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PCBs and OH-PCBs in polar bear mother–cub pairs: A comparative study based on plasma levels in 1998 and 2008

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ABSTRACT

The aim of this study was to examine the plasma concentrations and prevalence of polychlorinated biphenyls (PCBs) and hydroxylated PCB-metabolites (OH-PCBs) in polar bear (*Ursus maritimus*) mothers ($n=26$) and their 4 months old cubs-of-the-year ($n=38$) from Svalbard to gain insight into the mother–cub transfer, biotransformation and to evaluate the health risk associated with the exposure to these contaminants. As samplings were performed in 1997/1998 and 2008, we further investigated the differences in levels and pattern of PCBs between the two sampling years. The plasma concentrations of Σ_{21}PCBs (1997/1998: 5710 ± 3090 ng/g lipid weight [lw], 2008: 2560 ± 1500 ng/g lw) and Σ_6OH-PCBs (1997/1998: 228 ± 60 ng/g wet weight [ww], 2008: 80 ± 38 ng/g ww) in mothers were significantly lower in 2008 compared to in 1997/1998. In cubs, the plasma concentrations of Σ_{21}PCBs (1997/1998: 14680 ± 5350 ng/g lw, 2008: 6070 ± 2590 ng/g lw) and Σ_6OH-PCBs (1997/1998: 98 ± 23 ng/g ww, 2008: 49 ± 21 ng/g ww) were also significantly lower in 2008 than in 1997/1998. Σ_{21}PCBs in cubs was 2.7 ± 0.7 times higher than in their mothers. This is due to a significant maternal transfer of these contaminants. In contrast, Σ_6OH-PCBs in cubs were approximately 0.53 ± 0.16 times the concentration in their mothers. This indicates a lower maternal transfer of OH-PCBs compared to PCBs. The majority of the metabolite/precursor-ratios were lower in cubs compared to mothers. This may indicate that cubs have a lower endogenous capacity to biotransform PCBs to OH-PCBs than polar bear mothers. Exposure to PCBs and OH-PCBs is a potential health risk for polar bears, and the levels of PCBs and OH-PCBs in cubs from 2008 were still above levels associated with health effects in humans and wildlife.

1. Introduction

Polychlorinated biphenyls (PCBs) are still among the dominating persistent organic pollutants (POPs) in arctic mammals (Letcher et al., 2010), and the levels of PCBs in polar bears (*Ursus maritimus*) are among the highest reported in any species (e.g. Norstrom et al., 1998; Andersen et al., 2001; Dietz et al., 2004; Verreault et al., 2005a; Letcher et al., 2010; McKinney et al., 2011). In polar bears, PCBs and other POPs are suggested to cause adverse effects on the thyroid hormone system, sex steroid homeostasis, vitamin status, immune system, organ morphology and behaviour (e.g. Wiig et al., 1998; Skaare et al., 2001; Haave et al., 2003; Olsen et al., 2003; Oskam et al., 2003, 2004; Braathen et al., 2004; Lie et al., 2004, 2005; Sonne, 2010).

Adult polar bears have a well developed cytochrome P450 (CYP) enzyme system, which can metabolically biotransform PCBs to PCB-metabolites, and hydroxylated PCBs (OH-PCBs) are among the most common PCB-metabolites in polar bears (Letcher et al., 2000; Verreault et al., 2005b; Gebbink et al., 2008; Letcher et al., 2010). OH-PCBs are less hydrophobic than their parent compounds, and these metabolites bind to proteins and accumulate in blood rather than being associated with lipids as PCBs are (van den Berg, 1990; Lans et al., 1993, 1994). In adult polar bears from Svalbard (Norway), East-Greenland and Resolute Bay in the Canadian Arctic, the levels of OH-PCBs in plasma and whole blood have been reported to be even higher than the levels of PCBs in the same tissue (Sandau, 2000;Sandau et al., 2000; Sandala et al., 2004; Verreault et al., 2005b; Gebbink et al., 2008). The OH-PCBs detected in significant concentrations in polar bears and other mammals, including humans, are *meta-* or *para*-hydroxylated PCBs (Letcher et al., 2000). OH-PCBs have structural similarities to endogenous compounds, and *in vivo* and *in vitro* studies have demonstrated that OH-PCBs may disrupt the transport and

