**Persistent organic pollutants and mercury in dead and dying glaucous gulls (Larus hyperboreus) at Bjørnøya (Svalbard)**

Kjetil Sagerup [a,*], Lisa B. Helgason [b], Anuschka Polder [c], Hallvard Strøm [b], Terje D. Josefsen [d], Janneche U. Skåre [c,e], Geir W. Gabrielsen [b]

[a] Tromsø University Museum, NO-9037 Tromsø, Norway

[b] Norwegian Polar Institute, The Polar Environmental Centre, NO-9296 Tromsø, Norway

[c] Norwegian School of Veterinary Science, Pb 8146 Dep, NO-0033 Oslo, Norway

[d] National Veterinary Institute, NO-9292 Tromsø, Norway

[e] National Veterinary Institute, Pb 750 Centrum, NO-0106 Oslo, Norway

[*] Corresponding author. Tel.: +47 77645092; fax: +47 77645520. E-mail address: kjetil.sagerup@uit.no (K. Sagerup).

**Abstract**

Dead and dying glaucous gulls (Larus hyperboreus) were collected on Bjørnøya in the Barents Sea in 2003, 2004 and 2005. Autopsies of the seabirds only explained a clear death cause for three (14%) of the 21 birds. A total of 71% of the birds were emaciated. Liver and brain samples were analysed for organochlorine pesticides (OCPs), polychlorinated biphenyls (PCBs), polybrominated diphenyl ether (PBDEs), hexabromocyclododecanes (HBCDs) and mercury (Hg). Elevated levels of OCPs, PCBs, PBDEs and α-HBCD were found in liver and brain. Compared to a 1989-sample of dead and dying glaucous gulls, the congeners' composition tended to change toward more persistent compounds. The brain levels of OCPs and PCBs did not change between 1989 and 2003-2005, while the liver levels decreased significantly. The brain/liver ratio for PCB and PBDE significantly decreased with halogenations of the molecule, indicating a clear discrimination of highly halogenated PCBs and PBDEs entering the brain. There was further a clear negative correlation between contaminant concentrations and body condition. The brain levels were not as high as earlier published lethal levels of p,p′-DDE or PCB. However, more recent studies reported a range of sub-lethal OCP- and PCB-related effects in randomly sampled glaucous gulls. An additional elevation of pollutants due to emaciation may increase the stress of the already affected birds. The high brain levels of OCP, PCB and PBDE of present study might therefore have contributed to the death of weakened individuals of glaucous gull.

**Key words (3-6)**

Glaucous gull, Larus hyperboreus, PCB, PBDE, lethal levels?

## 1. Introduction

Dead and dying glaucous gulls (Larus hyperboreus) have over a period of 30 years regularly been found during the breeding seasons at Bjørnøya in the Barents Sea (Bogan and Bourne, 1972; Gabrielsen et al., 1995). The dead and dying gulls are usually found during the chick-rearing period in July, which is the most energy-demanding period in birds (Moe et al., 2002). Bogan and Bourne (1972) were the first to record polychlorinated biphenyls (PCBs) and 1,1,1-trichloro-2,2-bis(4-chlorophenyl)ethane (DDT) residues in seabirds of the European Arctic. They noted that one glaucous gull that was found lying above the seabird cliffs on Bjørnøya, showed failure of coordination and fell sideways when approached. This particular bird had elevated 1,1-dichloro-2,2-bis(4-chloro-phenyl) ethylene (p,p′-DDE) and PCB levels compared to other glaucous gulls sampled (Bogan and Bourne, 1972). Gabrielsen et al. (1995) also described glaucous gulls dying in convulsions at Bjørnøya in the late 80s. These birds also had high levels of PCBs and DDE in the liver and brain compared to randomly sampled gulls.

Bjørnøya is an important breeding location as the only island in the western Barents Sea midways between the coast of Norway and Spitsbergen. Approximately half a million pairs of seabirds breed at the southern part of the island (Strøm, 2006b). The main breeding species are common guillemot (Uria aalge), Brünnich's guillemot (Uria lomvia), black-legged kittiwake (Rissa tridactyla) and glaucous gull (Strøm, 2006b). The breeding population of glaucous gulls has decreased from about 2000 pairs in 1986 to about 650 pairs in 2006, a 65% decline during 20 years (Strøm, 2006b; HS, unpub. data). It is however not known if this decline is representative for the Barents Sea population of glaucous gull, or if it is a local phenomenon on Bjørnøya.

The glaucous gull is an apex predatory species feeding opportunistically from the marine food web. It's food ranges from low food chain items such as crustaceans, through fish to high food chain items such as seabird eggs, chicks and adults and carrion (Barry and Barry, 1990; Erikstad, 1990). The apex predatory feeding and a low metabolic capacity for POPs in fish-eating seabirds generally (Walker et al., 2006), and in glaucous gulls specially (Henriksen et al., 2000), makes this species sensitive to an accumulation of POPs. The European Arctic glaucous gulls accumulate some of the highest levels of persistent organic pollutants (POPs) relative to other circumpolar arctic species (AMAP, 2004; Borgå et al., 2005a). While the levels of legacy OCPs and PCBs are declining in Artic biota, new POPs like the PBDEs are increasing (Law et al., 2003; AMAP, 2004; Helgason et al., 2008, in press).

The body mass (BM) of adult seabirds fluctuates through the year (Coulson et al., 1983; Monaghan and Metcalfe, 1986). The BM decreases during the breeding season with the lowest BM and fat content at the end of the chick rearing period (Barrett et al., 1985; Mawhinney et al., 1999; Moe et al., 2002). The OCPs, PCBs and polybrominated diphenyl ethers (PBDEs) are fat-soluble (lipophilic) and a utilisation of the stored fat during breeding and chick-rearing releases the contaminants to the blood stream. The contaminants are those redistributed in the blood, where they become available for metabolism. The metabolites and persistent pollutants that are not metabolized can be redistributed to vital organs such as the liver and brain (Henriksen et al., 1996; van den Brink et al., 1998). When released from storage, the lipophilic xenobiotics, or their metabolites, may lead to toxic effects in the birds (Walker et al., 2006). More resent studies of breeding glaucous gulls have related the highest POP concentrations to increased nematode infections, decreased reproduction and survival, fluctuating

asymmetry in wing feathers, decreased feeding efficiency and reduced levels of thyroid- and testosterone hormones (Gabrielsen, 2007, and references therein).

Aims of the present study were to establish the cause of death of the glaucous gulls by performing autopsies and POP analyses. The dead and dying glaucous gulls were collected in breeding seasons 2003-2005 on Bjørnøya. Levels of OCP and PCB were compared to those found in 1989 (Gabrielsen et al., 1995) to evaluate toxicity and temporal changes of pollutant levels.

## 2. Materials and Methods

A total of 21 dead or dying adult glaucous gulls were sampled from Bjørnøya (74°21'N, 19°05'E) in 2003, 2004 and 2005. The dead gulls were found on different locations around the island, especially in the south. The carcasses were stored frozen (-20 ºC) until autopsy.

