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Anniina Holma-Suutari

HARMFUL AGENTS (PCDD/FS, PCBS, AND PBDES) IN FINNISH REINDEER (*RANGIFER TARANDUS TARANDUS*) AND MOOSE (*ALCES ALCES*)

*UNIVERSITY OF OULU GRADUATE SCHOOL;
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GAME AND FISHERIES RESEARCH INSTITUTE*

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Abstract

In Finland there is a food monitoring program which has found elevated dioxin and polychlorinated biphenyl concentrations in the muscle of semi-domesticated reindeer (*Rangifer tarandus tarandus*) calves. This led to further research on the concentrations of persistent organic pollutants in reindeer muscle, liver, and other internal organs. The research was further expanded on wild moose (*Alces alces*) muscle and liver.

The main objective of this thesis is to increase knowledge of polychlorinated dibenzo-*p*-dioxin (PCDD), polychlorinated dibenzofuran (PCDF), polychlorinated biphenyl (PCB), and polybrominated diphenyl ether (PBDE) pollution levels in the Finnish terrestrial environment, and in semi-domesticated reindeer and wild moose in particular. The research gives information of exposure conditions in the reindeer's food chain, as well as species differences and individual variation in accumulation and distribution of pollutants in reindeer and moose. Local differences between the contaminant concentrations were explored. Toxic equivalencies (TEQs) set by the World Health Organization (WHO) of PCDD/Fs and PCBs were calculated in order to assess the validity of selling reindeer and moose tissue.

It was observed that there is a species-, individual-, and tissue-specific accumulation of dioxins, dioxin-like PCB, and PBDE compounds in reindeer and moose. Varying exposure conditions mainly explain the differences, although taking into account the age of an individual animal, its metabolic patterns have a role, too. Reindeer placenta and milk proved to be important factors in the transporting of compounds from hind to calf. The highest PCDD/F and PCB concentrations (as WHO-TEQs) were observed in reindeer calves in the study area in which animals are fed in natural pastures only. Despite the findings, it was concluded that it is safe to eat reindeer and moose meat since the concentrations of dioxins and dioxin-like PCBs in the muscle are relatively low, and because of the low fat content in these animals. Reindeer liver, for its part, had quite a lot of dioxin-like compounds that may compromise its safety as food, at least on a regular basis.

The study shows that Finnish semi-domesticated reindeer and wild moose are good indicator species of POP contamination in a terrestrial environment, reindeer describing the situation in northern parts of the country especially.

Keywords: indicator species, intake, moose, muscle, PBDE, PCB, PCDD/F, persistent organic pollutants, reindeer, terrestrial environment, tissues

Holma-Suutari, Anniina, Haitalliset aineet (PCDD/F-, PCB- ja PBDE-yhdisteet) suomalaisessa porossa (*Rangifer tarandus tarandus*) ja hirvessä (*Alces alces*).
Oulun yliopiston tutkijakoulu; Oulun yliopisto, Luonnontieteellinen tiedekunta, Biologian laitos; Elintarviketurvallisuusvirasto Evira; Terveyden ja hyvinvoinnin laitos; Riista-ja kalatalouden tutkimuslaitos
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Tiivistelmä

Suomalaisessa ruokamonitorointitutkimuksessa löydettiin kohonneita dioksiinien ja polyklooratutujen bifenyylien pitoisuuksia puolivillin poron vasojen lihaksista. Se johti lisätutkimuksiin pysyvien orgaanisten yhdisteiden pitoisuuksista poron lihaksessa, maksassa ja muissa sisäelimissä. Tutkimus laajeni koskemaan myös hirven (*Alces alces*) lihasta ja maksaa.

Väitöskirjatyö lisää tietämystä polyklooratutujen dibentso-*p*-dioksiinien (PCDD), polyklooratutujen dibentsofuraanien (PCDF), polyklooratutujen bifenyylien (PCB) ja polybromattujen difenyylieettereiden (PBDE) pitoisuuksista suomalaisessa maaympäristössä ja erityisesti porossa ja hirvessä. Tutkimus antaa tietoa yhdisteille altistumisesta sekä viitteitä lajien- ja yksilöiden välisistä eroavai¬suuksista yhdisteiden kertymisessä ja niiden jakaantumisessa eri kudosten välillä. Alueellista vaihtelua yhdisteiden pitoisuuksissa selvitettiin myös. Maailman terveysjärjestön (WHO) asettamia PCDD/F- ja PCB-yhdisteiden toksisuusekvivalenttiarvoja (TEQ) tarkasteltaessa pystyttiin arvioimaan kemiallista elintarvikekelpoisuutta suhteessa EU:n antamiin sallittuihin pitoisuuksiin.

