Organic Pollutants in Northern Fulmars (*Fulmarus glacialis*) from Bjørnøya

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Photo: Gunnar Rise

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1. Preface

The aim of the present project was to determine a wide range of new and established organic pollutants in northern fulmars (*Fulmarus glacialis*) breeding on Bjørnøya. Current levels and congener patterns in northern fulmar liver samples were compared to previous reported levels in northern fulmar and other Arctic seabirds.

The present study was a collaboration between the Norwegian Polar Institute and the Norwegian Institute for Air Research (NILU). Funding was provided by the Norwegian Pollution Control Authority (SFT). The present study is part of an international collaboratory project examining the effects of persistent organic pollutants (POPs) in northern fulmars across Arctic regions. The international collaboratory project will assess the toxicological potential of measured POPs by examining the correlative relationships between levels of POPs and biomarkers (thyroid hormones, retinoids and 7-ethoxyresorufin-deethylase) in four different populations of northern fulmar in Canada (two colonies), Faeroe Islands and Norway.

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2. Contents

3. Summary

The aim of the present project was to determine a wide range of new and established persistent organic pollutants in northern fulmar (*Fulmarus glacialis*) breeding on Bjørnøya. All analyses were conducted on liver samples.

Legacy organochlorines (OCs)

Concentrations of ∑ polychlorinated biphenyl (PCB) in the northern fulmars of the present study were in the same range as previous reported ∑ PCB concentrations (egg, fat and liver samples) of northern fulmar from Jan Mayen and Canada. The ∑ PCB concentrations did not exceed any of the reproductive effect levels calculated for ∑ PCB exposure in bird eggs.

Liver concentrations of non-*ortho* PCB were two times higher than previous reports in northern fulmars from Canada and 7 – 11 times higher than reported levels in Brünnich's guillemot and black-legged kittiwake from Canada. Nevertheless, the liver concentrations in the present study were only 46 - 60 % of reported plasma levels of non-*ortho* PCBs in glaucous gull from Bjørnøya (lipid normalized).

Liver concentrations of ∑ polychlorinated dibenzo-p-dioxin/furan (PCDD/F) were 12 - 24 times higher than previous reports in black-legged kittiwake and Brünnich's guillemot from Canada, but comparable to levels reported in northern fulmars from Canada.

The highest toxic equivalents ∑ TEQ concentrations in the present study (range concentrations) were above the threshold liver levels for cytochrome P450 1A induction in terns. Furthermore, calculated TEQ concentrations of the present study exceeded threshold levels for various biological effects in bird eggs.

Levels of ∑ chlorbenzene (CBz) corresponded to levels observed in several species of birds. Furthermore, hexachlorbenzene (HCB) concentrations were well below the no-observed-effect-levels reported in kestrels.

∑ Chlordane (CHL) concentrations in the present study were 5 - 21 times higher than levels observed in little auk, Brünnich's guillemot, black guillemot and black-legged kittiwake. It has been suggested that northern fulmars may depart from other seabirds with respect to chlordane metabolism. In concert with this, the chlordane metabolite, oxychlordane, accounted for 83 % of ∑ CHL concentrations. Adult survival have been shown to be negatively related to oxychlordane in studies conducted on glaucous gull.

Relatively high levels of mirex and dieldrin were reported in the present study. Furthermore, reported levels of aldrin, isodrin, endrin and trifluralin were very low or non-detected.

∑ Dichlorodiphenyltrichloroethane (DDT) levels in the present study were only 8 - 13 % of plasma levels in glaucous gull from Bjørnøya, but comparable to levels reported in black-legged kittiwake.

The ∑ hexachlorocyclohexanes (HCH) concentrations in the northern fulmar of the present study were lower than reported concentrations of ∑ HCH in other species of seabirds.

Other organochlorines

Endosulfan levels in the present study were low (only detected in two individuals).

Concentrations of toxaphene in the present study were higher than current levels in glaucous gull plasma and polar bear fat (50 % and 0.03 % of the levels in the present study). The toxaphene concentrations in the present study were, however, lower than egg concentrations reported in black-backed gull and herring gull.

Brominated flameretardants

Concentrations of tribromoanisole (TBA), hexabromocyclododecane (HBCDD) and polybrominated biphenyls (PBB) in the present study were low.

Polybrominated diphenyl ethers (PBDE) were only detected in one individual of the present study. Only deca brominated diphenyl ether (BDE 209) was found in this bird and the concentrations were very high. Interestingly, this individual also carried high loads of non-*ortho* PCBs and PCDD/Fs. The northern fulmars special habitat and food specialization may explain the observed large differences in concentration from this individual and the other birds sampled. The northern fulmars covering distances that take them to the Kola peninsula and Finnmark, where they are exposed to local contamination. This may cause large differences in individual pollution load. Together, high levels of non-*ortho* PCB, PCDD, PCDF and BDE 209 may cause adverse biological effects in individuals of northern fulmars expressing unusually high levels of these compounds.

Perfluorinated alkyl substances (PFAS)

Of the 9 analysed PFAS only perfluorohexanoic acid (PFHxA), perfluorooctane sulfonate (PFOS) and perfluorooctane sulfonamide (PFOSA) were detected in the present study. Relatively low concentrations of PFHxS were found in all individuals.

The levels of PFOS in the present study were 0.2 – 28 % of the liver levels reported in seabirds from more polluted regions (Italy, USA and Korea), and only 2 % of the current plasma concentrations reported in glaucous gulls from Bjørnøya. Furthermore, liver concentrations of fur seal and polar bears from Alaska were 2.9 – 40 and 51 - 200 times higher, respectively, than the reported levels in the present study.

PFOSA was detected in 10 out of 15 birds, and the levels were low but comparable to reported liver concentrations in polar bears from Svalbard and Canada. Interestingly, PFOSA was not detected in a recent study on blood contaminants in glaucous gull from Bjørnøya.

There are few effect studies of PFAS exposure. However, data indicate that PFOS concentrations in wildlife are less than those required to cause adverse health effects in laboratory animals. The PFOS concentrations in the present study are well below those causing second generation effects in rats.

Mercury

Compared to other Arctic seabirds mercury levels in the present study were relatively high. Despite this, mercury levels do not exceed lethal threshold concentrations calculated for birds.

4. Sammendrag (Summary in Norwegian)

Formålet med denne studien var å kartlegge en rekke nye og etablerte organiske miljøgifter i hekkende havhest *(Fulmarus glacialis*) fra Bjørnøya. Alle analyser ble foretatt på leverprøver.

Rutinemessig analyserte organokloriner (OCs)

Konsentrasjoner av ∑ polyklorerte bifenyler (PCB) i havhest i denne studien tilsvarte tidligere rapporterte ∑ PCB-verdier (egg, fett og lever) i havhest fra Jan Mayen og Canada. Konsentrasjonen av ∑ PCB oversteg ikke de kalkulerte terskelverdier for reproduktive effekter kalkulert for fugleegg.

Leverkonsentrasjoner av non-*ortho* PCB var to ganger høyere enn tidligere rapporterte verdier i havhest fra Canada, og 7 – 11 ganger høyere enn rapporterte verdier i polarlomvi og krykkje fra Canada. De rapporterte verdier er derimot bare 46 - 50 % av verdier funnet i polarmåke fra Bjørnøya.

Leverkonsentrasjoner av ∑ polyklorerte dibenzo-p-dioksin/furan (PCDD/F) var 12 – 24 ganger høyere enn tidligere rapporterte verdier i krykkje og polarlomvi fra Canada. Nivåene i denne studien var tilsvarende rapporterte verdier på havhest i Canada.

De høyeste ∑ toksiske ekvivalenter (TEQ)-verdier i denne studien (maksimum-verdiene) var høyere enn leverterskelnivåer for cytochrome P450 1 A induksjon i terner. Videre var TEQ-verdiene i denne studien høyere enn kalkulerte terskelverdier for biologiske effekter kalkulert for fugleegg.

Verdier av ∑ klorbenzen tilsvarte rapporterte nivåer i andre fuglearter. Videre var heksaklorbenzenkonsentrasjoner i dette studiet lavere enn terskelverdiene for de såkalte "ingen-observert-skadelige-effekter-nivå" rapportert for fugl.

Konsentrasjon av ∑ klordan i denne studien var 5 – 21 ganger høyere enn observerte nivåer i alkekonge, polarlomvi, teist og krykkje. Tidligere studier har foreslått at havhest har klordan-metabolisme forskjellig fra andre sjøfugler. I overensstemmelse med dette svarte oxyklordan til 83 % av ∑ klordan konsentrasjonene. Oxyklordan har vist seg å korrelere negativt med overlevelse i voksne polarmåker.

I denne studien ble relativt høye nivåer av mirex og dieldrin rapportert. Videre var konsentrasjoner av aldrin, isodrin, endrin og trifluralin lave og under deteksjons-grensen.

Nivåer av ∑ diklordifenyltrikloretan (DDT) tilsvarte 8 – 13 % av rapporterte plasma-verdier (på lipidvektsbasis) i polarmåke fra Bjørnøya. Nivåene var imidlertid sammenlignbare med tidligere rapporterte verdier i krykkje.

Konsentrasjoner av heksaklorsykloheksan (HCH) i havhest fra denne studien var lavere enn rapporterte HCH-konsentrasjoner i andre fuglearter.

Andre organokloriner

Lave nivåer av endosulfan ble rapportert i denne studien (over deteksjonsgrensen i to individer).

Konsentrasjoner av toksafen i havhest fra denne studien var høyere enn rapporterte nivåer fra polarmåke (plasma) og isbjørn (fett) (50 % og 0.03 % av nivåene rapportert i dette studiet). Toksafen-konsentrasjoner i egg fra svartbak og sildemåke var derimot høyere.

Bromerte flammehemmere

Konsentrasjoner av tribromanisol (TBA), HBCDD og polybromerte bifenyler (PBB) i denne studien var lave.

Polybromerte difenyl-etere (PBDE) ble ikke detektert i noen av individene i denne studien med unntak av ett individ. Høye konsentrasjoner av dekabromdifenyleter (BDE 209) ble detektert. Individet med høye konsentrasjoner av BDE 209 hadde også høye verdier av non-*ortho* PCBer og PCDD/Fer. Havhestens spesielle habitat og matstrategi kan muligens forklare de observerte individuelle forskjellene. Havhesten kan bli utsatt for lokal forurensning fra områder langt fra Bjørnøya, slik som Kola-halvøya og Finnmarkskysten. Denne lokale forurensningen kan medføre store individuelle forskjeller med hensyn til miljøgiftsbelastning. Svært høye nivåer av non-*ortho* PCBer, PCDD, PCDF og BDE 209 kan medføre alvorlige biologiske effekter.

Perfluoralkylstoffer (PFAS)

I denne studien ble bare perfluorheksan syre (PFHxA), perfluoroktanyl-sulfonat (PFOS) og perfluoroktan-sulfonamide (PFOSA) påvist. Relativt lave verdier for PFHxS ble rapportert for alle individer.