### *2.1 Autopsies*

The autopsies were performed according to standard operating procedures at the National Veterinary Institute, Tromsø, Norway. Biometrical measurements were taken and the skins and skeletons (2003 and 2004) were preserved in the museum collection at Tromsø University Museum. Liver and brain samples were collected for POP and mercury (Hg) analysis and frozen at -20ºC until analysis. The sex was determined by gonad inspection. Tissue samples from heart, lung, liver, kidney, brain, pancreas, spleen, gonads, adrenal, thyroid and breast muscle were routinely fixed in phosphate-buffered formalin and prepared for histological examination. The degree of emaciation

was scaled from 0 (complete emaciation) to 5 (birds in good body condition), from an evaluation of amount of fat and muscles on the cadaver. Bacteriological examination was performed according to standard operating procedures when the macroscopic findings indicated a possible infection.

*2.2 Intestinal helminth counts*

The intestinal helminths were identified at the University of Tromsø, Norway. The intestine was cut in sections of about 10 cm. Each section was examined visually for macro-parasites. The intestinal contents were thereafter washed with water into two serially connected sieves, with mesh size 500- and 125 µm. The inner surfaces of the intestine were scraped with a spoon when washed. The surfaces of the oesophagus, pro-ventricle and ventricle were examined with a stereomicroscope to collect penetrating nematodes. The filtrate from each sieve was examined for helminths in a counting chamber using a stereomicroscope. The parasites were classified to phylum or class of nematoda, cestoda, trematoda and acanthocephalan. Nematode, trematode and acanthocephalan helminths are easy to quantify by counting the individuals. The cestodes are fragile and the scoleces could be hard to release from the intestine wall, resulting in a less precise cestode intensity measure. To reduce this imprecision the cestode intensity measures were always done by the same person (Sagerup et al., 2000).

*2.3 Chemical analyses of OCPs, PCBs and PBDEs*

The chemical analyses of OCPs, PCBs, PBDEs and hexabromocyclododecanes (HBCDs) were performed at the Laboratory of Environmental Toxicology at the Norwegian School of Veterinary Science, Oslo, Norway. The hexachlorobenzene (HCB), hexachlorocyclohexanes (α-, β- and γ-HCH), chlordanes (oxychlordane, cis-chlordane, trans-nonachlor), p,p′-DDE, mirex, PCB congeners; PCB-28, 52, 74, 66, 101, 99, 110, 149, 118, 114, 153, 105, 141, 138, 187, 183, 128, 156, 157, 180, 170, 194, 206 and 209, PBDE congeners; BDE-28, 47, 100, 99, 154, 153, 183, 208, 207, 206 and 209 and total HBCD (sum of α, β, γ- isomers) and the individual α, β, γ-HBCDs were analysed. The PCBs and PBDEs are presented in the order of appearance of the gas chromatograph. Principles of the analytical methods was described by Brevik (1978) and modified by Bernhoft et al. (1997). Details of the extraction, clean up, detection and calculation of OCPs, PCBs, PBDEs and HBCDs were previously described (Murvoll et al., 2005; Sørmo et al., 2006; Polder et al., 2008). Briefly, the samples were weighed, internal standards were added (PCB-29, 112 and 207, BDE-77, 119, 181 and $^{13}$C-BDE-209 (Cambridge Isotope Laboratories, Inc., Andover, MA, USA) and extracted twice with cyclohexane and acetone (3:2). The percentage extractable lipids were calculated gravimetrically by evaporation of an aliquot of the extract. The extracts were treated with concentrated ultrapure sulphuric acid (96%, Chemscan AS, Elverum, Norway) before analysis on a high resolution gas chromatograph (GC; Agilent 6890 Series gas chromatography system; Agilent Technologies, PA, USA) with an auto sampler (Agilent 7683 Series; Agilent Technologies) and two column (SPB-5 and SPB-1701, Supelco, Bellefonte, PA, USA) coupled to two $^{63}$Ni μ-electron capture detectors (Agilent 6890 μ-ECD). The separation and detection of BDE-28, 47, 100, 99, 154, 153, 183 and total HBCD was performed

on a GC (Hewlett Packard 6890 Series) equipped with a pulsed splitless injector (at 250°C) connected to a MS quadrupol detector (Agilent Technologies, Avondale, PA, USA). The mass spectrometer was operated in the electron capture mode with methane as buffer gas. The separation and detection of the nona-BDEs (BDE-208, 207, 206) and deca-BDE (BDE-209) were performed on a GC-MS (J&W Scientific, Agilent Technologies) with a DB-5-MS column (J&W Scientific, Agilent Technologies). The quantification standards were made using pure standards of BDE-206, 207, 208 and 209 (Cambridge Isotope Laboratories, Inc., Andover, MA, USA). The separation and detection of α-, β- and γ-HCBD were performed using an API 3000 LC-MS-MS system (triple quadrupole, Applied Biosystem, USA) connected to a C18 column. The detection limits for individual compounds were determined as three times the noise level and ranged from 0.03 to 0.69 ng/g wet weight (wet wt.).

The lab is accredited for determination of OCPs, PCBs, PBDEs and HBCD in biological material according to NS-EN ISO/IEC 17025 (TEST 137). Deviation in response of the GC system was controlled by analysing a standard for every $10^{th}$ sample. The reproducibility of the method was tested by analysing the laboratory's own reference sample of seal blubber which was analysed for each of the analytical series. For recovery tests, hen egg homogenates were spiked with OCs and BFRs. Several blanks (solvents) and one blank (hen egg) were included in every series. Results of the quality tests were within the laboratory's accredited requirements. The recoveries of the OCPs, PCBs and PBDEs analysed in the present study ranged from 97% to 145% and the recovery of α-HBCD was 76%. The analytical quality for OCs, PBDEs and HBCD in different biological matrices has been found satisfactory, including at very low levels, after participation in relevant intercalibration tests, such as

Quasimeme (2002, Exercise round 524); FIRE, 2003. Quasimeme, 2003 (Exercise 565, round 33), NIST/NOAA/MMHSRP 2003 and 2005, and an Interlaboratory Comparison on HBCD in Biological Samples by the Norwegian Institute of Public Health, Oslo, Norway.

*2.4 Chemical analyses of mercury*

Mercury levels were measured at the National Veterinary Institute, Oslo, Norway. The samples were digested (EN 14084:2003 ) by a mixture of nitric acid and hydrogen peroxide in a closed system using a microwave oven (Milestone), and analysed by cold vapour atomic absorption spectrometry (Varian Incorporated) using (tin(II) chloride (Merck) reduction at 253.7 nm and D2-background correction). For details, se Welz and Sperling (1999) and Sturman (1985).

Certified reference materials, such as TORT-2 and LUTS-1 from the National Research Council of Canada were used together with blanks and internal quality solutions. The detection limit of 3xSD of blank samples was 0.01 mg/kg. The measurement uncertainty is 40% in level 0.01-0.4 mg/kg and 20% in level 0.4-25 mg/kg wet wt. The analytical method is accredited after NS-EN ISO/IEC 17025 (Test nr.110) by the Norwegian Accreditation. The laboratory's accredited analytical quality has been approved in several international intercalibration tests (FAPAS, Quasimeme and National Food Administration of Sweden).

