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(2)	1.0	1.3
3,3',4,4',5-PeCB (126)	n.d.(9)		n.d.	n.d.(9)	n.d.(8)	7.9	13	12	24	25
3,3',4,4',5,5'-HxCB (169)	3.6		5	3.5	6.4	7.6	13	13	23	23
2,3,3',4,4'-PeCB (105)	206		281	226	341	403	630	608	1181	1519
2,3,4,4',5-PeCB (114)	64		67	56	92	112	211	206	445	467
2,3',4,4',5-PeCB (118)	898		1495	1316	2061	2414	4152	4050	8005	10539
2',3,4,4',5-PeCB (123)	n.d.(109)		n.d.(132)	n.d.(42)	53	n.d.(56)	58	59	132	142
2,3,3',4,4',5-HxCB (156)	513		582	367	954	1158	1905	1908	3346	3258
2,3,3',4,4',5'-HxCB (157)	125		136	97	212	252	467	446	764	759
2,3',4,4',5,5'-HxCB (167)	n.d.(132)		178	142	367	396	768	729	1278	1429
2,3,3',4,4',5,5'-HpCB (189)	46		70	43	122	138	240	243	361	351
Total non-ortho-PCB	4.8		5	3.5	6.4	15	26	25	48	50
Total mono-ortho-PCB	1852		2811	2246	4203	4873	8431	8249	15511	18463
Lower bound										
TEQ based on PCDD/PCDF	2.3		3.4	2.5	4.3	4.4	7.5	5.8	14	12
TEQ based on PCB	0.50		0.6	0.45	0.95	1.9	3.3	3.1	5.9	6.3
TEQ	2.8		4	3.0	5.3	6.3	11	8.9	20	19
Upper bound										
TEQ based on PCDD/PCDF	3.1		4.1	3.5	5.6	5.6	8.1	7.1	15	13
TEQ based on PCB	1.4		1.9	1.3	1.8	1.9	3.3	3.1	5.9	6.3
TEQ	4.6		6.0	4.9	7.3	7.6	11	10	21	19
Ratio upper / lower bound	163		150	164	140	120	105	115	104	104

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

South, Males PCDD/PCDFs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1 and 2	Pool 2 - not applicable	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.446		0.611	0.589	0.631	0.620	0.645	0.659	0.552	0.535
2,3,7,8-Tetra-CDD	n.d.(1.0)		n.d.(0.6)	n.d.(0.7)	n.d.(0.5)	n.d.(0.5)	1.0	0.79	1.2	n.d.(2)
1,2,3,7,8-Penta-CDD	1.5		1.1	1.1	1.3	1.8	3.2	2.6	3.7	4.3
1,2,3,4,7,8-Hexa-CDD	n.d.(2)		n.d.(0.9)	n.d.(1.0)	1.4	2.6	3.0	3.4	5.6	4.8
1,2,3,6,7,8-Hexa-CDD	3.9		6.6	5.7	11	11	20	19	27	28
1,2,3,7,8,9-Hexa-CDD	n.d.(2)		1.6	1.3	1.6	2.2	3.2	3.2	5.4	5.3
1,2,3,4,6,7,8-Hepta-CDD	14		18	14	20	21	26	31	40	40
OCDD	169		197	152	191	226	295	310	444	403
2,3,7,8-Tetra-CDF	n.d.(0.8)		n.d.(0.5)	n.d.(0.4)	n.d.(0.3)	n.d.(0.4)	n.d.(0.5)	n.d.(0.4)	n.d.(0.5)	n.d.(0.6)
1,2,3,7,8-Penta-CDF	n.d.(0.9)		n.d.(0.4)	n.d.(0.5)	n.d.(0.4)	n.d.(0.3)	n.d.(0.6)	n.d.(0.4)	n.d.(0.8)	n.d.(0.6)
2,3,4,7,8-Penta-CDF	0.90		1.0	0.86	1.1	1.8	2.2	1.7	2.9	3.2
1,2,3,4,7,8-Hexa-CDF	n.d.(1)		0.98	1.1	0.88	1.5	1.9	1.7	2.2	2.7
1,2,3,6,7,8-Hexa-CDF	n.d.(1)		1.0	0.65	0.84	1.3	1.3	1.7	2.2	2.2
1,2,3,7,8,9-Hexa-CDF	n.d.(2)		n.d.(1)	n.d.(1)	n.d.(3)	n.d.(3)	n.d.(5)	n.d.(6)	n.d.(10)	n.d.(10)
2,3,4,6,7,8-Hexa-CDF	n.d.(2)		n.d.(1)	n.d.(2)	n.d.(1)	n.d.(2)	n.d.(1)	n.d.(1)	n.d.(2)	n.d.(2)
1,2,3,4,6,7,8-Hepta-CDF	4.3		6.1	2.9	2.8	3.1	1.9	2.6	1.8	1.7
1,2,3,4,7,8,9-Hepta-CDF	n.d.(3)		n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)
OCDF	n.d.(5)		2.2	n.d.(1)	n.d.(2)	n.d.(1)	1.2	n.d.(1)	0.95	n.d.(2)
Total PCDD/PCDF	193		236	180	232	273	361	377	536	494
Lower bound										
TEQ based on PCDD/PCDF	2.5		2.9	2.6	3.6	4.9	8.6	7.4	11	11
TEQ based on PCB	0.48		2.0	0.63	1.2	1.3	4.2	3.6	6.3	6.3
TEQ	3.0		4.9	3.3	4.8	6.1	13	11	17	17
Upper bound										
TEQ based on PCDD/PCDF	4.6		3.9	3.7	4.6	5.8	9.3	8.2	12	14
TEQ based on PCB	1.5		2.1	1.7	1.9	2.2	4.2	3.6	6.3	6.3
TEQ	6.1		6.0	5.4	6.5	8.1	14	12	19	20
Ratio upper / lower bound	205		121	165	135	131	106	108	107	117

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

South, Males PCBs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1 and 2	Pool 2 - not applicable	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.446		0.611	0.589	0.631	0.620	0.645	0.659	0.552	0.535
3,3',4,4'-TCB (77)	n.d.(38)		n.d.(27)	n.d.(30)	n.d.(27)	n.d.(28)	n.d.(27)	n.d.(27)	n.d.(32)	n.d.(33)
3,4,4',5-TCB (81)	n.d.(2)		1.3	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	1.6	1.8
3,3',4,4',5-PeCB (126)	n.d.(11)		12	n.d.(10)	n.d.(7)	n.d.(10)	13	11	23	21
3,3',4,4',5,5'-HxCB (169)	5.0		7.6	6.8	9.2	11	20	17	28	30
2,3,3',4,4'-PeCB (105)	153		343	201	401	354	627	478	1205	1148
2,3,4,4',5-PeCB (114)	48		76	52	97	96	246	197	416	433
2,3',4,4',5-PeCB (118)	853		2592	1175	2132	2170	4147	3406	7922	7246
2',3,4,4',5-PeCB (123)	n.d.(61)		n.d.(330)	n.d.(52)	n.d.(40)	n.d.(50)	64	n.d.(74)	126	132
2,3,3',4,4',5-HxCB (156)	477		724	639	1279	1342	3328	2861	4201	4421
2,3,3',4,4',5'-HxCB (157)	122		191	145	271	310	701	630	924	1030
2,3',4,4',5,5'-HxCB (167)	143		348	173	344	390	860	765	1400	1494
2,3,3',4,4',5,5'-HpCB (189)	46		105	84	159	202	462	388	523	593
Total non-ortho-PCB	5.0		20	6.8	9.2	11	34	28	53	53
Total mono-ortho-PCB	1842		4380	2471	4684	4865	10436	8725	16716	16497
Lower bound										
TEQ based on PCDD/PCDF	2.5		2.9	2.6	3.6	4.9	8.6	7.4	11	11
TEQ based on PCB	0.48		2.0	0.63	1.2	1.3	4.2	3.6	6.3	6.3
TEQ	3.0		4.9	3.3	4.8	6.1	13	11	17	17
Upper bound										
TEQ based on PCDD/PCDF	4.6		3.9	3.7	4.6	5.8	9.3	8.2	12	14
TEQ based on PCB	1.5		2.1	1.7	1.9	2.2	4.2	3.6	6.3	6.3
TEQ	6.1		6.0	5.4	6.5	8.1	14	12	19	20
Ratio upper / lower bound	205		121	165	135	131	106	108	107	117