Työssä havaittiin laji-, yksilö-, ikä- ja kudosspesifistä dioksiinien, dioksiinienkaltaisten PCB- ja PBDE-yhdisteiden kerääntymistä porossa ja hirvessä. Tämä on todennäköisimmin seurausta vaihtelevasta altistumisesta yhdisteille, mutta myös lajien metabolisissa toiminnoissa yksilön eri ikäkausina voi olla eroavaisuuksia. Poron istukan ja maidon havaittiin olevan tärkeitä yhdisteiden kulkeutumisessa emolta sikiölle ja vasalle. Korkeimmat dioksiinien ja dioksiinien kaltaisten PCB-yhdisteiden konsentraatiot (WHO-TEQ-pitoisuuksina) havaittiin poron vasoilla tutkimusalueella, jossa eläimet olivat laiduntaneet ainoastaan luonnonlaitumilla.

Huolimatta havaituista haitta-ainepitoisuuksista todettiin, että sekä luonnostaan vähärasvaisen poron että hirvenlihan syönti on turvallista suhteellisen alhaisten dioksiinien ja dioksiininkaltaisten PCB-yhdisteiden pitoisuuksien perusteella. Poron maksa puolestaan sisälsi melko korkeita dioksiinien kaltaisten yhdisteiden pitoisuuksia, mikä voi vaikuttaa sen turvalliseen käyttöön elintarvikkeena ainakin usein syötynä.

Suomalainen puolivilli poro ja villi hirvi sopivat hyvin POP-kontaminoitumisen indikaattoreiksi maaympäristössä; poron erityisesti kuvaten tilannetta maan pohjoisosissa.

Asiasanat: altistuminen, dioksiinit, hirvi, indikaattorilajit, kudokset, lihas, maaympäristö, PBDE, PCB, PCDD/F, poro, pysyvät orgaaniset yhdisteet

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28.11.2014 Anniina Holma-Suutari

Abbreviations

AhR	Aryl hydrocarbon receptor
AhRE	AhR-responsive element
AMAP	Arctic monitoring and assessment program
ARNT	Aryl hydrocarbon nuclear translocator
BAT	Brown adipose tissue
BFRs	Brominated flame retardants
bw	Body weight
CYP450	Cytochrome P450 enzyme
DL-PCB	Dioxin-like polychlorinated biphenyl
DRE	Dioxin response element
EED	Environmental endocrine disruptor
I-TEQ	International toxic equivalent quantity
IUCN	World conservation union
lw	Lipid weight
Non-DL-PCB	Non-dioxin-like polychlorinated biphenyl
NO-PCB	Non-*ortho*-PCB
OH-radical	Hydroxyl radical
PBDE	Polybrominated diphenylether
PCB	Polychlorinated biphenyl
PCDD	Polychlorinated dibenzo-*p*-dioxin
PCDF	Polychlorinated dibenzofuran
PCDD/F	Polyclorinated dibenzo-*p*-dioxin and dibenzofuran
POP	Persistent organic pollutant
TCDD	Tetrachlorodibenzo-*p*-dioxin
TTR	Thyroid transporting protein
WHO	World Health Organization
WHO-TEQ	Toxic equivalent quantity defined by WHO
ww	Wet weight
XRE	Xenobiotic response element

List of original papers

This thesis contains the following original papers, which are referred to in the text by their Roman numerals.

I Suutari, A., Ruokojärvi, P., Kiviranta, H., Hallikainen, A., Laaksonen, S. (2009). Polychlorinated dibenzo-*p*-dioxins, dibenzofurans, and polychlorinated biphenyls in semi-domesticated reindeer (*Rangifer tarandus tarandus*) and wild moose (*Alces alces*) meat in Finland. Chemosphere 75, 617-622.

II Suutari, A., Ruokojärvi, P., Kiviranta, H., Verta, M., Korhonen, M., Nieminen, M., Laaksonen, S. (2011). Polychlorinated dibenzo-*p*-dioxins (PCDDs), dibenzofurans (PCDFs), polychlorinated biphenyls (PCBs), and polybrominated diphenyl ethers (PBDEs) in Finnish semi-domesticated reindeer (*Rangifer tarandus tarandus* L.). Environment International 37, 335-341.

III Suutari, A., Hallikainen, A., Ruokojärvi, P., Kiviranta, H., Nieminen, M. Laaksonen, S. (2012). Persistent Organic Pollutants in Finnish reindeer (*Rangifer tarandus tarandus* L.) and moose (*Alces alces*). Acta Veterinaria Scandinavica 54 (Suppl 1): S11.