*2.5 Statistics*

The concentration mean for a given compound was only determined if 60% or more of the samples had concentrations above the mean detection level (MDL) (Verreault et al., 2005c). These were the only compounds used in the further analyses. For the compounds in the statistical analysis, a randomly generated value between zero and the MDL replaced the samples below the MDL. Statistical analyses were carried out using the free statistical software R (R Development Core Team, 2008). The significance level was set to $\underline{P} < 0.05$.

Due to higher percentage of extractable liver lipids in 2003 than in 2004 and 2005 (one-way ANOVA: $F_{1,19} = 5.75$, $\underline{P} = 0.03$) and a large difference in liver lipids between 1989 and 2003-2005 (two-sample Wilcoxon test: $W = 231$, $\underline{P} < 0.0001$) the analyses were performed with lipid normalized data. Natural log transformation (ln) of lipid weight (lipid wt.) concentrations resulted in residuals with a constant variance and normal distribution. The linearity was evaluated by the means of diagnostic quantile-quantile plots, a graphical technique which plots the residuals against fitted values. A body condition index (BCI) of residuals from a linear regression of standardized BM and standardized size index was used. Analysis of variance (ANOVA) with sex and BCI as confounders was conducted to test for differences between years and the non-parametric Wilcoxon test for differences between sexes. Correlations were calculated with linear regressions or the non-parametric Spearman rank correlation.

## 3. Results

### *3.1 Autopsy findings*

Eight (38%) of the 21 birds were found to be completely (n = 4) or severely (n = 4) emaciated according to the subjective scaling of emaciation. Seven birds (33%) were classified as emaciated. Five of these showed various degrees of oedema in the lungs. Six birds (29%) were found to be in normal (n = 2), or slightly below normal condition (n = 4). Three of these birds had lung oedema. Plausible death causes were diagnosed for three birds (14%). This was anaemia, a broken wing and hemorrhagic enteritis. The two former individuals were completely emaciated whereas the latter was classified to be in normal condition.

Since the correlation between the subjective emaciations score and the calculated BCI ($t_{17} = 5.3$, $\underline{P} < 0.0001$, $R^2 = 0.62$) were good, the BCI was used as confounder in the further analysis. The heads were missing in 2 of the birds sampled and BCI could consequently not be calculated for these. The sample size were therefore 19 when BCI was included in the analyses. The BCI was positively correlated with extractable lipid content in brain ($t_{17} = 3.2$, $\underline{P} = 0.005$, $R^2 = 0.38$), but not with the extractable lipid percentage in liver ($t_{17} = 0.1$, $\underline{P} = 0.9$).

The examination of suspicious bacteriological signs did not result in any infection findings that could be related to the death of the birds. As an incidental finding, one or more cysts of Sarcocystis sp. were found in histological sections from breast muscles in five (24%) of the birds.

*3.2 Levels and congener pattern of POPs*

Mean hepatic concentrations of p,p′-DDE, $\Sigma PCB_{24}$ and $\Sigma BDE_{11}$ were 215, 1033 and 26 µg/g lipid wt. respectively (Table 1). The corresponding brain levels were 64, 255 and 3 µg/g lipid wt., respectively (Table 1). The concentrations of OCPs, PCBs or BDEs did not differ between the three collection years ($F_{1,19} < 2.47$, $P > 0.13$). The samples from these years were therefore treated as one sample. The mean hepatic and brain lipid wt. levels of all organic contaminants, except cis-chlordane, PCB-141, BDE-209 and α-HBCD from liver and PCB-110 and -206 from brain were approximately 1.4 times higher for males than for females, but none of the differences were significant. We therefore included both sexes in the statistical analysis, but included sex as a confounder. The most abundant OCPs were p,p′-DDE>oxychlordane>HCB, while PCB-153>138>180>118 were the most abundant PCBs and BDE-47>153>99=100 were the most abundant PBDEs (Table 1 and Figure 1) in both liver and brain. The α-HBCD was the only HBCD enantiomer found in the gulls (Table 1). The ΣOCP, ΣPCB and ΣPBDE make up 21%, 77% and 2%, respectively, of the sum of all organic contaminants in liver. The corresponding distribution in the brain was similar, 25%, 74% and 1%, respectively.

The concentrations of all xenobiotics were higher in the liver than in the brain, except for the BDE-154 which was below detection limit in liver (Table 1). The $\Sigma PCB_{24}$ and $\Sigma PBDE_{11}$ concentrations were for example 4 and 10 times higher in liver than in the brain, respectively. There was in addition a systematic decrease in brain/liver concentration ratio with an increase in the number of chlorines in the PCB molecules ($t_{22} = -3.3$, $P = 0.004$, $R^2 = 0.33$, Figure 2a). The brain concentrations were about 35% of the liver concentrations for tri- and tetra-chlorinated PCBs, and less than 10% for the

nona- and penta-chlorinated PCBs. The same pattern was found for bromine in PBDEs ($t_5$ = -6.8, P = 0.001, $R^2$ = 0.90, Figure 2b). The brain concentration was 12% of the liver concentration for the tri-brominated BDE-28 and only 2% for the nona-brominated BDE-207.

Generally, the highest lipid levels of contaminants were found in birds diagnosed as emaciated. The $\Sigma PCB_{24}$ and the $\Sigma BDE_{11}$ concentrations, both for liver and brain, were negatively correlated with BCI ($t_{17} < -2.3$, $P < 0.03$, $R^2 > 0.24$, Figure 3).

*3.3 Levels of Hg*

The mean Hg level was 1.5 μg/g wet wt. (SE = 0.2, range 0.3 − 4.3 μg/g wet wt.) in the liver of the glaucous gulls. There were no differences in concentration between the years 2003, 2004 and 2005 ($F_{1,19}$ = 0.005, P = 0.95) or between sexes ($F_{1,19} < 0.001$, P = 0.98).

*3.4 Intestinal helminths*

Nematodes were the most prevalent helminths, followed by cestodes, trematodes and acanthocephalans (Table 2). The nematode intensity of the 13 infected gulls range from 1 to 11 and one outlier had 233 nematodes. The trematodes and cestodes showed similar intensity ranges with one or two outliers, respectively. Only one gull was infected by acanthocephalans. The parasite intensity did not differ between the sampling years 2003-2005. No correlations to POP concentrations were found (Spearman rank correlation: rho < 0.22, P > 0.06).

*3.5 Comparisons to the 1989-sample*

A comparable sample of 11 dead and dying glaucous gulls was collected at Bjørnøya and Spitsbergen in 1989 (Gabrielsen et al., 1995). Levels of OCPs and PCBs were measured at the same laboratory and with the same method as the sample from 2003-2005. Three individual OCPs and 18 individual PCBs were measured in both samples and these were compared. The liver lipid wt. levels of all OCPs and individual PCB congeners, except PCB-187, were significantly higher in 1989 than in 2003-2005 (Figure 1a). The mean $\Sigma PCB_{18}$ hepatic lipid wt. level was 8 times higher in 1998 than in 2003-2005. The brain lipid wt. levels of all OCPs and PCB-74, 153, 105, 138, 183, 180, 170 and 209 were however, not different in 1989 and 2003-2005 (Figure 1b). Due to increased concentrations in three of the most abundant PCBs (153, 138, 180), the brain mean $\Sigma PCB_{18}$ lipid wt. level was exactly the same in 1989 and 2003-2005 (Figure 1b).