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

RURAL, Females PCDD/PCDFs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.545	0.499	0.524	0.542	0.577	0.556	0.647	0.659	0.615	0.566
2,3,7,8-Tetra-CDD	0.6	1.	0.6	0.6	1.0	1.	1.00	0.94	2.1	2.3
1,2,3,7,8-Penta-CDD	1.0	1.2	1.0	1.	1.6	2.4	2.7	2.7	5.4	5.2
1,2,3,4,7,8-Hexa-CDD	1.	1.	1.	1.	1.9	1.	2.4	1.7	3.3	5.1
1,2,3,6,7,8-Hexa-CDD	5.3	4.7	5.6	3.8	9.8	10	17	16	29	30
1,2,3,7,8,9-Hexa-CDD	1.5	2.4	1.8	1.8	2.6	2.8	3.4	4.2	5.5	6.4
1,2,3,4,6,7,8-Hepta-CDD	11	13	13	12	22	24	32	34	47	48
OCDD	163	165	166	178	280	309	346	348	503	499
2,3,7,8-Tetra-CDF	0.7	0.5	0.6	0.5	0.4	0.5	0.5	0.5	0.5	1.
1,2,3,7,8-Penta-CDF	0.6	0.5	0.89	0.5	0.7	0.83	0.6	0.5	0.4	3.
2,3,4,7,8-Penta-CDF	0.83	0.70	0.78	0.80	1.3	1.4	2.1	2.0	3.8	4.4
1,2,3,4,7,8-Hexa-CDF	1.0	0.7	1.00	0.8	1.5	1.5	2.0	1.6	3.1	3.5
1,2,3,6,7,8-Hexa-CDF	1.0	0.7	0.74	0.7	1.2	1.3	1.8	1.7	2.3	2.9
1,2,3,7,8,9-Hexa-CDF	1.	1.	1.	1.	1.	1.	1.	1.	1.	3.
2,3,4,6,7,8-Hexa-CDF	1.	2.	2.	1.	1.	1.	1.	1.	1.	1.
1,2,3,4,6,7,8-Hepta-CDF	2.7	1.9	2.2	1.8	5.9	2.5	2.0	2.1	1.8	2.4
1,2,3,4,7,8,9-Hepta-CDF	1.	2.	2.	2.	2.	2.	2.	2.	2.	3.
OCDF	3.	3.	3.	4.	15	5.	3.	4.	3.	7.
Total PCDD/PCDF	188	189	193	198	343	356	413	414	606	609
Lower bound										
TEQ based on PCDD/PCDF	2.5	2.4	2.6	1.1	5.3	5.1	7.8	7.5	14	15
TEQ based on PCB	4.7	0.95	0.78	3.1	1.4	1.6	4.2	4.5	7.7	12
TEQ	7.2	3.4	3.3	4.2	6.7	6.7	12	12	22	27
Upper bound										
TEQ based on PCDD/PCDF	3.6	4.1	3.6	3.4	5.6	6.6	8.1	7.8	15	16
TEQ based on PCB	4.7	2.7	2.5	3.1	3	3.2	4.2	4.5	7.7	12
TEQ	8.3	6.8	6.1	6.5	8.6	9.8	12	12	22	27
Ratio upper / lower bound	115	201	182	154	128	147	103	103	101	103

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

RURAL, Females PCBs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.545	0.499	0.524	0.542	0.577	0.556	0.647	0.659	0.615	0.566
3,3',4,4'-TCB (77)	n.a.	n.d.(42)	40.	39.	37.	38.	33.	32.	n.d.(34)	n.a.
3,4,4',5-TCB (81)	n.a.	n.d.(1)	1.	1.	1.	1.	1.	1.	1.2	n.a.
3,3',4,4',5-PeCB (126)	30	18.	17.	25	15.	16.	16	18	32	50
3,3',4,4',5,5'-HxCB (169)	6.5	4.1	4.8	3.8	8.0	9.6	14	16	24	31
2,3,3',4,4'-PeCB (105)	694	603	308	349	572	540	930	906	1918	2700
2,3,4,4',5-PeCB (114)	128	130	76	67	179	184	337	317	673	921
2,3',4,4',5-PeCB (118)	3865	2769	1552	1573	3046	2923	5497	5471	11030	15023
2',3,4,4',5-PeCB (123)	122	43	55.	54.	30	52	63	76	168	284
2,3,3',4,4',5-HxCB (156)	1826	796	823	555	1450	1732	2572	2884	4151	6325
2,3,3',4,4',5'-HxCB (157)	280	181	169	120	314	385	559	591	941	1345
2,3',4,4',5,5'-HxCB (167)	920	304	343	188	445	548	763	880	1399	2283
2,3,3',4,4',5,5'-HpCB (189)	246	59	94	53	143	175	230	282	353	500
Total non-ortho-PCB	37	4.1	4.8	29	8.0	9.6	30	34	57	82
Total mono-ortho-PCB	8080	4885	3364	2903	6178	6540	10951	11407	20633	29380
Lower bound										
TEQ based on PCDD/PCDF	2.5	2.4	2.6	1.1	5.3	5.1	7.8	7.5	14	15
TEQ based on PCB	4.7	0.95	0.78	3.1	1.4	1.6	4.2	4.5	7.7	12
TEQ	7.2	3.4	3.3	4.2	6.7	6.7	12	12	22	27
Upper bound										
TEQ based on PCDD/PCDF	3.6	4.1	3.6	3.4	5.6	6.6	8.1	7.8	15	16
TEQ based on PCB	4.7	2.7	2.5	3.1	3	3.2	4.2	4.5	7.7	12
TEQ	8.3	6.8	6.1	6.5	8.6	9.8	12	12	22	27
Ratio upper / lower bound	115	201	182	154	128	147	103	103	101	103

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

RURAL, Males PCDD/PCDFs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.512	0.485	0.548	0.498	0.713	0.675	0.752	0.724	0.625	0.591
2,3,7,8-Tetra-CDD	0.8	0.7	0.6	0.8	0.67	0.6	0.99	1.1	1.1	1.7
1,2,3,7,8-Penta-CDD	1.1	1.3	0.81	1.4	1.7	2.1	2.6	2.6	2.8	3.9
1,2,3,4,7,8-Hexa-CDD	2.	2.	1.	2.	1.	1.5	2.3	1.9	3.1	3.4
1,2,3,6,7,8-Hexa-CDD	5.2	5.5	5.0	6.1	7.7	12	16	14	19	21
1,2,3,7,8,9-Hexa-CDD	1.7	1.5	1.7	1.9	1.9	1.0	2.7	2.6	3.2	4.4
1,2,3,4,6,7,8-Hepta-CDD	13	11	14	13	16	18	23	20	27	33
OCDD	166	161	180	160	177	181	228	213	294	306
2,3,7,8-Tetra-CDF	0.4	0.4	0.6	0.4	0.30	0.4	0.38	0.4	0.4	0.41
1,2,3,7,8-Penta-CDF	0.6	0.5	0.5	0.5	0.5	0.4	0.5	0.3	0.3	0.4
2,3,4,7,8-Penta-CDF	1.1	0.87	0.85	1.0	1.2	1.4	1.6	1.8	2.4	3.1
1,2,3,4,7,8-Hexa-CDF	1.1	1.	0.94	0.9	0.77	1.1	1.2	1.2	1.6	2.0
1,2,3,6,7,8-Hexa-CDF	1.3	0.8	1.1	0.95	1.2	1.2	1.5	1.4	1.7	2.0
1,2,3,7,8,9-Hexa-CDF	2.	2.	2.	1.	1.	1.	2.	5.	3.	7.
2,3,4,6,7,8-Hexa-CDF	1.	1.	1.	1.	1.	1.	0.9	1.	1.	1.
1,2,3,4,6,7,8-Hepta-CDF	5.4	3.7	2.7	5.8	2.7	2.6	2.4	2.6	1.4	1.9
1,2,3,4,7,8,9-Hepta-CDF	3.	2.	2.	3.	2.	2.	2.	1.	1.	2.
OCDF	8.	5.	5.	6.	5.	3.	5.	2.3	2.	5.
Total PCDD/PCDF	196	185	207	190	212	221	283	264	357	383
Lower bound										
TEQ based on PCDD/PCDF	2.8	2.6	2.3	3.0	4.4	4.7	7.1	6.9	8.2	11
TEQ based on PCB	1.0	0.66	0.79	1.0	2.3	2.4	4.4	4.5	7.4	7.1
TEQ	3.8	3.3	3.1	4.0	6.6	7.1	11	11	16	18
Upper bound										
TEQ based on PCDD/PCDF	4.3	4.1	3.4	4.4	4.8	5.6	7.4	7.5	8.7	12
TEQ based on PCB	2.2	1.9	1.9	2.2	2.3	2.4	4.4	4.5	7.4	7.2
TEQ	6.5	6	5.3	6.6	7.1	8	12	12	16	19
Ratio upper / lower bound	168	183	172	165	106	113	103	106	103	105