IV Holma-Suutari, A., Ruokojärvi, P., Laaksonen, S., Kiviranta, H., Nieminen, M., Viluksela, M., Hallikainen, A. (2014). Persistent organic pollutant levels in semi-domesticated reindeer (*Rangifer tarandus tarandus* L.), feed, lichen, blood, milk, placenta, foetus and calf. Science of the Total Environment 476-477, 125-135.

V Holma-Suutari, A., Ruokojärvi, P., Kiviranta, H., Laaksonen, S., Nieminen, M., Viluksela, M., Hallikainen, A. (2014). Polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs), polychlorinated biphenyls (PCBs) and polybrominated diphenyl ethers (PBDEs) in tissues of Finnish semi-domesticated reindeer (*Rangifer tarandus tarandus* L.) and wild moose (*Alces alces*). Manuscript.

Author contribution

Article	Data collection	Analysis	Manuscript writing	Critical review and approval of the final manuscript
I	AH, SL, MN	PR	AH-S	AH-S, AH, PR, HK, SL
II	SL, MN	PR	AH-S	AH-S, PR, HK, MV, MK, MN, SL
III	AH, SL, MN	PR	AH-S	AH-S, AH, PR, HK, MN, SL
IV	SL, AH-S	PR	AH-S	AH-S, AH, PR, HK, MN, SL, MVi
V	SL, AH-S	PR	AH-S	AH-S, AH, PR, HK, MN, SL, MVi

AH-S= Anniina Holma-Suutari, MN= Mauri Nieminen, PR= Päivi Ruokojärvi, AH= Anja Hallikainen, HK= Hannu Kiviranta, SL= Sauli Laaksonen, MV= Matti Verta, MK= Markku Korhonen, MVi= Matti Viluksela

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1 Introduction

POPs, such as polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs), polychlorinated biphenyls (PCBs), and polybrominated diphenylethers (PBDEs) (Fig. 1. a, b, c, d), are commonly found in the food chains of northern terrestrial animals. Levels and patterns of POPs show variability between sampling sites, species, and individuals. Chemical and its stability, as well as the metabolic activity of different POPs, affect variations in their measured levels in different organs. Animals may have varying concentrations and compositions of compounds stored in their body and that may cause effects in different manners. Many ecological and physiological characteristics of animals affect the end point of chemical exposure. For instance, fat accumulation and depletion is a significant factor in the bioaccumulation process of lipophilic contaminants (AMAP, 2004).

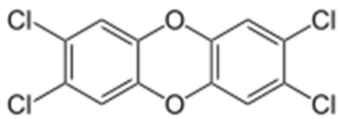
There are also many natural and anthropogenic factors that may affect the exposure and fate of environmental pollutants. Warming climate, changes in the food web structure and in prey-predator relationships, and an introduction of new pathogens are examples of possible factors contributing to exposure to these pollutants in the future. Species with strong seasonal adaptations, such as fat accumulation and mobilization, may be threatened by increased toxic load. Redistribution of accumulated contaminants may make animals sensitive to harmful effects caused by POPs (Jensen, 2006; Letcher *et al.*, 2010). For example, by losing fat and/or weight, POP storage in the body might be mobilized and transported to different organs.

Because of increased environmental stress, the health, reproduction potential, and survival of exposed species are affected. The vulnerability of fetuses and neonates is a cause for concern because contaminants may transfer from mother to offspring at a time of critical developmental (Fisk *et al.*, 2005; Main *et al.*, 2007; Birnbaum, 2013). It is important to understand that health effects of exposure during development processes can be observed long after the actual exposure has stopped. The main reason for human exposure to environmental contaminants in northern territories is the consumption of traditional food: fish, marine mammals, terrestrial mammals, and birds (Jensen *et al.*, 1997; Van Oostdam *et al.*, 1999; Hanssen, 2006; Hlimi et al., 2012). Bioaccumulation properties of POPs and their transfer through the food web have been quite thoroughly studied. However, the balance of this research has been on aquatic ecosystems rather than terrestrial ones (Jones & de Voogt, 1999).

In the annual EU food monitoring program conducted in Finland from 2003 to 2005, it was observed that the muscle meat of reindeer calves contained WHO-PCDD/F-PCB-TEQs in the range from 3.5 to 6.7 pg/g fat. PCDD/Fs and PCBs were also found from the muscle meat of adult reindeer and moose, but the levels were lower than in reindeer calves (Kiviranta *et al*., 2006).