The PCB profile both for liver and brain changed, however, from 1989 to 2003-2005. The percentage contents of total PCB increased for the PCB-153, 138, 187 and 180, decreased for the PCB-101, 118, 105, 128, 156, 170 and 194 and were less than 1% for the PCB-28, 52, 74, 114, 183, 157 and 209 (Figure 4).

## 4. Discussion

Most of the sick and dead birds from the present study were in poor body condition. The eight birds classified as completely or severely emaciated could have died due to lack of body reserves. However, the cause of emaciation was unknown. Only two birds had normal body condition according to breast muscular size and fat reserves. The Sarcocystis sp. muscle cysts reported in five of the birds are normally not of clinical significance. The poor body condition is in agreement to dead glaucous gulls collected on Bjørnøya in 1989 (Gabrielsen et al., 1995) and with a sample of dead ring-billed gulls (Larus delawarensis) from Ontario Canada that also was suspected to be affected by OCs (Sileo et al., 1977).

Some of the studied birds were observed as they became sick and died. They typically lost their ability to fly, had tremors and trouble to maintain balance. This abnormal behaviour usually started two to three days before they died. The sick birds often deserted their nest and moved to nearby freshwater, or simply sat on the tundra until they died. These symptoms were the same as those described for the highly p,p′-DDE and PCB-contaminated glaucous gull sampled on Bjørnøya in 1972 (Bogan and Bourne, 1972). The described behavioural patterns were also similar to those of four passerine bird species experimentally fed PCB-containing food (Stickel et al., 1984) and with moribund ring-billed gulls (Sileo et al., 1977).

All studied birds were adults. They were all found on breeding ground and were assumed to be breeding birds. The collection dates were all in last part of June and in July. The peak hatching for glaucous gull at Bjørnøya is late June, indicating that death of adults starts after hatching. The time span for sampling was similar to that of ring-

billed gulls found dead in Ontario, Canada (Sileo et al., 1977). The latter birds were collected in July, August and September, i.e. the late chick-rearing period or soon after (Sileo et al., 1977). It is possible that gulls dying in May were not found since the scientists arrives the island in late May. However, the scavenging birds on Bjørnøya (great skua Stercorarius skua, arctic skua S. parasiticus and glaucous gull) are not able to carry the heavy glaucous gulls away, and the arctic fox (Alopex lagopus) has not been observed to carry away dead gulls on Bjørnøya. Besides, arctic foxes have easy access to seabird eggs from the middle of May. We then think that the death of breeding glaucous gulls starts around hatching and continues during the chick-rearing period. Chick-rearing is the most energy demanding period for seabirds (Monaghan and Metcalfe, 1986; Moe et al., 2002), and the low body weights and depleted fat reserves of the studied gulls suggests that these individuals were not able to compensate for their energetic needs by foraging.

Long-term population monitoring of seabirds on Bjørnøya indicates a strong decline in the breeding population of glaucous gulls in the period 1986-2006 (Strøm, 2006b). The populations of other surface-feeders such as the black-legged kittiwake have not declined, suggesting that the decline of glaucous gull is not caused by food shortage (Strøm, 2006b). The number of arctic foxes has, however, increased (Strøm, 2006a). These arctic foxes predate glaucous gull nests. Since the glaucous gull breeds on flat land and has problems defending their nests against the arctic fox, their eggs are an easy meal. Whether the arctic fox influences the glaucous gull population is however not known.

*4.1 Levels and effects*

The hepatic levels of OCPs, PCBs and PBDEs were 10-40 times higher in present study than previously reported in randomly sampled glaucous gulls from the Barents Sea area (Henriksen et al., 2000; Borgå et al., 2001; Haukås et al., 2007). For example the latest published hepatic mean levels of PCB vary: $\Sigma PCB_9$ 75.3 µg/g lipid wt. (Henriksen et al., 2000), $\Sigma PCB_{30}$ 130.4 µg/g lipid wt. (Borgå et al., 2001), $\Sigma PCB_{24}$ 30.7 µg/g lipid wt. (Herzke et al., 2003) and $\Sigma PCB_{13}$ 27.1 µg/g lipid wt. (Haukås et al., 2007), while our hepatic mean of $\Sigma PCB_{24}$ was 1033 µg/g lipid wt. To our knowledge, only one study has measured the brain level of OCPs and PCBs in a random sample of glaucous gull from the European Arctic and the mean brain levels of p,p′-DDE and $\Sigma PCB_{19}$ were 2.2 and 6.0 µg/g lipid wt., respectively (Savinova et al., 1995). The brain levels of p,p′-DDE and PCBs in our sample are then 30 and 40 times higher, respectively, in the sick and dead glaucous gulls (Table 1) than in a random sample of glaucous gulls from the Barents Sea.

The levels of PBDE were also high in this study. The mean lipid wt. liver level of BDE-47 and -99 were up to 40 times higher than previously measured levels in glaucous gull liver (Herzke et al., 2003; Haukås et al., 2007). It was also about 400 and 1500 times higher, respectively, than previously reported in black guillemot (Cepphus grylle) (Haukås et al., 2007). Kelly et al. (2008) reported that BDE-47 was the only DBE-congener with a biomagnification factor greater than 1 in an arctic marine food web. This could explain some of the observed dominance of BDE-47 in aquatic ecosystems (de Wit, 2002; de Wit et al., 2006; Sørmo et al., 2006). Other factors such as bioavailability, uptake efficiency and metabolism may also influence the PBDE profiles. In this study, three HBCD enantiomers were measured, but only α-HBCD was

detected (Table 1). This corroborates a previous finding in glaucous gulls (Verreault et al., 2007).

In birds, lethal residual brain levels are about 150-250 and 300 µg/g wet wt. for p,p′-DDE and PCB, respectively (Sileo et al., 1977; Ohlendorf et al., 1981; Stickel et al., 1984). However, great cormorants (Phalacrocorax carbo sinensis) dosed experimentally with the technical PCB mixture Clophen-A60 died with lower brain residue levels (76-180 µg/g wet wt.) (Koeman et al., 1973). The maximum brain levels of p,p′-DDE and $\Sigma PCB_{24}$ in this study were, however, only 14 and 45 µg/g wet wt.. Present brain levels were therefore less than 10-20% of the generally accepted lethal levels and 25-60% of the suggested cormorant lethal levels. It seems therefore unlikely that the p,p′-DDE or PCB levels alone could have caused the death of the birds. However, the total number of pollutants in the studied birds was high (Table 1) and the cocktail of these xenobiotics and their metabolites (Verreault et al., 2005a, 2005b) can increase the birds' stress levels during the hard working chick-rearing period.

Effects of POPs in adult glaucous gulls have further been documented at many biological organizational levels, including endocrine, immunological, bioenergetics, enzymatic, behavioural, reproductive, survival and developmental, reviewed by Bustnes (2006) and Gabrielsen (2007). These correlative studies showed that the highest levels of POPs within the apparently healthy glaucous gulls were associated with adverse effects. It is therefore reasonable to claim that the up to 40 times elevation of POP levels could increase the stress in glaucous gulls already stressed by high POP levels.