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed

RURAL, Males PCBs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.512	0.485	0.548	0.498	0.713	0.675	0.752	0.724	0.625	0.591
3,3',4,4'-TCB (77)	n.d.(28)	n.d.(30)	n.d.(27)	n.d.(29)	n.d.(21)	n.d.(22)	n.d.(19)	n.d.(20)	n.d.(23)	n.d.(24)
3,4,4',5-TCB (81)	1.4	n.d.(1)	1.5	1.5	1.0	1.2	2.0	4.3	2.1	2.2
3,3',4,4',5-PeCB (126)	n.d.(11)	n.d.(12)	n.d.(11)	n.d.(12)	10	11	17	16	27	30
3,3',4,4',5,5'-HxCB (169)	4.6	4.9	5.6	6.2	9.7	9.1	16	17	22	25
2,3,3',4,4'-PeCB (105)	478	295	307	492	427	542	871	799	1398	1271
2,3,4,4',5-PeCB (114)	134	81	74	106	129	132	281	319	561	473
2,3',4,4',5-PeCB (118)	2377	1431	1991	2148	1980	2293	4738	4451	8569	7984
2',3,4,4',5-PeCB (123)	n.d.(153)	n.d.(81)	n.d.(177)	n.d.(110)	n.d.(98)	n.d.(82)	n.d.(134)	n.d.(98)	n.d.(202)	n.d.(191)
2,3,3',4,4',5-HxCB (156)	990	642	750	1003	1301	1335	2990	3204	5139	4493
2,3,3',4,4',5'-HxCB (157)	258	163	161	238	308	309	684	706	1091	958
2,3',4,4',5,5'-HxCB (167)	226	n.d.(268)	217	n.d.(341)	222	201	688	694	1316	1231
2,3,3',4,4',5,5'-HpCB (189)	41	n.d.(66)	69	64	123	128	301	321	403	431
Total non-ortho-PCB	6.0	4.9	7.0	7.7	21	21	35	38	51	57
Total mono-ortho-PCB	4503	2612	3569	4051	4490	4940	10552	10494	18477	16841
Lower bound										
TEQ based on PCDD/PCDF	2.8	2.6	2.3	3.0	4.4	4.7	7.1	6.9	8.2	11
TEQ based on PCB	1.0	0.66	0.79	1.0	2.3	2.4	4.4	4.5	7.4	7.1
TEQ	3.8	3.3	3.1	4.0	6.6	7.1	11	11	16	18
Upper bound										
TEQ based on PCDD/PCDF	4.3	4.1	3.4	4.4	4.8	5.6	7.4	7.5	8.7	12
TEQ based on PCB	2.2	1.9	1.9	2.2	2.3	2.4	4.4	4.5	7.4	7.2
TEQ	6.5	6.0	5.3	6.6	7.1	8.0	12	12	16	19
Ratio upper / lower bound	168	183	172	165	106	113	103	106	103	105

Data in pg g^{-1} lipid based

n.d. = not detected,
detection limit in ()
n.a. = not analysed

North East, Females PCDD/PCDFs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.488	0.492	0.571	0.553	0.651	0.605	0.665	0.652	0.652	0.639
2,3,7,8-Tetra-CDD	n.d.(0.7)	n.d.(0.7)	n.d.(0.6)	n.d.(0.5)	n.d.(1)	n.d.(0.6)	1.2	1.1	2.3	1.8
1,2,3,7,8-Penta-CDD	1.0	n.d.(0.8)	1.5	1.2	2.1	1.3	2.9	2.7	4.1	4.8
1,2,3,4,7,8-Hexa-CDD	n.d.(1)	n.d.(1)	n.d.(0.8)	n.d.(1.0)	1.7	n.d.(2)	2.6	2.3	3.2	3.4
1,2,3,6,7,8-Hexa-CDD	4.8	3.8	5.8	7.0	11	9.6	16	18	28	27
1,2,3,7,8,9-Hexa-CDD	1.4	1.4	2.0	2.0	2.3	1.9	3.5	3.1	5.4	4.1
1,2,3,4,6,7,8-Hepta-CDD	11	13	15	16	20	20	30	27	43	46
OCDD	141	141	161	172	233	239	344	278	409	480
2,3,7,8-Tetra-CDF	n.d.(0.4)	n.d.(0.5)	n.d.(0.4)	n.d.(0.4)	n.d.(0.4)	n.d.(0.4)	n.d.(0.4)	n.d.(0.4)	n.d.(0.4)	n.d.(0.4)
1,2,3,7,8-Penta-CDF	n.d.(0.4)	n.d.(0.7)	n.d.(0.4)	n.d.(0.4)	n.d.(0.5)	n.d.(0.6)	n.d.(0.3)	n.d.(0.4)	n.d.(0.5)	n.d.(0.4)
2,3,4,7,8-Penta-CDF	0.97	0.62	0.99	0.82	1.4	1.5	2.2	2.0	3.4	3.7
1,2,3,4,7,8-Hexa-CDF	0.89	n.d.(0.9)	0.80	0.76	1.4	1.2	2.0	1.9	2.7	2.8
1,2,3,6,7,8-Hexa-CDF	0.88	n.d.(0.7)	0.68	0.88	1.1	1.2	1.5	1.6	2.3	1.8
1,2,3,7,8,9-Hexa-CDF	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(2)	n.d.(3)	n.d.(3)	n.d.(4)	n.d.(5)	n.d.(7)	n.d.(11)
2,3,4,6,7,8-Hexa-CDF	n.d.(2)	n.d.(1)	n.d.(2)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)
1,2,3,4,6,7,8-Hepta-CDF	3.0	1.9	2.9	2.1	2.0	1.5	1.7	1.7	1.8	1.3
1,2,3,4,7,8,9-Hepta-CDF	n.d.(1)	n.d.(2)	n.d.(1)	n.d.(2)	n.d.(1)	n.d.(2)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(2)
OCDF	n.d.(2)	n.d.(5)	n.d.(2)	n.d.(3)	n.d.(1)	n.d.(5)	n.d.(1)	n.d.(2)	n.d.(3)	n.d.(5)
Total PCDD/PCDF	165	161	191	202	276	278	408	339	505	577
Lower bound										
TEQ based on PCDD/PCDF	2.4	0.99	3.1	2.9	4.8	3.7	8.2	7.7	13	13
TEQ based on PCB	0.74	0.57	0.61	0.80	1.4	1.4	4.8	2.7	11	8.1
TEQ	3.2	1.6	3.7	3.7	6.2	5.2	13	10	24	21
Upper bound										
TEQ based on PCDD/PCDF	3.6	3.2	4.1	3.9	6.7	5.0	9.2	8.4	14	14
TEQ based on PCB	2.0	2	1.7	2.2	2.3	2.7	4.7	3.9	11	8.1
TEQ	5.5	5.3	5.8	6.1	9.0	7.7	14	12	25	22
Ratio upper / lower bound	174	337	156	165	145	149	108	118	104	106