In this thesis the levels and distribution of PCDD/Fs, PCBs, and PBDEs in terrestrial food chains of Finnish semi-domesticated reindeer (*Rangifer tarandus tarandus*) and wild moose (*Alces alces*) have been investigated. Because of economic, cultural, and environmental importance of reindeer and moose in Finland, it is essential to get knowledge these pollutants' occurrences in these species. The congener profiles of contaminants were explored and compared within and between species to demonstrate if there was variation between different species, or between different individuals of the same species. Among the objectives of the study was also to detect the placental and lactational transfer of PCDD/Fs, PCBs, and PBDEs, and to develop a picture of early-life exposure to studied POPs. The primary model animal for this effort was the semi-domesticated reindeer, whose suitability as a model is excellent because of its strong adaptation to the seasonal cycle with fat accumulation and depletion and lipid-rich milk.

In addition to finding out the occurrence, levels, and distribution of PCDD/Fs, PCBs, and PBDEs in the terrestrial environment and in its indicator species reindeer and moose, this thesis discusses chemical safety when consuming reindeer and moose muscle meat and liver as food.



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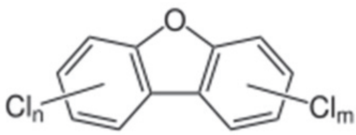

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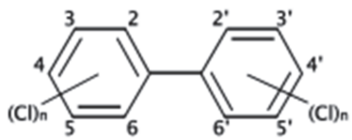

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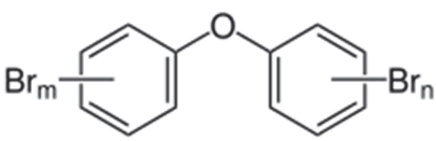

d.)

Fig. 1. a.) TCDD, the most toxic congener of PCDDs; b.) A common structure of PCDFs; c.) A common structure of PCBs; d.) A common structure of PBDEs.

2 Review of the literature

The following review of the literature describes basic knowledge and characteristics related to environmental contaminants, including this study. It also summarizes ecological and physiological characteristics of the studied indicator species reindeer and moose and brings up what is already known about the topic of interest in this study: levels and distribution of PCDD/Fs, PCBs, and PBDEs in the animal species in northern environments.

2.1 PCDD/Fs, PCBs, and PBDEs

Polychlorinated dibenzo-*p*-dioxins (PCDDs), dibenzofurans (PCDFs), and polychlorinated biphenyls (PCBs) are chlorine-containing chemicals which belong to the group of Persistent Organic Pollutants (POPs), which are characterized by long-range transport, persistence in their environments, bioaccumulation, and adverse health effects. Most PCDD/Fs and PCBs are extremely hydrophobic and resistant to biodegradation in soils and sediments. PCDD/Fs and PCBs are listed in the Stockholm Convention, whose aim is to reduce contamination by identifying and banning selected POPs (UNEP, 2001). Polybrominated diphenyl ethers (PBDEs) are in the family of bromine-containing compounds used as flame retardants (Renner, 2000; Bergman *et al*., 2012), part of which are classified as POPs. PBDEs are stable lipophilic substances. However, they may be less stable than dioxins and PCBs. They resemble PCBs in their structure and physical and chemical characteristics, which encourage long-range transport and accumulation (AMAP, 2003, 2004). PCDD/Fs, PCBs, and PBDEs are on the list of priority pollutants established by circumpolar and other governments for the regulation of environmental pollutants (AMAP, 1998; de Wit & Muir, 2010).

The northern circumpolar region is an important indicator area for the evaluation and research of POPs and related substances (de Wit *et al*., 2006). The levels of persistent organic pollutants are generally lower in the northern environment than in more temperate regions, but in certain species and at some locations the levels have been observed to be high (AMAP, 1998, 2004). In general, the time series of these legacy POP levels in biota mostly show significantly decreasing trends, with few examples of increasing trends. In contrast, some new chemicals, such as brominated flame retardants (BFRs) have been increased globally, especially in biota from northern regions (Ikonomou *et* *al*., 2002). An assessment of the state of the environment and ecosystem is one of the main goals of the European Union dioxin strategy (EU, 2012).

2.1.1 Structures and qualities

PCDDs and PCDFs (PCDD/Fs) belong to structurally similar families. Their molecule structure is planar and is a composition of three rings, which could be connected with 1-8 chlorine atoms (UNEP, 2001). There are 75 chlorinated dibenzo-*p*-dioxin congeners and 135 chlorinated dibenzofuran congeners; 7 PCDD and 10 PCDF congeners are toxicologically significant. PCDD/Fs are distributed in the atmosphere between gas and particle phases (AMAP, 2004), and accumulate in biological material via wet deposition, dry particulate deposition, and dry gaseous deposition (McLachlan & Hutzinger, 1990). In particular, the 2, 3,7,8-tetrachlorinated congeners are known to bioaccumulate (AMAP, 2003) when the non-2,3,7,8-Cl-substituted congeners are readily degraded by vertebrates (Opperhuizen & Sijm, 1990). PCDD/Fs are hydrophobic, lipophilic, resistant to biodegradation, and persistent in the environment. Water solubility of PCDD/Fs decreases when the chlorination degree increases (UNEP, 2001). The atmospheric half-life for PCDD/Fs ranges from one to three weeks (Mackay *et al*., 1992). Half-lives in the soil are considerably longer (Sinkkonen & Paasivirta, 2000).

