

ACCUMULATION AND TRANSFER OF CONTAMINANTS IN KILLER WHALES
(*ORCINUS ORCA*) FROM NORWAY: INDICATIONS FOR
CONTAMINANT METABOLISM

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Abstract—Blubber tissue of one subadult and eight male adult killer whales was sampled in Northern Norway in order to assess the degree and type of contaminant exposure and transfer in the herring–killer whale link of the marine food web. A comprehensive selection of contaminants was targeted, with special attention to toxaphenes and polybrominated diphenyl ethers (PBDEs). In addition to assessing exposure and food chain transfer, selective accumulation and metabolism issues also were addressed. Average total polychlorinated biphenyl (PCB) and pesticide levels were similar, approximately 25 $\mu\text{g/g}$ lipid, and PBDEs were approximately 0.5 $\mu\text{g/g}$. This makes killer whales one of the most polluted arctic animals, with levels exceeding those in polar bears. Comparing the contamination of the killer whale's diet with the diet of high-arctic species such as white whales reveals six to more than 20 times higher levels in the killer whale diet. The difference in contaminant pattern between killer whales and their prey and the metabolic index calculated suggested that these cetaceans have a relatively high capacity to metabolize contaminants. Polychlorinated biphenyls, chlordanes, and dichlorodiphenyldichloro-ethylene (DDE) accumulate to some degree in killer whales, although toxaphenes and PBDEs might be partly broken down.

Keywords—Killer whale Polychlorinated biphenyls Pesticides Polybrominated diphenyl ethers Accumulation

INTRODUCTION

Halogenated organic contaminants (HOCs), like polychlorinated biphenyls (PCBs) and chlorinated pesticides, are a continuing threat to the health of animals as well as humans. These chemicals are lipophilic, persistent, and accumulate through the food chain. The marine environment is particularly vulnerable to HOCs because it acts as the final sink and consequently contains the major portion of these compounds [1]. Concern is growing about new chemicals in the environment, such as polybrominated diphenyl ethers (PBDEs). These compounds are used to make consumer products more fire safe, but until recently, they appeared to double their concentration in the environment every five years ([2–4]; for reference 4, see [http://amap.no/documents/index.cfm?dirsub=%2FAMAP Assessment 2002](http://amap.no/documents/index.cfm?dirsub=%2FAMAP%20Assessment%202002)—Persistent Organic Pollutants in the Arctic&sort=default).

Marine predators, such as seals and whales, are exposed to relatively high HOC concentrations. Several studies have indicated that this may result in adverse effects, specifically targeting the endocrine and immune systems [5–9]. Killer whales (*Orcinus orca*) are widely distributed toothed cetaceans inhabiting all oceans [10]. They are capable of surviving on a variety of prey and the different subpopulations are adapted to feeding off the resources available to them in their specific home range. For example, several ecotypes of killer whales have been identified off the coasts of British Columbia and southern Alaska as residents and transients. They are genetically distinct and differ in various aspects of morphology,

vocalization patterns, habitat use, and diet [11]. Killer whales occur throughout the Northeast Atlantic; one of the populations in these waters is specialized in feeding on Norwegian spring-spawning herring (*Clupea harengus*). Although their exact geographic range is not known, these animals follow the seasonal migrations of the herring throughout their annual movements in the Norwegian and Barents Seas. Due to their continuous efforts to follow migrating herring, these whales are thought to feed predominantly on this fish species [12,13]. It has been estimated that over 1,500 killer whales might be present in Norwegian coastal waters when herring is abundant [13].

Norway is one of the few countries where killer whales have been hunted in recent decades, although there is no killer whale hunt at present. Yearly catch records of killer whales averaged 57 animals between 1938 and 1980 [14]. Overfishing drove the complete collapse of the Norwegian spring-spawning herring stock in the late 1960s and, in response, more than 700 whales were culled between 1969 and 1980 [14,15].

Killer whales accumulate relatively large amounts of contaminants [4,9,16]. Along North America's west coast they are highly polluted with PCBs, dioxins [9], and PBDEs as compared to other marine mammals, with levels exceeding 200 mg/kg for PCBs [16]. No data exist on pollution in killer whales from Norway. Therefore, the current study was initiated to assess the degree and type of contaminant exposure, accumulation, and transfer in the herring–killer whale link of the marine food web. Free-ranging animals were sampled using remote biopsy, allowing inference on the pollutant loads of a sample of apparently healthy animals. A comprehensive se-

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lection of contaminants was targeted, including PBDEs and toxaphenes. In addition to assessing exposure and food chain transfer, selective accumulation and metabolism issues were addressed.

MATERIALS AND METHODS

Sample collection

Blubber samples were collected from eight adult male killer whales that were identified by their large dorsal fins, and one juvenile in the Tysfjorden area, Norway, in fall 2002, using a biopsy system (Paxarms, Timaru, New Zealand) for small cetaceans with a biopsy point of 8 mm [17]. Animals were darted from a distance of approximately 10 to 15 m, and the resulting samples (0.5–1 g), originating from the first 2 to 4 cm of the outer blubber, were wrapped in aluminum foil and frozen at -20°C . Photographs to identify individual animals were collected during sampling [18].