Nivåene av PFOS var 0.2 – 28 % av rapporterte leverkonsentrasjoner i sjøfugl fra mer forurensede miljøer (Italia, USA og Korea), og bare 2 % av plasmakonsentrasjoner nylig rapportert i polarmåke fra Bjørnøya. Rapporterte verdier for pelssel og isbjørn fra Alaska var 2.9 – 40 og 51 – 200 ganger høyere enn PFOS-konsentrasjoner rapportert i denne studien.

I 10 av 15 havhest ble PFOSA detektert. Nivåene var sammenlignbare med rapporterte konsentrasjoner i isbjørn fra Svalbard og Canada. I en nylig studie gjort på polarmåke fra Bjørnøya ble PFOSA ikke detektert i et eneste individ.

Det eksisterer få studier på effekter av PFAS-eksponering. Det er imidlertid foreslått at PFOS-konsentrasjoner i fritt levende dyr ikke er høye nok til å skape alvorlige helseeffekter. Rapporterte PFOS-konsentrasjoner i denne studien er adskillig lavere enn estimerte terskelverdier for andregenerasjonseffekter i rotte.

Kvikksølv

Kvikksølvnivåene i denne studien er relativt høye sammenlignet med andre sjøfugl. Til tross for høye kvikksølv nivå, oversteg ikke nivå av kvikksølv de kalkulerte terskelverdier for overlevelse i fugl.

5. Rationale

Even though the industry in the Arctic is modest virtually all POPs detected at southern latitudes have been reported in the Arctic biota. Levels of persistent organic pollutants in seabirds are generally related to trophic level and migration pattern, with highest concentrations found in great skuas (*Stercorarius parasiticus*), great black-backed gull (*Larus marinus)* and glaucous gulls (*Larus hyperboreus)* (AMAP 2004). Due to restrictions in production and use of chemicals such as polychlorinated biphenyls (PCB) and dichlorodiphenyltrichloroethane (DDTs), levels start to decline in biota. However, there is evidence that new contaminants, i.e. perfluoralkylated substances (PFAS) and brominated flame retardants (BFR) are on the rise (AMAP 2004).

Bourne and Bogan (1972) reported that northern fulmar from Bjørnøya carried heavy loads of POPs. Since then there have been few studies describing POP concentrations in northern fulmars from the European Arctic. The northern fulmar is one of the most long-lived bird species (Hatch and Nettleship 1998) and it has been suggested that they may reach 50 years of age. Ringed birds have been reported still on their nest 39 years after the first observation (del Hoyo et al. 1992). Northern fulmars lay one egg only and they start to breed when they are 8-10 years old (Hatch and Nettleship 1998), typically on cliffs and rock faces (del Hoyo et al. 1992). The fulmars catch prey in the upper part of the water column such as fish, squid, copepods and amphipods, but fish offal and carrion is also included in their diet (del Hoyo et al. 1992; Hatch and Nettleship 1998). They are typical marine birds and spend most of their time at sea, retreating south as the sea freezes, but never to areas of warm water masses (del Hoyo et al. 1992). Because the northern fulmars do not migrate to more southern regions during the winter season this species is an interesting candidate for contaminant monitoring in the Arctic.

Fisk et al. (2001a) suggested that the northern fulmars had a different metabolism of chlordane components compared to other avian species. They reported that the relative proportions of chlordane components in seabirds were related to phylogeny. Furthermore, Fisk et al. (2001b) observed higher POP concentrations in northern fulmars than expected based on the POP trophic level relationship. This is in line with the findings of Braune and Simon (2003) of higher concentrations of dioxin, furans and TEQ (toxic equivalents) values in northern fulmars compared to species of the same trophic level, i.e. black-legged kittiwakes (*Rissa tridactyla)* and Brünnich's guillemots (*Uria lomvia*).

Relatively high concentrations of POPs have been reported in well studied species such as polar bears (*Ursus maritimus*) and glaucous gulls with indications of biological effects associated with these high levels of POPs (Sagerup et al. 2000; Lie et al. 2002; Bustnes et al. 2003 and Verreault et al. 2004a). Furthermore, studies of free-ranging birds have revealed changes in thyroid function, disruption of retinoid homeostasis and organochlorine (OC) induced cytochrome P450 activity (Moccia et al. 1986; Yamashita et al. 1992; review by Fox 1993). So far, no effect studies are reported in northern fulmars (AMAP 2004).

Northern fulmar is an interesting candidate for the study of POP levels in the Arctic. This is due to their suggested different metabolic abilities, the lack of data on POP levels and the fact that the northern fulmar is an all year resident to the northern regions. The main objective of this study was to determine a wide range of new and established organic pollutants in northern fulmars breeding on Bjørnøya. These current levels and congener patterns were compared to previous reported levels in northern fulmar and other Arctic seabirds.

6. Materials and methods

6.1. Sampling

A total of 15 breeding northern fulmars (six females and nine males) were captured in June-July 2003 from two colonies (Kapp Nilsson and Kapp Heer) on Bjørnøya (Figure 1). After capture the northern fulmars were killed and the liver collected from all individuals. The liver samples were stored at - 20°C or below during fieldwork, shipping and prior to the chemical analyses. Northern fulmars may become very old. Unfortunately, the birds sampled in the present study could not be aged since there are no good methods of age determination in birds (except ringing). However, since all birds were breeding they were all estimated to be older than eight years old.

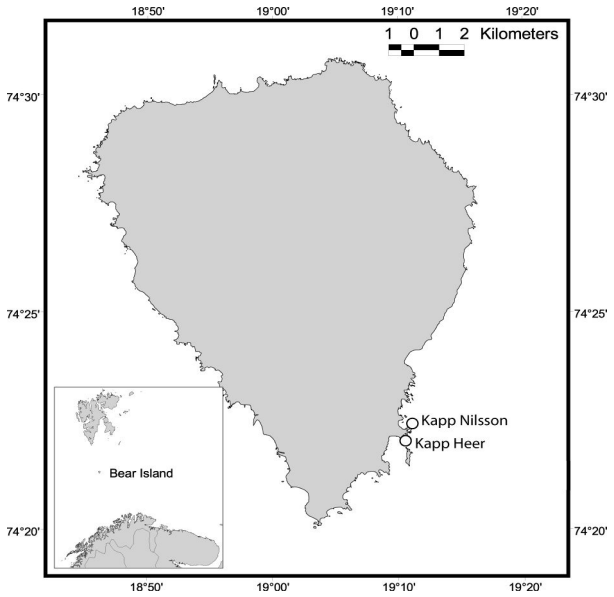

Figure 1. *Map of sampling colonies on Bjørnøya (Kapp Nilsson and Kapp Heer).*

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6.2. Chemical analyses

The chemical analyses were performed at the Norwegian Institute for Air Research at Kjeller, Norway. The homogenized samples were divided into four subsamples which were used for the chemical analyses of (1) organochlorines including BFR, (2) polychlorinated dibenzo-p-dioxins/furnas (PCDD/PCDF) and non-ortho PCB, (3) PFAS and (4) Mercury (Hg).

6.2.1. Extraction and cleanup of OCs and BFRs

A 'multi-analysis' method was used to analyse a large spectrum of OCs and BFRs as shown in Table 1.

The sample was homogenized with sodium sulphate. The mixture was filled into a glass column and an internal standard mixture containing ^{13}C-labeled was added on top of the column. The lipophilic compounds were eluted by a slow flow of cyclohexane. Lipids were removed by gel permeation chromtography (GPC) on a 50 g Biobeads SX-3 with cyclohexane/ethylacetate (50/50). The POP fraction was cleaned additionally by chromatography on silica gel. Before quantification 1,2,3,4-tetrachloronaphthalene was added as recovery standard.

6.2.2. Extraction and cleanup of PCDD/PCDF and non-*ortho* PCB

A sample of 5–20 g, spiked with ^{13}C-labelled 2,3,7,8-chloro substituted PCDD and PCDF congeners and 13C-labelled non-*ortho* PCB, was ground with dried sodium sulphate in a household mixer and placed on top of the first column for extraction and clean-up. The column was filled with 650 ml cyclohexane/dichloromethane (DCM) 1 +1. The sample was passed through column 1 (30 g of silica, and 30 g of KOH-coated silica, bottom to top) and column 2 (20 g KOH-coated silica and 30 g silica, bottom to top) and eluted into column 3 (activated charcoal (AX21) suspended on glass fibres). Undesired matrix compounds were removed from column 3 with 75 ml cyclohexane/ DCM 1+ 1 and 75 ml DCM. PCB, PCDD/PCDF and non-*ortho* PCB were eluted with 40 ml toluene in reverse flow direction. A final clean-up was performed on two small columns (Pasteur pipettes) filled with sulphuric acid coated silica and aluminium oxide. The dissolved sample was placed on the first column and PCDD/F and non-*ortho* PCB were eluted into the aluminium oxide layer with hexane. The second pipette was first eluted with 5 ml hexane/DCM 98+ 2 (discarded) and then with 5 ml hexane/DCM 1+ 1 (PCDD/F, non-*ortho* PCB). Just before quantification, the sample was spiked with a recovery control standard.

6.2.3. PFAS extraction and clean-up

The samples were homogenized in high-purity lab water (one part sample, five parts MilliQ-water; MilliQ, Millipore Corp.) using an Ultra-Turrax homogenizer. From this homogenate 1 g sample in a PP-centrifuge tube was spiked with 20 µl internal standard (corresponding to 20 ng/g liver) and 2.7 g 2 mM NH4OAc in methanol/water (1:1) was added. The mixture was extracted three times 10 min in ultrasonic bath. Before transferring to an autosampler vial the solution was filtrated through micron centrifuge filter (14000 rpm, 15-45 min).

6.2.4. Quantification of the organic compounds by GC/MS

The separation of all the organic compounds, with the exception of PFAS, was performed by high-resolution gas chromatography (HRGC) on a Hewlett-Packard HP 6890 with splitless injection of 1 µl aliquot of the sample extract and helium as a carrier gas. For detection and quantification the following mass spectrometers were used: a Hewlett-Packard HP5973MSD low resolution mass spectrometer running in the negative ion chemical ionisation mode (LRMS-NICI) and a Micromass AutoSpec (formerly VG Analytical AutoSpec) high resolution mass spectrometer (res> 10 000) running in the electron impact mode (HRMS-EI). For more details see Table 1.

6.2.5. Quantification of the organic compounds by LC/MS

An Agilent high performance liquid chromatography system (HPLC, 1100 series, Agilent, Palo Alto, CA) in combination with a time-of-flight high resolution mass spectrometer (LC-TOF: Waters-Micromass, Manchester, UK) was used for quantification of hexabromocyclododecane (HBCDD) and PFAS. The compounds of interest were transferred

into the TOF high-resolution mass spectrometer. HBCDD and PFAS were quantified with electrospray ionisation (ESI) in negative ion mode. For more details see Table 1.