PCBs consist of 209 different congeners, of which 12 congeners' toxic properties equate the properties of PCDD/Fs, and are called dioxin-like PCBs (DL-PCBs). DL-PCBs are either co-planar non-*ortho*-PCBs (4) or mono-*ortho*-PCBs (8) with biological activity (Ahlborg *et al*., 1994; EU, 2006). There are a different number of chlorine atoms in varying positions in the PCB molecule that affect the physical and chemical properties of a compound. The lack of chlorine atom substituents in the *ortho* positions permits non-*ortho*-PCBs to assume a planar configuration similar to that of PCDD/Fs (AMAP, 1998).

Individual PCB congeners and commercial formulations do not reflect the composition of environmental exposure sources; each environmental exposure matrix contains a unique mixture of PCB congeners (Kostyniak *et al*., 2005). PCBs (DL-PCBs and non-DL-PCBs) accumulate in biological material via the same deposition mechanisms as PCDD/Fs. Water solubility of PCBs is scarce; it decreases when the chlorination degree increases, which happens with the volatilization degree, too. The more chlorine atoms in the molecule, the more they accumulate in organic material and sediments in aquatic environments (UNEP, 2001). The molecular weights of PCB congeners are correlated with their tendency to adsorb to plant and soil surfaces (AMAP, 1998). OH-radical reactions are the major removal process for PCBs in the atmosphere, with half-lives ranging from 2 days to over 30 days, depending on the congener. These half-lives could be much longer for PCBs in the northern atmosphere because of lower OH-radical concentrations at the poles most of the year (Franklin *et al*., 2000). The half-lives in the soil are much longer for PCBs than half-lives in the atmosphere (Sinkkonen & Paasivirta, 2000).

PBDEs are stable lipophilic aromatic compounds that resemble PCBs in their structure and characteristics (Bergman *et al*., 2012). PBDEs consist of 209 different congeners, which have different degrees of bromine atom substitutions on the 2 phenyl rings. Presently used commercial PBDE mixtures, Deca-, Octa-, and Penta-BDEs, contain limited numbers of congeners. Deca-BDE contains an almost fully substituted Deca-BDE (BDE-209) (Chen & Bunce, 2003). PBDEs are persistent in the environment and are accumulated in organisms (Mikula & Svobodová, 2006). Lower brominated congeners in particular are known to bioaccumulate (AMAP, 2004). PBDEs are hydrophobic; higher brominated diphenyl ethers dissolve poorly in water, so their availability from the environment is scarce and they are not usually detected in animal and human samples (Vartiainen *et al*., 2001). However, BDE-209 for example, may debrominate to form less-brominated congeners which are more bioavailable than the original BDE-compound (Söderström *et al*., 2004). A part of PBDE compounds degrade in oxygen-rich circumstances in the soil (half-life 130–700 days), but BDE-209 is very stable (Nyholm *et al*., 2010).

2.1.2 Sources and migration

PCDD/Fs are unintentional by-products of industrial processes such as the production of chlorinated chemicals, e.g., chlorophenoxy herbicides, chlorophenol wood preservatives, PCB formulations, pesticides, and municipal, hospital, hazardous, and industrial waste incineration processes with chlorine present (AMAP, 1998; Alcock & Jones, 1996; Fiedler, 1996). Other sources of PCDD/Fs are the fuel combustion of chlorine scavengers containing motor vehicle and metallurgical industries. Pulp and paper mills that use chlorine in the bleaching process lead to PCDD/F inputs into aquatic environments (AMAP, 2004). Further sources are backyard waste and wood burning (UNEP, 2002; USEPA, 2000). In Finland the major source of PCDD/Fs has been the production of chlorophenol compounds (Hallikainen *et al*., 2004). The present sources in Finland are energy production, metal industry, and incineration processes of households.

Global emissions of PCDD/Fs are considered to be dominated by emissions into the atmosphere (Brzuzy & Hites, 1996). Many countries have set limitations for PCDD/F emissions to air via incineration processes; in Finland it is 0.1 ng/m^3 I-TEQ (VNp 842/1997). PCDD/F emissions in air have fallen in the range of 30–34 g I-TEQ per year in Finland. The major part (25 g I-TEQ) comes from energy production processes. The remarkable PCDD/F loader in the Finnish aquatic environment is the river Kymijoki, where there are about 6 000 kg of PCDD/Fs in the contaminated sediments. The highest PCDD/F concentrations in sediment reach 350 µg/kg I-TEQ (NIP, 2006).