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

North East, Females PCBs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.488	0.492	0.571	0.553	0.651	0.605	0.665	0.652	0.652	0.639
3,3',4,4'-TCB (77)	n.d.(33)	n.d.(32)	n.d.(28)	n.d.(29)	n.d.(27)	n.d.(25)	n.d.(27)	n.d.(24)	n.d.(32)	n.d.(24)
3,4,4',5-TCB (81)	n.d.(2)	n.d.(1)	n.d.(1)	n.d.(2)	n.d.(2)	n.d.(1)	n.d.(2)	n.d.(1)	2.3	1.4
3,3',4,4',5-PeCB (126)	n.d.(12)	n.d.(15)	n.d.(10)	n.d.(14)	n.d.(9)	n.d.(13)	8.6	n.d.(12)	39	31
3,3',4,4',5,5'-HxCB (169)	5.0	3.4	4.3	4.8	8.2	7.5	14	15	24	23
2,3,3',4,4'-PeCB (105)	226	219	297	370	566	601	1044	898	2707	1997
2,3,4,4',5-PeCB (114)	84	69	72	92	167	158	389	327	1053	669
2,3',4,4',5-PeCB (118)	1346	1063	1422	1693	2964	3198	6308	5646	15428	12042
2',3,4,4',5-PeCB (123)	n.d.(64)	n.d.(41)	n.d.(50)	n.d.(45)	n.d.(69)	n.d.(52)	96	60	241	112
2,3,3',4,4',5-HxCB (156)	788	601	571	805	1448	1499	3170	2760	7342	4883
2,3,3',4,4',5'-HxCB (157)	166	131	129	168	294	296	687	576	1519	1003
2,3',4,4',5,5'-HxCB (167)	195	141	171	195	408	434	889	812	2042	1563
2,3,3',4,4',5,5'-HpCB (189)	69	38	52	68	138	144	266	259	464	411
Total PCDD/PCDF	165	161	191	202	276	278	408	339	505	577
Total non-ortho-PCB	5.0	3.4	4.3	4.8	8.2	7.5	23	15	65	55
Total mono-ortho-PCB	2875	2262	2715	3391	5986	6330	12849	11337	30798	22680
Lower bound										
TEQ based on PCDD/PCDF	2.4	0.99	3.1	2.9	4.8	3.7	8.2	7.7	13	13
TEQ based on PCB	0.74	0.57	0.61	0.80	1.4	1.4	3.9	2.7	11	8.1
TEQ	3.2	1.6	3.7	3.7	6.2	5.2	12	10	24	21
Upper bound										
TEQ based on PCDD/PCDF	3.6	3.2	4.1	3.9	6.7	5.0	8.8	8.4	14	14
TEQ based on PCB	2.0	2	1.7	2.2	2.3	2.7	3.9	3.9	11	8.1
TEQ	5.5	5.3	5.8	6.1	9.0	7.7	13	12	25	22
Ratio upper / lower bound	174	337	156	165	145	149	105	118	104	106

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

North East, Males PCDD/PCDFs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.486	0.480	0.618	0.566	0.680	0.699	0.785	0.719	0.656	0.579
2,3,7,8-Tetra-CDD	n.d.(0.6)	n.d.(0.7)	n.d.(0.7)	n.d.(0.6)	n.d.(0.5)	n.d.(0.6)	0.96	1.1	1.3	1.1
1,2,3,7,8-Penta-CDD	1.3	1.4	n.d.(0.9)	1.1	1.2	1.4	2.7	2.9	3.1	3.6
1,2,3,4,7,8-Hexa-CDD	n.d.(2)	n.d.(2)	n.d.(1)	n.d.(2)	n.d.(1)	n.d.(2)	1.9	2.1	2.5	n.d.(2)
1,2,3,6,7,8-Hexa-CDD	5.8		5.4	3.5	9.2	8.9	14	15	19	21
1,2,3,7,8,9-Hexa-CDD	1.6	n.d.(1)	n.d.(1)	1.4	n.d.(1)	n.d.(1)	2.2	2.5	2.7	3.2
1,2,3,4,6,7,8-Hepta-CDD	12	9.7	11	10	14	16	20	23	26	32
OCDD	168	125	134	120	150	170	204	257	260	314
2,3,7,8-Tetra-CDF	n.d.(0.4)	n.d.(0.7)	n.d.(0.4)	n.d.(0.4)	n.d.(0.3)	n.d.(0.5)	n.d.(0.3)	n.d.(0.7)	n.d.(0.5)	n.d.(0.7)
1,2,3,7,8-Penta-CDF	n.d.(0.5)	n.d.(0.5)	n.d.(0.4)	n.d.(0.5)	n.d.(0.4)	n.d.(0.4)	n.d.(0.4)	n.d.(0.4)	n.d.(0.3)	n.d.(0.6)
2,3,4,7,8-Penta-CDF	0.81	0.88	1.1	0.96	1.3	1.4	1.6	2.1	2.4	2.8
1,2,3,4,7,8-Hexa-CDF	n.d.(1)	1.1	n.d.(0.8)	n.d.(0.9)	n.d.(1)	1.7	1.2	2.0	2.2	2.2
1,2,3,6,7,8-Hexa-CDF	0.91	0.84	0.91	0.84	0.98	1.0	1.2	1.3	2.0	2.1
1,2,3,7,8,9-Hexa-CDF	n.d.(2)	n.d.(1)	n.d.(2)	n.d.(2)	n.d.(4)	n.d.(3)	n.d.(8)	n.d.(9)	n.d.(6)	n.d.(15)
2,3,4,6,7,8-Hexa-CDF	n.d.(1)	n.d.(2)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(0.9)	n.d.(1)	n.d.(1)	n.d.(1)
1,2,3,4,6,7,8-Hepta-CDF	4.7	4.5	2.6	2.2	1.2	1.8	1.3	2.2	1.7	2.2
1,2,3,4,7,8,9-Hepta-CDF	n.d.(3)	n.d.(2)	n.d.(3)	n.d.(2)	n.d.(3)	n.d.(2)	n.d.(3)	n.d.(3)	n.d.(1)	n.d.(4)
OCDF	n.d.(7)	n.d.(4)	n.d.(6)	n.d.(5)	n.d.(5)	n.d.(5)	n.d.(5)	n.d.(6)	n.d.(2)	n.d.(9)
Total PCDD/PCDF	195	147	155	140	178	203	251	311	324	383
Lower bound										
TEQ based on PCDD/PCDF	2.7	2.5	1.3	2.3	3.1	3.5	6.7	7.6	8.7	9.3
TEQ based on PCB	0.65	0.65	0.90	0.66	1.5	2.4	4.0	6.0	7.1	7.2
TEQ	3.4	3.2	2.2	2.9	4.6	5.9	11	14	16	16
Upper bound										
TEQ based on PCDD/PCDF	4.0	3.9	3.6	3.5	4.5	4.9	7.7	8.7	9.5	11
TEQ based on PCB	1.8	1.6	2.1	1.5	2.6	2.4	4.0	6.1	7.1	7.2
TEQ	5.9	5.6	5.8	5	7.2	7.3	12	15	17	19
Ratio upper / lower bound	174	175	263	172	157	125	109	108	105	112

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

North East, Males PCBs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.486	0.480	0.618	0.566	0.680	0.699	0.785	0.719	0.656	0.579
3,3',4,4'-TCB (77)	n.d.(25)	n.d.(35)	n.d.(28)	n.d.(29)	n.d.(20)	n.d.(24)	n.d.(18)	n.d.(23)	n.d.(25)	n.d.(29)
3,4,4',5-TCB (81)	n.d.(1)	n.d.(1)	n.d.(2)	n.d.(2)	n.d.(1)	n.d.(1)	n.d.(1)	1.7	1.1	2.3
3,3',4,4',5-PeCB (126)	n.d.(12)	n.d.(10)	n.d.(12)	n.d.(8)	n.d.(11)	8.3	10	19	27	25
3,3',4,4',5,5'-HxCB (169)	4.9	4.9	6.5	5.8	11	11	18	19	24	24
2,3,3',4,4'-PeCB (105)	200	233	296	245	354	447	682	1217	1307	1316
2,3,4,4',5-PeCB (114)	84	77	83	63	124	153	302	400	476	495
2,3',4,4',5-PeCB (118)	971	1268	1449	1246	2012	2431	4341	7385	8372	8700
2',3,4,4',5-PeCB (123)	n.d.(56)	n.d.(59)	n.d.(67)	n.d.(44)	n.d.(50)	n.d.(52)	n.d.(64)	96	134	111
2,3,3',4,4',5-HxCB (156)	699	657	1008	682	1786	1824	3547	4707	4798	5250
2,3,3',4,4',5'-HxCB (157)	164	157	200	144	353	359	700	947	1007	1074
2,3',4,4',5,5'-HxCB (167)	119	185	168	182	336	399	716	1175	1327	1424
2,3,3',4,4',5,5'-HpCB (189)	50	54	92	68	189	194	328	419	464	487
Total non-ortho-PCB	4.9	4.9	6.5	5.8	11	19	28	39	52	51
Total mono-ortho-PCB	2288	2632	3296	2629	5155	5806	10617	16347	17885	18858
Lower bound										
TEQ based on PCDD/PCDF	2.7	2.5	1.3	2.3	3.1	3.5	6.7	7.6	8.7	9.3
TEQ based on PCB	0.65	0.65	0.90	0.66	1.5	2.4	4.0	6.0	7.1	7.2
TEQ	3.4	3.2	2.2	2.9	4.6	5.9	11	14	16	16
Upper bound										
TEQ based on PCDD/PCDF	4.0	3.9	3.6	3.5	4.5	4.9	7.7	8.7	9.5	11
TEQ based on PCB	1.8	1.6	2.1	1.5	2.6	2.4	4.0	6.1	7.1	7.2
TEQ	5.9	5.6	5.8	5	7.2	7.3	12	15	17	19
Ratio upper / lower bound	174	175	263	172	157	125	109	108	105	112