Whole herring, collected from fishing vessels at three different locations within the Tysfjord/Vestfjord complex, were sampled; six individual herring were pooled per location and homogenized using a professional meat grinder. A subsample of approximately 10 g was used for contaminant analyses.

Chemical analyses

All chemicals were of pesticide grade and commercially available from Sigma (St. Louis, MO, USA) and Merck (Darmstadt, Germany). Whale blubber and fish samples were analyzed for PCBs (32 congeners), toxaphenes (6 congeners), dichlorodiphenyldichloro-ethylene (DDE), chlordanes, and PBDEs (10 congeners). Approximately 0.5 to 1 g of the samples was used and analyzed as described previously [19]. The detection limit was calculated at a signal-to-noise (S/N) ratio of 3 ($S/N > 3$) and depended on the amount of lipids extracted. A blank sample was run with each set of samples; no blank levels $> 10\%$ of the levels measured in the samples were observed in the five laboratory blanks. In addition 10 in-house reference material samples (human adipose tissue or serum) were run with satisfactory results (relative standard deviation $< 15\%$). The laboratory has taken part in both the Arctic Monitoring and Assessment Programme 2002 (Institut national de santé publique du Québec) and the Bromine and Science Environmental Forum/QUASIMEME 2002 (<http://www.quasimeme.org>) interlaboratory comparison studies with good results Z -scores < 2 .

Data management

Based on the chlorine substitution pattern on the *ortho* (*o*), *meta* (*m*), and *para* (*p*) positions, four groups of PCB congeners, with a different resistance towards metabolic breakdown (metabolic groups), were assigned [20–22]. Group I comprised congeners with no vicinal H-atoms in *o,m* or *m,p* positions, considered persistent. Group II comprised congeners with vicinal *m,p* H-atoms and 0–1 *o*-Cl (IIA), 2 *o*-Cl (IIB), or 3 *o*-Cl (IIC), considered to be metabolized by cytochrome P450 (CYP) 2B/3A and to a lesser extent (if a maximum of 1 *o*-Cl is present) CYP1A enzymes. Group IIIA comprised congeners with no vicinal H-atoms at *m,p* positions and a maximum of 1 *o*-Cl, considered to be metabolized by CYP1A. Group IIIB comprised congeners with no vicinal H-atoms at *m,p* positions and 2 or more *o*-Cl, considered persistent.

Geometric means and 95% confidence intervals were calculated for concentrations of individual contaminants. The relative contribution of metabolic groups (PCBs) or individual

compounds (pesticides) to the main contaminant groups, as well as differences in contaminant pattern between the whales and their food, was calculated.

Multivariate data visualization techniques were used to explore the manner in which the proportion of contaminant mixtures differed between killer whales and herring. The mixtures occurring in whale and herring were examined using nonmetric multidimensional scaling [23] on row-standardized data. Proportions were calculated separately for each row (i.e., for each whale or fish sample) [24]. All analyses used Bray-Curtis dissimilarity matrices. Results of the nonmetric multidimensional scaling were centered and then rotated so that variance was maximized along the primary axis. Groups of PCBs (metabolic groups), toxaphenes, PBDEs, and chlordanes were analyzed separately. The PCB congeners were grouped as described above.

The relative contribution of each compound, expressed as the ratio contaminant/PCB 153, R_{153} , was calculated for the whales ($R_{153\text{predator}}$) as well as in their main prey items (i.e., herring, $R_{153\text{prey}}$). The relative metabolic index ([MI], $R_{153\text{predator}}/R_{153\text{prey}}$) [21,24] was calculated. The MI expresses the presence of individual compounds in predators versus prey relative to PCB 153. Values above 1 indicated that a compound accumulates more than PCB 153, and values below 1 indicated less accumulation than PCB 153.

Indices were analyzed using linear models [25]. Pesticides (toxaphenes, chlordanes), PCBs, and PBDEs were analyzed separately. The MI values were square-root transformed for analyses. Fixed effects were species, contaminant congeners, and their interaction. Individual samples (of whales and fish) were treated as nested within species. The relative influence of fixed effects was tested using model selection [25].

Data analyses were carried out using libraries: Modern Applied Statistics with S-plus Ver 7.2 to 24 [25], Lattice Ver 0.12 to 11 [26], and Vegan Ver 1.6 to 10 [27] of R 2.2.1 (R Development Core Team 2005; <http://www.R-project.org> and <http://cc.oulu.fi/~jarioksa/>) running under Microsoft Windows XP.

RESULTS

Animals' ages

Good identification photographs were collected for five of the eight adult males sampled. Long-term photo records showed that these whales were at least 25 years of age. The three unidentified males were adults, but their approximate age is uncertain. The one juvenile animal sampled must have been less than six years of age.