PCBs are intentionally produced chemicals which have many useful characteristics, e.g., stability, non-flammability, and electrical insulating properties. PCBs have had many applications, including functioning as dielectric fluids in transformers and capacitors, as heat exchange fluids, and in plastics, sealants, and lubricating oils (AMAP, 2003). PCBs have been used world-wide in large quantities since the 1930s (Tanabe, 1988; Safe, 1994). Worldwide production of PCBs is estimated to be 1.3 million tons (Breivik *et al*., 2002). The use of PCBs has been banned in Western Europe and North America since the 1970s, but they are still released into the environment via leaking and degrading systems and are distributed through atmospheric transport (Tanabe *et al*., 1994; Letcher, 1996; Van Oostdam *et al*., 2005).

In Finland, the production, transport, sale, and abandonment of PCB-containing products was forbidden in the beginning of the 1990s. The major source of PCB exposure seems to be the environmental recycling of PCBs from former usage, and the northern ecosystem is polluted primarily through long-range transport from southern regions (AMAP, 2003). The levels of PCBs have been slowly decreasing in the environment due to legislation banning their use (Dallaire *et al*., 2002).

PBDEs are the subgroup of brominated flame retardants (BFRs), which account for 39% of worldwide flame retardant production (Darnerud *et al*., 2001; de Wit, 2002; Rahman *et al*., 2001). In Europe the production of BFRs is 10% of the total consumption (Van den Veen & de Boer, 2012). These substances are used in a number of industries in the manufacture of synthetic materials to reduce the risk of fires. They are used, for example, in building materials, electronic equipment, household furnishings, and textile coatings (Van Esch, 1994;

Darnerud *et al.*, 2001). PBDE-containing products are disposed of in the normal domestic waste stream in landfills and incineration processes. Congeners with more than three bromine atoms are persistent in the environment, and the introduction of these chemicals into many products may be a considerable long-term diffuse source of PBDE emissions into the environment (IPCS, 1994). In addition, there is a concern that BDE-209 debrominates to form less-brominated BDE congeners which are more bioavailable than BDE-209 itself (Law *et al.*, 2006).

The increasing use of BFRs in modern societies has led to increases of PBDEs in the environment. PBDEs are diffused by leaching from products into wastewater streams from users, households, and industries (Law *et al.*, 2006). PBDEs are found in remote areas of the Arctic which supports the indications that substances are globally distributed in the environment (AMAP, 2003). In Arctic areas, there may be much less photodegradation because of the lack of sunlight in wintertime. In some cases in Europe, slowing trends have been observed in PBDE concentrations, possibly because of the cessation of the manufacture and use of the penta- and octabromodiphenyl ether mixtures in the EU. However, in Arctic areas there are rapidly rising concentrations of the penta-mix formulation, especially in Canada (Law *et al.*, 2003). In North America, PBDE concentrations have been increased in many different samples, including human blood, in 1970–2005 (Birnbaum, 2013).

POPs enter the northern regions by different physical pathways via air currents, ocean currents, rivers, and transpolar ice movements. Biotic transport via migratory animals may also be a significant way to transport pollutants (AMAP, 2004). The present levels of POPs in the northern regions are mostly explained by long-range transport from lower latitudes (Hansen *et al.*, 1996; de March *et al.*, 1998; Macdonald *et al.*, 2000). The "global fractionation hypothesis" is the most widely accepted model about atmospheric long-range transport and distribution of POPs (Wania & Mackay, 1995, 1996; Li *et al.*, 2010). The hypothesis explains that transport is a complex phenomenon depending on physical-chemical properties, such as solubility, molecule size, and the vapor pressure of the contaminant. Highly volatile compounds, such as some lower-chlorinated PCBs, are taken directly into the gaseous phase and transported into the deposition regions. Semi-volatile compounds, as some highly chlorinated PCBs, are distributed between airborne particles and a gaseous phase depending on temperature, and could be deposited into seawater or soil via rain. During favorable weather conditions the compounds evaporate again into the atmosphere and are transported further. This kind of remobilization is called the "grasshopper effect:" the compounds could be categorized to "one-hop" or "multi-hop" types, depending on their physical-chemical characteristics (AMAP, 1998, 2004).

However, there are sources of POPs in Arctic regions which may have local significance, e.g., mineral exploration, coal mining, harbors, landfills, smelters, industry, and former military bases have been identified as sources of POPs (Schlabach & Skotvold, 1996a, 1996b; de March *et al.*, 1998; AMAP, 2004). Consequently, the distribution of POPs is not uniform in the Arctic area, since there is a geographical variation in levels resulting from point sources and from environmental convergence mechanisms. Some locally significant point sources have been identified, e.g., the Kola Peninsula, Norilsk in Russia, and Eastern Finnmark in Norway (UNEP, 2002).