The higher POP levels in liver than in brain and the systematic decrease in brain/liver concentration ratios with an increase in numbers of halogens (Figure 2) suggest that brain tissue is better protected against lipid soluble contamination than liver tissue. This has been reported in several species, and has been suggested to be a result of the blood-brain barrier (Bachour et al., 1998). Different lipid groups have also different affinity for different POPs (Kawai et al., 1988). The brain of glaucous gulls contains relatively more polar phospholipids and cholesterol compared to liver, muscle and fat tissue (Halvorsen, 1997). The accumulation of the relatively polar low-halogenated compounds in the brain could therefore also be the result of solubility. Whether lower concentrations and different congener compositions in the brain are due to selective transport through the blood-brain barrier, different lipid composition in the brain compared to other tissues, or a combination of these or other factors remains to be solved.

The negative relationship between BCI and PCB/PBDE concentrations (Figure 3) results probably from the redistribution of lipophilic contaminants. The half-life of the present PCB-congeners are around, or more than, a year (Drouillard and Norstrom, 2003). The mobilization of fat reserves during incubation and chick-rearing is therefore much faster than the bird's capacity to metabolize the released PCB. The mobilization of deposited fat therefore increased the concentrations of lipophilic pollutants in remaining fats, such as in the liver or brain.

*4.2 Mercury*

The levels of Hg were similar to those reported in other arctic seabird species and those previously reported in glaucous gulls (Savinov et al., 2003; Borgå et al., 2006). The Hg levels in the present study were, however, below the levels known to cause effects on terrestrial bird hatching success and well below the threshold level for lethal effects (Derome et al., 2005). Savinov et al. (2003) concluded that the Hg concentrations in arctic seabirds were mainly a result of natural background levels rather than from pollution input.

*4.3 Intestinal parasites*

No relationship between intestinal helminth intensities and OCP, PCB, PBDE or α-HBCD levels were found in the present study. This result is in agreement with glaucous gulls sampled in August 2001 at Barentsburg, Svalbard, where correlations between pollutant levels and parasites also were absent (Sagerup et al., submitted). Positive correlations between OC concentrations and nematode intensity were, however, found in breeding glaucous gulls (Sagerup et al., 2000). The ΣPCB liver levels of the three studies were 1033 µg/g lipid wt. (Table1), 29 µg/g lipid wt. (Sagerup et al., submitted) and 75 µg/g lipid wt. (Sagerup et al., 2000), respectively. The latter two studies illustrate two important aspects of toxicological field studies, concentrations of pollutants and confounding factors. An adverse effect was found in the most polluted sample, but, at the same time, these gulls were breeding. Since breeding is energetically costly and involves hormones that also affect the immune system, the threshold levels for influencing the immune system could be lower at this time of the year. In the present study, a reduced infection rate due to less feeding as the birds grow thinner, could have influenced the helminth number. The BCI indicates also

that the individuals' emaciation was uneven, suggesting that possible correlations could be have been lost during the emaciation period.

*3.5 Comparisons to the 1989-sample*

The declined liver lipid wt. levels of OCPs and PCBs from 1989 to 2003 corroborates the general decline in PCBs and DDTs in the northern hemisphere (Muir et al., 1992; Bignert et al., 1998; AMAP, 2004). That the most persistent PCB-153, 138 and 180 congeners (Borlakoglu et al., 1990; Borgå et al., 2005b) were more abundant in the 2003-2005 sample than in the 1989 sample (Figure 4) indicates further that the supply of new PBCs to the biota is decreasing.

The constant brain levels during the same period, suggest that the organochlorine concentrations in brain increase to the same level in emaciating glaucous gulls, independently of total OC amounts in the body. This observation raises the question whether the halogenated organic contaminants could contribute to the death in these glaucous gulls.

It is natural that old or weak birds die during the incubation- and chick-rearing periods, but the observed inactivity, poor balance and convulsions suggest an effect of toxins. We therefore conclude that contamination levels add supplementary stress to those glaucous gulls that deplete their body reserves during breeding and chick-rearing. Even though the brain residues of p,p′-DDE and PCBs were lower than earlier published deadly levels in birds, we propose that some glaucous gulls pass a point where the combined stress effect of reduced body reserves and high POP concentrations, become mortal.

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Table 1: The mean (± standard error) and range (µg/g lipid wt.) of persistent organic pollutants in liver and brain of glaucous gull found dead or dying at Bjørnøya in the Barents Sea.

| | Liver | | | Brain | | |
|---|---|---|---|---|---|---|
| **Compound** | **n** [a] | **Mean ± SE** | **Range** [a] | **n** [a] | **Mean ± SE** | **Range** [a] |
| lipid % | 21/21 | 4.8 ± 0.4 | 2.9 – 10.4 | 21/21 | 7.1 ± 0.1 | 6.2 – 8.3 |
| HCB | 21/21 | 26.4 ± 9.4 | 2.6 – 206.4 | 21/21 | 8.3 ± 1.7 | 0.8 – 27.0 |
| α-HCH | 19/21 | 0.1 ± 0.0 | <MDL – 0.1 | 18/21 | 0.004 ± 0.0 | <MDL – 0.02 |
| β-HCH | 21/21 | 1.0 ± 0.2 | 0.1 – 3.3 | 21/21 | 0.2 ± 0.1 | 0.01 – 0.9 |
| γ-HCH | 0/21 | - | - | 1/21 | - | <MDL – 0.02 |
| oxychlordane | 21/21 | 28.9 ± 6.1 | 2.5 – 103.9 | 21/21 | 11.6 ± 2.9 | 0.7 – 42.1 |
| cis-chlordane | 20/21 | 0.4 ± 0.1 | <MDL – 1.1 | 21/21 | 0.3 ± 0.1 | 0.03 – 1.0 |
| trans-nonachlor | 21/21 | 0.5 ± 0.1 | 0.1 – 1.5 | 21/21 | 0.3 ± 0.1 | 0.03 – 1.0 |
| p.p′-DDE | 21/21 | 215.1 ± 43.2 | 17.2 – 732.3 | 21/21 | 63.8 ± 14.5 | 4.4 – 232.0 |
| Mirex | 21/21 | 13.0 ± 2.5 | 1.1 – 35.1 | 21/21 | 2.9 ± 0.6 | 0.2 – 8.2 |
| PCB-28 | 21/21 | 1.5 ± 0.3 | 0.2 – 3.8 | 21/21 | 0.6 ± 0.1 | 0.04 – 2.0 |
| PCB-52 | 21/21 | 0.7 ± 0.1 | 0.1 – 1.5 | 21/21 | 0.1 ± 0.0 | 0.02 – 0.4 |
| PCB-74 | 21/21 | 6.4 ± 1.1 | 0.9 – 16.7 | 21/21 | 2.7 ± 0.6 | 0.3 – 8.9 |
| PCB-66 | 21/21 | 6.8 ± 1.2 | 1.0 – 16.4 | 21/21 | 2.5 ± 0.5 | 0.2 – 8.1 |
| PCB-101 | 21/21 | 4.1 ± 0.7 | 0.4 – 9.2 | 21/21 | 0.5 ± 0.1 | 0.05 – 1.4 |
| PCB-99 | 21/21 | 37.4 ± 6.6 | 4.2 – 90.4 | 21/21 | 12.7 ± 2.6 | 0.9 – 39.1 |
| PCB-110 | 21/21 | 1.7 ± 0.3 | 0.2 – 5.1 | 21/21 | 0.2 ± 0.0 | 0.02 – 0.9 |
| PCB-149 | 21/21 | 4.6 ± 0.9 | 0.8 – 15.4 | 21/21 | 1.6 ± 0.4 | 0.1 – 6.7 |
| PCB-118 | 21/21 | 64.0 ± 11.9 | 6.6 – 176.3 | 21/21 | 20.7 ± 4.5 | 1.5 – 73.2 |
| PCB-114 | 21/21 | 2.9 ± 0.5 | 0.3 – 7.3 | 21/21 | 0.9 ± 0.2 | 0.1 – 3.2 |
| PCB-153 | 21/21 | 297.3 ± 55.0 | 26.2 – 770.1 | 21/21 | 87.6 ± 17.9 | 0.6 – 253.7 |
| PCB-105 | 21/21 | 15.5 ± 2.7 | 1.9 – 43.3 | 21/21 | 10.7 ± 6.1 | 0.4 – 131.1 |
| PCB-141 | 21/21 | 0.7 ± 0.1 | 0.1 – 2.0 | 16/21 | 0.02 ± 0.0 | <MDL – 0.1 |
| PCB-138 | 21/21 | 263.9 ± 124.3 | 15.1 – 2 696.6 | 21/21 | 41.9 ± 8.4 | 2.8 – 114.5 |
| PCB-187 | 21/21 | 17.6 ± 3.5 | 2.3 – 53.7 | 21/21 | 4.9 ± 1.1 | 0.4 – 17.7 |
| PCB-183 | 21/21 | 24.9 ± 4.5 | 2.1 – 58.2 | 21/21 | 6.5 ± 1.3 | 0.4 – 16.0 |
| PCB-128 | 21/21 | 8.7 ± 1.4 | 1.2 – 21.0 | 21/21 | 3.4 ± 0.8 | 0.2 – 11.1 |
| PCB-156 | 21/21 | 19.5 ± 4.0 | 0.3 – 58.1 | 21/21 | 5.1 ± 1.1 | 0.3 – 15.7 |