Contaminant concentrations

The PCB blubber concentrations ranged between 16 and 44 $\mu\text{g/g}$ lipid (lower and upper level, respectively of 95% confidence interval) in the killer whales, although PCB concentrations in herring were approximately 1,000 times lower on a wet weight basis and approximately 100 times lower on a lipid weight basis (Table 1). Ninety-five percent confidence intervals (lower and upper level, respectively) for DDE, chlordanes, and toxaphenes were between approximately 7 and 20, 4 and 10, and 6 and 12 $\mu\text{g/g}$ lipid, respectively, in the whales with levels in their food substantially lower (Table 2). In contrast to PCBs and pesticides, blubber PBDE concentrations were relatively low in killer whales; the 95% confidence interval (lower and upper level, respectively) was between 350

Table 1. Geometric mean polychlorinated biphenyl (PCB) concentrations (ng/g lipid) and 95% confidence limits in blubber of adult male killer whales (KW) and their main food, herring (ng/g wet wt). Average lipid content of herring = 12.3%

PCB group ^b	28 IIIA	52 IIB	66 IIIA	74 IIIA	99 IIIB	101 IIB	105 IIIA	110 IIB	118 IIIA	128 IIIB	138 IIIB	141 IIB
KW												
Mean	23.1	2,036	113	113	1,053	857	391	78	1,430	180	6,830	ND ^c
95% Low	4.2	1,263	77.1	83.2	622	614	290	48	1,062	117	4,109	—
95% High	128.7	2,181	166	155	1,783	1,198	526	126	1,926	278	11,354	—
Herring												
Mean	0.51	1.7	1.8	0.5	1.2	1.8	0.70	1.4	1.7	0.5	3.0	0.26
95% Low	0.39	1.4	1.6	0.3	1.0	1.4	0.50	1.1	1.2	0.4	2.4	0.2
95% High	0.66	1.9	2.0	0.7	1.6	2.3	0.90	1.8	2.4	0.6	3.7	0.3
MI ^d	<0.01	0.5 ± 0.1	<0.01	0.1 ± 0.0	0.3 ± 0.0	0.2 ± 0.1	0.2 ± 0.1	<0.01	0.3 ± 0.1	0.1 ± 0.0	0.8 ± 0.1	<0.01

^a Sum of PCBs 195, 196, 197, 199, 201, 202, and 206–209. The metabolic groups are based on the chlorine substitution pattern of the PCB congener.

^b Group I = no vicinal H-atoms in *o,m* or *m,p* positions; group II = congeners with vicinal *m,p* H-atoms; group IIIA = congeners with no vicinal H-atoms at *m,p* positions and a maximum of 1 *o*-Cl; group IIIB = congeners with no vicinal H-atoms at *m,p* positions and 2 or more *o*-Cl.

^c ND = nondetectable.

^d Metabolic index (MI) is calculated as the ratio of contaminant *x*/PCB 153, R_{153} , in predator and prey (i.e., $R_{153\text{predator}}/R_{153\text{prey}}$).

and 650 ng/g lipid, with levels in their food substantially lower (Table 3).

Contaminant patterns

Toxaphene2, *cis*-chlordane, and PBDE 138 were removed from data matrices, because these compounds were undetectable or occurred only in very low amounts in most samples. Shepard plots (not shown) [25] indicated all nonmetric multidimensional scaling ordinations were good representations of the data.

PCB pattern

Average proportion of metabolic PCB groups showed that persistent PCBs from groups I and IIIB contributed over 80% of total PCBs in killer whales and less than 45% in herring (Fig. 1a). In contrast, less persistent PCBs from groups II and IIIA were proportionally less present in whales as compared to herring (~20 vs 50% of total PCBs; Fig. 1a). Biplots of PCB patterns in killer whales and herring confirmed this difference and showed a clear separation between herring and killer whales, with a tendency for higher proportions of PCB groups I and IIIB in killer whales and higher proportions of PCB groups IIB, IIC, and IIIA in herring (Fig. 2a).

Chlordane pattern

Average proportions of chlordanes showed that *trans*-nonachlor contributed almost 80% to total chlordanes in killer whales and approximately 46% in herring. In contrast, *trans*-chlordane and *cis*-nonachlor constituted on average less than 4% each of total chlordane levels in killer whales, although in herring these compounds comprised 40 and 10%, respectively, of total chlordanes (Fig. 1b). Biplots of chlordane patterns in killer whale and herring showed a clear separation between these species, with higher proportions of *trans*-nonachlor and heptachlor epoxide in the whales (Fig. 2b).

Toxaphene pattern

Toxaphenes 26 and 50 were the only toxaphene congeners that contributed more to total toxaphenes in whales than in herring; together they made up approximately 55% in whales, versus 40% in herring. The other toxaphenes were relatively more present in herring (Fig. 1c). The biplot shows a clear

separation (except for one killer whale that is clearly an outlier) between killer whale and herring, with a tendency for the proportion of toxaphene 26 and 50 to be higher in killer whales, and toxaphene 40/41, 44, and 62 show higher proportions in herring (Fig. 2c).

PBDE pattern

Mean higher proportions of PBDE 99, 100, and 154 were found in killer whale (40%) as compared to herring (15%; Fig. 1d). The biplot shows a separation between killer whales and herring for the PBDE pattern, although there is more variation along the first dimension when compared to the other contaminants. Proportions of PBDE 47 and 28 tended to be higher in herring, although PBDE 99, 100, 153, and 154 tended to be higher in the killer whales (Fig. 2d).

