Halogenated Organic Contaminants and Metabolites in Blood and Adipose Tissues of Polar Bears (Ursus maritimus) From Svalbard

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Photo: Magnus Andersen

Geir W. Gabrielsen, Lisa B. Knudsen, Jonathan Verreault, Kerstin Push, Derek D.C. Muir, Robert J. Letcher

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1. Preface

The aim of the present project was to determine a wide range of new and established persistent organic contaminants and their metabolites in polar bears (*Ursus maritimus*) from Svalbard. Current levels and congener patterns are examined in polar bear adipose tissue and blood samples, and are compared to levels measured in previous studies of polar bears and other Arctic animals.

The present study involves the collaboration between the Norwegian Polar Institute and leading international laboratories: the National Laboratory for Environmental Testing (NLET) at the National Water Research Institute (NWRI), (Canada), Great Lakes Institute for Environmental Research (GLIER), (Canada), Centre d'Expertise en Analyse Environnementale du Québec (CEAEQ), (Canada) and AXYS Analytical, (Canada). The Norwegian Pollution Control Authority (SFT) and the Norwegian Polar Institute's Ecotoxicology Programme provided funding.

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Geir W. Gabrielsen
Research Leader Ecotoxicology Programme, Norwegian Polar Institute (Tromsø, Norway)

Lisa Bjørnsdatter Knudsen
Master student, Norwegian Polar Institute (Tromsø, Norway)

Jonathan Verreault
PhD Candidate, Norwegian Polar Institute (Tromsø, Norway)

Kerstin Pusch
Norwegian Polar Institute (Tromsø, Norway)

Derek D.C. Muir
Research Scientist, National Water Research Institute, Environment Canada (Burlington, ON, Canada)

Robert J. Letcher
Assistant Professor, Great Lakes Institute for Environmental Research (GLIER) and the Department of Chemistry and Biochemistry, University of Windsor (Windsor, ON, Canada)

2. Content

3. Summary

The objective of the present study was to determine a wide range of new and established persistent organic contaminants and their metabolites in polar bear (*Ursus maritimus*) adipose tissue and blood samples from Svalbard.

The present analyses of fat and plasma samples of polar bears showed the presence of compounds that have been routinely analysed and other less studied contaminants such as polychlorinated naphthalene (PCNs), toxaphenes, metabolites, phenolics, brominated flame retardants (BFR), and perfluorinated alkyl substances. In the present study polychlorinated biphenyl (PCB) was the major class of contaminants. Svalbard polar bears of the present study carried higher loads of higher chlorinated PCBs, nonachlor and oxychlordane compared to polar bears from Arctic regions of Alaska and Canada. Furthermore, the Svalbard population had higher mean concentrations of ∑ PCB and ∑ methylsulfone ($MeSO_2$)-PCB compared to polar bear populations from Alaska, East Greenland and Canada. The polar bears examined in the present study exceed the lowest-adverse-observed-effect levels (LOAEL), no-observed-adverse-effect level (NOAEL) and no-observed-effect level for short-time memory (measured in monkeys), visual memory (measured in monkey) and vitamin A reduction (measured in European otter), respectively. Coplanar PCB concentrations in the present study are comparable with Canadian and eastern Greenland polar bears. However, the levels were considerably lower than reported concentrations in glaucous gull from Bear Island and harbour porpoise from the Norwegian Sea. Calculated toxic equivalent concentrations (including coplanar PCBs, polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans) of the present study did not exceed the threshold level for immune suppression in captive harbour seals.

Hexachlorobutadiene, pentachloroanisole and endosulfan were detected in the polar bears of the present study, however, at low concentrations. Toxaphene concentrations in polar bears of the present study were comparable to previous reported levels in Arctic wildlife. Furthermore, PCN levels reported in the present study are higher than concentrations reported in beluga whale.

Relatively high concentrations of hydroxy (HO)-PCBs were detected in the present study compared to other animals, whereas relatively low concentrations of polybrominated diphenyl ethers (PBDEs) were found in the present study. Furthermore, only two (out of 14) analysed BFR metabolites were detected. These two metabolites were reported in two of the 15 polar bears analysed in the present study.

Plasma levels of perfluorooctane sulfonate (PFOS) were higher than those reported in ringed seals. PFOS plasma concentrations are reported to be one of the most prominent contaminants in polar bears, but the levels were well below estimated NOAEL and LOAEL for second-generation effects in rat.

4. Sammendrag

Formålet med dette studiet var å kartlegge en rekke nye og etablerte persistente organiske miljøgifter og deres metabolitter i fettvev og blodprøver fra isbjørn (*Ursus maritimus)* på Svalbard.

Analyser av fettvev og plasmaprøver fra isbjørn viste tilstedeværelse av forbindelser som rutinemessig blir analysert og andre mindre studerte forbindelser som polyklorerte naftalener (PCNs), toksafen, metabolitter, fenoler, bromerte flammehemmere (BFR) og perfluorerte alkylerte stoffer. Polyklorerte bifenyler (PCB) var den dominerende gruppen av miljøgifter. Isbjørn fra Svalbard hadde en høyere andel av høy klorerte PCBer, nonaklor og oksyklordan sammenliknet med isbjørner fra Alaska og Canada. Videre, hadde isbjørn fra Svalbard høyere konsentrasjoner av $\sum$ PCB og $\sum$ metylsulfon ($MeSO_2$)-PCB sammenliknet med isbjørn fra Alaska, Øst-Grønland og Canada. Isbjørnene i dette studiet overstiger lavest-skadelig-observerte-effekt nivå (LOAEL), ingen-observert-skadelig-effekt-nivå (NOAEL) og ingen-observert-effekt-nivå for henholdsvis korttids hukommelse (studier av aper), visuell hukommelse (studier av aper) og vitamin A reduksjon (studier av europeisk oter). Koplanare PCB konsentrasjoner i dette studiet er sammenlignbart med isbjørn fra Canada og Øst-Grønland. Nivåene er imidlertid betydelig lavere enn rapporterte nivåer i polarmåke fra Bjørnøya og nise fra Norskehavet. Kalkulerte toksiske ekvivalenter (TEQ) (inkludert koplanare PCBer, polyklorerte dibenzo-p-dioksin og polyklorerte dibenzofuraner) i dette studiet oversteg ikke terskelnivåer for immunforstyrrelser fra niser i fangenskap.

Heksaklorbutadien, pentakloranisole og endosulfan ble funnet hos isbjørn i dette studiet. Konsentrasjonene var imidlertid lave. Toksafen konsentrasjoner i isbjørn i dette studiet er sammenlignbart med tidligere rapporterte verdier i arktiske dyr. Videre er rapporterte PCN nivåer i dette studiet høyere enn rapporterte nivåer i hvithval.
I dette studiet ble det funnet relativt høye nivåer av hydroksy (HO)-PCBer sammenliknet med andre dyr. Relativt lave verdier av polybromerte difenyl etere ble rapportert. Videre ble bare 2 (ut av 14) analyserte BFR metabolitter funnet i prøvematerialet, og disse to ble påvist i 2 av 15 analyserte isbjørn.

Plasma nivåer av perfluoroktan sulfonat (PFOS) var høyere enn rapporterte nivåer i ringsel. Plasma konsentrasjoner av PFOS er en av de mest fremtredende miljøgifter i isbjørn. Likevel er PFOS nivåene atskillig lavere enn estimerte NOAEL og LOAEL for andregenerasjons effekter i rotte.

5. Background

The polar bear (*Ursus maritimus*) in the Arctic marine food chain was found to accumulate high levels of halogenated organic contaminants such as polychlorinated biphenyls (PCBs) (Bernhoft et al. 1997; Andersen et al. 2001). Due to their lipophilicity and persistence, halogenated organic contaminants have high bioaccumulative potential and biomagnify at higher trophic levels in arctic food webs (Muir et al. 1988; Borgå et al. 2001; Fisk et al. 2001). Therefore, the polar bear which is feeding at the top of the Arctic marine food chain achieves some of the highest concentrations of contaminants of any Arctic mammal species (de March et al. 1998; AMAP 2004). Adverse biological effects observed in polar bears have been linked to the exposure to these contaminants (de March et al. 1998; AMAP 2004). Due to its wide distribution throughout the Arctic and sub-Arctic and to its key role as apex predator of the marine environment, the polar bear serves as an ideal candidate for assessing spatial and temporal trends of contaminants for certain compound classes such as PCBs and chlordane-related compounds (de March et al. 1998; AMAP 2004). Routinely analysed contaminants like PCBs, dichlorodiphenyltrichloroethane (DDTs) and toxaphenes have been linked to reproductive, developmental, thyroid and retinol effects, cancer, immunosuppression and endocrine modulation (AMAP 2004). In recent years, metabolites of PCBs have been found to be toxic and to accumulate in the environment. The transformation of PCBs is part of a detoxification process and results in the formation of more polar metabolites. The increased polarity increases the likelihood for elimination from the organism. These metabolites, i.e. hydroxy (HO-) PCBs, methyl sulfone ($MeSO_2$-) PCBs and $MeSO_2$-p,p'-DDE are detected in a growing number of species (Letcher et al. 2000). Recently, several studies have demonstrated the toxicological potential of $MeSO_2$-PCBs, like tissue selective retention via non-covalent protein binding, induction of cytochrome (CYP) enzymes, and endocrine-related effects (Letcher et al. 2000). Furthermore, HO-PCBs are suggested to bind with the thyroid hormone thyroxin (T4) transport proteins and interact with thyroid and estrogen hormone receptors (Sandau 2000).