Table 1. *List of analytes determined in liver tissue of northern fulmars captured on Bjørnøya and quantification method used. Methods used: gas chromatograph (GC)-high resolution-mass spectrometer (HRMS) in the electron chemical ionization mode (EI), GC-low resolution (LR)-MS in the negative ion chemical ionization mode (NICI) and liquid chromatograph (LC) combined with time of flight (TOF) MS. For a complete list of congeners analysed see Appendix A - C.*

SUBSTANCES	METHOD	GC or LC-column
Organochlorines (OCs)		
Legacy OCs		
Polychlorinated biphenyl (∑ PCB)	GC/HRMS-EI	SGE HT-8 (50m*0,22mm*0,15µm)
Non-ortho PCBs[1]	GC/HRMS-EI	Rtx2330 (30m*0,18mm*0,11µm)
Polychlorinated dibenzo-p-dioxins (PCDDs)	GC/HRMS-EI	Rtx2330 (30m*0,18mm*0,11µm)
Polychlorinated dibenzofurans (PCDFs)	GC/HRMS-EI	Rtx2330 (30m*0,18mm*0,11µm)
Hexachlorobenzene (HBC)	GC/HRMS-EI	SGE HT-8 (50m*0,22mm*0,15µm)
Chlorobenzene (CBz)	GC/HRMS-EI	SGE HT-8 (50m*0,22mm*0,15µm)
Chlordanes and metabolites (CHL)	GC/LRMS-NICI	Ultra-II (25m*0,2mm*0,11µm)
Mirex	GC/LRMS-NICI	Ultra-II (25m*0,2mm*0,11µm)
Dieldrin	GC/LRMS-NICI	Ultra-II (25m*0,2mm*0,11µm)
Aldrin	GC/LRMS-NICI	Ultra-II (25m*0,2mm*0,11µm)
Isodrin	GC/LRMS-NICI	Ultra-II (25m*0,2mm*0,11µm)
Endrin	GC/LRMS-NICI	Ultra-II (25m*0,2mm*0,11µm)
Trifluralin	GC/LRMS-NICI	Ultra-II (25m*0,2mm*0,11µm)
Dichlorodiphenyltrichloroethane (DDT) and metabolites	GC/HRMS-EI	HP-1 (25m*0,2mm*0,25µm)
Hexachlorocyclohexanes (HCH)	GC/HRMS-EI	HP-1 (25m*0,2mm*0,25µm)
Other OCs		
Endosulfans	GC/LRMS-NICI	Ultra-II (25m*0,2mm*0,11µm)
Polychlorobornanes and camphenes (Toxaphene)	GC/LRMS-NICI	Ultra-II (25m*0,2mm*0,11µm)
Brominated flame retardants (BFR) and tribromoanisole	GC/HRMS-EI	Ultra-II (25m*0,2mm*0,11µm)
Hexabromocyclododecane (HBCDD)	LC/MS-TOF	XTerra C_{18} RP (150mm*2,1mm 3,5 µm particle size)
Perfluorinated alkyl substances (PFAS)	LC/MS-TOF	ACE C_{18} RP (150mm*2,1mm 3 µm particle size)

6.2.6. Mercury (Hg) - sample preparation and extraction

For each fulmar sample a total of 0.5 g was weighed, and a mixture of supra pure HNO_3 and H_2O_2 added. Furthermore, the northern fulmar samples, reference material and two control samples were dissoluted under pressure at 180°C. All samples, reference material and control samples were thereafter diluted in acid rinsed test tubes.

6.2.7. Mercury (Hg) - quantification

A total of 25 ml of the extract from all samples were transferred to pre-rinsed (with BrCl) test tubes and diluted to 50 ml. All extracts were added BrCl in order to reduce Hg-compounds to water soluble compounds. These water soluble compounds were reduced further to Hg by $SnCl_2$. After reduction, Hg(0) were separated from the liquid phase in a phase separator. Furthermore, the sample were concentrated on a gold trap and Hg quantified by use of atomfluorescence spectrophotometer.

6.2.8. Quality control and measurement uncertainty

A rigorous quality control plan was applied based on the recommendations of the Arctic Monitoring and Assessment Programme (AMAP) and on the requirements in the European quality norm EN 17049. Particular focus was given to sample storage and transport with special equipment to maintain freezing conditions during storage and transport from the point of sampling to the laboratory. The use of isotopic labelled internal standards for quantification of POPs and the frequent control of complete method blank values insured a high quality of the analytical results. Blank values were not subtracted.

The methods used by NILU are routinely tested, i.e. by participation in intercalibration. Estimated uncertainty (95 % confidence interval) of the organic compounds with the exception of PBDE, HBCDD and PFAS was in the range of 20 % to 30 %. For polybrominated diphenylethers (PBDE) the estimated uncertainty was 30 to 40 %. The results for HBCDD and PFAS are to be regarded as semiquantitative. For Hg the measurement uncertainty is estimated to 10 %.

6.3. Statistics

Analyses of variance (ANOVA) was conducted to test for possible differences between male and female northern fulmars regarding POP concentrations (both lipid weight and wet weight) in liver tissues (Statistica 6.1 2003 for Windows ® (Stat soft Inc). No differences in male and female northern fulmar were found, and males and females were consequently treated as one homogenous group (except for lipid normalized mirex and HBCDD concentrations). Hence, lipid normalized concentrations of mirex and HBCDD were given for both male and female northern fulmar (Table 3). ANOVA was also conducted to test for difference in POP concentrations between the birds in the two colonies. Differences were only found for Hg. Hg concentrations were given separately for the two colonies (Table 2).

7. Results

The chemical analyses of liver from northern fulmar revealed the presence of legacy OCs and new contaminants such as BFR and PFAS (Table 2 and 3). The concentrations of individual congeners are listed in Appendix A - C. Statistical significant differences in POP levels between male and females northern fulmars were only found for lipid normalized mirex and HBCDD concentrations. Lipid normalized mirex and HBCDD concentrations were consequently given both for male and female northern fulmar (Table 3). Furthermore, statistical significant differences between colonies were calculated for mercury and given separately for the two colonies.

***Table 2**: Arithmetic means (only for samples above the detection limit) for sum (∑) concentrations of different analytes, with standard deviation (± SD) and ranges (min-max), in liver samples of northern fulmars from Bjørnøya. Concentrations in pg/g wet weight for PCDDs, PCDFs, non-ortho PCBs and TEQ concentrations (marked with *), in μg/g (=mg/kg) wet weight for mercury (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), B (BFR and tribromoanisole) and C (PFAS) for congeners constituting the different groups, the specific concentration of the congeners of each group, and concentration uncertainty due to deviations from NILU's quality control.*

Congeners	N	Mean ± SD ng/g ww	Min – Max ng/g ww
Lipid % blood	15	4.3 ± 1.2	2.6 – 7.8
Organochlorines (OCs)			
Legacy OCs			
∑ Polychlorinated biphenyl (∑ PCB) (32 congeners)[1]	15/15	282 ± 70.2	141 – 420
∑ Extended PCB (80 congeners)[2]	15/15	308 ± 76.5	156 – 457
∑ Non-ortho PCBs[3] pg/g ww*	15/15	2 270 ± 1565	832 – 7 209
∑ Polychlorinated dibenzo-p-dioxins (PCDDs)[4] pg/g ww*	15/15	162 ± 262	24.7 – 1 079
∑ Polychlorinated dibenzofurans (PCDFs)[5] pg/g ww*	15/15	317 ± 550	48.4 – 2 252
∑ $TEQ_{PCDD,PCDF,non\text{-}ortho\ PCBs}$ pg/g ww*	15/15	288 ± 341	81.0 – 1465
∑ $TEQ_{mono\text{-}ortho}$ pg/g ww*	15/15	9.1 ± 2.1	4.8 – 12.3
∑ Chlorobenzene (CBz)[6]	15/15	25.2 ± 5.2	14.2 – 33.7
∑ Chlordanes and metabolites (CHL)[7]	15/15	140 ± 43.9	65.7 – 197
Mirex	15/15	5.3 ± 2.3	2.0 – 10.7
Dieldrin	15/15	23.4 ± 11.4	6.4 – 49.7
Aldrin	1/15	0.12	–
Isodrin	0/15	–	–
Endrin	15/15	2.2 ± 1.3	0.8 – 6.0
Trifluralin	2/15	–	0.03 – 0.04
∑ Dichlorodiphenyltrichloroethane (DDT) and metabolites[8]	15/15	53.4 ± 20.5	37.0 – 113
∑ Hexachlorocyclohexanes (HCH)[9]	4/15	0.76 ± 0.25	0.5 – 1.0

Other OCs			
∑ Endosulfans[10]	2/15	–	0.01 – 0.02
∑ Polychlorobornanes and camphenes (Toxaphene)[11]	15/15	18.1 ± 11.2	6.5 – 53.9
Brominated flame retardants (BFR) and tribromoanisole			
Tribromoanisole (TBA)	7/15	0.02 ± > 0.01	0.02 – 0.03
∑ Hexabromocyclododecane (HBCDD)[12]	14/15	0.65 ± 0.7	0.2 – 2.22
∑ Brominated biphenyl (PBB)[13]	11/15	0.29 ± 0.09	0.21 – 0.44
∑ Polybrominated diphenyl ethers (PBDEs)[14]	1/15	206	–
Perfluorinated alkyl substances (PFAS)			
Perfluorohexanoic acid (PFHxA)	0/15	–	> 4.2
Perfluoroheptanoic acid (PFHpA)	0/15	–	> 3.1
Perfluorooctanoic acid (PFOA)	0/15	–	> 3.5
Perfluorononanoic acid (PFNA)	0/15	–	> 3.1
Perfluorobutane sulfonate (PFBS)	0/15	–	> 1.4
Perfluorohexane sulfonat (PFHxS)	15/15	1.0 ± 0.3	0.5 – 1.6
Perfluorooctane sulfonate (PFOS)	13/15	3.4 ± 2.2	0.8 – 8.3
Perfluorodecane sulfonate (PFDcS)	0/15	–	> 0.9
Perfluooctane sulfonamide (PFOSA)	10/15	2.5 ± 1.9	0.6 – 5.9
Mercury (Hg) µg/g ww**			
Mercury	15/15	3.0 ± 2.7	1.2 – 12.2
Mercury, Kapp Nilsson	5/15	4.9 ± 4.2	1.4 – 12.2
Mercury, Kapp Heer	10/15	2.1 ± 0.7	1.2 – 3.4

[1] ∑ PCB: sum of PCB no. IUPAC (international union of pure and applied chemistry): 18, 28, 31, 33, 37, 47, 52, 66, 74, 99, 101, 105, 114, 118, 122, 123, 128, 138, 141, 149, 153, 156, 157, 167, 170, 180, 183, 187, 189, 194, 206 and 209.
[2] ∑ Ext. PCB: extended sum of PCB no. IUPAC: 16, 17, 22, 24, 25, 26, 27, 32, 40, 41/71, 42, 44, 45, 49, 64, 70/76, 84/92, 85, 87, 97, 110, 130, 132, 133, 134/131, 136, 137, 144, 146, 151, 158, 171, 172, 174, 176, 177, 178, 179, 185, 190, 195, 196, 199, 200, 201, 202, 203 + 18, 28, 31, 33, 37, 47, 52, 66, 74, 99, 101, 105, 114, 118, 122, 123, 128, 138, 141, 149, 153, 156, 157, 167, 170, 180, 183, 187, 189, 194, 206 and 209.
[3] PCB no. IUPAC: 77, 81, 126 and 169.
[4] 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, 1,2,3,4,7,8-HxCDD, 1,2,3,6,7,8-HxCDD, 1,2,3,7,8,9-HxCDD, 1,2,3,4,6,7,8-HpCDD and OCDD.
[5] 2,3,7,8-TCDF, 1,2,3,7,8-PeCDF, 2,3,4,7,8-PeCDF, 1,2,3,4,7,8-HxCDF, 1,2,3,6,7,8-HxCDF, 1,2,3,7,8,9-HxCDF, 2,3,4,6,7,8-HxCDF, 1,2,3,4,6,7,8-HpCDF, 1,2,3,4,7,8,9-HpCDF and OCDF.
[6] Hexachlorbenzene (HCB) and pentachlorbenzene (PeCBz).
[7] Heptachlor, heptachlor-exo-epoxide, heptachlor-endo-epoxide, trans-chlordane, cis-chlordane, trans-nonachlor, cis-nonachlor, chlordene and oxychlordane.
[8] p,p'-DDT, o,p'-DDT, p,p'-DDE, o,p'-DDE, p,p'-DDD and o,p'-DDD.
[9] α-HCH, β-HCH and γ-HCH.
[10] Endosulfan-I, endosulfan-II and endosulfan sulphate.
[11] Parlar 26, 32, 50 and 62.
[12] α-HBCDD, β-HBCDD and γ-HBCDD.
[13] BB 15 and BB 153.
[14] BDE 28, 66, 49+71, 77, 85, 99, 100, 119, 138, 153, 154, 183, 196, 206 and 209.