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

West, female PCDD/PCDFs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1 and 2	Pool 2 - not applicable	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.496		0.566	0.572	0.547	0.580	0.667	0.706	0.606	0.633
2,3,7,8-Tetra-CDD	n.d.(1)		n.d.(0.6)	n.d.(0.7)	n.d.(0.7)	n.d.(0.8)	n.d.(0.7)	0.57	1.0	1.3
1,2,3,7,8-Penta-CDD	n.d.(1)		0.97	1.2	1.3	1.3	2.0	2.0	3.1	3.9
1,2,3,4,7,8-Hexa-CDD	n.d.(2)		n.d.(1)	n.d.(1)	n.d.(1)	1.9	n.d.(1)	2.3	3.2	3.2
1,2,3,6,7,8-Hexa-CDD	3.3		4.8	5.9	7.4	9.7	14	13	37	38
1,2,3,7,8,9-Hexa-CDD	n.d.(1)		1.2	1.2	2.0	1.5	1.7	2.6	4.9	5.0
1,2,3,4,6,7,8-Hepta-CDD	12		16	17	21	25	27	35	63	56
OCDD	125		157	166	242	268	292	336	514	457
2,3,7,8-Tetra-CDF	n.d.(0.8)		n.d.(0.4)	n.d.(0.5)	n.d.(0.4)	n.d.(0.6)	n.d.(0.4)	n.d.(0.3)	n.d.(0.4)	n.d.(0.4)
1,2,3,7,8-Penta-CDF	n.d.(0.8)		n.d.(0.6)	n.d.(0.5)	n.d.(0.7)	n.d.(0.6)	n.d.(0.5)	n.d.(0.4)	n.d.(0.5)	n.d.(0.4)
2,3,4,7,8-Penta-CDF	0.80		0.95	0.89	1.4	1.3	1.8	1.8	4.1	3.9
1,2,3,4,7,8-Hexa-CDF	n.d.(1)		n.d.(0.7)	n.d.(0.8)	1.2	1.4	2.0	1.6	3.1	2.6
1,2,3,6,7,8-Hexa-CDF	n.d.(0.8)		0.84	0.78	1.4	1.4	1.3	1.4	3.1	2.1
1,2,3,7,8,9-Hexa-CDF	n.d.(2)		n.d.(1)	n.d.(1)	n.d.(3)	n.d.(3)	n.d.(6)	n.d.(6)	n.d.(8)	n.d.(9)
2,3,4,6,7,8-Hexa-CDF	n.d.(2)		n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1.0)	n.d.(0.9)	n.d.(1)	n.d.(1)
1,2,3,4,6,7,8-Hepta-CDF	6.3		4.3	4.1	4.0	2.0	2.0	4.6	2.1	1.9
1,2,3,4,7,8,9-Hepta-CDF	n.d.(2)		n.d.(1)	n.d.(2)	n.d.(1)	n.d.(2)	n.d.(2)	n.d.(1)	n.d.(1)	n.d.(2)
OCDF	n.d.(4)		n.d.(2)	n.d.(3)	n.d.(2)	n.d.(4)	n.d.(3)	n.d.(3)	n.d.(2)	n.d.(4)
Total PCDD/PCDF	148		186	197	281	313	344	400	638	576
Lower bound										
TEQ based on PCDD/PCDF	0.93		2.4	2.6	3.5	3.8	5.1	6.0	12	13
TEQ based on PCB	0.43		1.4	0.68	2.3	2.1	3.0	3.2	6.4	6.5
TEQ	1.4		3.7	3.3	5.8	5.9	8.1	9.2	18	19
Upper bound										
TEQ based on PCDD/PCDF	4.3		3.4	3.9	4.9	5.1	6.7	6.7	13	14
TEQ based on PCB	1.6		1.4	1.4	2.3	2.1	3.0	3.2	6.4	6.5
TEQ	5.9		4.8	5.3	7.2	7.2	9.7	9.9	19	21
Ratio upper / lower bound	433		129	159	124	122	120	108	105	105

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

West, female PCBs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1 and 2	Pool 2 - not applicable	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.496		0.566	0.572	0.547	0.580	0.667	0.706	0.606	0.633
3,3',4,4'-TCB (77)	n.d.(33)		n.d.(20)	n.d.(20)	n.d.(21)	n.d.(19)	n.d.(17)	n.d.(16)	n.d.(19)	n.d.(18)
3,4,4',5-TCB (81)	n.d.(1)		n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	1.8	1.2
3,3',4,4',5-PeCB (126)	n.d.(12)		7.8	n.d.(7)	10	9.6	10	12	28	31
3,3',4,4',5,5'-HxCB (169)	4.1		4.4	5.4	9.6	9.4	16	15	25	24
2,3,3',4,4'-PeCB (105)	148		289	257	434	402	477	550	1442	1412
2,3,4,4',5-PeCB (114)	46		67	73	137	111	202	236	477	457
2,3',4,4',5-PeCB (118)	847		1490	1368	2569	2472	3284	3573	8891	8982
2',3,4,4',5-PeCB (123)	n.d.(113)		n.d.(53)	n.d.(54)	n.d.(58)	n.d.(51)	26	35	100	95
2,3,3',4,4',5-HxCB (156)	415		501	660	1273	1065	2047	2063	3232	2966
2,3,3',4,4',5'-HxCB (157)	106		130	163	306	253	497	496	798	756
2,3',4,4',5,5'-HxCB (167)	194		226	251	480	460	785	730	1418	1267
2,3,3',4,4',5,5'-HpCB (189)	53		63	83	173	157	343	286	396	336
Total non-ortho-PCB	4.1		12	5.4	20	19	26	26	55	55
Total mono-ortho-PCB	1810		2765	2855	5373	4920	7662	7969	16755	16271
Lower bound										
TEQ based on PCDD/PCDF	0.93		2.4	2.6	3.5	3.8	5.1	6.0	12	13
TEQ based on PCB	0.43		1.4	0.68	2.3	2.1	3.0	3.2	6.4	6.5
TEQ	1.4		3.7	3.3	5.8	5.9	8.1	9.2	18	19
Upper bound										
TEQ based on PCDD/PCDF	4.3		3.4	3.9	4.9	5.1	6.7	6.7	13	14
TEQ based on PCB	1.6		1.4	1.4	2.3	2.1	3.0	3.2	6.4	6.5
TEQ	5.9		4.8	5.3	7.2	7.2	9.7	9.9	19	21
Ratio upper / lower bound	433		129	159	124	122	120	108	105	105