Several studies have reported levels of routinely analysed compounds, (i.e. PCB and pesticides), in Arctic biota. In the Arctic there are uncertainty of levels in biota and spatial distribution of compounds such as organochlorine (OC) metabolites, polychlorinated naphtalenes, toxaphenes, short-chained paraffins, brominated flame retardants (BFR), metabolites of BFR and perfluorinated alkylated substances. In order to address this lack of knowledge we have determined a wide range of established and new organohalogen contaminants, and metabolites in polar bear adipose tissue and blood samples from Svalbard.

6. Materials and methods

6.1. Sampling

Blood and fat biopsy samples of 15 polar bears from Svalbard were collected in April 2002. All the individuals were females between 2 and 25 years of age. For details on capture methods and sample collection, see Verreault et al. (2004a).

6.2. Chemicals analysed

PCBs, OC pesticides and byproducts, and other OCs, OC metabolites, BFR, metabolites of polybrominated diphenyl ethers (PBDEs) and perfluorinated alkylated substances (PFAS) were analysed in the present study. For details on analysed contaminants and methods see Table 1.

6.2.1. Methods for chemical analyses

Legacy OCs (except coplanar PCBs and polychlorinated dibenzo-p-dioxin (PCDDs)/furans (PCDFs)), BFR and PFAS in polar bear fat were determined by the National Laboratory for Environmental Testing at NWRI using standard procedures. These methods are summarized here:

1. *Sample preparation and extraction*

Samples were homogenized prior to extraction in a small blender. Internal recovery surrogates of 1,3,5-tribromobenzene, 1,2,4,5-tetra-bromobenzene, delta-hexachlorocyclohexanes (HCH), endrin ketone, PCB-30 and PCB-204 were added at the extraction step. Homogenized tissue was mixed with precleaned sodium sulfate to form a dry powder and Soxhlet extracted for 6 hrs with dichloromethane (DCM).

2. *Removal of Interferences*

The DCM solution was reduced to approximately 2 ml. The extract was applied to the top of a gel permeation column (GPC) to remove lipids using hexane: DCM (1:1) as elution solvent. Extractable lipids were determined gravimetrically on the first 150 ml of GPC eluate by evaporating off the solvent. The GPC eluate was reduced to small volume, quantitatively exchanged into hexane and chromatographed on activated Silica Gel (8 g in a 1.1 cm diameter chromatographic column) and eluted with hexane (F1) followed by DCM:hexane (50:50) (F2) to separate PCBs from most other organochlorine pesticides and byproducts. This latter procedure has been used successfully to separate 100% of technical toxaphene from PCBs (NLET 1997). Final extracts were stored at 4 ^{o}C in a fridge. Prior to instrumental analysis they were reduced to an appropriate final volume under gentle nitrogen stream.

3. *Instrument analysis*

Gas chromatograph (GC) -electron capture detection: PCB congeners and organochlorine pesticides were determined by high resolution capillary GC with electron capture detection using a Hewlett Packard 6890 GC equipped with a 30 m x 0.25 mm, 0.25 μm film thickness DB-5 column programmed at 15 ^{o} C/min to 150 ^{o}C and 3 ^{o}C/min to 265 ^{o}C. Carrier gas was H_2 (about 1 ml/min) and make-up gas was N_2 (40 ml/min). PCB congeners and OC pesticides were quantified by GC-electron capture detection (ECD) using a series of authentic external standards. PBDE (including BDE209 and HBCDD) analyses were carried out using an Agilent 6890 GC-5973 MSD. The GC separation was performed on an Agilent HP5-MS capillary column (30 m x 0.25 mm x 0.25 μm). Helium was used as carrier gas and separation was performed at a constant flow of 1.2 ml/min. Injection of 2 μl was performed in pulsed splitless mode at 25 psi for 1.0 min at an injector temperature of 250 ^{o}C. The initial column temperature was 80 ^{o}C for 2 min, 10 ^{o}C/min to 120 ^{o}C and 3 ^{o}C/min to 285 ^{o}C, which was held for 15 min. The mass spectrometer was operated in the NCI (negative chemical

ionization) mode with methane as the buffer gas. The temperatures were 106, 150 and 300 ^{o}C for the quadrupole, the ion source and the interface, respectively. All PBDEs were monitored at m/z 79 and 81. PBDEs were quantified using an external standard consisting of 32 congeners. Chlorinated paraffins were analysed by GC-high resolution MS using metastable atom bombardment ionization (MAB).

OC metabolites and hydroxyl-PBDEs concentrations in polar bear plasma were analysed by the Great Lakes Institute for Environmental Research (GLIER). Surrogate standard recovery was consistently >80%. Quantification was via an internal standard approach using relative response factors to the I.S. 4-HO-CB72. Detection limit was 0.001 ng/g w.w. There is no standard for the 3'-HO-CB187 congener, so the relative response factor of 4-HO-CB187 was used for the quantification. 4-HO-CB187 was used because it has the same structure, except the HO-group is on another position. There is no standard for the 4'-HO-CB172 congener, so the relative response factor of 3'-HO-CB180 is used for the quantification. 3'-HO-CB180 is used because it has almost the same structure as 4'-HO-CB172. The only difference is that the HO-group and a Cl have swapped positions. For 4,4'-di-HO-CB202 and 4'-HO-CB208 the relative response factor of 4-HO-CB193 is used for the quantification. See Maervoet et al. (2004) for full chemical structures corresponding to the name abbreviations.

PCDD/Fs were determined by AXYS analytical (Sidney BC) using US EPA methods 9290A and 1688 for PCDD/Fs, slightly modified to include *no*-PCBs (coplanar PCBs) (US EPA 1998; 1999). Chlorinated paraffins in fat extracts were determined by CEAEQ using GC-high resolution metastable atom bombardment mass spectrometry as described by Moore et al. (2004).

Laboratory analyses were performed during January and February and June-August 2004 at NWRI, GLIER, AXYS and CEAEQ. The participating laboratories NLET, CEAEQ and AXYS are accredited under the Canadian Association for Environmental Analysis Laboratories (CAEAL). GLIER and NWRI are certified as laboratories for determination of PCBs and OC pesticides according to the requirements of the CAEAL program of the Canadian Standards Association, and are participants in the Northern Contaminant Program's (Indian and Northern Affairs of Canada, Ottawa, ON, Canada) Quality Assurance Program. CAEAL is the Canadian equivalent of QUASIMEME (Quality Assurance of Information for Marine Environmental Monitoring in Europe) and ISO9000 (International Organization for Standardization in Europe). The labs of the Canadian consortium also participated in QUASIMEME and are also participating in the inter-laboratory program for analytical quality within the Northern Contaminant Program.

Standard procedures were used to ensure quality assurance and control, and the results were within the laboratories accredited requirements for precision, linearity and sensitivity. A standard reference material (SRM) (1588a: cod liver oil) from the National Institute of Standards and Technology (Gaithersburg, MD, US) was used to confirm the accuracy and reproducibility of the analytical methods.

For legacy OC, concentrations were within acceptable range of the SRMs certified values. The mean recoveries (± 1 standard deviations) of 1,3,5-tribromobenzene and $MeSO_2$-PCB-IS internal standards were 83.8 ± 15% and 68.4 ± 13.1%, respectively. The minimum detection limit for individual analytes of PCBs, OC pesticides, and methyl sulfones was determined as ten times the noise level at the baseline and was on average 0.01 ng/g lipid weight. The repeatability (inter-day comparison) of the GC-performance was tested by repeated injection (duplicate) of standard compounds and polar bear samples at regular intervals of time. Method blank samples were also run with each block of 5 samples to test for interferences. Duplicate standard compounds and polar bear fat samples demonstrated 5% or less variation of contaminant levels. All results were routinely blank corrected by substracting

an average blank value. However blank analyses indicated very low background contributions representing < 1 % of PCBs, OC pesticides, total PBDEs and HBCDD in polar bear fat. Blank values for polychlorinated naphtalenes ∑ PCNs, ∑ PCDD/Fs and ∑ coplanar PCBs averaged 17, 11 and 6 % of measured values in polar bear fat. Blank values for PFOA averaged 30 % of measured values while PFOS and PFOSA were undetectable in blanks. Chlorinated paraffins (C_{10}-C_{13}) were not detected in blanks. Concentrations of the analytes are expressed in ng/g wet weight (w.w.).