Percentage of PCB 153 in killer whale and herring

Model selection demonstrated that, for all pollutants, models that incorporated an interaction term represented the data best. That is, the proportions of congeners of organic pollutants (relative to PCB153) present in samples differed significantly between samples from whales and from herring.

The percentage of PCB 153 for most metabolic group I PCBs differed only slightly in herring and killer whales (Fig. 3a) and, consequently, the MI values were close to 1 (Table 1). In contrast, PCB groups II and IIIA showed a different pattern: The percentage of PCB 153 for individual congeners showed higher values in herring as compared to whales for almost all compounds (Fig. 3a), resulting in MI values lower than 1 (Table 1). Polychlorinated biphenyl group IIIB generally showed somewhat higher values for herring than for the whales, and the MI was around 0.5 on average (Table 1).

The pesticides showed a pattern similar to PCB groups II and IIIA: Almost all compounds showed a higher percentage of PCB 153 in herring than in killer whales (Fig. 3b), resulting in MIs well below 1 (Table 2). *trans*-Nonachlor and heptachlor epoxide showed MIs close to or above 1. The MI for all toxaphenes was well below 1, yet toxaphene 26 and 50 showed the highest values (Table 2).

Most PBDEs showed a lower percentage of PCB 153 in whales than in herring (Fig. 3c), resulting in MIs well below 1, with highest values for PBDE 99 (Table 3).

Table 1. Extended

153 I	156 IIIA	157 IIIA	170 IIIB	172 IIIB	174 IIC	177 IIIB	180 I	183 I	187 I	194 I	Σ ^a I	Σ
6,766	51.2	25.3	538	102	202	223	2,358	658	1,720	294	452	26,940
4,020	8.0	4.5	318	61.1	119	134	1,409	393	1,047	178	257	16,613
11,388	329	143.7	911	169	342	371	3,943	1,100	2,823	485	795	43,687
2.5	0.14	0.0	0.31	0.06	0.05	0.3	0.71	0.28	0.79	0.05	0.18	21.0
1.7	0.10	0.0	0.30	0.05	0.01	0.2	0.60	0.20	0.70	0.04	0.15	17.4
3.8	0.20	0.0	0.40	0.07	1.7	0.4	0.80	0.40	0.90	0.07	0.23	25.5
1.0 ± 0.0	0.3 ± 0.2	0.3 ± 0.2	0.6 ± 0.1	0.6 ± 0.2	1.5 ± 0.3	0.3 ± 0.0	1.3 ± 0.2	0.9 ± 0.1	0.8 ± 0.1	2.2 ± 0.6	—	—

DISCUSSION

The current study is the first to report on contaminant-related issues in Norwegian killer whales. In addition, to our knowledge, this is the first study on killer whales addressing congener-specific accumulation, prey to predator transfer, and metabolism of a comprehensive selection of contaminants. Studies on the diet of Norwegian killer whales have shown that, besides herring, other prey items such as other fish species and sea birds occasionally are taken as well [12]. Because the presence of these killer whales is closely associated with the presence of herring [12,13], it is conceivable that herring constitutes the major part of their diet. Therefore, Norwegian killer whales offer a rare opportunity to study contaminant transfer from prey to predator as well as contaminant metabolism, expressed as MI. However, because no history of environmental exposure during the killer whale's lifetime is available, we are

dealing with a contaminant burden from the past that is hard to quantify and will influence the MI values to some (unknown) degree.

Compared to seals and other cetaceans, levels of all compounds measured were particularly high in the killer whales. Harbor seals (*Phoca vitulina*) from the same area, sampled in the late eighties [28], showed 6, 8, and 12 times lower levels of PCB 153, DDE, and *trans*-nonachlor, i.e., approximately 1,000, 1,450, and 450 ng/g lipid, respectively, in spite of declining PCB and pesticide concentrations in the Arctic over the last decades [4]. Likewise, also arctic marine mammals generally showed lower levels than the killer whales. Adult male white whales (*Delphinapterus leucas*) from Svalbard also showed two to five times lower concentrations for all major contaminants: Sum PCBs, DDE, and sum chlordanes were 5,108, 3,915, and 2,872 ng/g lipid, respectively [29]. Polar bears from Svalbard notoriously are polluted; animals sampled in the mid-nineties showed similar PCB 153 levels, but 50 to 100 times lower levels of pesticides and 10 times lower levels of PBDE 47 [30,31] than the killer whales from the current study. Also the particularly polluted marine mammals from the Gulf of St. Lawrence, Canada, showed generally lower levels than the killer whales from the present study [32–34]. Stranded white whales sampled in the 1990s [34] showed approximately half of the toxaphene levels, and animals live-sampled in the late 1990s showed approximately 4 times lower chlordanes, 2.5 times lower DDE, and 2.5 times lower PCB concentrations than the killer whales from the present study [33].

Overall, contaminant levels in Norwegian killer whales are currently among the highest recorded for any arctic marine mammal species. This most likely is linked to their main food source, herring, which showed 10 to 15 times higher HOC levels than, for example, polar cod from Svalbard [19], the dominant food from the white whales.