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

West, male PCDD/PCDFs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1 and 2	Pool 2 - not applicable	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.444		0.520	0.472	0.651	0.559	0.677	0.647	0.638	0.551
2,3,7,8-Tetra-CDD	n.d.(1.0)		n.d.(0.5)	n.d.(0.6)	n.d.(0.6)	1.5	n.d.(0.4)	n.d.(0.4)	0.86	n.d.(0.8)
1,2,3,7,8-Penta-CDD	n.d.(1)		0.60	0.89	1.8	1.9	2.4	2.1	2.9	3.7
1,2,3,4,7,8-Hexa-CDD	n.d.(2)		0.60	n.d.(1)	0.66	1.2	2.2	1.7	2.8	4.0
1,2,3,6,7,8-Hexa-CDD	4.8		4.9	3.6	8.4	10	13	13	31	26
1,2,3,7,8,9-Hexa-CDD	n.d.(2)		0.89	0.90	0.92	1.8	0.67	1.9	2.3	1.9
1,2,3,4,6,7,8-Hepta-CDD	14		13	12	20	23	24	19	34	33
OCDD	151		142	144	188	266	238	194	318	280
2,3,7,8-Tetra-CDF	n.d.(0.7)		n.d.(0.3)	n.d.(0.4)	n.d.(0.4)	n.d.(0.4)	0.38	n.d.(0.3)	n.d.(0.4)	n.d.(0.5)
1,2,3,7,8-Penta-CDF	n.d.(0.8)		n.d.(0.4)	n.d.(0.4)	n.d.(0.3)	n.d.(0.3)	n.d.(0.3)	n.d.(0.3)	n.d.(0.4)	n.d.(0.6)
2,3,4,7,8-Penta-CDF	1.1		1.0	0.86	1.5	1.9	2.2	2.0	3.1	3.0
1,2,3,4,7,8-Hexa-CDF	n.d.(1)		0.71	0.81	1.5	1.8	1.8	1.6	2.2	2.1
1,2,3,6,7,8-Hexa-CDF	n.d.(1)		0.70	0.91	1.3	1.4	1.3	1.4	2.2	2.5
1,2,3,7,8,9-Hexa-CDF	n.d.(3)		n.d.(2)	n.d.(1)	n.d.(5)	n.d.(5)	n.d.(9)	n.d.(5)	n.d.(11)	n.d.(15)
2,3,4,6,7,8-Hexa-CDF	n.d.(2)		n.d.(2)	n.d.(2)	n.d.(1)	n.d.(2)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(2)
1,2,3,4,6,7,8-Hepta-CDF	5.0		3.9	4.3	5.2	3.1	4.6	1.8	2.3	2.3
1,2,3,4,7,8,9-Hepta-CDF	n.d.(4)		n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(2)
OCDF	n.d.(7)		n.d.(2)	n.d.(2)	3.0	n.d.(2)	4.1	n.d.(1)	n.d.(2)	n.d.(3)
Total PCDD/PCDF	175		168	168	232	314	294	238	401	359
Lower bound										
TEQ based on PCDD/PCDF	1.2		2.1	2.1	4.1	6.4	5.7	5.3	9.8	9.2
TEQ based on PCB	0.86		0.74	0.64	2.4	2.7	4.1	4.0	6.8	6.9
TEQ	2.1		2.8	2.8	6.4	9.1	9.8	9.4	17	16
Upper bound										
TEQ based on PCDD/PCDF	4.5		3	3.2	5.3	7.1	7.2	6.4	11	12
TEQ based on PCB	1.9		1.7	1.6	2.4	2.7	4.1	4.0	6.8	6.9
TEQ	6.5		4.7	4.9	7.7	9.8	11	10	18	19
Ratio upper / lower bound	311		167	176	120	108	115	112	108	116

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()

n.a. = not analysed

M = Maximum data

West, male PCBs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1 and 2	Pool 2 - not applicable	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.444		0.520	0.472	0.651	0.559	0.677	0.647	0.638	0.551
3,3',4,4'-TCB (77)	n.d.(30)		n.d.(32)	n.d.(34)	n.d.(25)	n.d.(29)	n.d.(24)	n.d.(25)	n.d.(26)	n.d.(30)
3,4,4',5-TCB (81)	n.d.(1)		n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	1.0	1.1	1.2
3,3',4,4',5-PeCB (126)	n.d.(10)		n.d.(9)	n.d.(10)	9.3	11	14	15	29	29
3,3',4,4',5,5'-HxCB (169)	7.3		7.0	7.2	13	16	23	20	30	32
2,3,3',4,4'-PeCB (105)	139		285	186	371	415	582	528	1067	1153
2,3,4,4',5-PeCB (114)	77		60	58	123	119	234	206	411	401
2,3',4,4',5-PeCB (118)	1048		1120	795	1946	2254	3691	3325	6600	6865
2',3,4,4',5-PeCB (123)	n.d.(36)		n.d.(106)	n.d.(115)	n.d.(82)	n.d.(98)	n.d.(80)	n.d.(84)	n.d.(159)	n.d.(135)
2,3,3',4,4',5-HxCB (156)	983		801	697	1598	1731	3097	2902	4310	4163
2,3,3',4,4',5'-HxCB (157)	245		182	162	340	362	656	598	936	963
2,3',4,4',5,5'-HxCB (167)	285		152	n.d.(139)	367	430	832	724	1409	1260
2,3,3',4,4',5,5'-HpCB (189)	133		105	84	233	276	452	403	525	541
Total non-ortho-PCB	7.3		7.0	7.2	23	27	37	37	59	62
Total mono-ortho-PCB	2909		2705	1982	4979	5587	9544	8686	15258	15346
Lower bound										
TEQ based on PCDD/PCDF	1.2		2.1	2.1	4.1	6.4	5.7	5.3	9.8	9.2
TEQ based on PCB	0.86		0.74	0.64	2.4	2.7	4.1	4.0	6.8	6.9
TEQ	2.1		2.8	2.8	6.4	9.1	9.8	9.4	17	16
Upper bound										
TEQ based on PCDD/PCDF	4.5		3	3.2	5.3	7.1	7.2	6.4	11	12
TEQ based on PCB	1.9		1.7	1.6	2.4	2.7	4.1	4.0	6.8	6.9
TEQ	6.5		4.7	4.9	7.7	9.8	11	10	18	19
Ratio upper / lower bound	311		167	176	120	108	115	112	108	116

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

South East, females PCDD/PCDFs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.526	0.525	0.679	0.600	0.698	0.666	0.712	0.712	0.685	0.673
2,3,7,8-Tetra-CDD	n.d.(0.6)	n.d.(0.5)	n.d.(0.4)	0.54	0.90	0.85	1.4	1.1	2.2	2.2
1,2,3,7,8-Penta-CDD	0.90	0.84	1.00	0.88	1.6	1.6	2.4	1.8	4.0	4.6
1,2,3,4,7,8-Hexa-CDD	n.d.(1)	n.d.(0.9)	0.80	1.1	1.2	1.8	2.1	1.6	4.1	4.5
1,2,3,6,7,8-Hexa-CDD	3.8	3.1	5.4	5.8	11	11	18	15	29	32
1,2,3,7,8,9-Hexa-CDD	1.0	0.96	1.6	1.1	1.7	1.8	2.6	2.2	5.6	5.7
1,2,3,4,6,7,8-Hepta-CDD	9.2	12	15	15	22	27	25	28	46	45
OCDD	106	110	148	145	224	220	248	259	397	435
2,3,7,8-Tetra-CDF	n.d.(0.7)	n.d.(0.7)	n.d.(0.6)	n.d.(0.7)	0.43	n.d.(0.5)	n.d.(0.6)	0.49	0.58	n.d.(0.5)
1,2,3,7,8-Penta-CDF	0.92	0.73	1.1	1.2	0.46	1.6	n.d.(0.4)	0.87	0.73	2.0
2,3,4,7,8-Penta-CDF	0.99	0.92	1.1	0.85	1.6	1.6	2.4	2.0	4.4	4.3
1,2,3,4,7,8-Hexa-CDF	0.90	0.64	1.5	0.72	1.3	1.6	1.8	2.8	4.1	4.0
1,2,3,6,7,8-Hexa-CDF	n.d.(0.5)	0.70	0.94	1.2	1.4	0.94	1.9	1.3	2.9	2.9
1,2,3,7,8,9-Hexa-CDF	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(4)	n.d.(2)	n.d.(6)	n.d.(7)	n.d.(6)
2,3,4,6,7,8-Hexa-CDF	n.d.(1)	n.d.(1)	1.5	1.4	2.1	1.5	2.3	3.1	3.3	3.6
1,2,3,4,6,7,8-Hepta-CDF	2.2	3.9	3.3	4.3	2.8	2.9	2.2	2.3	2.3	2.3
1,2,3,4,7,8,9-Hepta-CDF	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)
OCDF	1.9	n.d.(2)	n.d.(1)	1.7	1.2	1.7	n.d.(1)	1.4	1.7	n.d.(2)
Total PCDD/PCDF	128	133	180	181	274	275	309	324	505	548
Lower bound										
TEQ based on PCDD/PCDF	2.1	2.0	3.0	3.3	5.6	5.5	8.1	6.9	14	15
TEQ based on PCB	3.1	4.4	3.6	4.7	7.8	5.0	6.1	8.1	11	5
TEQ	5.3	6.4	6.5	7.9	13	10	14	15	25	20
Upper bound										
TEQ based on PCDD/PCDF	3.2	2.9	3.5	3.4	5.7	5.9	8.4	7.5	15	17
TEQ based on PCB	3.1	4.4	3.6	4.7	7.8	5.0	6.1	8.1	11	11
TEQ	6.3	7.3	7.1	8.1	14	11	15	16	25	28
Ratio upper / lower bound	120	114	109	102	101	104	102	104	100	140