***Table 3**: Arithmetic means (only for samples above the detection limit) for sum (∑) concentrations of different analytes, with standard deviation (± SD) and ranges (min-max), in liver samples of northern fulmars from Bjørnøya. Concentrations in pg/g lipid weight for PCDDs, PCDFs, non-ortho PCBs and TEQ concentrations (marked with *) and in ng/g lipid 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). List of congeners constituting the different groups see Table 2.*

Congeners	N	Mean ± SD ng/g lw	Min – Max ng/g lw
Organochlorines (OCs)			
Legacy OCs			
∑ Polychlorinated biphenyl (∑ PCB) (32 congeners)	15/15	6657 ± 1204	4873 – 9164
∑ Extended PCB (80 congeners)	15/15	7273 ± 1306	5264 – 10 013
∑ Non-ortho PCBs pg/g lw*	15/15	55 317 ± 40 189	19 066 – 183 905
∑ Polychlorinated dibenzo-p-dioxins (PCDDs) pg/g lw*	15/15	3972 ± 6692	664 – 27519
∑ Polychlorinated dibenzofurans (PCDFs) pg/g lw*	15/15	7793 ± 14 078	1192 – 57 448
∑ $TEQ_{PCDD,PCDF,non\text{-}ortho\ PCBs}$ pg/g lw*	15/15	7041 ± 8737	2123 – 37 384
∑ $TEQ_{mono\text{-}ortho}$ pg/g lw*	15/15	216 ± 42.8	154 - 302
∑ Chlorobenzene (CBz)	15/15	602 ± 117	423 – 802
∑ Chlordanes and metabolites (CHL)	15/15	3363 ± 1065	1425 – 5047
Mirex	15/15	124 ± 45.5	67 – 235
Mirex males	9/9	143 ± 46.0	90.9 – 235
Mirex females	6/6	94.8 ± 26.7	67.0 – 141
Dieldrin	15/15	563 ± 270	162 – 1218
Aldrin	1/15	3.4	–
Isodrin	0/15	–	–
Endrin	15/15	51.0 ± 17.4	20.7 – 79.6
Trifluralin	2/15	–	0.7 – 1.1
∑ Dichlorodiphenyltrichloroethane (DDT) and metabolites	15/15	1289 ± 524	867 – 2880
∑ Hexachlorocyclohexanes (HCH)	4/15	15.7 ± 4.9	12.9 – 23.0
Other OCs			
∑ Endosulfans	2/15	–	0.28 – 0.50
∑ Polychlorobornanes and camphenes (Toxaphene)	15/15	406 ± 137	167 – 688
Brominated flame retardants (BFR) and tribromoanisole			
Tribromoanisole (TBA)	7/15	0.6 ± 0.1	0.4 – 0.8
∑ Hexabromocyclododecane (HBCDD)	14/15	14.8 ± 14.9	3.8 – 61.6
∑ Hexabromocyclododecane (HBCDD) males	9/9	9.7 ± 5.0	3.8 – 18.6
∑ Hexabromocyclododecane (HBCDD) females	5/6	23.9 ± 22.6	7.96 – 61.6
∑ Brominated biphenyl (BB)	11/15	6.6 ± 1.8	4.2 – 10.5
∑ Polybrominated diphenyl ethers (PBDEs)	1/15	5255	–

8. Discussion

8.1. Organochlorines (OCs)

It has been suggested that OC body burden in avian species acquire equilibrium with OC levels in their diet. This conclusion is based on the fact that no significant fluctuations in mean residue levels of persistent organochlorines (e.g. PCB 153) in adult age classes have been observed (Elliot and Shutt 1993). In light of this, the levels of the more persistent OCs in the long-lived northern fulmars would be expected to obtain equilibrium with the dietary input and maintain an apparent steady-state level. The persistent and the non-persistent OC levels in northern fulmars of the present study were consequently expected to be independent of age (because all birds were older than eight years, i.e. breeders). A relatively low OC variance supports this hypothesis in the present study (except for the PCDD/F and non-*ortho* PCB concentrations, see explanation 8.1.1. PCDD/F and non-*ortho* PCB).

8.1.1. Legacy OCs

Sum PCB

$\sum PCB_{32}$ concentrations in the northern fulmars of the present study ranged from 4873 ng/g lw to 9164 ng/g lw, with a mean concentration of 6657 ng/g lw. This is in the same range as previously reported ∑ PCB concentrations (2372 ng/g lw to 14 292 ng/g lw) in egg, fat and liver samples of northern fulmar from Jan Mayen and Canada (Gabrielsen et al. 1997; Braune et al. 2001; Buckman et al. 2004). The PCB profile was dominated by PCB 153, 118 and 180. Together these congeners contributed to 60% of ∑ PCB. Compared to lipid normalized plasma levels of ∑ PCB in glaucous gull from Bjørnøya, liver ∑ PCB concentrations in the present study were low (only 13 % and 21 % of the levels in male and female glaucous gull, respectively) and showed less individual variance (Verreault et al. 2004b). In a study of Henriksen et al. (1998) POP concentrations in blood of glaucous gulls correlated well with body burden. Therefore lipid normalized blood concentrations in glaucous gull were expected to be representative as a measure of the body burden (equal to liver concentrations). ∑ PCB liver concentrations (lipid normalized) in the present study were comparable to liver concentrations of black-legged kittiwakes from Jan Mayen (72 % of the levels in black-legged kittiwakes), lower than liver (lw) levels reported in black-legged kittiwakes from the Barents Sea (37% of the levels in black-legged kittiwakes), but 3.7 times higher than liver concentrations found in Brünnich's guillemots from the Barents Sea (1782 ng/g lw and 6657 ng/g lw for Brünnich's guillemot and northern fulmars, respectively) (Gabrielsen et al. 1997; Borgå et al. 2001).

Most effect studies of Arctic wildlife have been carried out measuring the effect of PCB exposure. Both mean and range wet weight concentrations of $\sum PCB_{32}$ and $\sum PCB_{80}$ in the present study were well below the no-observed-effect level (NOEL) and lowest-observed-effect level (LOEL) for reproductive effects in eggs of fish-eating and predatory birds. The relatively low levels of PCBs in adult northern fulmar indicate that northern fulmar eggs probably do not contain elevated PCB levels, and that eggs most likely are well below thresholds levels for reproductive effects (AMAP 2004).

Non-ortho PCBs

Non-*ortho* PCB concentrations in the present study were two times higher than previous reported liver concentrations in northern fulmars from Canada on a wet weight basis (2270 pg/g ww and 1060 pg/g ww, for the northern fulmars in the present study and Canada, respectively) (Braune and Simon 2003). Furthermore, liver concentrations of ∑ non-*ortho*

PCBs in the present study were only 60 % and 45 % of the reported blood concentrations in male and female glaucous gull from Bjørnøya, respectively (92 ng/g lw and 121 ng/g lw for male and female glaucous gull) (Verreult et al. 2004b). On the other hand, they were much higher than liver ∑ non-*ortho* PCB concentrations (lipid normalized) reported in Brünnich's guillemot and black-legged kittiwake from Canada (11 and 7 times higher, respectively) (Braune and Simon 2003).

The temporal trend of non-*ortho* PCBs are confounding. Whereas, liver concentrations of non-*ortho* PCB in black-legged kittiwake and Brünnich's guillemot decreased from 1975 to 1993, there was a dramatic increase of non-*ortho* PCBs concentrations in northern fulmar liver samples during the same period. The increase in ∑ non-*ortho* PCB was primarily driven by increases in PCB 126 and, in particular, PCB 169 (Braune and Simon 2003). In the present study these two congeners were definitely the most important congeners constituting ∑ non-*ortho* PCB as well, making up 99 % of ∑ non-*ortho* PCB.

The dramatic increase in ∑ non-*ortho* PCB in northern fulmar from 1975 to 1993 could be explained by i) a delay in PCB arrival to the Arctic (because PCBs have been banned and a decrease in PCB concentrations have been observed at temperate latitudes) or ii) change in the northern fulmars migratory habits. If there were a delay in transport of PCBs into the Arctic, this should be reflected in the two other species examined, i.e. black-legged kittiwake and Brünnich's guillemot as well. However, the black-legged kittiwake is not as resident to the Arctic as the northern fulmar and could follow more southern temporal trends, i.e. decrease in non-*ortho* PCB (Braune and Simon 2003). Furthermore, stable isotope analyses ($\delta^{15}N$) disprove that the trend of a decrease/increase in contaminant concentrations in northern fulmar, black-legged kittiwake and Brünnich's guillemot eggs between 1975 and 1998 were the result of progressive shift in trophic level (Braune et al. 2001).

High levels of non-*ortho* PCB concentrations in the northern fulmar compared to the black-legged kittiwake and the Brünnich's guillemot, could be due to a lower metabolic capacity of these congeners in the northern fulmars compared to the black-legged kittiwake and the Brünnich's guillemot. This is difficult to infer without studying the biotransformation potential of the species. It is interesting though that the basic metabolic rate has been shown to be lower in Procellariiformes compared to Charadriiformes (Ellis and Gabrielsen 2001). In a Canadian study from 1975, lower liver concentrations of non-*ortho* PCBs were reported in northern fulmars compared to the black-legged kittiwake and Brünnich's guillemot. However, lower biotransformation potential in the northern fulmars may result in a greater bioaccumulation over time. This could explain the relatively low levels (compared to Brünnich's guillemot and black-legged kittiwake) of non-*ortho* PCB in 1975 compared to 1993 (Braune and Simon 2003).

The results from the seabird study of Braune and Simon (2003) point out the importance of species-specific differences with regards to spatial time trend analyses.