Only killer whales from the Northeast Pacific showed comparable or higher HOC levels. Northern residents, a group of killer whales mainly feeding on relatively polluted Pacific salmon, sampled in the mid-nineties, showed similar PCB levels as the Norwegian whales [9]. On the other hand, transient killer whales, mainly feeding on other marine mammals [9], showed approximately seven times higher PCB levels [9].

The relatively low PBDE levels in Norwegian versus North American (Pacific) killer whales may indicate that these compounds are more prevalent in the Pacific marine system than in the Northeast Atlantic. This is consistent with fewer re-

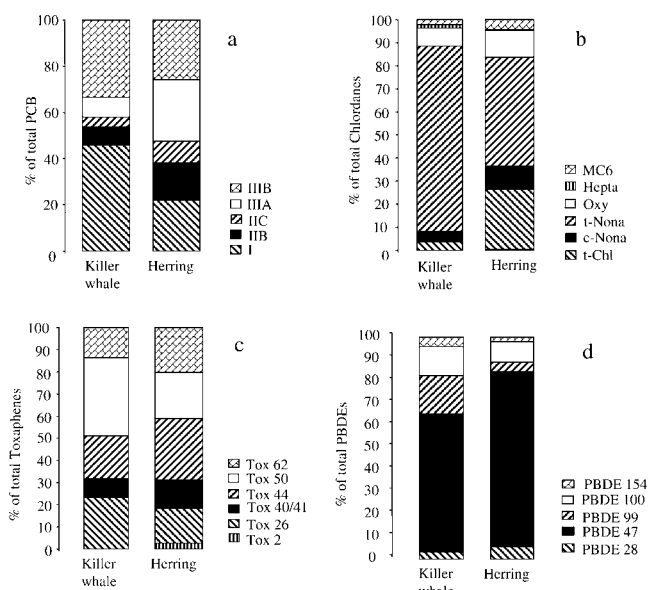


Fig. 1. Contaminant patterns in killer whale and herring for different contaminant groups. (a) Polychlorinated biphenyl (PCB) groups, as a percentage of total PCBs. Group I: No vicinal H-atoms in *o,m* or *m,p* positions; group II: Vicinal *m,p* H-atoms and 0-3 *o*-Cl's; group IIIA: No vicinal H-atoms at *m,p* positions, and a maximum of 1 *o*-Cl; group IIIB: No vicinal H-atoms at *m,p* positions, and 2 or more *o*-Cl's. (b) Chlordane pattern for *trans*-chlordane (t-chl), *cis*- and *trans*-nonachlor (c-nona and t-nona), oxychlordanes (oxy), heptachlorepoxyde (hepta), and MC6. (c) Toxaphene (Tox) pattern for congeners 2, 26, 40/41, 44, 50, and 62. (d) Polybrominated diphenyl ether (PBDE) pattern for congeners 28, 47, 99, 100, and 154.

Table 2. Geometric mean pesticide concentrations (ng/g lipid) and 95% confidence limits in blubber of adult male killer whales (KW) and their main food, herring (ng/g wet wt). Average lipid content of herring = 12.3%; average lipid content of herring = 12.3%

Pesticide ^a	c-chl	t-chl	c-nona	t-nona	oxy	hepta	MC6	Σ chl
KW								
Mean	ND ^b	250	282	5,207	515	87.8	138	6,565
95% Low	ND	165	137	3,395	340	51.2	90.2	4,281
95% High	ND	378	583	7,986	781	151	212	10,069
Herring								
Mean	ND	0.6	2.8	0.7	0.02	0.2	6.0	8.8
95% Low	ND	0.4	2.1	0.6	0.00	0.2	4.5	1.6
95% High	ND	0.9	3.6	0.8	0.32	0.3	7.9	46.8
MI ^c	—	0.06 ± 0.02	0.24 ± 0.17	0.72 ± 0.18	0.29 ± 0.10	1.82 ± 0.87	0.22 ± 0.06	—

^a c = *cis*; t = *trans*; chl = chlordane; nona = nonachlor; oxy = oxychlordane; hepta = heptachlor epoxide; Σ chl = total chlordane; DDE = dichlorodiphenyldichloro-ethylene; Tox. = toxaphene congener (Parlar 26, 32, 40, 44, 50); Σ Tox. = total toxaphene levels; Σ Pest. = total pesticide levels.

^b ND = nondetectable.

^c Metabolic index (MI) is calculated as the ratio of contaminant *x*/PCB 153, R_{153} , in predator and prey (i.e., $R_{153\text{predator}}/R_{153\text{prey}}$).

restrictions on use of these compounds in North America as compared to Europe.

Harbor porpoises (*Phocoena phocoena*) stranded on the Belgium coast on average showed approximately 2,000 ng/g lipid PBDEs [35], i.e., approximately 4 times higher as compared to killer whales from the present study. We have to keep in mind though that levels in stranded animals may not be representative for the population due to possible associated diseases and emaciation, and a consequent increase in contaminant levels, in stranded specimens. In addition, liver and blubber might contain different PBDE levels and as such might not be comparable. In a different study on stranded harbor porpoises as well as individuals accidentally caught in gill nets

in European waters, differences in body condition were accounted for and revealed PBDE levels in blubber of approximately 170 ng/g lipid [36], approximately a third of that in killer whales.