| | | | | | | |
|---|---|---|---|---|---|---|
| PCB-157 | 21/21 | 5.0 ± 0.9 | 0.5 – 13.7 | 21/21 | 1.3 ± 0.3 | 0.1 – 4.3 |
| PCB-180 | 21/21 | 168.2 ± 33.3 | 10.1 – 453.0 | 21/21 | 36.7 ± 7.4 | 1.6 – 91.5 |
| PCB-170 | 21/21 | 46.8 ± 8.9 | 3.0 – 123.5 | 21/21 | 10.3 ± 2.1 | 0.4 – 27.7 |
| PCB-194 | 21/21 | 27.7 ± 5.7 | 1.3 – 72.5 | 21/21 | 3.7 ± 0.7 | 0.2 – 8.4 |
| PCB-206 | 21/21 | 4.5 ± 0.9 | 0.3 – 11.1 | 21/21 | 0.4 ± 0.1 | 0.04 – 1.0 |
| PCB-209 | 21/21 | 2.5 ± 0.4 | 0.2 – 5.3 | 21/21 | 0.2 ± 0.0 | 0.1 – 0.5 |
| $\Sigma PCB_{24}$ | 21/21 | 1 033.0 ± 227.7 | 84.1 – 4 274.0 | 21/21 | 255.0 ± 52.5 | 16.7 – 711.5 |
| BDE-28 | 21/21 | 0.2 ± 0.0 | 0.01 – 0.6 | 21/21 | 0.02 ± 0.0 | 0.0004 – 0.1 |
| BDE-47 | 21/21 | 14.2 ± 3.4 | 1.2 – 56.8 | 21/21 | 1.5 ± 0.3 | 0.1 – 5.9 |
| BDE-100 | 21/21 | 3.2 ± 0.8 | 0.2 – 12.5 | 21/21 | 0.3 ± 0.1 | 0.03 – 1.2 |
| BDE-99 | 21/21 | 3.2 ± 0.9 | 0.2 – 16.3 | 21/21 | 0.3 ± 0.1 | 0.02 – 1.7 |
| BDE-154 | 0/21 | - | - | 21/21 | 0.1 ± 0.0 | 0.006 – 0.3 |
| BDE-153 | 21/21 | 4.5 ± 1.3 | 0.1 – 21.3 | 21/21 | 0.4 ± 0.1 | 0.02 – 1.7 |
| BDE-183 | 16/21 | 0.2 ± 0.1 | <MDL – 1.2 | 21/21 | 0.08 ± 0.0 | 0.0008 – 0.03 |
| BDE-208 | 16/21 | 0.02 ± 0.0 | <MDL – 0.1 | 5/21 | - | <MDL – 0.001 |
| BDE-207 | 19/21 | 0.04 ± 0.0 | <MDL – 0.2 | 14/21 | 0.001 ± 0.0 | <MDL – 0.003 |
| BDE-206 | 1/21 | - | <MDL – 0.007 | 0/21 | - | - |
| BDE-209 | 19/21 | 0.2 ± 0.1 | <MDL – 2.6 | 5/21 | - | <MDL – 0.01 |
| $\Sigma BDE_{11}$ | 21/21 | 25.8 ± 6.3 | 1.8 – 102.8 | 21/21 | 2.5 ± 0.6 | 0.2 – 10.9 |
| α-HBCD | 21/21 | 3.0 ± 0.9 | 0.2 – 15.0 | 21/21 | 0.1 ± 0.0 | 0.005 – 0.5 |
| β-HBCD | 0/21 | - | - | 0/21 | - | - |
| γ-HBCD | 0/21 | - | - | 0/21 | - | - |

[a] MDL = median detection limit. n = samples above MDL/ samples analysed.

Table 2: The prevalence (% infected), mean abundance, mean intensity and maximum intensity of intestinal parasites in adult glaucous gulls from Bjørnøya, Barents Sea. Mean abundance = number of parasite individuals/n, mean intensity = number of parasite individuals/ number of infected hosts (Bush et al., 1997). N = 21.

| Parasite group | Prevalence | Mean abundance | Mean intensity | Max intensity |
|---|---|---|---|---|
| Cestode | 48 | 38 | 79 | 529 |
| Nematode | 62 | 13 | 21 | 233 |
| Trematode | 43 | 7 | 17 | 88 |
| Acanthocephalan | 5 | - | 8 | 8 |