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

South East, females PCBs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.526	0.525	0.679	0.600	0.698	0.666	0.712	0.712	0.685	0.673
3,3',4,4'-TCB (77)	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.d.
3,4,4',5-TCB (81)	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.d.
3,3',4,4',5-PeCB (126)	24 (M)	34 (M)	25 (M)	37 (M)	41 (M)	32 (M)	31 (M)	48 (M)	56 (M)	n.d.
3,3',4,4',5,5'-HxCB (169)	4.8	5.3	6.8	7.2	12	11	17	15	23	20
2,3,3',4,4'-PeCB (105)	443	470	609	538	1588	870	973	1370	2201	2167
2,3,4,4',5-PeCB (114)	91	121	112	82	391	182	321	386	664	757
2,3',4,4',5-PeCB (118)	2134	2674	3195	2512	9578	4779	6510	7668	13892	13176
2',3,4,4',5-PeCB (123)	147	177	88	98	257	98	150	203	318	299
2,3,3',4,4',5-HxCB (156)	616	935	925	826	3659	1688	3107	3173	4796	5259
2,3,3',4,4',5'-HxCB (157)	142	205	205	169	791	382	665	717	1086	1244
2,3',4,4',5,5'-HxCB (167)	199	301	365	322	1447	647	1147	1088	1833	1826
2,3,3',4,4',5,5'-HpCB (189)	37	65	96	82	372	172	360	270	402	416
Total non-ortho-PCB	28	39	32	45	53	43	48	63	51	20
Total mono-ortho-PCB	3809	4948	5596	4628	18083	8818	13234	14875	25192	25144
Lower bound										
TEQ based on PCDD/PCDF	2.1	2.0	3.0	3.3	5.6	5.5	8.1	6.9	14	15
TEQ based on PCB	3.1	4.4	3.6	4.7	7.8	5.0	6.1	8.1	11	5
TEQ	5.3	6.4	6.5	7.9	13	10	14	15	25	20
Upper bound										
TEQ based on PCDD/PCDF	3.2	2.9	3.5	3.4	5.7	5.9	8.4	7.5	15	17
TEQ based on PCB	3.1	4.4	3.6	4.7	7.8	5.0	6.1	8.1	11	11
TEQ	6.3	7.3	7.1	8.1	14	11	15	16	25	28
Ratio upper / lower bound	120	114	109	102	101	104	102	104	100	140

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

South East, males PCDD/PCDFs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.540	0.495	0.520	0.537	0.658	0.677	0.678	0.651	0.574	0.563
2,3,7,8-Tetra-CDD	n.d.(0.8)	n.d.(0.8)	n.d.(0.5)	n.d.(0.7)	0.59	n.d.(0.7)	0.71	1.2	2.1	1.1
1,2,3,7,8-Penta-CDD	1.3	n.d.(1)	1.1	1.7	1.6	1.5	2.2	2.3	4.0	3.1
1,2,3,4,7,8-Hexa-CDD	n.d.(1.0)	n.d.(1)	n.d.(0.8)	1.4	1.0	1.4	2.1	2.1	2.3	2.8
1,2,3,6,7,8-Hexa-CDD	5.4	4.1	5.3	5.8	9.3	11	16	16	22	22
1,2,3,7,8,9-Hexa-CDD	n.d.(0.8)	n.d.(1)	1.1	1.2	1.0	2.0	1.6	1.8	3.3	3.0
1,2,3,4,6,7,8-Hepta-CDD	12	11	11	13	15	19	25	22	28	28
OCDD	136	121	117	141	155	169	232	241	284	250
2,3,7,8-Tetra-CDF	0.69	n.d.(0.6)	n.d.(0.5)	n.d.(0.6)	n.d.(0.4)	0.73	0.59	n.d.(0.5)	n.d.(0.6)	n.d.(0.4)
1,2,3,7,8-Penta-CDF	1.6	n.d.(0.7)	0.79	n.d.(1)	1.5	0.95	n.d.(0.4)	n.d.(0.7)	0.76	n.d.(0.6)
2,3,4,7,8-Penta-CDF	1.0	0.74	1.2	1.3	1.8	2.1	2.5	2.7	3.3	3.3
1,2,3,4,7,8-Hexa-CDF	1.5	n.d.(1)	1.2	0.92	1.9	1.8	2.3	2.3	3.3	2.4
1,2,3,6,7,8-Hexa-CDF	0.99	n.d.(0.7)	1.3	0.91	1.2	2.2	1.9	1.7	2.2	1.7
1,2,3,7,8,9-Hexa-CDF	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(2)	n.d.(2)	n.d.(2)	n.d.(4)	n.d.(3)
2,3,4,6,7,8-Hexa-CDF	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1.0)	n.d.(0.9)	1.7	n.d.(1.0)	n.d.(1)	n.d.(1)
1,2,3,4,6,7,8-Hepta-CDF	8.2	6.4	5.1	5.0	3.6	5.9	2.5	2.8	2.7	2.6
1,2,3,4,7,8,9-Hepta-CDF	n.d.(1)	n.d.(2)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)	n.d.(1)
OCDF	n.d.(2)	n.d.(3)	n.d.(1)	n.d.(2)	1.5	4.3	n.d.(2)	n.d.(2)	n.d.(2)	n.d.(2)
Total PCDD/PCDF	169	144	146	172	195	221	291	296	358	319
Lower bound										
TEQ based on PCDD/PCDF	3.0	0.96	2.8	3.6	4.8	4.7	7.1	7.5	11	9.4
TEQ based on PCB	1.3	0.79	0.89	2.9	3.1	4.8	5.7	6.2	9.6	8.0
TEQ	4.3	1.8	3.7	6.5	8.0	9.5	13	14	21	17
Upper bound										
TEQ based on PCDD/PCDF	5.3	3.5	3.6	4.6	5.1	5.7	7.3	7.9	12	9.9
TEQ based on PCB	2.7	2.3	2.3	2.9	3.1	4.8	5.7	6.2	9.6	8
TEQ	8.0	5.8	5.9	7.6	8.2	11	13	14	22	18
Ratio upper / lower bound	186	331	162	116	104	110	102	103	103	103

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

South East, males PCBs	<16 years		16-30 years		31-45 years		46-60 years		> 60 years	
	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2	Pool 1	Pool 2
lipid content [%]	0.540	0.495	0.520	0.537	0.658	0.677	0.678	0.651	0.574	0.563
3,3',4,4'-TCB (77)	n.a.	n.d.(41)	n.d.(39)	n.d.(37)	n.d.(31)	n.a.	n.d.(30)	n.d.(31)	n.d.(34)	n.d.(36)
3,4,4',5-TCB (81)	n.a.	1.1	n.d.(1)	1.1	1.0	n.a.	1.5	1.4	2.7	2.5
3,3',4,4',5-PeCB (126)	n.d.	n.d.(15)	n.d.(14)	15	13	28	23	27	39	33
3,3',4,4',5,5'-HxCB (169)	7.0	5.2	8.6	9.8	14	15	20	24	28	28
2,3,3',4,4'-PeCB (105)	806	326	348	644	618	843	990	1219	2216	1607
2,3,4,4',5-PeCB (114)	128	72	74	114	146	186	284	324	618	495
2,3',4,4',5-PeCB (118)	3874	1641	1833	3165	3407	3877	5759	7415	13293	9958
2',3,4,4',5-PeCB (123)	n.d.(213)	63	65	111	92	126	133	121	203	168
2,3,3',4,4',5-HxCB (156)	1161	792	848	1381	1843	2073	3863	3598	5614	4701
2,3,3',4,4',5'-HxCB (157)	254	192	209	304	412	468	853	840	1328	1151
2,3',4,4',5,5'-HxCB (167)	414	230	308	396	518	735	1084	1260	1969	1619
2,3,3',4,4',5,5'-HpCB (189)	79	53	107	134	225	239	404	417	528	472
Total non-ortho-PCB	7	6.3	8.6	26	28	43	44	52	70	63
Total mono-ortho-PCB	6715	3368	3790	6248	7261	8548	13371	15194	25770	20171
Lower bound										
TEQ based on PCDD/PCDF	3.0	0.96	2.8	3.6	4.8	4.7	7.1	7.5	11	9.4
TEQ based on PCB	1.3	0.79	0.89	2.9	3.1	4.8	5.7	6.2	9.6	8.0
TEQ	4.3	1.8	3.7	6.5	8.0	9.5	13	14	21	17
Upper bound										
TEQ based on PCDD/PCDF	5.3	3.5	3.6	4.6	5.1	5.7	7.3	7.9	12	9.9
TEQ based on PCB	2.7	2.3	2.3	2.9	3.1	4.8	5.7	6.2	9.6	8
TEQ	8.0	5.8	5.9	7.6	8.2	11	13	14	22	18
Ratio upper / lower bound	186	331	162	116	104	110	102	103	103	103