Polychlorinated dibenzo-p-dioxins/furans

∑ PCDD/F concentrations in the northern fulmar of the present study were 3972 pg/g lw and 7793 pg/g lw, respectively. This is 1.6 times higher and similar (ratio of 1) to reported liver concentrations in northern fulmars from Canada (Braune and Simon 2003). The lipid normalized levels of ∑ PCDD/F in the present study were 20 and 12 times higher, respectively, than reported liver levels in black-legged kittiwakes from Canada. Furthermore, the reported PCDD/F levels were 24 and 18 times higher than lipid normalized concentrations in Brünnich's guillemot from Canada (Braune and Simon 2003). Braune and Simon (2003) reported an decrease in PCDD/F liver concentrations in Canadian northern fulmars during the period 1975 to 1993.

Higher levels of PCDD/Fs in northern fulmars (in the present study and in the study by Braune and Simon 2003) compared to black-legged kittiwake and Brünnich's guillemot could be due to i) a much greater exposure to these compounds – possibly due to different migration pattern, or ii) because the northern fulmar has lower capacity to metabolize these compounds compared to the black-legged kittiwake and Brünnich's guillemot (Braune and Simon 2003).

Through time, omnivorous and herbivore species have evolved a more efficient biotransformation capacity than more specialized feeders (Walker 1990, Fossi et al. 1995). Thus there exists today a wide range of metabolic capacities in different species of birds. Furthermore, it has been suggested that fish-eating bird species have slow rates of metabolism which would allow enhanced bioaccumulation of certain PCBs, PCDDs and PCDFs (Walker 1990). There were observed relatively large standard deviations for the ∑ non-*ortho* PCBs and for the ∑ PCDD/F, compared to other compounds, suggesting a intraspecific variation in biotransformation potential of these congeners (Sanderson et al. 1998; Kennedy et al. 2003a). Again, it is difficult to conclude anything without examining the biotransformation potential of northern fulmars.

Dioxins and dioxin-like congeners (i.e. non-*ortho* 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). Induction of hepatic EROD activity by OC has been reported in several studies including free-ranging chicks and birds (Yamashita et al. 1992; Boseveld et al. 1995; Elliot et al. 1996). Ah-receptor mediated effects may account for biological and toxic effects. When applying toxic equivalent concentrations, the toxicity of a compound relative to dioxin is determined and threshold level of Ah-mediated biological effects can be calculated.

Toxic equivalency factors (TEQ)

When calculating toxic equivalent concentrations in the present study by using toxic equivalency factors (TEF) from World Health Organization (see Van den Berg et al. 1998), mean $TEQ_{PCDD/F,non\text{-}ortho PCB}$ concentrations of 288 ± 341 pg/g ww were obtained (range of 81 to 1465 pg/g ww). TEQ levels for mono-*ortho* PCBs were relatively low (9.1 ng/g ww) compared to $TEQ_{PCDD/F,non\text{-}ortho PCB}$ concentrations. ∑ $TEQ_{PCDD/F,non\text{-}ortho PCB}$ concentrations in the present study were comparable to liver TEQ concentrations calculated for Canadian northern fulmars (Bjørnøya/Canada: ratio of 0.9). However, these levels were higher than the reported lipid normalized liver levels in both black-legged kittiwake and Brünnich's guillemot from Canada (6 and 10 times higher, respectively) (Braune and Simon 2003). The mean ∑ TEQ values calculated in the present study are well below LOEL for liver CYP 1A induction in terns (*Sterna hirundo*) (25 000 ng/g lw). However, considering range TEQ concentrations (2123 to 37 384 pg/g lw), some individuals are above the liver TEQ level threshold for CYP 1A induction. Furthermore, ∑ TEQ wet weight concentrations exceed several threshold levels for biological effects in bird eggs (Table 4) (AMAP 2004). OC and TEQ levels are probably lower in eggs/embryos than in adult northern fulmars. Nevertheless, due to high TEQ levels observed in livers of adult northern fulmar, eggs from fulmars might be associated with TEQ levels above thresholds for effects. In concert with this, TEQ concentrations in Canadian northern fulmar eggs were above LOAEL for reproductive effects in wood duck (a sensitive species) (AMAP 2004).

It is obvious from Table 4 that differences between species of birds exist with regard to sensitivity of PCB, PCDD and PCDF exposure. Information on biological effects and threshold levels of biological effects after POP exposure for northern fulmar does not exist. Accordingly, it is difficult to know if the northern fulmars are as sensitive as the birds included in Table 4.

***Table 4**: Threshold TEQ egg concentrations calculated for various avian effects. NOAEL = no-observed-adverse-effect-level, LOAEL = lowest-observed-adverse-effect-level, NOEL = no-observed-effect-level, LOEL = lowest-observed-effect-level, LD_{50} = lethal dose that kills 50 % of the animals. The highest levels (range concentrations) reported in the northern fulmars exceed all threshold concentrations given in the table. Mean concentrations reported in the northern fulmars do not exceed the LD_{50} values, but they exceed all the other biological effects reported in the table.*

English species name	Latin species name	Threshold level of effect	Biological effect	Threshold concentrations
Double-crested cormorants	*Phalacrocorax auritis*	NOAEL	reproduction	4.6 pg/g ww
Herring gull	*Larus argentatus*	NOAEL	reproduction	10 pg/g ww
Wood-duck	*Aix sponsa*	LOAEL	reproduction	20 pg/g ww
Bald eagle	*Haliaeetus leucocephalus*	NOEL	CYP P450 1A induction	100 pg/g ww
Bald eagle	*Haliaeetus leucocephalus*	LOEL	CYP P450 1A induction	210 pg/g ww
Forster's tern	*Sterna forsteri*	NOAEL	reproduction	200 pg/g ww
Bald eagle	*Haliaeetus leucocephalus*	LOAEL	reproduction	200 pg/g ww
Caspian tern embryos	*Hydroprogne caspia*	LD_{50}	-	750 pg/g ww
Double-crested cormorants embryos	*Phalacrocorax auritis*	LD_{50}	-	550 pg/g ww

Chlorbenzene

∑ CBz concentrations in the present study were 602 ng/g lw. HCB accounted for 97 % of ∑CBz concentration (Appendix A.4.). The ∑ CBz concentrations were comparable to reported liver concentrations of ∑ CBz in northern fulmars from Canada (487 ng/g lw) (Buckman et al. 2004). Furthermore, the concentrations in the present study corresponded to reported liver concentrations in ivory gull (*Pagophila eburnean*) and glaucous gull from Canada (591 ng/g lw and 541 ng/g lw, respectively) (Buckman et al. 2004). However, the concentrations in the present study were 8.3, 2.1, 2.3 and 1.9 times higher than reported levels (on lipid weight basis) in little auk (*Alle alle)*, Brünnich's guillemot, black guillemot *(Cepphus grylle)* and black-legged kittiwakes (Buckman et al. 2004).

The levels of HCB in the present study were well below the NOEL reported in kestrels (*Falco tinnunculus*) (AMAP 2004).

Chlordanes

∑ CHL concentrations in the present study corresponded to liver concentrations reported in studies of northern fulmars from Canada and Jan Mayen (Gabrielsen et al. 1997; Buckman et al 2004). Furthermore, lipid normalized concentrations of ∑ CHL in the present study were higher than ∑ CHL liver concentrations in other seabirds, such as little auk, Brünnich's guillemot, black guillemot and black-legged kittiwake (13, 21, 4.6 and 5.3 times higher, respectively) (Buckman et al. 2004). The ∑ CHL concentrations were, however, comparable to plasma levels of ∑ CHL reported in glaucous gull from Bjørnøya (2809 and 2068 ng/g lw in males and females, respectively) (Verreault et al. 2004b). In a study of temporal trends of ∑ CHL in eggs of Brünnich's guillemot, northern fulmars and black-legged kittiwakes, ∑ CHL was found to be decreasing in black-legged kittiwakes, but not in Brünnich's guillemot and northern fulmar (Braune et al. 2001).

Oxychlordane in northern fulmar accounted for 83 % of ∑ CHL. Oxychlordane concentrations in the preset study were comparable to reported plasma levels in glaucous gull from Bjørnøya (2521 ng/g lw and 2630 ng/g lw for females and males, respectively) (Verrault et al. 2004a). Fisk et al. (2001a) reported that the northern fulmar had the highest percentage of oxychlordane relative to ∑ chlordane compared to little auk, Brünnich's guillemot, black guillemot, black-legged kittiwake, ivory gull and glaucous gull. They suggested that northern fulmars departed from other seabirds with respect to chlordane metabolism and that chlordane metabolism in seabirds was related to phylogeny.

A number of chlordane components and metabolites are toxic and carcinogenic and have been shown to decrease reproduction, induce cytochrome P450 2B and suppress the immune system (AMAP 2004). In long-lived birds, adult survival probability is the parameter to which the population growth rate is most sensitive. In a study of glaucous gull on Bjørnøya, adult survival was negatively related to blood concentrations of several OCs, and especially to oxychlordane. Survival probability was reduced by 29 % in females and 16 % in males as oxychlordane concentrations in the blood increased 10-fold (Bustnes et al. 2003). Northern fulmars in the present study might be affected by relatively high levels of oxychlordane. However, there were no indications of decreased adult survival in the colony on Bjørnøya. It is possible that the glaucous gull in the study of Bustnes et al. (2003) experienced decreased adult survival due to a combination of several factors. It might be that oxychlordane i) was correlating with other congeners that influenced adult survival, or ii) that oxychlordane together with very high levels of other OCs had strong synergistic effects on adult survival.

Mirex, dieldrin, aldrin, isodrin, endrin, trifluralin

Mirex concentrations in the present study (Tables 2 and 3) were comparable to lipid normalized concentrations in liver samples from Canadian northern fulmars (124 ng/g lw and 108 ng/g lw for the northern fulmars from Bjørnøya and Canada, respectively) (Buckman et al. 2004). Furthermore, lipid normalized mirex levels in the present study were 43 % lower than liver concentrations of mirex in northern fulmars from Jan Mayen sampled in 1995-1996 (Gabrielsen et al. 1997). Additionally, mirex concentrations in the present study were higher than current lipid normalized plasma concentrations in glaucous gull (5.4 and 5.6 times higher than levels reported in male and female glaucous gull, respectively), but comparable to liver concentrations reported in glaucous gull from Canada (168 ng/g lw) (Buckman et al. 2004; Verreult et al. 2004b). Furthermore, lipid normalized mirex concentrations in livers of little auks, Brünnich's guillemots, black guillemots and black-legged kittiwakes from Canada were 95 %, 91 %, 70 % and 40 % lower, respectively than reported levels in northern fulmars (Buckman et al. 2004). Chronic exposure to mirex may lead to body weight loss, various signs of liver toxicity, and induced monooxygeneases (AMAP 1998).

Dieldrin concentrations in the present study (Tables 2 and 3) were comparable to liver concentrations reported in northern fulmars from the Canadian Arctic (539 ng/g lw) (Buckman et al. 2004). Furthermore, dieldrin concentrations in the present study were relatively high compared to lipid normalized liver concentrations of little auks, Brünnich's guillemots, black guillemots and black-legged kittiwakes from the Canadian Arctic (5.1, 5.6, 2.4 and 1.8 times higher, respectively) (Buckman et al. 2004). Lipid normalized concentrations of dieldrin in glaucous gull from Bjørnøya (plasma), glaucous gull from Canada (liver) and ivory gull (liver) from Canada, were 63 – 68 % lower, 10 % higher and 53 % higher than reported concentrations in the present study, respectively (Buckman et al. 2004). Dieldrin has been reported to be toxic and effects such as disturbance in reproduction, effects on the immune system and increased cytochrome P450 activity have been reported in experimental animals dosed with dieldrin (AMAP 1998).