The lower relative presence of some compounds in killer whales versus herring, quantified as MI, may indicate contaminant metabolism [20–22,31]. However, although killer whales may feed on additional prey items, for the MI calculations we assumed a 100% herring diet. The MI has the important advantage over the more commonly used biomagnification factor in that it is not influenced by fluctuating HOC concentrations, for example due to fluctuations in fat content, in an individual. In addition, a more meaningful biomagnification factor should

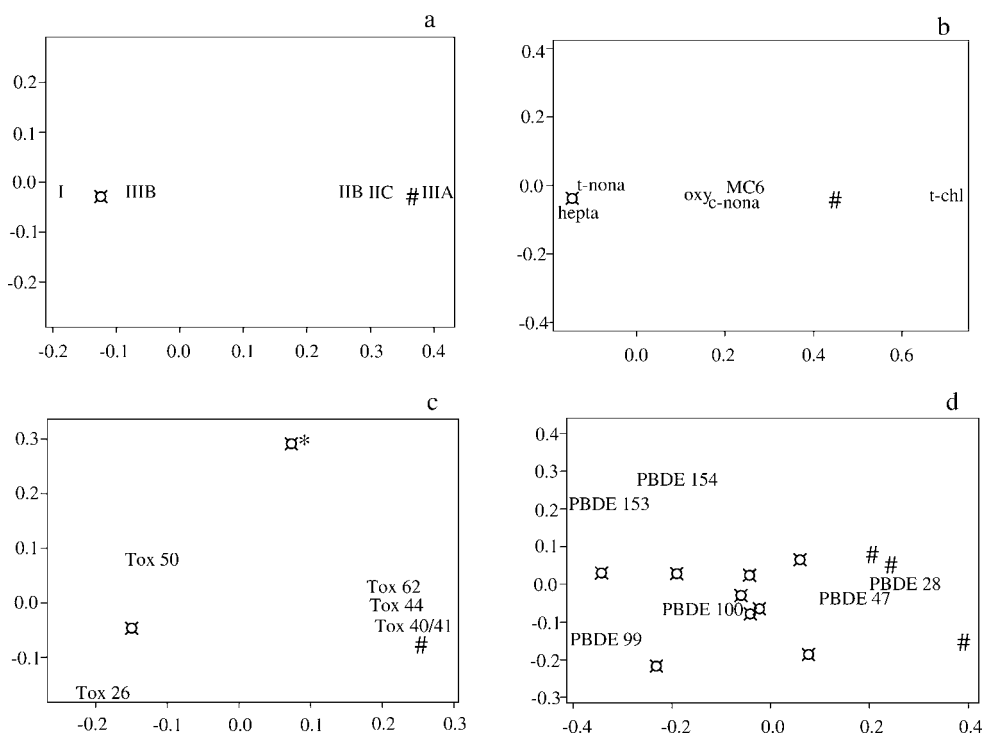


Fig. 2. Nonmetric multidimensional scaling (NMDS) plots showing differences in contaminant patterns between killer whales and herring. (a) Polychlorinated biphenyl (PCB) groups, expressed as a percentage of total PCBs. Group I: no vicinal H-atoms in *o,m* or *m,p* positions; group II: vicinal *m,p* H-atoms and 0–3 *o*-Cl; group IIIA: no vicinal H-atoms at *m,p* positions and a maximum of 1 *o*-Cl; group IIIB: PCBs with no vicinal H-atoms at *m,p* positions and 2 or more *o*-Cl. (b) Chlordane pattern for *trans*-chlordane (t-chl), *cis*- and *trans*-nonachlor (c-nona and t-nona), oxychlordane (oxy), heptachlorepoide (hepta), and MC6. (c) Toxaphene pattern for congeners 2, 26, 40/41, 44, 50, and 62. (d) Polybrominated diphenyl ether (PBDE) pattern for congeners 28, 47, 99, 100, and 154. # = herring; α = killer whales; α* = outlier killer whale.

Table 2. Extended

<i>p,p'</i> -DDE	Tox. 2	Tox. 26	Tox. 40/41	Tox. 44	Tox. 50	Tox. 62	Σ Tox.	Σ Pest.
11,652	5.4	1,857	669	1,532	2,803	1,069	8,206	26,951
6,966	3.2	1,096	467	1,050	1,969	784	5,728	17,573
19,490	9.3	3,144	958	2,236	3,991	1,458	11,756	41,333
8.8	0.6	3.4	2.7	6.0	4.4	4.3	21.6	37.1
7.4	0.4	1.5	1.6	2.8	2.1	1.7	10.3	19.7
10.4	0.9	7.4	4.5	12.6	9.2	10.8	45.0	69.9
0.50 ± 0.07	0.01 ± 0.01	0.23 ± 0.08	0.11 ± 0.07	0.11 ± 0.07	0.25 ± 0.08	0.11 ± 0.06	—	—