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metabolism of thyroid hormones, vitamin homeostasis, the oestrogen cycle, interrupt intercellular communication, and development of the nervous system (Brouwer and van den Berg, 1986; Rickenbacher et al., 1986; Lans et al., 1993; Brouwer et al., 1998; Schuur et al., 1998, 1999; Cheek, 1999; Kester et al., 2000; Machala et al., 2003, 2004; Meerts et al., 2004; Ptak et al., 2005; Gutleb et al., 2010).

In mammals, PCBs are transferred from mothers to their offspring through the umbilical cord and via the milk (Guvenius et al., 2003; Sørmo et al., 2003a; Greig et al., 2007; Park et al., 2008). The milk of polar bears is lipid-rich and contains high levels of hydrophobic contaminants such as PCBs (Bernhoft et al., 1997; Polischuk et al., 1995, 2002). Levels of PCBs in polar bear milk are higher than in the adult diet and the PCB levels in suckling cubs exceed the levels in their mothers (Bernhoft et al., 1997; Sandau, 2000; Polischuk et al., 2002). Contaminant levels in umbilical blood and the foetuses of polar bears are unreported. Because the polar bear foetuses are small and have minor fat reservoirs (Blix and Lentfer, 1979) for storage of hydrophobic POPs, the POPs entering their body may be more bioavailable to exert toxic effects. Hence, developing polar bears may be of particular risk from harmful effects of these pollutants (Bernhoft et al., 1997; Wiig et al., 1998; Polischuk et al., 2002). Exposure to POPs during critical developmental periods has been associated with negative health effects in wildlife, experimental animals, and humans (Colborn et al., 1993; Peterson et al., 1993; Brouwer et al., 1995, 1998; Huisman et al., 1995; Ulbrich and Stahlmann, 2004; Grandjean and Landrigan, 2006).

Because of the important role of thyroid hormones in normal growth and development of mammals, concern has been expressed about the thyroid disruptive effects of PCBs and OH-PCBs in polar bear cubs (Jenssen, 2006). Furthermore, there are indications of reproductive effects and decreased cub survival in polar bears that may be linked to high concentrations of contaminants (Derocher et al., 2003). Human infants exposed to PCBs and OH-PCBs prenatally show lower birth weight, smaller head circumference and alterations in the thyroid hormone homeostasis (Fein et al., 1984; Rylander et al., 1996; Sandau et al., 2002), and exposed children show altered neural development, cognitive, motor and learning abilities (Schantz et al., 2003; Grandjean and Landrigan, 2006; Maervoet, 2007; Park et al., 2009; Roze et al., 2009). In a recent study, OH-PCBs have been associated with alterations in the thyroid hormone homeostasis in hooded seal (*Cystophora cristata*) pups from East-Greenland (Gabrielsen et al., 2011). There are, however, few reports on PCB and OH-PCB levels in cubs-of-the-year (Sandau, 2000; Polischuk et al., 2002) and there is a lack of knowledge on mother–cub transfer of these two groups of endocrine disrupting compounds.

Starting in the 1970s, the production and use of PCBs have gradually been banned in most countries, and following the ratification of the Stockholm Convention in 2004, production, use and release of PCBs have been globally banned (www.pops.int). Thus, the levels of PCBs in adult polar bears have declined at least since the 1990s (Braune et al., 2005; Verreault et al., 2005a; McKinney et al., 2011). However, there are no reports on changes in levels of OH-PCBs in neither adult polar bears or in cubs. Information on changes in the levels of these environmental pollutants in polar bear mothers and their offspring are important for assessing the risk of exposure, and to assess to which extent international regulatory treaties and national bans of production, use and release of environmental pollutants affect levels in arctic wildlife and ecosystems.