Table 1: *List of analytes determined in blood and adipose tissue samples of polar bears from Svalbard, analytical laboratories involved and methods used. Methods: gas chromatograph (GC) with a micro electron capture detector (μECD), GC-mass spectrometer (GC-MS) in the negative chemical ionization mode using a buffer gas in the electron impact source (GC-(ECNI)MS, GC-high resolution-MS (GC-HRMS), GC-HR-metastable atom bombardment-MS (GC-HR-MAB-MS) or liquid chromatograph (LC) – tandem MS (LC-MS-MS).*

PARAMETER	LABORATORY	METHODS
ORGANOCHLORINES (OC) AND METABOLITES		
Legacy OCs (routinely analysed)		
Polychlorinated biphenyls (PCBs)	NLET/NWRI	GC-μECD
Coplanar PCBs	AXYS	GC-HRMS
Polychlorinated dibenzo-p-dioxins (PCDDs)	AXYS	GC-HRMS
Polychlorinated dibenzofurans (PCDFs)	AXYS	GC-HRMS
Chlorobenzene (CBz)	NLET/NWRI	GC-μECD
Chlordanes and metabolites (CHL)	NLET/NWRI	GC-μECD
Aldrin	NLET/NWRI	GC-μECD
Endrin	NLET/NWRI	GC-μECD
Dieldrin	NLET/NWRI	GC-μECD
Mirex	NLET/NWRI	GC-μECD
Octachlorostyrene	NLET/NWRI	GC-μECD
Dichlorodiphenyltrichloroethane (DDT) and metabolites	NLET/NWRI	GC-μECD
Hexachlorocyclohexanes (HCH)	NLET/NWRI	GC-μECD
Other OCs		
Hexachlorobutadiene (HCBD)	NLET/NWRI	GC-(ECNI)MS
Pentachloroanisole	NLET/NWRI	GC-μECD
Endosulfans	NLET/NWRI	GC-μECD
Polychlorinated naphthalenes (PCNs)	AXYS	GC-(ECNI)MS
Polychlorobornanes and camphenes (Toxaphene)	NWRI	GC-(ECNI)MS
Short-chain chlorinated paraffins (SCCPs)	CEAEQ	GC-HR-MAB-MS
Medium-chain chlorinated paraffins (MCCPs)	CEAEQ	GC-HR-MAB-MS
Metabolites and other phenolics		
Hydroxy PCBs (HO-PCBs)	GLIER	GC-(ECNI)MS/GC-μECD
Methylsulfone PCBs ($MeSO_2$-PCBs)	GLIER	GC-(ECNI)MS/GC-μECD
Methylsulfone DDE ($MeSO_2$-p,p'-DDE)	GLIER	GC-(ECNI)MS
Pentachlorophenol (PCP)	GLIER	GC-(ECNI)MS/GC-μECD
HO-heptachlorostyrene (HO-HpCS)	GLIER	GC-(ECNI)MS/GC-μECD
BROMINATED FLAME RETARDANTS (BFR) AND METABOLITES		
BFR		
Polybrominated diphenyl ethers (PBDEs)	NLET/NWRI	GC-(ECNI)MS
Hexabromocyclododecane (HBCDD)	NLET/NWRI	GC-(ECNI)MS
Metabolites		
Hydroxy PBDEs (HO-BDEs)	GLIER	GC-(ECNI)MS

PERFLUORINATED ALKYL SUBSTANCES (PFAS)		
Perfluorooctane sulfonate (PFOS)	NWRI	LC-MS-MS
Perfluorooctanoic acid (PFOA)	NWRI	LC-MS-MS
Perfluooctanesulfamide (PFOSA)	NWRI	LC-MS-MS

7. Results

The analyses of plasma and fat samples of polar bears report the presence of several new contaminants and metabolites (Table 2). The concentrations of individual congeners are listed in Appendix A - C. Concentrations were measured in plasma for the compound HO-PCB, PCP, 4-HO-HpCS, PFOS, PFOA, PFOSA and hydroxy-PBDEs. For the remaining congeners, concentrations were measured in fat.

***Table 2**: Arithmetic means (only for samples above the detection limit) with standard deviation (± SD) and ranges (min-max), for the sum (∑) concentrations of different analytes, in plasma and fat samples of polar bears from Svalbard. Concentrations in pg/g wet weight for PCDDs, PCDFs and coplanar PCBs (marked with *) and in ng/g wet weight for all the other compounds. The number of samples above the detection limit of the analysed contaminant is shown relatively to the number of samples analysed (N). See Appendix A (OCs and metabolites), B (BFR and metabolites) and C (PFAS) for congeners constituting the different groups and the specific concentrations of the congeners of each group.*

Congeners	N	Mean ± SD	Min – Max
Lipid % fat	15	56.3 ± 15.1	23.0 – 80.8
Lipid % blood	15	1.0 ± 0.7	0.5 – 3.6
Organochlorines (OCs) and metabolites			
Legacy OCs			
∑ Polychlorinated biphenyl (∑ PCB) (99 congeners)[1]	15/15	3636 ± 1316	1315 – 5658
∑ Coplanar PCBs*	15/15	181 ± 91.3	81.6 – 390
∑ Polychlorinated dibenzo-p-dioxins (PCDDs)*	15/15	24.7 ± 41.5	0.9 – 173
∑ Polychlorinated dibenzofurans (PCDFs)*	15/15	6.5 ± 4.8	2.6 – 21
∑ Chlorobenzene (CBz)	15/15	110 ± 56.5	50.2 – 264
∑ Chlordanes and metabolites (CHL)	15/15	871 ± 311	423 – 1329
Aldrin	15/15	3.3 ± 1.9	1.0 – 6.6
Endrin	7/15	2.8 ± 1.3	1.8 – 5.2
Dieldrin	15/15	92.1 ± 31.0	29.6 – 142
Mirex	15/15	6.8 ± 4.1	0.8 – 14.7
Octachlorostyrene	15/15	9.6 ± 3.9	3.3 – 19.8
∑ Dichlorodiphenyltrichloroethane (DDT) and metabolites	15/15	131 ± 59.0	22.3 – 244
∑ Hexachlorocyclohexanes (HCH)	15/15	41.5 ± 16.0	14.5 – 79.2
Other OCs			
∑ Hexachlorobutadiene (HCBD)	5/15	3.7 ± 2.4	1.2 – 8.9
Pentachloroanisole	15/15	1.9 ± 1.2	0.8 – 5.2
∑ Endosulfans	15/15	4.8 ± 2.8	1.3 – 10.5
∑ Polychlorinated naphtalenes (PCNs)	14/14	4.4 ± 7.3	0.7 – 29.3
∑ Polychlorobornanes and camphenes (Toxaphene)	15/15	26.8 ± 17.3	5.4 – 77.9
∑ Short-chain chlorinated paraffins (SCCPs) (26 congeners)[2]	7/15	0.5 ± 1.0	0.004 – 2.6
∑ Medium-chain chlorinated paraffins (MCCPs)	0/15	–	< 0.0005

Metabolites and other phenolics			
∑ Hydroxy PCBs (HO-PCBs)	15/15	93.5 ± 55.4	2.6 – 235
∑ Methylsulfone PCBs ($MeSO_2$-PCBs)	15/15	133 ± 77.3	27.1 – 273
Methylsulfone DDE ($MeSO_2$-p,p'-DDE)	3/15	1.3 ± 0.1	1.2 – 1.3
Pentachlorophenol (PCP)	14/15	0.3 ± 0.2	0.1 – 0.8
HO-heptachlorostyrene (HO-HpCS)	15/15	8.8 ± 3.7	0.1 – 14.2
Brominated flame retardants (BFR) and metabolites			
BFR			
∑ Polybrominated diphenyl ethers (PBDEs)[3]	15/15	29.0 ± 11.5	14.6 – 48.0
Hexabromocyclododecane (HBCDD)	15/15	25.6 ± 9.0	9.7 – 44.8
Metabolites			
∑ Hydroxy PBDEs (HO-BDEs)	2/15	0.2 ± 0.04	0.17 – 0.22
Perfluorinated alkyl substances (PFAS)			
∑ Perfluorooctane sulfonate (PFOS)	14/14	97.2 ± 22.9	56.7 – 150
∑ Perfluorooctanoic acid (PFOA)	14/14	1.6 ± 0.7	0.7 – 2.8
∑ Perfluooctanesulfamide (PFOSA)	9/14	0.6 + 0.1	0.5 – 0.8

[1] ∑ PCB: sum of PCB no. IUPAC (international union of pure and applied chemistry): 1, 3, 7-9, 6, 8-5, 19, 12-13, 18, 15-17, 24-27, 16-32, 24-29, 26, 25, 31-28, 33-20, 53, 51, 22, 45, 46, 52, 43, 49, 47-48, 44, 59, 42, 71-41-64, 40, 100, 63, 74, 70-76-98, 66, 95, 91, 55, 66-60, 92, 84, 101, 99, 119, 83, 97, 81-87, 85, 136, 110, 82, 151, 135-144, 147, 107, 149, 118, 133, 114, 134-131, 146, 153, 105, 141, 179, 137, 130, 163-138, 158, 129, 178, 175, 182-187, 183, 128, 167, 185, 174, 177, 202, 156, 173, 157-201, 172, 180, 193, 191, 201, 170-190, 198, 200, 203-196, 189, 208-195, 207, 194, 205, 206 and 209.

[2] $C_{10}H_{19}Cl_3$, $C_{10}H_{18}Cl_4$, $C_{10}H_{17}Cl_5$, $C_{10}H_{16}Cl_6$, $C_{10}H_{15}Cl_7$, $C_{10}H_{14}Cl_8$, $C_{10}H_{13}Cl_9$, $C_{11}H_{21}Cl_3$, $C_{11}H_{20}Cl_4$, $C_{11}H_{19}Cl_5$, $C_{11}H_{18}Cl_6$, $C_{11}H_{17}Cl_7$, $C_{11}H_{16}Cl_8$, $C_{11}H_{15}Cl_9$, $C_{12}H_{22}Cl_4$, $C_{12}H_{21}Cl_5$, $C_{12}H_{20}Cl_6$, $C_{12}H_{19}Cl_7$, $C_{12}H_{18}Cl_8$, $C_{12}H_{17}Cl_9$, $C_{13}H_{24}Cl_4$, $C_{13}H_{23}Cl_5$, $C_{13}H_{22}Cl_6$, $C_{13}H_{21}Cl_7$, $C_{13}H_{20}Cl_8$ and $C_{13}H_{19}Cl_9$.