not be calculated on lipid-based concentrations in individual tissues, but it should be based on the total amount of pollutant per unit of body mass, i.e., whole herring and whole killer whale, as demonstrated before [37]. If calculated based on whole animals, biomagnification factor and MI have been shown to be very similar [37]. Yet, like the biomagnification factor, MI potentially might be biased due to individual differences in diet or if dietary components are present that have not been accounted for. Nevertheless, due to their close geographic association with the herring schools year-round, it is widely assumed that by far the main part of the killer whales' diet consists of herring [12,13]. In addition, comparing the contaminant levels obtained in adult male beluga whales [29] with those in the killer whales showed that the levels in killer whales are plausible in the sense that the assumed dominant diet of killer whales (i.e., herring) is approximately 10 to 15 times more polluted than the dominant diet of beluga whales (i.e., polar cod), resulting in killer whales being two to five times more polluted than beluga whales. Nevertheless, to get a better understanding of possible additional food items in killer whales, future trophic level studies will be carried out. Generally, MI values corresponded well with the general persistence of most HOCs. Both contaminant patterns and MI values indicated accumulation of PCB groups I and IIIB and metabolism of PCB groups II and IIIA in whales. Compared to white whales from Svalbard [19], the MIs in killer whales suggested a similar accumulation for persistent PCB from group I and IIIB, i.e., MI was approximately one in both species, indicating accumulation similar to PCB 153.

In contrast, PCBs from groups II and IIIA showed substantially lower MIs in killer whales than white whales, in-

dicating a higher ability of killer whales to metabolize these compounds. Cetaceans in general are thought to have a lower capability to metabolize specifically group II PCBs [38,39]. Metabolism of these PCBs involves CYP 2B/3A enzymes [20,21], but both the PCB patterns as well as the MIs indicate that killer whales might be more capable of metabolizing these compounds than other cetaceans.

Like PCBs, pesticides showed indications for metabolism in killer whales. Differences in HOC patterns between herring and whales and low MIs suggested that most chlordanes, except *trans*-nonachlor, can be metabolized in killer whales. Similar to most chlordanes, the MI below 1 for DDE indicates that this DDT metabolite might be broken down to some degree in killer whales, a similar result as was found in white whales [19].

Metabolism of most toxaphenes was suggested by lower proportions in killer whales as compared to herring and relatively low MIs for most congeners in killer whales. Overall, MIs were lower in killer whales than in white whales, suggesting a higher ability to metabolize chlordanes than white whales.

Similar to the pesticides, the overall low MIs for most PBDEs suggested low bioaccumulation of these compounds in killer whales. Only PBDE 99, with an MI around 0.6, might have some bioaccumulative potential. In contrast, white whales showed MI values around or above 1 for PBDE 47, 99, and 100 [31]. Similar to polar bears [31], killer whales seem to be able to metabolize most PBDEs.

CONCLUSION

Killer whales hold the gloomy record of most-polluted European arctic mammal. At the same time, they seem to have

Table 3. Geometric mean concentrations (ng/g lipid) and 95% confidence limits of polybrominated diphenyl ethers (PBDEs) in blubber of killer whales (KW) and their main food, herring (pg/g wet wt), from Norway. MI = metabolic index (mean ± SD) for PBDEs in killer whales. ND = nondetectable; average lipid content of herring = 12.3%

PBDE	Tetra-Br			Penta-Br			Hexa-Br		Hepta-Br		Σ PBDE
	28	47	66	85	99	100	138	153	154	183	
KW											
Mean	14.3	275	ND	ND	76.1	58.7	ND	1.5	18.0	ND	475
95% Low	10.9	204	ND	ND	53.8	41.2	ND	0.2	3.4	ND	348
95% High	18.6	370	ND	ND	107.6	83.6	ND	12.7	96.2	ND	647
Herring											
Mean	77.0	1,087	1.2	1.2	56.6	127.4	0.0	1.5	18.0	ND	1,484
95% Low	65.9	925.2	1.1	1.1	47.6	114.0	0.0	0.2	3.4	ND	1,184
95% High	89.9	1,277	1.4	1.4	67.3	142.5	0.1	12.7	96.2	ND	1,861
MI ^a	0.09 ± 0.05	0.12 ± 0.07	—	—	0.59 ± 0.31	0.20 ± 0.10	—	0.29 ± 0.29	0.13 ± 0.10	—	—

^a Metabolic index (MI) is calculated as the ratio of contaminant *x*/PCB 153, R_{153} , in predator and prey (i.e., $R_{153\text{predator}}/R_{153\text{prey}}$).