The objective of the study was to examine the levels and prevalence of PCBs, OH-PCBs in polar bears mother and their suckling cubs to gain insight into the mother–cub transfer, biotransformation and to evaluate the health risk associated with the exposure to these contaminants. Thus, contaminant levels were analysed in plasma samples from live-caught female polar bears from Svalbard and their cubs (~4 months old, i.e. cubs-of-the-year), shortly after den emergence. As samples were obtained in 1997/1998 and 2008, we further investigated the differences in levels and patterns of PCBs and OH-PCBs in mothers and cubs from the two sampling periods.

2. Material and methods

2.1. Field sampling

In April 1997, 1998 and 2008, blood samples were collected from 3 polar bear mothers with 5 cubs, 13 mothers with 17 cubs, and 10 mothers with 16 cubs, respectively, at Svalbard, Norway. Cubs were approximately 4 months old at sampling (i.e. cubs-of-the-year), and litters comprised of either one ($n=14$) or two cubs ($n=12$). Because the quantitative rates of change over time in contaminant levels in polar bears are slow (AMAP, 2004), bears sampled in 1997 and 1998 were pooled into one group hereafter termed “1998”. Thus, the bears were divided into 4 groups based on age and sampling year: Mothers 1998, Mothers 2008, Cubs 1998, and Cubs 2008. In 1998, animals were sampled at Hopen and Edgeøya, whereas in 2008 animals were sampled at Spitsbergen and Edgeøya (Fig. 1). Latitude and longitude co-ordinates were recorded for all bears (Table 1; Fig. 1).

Capture and handling procedures followed standard protocols (Stirling et al., 1989; Derocher and Wiig, 2002) and were approved by the National Animal Research Authority (NARA), Norway. Following immobilization, and as part of the routine measurements of polar bears, a selection of morphometric variables representing the bears body size and head size were collected. Dorsal straight-line body length (SL), head length (HL) and zygomatic width (ZW) were measured in all bears, axillary girth (AG) was recorded in all mothers, and body mass (BM) was recorded by spring scale for all cubs (Derocher et al., 2005) (Table 1). Because BM was measured only for mothers from 2008, BM for all mothers was estimated based on SL and AG using a morphometric equation (Derocher and Wiig, 2002) before further recalculation into body condition index (BCI) using a BCI equation developed for polar bears (Cattet et al., 2002). The calculated BCI closely represent the polar bears true body condition. For some mothers, age was known because they had been caught previously, during their first two years of life. For the remaining mothers, age was estimated by counting annual growth layers in the cementum of an extracted vestigial premolar (Calvert and Ramsay, 1998; Christensen-Dalsgaard et al., 2010). All capture dates were transformed to capture day (1–365). Detailed information on capture day, age, and morphometric variables for the 4 groups are listed in Table 1.

Blood was collected from the femoral vein into heparinised Venoject® tubes (10 mL, Thermo Electron Corporation, Belgium) and separated into plasma and blood cells by centrifugation (3500 rpm, 10 min) within 8 h after sampling. Plasma samples were transferred to cryogenic vials and stored at −20 °C in the field and then at −70 °C in the lab freezer until analysis.

2.2. Contaminant analysis

The analyses of PCBs and OH-PCBs in the plasma samples were performed at the Laboratory of Environmental Toxicology at the Norwegian School of Veterinary Science (Oslo, Norway).

2.2.1. Extraction of PCBs and OH-PCBs

The multicomponent method used for extraction, determination of plasma lipid percentage (PL%), clean-up and analyses of the PCBs is based on the procedure originally described by Brevik (1978) with modifications described by Bernhoft et al. (1997) and Andersen et al. (2001), and extended to include OH-PCBs as described by Løken et al. (2006) and Berg et al. (2010). Briefly, the samples (~3 g of plasma) were added an internal standard (I.S.) mix consisting of PCB-29, PCB-112, PCB-207 (Ultra Scientific, RI, USA), 4′-OH-