[3] BDE 10, 7, 11, 8, 12-13, 15, 30, 32, 28-33, 35, 37, 75, 71, 66, 47, 49, 77, 100, 119, 99, 116, 85, 155-126, 105, 154, 153, 140, 138, 166, 183, 181, 190 and 209 (the congeners that have concentrations above detection limit are reported in Appendix B.1.)

8. Discussion

8.1. Organochlorines (OCs) and metabolites

8.1.1. Legacy compounds

Polar bears analysed in the present study were compared with polar bears from sub Arctic and Arctic regions of Alaska, Canada and East Greenland both for spatial and temporal trends of chlorinated hydrocarbon (∑ CHL, ∑ HCH, HCB, ∑ CBz, ∑ DDT, $\sum_{42}$PCB, Mirex, dieldrin, octachlorostyrene) and metabolite (p,p'-DDE, oxychlordane, 3-$MeSO_2$-p,p'-DDE, ∑ $MeSO_2$-PCB) concentrations (Verreault et al. 2004a). Briefly, the Svalbard and Greenland population of polar bear had higher loads of DDT-related compounds, nonachlor, oxychlordane and higher-chlorinated PCB congeners. Furthermore, the Svalbard population had higher mean concentration of ∑ PCB and ∑ $MeSO_2$-PCB compared to the other populations investigated in the study. The distribution of ∑ CHL, ∑ CBz, mirex and dieldrin, on the other hand, was relatively uniform between populations of polar bears. Temporal assessment including polar bear samples collected between 1989 to 1993 and 1996 to 2002 showed a general decrease for age-adjusted mean concentrations of ∑ CHL, p,p'-DDE, ∑ PCB, ∑ $MeSO_2$-PCB and 3-$MeSO_2$-p,p'-DDE (Verreault et al. 2004a). Most studies of biological effects in polar bears have studied the effect of environmental levels of PCBs. However, Skaare et al. (2001) reported correlations between PCB, DDE, HCB and the ratio of total T4: free T4, and a correlation between retinol in plasma and HCH. Furthermore, Lie et al. (2004) reported that ∑ OC pesticides (HCB, α-HCH, β-HCH, oxychlordane, trans-nonachlor and p,p'-DDE) contributed significantly to the variation in immunological responses in polar bears from Svalbard and Churchill, Canada. When comparing ∑ PCB (arithmetic mean concentrations of 6458 ng/g l.w.) levels in the present study with no-observed-effect-level (NOEL), no-observed-adverse-effect-level (NOAEL), lowest-observed-effect-level (LOEL) and lowest-observed-adverse-effect-level (LOAEL) in European otter (*Lutra lutra*), mink (*Mustela vison)*, rhesus monkey, harbour seals (*Phocoena phocoena)* and ringed seals (*Phoca hispida*) (AMAP 2004) the polar bears examined in the present study exceed:

- LOAEL for short-time memory reported in rhesus monkey (500-1000 ng/g l.w., blood serum)
- NOAEL for visual memory reported in rhesus monkey offspring (1000 ng/g l.w., blood serum)
- NOEL for vitamin A reduction reported in otter (4000 ng/g l.w., liver)

If range concentrations were considered for ∑ PCB (2336 – 10 050 ng/g l.w.) the polar bears with highest ∑ PCB concentrations would additionally exceed:

- NOEL for reproduction in otter (7500 ng/g l.w., muscle)
- NOEL for kit survival in mink (9000 ng/g l.w., muscle)

Lie et al. (2000) reported that cubs-of-the year and yearlings had higher ∑ PCB blood plasma concentrations than adult polar bears. These authors suggested that this might indicate a higher risk for toxic effects in newborns. Cubs and yearlings were not included in the present study. Age effects in adult females on the other hand, have not been observed (AMAP 2004).

Unfortunately, there are few reportings of coplanar PCB concentrations in Arctic marine mammals. However, the concentrations of coplanare PCBs (CB – 77, 81, 126, 169) from the present study (321 pg/g l.w.) were intermediary compared to concentrations reported in Canadian and eastern Greenland polar bears (Letcher et al. 1996; Sonne et al. 2004). These concentrations are relatively low compared to the coplanar (CB – 77, 81, 126, 169)

concentrations recently reported in glacous gulls (*Larus hyperboreus*) from Bear Island (42.7 to 309 ng/g l.w.) (Verreault et al. 2004b) and harbour porpoise (*Phoceana phoceana*) from the Norwegian Sea (1563 pg/g l.w.) (Berggren et al. 1999).

∑ PCDD and ∑ PCDF concentrations in the polar bears from Svalbard were ranging from 0.9 – 173 pg/g w.w. and 2.6 – 21 pg/g w.w. for ∑ PCDD and ∑ PCDF, respectively. PCDD/Fs have been reported recently in polar bear liver from Alaska (Corsolini et al. 2002), with mean ∑ PCDDs and ∑ PCDFs concentration higher than that reported in the present study. However, PCDD/Fs may be preferentially accumulated in liver relative to subcutaneous fat and thus the comparison may not be entirely valid (Muir, DCG., personal communication). When comparing congener specific concentrations of the present study with Greenland polar bears and Canadian polar bears on a lipid weight basis, the levels in the present study are higher for all identical congeners analysed (1,2,3,4,7,8-HxCDD, 1,2,3,4,6,7,8-HpCDD, OCDD, 2,3,4,7,8-PeCDF, 1,2,3,4,7,8,9-HpCDF and OCDF) in the Greenland polar bears (Sonne et al. 2004), but comparable with the Canadian polar bear concentrations (2,3,7,8-TCDD and 1,2,3,7,8-PeCDD) (AMAP 2004). Compared to harbour porpoise from the Norwegian Sea the ∑ PCDD (2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, 1,2,3,6,7,8-HxCDD, 1,2,3,4,6,7,8-HpCDD and OCDD) levels of the present study are higher (6.32 pg/g l.w. for harbour porpoise versus 45 pg/g l.w. for polar bears of the present study), whereas ∑ PCDF (2,3,7,8-TCDF, 2,3,4,7,8-PeCDF and 1,2,3,6,7,8-HxCDF) are in the same range, with mean concentrations of 7.8 pg/g l.w. and 5.7 pg/g l.w. for harbour porpoise and polar bear, respectively (Berggren et al. 1999). Dioxins and dioxin-like congeners (i.e. coplanar PCBs) bind effectively to the aryl hydrocarbon (Ah) receptor and stimulate enzymes involved in biotransformation, normally measured as hepatic 7-ethoxyresorufin-*O*-deethylase (EROD) activity (Parkinson 2001). Polar bear have a superior ability to biotransform OCs (AMAP 2004). The PCB burden in polar bears is dominated by a small number of congeners, much less than that observed in the major prey item, i.e. the ringed seal (Muir et al. 1988). This may explain the low levels of coplanar PCBs compared to other species such as harbour porpoise and glaucous gull. In concert with this, a positive correlation between ∑ PCB, (with strongest correlation with PCB 156), and cytochrome P450 1A1 (increased by induction through the Ah receptor) have been observed in polar bears (Skaare et al. 2000). When calculating the toxic equivalent (TEQ) concentrations in the present study by using toxic equivalency factors (TEF) from World Health Organization as given in Van den Berg et al. (1998), mean $TEQ_{PCDD/F,non\text{-}ortho PCB,mono\text{-}ortho PCB}$ concentrations of 27.1 ± 7.3 pg/g l.w were obtained (range of 12.2 to 41.7 pg/g l.w.). Comparing these TEQ concentrations with TEQ thresholds for immune suppression in captive harbour seals (AMAP 2004), none of the polar bears exceeds the threshold levels.

8.1.2. Other OCs

There are no studies available on levels of HCBD, pentachloroanisole and α-/β-endosulfan in polar bears. However, if HCBD are compared to other species such as glaucous gull the concentrations in polar bear are higher (not detected in glaucous gulls versus 6.6 ng/g l.w. in polar bears). In rats HCBD cause damage to the kidney tubules which lead to carcinoma of the proximal tubules (Parkinson 2001). No effect studies of HCBD exposure are reported in Arctic animals (AMAP 2004) and there have generally been reported low levels of HCBD in arctic biota and in the North Atlantic Ocean (AMAP 2004). Endosulfan and pentachloranisole were among the top five pesticides measured in air in Arctic Canada. Furthermore, endosulfan is widespread in Arctic seawater, with no observed geographical trends (AMAP 2004). In the present study endosulfan concentrations have mean concentrations of 8.5 ng/g l.w. This is slightly lower than the observed concentrations in male beluga whales *(Delphinapterus leucas)* analysed in 1996/1997 (range of approximately 12 to

16 ng/g l.w.) (AMAP 2004). In the present study α-endosulfan constituted on average 57 % of ∑ endosulfan. Compared to concentrations found in minke whales (*Balaenoptera acutorostrata)* from the North Sea, the reported α-endosulfan is slightly higher in the present study (2.4 ng/g l.w. in the minke whales (sampled in 2003) compared to 6.7 ng/g l.w. in the polar bears of the present study), whereas α-endosulfan levels of minke whales from Svalbard were detected at very low concentrations (≈ 0) (AMAP 2004). It is noteworthy that a 2.1 fold increase in age-adjusted mean concentrations of endosulfan in male belugas was observed over a 15 year period (1982-1997) (AMAP 2004). Reproductive and immune effects, as well as an increased CYP P450-dependent monooxygenase have been observed in laboratory fish and mammals exposed to endosulfan (de March et al. 1998).