2.1.3 A fate of xenobiotics in organisms

The levels of xenobiotics in an organism are the result of different factors: 1) uptake (absorption), 2) distribution, 3) metabolism, 4) excretion, and 5) stability (AMAP, 2004). Xenobiotics are mostly passively absorbed into the organism according to their physico-chemical properties, but their metabolism, detoxification, and elimination are active processes, most often very slow in persistent substances. Absorption (or excretion) of PCDD/Fs and PCBs has been observed to be both congener-dependent and related to the concentration of contaminants in blood and the congener-specific body burden. Potential routes are gastrointestinal, dermal, and transpulmonary absorption (Olson, 2003). After absorption of POPs from the gastrointestinal tract they are transported in blood and lymphatic streams, mainly bound with lipoproteins and chylomicrons (Lakshman *et al.*, 1986; Marinovich *et al.*, 1983; Moser & McLachlan, 2001). Also transthyretin (TTR) participates in the transportation of POPs. Some hydroxylated PBDE metabolites are also found capable of binding to TTR, while unhydroxylated PBDEs do not (Meerts *et al.*, 2000). OH-PBDEs and MeO-PBDEs are the metabolic products originated from PBDEs. The origin of these substances can be natural, anthropogenic, or both (Söderström *et al.*, 2004).

The metabolism of POPs occurs mainly in the liver via a two-phase process. These processes are catalyzed by liver enzymes such as the cytochrome P450 containing mono-oxygenases (Nebert & Gonzalez, 1987). In addition to detoxification, the enzymatic processes can also create reactive intermediates that may be mutagenic and/or carcinogenic (AMAP, 2004).

In marine mammals, lipophilic xenobiotics are primarily stored in the blubber but are metabolized in the liver. Most marine mammals undergo extensive seasonal changes in their fat reserves, because they fast during the breeding, lactation, and moulting seasons, and that can cause a release of stored contaminants into the body (O'Shea, 1999). This is also true for other Arctic animals, including terrestrial ones; their lipid dynamics can affect the distribution of stored contaminants. As the fat is utilized for energy production during fasting, the contaminant concentration in the remaining fat will increase, leading to new equilibrium between fat, blood lipids, and the lipids of other organs (Malcolm *et al*., 2003). On the other hand, in a fasting experiment with harp seals (*Phoca groenlandica*), it has been observed that POP concentrations in blubber have not changed, but blood levels have increased during the fast (Lydersen *et al*., 2002).

A liver and adipose tissue distribution of PCDD/Fs has been investigated with rats following the chronic ingestion of contaminated milk (Laurent *et al*., 2005). The PCDD/F congener properties and tissue characteristics seemed to be state the distribution to tissues. An increase in the chlorination degree of PCDDs caused a decrease in bonding in the adipose tissue, while it facilitated bonding in the liver.

Concentrations of 2,3,7,8-substituted PCDDs, PCDFs, and DL-PCBs have been measured in beef cattle calves to determine the distribution of contaminants (Hirako, 2007). Of 29 congeners analyzed, 19, 20, and 28 were detected in the blood, testes, and adipose tissue, respectively. It was demonstrated that PCDDs, PCDFs, and DL-PCBs leave the circulation and accumulate in the testes and adipose tissue in bovine calves. More PCDDs and PCDFs had accumulated in the testes than in adipose tissue, but more DL-PCBs were found in adipose tissue than in the testes.

PCB levels in the tissues of male bovine animals have been observed to correlate well with each other. Correlation was strong between the ear fat and perirenal, peritoneal, and neck fat, and with ear fat and neck meat, shoulder meat, and top side meat. Thus the other matrices that originated from the same animal had a PCB concentration in the same range, but the liver was an exception; it has higher concentrations in all studied bovine animals (Marchand *et al*., 2007).

In a study of Abraham *et al*. (1990), a mixture of PCDDs and PCDFs was subcutaneously administered to marmoset monkeys (*Callithrix jacchus*) and concentrations in different tissues were measured seven days after treatment. Only 2,3,7,8-substituted congeners were able to be detected in high amounts. The highest concentrations were detected in hepatic and adipose tissue; corresponding values in kidney, brain, lung, heart, thymus, or testes were clearly lower.