There are no studies available on aldrin, isodrin, endrin and trifluaralin levels in northern fulmars. Reported levels of aldrin, isodrin, endrin and trifluaralin in the present study were low, and non detect for isodrin and trifluralin (Tables 2 and 3). Chronic exposure to aldrin leads to signs of liver toxicity, however, aldrin is oxididized to dieldrin in plants and animals and is rarely found in samples from living organisms (AMAP 1998).

Dichlorodiphenyltrichloroethane (DDT) and metabolites

∑ DDT concentrations in the present study were comparable to liver concentrations of black-legged kittiwake (1471 and 1289 ng/g lw in kittiwakes and fulmars, respectively) from Svalbard, but they were only 8 % and 13 % of the reported lipid normalized blood levels in male and female glaucous gull from Bjørnøya (Borgå et al. 2001; Verreult et al. 2004b).

Liver concentrations of ∑ DDT in northern fulmars from Canada have been reported to be higher than concentrations of ∑ PCB (Buckman et al. 2004). In the present study ∑ DDT was only 19 % of ∑ PCB concentrations and considerably lower than reported liver concentrations in Canadian northern fulmars (Buckman et al. 2004). There have been reported a lack in definitive geographical differences in ∑ DDT concentrations between the central Barents Sea and the Northwater Polynya. Birds such as glaucous gull and little auk from the central Barents Sea contained higher ∑ DDT concentrations compared to the Northwater Polynia. ∑ DDT concentrations in Black guillemot from the Northwater Polynya were higher than the concentrations reported in the central Barents Sea, whereas no spatial differences were observed in Brünnich's guillemot and kittiwake (Borgå et al. 2004).

In a study by Braune et al. (2001) of temporal trends in Arctic seabird eggs ∑ DDT levels decreased from 1975 to 1998 in northern fulmar, black-legged kittiwake and Brünnich's guillemot. Highest DDT concentrations were reported for the northern fulmar.

In birds, DDT has been shown to affect reproduction, the immune system, the adrenal and thyroid hormones. For example p,p'-DDE has been shown to cause significant egg-shell thinning in DDE-treated birds, but also in wild birds significant correlations between DDE and eggshell thinning have been reported (AMAP 1998). The DDT levels in the present study were relatively low and biological effects were consequently not expected.

Hexachlorocyclohexanes (HCH)

Seabirds are apparently able to efficiently metabolise α and γ-HCH, but not β-HCH. In concert with this, only β-HCH were found in the present study. Liver HCH concentrations reported in the present study were relatively low compared to other seabirds, and only 14 % and 21 % of reported plasma HCH levels in male and female glaucous gull from Bjørnøya, respectively (AMAP 2004; Verreault et al. 2004b).

∑ HCH levels have been reported to be higher in seabirds from the Canadian Arctic compared to seabirds from the European Arctic (Borgå et al. 2004). Higher concentrations of HCH in the Canadian Arctic compared to the European Arctic biota are suggested to be caused by the closer proximity to Asia (were HCH has been used until recently) (AMAP 2004).

8.1.2. Other organochlorines (OCs)

Endosulfan

There are no studies available on endosulfan levels in northern fulmar (to our knowledge). Endosulfan was only detected in two individuals in very low concentrations. Interestingly, endosulfan was among the top five pesticides in air measurements from Arctic Canada. Furthermore, endosulfan is widespread in Arctic seawater, with no observed geographical trends (AMAP 2004). It is noteworthy that a 2.1 fold increase in age-adjusted mean concentrations of endosulfan in male belugas *(Delphinapterus leucas)* was observed

over a 15 year period (1982-1997) (AMAP 2004). The low detection of endosulfan in northern fulmar from Bjørnøya may be caused by i) low presence of endosulfan in the waters around Bjørnøya, or ii) that northern fulmars metabolize endosulfan more effectively than marine mammals.

Toxaphene

The northern fulmars from the present study had toxaphene concentrations in the range of 167 to 688 ng/g lw (mean concentrations of 406 ng/g lw). The four parlars (parlar 26, 32, 50 and 62) constituting ∑ toxaphene were contributing 35 %, 1%, 51% and 13%, respectively to ∑ toxaphene. There are few publications of toxaphene levels in Arctic seabirds. However, the levels of parlar 26, 32 and 50 in the present study were 15 % of liver wet weight concentrations in the 1995 samples (only three birds) of glaucous gull from Bjørnøya, but comparable to liver levels reported in glaucous gull sampled in 1999 (15 samples) (27.5 ng/g ww) (Herzke et al. 2003). Furthermore, current concentrations of parlar 26, 50 and 62 in glaucous gulls from Bjørnøya (plasma) and polar bear (fat) from Svalbard (excluded Bjørnøya) were lower (50 % and 0.03 % of the levels in the present study) than the reported levels in the present study (Verreault et al. 2004b; Gabrielsen et al. 2004). On the other hand, egg concentrations in great black-backed gull and herring gull were higher than reported levels in the present study (concentrations of 165 to 200 ng/g ww, lipid % concentrations were not reported, but in seabird eggs they are typically 10 to 15 %) (Geir Wing Gabrielsen, unpublished results). Yet, the results could be influenced by the different matrices used in the studies (liver versus blood, fat and eggs).

Toxaphene has been shown to retard growth and morphological development in laboratory rats, increase thyroid stimulating hormone and are a potent carcinogen. Unfortunately, no effect studies of toxaphene have been reported in Arctic animals. However, it has been suggested that toxaphene would not induce CYP P450 enzymes in free-ranging animals exposed to toxaphene at environmental levels (AMAP 2004). Due to very limited knowledge of biological effects of toxaphene in Arctic animals it is difficult to conclude if northern fulmars of the present study are susceptible to effects associated with toxaphene exposure. The levels reported are low, but higher than reported levels in species containing high concentrations of POPs, such as polar bear and glaucous gull. This might indicate that northern fulmars have a low ability to metabolize these compounds compared to polar bears and glaucous gulls. Alternatively these species may have a high metabolic capacity for these compounds.

8.2. Brominated flame retardants (BFR) and tribromoanisole

Tribromanisole, polybrominated biphenyls and hexabromocyclododecane

There are few reports on tribromoanisole (TBA), hexabromocyclododecane (HBCDD) and polybrominated biphenyls (PBB) in Arctic animals. The levels of TBA and PBB 153 reported in the present study are low (0.6 and 6.6 ng/g lw, respectively) and only detected in 7 and 11 out of 15 analysed samples, respectively. Compared to plasma concentrations of PBB 101 and PBB153/BDE 154 reported in glaucous gull from Bjørnøya, the PBB concentrations in the present study were low (13.2 to 16.2 and 121 ng/g lw (plasma) for PBB 101 and PBB 153/BDE 154 in glaucous gull) (Verreault et al. 2004b). HBCDD was detected with mean concentrations of 14.8 ng/g lw. The HBCDD levels in the present study were lower than the lipid normalized blood levels reported in glaucous gull from Bjørnøya (only 28 % - 40 of the HBCDD levels in glaucous gull) (Verreult et al. 2004b). However, the levels could be influenced by the different matrixes used (blood versus liver).

Polybrominated diphenyl ethers (PBDE)

∑ PBDE (of BDE 47 and 99) has been reported in liver samples of Brünnich's guillemot and black guillemot from Greenland (AMAP 2004). Furthermore, ∑ $PBDE_{28+47+99+100+154/bb153+153+138+183+209}$ plasma concentrations of 1596 and 1138 ng/g lw for were reported for male and female glaucous gull from Bjørnøya (Verreault et al. 2004b). In the present study only one congener of BDE was detected in one bird, i.e. BDE 209 (5255 ng/g lw). The BDE 209 concentrations in this bird were almost as high as ∑ PCB concentrations and the bird carried additionally very high loads of non-*ortho* PCBs, PCDD and PCDFs (183 905, 27 519, 57 448 pg/g lw, respectively). Other POP concentrations in this single bird seemed relatively normal. Habitat and food specialization may influence the observed large difference in the organic pollutant concentrations between individuals. Northern fulmars combine relatively short foraging trips with longer foraging trips lasting two to three days and travel as far away as 570 to 580 km from Bjørnøya (Weimerskirch et al. 2001). The birds might consequently be exposed to local contaminants (for example at the Kola peninsula or the coast of Finnmark), and this may cause the large differences in individual concentrations of some congeners. Other factors that may cause large differences in PBDE concentration between individuals could be scavenging on dead animals or feeding garbage.

In a study on glaucous gull from Bjørnøya by Herzke et al. (2003), elevated levels of BDE 47 and 99 were found in the intestinal contents compared to reported liver concentrations. Herzke et al. (2003) suggested a poor uptake of PBDE congeners through the gastro intestinal membranes (Herzke et al. 2003). They suggested that the size of the brominated molecules, as well as shape and polarity, were responsible for differences in congener specific accumulation. A poor uptake through the gastro intestinal membranes could cause the non-appearing levels of PBDEs in northern fulmars of the present study as well. However, this cannot be concluded without analysing the intestine content of the northern fulmars.

Higher levels of PBDEs reported in the glaucous gull compared to the northern fulmars could be due to several reasons: i) due to the different matrixes used for the analyses (blood versus liver), ii) due to difference in feeding ecology (glaucous gull is a typical predator and generalist), iii) due to differing metabolism and/or iii) due to their winter migration. Verreault et al. (2004b) suggested that the glaucous gull had a lower ability to metabolise PBDEs relative to e.g. marine mammals such as the polar bear. Furthermore, Henriksen et al. (2000) reported a low 7-ethoxyresorufin-*O*-deetthylase activity (enzyme activity in the liver) and suggested that this could indicate a limitations in the glaucous gull's metabolic capability, and eventually contribute to the high accumulation of OCs in the glaucous gull. It is possible that the northern fulmars of the present study were more effectively metabolising the PBDEs compared to the glaucous gull in the study of Verreult et al. (2004b). However, this is impossible to conclude without analysing metabolites of PBDEs. Due to little knowledge about the winter migration of the northern fulmars it is difficult to interpret if the winter migrating results in the observed difference in PBDE levels. Ringing evidence have suggested that northern fulmars do not truly migrate and that recoveries such as Norway to Faroes and Netherlands, represent pelagic dispersal (Hatch and Nettleship 1998). Most of the glaucous gull breeding in the Barents Sea region winter mainly in the northern part of the Atlantic Ocean (Anker-Nilssen et al. 2000).