Pentachloranisole is a transformation product of pentachlorophenol (Muir DCG, personal communication). Low levels of pentachloroanisole were found in the polar bears of the present study (Table 2). Furthermore, low levels of pentachloroanisole have been reported in blubber of beluga whales and ringed seals from the Canadian Arctic (Muir, DCG., personal communication). Pentachloroanisole is found in the Arctic atmosphere (AMAP 2004) and thus may accumulate through the marine food chain. It may also be formed from biomethylation of pentachlorophenol, which is a suggested degradation product of HCB (Muir DCG, personal communication).

In the present study 13 toxaphene congeners were detected, with b8-1413 (parlar 26), b8-1414/b8-1945 and b9-1679 (parlar 50) as the toxaphene congeners of highest concentrations. There have been few reports of toxaphene levels in polar bears, and only parlar 26, 50 and 62 have been reported previous in polar bear populations from Svalbard (Føreid et al. 2000). Concentrations of parlar 26 and 50 in the female polar bear population studied by Føreid et al. (2000) corresponded to the concentrations found in the present study (parlar 26: 5.7 ng/g l.w. versus 5.9 ng/g l.w in the study of Føreid et al. (2000) and the present study, respectively. Parlar 50: 3.6 ng/g l.w. versus 6.2 ng/g l.w. in the study of Føreid et al. (2000) and the present study, respectively). However, the toxaphene levels in polar bears from Svalbard is 2-3 times higher compared to unpublished data from polar bears in Alaska (Muir, DCG., personal communication). The concentrations of parlar 26 and 50 in the present study are comparable to reported levels in ringed seal from Svalbard (Wolkers et al. 1998; Føreid et al. 2000). No effect studies of toxaphene are reported in Arctic animals, but toxaphene have been shown to retard growth and morphological development in laboratory rats. Furthermore, induction of CYP P 450 activity in laboratory animals was only observed after administering doses that were close to those known to cause mortality in laboratory rats. It was suggested that toxaphene would not induce CYP P450 enzymes in free-ranging animals exposed to toxaphene levels at environmental levels (AMAP 2004).

The PCN profile was dominated by the congeners, diPCN 3, diPCN 5 and diPCN 6, which made up more than 70% of the ∑ PCN. The presence of the dichloronaphthalenes was unexpected. Possibly they are dechlorination products of higher chlorinated congeners (Muir, DCG., personal communication). PCN has been reported in Alaska polar bear liver samples (N=5) with mean concentrations of 3.2 ng/g l.w. (Corsolini et al. 2002). Only penta-, hexa- and heptachloronaphthalenes were determined in the study by Corsolini et al. (2002), where pentachloronaphthalenes were the major components. Compared to other marine species such as beluga whales the PCN concentrations in polar bears are higher (beluga whale have PCN concentrations of 0.04 to 0.4 ng/g l.w. compared to 1.2 to 52 ng/g l.w. in the polar bears of the present study) (AMAP 2004). PCNs are planar and seem to exert its effect via the Ah receptor. Chronic exposure to PCN has led to similar effects to those seen for dioxins or dioxin-like congeners. The most toxic PCN are the penta and hexa congeners. There are few effect studies of mammals on PCN, but it was reported 16-days LD50 (lethal dose that kills 50 % of the animals) of 4.2 ng/egg for medaka (*Oryzias latipes*) eggs injected with doses

from 0.3 to 30 ng/egg of Halowax 1014 (mixture of PCN), whereas premature hatching of embryos at all doses were reported for Halowax 1013. Halowax 14 was found to be the most toxic mixture, however, 5.585-fold less potent than TCDD (Villalobos et al. 2000). The study of Villalobos et al. (2000) suggests that some mixtures of PCN are very potent in causing biological effects in animals.

Chlorinated paraffins seem to be less toxic than other persistent organic pollutants. In a Canadian study of atmospheric levels of SCCPs the concentrations were quantified above detection limits only in the summer months (AMAP 2004). In the present study SCCP were detected at very low concentrations in the polar bear fat samples. The profile of SCCP chain length did not resemble technical products, which consist mainly of Cl_5 to Cl_9 substituted decanes to tridecanes. Only trichloro and tetrachloroundecanes were detected in the present study. Studies of beluga whale and ringed seals have revealed SCCP concentrations present in the same fraction as toxaphene, however, they are not detected in seabird eggs (Tomy et al. 1999; Muir et al. 2004). Very low and virtually non detected SCCP is not surprising since they normally are readily oxidized by most homeotherms (Muir et al. 1999).

8.2.3. Metabolites

The major compounds making up ∑ HO-PCB (sum of 11 congeners) in plasma were 4-HO-CB 112, 4-HO-CB 107, 4-HO-CB 172, 4-HO-CB 193 and 4-HO-CB 187, the latter constituting 41 % of the ∑ HO-PCB. 4-HO-CB 187 have been reported to be the HO-PCB metabolite of highest concentrations in Arctic mammals (AMAP 2004). Mean concentrations of ∑ OH-PCB in a study of polar bear plasma from Resolute in Canada and Svalbard were 57 ng/g w.w. and 218 ng/g w.w, respectively (AMAP 2004). The ∑ OH-PCB in plasma of polar bear in the present study had intermediary concentrations of these two populations (Table 2). Interestingly, there have been reported significantly higher concentrations of ∑ HO-PCB in females compared to males, and it has been suggested that females have a higher binding affinity for OH-PCBs in plasma or a higher capacity to form OH-PCBs than males (AMAP 2004). The ∑ OH-PCBs concentrations found in ringed seals have been shown to be 1000 times lower than the levels found in polar bear (AMAP 2004). Furthermore, the polar bear of the present study have much higher concentrations of HO-PCBs than observed concentrations in birds (Verreault et al. 2004b).

Sandau et al. (2000) identified HO-HpCS in polar bear plasma. Mean HO-Hp-CS concentrations of 9.1 ng/g w.w. were reported in polar bear, whereas PCP concentrations were reported to be in the range 0.09 to 0.53 ng/g w.w (Sandau et al. 2000). This corresponds to the concentrations found in the present study, 8.8 ng/g w.w and 0.3 ng/g w.w. for HO-HpCS and PCP, respectively. Furthermore, HO-HpCS concentrations in the present study are higher than reported levels in ringed seal plasma and female glaucous gull plasma from Bear Island (0.06 ng/g w.w. and 0.17 ng/g w.w., respectively). The PCP concentrations reported in female glaucous gull and ringed seal are comparable with the reported concentrations in the present study (Verreault et al. 2004b; Sandau 2000).

The methyl sulfone profile was dominated by the congeners 3-$MeSO_2$-CB-101, 4-$MeSO_2$-CB-101, 4-$MeSO_2$-CB-87 and 4-$MeSO_2$-CB-149. Together, these methyl sulfones made up more than 40% of ∑ $MeSO_2$-PCB**.** As discussed in section 8.1.1, concentrations of ∑ $MeSO_2$-PCB were highest in the Svalbard population of polar bears, compared to the other Arctic populations (Alaska, Canada and East Greenland). Furthermore, time-trend analysis showed a general decrease for age-adjusted mean concentrations of ∑ $MeSO_2$-PCB and 3-$MeSO_2$-p,p'-DDE (Verreault et al. 2004a). Levels of ∑ $MeSO_2$-PCB in the present study are much higher than the observed plasma levels of humans (Letcher et al. 2000). Furthermore, the levels of ∑ $MeSO_2$-PCB in polar bears are higher compared to levels of ∑ $MeSO_2$-PCB observed in blubber of ringed seals from the Canadian Arctic. Ringed seals and grey seal

(*Halichoerus grypus*) blubber/liver from the Baltic Sea, on the other hand, have higher concentrations than the polar bears of the present study (Letcher et al. 2000). It is suggested that a significant proportion of methylsulfone-PCB metabolites are bioaccumulated from seals (Norstrom 2001).

The $MeSO_2$-p,p'-DDE concentrations of the polar bears in the present study were comparable to concentrations reported in polar bears from the Canadian arctic. (However, $MeSO_2$-p,p'-DDE were only detected in 3 out of 15 analysed polar bears). Furthermore, these concentrations are higher compared to the lower trophic levels in the polar bear food chain, i.e. polar cod (*Boregadus saida*) and ringed seal (Letcher et al. 1998).

A competition between T4 and hydroxylated OCs (a result of the biotransformation) for binding sites on the TTR (transthyretin) protein may lead to thyroid hormone disruption (Brouwer et al. 1998). Sandau et al. (2000) found that the binding affinity of 4-OH-HpCS to the transthyretin receptor was 1.1 times that of thyroxin (T4). Though, it is interestingly that when examining a large data set (N=60) of polar bears from Svalbard and Canada, plasma T4 concentrations were correlating negatively with persistent and non-persistent PCB congeners, but not with HO-PCBs (AMAP 2004). Furthermore, HO-PCB metabolites caused indirect estrogen effects in laboratory mammals in addition to effects on the thyroxine metabolism (Letcher et al. 2000; Sandau 2000). Several studies have demonstrated the toxicological potential of $MeSO_2$-PCBs, such as tissue selective retention via non-covalent protein binding, thyroid hormone reduction, increased thyroid gland weight and induction of the cythochrome P450 family. Furthermore, *in vitro* studies of humans and mouse have revealed endocrine related implications due to the affinity of several persistent $MeSO_2$-PCBs to the glucocorticoid receptor (Letcher et al. 2000).