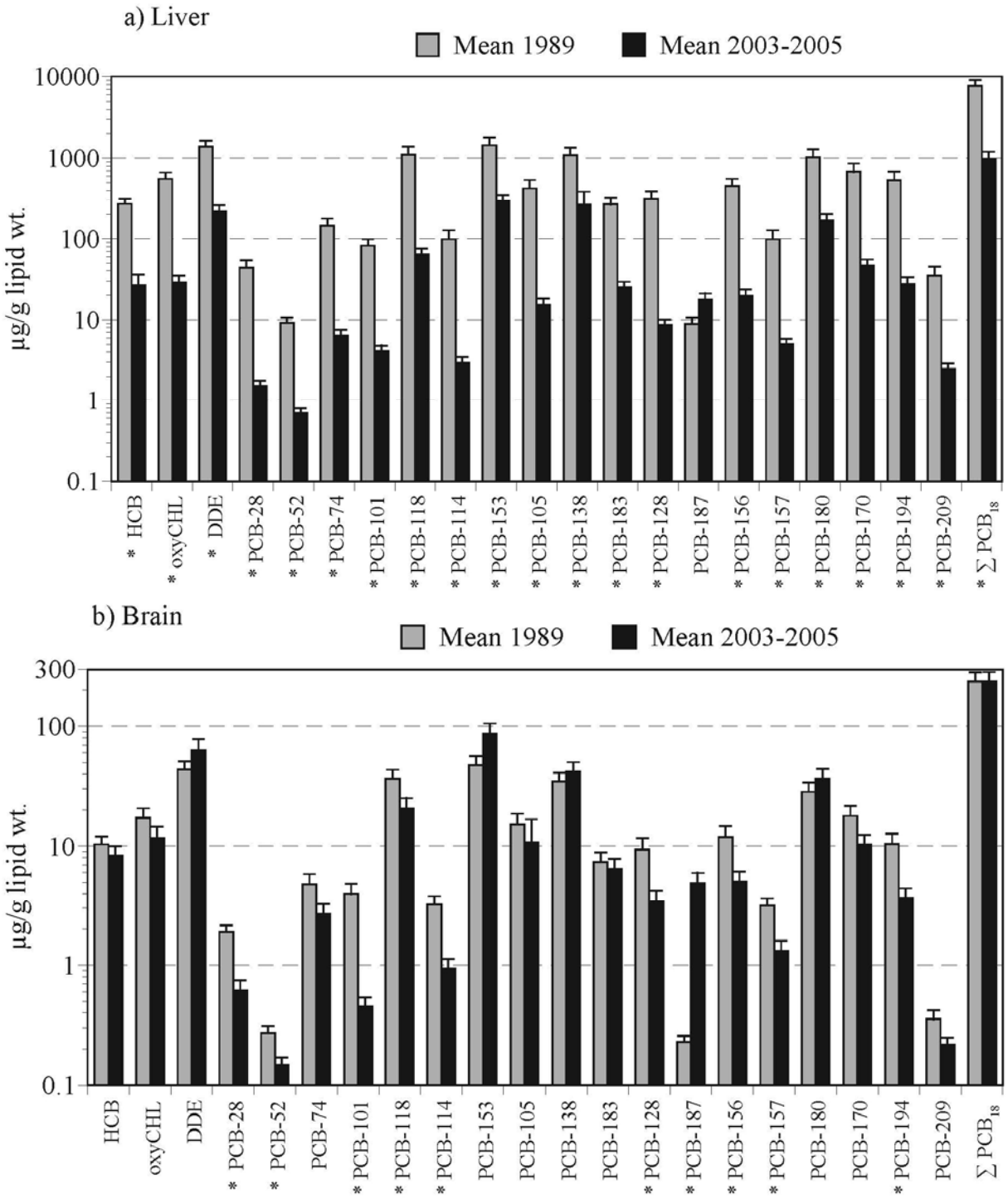


Figure 1: The mean and summarized mean (+ standard error) µg/g lipid wt. of three organochlorine pesticides and 18 polychlorinated biphenyls (PCB) in a) liver and b) brain samples from glaucous gulls found dead or dying at Bjørnøya and Spitsbergen in 1989 (n = 11) and Bjørnøya in 2003-2005 (n = 21). * Significant different ($\underline{P} < 0.05$). Data for 1989 from Gabrielsen et al. (1995).

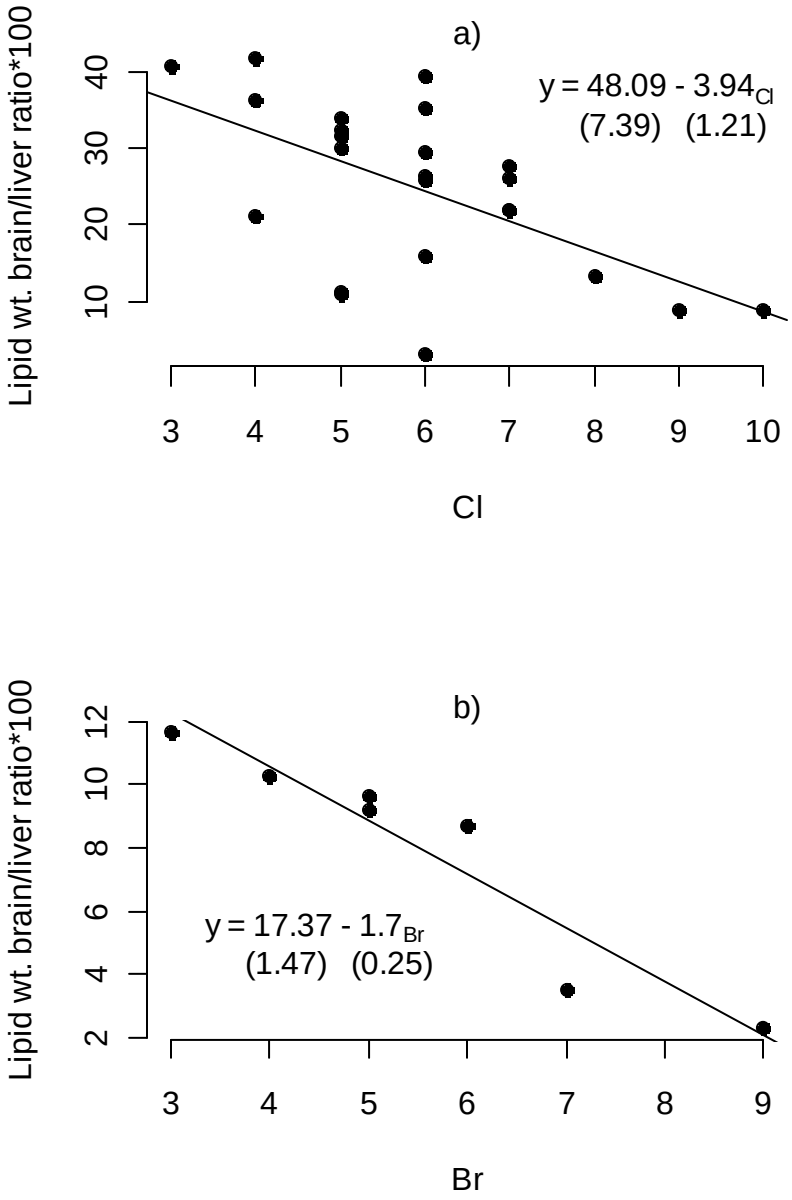


Figure 2: Scatter plot and regression line for the brain/liver concentration percentage (lipid wt.) to the a) numbers of chlorines (Cl) in the PCB molecule and b) numbers of bromines (Br) in the PBDE molecule from glaucous gulls found dead or dying at Bjørnøya 2003-2005 (n = 24 (PCBs) and n = 7 (PBDEs)). The regression line is given with its estimated standard errors below the equation.