Similar toxicity of PCB and PBDE are expected due to molecular analogies between these two compounds. Exposure to BDE 209 has been found to induce thyroid hyperplasia, heptacellular and thyroid adenomas and carcinomas in mice. Furthermore, BDE 47 has been shown to reduce T4 levels in rats. It has been suggested that this is a result of BDE metabolites competing for binding sites on the transthyrethin (TTR) (AMAP 2004). The

results of the present study demonstrate that northern fulmars from Bjørnøya generally contain low concentrations of PBDEs. However, some birds in the colony may become heavily exposed to specific congeners of PBDEs, i.e. BDE 209, possibly from local contamination far away from the breeding colony, from scavenging of dead animals or eating garbage. This suggests that high levels of PCDDs, PCDFs, non-*ortho* PCBs and BDE 209 might lead to adverse biological effects in some individuals with exceptionally high levels of these compounds.

8.3. Perfluorinated alkyl substances (PFAS)

Of nine analysed perfluorinated alkyl substances, only three were detected in the northern fulmars of the present study (Table 2). Perfluorohexane sulfonate (PFHxS) was reported in all individuals with mean concentrations of 1.0 ng/g ww. Reported levels of PFHxS in glacous gull blood were comparable on a wet weight basis (0.97 ng/g ww) (Verreault et al. 2004b). However, due to the special physical properties of PFAS, it is difficult to compare blood concentrations in glaucous gull with liver concentrations. Liver concentrations in glaucous gull would be expected to be higher than observed plasma levels.

Perfluorooctane sulfonate (PFOS) in the northern fulmars of the present study was detected in 13 out of the 15 analysed northern fulmars. The levels of PFOS were low (3.4 ng/g ww) and much lower than reported liver levels in seabirds from more polluted and industrialized regions, such as the USA, Italy and Korea (concentrations ranging from < 12 to 1780 ng/g ww) (Giesy and Kannan 2001; Kannan et al. 2001a). There are few publications on PFOS concentrations in Arctic marine animals. PFOS levels in the northern fulmars of the present study were only 2 % of current ww concentrations reported in plasma samples of glaucous gull from Bjørnøya (be aware of the different matrixes used, blood versus liver) (Verreult et al. 2004b). Kannan et al. (2001b) reported liver PFOS concentrations in northern fur seal (*Callorhinus ursinus*) and polar bear from Alaska in the range, < 10 – 122 ng/g ww and 175 to 678 ng/g ww, respectively. PFOS concentrations in the present study were lower (range concentrations in the present study of 0.7 – 8.3 ng/g ww). The results of the present study supports the suggested hypothesis of bioaccumulation of PFOS in the trophic marine food chain (Giesy and Kannan 2001).

Perfluoroctane sulfonamide (PFOSA) was detected in 10 out of 15 analysed northern fulmars (Table 2). PFOSA was not detected in plasma of glacous gull from Bjørnøya. The PFOSA levels in the present study were comparable to reported liver wet weight levels of PFOSA in polar bears from the North American Arctic and the European Arctic with range concentrations in the order non-detect to 8.5 ng/g ww (Smithwich et al. 2004).

There are few data on effects of PFAS in animals. The mechanism of toxicity of perfluoroalkylated substances are not well understood, but they are suggested to be peroxisome proliferators (Hekster et al. 2003). Data indicate that the concentrations of PFOS in wildlife are less than those required to cause adverse effects in laboratory animals (Giesy and Kannan 2001). In concert with this, the levels in the present study were well below the NOAEL (15 000 ng/g ww in liver) and LOAEL (58 000 ng/g ww in liver) for second-generation effects in rats (AMAP 2004).

8.4. Mercury (Hg)

Mercury was detected in all birds with mean liver concentrations of 3.1 ± 2.7 μg/g ww. However, there was a statistical significant difference in concentrations measured in the individuals from the two different colonies. Birds from Kapp Nilsson (N = 5) and Kapp Heer (N = 10) had mean liver concentrations of 4.9 ± 4.2 and 2.1 ± 0.7, respectively. This was higher than quantified liver levels of mercury in several species of Arctic seabirds, but much lower than reported liver levels in great cormorant (*Phalococorax carbo*) from Greenland (10.6 μg/g ww), except for one bird from the colony of Kapp Nilsson with mean levels of mercury of 12.2 μg/g ww (AMAP 1998). In glaucous gull the liver concentrations of mercury, on the other hand, were comparable to the levels found in the northern fulmars (AMAP 1998). The reason for a colony difference in levels of mercury could be explained by different age demography in the two colonies. Most studies do not report on age dependent accumulation of mercury. However, some studies have reported a age-dependent accumulation of mercury in birds (Wenzel and Gabrielsen 1995; AMAP 1998).

Recent studies have uncovered that the Arctic may become a global sink for atmospheric mercury during polar sunrise (gaseous elemental mercury is oxidized and converted to more reactive and short lived species of mercury, such as reactive gaseous mercury and particulate mercury). Data of atmospheric mercury levels at the Zeppelin Station for Atmospheric Monitoring and Research, Ny-Ålesund, confirm that the atmospheric mercury depletions/deposition episodes also are extended to the Svalbard area. Furthermore, an increase in concentrations of mercury was observed in surface snow from the polar night to the Arctic spring. This snow-bound mercury may end up in runoff when the snow melts in the spring (Berg and Aspmo 2003).

Birds assimilate organic forms of Hg more readily than inorganic compounds. Methylmercury is distributed evenly in tissues while inorganic forms accumulate primarily in liver and kidneys. Generally, organic Hg is more toxic than inorganic salts. Laboratory studies have identified a number of effects after Hg exposure such as: effects of enzyme systems, decreased cardiovascular function, changed blood parameters, effects of the immune responses and decreased kidney function and structure (AMAP 1998).

Threshold levels for lethal effects after mercury exposure in free ranging birds have been calculated. The birds of the present study are well below this level (> 30 μg/g = lethal threshold) (AMAP 1998). Nevertheless, malnutrition and mortality from chronic diseases were reported in great white herons if liver concentrations exceeded 6 μg/g ww (Spalding et al. 1994). Furthermore, embryo mortality and brain lesions with liver embryo concentrations have been observed with liver concentrations of about 11 μg/g and above, whereas concentrations of 1 to 2 mg/kg ww was associated with behavioural effects (Wolfe et al. 1998). Even though concentrations in adult northern fulmar of the present study were high, it is unknown if these high levels are expressed in the embryos as well. Furthermore, comparisons with threshold levels reported for various species of birds should be done with caution. There are large differences in sensitivity to pollutant exposure between avian species. In Procellariformes demethylation of organic Hg appears to be a significant detoxification strategy (Wolfe et al. 1998). The capability to demethylating the more toxic organic mercury have been suggested to be a way of coping with high levels of mercury. This may explain why some birds with high levels (much higher than the levels in the present study) of mercury show no detrimental effects (Muirhead and Furness 1988). Muirhead and Furness (1988) highlighted the danger of attributing high levels of toxic metals in seabirds to toxic effects and suggested that high levels of mercury in seabirds could be due to natural processes and result from accumulation and storage processes.

9. Conclusions

- ∑ PCB in northern fulmar did not exceed any of the threshold levels of biological effects calculated for ∑ PCB in bird eggs.

- Very high TEQ levels were reported in the present study.

- The highest ∑ TEQ concentrations in the present study (range concentrations) are above the threshold liver levels for CYP 1A induction in terns. Furthermore, the ∑ TEQ levels exceeded threshold levels of various biological effects calculated for bird eggs.

- High oxychlordane concentrations were found in the present study.

- The relative contribution of oxychlordane to ∑ chlordane concentrations were also high (83 % of ∑ chlordane), supporting the hypothesis of different (i.e. higher) chlordane metabolism in northern fulmars compared to other fish-eating birds.

- PBDEs were non-detect in the northern fulmars of the present study, with the exception of one bird. This bird had extremely high concentrations of BDE 209, whereas other PBDE congeners were non-detect.

- PHxS, PFOS and PFOSA were the only PFAS detected in the northern fulmars of the present study. Relatively low concentrations were reported.

- Mercury levels in the northern fulmars were high compared to other species of Arctic seabirds, but below reported lethal threshold levels in birds.

10. References

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11. Appendix

Arithmetic means (only for samples above detection limit) with standard deviation (+SD) and ranges (min-max) for the different analytes in liver samples of northern fulmars breeding on Bjørnøya. Concentrations in pg/g wet weight for PCDDs, PCDFs and non-ortho PCBs and in ng/g wet weight for all other compounds. The number of samples above the detection limit of the analysed POP is shown relative to the number of samples analysed (N). Furthermore, deviation of more than 20 % from theoretical value of isotopes = (i) is reported.

Appendix A. Legacy OCs

Appendix A.1. *Legacy OCs: Polychlorinated biphenyls (PCBs, CB) in ng/g wet weight*

Congeners	N	(I)	Liver samples Mean ± SD	Min – Max
Lipid %	15	–	4.3 ± 1.2	2.6 – 7.8
CB 18	0/15	–	–	–
CB 28	15/15	–	1.7± 0.5	0.6 – 2.6
CB 31	4/15	–	0.1 ± 0.02	0.08 – 0.13
CB 33	0/15	–	–	–
CB 37	5/15	–	0.04 ± 0.01	0.03 – 0.05
CB 47	15/15	–	0.7 ± 0.2	0.4 – 1.2
CB 52	0/15	–	–	–
CB 66	15/15	–	3.5 ± 0.97	2.2 – 5.1
CB 74	15/15	–	4.0 ± 0.9	2.6 – 5.5
CB 99	15/15	–	11.6 ± 2.8	6.9 – 17.3
CB 101	1/15	–	0.4	–
CB 105	15/15	–	10.2 ± 2.1	6.1 – 12.9
CB 114	15/15	–	0.8 ± 0.2	0.4 – 1.0
CB 122	5/15	(1)	0.02 ± 0.01	0.01 – 0.04
CB 118	15/15	–	34.9 ± 8.0	19.8 – 47.9
CB 123	15/15	–	0.5 ± 0.1	0.3 – 0.7
CB 141	12/15	–	0.1 ± 0.04	0.1 – 0.2
CB 149	12/15	–	0.3 ± 0.09	0.2 – 0.5
CB 153	15/15	–	89.4 ± 22.2	43.8 – 132
CB 138	15/15	–	34.9 ± 8.0	19.4 – 47.0
CB 167	15/15	–	3.8 ± 1.0	1.9 – 5.2
CB 128	15/15	–	7.3 ± 1.3	4.3 – 9.3
CB 156	15/15	–	6.6 ± 1.7	3.1 – 9.3
CB 157	15/15	–	1.5 ± 0.4	0.7 – 2.1
CB 170	15/15	–	13.3 ± 4.3	4.3 – 22.7
CB 180	15/15	–	42.7 ± 13.7	16.8 – 75.8
CB 183	15/15	–	4.9 ± 1.5	1.9 – 8.3
CB 187	15/15	–	0.3 ± 0.2	0.1 – 0.6
CB 189	15/15	–	0.7 ± 0.2	0.3 – 1.2
CB 194	15/15	–	5.7 ± 2.3	2.1 – 10.6
CB 206	15/15	–	1.6 ± 0.5	0.7 – 2.4
CB 209	15/15	–	1.5 ± 0.6	0.4 – 2.2
$\sum TEQ_{mono\text{-}ortho}$	15/15	–	9.1 ± 2.1	4.8 – 12.3