8.2. Brominated flame retardants (BFR) and metabolites

The ∑ PBDE concentrations were relatively low and the pattern was dominated by BDE-47, accounting for more than 80% of the analysed PBDEs. This is consistent with the findings of van Bavel et al. (2001) in polar bears from Svalbard were the congener pattern was dominated by BDE-47 and a methoxy TeBDE. The concentrations of BDE 47 in the present study and in the study of van Bavel et al. (2001) are higher than reported levels in ringed seal, minke whale and beluga whales from the Canadian Arctic, Barents Sea and Candian Arctic, respectively (AMAP 2004). The beluga whales from Svalbard and glaucous gulls from Bear Island, on the other hand, have higher concentrations of BDE 47 than the reported levels in the present study (van Bavel et al. 2001; Verreault et al. 2004b). The findings of a dominating pattern of BDE-47 and methoxy TeBDE in polar bears from Svalbard (van Bavel et al. 2001) may indicate that polar bears are effectively metabolizing PBDEs. However, in the present study the concentrations of HO-BDE were low and out of 14 analysed metabolites, only 2 of them were above (but of low concentrations) the detection limit (and only detected in 2 out of 15 analysed polar bears). HBCDD concentrations in the polar bears from Svalbard were in the same range as glaucous gulls from Bear Island (polar bear range of 17 to 80 ng/g l.w., whereas the glaucous gull have a range of 6 to 122 ng/g l.w.).

Because of molecular analogies between PCBs and PBDEs comparable toxic effects are expected. Exposure to decaBDEs has been found to induce thyroid hyperplasia, heptacellular and thyroid adenomas and carcinomas in mice (AMAP 2004). However, decaBDE (BDE 209) were not detected in the present study. Furthermore, BDE 47 has been shown to reduce the T4 and vitamin A levels in rats. It has been suggested that this is a result of BDE metabolites competing with T4 for binding sites on the TTR (AMAP 2004).

8.3. Perfluorinated alkylated substances (PFAS)

In a circumpolar study of perfluorinated acids in polar bear, the plasma wet weight concentrations of PFAS of the present study were converted to liver concentrations by using conversion factors obtained from Alaskan polar bears (Smithwick et al. 2004). Reported liver concentrations of PFOS for polar bears from the North American Arctic and European Arctic were in the range 729 ng/g w.w to 2730 ng/g w.w. (Smithwick et al. 2004). Liver concentrations of PFOS are thus one of the most prominent contaminants in polar bear liver. A geographical trend of PFOS concentrations in the liver samples was suggested, with polar bears from South Hudson Bay and East Greenland having significantly higher concentrations than polar bears from Svalbard, High Arctic and western Northwest Territories. The high concentrations found in polar bear liver from South Hudson Bay and East Greenland was suggested to be due to the proximity to sources in Europe and Eastern North America (Smithwick et al. 2004). In a study by Kannan et al. (2001) PFOS concentrations in polar bear plasma from Beaufort Sea were reported to be in the range 26-52 ng/ml. (Kannan et al. 2001). Thus concentrations of PFOS in plasma of Svalbard polar bears appear to be 2-3 times higher than those in Alaska (Table 2). Compared to plasma concentrations of ringed seal from Svalbard sampled in 1996 and 1998 (8.1 and 10.1 ng/ml, respectively) (Kannan et al. 2001) the concentrations of the polar bears in the present study are much higher (Table 2) suggesting that PFOS have a biomagnification potential (Giesy and Kannan 2001). Animals from more polluted and industrialized regions, such as North American Great Lakes, Baltic Sea and Mediterranean Sea, carried higher loads of PFOS than animals from more remote locations (Giesy and Kannan 2001).

Other PFAS, such as PFOA and PFOSA (a possible precursor of PFOS) have been detected at low levels in polar bear blood plasma, but have not been reported. It is difficult to compare blood concentrations with liver concentrations due to the special physical properties of PFAS. However, Smithwick et al. (2004) converted the plasma concentrations in the present study to liver concentrations. In this circumpolar study, liver concentrations of PFOA and PFOSA from the North American Arctic and European Arctic were in the range 2.2 to 36 ng/g w.w. and non detect to 8.5 ng/g w.w., respectively. Age trends were observed for PFOS concentrations in the Canadian polar bears, but were not reported for the Svalbard population (Smithwick et al. 2004). Other studies have not reported age-dependent increase in PFOS concentrations (Kannan et al. 2001).

There have not been many effect studies of PFOS, but NOAEL and LOAEL for second-generation effects of PFOS concentration (wet weight liver) in rats have been estimated. The PFOS levels reported in polar bear plasma of the present study (converted to liver concentrations by using conversion factors; 1290 ng/g w.w.) (Smithwick et al. 2004) are well below the estimated NOAELs and LOAELs estimated (15 000 and 58 000 ng/g w.w. in liver, respectively) for second-generation effects in rats (AMAP 2004).

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10. Appendix

Arithmetic means (only for samples above the detection limit) with standard deviation (± SD) and ranges (min-max), for the different analytes, in plasma and fat samples of polar bears from Svalbard. Concentrations in pg/g wet weight for PCDDs, PCDFs and co-planar PCBs and in ng/g wet weight for all the other compounds. The number of samples above the detection limit of the analysed contaminant is shown relatively to the number of samples analysed (N).

Appendix A. Organochlorines and metabolites

Appendix A.1. *Coplanar polychlorinated biphenyls (PCBs) in pg/g wet weight*

		Fat sampels	
Congeners	**N**	**Mean ± SD**	**Min – Max**
Lipid %	15	56.3 ± 15.1	23 – 80.8
CB 77	15/15	39.4 ± 21.0	19.0 – 103
CB 81	11/15	5.1 ± 1.9	2.4 – 9.1
CB 126	15/15	32.3 ± 13.2	9.1 – 59.6
CB 169	15/15	105 ± 71.5	29.0 – 241

Appendix A.2. *Polychlorinated dibenzo-p-dioxins (PCDDs, CDD) and polychlorinated dibenzofurans (PCDFs) in pg/g wet weight*

		Fat samples	
Congeners	**N**	**Mean ± SD**	**Min – Max**
Lipid %	15	56.3 ± 15.1	23 – 80.8
2,3,7,8-TCDD	12/15	1.6 ± 0.8	0.5 – 2.7
1,2,3,7,8-PeCDD 3	9/15	1.0 ± 0.5	0.5 – 1.7
1,2,3,4,7,8-HxCDD	6/15	1.1 ± 0.8	0.5 – 2.5
1,2,3,6,7,8-HxCDD	13/15	2.4 ± 1.1	0.9 – 4.1
1,2,3,7,8,9-HxCDD	6/15	0.6 ± 0.4	0.3 – 1.3
1,2,3,4,6,7,8-HpCDD	14/15	3.5 ± 2.8	0.9 – 12.3
OCDD	15/15	16.8 ± 39.2	0.9 - 158
2,3,7,8-TCDF	8/15	1.4 ± 0.9	0.5 – 3.5
1,2,3,7,8-PeCDF	6/15	0.9 ± 0.5	0.5 – 1.7
2,3,4,7,8-PeCDF	13/15	1.8 ± 1.1	0.8 – 4.1
1,2,3,4,7,8-HxCDF	0/15	–	< 0.3
1,2,3,6,7,8-HxCDF	6/15	0.9 ± 0.6	0.5 – 1.8
1,2,3,7,8,9-HxCDF	1/15	1.0	< 0.3
2,3,4,6,7,8-HxCDF	13/15	1.0 ± 0.6	0.4 – 2.6
1,2,3,4,6,7,8-HpCDF	10/15	1.6 ± 0.8	0.6 – 3.5
1,2,3,4,7,8,9-HpCDF	5/15	1.7 ± 1.2	0.6 – 3.4
OCDF	6/15	1.6 ± 1.2	0.6 – 3.9

***Appendix A.3**. Chlorobenzenes (CBz): dichlorobenzene (DCB), trichlorobenzene (TCB), tetra-TCB, penta-TCB), hexachlorobenzene (HCB) in ng/g wet weight*

		Fat samples	
Congeners	**N**	**Mean ± SD**	**Min – Max**
Lipid %	15	56.3 ± 15.1	23 – 80.8
1,3-DCB	0/15	–	< 0.004
1,4-DCB	15/15	39.3 ± 31.4	6.9 – 112
1,2-DCB	3/15	12.1 ± 14.9	2.0 – 29.2
1,3,5-TCB	7/15	0.9 ± 1.0	0.2 – 2.9
1,2,4-TCB	14/15	0.9 ± 0.5	0.1 – 2.2
1,2,3-TCB	14/15	1.2 ± 0.5	0.3 – 2.2
1,2,3,4-TTCB	5/15	0.2 ± 0.1	0.1 – 0.3
PECB	15/15	7.9 ± 2.8	1.4 – 13.3
HCB	15/15	58.1 ± 33.7	9.9 – 139.8

***Appendix A.4**. Chlordanes (CHL) and metabolites: heptachlor, heptachlor epoxide, a-chlordane, g-chlordane, trans-nonachlor, cis-nonachlor, oxychlordane, methoxychlor in ng/g wet weight*

		Fat samples	
Congeners	**N**	**Mean ± SD**	**Min – Max**
Lipid %	15	56.3 ± 15.1	23 – 80.8
Heptachlor	6/15	0.1 ± 0.1	0.1 – 0.4
Heptachlor epoxide	15/15	54.5 ± 19.9	22.5 – 91.0
a-chlordane	10/15	1.0 ± 0.8	0.1 – 2.0
g-chlordane	15/15	36.5 ± 31.2	3.5 – 101
Trans-nonachlor	14/15	90.1 ± 46.6	8.5 – 152
Cis-nonachlor	14/15	3.9 ± 3.4	0.3 – 9.7
Oxychlordane	5/15	687 ± 238	346 – 1060
Methoxychlor	0/15	–	< 0.004