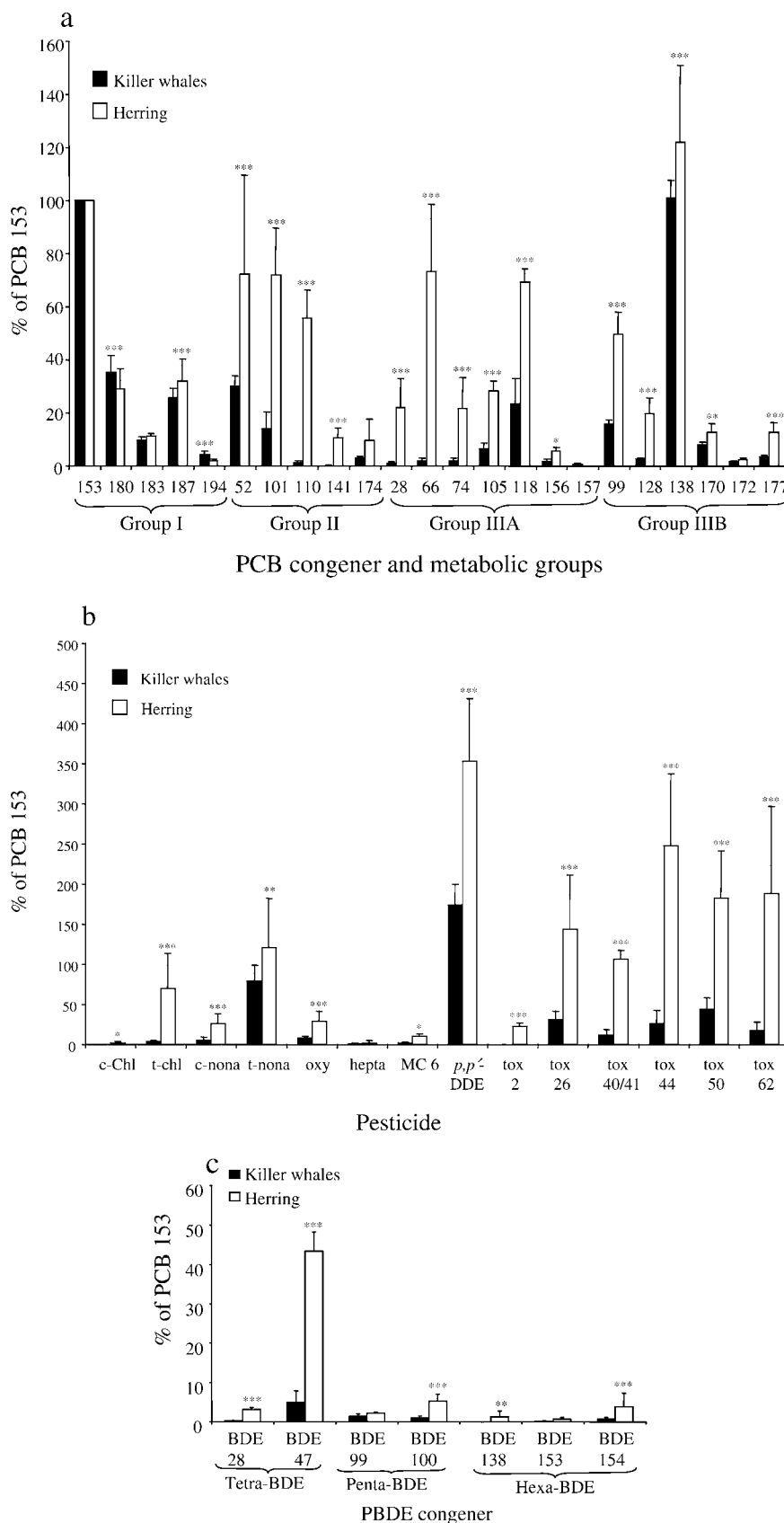


Fig. 3. (a) Congener-specific polychlorinated biphenyl (PCB) pattern, expressed as a percentage of PCB 153, in killer whale and herring. Group I: no vicinal H-atoms in *o,m* or *m,p* positions; group II: vicinal *m,p* H-atoms and 0-3 *o*-Cl; group IIIA: no vicinal H-atoms at *m,p* positions and a maximum of 1 *o*-Cl; group IIIB: no vicinal H-atoms at *m,p* positions and 2 or more *o*-Cl. (b) Congener-specific pesticide pattern, expressed as a percentage of PCB 153, in killer whale and herring. t-chl = *trans*-chlordan; c-nona and t-nona = *cis*- and *trans*-nonachlor; oxy = oxychlorane; hepta = heptachlorepoxide (hepta). (c) Congener-specific polybrominated diphenyl ether (PBDE) pattern, expressed as a percentage of PCB 153, in killer whale and herring. (a,b,c) * = difference between killer whale and herring significant at $p < 0.05$; ** = difference between killer whale and herring significant at $p < 0.01$; *** = difference between killer whale and herring significant at $p < 0.001$.

a substantial capacity to metabolize a wide range of contaminants, as indicated by the change in contaminant pattern as well as MI. These results indicate that PCBs, chlordanes, and DDE accumulate to some degree in killer whales, and toxaphenes and PBDEs might be partly broken down. This is an unexpected result because other cetaceans, such as white whales and several species of dolphins, showed a much lower capability to metabolize contaminants. Comparing the contamination of the killer whale's diet with the diet of high-arctic species such as white whales, reveals six to more than 20 times higher levels in the killer whale diet. Consequently, levels in killer whales are between five and eight times higher than in white whales. Despite the killer whale's capability of metabolizing a wide range of compounds, the contamination of their food source results in record-high pollution levels.

SUPPORTING INFORMATION

Table S1. Details of the nested linear models to assess if the contaminant pattern (proportions relative to polychlorinated biphenyl [PCB] 153) in killer whales differs from that in herring. Estimate is the coefficient estimated by the model. Negative values indicate that proportion in whales were lower than in herring, positive values indicate the opposite. Probabilities are given in the final column of each table. *** is $p < 0.001$; ** is $p < 0.01$; * is $p < 0.05$.

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