Appendix A.2. *Legacy OCs: Non-ortho polychlorinated biphenyls (PCBs) in pg/g wet weight*

Congeners	N	(I)	Liver samples Mean ± SD	Min – Max
Lipid %	15	–	4.3 ± 1.2	2.6 – 7.8
PCB-77	15/15	–	15.4 ± 7.2	5.0 – 30.8
PCB-81	15/15	–	14.2 ± 3.5	8.25 – 21.8
PCB-126	15/15	–	1191 ± 607	508 – 2831
PCB-169	15/15	–	1049 ± 985	290 – 4356
$TEQ_{non\text{-}ortho\ PCBs}$	15/15		130 ± 70.0	53.7 – 327

Appendix A.3. *Legacy OCs: Polychlorinated dibenzo-p-dioxins (PCDDs, CDD) and polychlorinated dibenzofurans (PCDFs) in pg/g wet weight*

Congeners	N	(I)	Liver samples Mean ± SD	Min – Max
Lipid %	15	–	4.3 ± 1.2	2.6 – 7.8
2,3,7,8-TCDD	15/15	(1)	1.7 ± 0.38	0.9 – 2.3
1,2,3,7,8-PeCDD 3	15/15	(1)	21.4 ± 34.1	3.4 – 140
1,2,3,4,7,8-HxCDD	14/15	(4)	15.2 ± 33.1	0.9 – 128
1,2,3,6,7,8-HxCDD	15/15	–	112 ± 180	15.0 – 744
1,2,3,7,8,9-HxCDD	15/15	(3)	3.0 ± 3.4	0.5 – 14.3
1,2,3,4,6,7,8-HpCDD	15/15	–	8.8 ± 11.5	1.3 – 47.5
OCDD	14/15	(3)	1.5 ± 2.2	0.15 – 8.1
TEQ_{PCDD}	15/15	–	36.0 ± 55.8	7.4 – 231
2,3,7,8-TCDF	15/15	–	0.9 ± 0.4	0.3 – 1.6
1,2,3,7,8-PeCDF	14/15	(3)	0.5 ± 0.1	0.3 – 0.7
2,3,4,7,8-PeCDF	15/15	–	228 ± 421	30.4 – 1712
1,2,3,4,7,8-HxCDF	15/15	–	30.0 ± 51.7	4.0 – 212
1,2,3,6,7,8-HxCDF	15/15	–	32.6 ± 46.4	4.5 – 193
1,2,3,7,8,9-HxCDF	4/15	(3)	0.2 ± 0.08	0.1 – 0.3
2,3,4,6,7,8-HxCDF	15/15	–	20.4 ± 26.2	3.6 – 110
1,2,3,4,6,7,8-HpCDF	15/15	(1)	4.6 ± 4.8	1.3 – 20.4
1,2,3,4,7,8,9-HpCDF	14/15	(2)	0.6 ± 0.5	0.1 – 2.0
OCDF	11/15	(4)	0.4 ± 0.4	0.07 – 1.2
TEQ_{PCDF}	15/15	–	122 ± 223	17.4 – 908

Appendix A.4. *Legacy OCs: Chlorbenzenes: hexachlorbenzene (HCB) and pentachlorbenzene (PeCBz) in ng/g wet weight*

Congeners	N	(I)	Liver samples Mean ± SD	Min – Max
Lipid %	15	–	4.3 ± 1.2	2.6 – 7.8
HCB	15/15	–	24.7 ± 5.0	14.2 – 33.7
PeCBz	8/15	–	0.8 ± 0.1	0.5 – 0.9

Appendix A.5. *Legacy OCs: Chlordanes (CHL) and metabolites: Heptachlor, heptachlor-exo-epoxide, heptachlor-endo-epoxide, trans-chlordane, cis-chlordane, trans-nonachlor, cis-nonachlor, chlordene and oxychlordane in ng/g wet weight*

Congeners	N	(I)	Liver samples Mean ± SD	Min – Max
Lipid %	15	–	4.3 ± 1.2	2.6 – 7.8
Heptachlor	0/15	–	–	–
Heptachlor-exo-epoxide	15/15	(15)	21.0 ± 7.8	8.1 – 32.5
Heptachlor-endo-epoxide	0/15	–	–	–
Trans-chlordane	0/15	–	–	–
Cis-chlordane	0/15	–	–	–
Trans-nonachlor	14/15	–	3.5 ± 2.3	1.6 – 11.0
Cis-nonachlor	11/15	–	0.2 ± 0.16	0.08 – 0.6
Chlordene	6/15	(6)	0.07 ± 0.06	0.03 – 0.2
Oxychlordane	15/15	–	116 ± 35.9	54.5 – 169

Appendix A.6. *Legacy OCs: Mirex, dieldrin, aldrin, isodrin, endrin and trifluralin in ng/g wet weight*

Congeners	N	(I)	Liver samples Mean ± SD	Min – Max
Lipid %	15	–	4.3 ± 1.2	2.6 – 7.8
Mirex	15/15	–	5.3 ± 2.3	2.0 – 10.7
Dieldrin	15/15	(1)	23.4 ± 11.4	6.4 – 49.7
Aldrin	1/15	–	0.12	–
Isodrin	0/15	–	–	–
Endrin	15/15	(15)	2.2 ± 1.3	0.8 – 6.0
Trifluralin	2/15	(1)	–	0.03 – 0.04

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

Congeners	N	(I)	Liver samples Mean ± SD	Min – Max
Lipid %	15	–	4.3 ± 1.2	2.6 – 7.8
α-HCH	0/15	–	–	–
β-HCH	4/15	–	0.76 ± 0.3	0.5 – 1.0
γ-HCH	0/15	–	–	–

Appendix A.8. *Legacy OCs: Dichlorodiphenyltrichloroethane (DDT, ortho (o) and para (p) and metabolites (DDE and DDD) in ng/g wet weight*

Congeners	N	(I)	Liver samples Mean ± SD	Min – Max
Lipid %	15	–	4.3 ± 1.2	2.6 – 7.8
o,p'-DDE	6/15	(4)	0.02 ± > 0.01	0.01 – 0.02
p,p'-DDE	15/15	(11)	49.8 ± 19.7	34.7 – 109
o,p'-DDD	9/15	(2)	0.01 ± > 0.01	0.01 – 0.02
p,p'-DDD	15/15	–	1.2 ± 0.3	0.7 – 1.6
o,p'-DDT	13/15	(13)	0.03 ± 0.01	0.02 – 0.07
p,p'-DDT	15/15	–	2.5 ± 1.4	1.1 – 6.6

Appendix A.9. *Other OC: Endosulfans: endosulfan-I, endosulfan-II and endosulfan sulphate in ng/g wet weight*

Congeners	N	(I)	Liver samples Mean ± SD	Min – Max
Lipid %	15	–	4.3 ± 1.2	2.6 – 7.8
Endosulfan-I	2/15	–	–	0.01 – 0.02
Endosulfan-II	0/15	–	–	–
Endosulfan sulphate	0/15	–	–	–

Appendix A.10. *Other OC: Toxaphenes: Parlar 26, 32, 50 and 62 in ng/g wet weight*

Congeners	N	(I)	Liver samples Mean ± SD	Min – Max
Lipid %	15	–	4.3 ± 1.2	2.6 – 7.8
Parlar 26	15/15	–	6.4 ± 3.4	2.3 – 16.7
Parlar 32	3/15	(3)	0.2 ± 0.06	0.2 – 0.3
Parlar 50	15/15	–	9.4 ± 6.3	2.9 – 30.1
Parlar 62	14/15	(5)	2.4 ± 1.6	1.1 – 6.9

Appendix B. Brominated flame retardants (BFR) and tribromoanisole

Appendix B.1. *BFR: Polybrominated diphenylethers (PBDEs, BDE), Brominated biphenyls (BB0), tribromanisol (TBA) and hexabromocyclodecane (HBCDD) in ng/g wet weight*

Congeners	N	(I)	Liver samples Mean ± SD	Min – Max
Lipid %	15	–	4.3 ± 1.2	2.6 – 7.8
TBA	7/15	(5)	0.02 ± > 0.01	0.02 – 0.03
α-HBCDD	14/15	–	0.5 ± 0.5	0.2 – 2.22
β-HBCDD	2/15	–	–	0.1 – 0.4
γ-HBCDD	3/15	–	–	0.2 – 1.1
BB 15	0/15	–	–	–
BB 153	11/15	–	0.29 ± 0.09	0.21 – 0.44
BDE 28	0/15	–	–	–
BDE 47	0/15	–	–	–
BDE 66	0/15	–	–	–
BDE 49 + 71	0/15	–	–	–
BDE 77	0/15	–	–	–
BDE 85	0/15	–	–	–
BDE 99	0/15	–	–	–
BDE 100	0/15	–	–	–
BDE 119	0/15	–	–	–
BDE 138	0/15	–	–	–
BDE 153	0/15	–	–	–
BDE 154	0/15	–	–	–
BDE 183	0/15	–	–	–
BDE 196	0/15	–	–	–
BDE 206	0/15	–	–	–
BDE 209	1/15	–	206	–

Appendix C. Perfluorinated alkylated substances (PFAS)

Appendix C.1. *Perfluorohexanoic acid (PFHxA), perfluoroheptanoic acid (PFHpA), perfluorooctanoic acid (PFOA), perfluorononanoic acid (PFNA), perfluorobutane sulfonate (PFBS), perfluorohexane sulfonat (PFHxS), perfluorooctane sulfonate(PFOS), perfluorodecane sulfonate (PFDcS) and perfluooctane sulfonamide (PFOSA) in ng/g wet weight*

		Liver samples	
Congeners	**N**	**Mean ± SD**	**Min – Max**
PFHxA	0/15	–	> 4.2
PFHpA	0/15	–	> 3.1
PFOA	0/15	–	> 3.5
PFNA	0/15	–	> 3.1
PFBS	0/15	–	> 1.4
PFHxS	15/15	1.0 ± 0.3	0.5 – 1.6
PFOS	13/15	3.4 ± 2.2	0.8 – 8.3
PFDcS	0/15	–	> 0.9
PFOSA	10/15	2.5 ± 1.9	0.6 – 5.9

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Tittel - norsk og engelsk Kartlegging av organiske miljøgifter i havhest (*Fulmarus glacialis*) fra Bjørnøya Organic pollutants in northern fulmars (*Fulmarus glacialis*) from Bjørnøya
Sammendrag – summary In the present study a wide range of persistent organic pollutants were determined in northern fulmar livers. Current levels and congener patterns were examined in northern fulmar livers and compared to previous reports on contaminant levels in northern fulmar and other seabirds of interest. There are few data on contaminant levels in northern fulmars from the European Arctic, and the effects associated with contaminant exposure have never been assessed in the northern fulmar. The data obtained from the present study will be included in a circumpolar study (Canada, Faroe Islands and Bjørnøya) assessing the effects on biomarkers such as plasma thyroid hormones, plasma and liver retinoid concentrations and hepatic enzyme activity (7-ethoxyresorufin-*O*-deethylase, EROD) with increasing levels of organic pollutants.

4 emneord Havhest, Arktis, nye miljøgifter, Svalbard	4 subject words Northern fulmar, Arctic, new contaminants, Svalbard