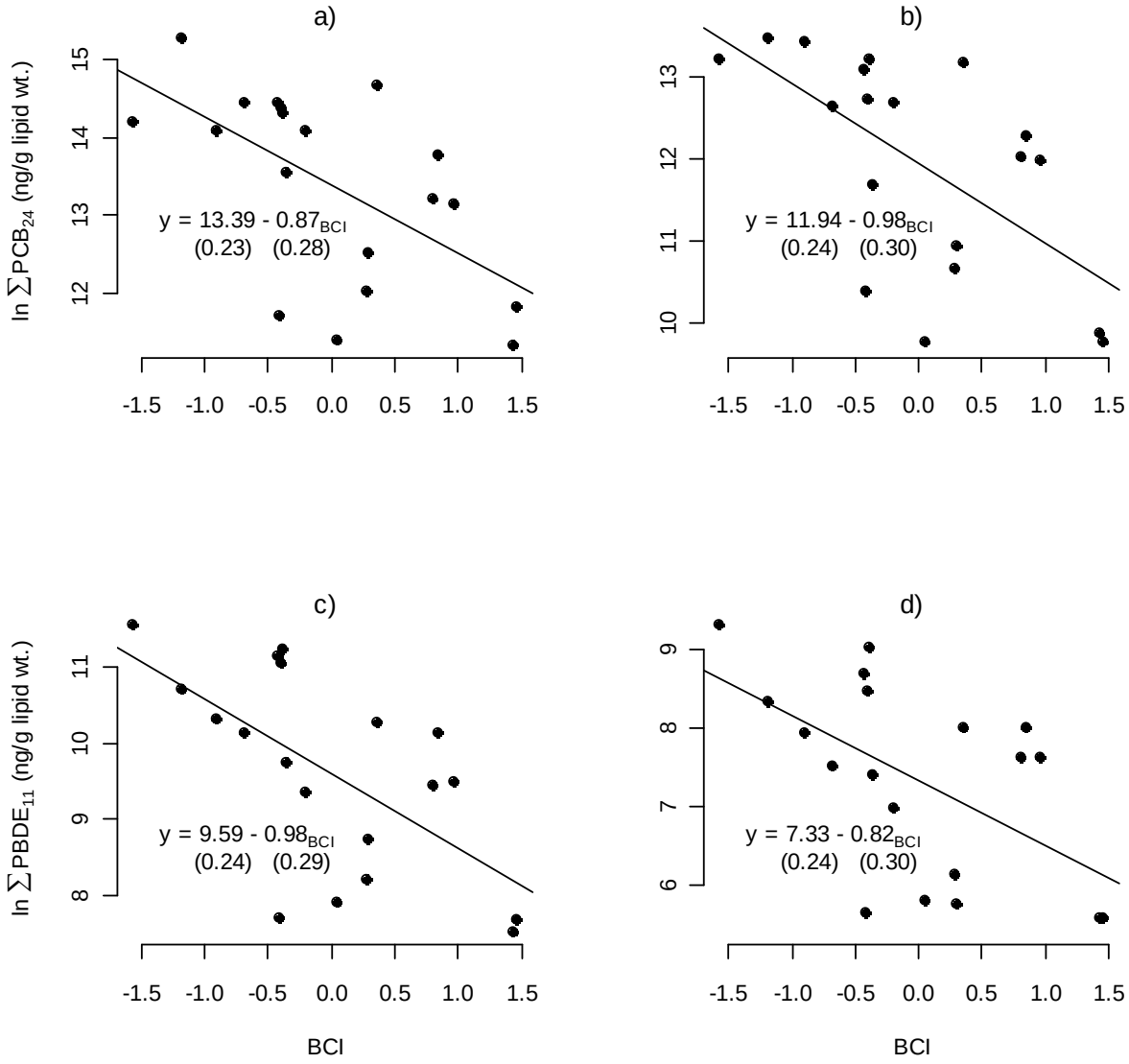


Figure 3: Scatter plot and regression line for natural logarithm (ln) $\Sigma PCB_{24}$ and $\Sigma BDE_{11}$ (ng/g lipid wt.) in glaucous gulls found dead or dying at Bjørnøya 2003-2005 (n = 21). Liver samples; (a) and (c), brain samples; (b) and (d). The regression line is given with its estimated standard errors below the equation.

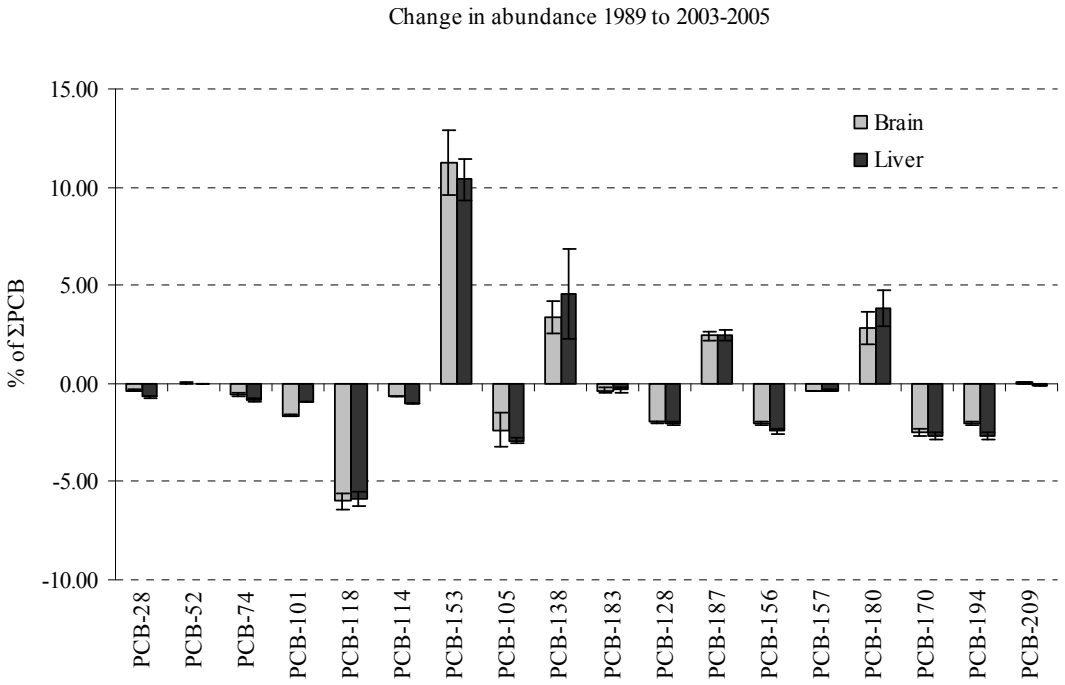


Figure 4: The mean lipid wt. concentration changes (+ standard error) in percentage of $\Sigma PCB_{18}$ for individual PCB congeners in brain and liver samples from glaucous gulls found dead or dying at Bjørnøya and Spitsbergen in 1989 (n = 11) and 2003-2005 (n = 21). Data for 1989 from Gabrielsen et al. (1995).