***Appendix A.5**. Aldrin, Endrin, dieldrin, mirex and octachlorostyrene in ng/g wet weight*

		Fat samples	
Congeners	**N**	**Mean ± SD**	**Min – Max**
Lipid %	15	56.3 ± 15.1	23 – 80.8
Aldrin	15/15	3.3 ± 1.9	1.0 – 6.6
Endrin	7/15	2.8 ± 1.3	1.8 – 5.2
Dieldrin	15/15	92.1 ± 31.0	29.6 – 142
Mirex	15/15	6.8 ± 4.1	0.8 – 14.7
Octachlorostyrene	15/15	9.6 ± 3.9	3.3 – 19.8

***Appendix A.6**. Dichlorodiphenyltrichloroethane (DDT, ortho (o) and para (p)) and metabolites (DDE and DDD)) in ng/g wet weight*

Congeners	N	Fat samples Mean ± SD	Min – Max
Lipid %	15	56.3 ± 15.1	23 – 80.8
o,p-DDT	0/15	–	< 0.004
p,p-DDT	15/15	9.7 ± 7.2	1.3 – 27.5
o,p-DDD	11/15	3.7 ± 3.7	0.7 – 11.7
p,p-DDD	6/15	8.3 ± 3.4	2.4 – 12.9
o,p-DDE	1/15	2.5	–
p,p-DDE	15/15	115.2 ± 49.7	20.9 – 202

***Appendix A.7**. Hexachlorocyclohexanes (HCH) in ng/g wet weight*

Congeners	N	Fat samples Mean ± SD	Min – Max
Lipid %	15	56.3 ± 15.1	23 – 80.8
a-HCH	15/15	11.1 ± 4.1	1.8 – 18.1
b-HCH	15/15	29.7 ± 13.1	11.5 – 61.1
g-HCH	12/15	0.7 ± 0.4	0.1 – 1.2

***Appendix A.8**. Hexachlorobutadiene (HCBD), pentachloroanisole and endosulfans in ng/g wet weight*

Congeners	N	Fat samples Mean ± SD	Min – Max
Lipid %	15	56.3 ± 15.1	23 – 80.8
HCBD	5/15	3.7 ± 2.4	1.2 – 8.9
Pentachloroanisole	15/15	1.9 ± 1.2	0.8 – 5.2
a-endosulfan	15/15	3.8 ± 2.2	1.3 – 7.8
b-endosulfan	5/15	2.9 ± 0.8	2.2 – 4.3

***Appendix A.9.** Polychlorinated naphthalenes (PCNs) in ng/g wet weight*

Congeners	N	Fat samples Mean ± SD	Min – Max
Lipid %	15	56.3 ± 15.1	23 – 80.8
DiPCN 3	14/14	0.9 ± 1.5	0.1 – 6.1
DiPCN 5	14/14	1.1 ± 2.1	0.1 – 8.4
DiPCN 6	14/14	1.1 ± 2.0	0.1 – 7.9
DiPCN 9	4/14	0.2 ± 0.3	0.02 – 0.6

DiPCN 10	14/14	0.3 ± 0.5	0.03 – 1.9
TriPCN 13	14/14	0.03 ± 0.05	0.003 – 0.2
TriPCN 14	14/14	0.1 ± 0.2	0.04 – 1.0
TriPCN 15	14/14	0.03 ± 0.07	0.004 – 0.3
TriPCN 16	14/14	0.05 + 0.1	0.006 – 0.4
TriPCN 17	14/14	0.07 + 0.2	0.006 – 0.6
TriPCN 18	12/14	0.02 ± 0.03	0.005 – 0.09
TriPCN 19	14/14	0.08 ± 0.2	0.007 – 0.7
TriPCN 20	0/14	–	< 0.001
TriPCN 21	0/14	–	< 0.001
TriPCN 22	2/15	0.05 ± 0.05	0.01 – 0.08
TriPCN 23	14/14	0.02 ±0.01	0.01 – 0.07
TriPCN 24	0/14	–	< 0.001
TriPCN 25	0/14	–	< 0.001
TriPCN 26	14/14	0.04 ± 0.08	0.003 – 0.3
TetraPCN 27	10/14	0.02 ± 0.02	0.005 – 0.006
TetraPCN 28	14/14	0.03 ± 0.02	0.007 –0.08
TetraPCN 29	0/14	–	< 0.001
TetraPCN 30	0/14	–	< 0.001
TetraPCN 31	8/14	0.02 ± 0.03	0.005 –0.09
TetraPCN 32	3/14	0.008 ± 0.002	0.006 –0.009
TetraPCN 33	14/14	0.03 ± 0.03	0.007 –0.1
TetraPCN 34	0/14	–	< 0.001
TetraPCN 35	14/14	0.02 ± 0.01	0.002 – 0.04
TetraPCN 36	3/14	0.01 ± 0.009	0.003 – 0.02
TetraPCN 37	0/14	–	< 0.001
TetraPCN 38	14/14	0.05 ± 0.03	0.007 – 0.1
TetraPCN 39	0/14	–	< 0.001
TetraPCN 40	0/14	–	< 0.001
TetraPCN 41	1/14	0.006	–
TetraPCN 42	11/14	0.008 ± 0.006	0.003 – 0.02
TetraPCN 43	0/14	–	< 0.001
TetraPCN 44	2/14	0.01 ± 0.01	0.006 – 0.02
TetraPCN 45	0/14	–	< 0.001
TetraPCN 46	14/14	0.03 ± 0.01	0.009 – 0.05
TetraPCN 47	11/14	0.01 ± 0.005	0.002 –0.02
TetraPCN 48	0/14	–	< 0.001
PentaPCN 49	1/14	0.003	< 0.001
PentaPCN 50	14/14	0.07 ± 0.06	0.008 – 0.2
PentaPCN 51	0/14	–	< 0.001
PentaPCN 52	14/14	0.02 ± 0.03	0.007 – 0.01
PentaPCN 53	7/14	0.01 ± 0.007	0.004 – 0.02
PentaPCN 54	3/14	0.02 ± 0.01	0.006 – 0.03
PentaPCN 55	0/14	–	< 0.001
PentaPCN 56	3/14	0.03 ± 0.007	0.02 – 0.04
PentaPCN 57	13/14	0.01 ± 0.01	0.004 – 0.04
PentaPCN 58	14/14	0.04 ± 0.07	0.005 – 0.3
PentaPCN 59	6/14	0.007 ± 0.002	0.004 – 0.01
PentaPCN 60	0/14	–	< 0.001
PentaPCN 61	4/14	0.005 ± 0.002	0.003 – 0.007
PentaPCN 62	4/14	0.009 ± 0.009	0.003 – 0.02
Hexa PCN 63	2/14	0.004 ± 0.001	0.003 – 0.005
Hexa PCN 64	9/14	0.005 ± 0.002	0.002 – 0.007
Hexa PCN 66	14/14	0.009 ± 0.004	0.004 – 0.02
Hexa PCN 67	0/14	–	< 0.001
Hexa PCN 68	0/14	–	< 0.001
Hexa PCN 69	3/14	0.003 ± 0.002	0.002 – 0.005
Hexa PCN 70	6/14	0.003 ± 0.002	0.002 – 0.006

Hexa PCN 71	8/14	0.005 ± 0.003	0.002 – 0.01
Hexa PCN 72	0/14	–	< 0.001
HeptaPCN 73	14/14	0.02 ± 0.01	0.005 – 0.04
HeptaPCN 74	0/14	–	< 0.002
OctaPCN 75	14/14	0.009 ± 0.004	0.003 – 0.016

Appendix A.10. *Toxaphenes and short chain chlorinated paraffins (SCCPs, C_{10}-C_{13} chloroalkanes) in ng/g wet weight*

		Fat samples	
Congeners	**N**	**Mean ± SD**	**Min – Max**
Lipid %	15	56.3 ± 14.1	23.0 – 80.8
b7-1001	6/15	0.4 ± 0.3	0.2 – 1.0
b8-1413 (26)	15/15	3.3 ± 3.8	0.02 – 12.7
b8-1412	15/15	2.1 ± 2.3	0.3 – 8.9
b7-1450	1/15	0.9	–
b7-515	0/15	–	< 0.1
b7-1474/ b7-1440/ b7-495	2/15	1.1 ± 1.4	0.1 – 2.1
b8-789	14/15	3.4 ± 3.1	0.6 – 12.4
b7-1059a	0/15	–	< 0.1
b8-531	1/15	0.48	–
b8-1414/ b8-1945	15/15	4.0 ± 2.0	0.6 – 9.8
b8-806	7/15	0.3 ± 0.2	0.1 – 0.8
b8-2229	15/15	7.9 ± 4.0	1.4 – 19.1
b8-810	2/15	1.5 ± 1.4	0.5 – 2.5
b9-1679 (50)	15/15	3.5 ± 4.5	0.2 – 16.7
b9-718	0/15	–	< 0.1
b8-1471	5/15	1.0 ± 0.2	0.7 – 1.4
b9-743/ b9-2006	0/15	–	< 0.1
b9-1046	0/15	–	< 0.1
b9-715	15/15	1.8 ± 0.9	0.2 – 3.8
b9-1025 (62)	0/15	–	< 0.1
C10 (C13-C19)	0/15	–	< 0.0005
C11 (C13-C19)	5/15	0.5 ± 1.0	0.004 – 2.6
C12 (C14-C19)	0/15	–	< 0.0005
C13 (C14-C19)	0/15	–	< 0.0005