Data in pg g^{-1} lipid based

n.d. = not detected, detection limit in ()
n.a. = not analysed
M = Maximum data

Appendix G International data

A comparison of Australian levels of PCDD/PCDFs with other countries

Country	Sampling year	Comments	No. of samples	Total PCDD	Total PCDF	Total PCDD/PCDF	Total TEQ PCDD/PCDF	Reference
U.S.	unknown		16	734.9^	43.9^	778.8^	19.1	Tepper A (1997)
U.S.	1995/1996	pre-delivery	5	465.0	26.0	491.0	12.1**	Schecter et al (1998)
	1995/1996	post-delivery	5	301.0	23.5	324.5	10**	Schecter et al (1998)
Finland	1993		18	790.1	102.7	893.7	50.4	Kontsas H et al (1998)
Spain	1995		97	610.3	27.9	640.3	13.4	Gonzalez CA et al (2000)
	1997		91	na	na	na	16.7	Gonzalez CA et al (2000)
Spain	1993		11	515.3	66.7	582.0	15.7	Jimenez B et al (1996)
Spain	unknown		20	na	na	na	27.0	Schuhmacher M et al (1999)
Belgium	1999		47	na	na	na	48.6 *	Covaci A et al (2001)
Norway	1992		10	562.8	72.8	631.1	21.4 #	Johansen R et al (1996)
Germany	1989		228	843.7	98.2	942.2	43.7	Wittsiepe J et al (2000)
	1992		157	627.1	76.0	703.2	38.1	Wittsiepe J et al (2000)
	1993		17	454.5	79.7	534.5	29.1	Wittsiepe J et al (2000)
	1994		74	317.7	59.0	376.7	29.1	Wittsiepe J et al (2000)
	1995		69	382.1	49.4	431.6	24.1	Wittsiepe J et al (2000)
	1997/1998		9	414.8	37.3	452.0	20.7	Wittsiepe J et al (2000)
	1989-1998		744	596.9	74.6	671.7	35.6	Wittsiepe J et al (2000)
Germany	unknown		16	na	na	na	18.5	Menzel HM et al (1998)
Germany	1994		134	462.8	46.0	508.8	19.1	Paepke O et al (1996)
Germany	unknown	adults	15	511.2	46.2	573.1	18.4	Wuthe J et al (1996)
	unknown	< 12 yrs urban/industrial 1	45	302.7	24.0	326.7	7.3	Wuthe J et al (1996)
	unknown	< 12 yrs urban/industrial 2	79	290.1	28.0	318.1	8.2	Wuthe J et al (1996)
	unknown	<12 yrs industrial/rural 1	39	273.2	30.2	303.4	10.0	Wuthe J et al (1996)
	unknown	<12 yrs industrial/rural 2	44	230.6	30.0	260.6	9.0	Wuthe J et al (1996)
	unknown	< 12 yrs rural 1	46	246.3	42.7	289.0	9.3	Wuthe J et al (1996)
	unknown	< 12 yrs rural 2	33	303.1	30.7	333.8	10.1	Wuthe J et al (1996)
Japan	unknown		20	361.3	39.3	400.6	20.3**	Kumagai S et al (2002)

Country	Sampling year	Comments	No. of samples	Total PCDD	Total PCDF	Total PCDD/PCDF	Total TEQ PCDD/PCDF	Reference
Japan	1998		na	na	na	na	11*	Ueda et al (1999)
China	unknown	15-19 years	50	126.0	21.7	148.0	4.8	Schecter AJ et al (1996)
	unknown	35-70 years	51	149.5	26.2	178.0	6.4	Schecter AJ et al (1996)
New Zealand	1996/1997		1834	na	na	459.0	12.7*	Buckland et al (2001)
New Zealand	1992/1993		28	841.7	24.9	866.6	11.6 **	Hannah et al (1994)

Concentration expressed as mean in pg g^{-1} lipid adjusted unless otherwise specified
TEQ expressed using I-TEF unless otherwise specified
*WHO
TEQ
** TEF unknown
^ pg g^{-1} median
na = not assessed
# Nordic TEQ

A comparison of Australian levels of PCBs with other countries

Country	Sampling year	Comments	No. of samples	PCB 126	PCB 169	PCB 118	Sum PCBs	TEQ coplanar PCBs	TEQ total PCBs	Reference
U.S.	unknown		16	18	27	na	na	na	na	Kang D et al (1997)
U.S.	1991/1993		7	na	na	28.5*	na	na	na	Greizerstein HB et al (1999)
U.S.	1995/1996	pre-delivery	5	21.7	9	na	na	2.26	na	Schecter et al (1998)
		post-delivery	5	16.3	6.7	na	na	1.7	na	Schecter et al (1998)
U.S.	1995		150	10.8	15.7	na	na	Na	na	Shadel et al (2001)
Finland	1993		18	69.4	82.8	na	na	Na	11.1*	Kontsas H et al (1998)
Spain	1993		11	55.21	30.26	na	na	7.03*	na	Jimenez B et al (1996)
Spain	1995		97	na	na	na	1.76#	Na	na	Gonzalez CA et al (2000)
	1997		91	na	na	na	1.99#	Na	na	Gonzalez CA et al (2000)
Belgium	1996/1998		96	na	na	27.3*	na	Na	na	Pauwels A et al (2000)
Belgium	1999		47	na	na	na	550.6*	Na	25.8	Covaci A et al (2001)
Norway	1992		10	93.4	70.1	29.3*	1344.2*	Na	45	Johansen R et al (1996)
Germany	1994		104	80.3	101.8	na	na	Na	na	Paepke O et al (1996)
Germany	unknown	adults	15	67.3*	116.2*	34.2*	na	Na	na	Wuthe J et al (1996)
	unknown	< 12 yrs urban/industrial 1	45	37.6	24.6	13.2*	na	Na	na	Wuthe J et al (1996)
	unknown	< 12 yrs urban/industrial 2	79	41.9	29.4	14.6*	na	Na	na	Wuthe J et al (1996)
	unknown	< 12 yrs industrial/rural 1	39	52.6	37.4	11.8*	na	Na	na	Wuthe J et al (1996)
	unknown	< 12 yrs industrial/rural 2	44	44.8	30.3	7.9*	na	Na	na	Wuthe J et al (1996)
	unknown	< 12 yrs rural 1	46	49.4	36.7	18.4*	na	Na	na	Wuthe J et al (1996)
	unknown	< 12 yrs rural 2	33	45.2	34	20.6*	na	Na	na	Wuthe J et al (1996)
Japan	1993/1994		50	46	23	na	na	Na	21	Iida et al (1999)
Japan	1998		253	na	na	na	na	7.3	na	Ueda et al (1999)
New Zealand	1996/1997		1834	30	20	na	na	Na	6.86	Buckland et al (2001)

Concentration expressed in pg g^{-1} lipid unless otherwise specified

TEQ expressed using WHO- TEF unless otherwise specified

na = not assessed # = ug l^{-1} *ng g^{-1} lipid * I-TEQ