Appendix A.11. *Hydroxylated PCBs (HO-PCBs) in ng/g wet weight*

		Plasma samples	
Congeners	**N**	**Mean ± SD**	**Min – Max**
Lipid %	15	1.0 ± 0.7	0.5 – 3.6
4'-HO-CB 121	0/15	–	< 0.001
3'-HO-CB 85	0/15	–	< 0.001
4'-HO-CB 120	4/15	0.6 ± 0.5	0.2 – 1.3
4-HO-CB 112	6/15	11.4 ± 23.7	1.0 – 59.7
4-HO-CB 107	15/15	7.9 ± 6.1	0.3 – 27.0
4-HO-CB 165	15/15	6.0 ± 5.4	0.1 – 18.3
3'-HO-CB 138	2/15	0.8 ± 0.1	0.7 – 0.9

4'-HO-CB 130	15/15	4.5 ± 10.4	0.2 – 42.0
3'-HO-CB 187	15/15	2.9 ± 1.4	0.5 – 5.3
4-HO-CB 187	15/15	38.4 ± 23.6	0.3 – 95.5
4'-HO-CB 159	15/15	0.4 ± 0.2	0.1 – 1.0
3'-HO-CB 180	14/15	1.2 ± 0.6	0.3 – 2.9
4-HO-CB 172	15/15	8.2 ± 4.7	0.2 – 18.1
4-HO-CB 193	15/15	12.3 ± 5.6	0.1 – 23.1
4,4' di-HO-CB 202	15/15	3.6 ± 2.5	0.2 – 9.1
4'-HO-CB 208	15/15	3.5 ± 1.8	0.1 – 7.7

Appendix A.12. *Methylsulfonyl PCBs ($MeSO_2$-PCBs) and methylsulfonyl DDE ($MeSO_2$-DDE) in ng/g wet weight*

		Fat samples	
Congeners	**N**	**Mean ± SD**	**Min – Max**
Lipid %	15	56.3 ± 14.1	23.0 – 80.8
3-$MeSO_2$-CB 52	0/15	–	< 0.003
3-$MeSO_2$-CB 49	15/15	7.3 ± 4.3	1.6 – 15.4
4-$MeSO_2$-CB 52	0/15	–	< 0.003
4-$MeSO_2$-CB 49	15/15	7.9 ± 4.5	1.9 – 15.6
3-$MeSO_2$-CB 64	15/15	2.2 ± 1.9	0.4 – 6.9
4-$MeSO_2$-CB 64	15/15	3.5 ± 2.3	0.9 – 8.0
3-$MeSO_2$-CB 91	15/15	2.5 ± 2.0	0.4 – 7.0
4-$MeSO_2$-CB 91	15/15	3.1 ± 1.9	0.7 – 6.1
3-$MeSO_2$-CB 70	15/15	4.0 ± 2.1	0.9 – 8.1
3-$MeSO_2$-CB 101	15/15	19.7 ± 10.8	4.6 – 38.5
4-$MeSO_2$-CB 70	15/15	3.8 ± 2.1	0.8 – 8.0
4-$MeSO_2$-CB 101	15/15	16.3 ± 11.4	2.8 –36.2
3-$MeSO_2$-CB 87	15/15	5.5 ± 3.7	1.1 – 13.2
3-$MeSO_2$-CB 110	0/15	–	< 0.003
3-$MeSO_2$-CB 149	15/15	2.6 ± 1.4	0.7 – 5.5
4-$MeSO_2$-CB 110	15/15	5.1 ± 2.8	1.2 – 9.8
4-$MeSO_2$-CB 87	15/15	20.4 ± 14.0	3.4 – 50.3
4-$MeSO_2$-CB 149	15/15	16.2 ± 9.0	3.3 – 31.5
3-$MeSO_2$-CB 132	9/15	0.7 ± 0.3	0.3 – 1.0
4-$MeSO_2$-CB 132	15/15	5.4 ± 3.0	1.0 – 11.1
3-$MeSO_2$-CB 141	1/15	1.4 ± 1.0	0.4 – 3.5
4-$MeSO_2$-CB 141	15/15	4.7 ± 3.4	0.7 – 11.6
4-$MeSO_2$-CB 174	15/15	1.6 ± 0.9	0.3 – 3.7
3-$MeSO_2$-4,4'-DDE	3/15	1.3 ± 0.1	1.1 – 1.3

Appendix A.13. *Pentachlorophenol (PCP) and HO-heptachlorostyrene (4-HO-HpCS) in ng/g wet weight*

Congeners	N	Plasma samples Mean ± SD	Min – Max
Lipid %	15	1.0 ± 0.7	0.5 – 3.6
PCP	14/15	0.3 ± 0.2	0.1 –0.8
4-HO-HpCS	15/15	8.8 ± 3.7	0.1 – 14.2

Appendix B. Brominated flame retardants and metabolites (BFR)

Appendix B.1. *Polybrominated diphenylethers (PBDEs) congeners and hexabromocyclododecane (HBCDD) in ng/g wet weight*

Congeners	N	Fat samples Mean ± SD	Min – Max
Lipid %	15	56.3 ± 15.1	23.0 – 80.8
BDE 17	0/15	–	< 0.1
BDE 28/33	0/15	–	< 0.1
BDE 71	0/15	–	< 0.1
BDE 47	15/15	24.1 ± 10.3	9.5 – 41.7
BDE 66	0/15	–	< 0.1
BDE 100	11/15	0.9 ± 0.4	0.4 – 1.6
BDE 99	13/15	2.1 ± 1.1	0.7 – 4.7
BDE 85	0/15	–	< 0.1
BDE 154	0/15	–	< 0.1
BDE 153	13/15	2.9 ± 1.2	1.4 – 5.8
BDE 138	0/15	–	< 0.1
BDE 183	0/15	–	< 0.1
BDE 190	0/15	–	< 0.1
BDE 209	0/15	–	< 0.1
HBCDD	15/15	25.6 ± 9.0	9.7 – 44.8

Appendix B.2. *Hydroxylated PBDEs (HO-BDEs) in ng/g wet weight*

Congeners	N	Plasma samples Mean ± SD	Min – Max
Lipid %	15	1.0 ± 0.7	0.5 – 3.6
6'-HO-BDE 17	0/15	–	< 0.001
4'-BDE 17	0/15	–	< 0.001
6'HO-BDE 49	0/15	–	< 0.001
2'-HO-BDE 68	0/15	–	< 0.001
6-HO-BDE 47	0/15	–	< 0.001
3-HO-BDE 47	0/15	–	< 0.001
5-HO-BDE 47	0/15	–	< 0.001
4'-HO-BDE 49	2/15	0.1 ± 0.04	0.08 – 0.13

4-HO-BDE 42	2/15	0.09 ± < 0.00	0.09 – 0.09
6-HO-BDE 90	0/15	–	< 0.001
6-HO-BDE 99	0/15	–	< 0.001
2-HO-BDE 123	0/15	–	< 0.001
6-HO-BDE 85	0/15	–	< 0.001
6-HO-BDE 137	0/15	–	< 0.001

Appendix C. Perfluorinated alkylated substances (PFAS)

Appendix C.1. *Perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and perfluooctanesulfamide (PFOSA) in ng/g wet weight*

		Plasma samples	
Congeners	**N**	**Mean ± SD**	**Min – Max**
PFOS	14/14	97.2 ± 22.9	56.7 – 150
PFOA	14/14	1.6 ± 0.7	0.7 – 2.8
PFOSA	9/14	0.6 ± 0.1	0.5 – 0.8

Statens forurensningstilsyn (SFT)

Postboks 8100 Dep. 0032 Oslo
Besøksradresse: Strømsveien 96

Telefon: 22 57 34 00
Telefaks: 22 67 67 06
E-post: postmottak@sft.no
Internett: www.sft.no

Utførende institusjon Norsk Polarinstitutt (Tromsø)	Kontaktperson SFT Linn Bryhn Jacobsen	ISBN-nummer 82-7655-490-3
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Forfattere
Geir Wing Gabrielsen, Lisa Bjørnsdatter Knudsen, Jonathan Verreault, Kerstin Pusch, Derek DC. Muir & Robert J. Letcher

Tittel - norsk og engelsk
Halogenerte organiske miljøgifter og deres metabolitter i blod og fettvev fra isbjørn (*Ursus* maritimus) på Svalbard
Halogenated organic contaminants and metabolites in blood and adipose tissues of polar bears (*Ursus maritimus*) from Svalbard

Summary
In the present project a wide range of persistent organic contaminants (standard and emerging) and their metabolites in polar bears (*Ursus maritimus*) from Svalbard have been determined. Current levels and congener patterns were examined in polar bear adipose tissue and blood samples. These values were compared to levels measured in other studies from polar bears. At present, results for some of these emerging compounds are not available for polar bears. It is therefore important to screen these compounds so it is possible in later studies to link current levels of these compounds with possible biological effects. Further on, the data obtained from Svalbard will be added to a comprehensive assessment on contaminants of polar bears from Arctic regions (Greenland, Canada and Alaska).

4 emneord isbjørn, arktis, nye miljøgifter, nivå	4 subject words polar bear, Arctic, emerging contaminants, levels