POP concentrations and variability in congener profiles may be associated with biological factors such as age, diet, and body condition, which can affect the distribution of compounds among the different organs and tissues (Colabuono *et al*., 2012). The major excretion route of POPs and their metabolites is via feces, and to some extent urine. Some of this is passive diffusion over the gut membrane and some from bile excretion of metabolites. It has been shown that if dietary levels exceed equilibrium concentration, then there is a net uptake leading to an increase in body burden. If the dietary levels are below the equilibrium concentration, then there is a net excretion and a drop in body burden (Moser & McLachlan, 2001).

The half-lives of PCDD/Fs and PCBs are long in the human body, on average 5 to 10 years (Tuomisto *et al*., 2011). For example, the half-life of TCDD is 2 840 days on average in an adult human, but only 19 days in a Sprague-Dawley rat (Geyer *et al*., 2002). PCB half-life has been reported to be 54–124 days in the Sprague-Dawley rat (Öberg *et al*., 2002).

Some background concentrations of POPs in different animal species are shown in Table 1. In Finland, PCDD/F concentration has been observed to be 29 pg/g WHO-TEQ in human fat tissue. In the same tissue the WHO-PCB-TEQ level has been 20.7 pg/g fat. The WHO-TEQ of PCDD/F concentration in human breast milk in Finland has been 5–7 pg/g fat, while in cow's milk the concentration has been 0.1–0.2 pg/g fat (Kiviranta, 2005).

Table 1. PCDD/F and PCB levels in different animal species.

Species	Sampling site	Year	Tissue	WHO-PCDD/F-TEQ pg/g fat	WHO-PCB-TEQ pg/g fat	Source
Hare	Russia	2001	Muscle	30	N.A.	1
Sheep	Finland	2003	Muscle	0.25	N.A.	2
Cow	Finland	2003	Muscle	0.24	0.13	2
Pig	Finland	2003	Muscle	0.20	0.06	2
Pig	Finland	2003	Liver	0.83	N.A.	2
Chicken	Finland	2003	Muscle	0.40	0.14	2
Reindeer	Russia	2001	Muscle	20	N.A.	1
Reindeer	Russia	2001	Liver	105	N.A.	1
Moose	Finland	2004	Muscle	0.35	0.92	2

N.A.=Not available. 1=RAIPON/AMAP/GEF/Project, 2001. 2=Kiviranta *et al*., 2006.

Female mammals transfer POPs via the placenta (Foster *et al*., 2000; Waliszewski *et al.*, 2000) and through milk (Nair *et al*., 1996; Anderson & Wolff, 2000). Many northern mammals, like seals, have very high fat content in their milk in order to facilitate rapid growth of the young during the short growing period; therefore the excretion of POPs via milk is important (AMAP, 2004). Studies support the idea that the transfer of POPs is more important via lactation than placental transfer. Lactation of females is an effective way to get rid of the contaminant load (Krowke *et al*., 1990).

2.1.4 Toxicity and adverse effects

The Toxic Equivalent Quantity (TEQ) is used when calculating the total amount of toxic PCDD/F and PCB congeners in the sample (Van den Berg *et al*., 2006). It is based on a concept of 2,3,7,8-TCDD Toxic Equivalency Factor (TEF), which is a certain value given to a congener founded its toxic potential by the World Health Organization (WHO). A congener-specific concentration level is multiplied by TEF and that gives the total TEQ value. The most toxic member of the class of planar halogenated aromatic hydrocarbons is 2,3,7,8-TCDD and at the same time it is also the most toxic synthetic compound (Kerkvliet, 1995). The last re-evaluation of TEFs was implemented in June 2005 (Van den Berg *et al*., 2006). The TEQ value is used, e.g., to assess the food consumption recommendations and maximum levels allowed in the food and foodstuffs (Hallikainen *et al*., 2004).

The toxic effects of PCDD/Fs and DL-PCBs occur via an Aryl Hydrocarbon receptor (AhR). AhR acts as a ligand-activated transcription factor located in cytosol (IPCS, 1993), is involved in the regulation of a large number of genes (Martinez *et al*., 2003), and resembles the steroid hormone receptor as its function (Cuthill *et al*., 1988; Whitlock, 1990). After the binding of an appropriate compound (ligand) to the AhR it produces a heterodimer with the aryl hydrocarbon nuclear translocator (ARNT). The heterodimer, an AhR/ARNT complex, integrates with a xenobiotic response element (XRE, also known as the AhR-responsive element, AhRE, or dioxin response element, DRE) in the cell nucleus, and regulates the expression of the target genes. As a consequence, e.g., the phase I cytochrome P450 enzymes CYP1A1, CYP1A2, and CYP1B1 are induced (Mikula & Svobodová, 2006; Webster & Commoner, 2003), and many biological responses get started, affecting growth and developmental differentiation (Vanden Heuvel & Lucier, 1993).

Differences in toxic potencies of various PCDD/F and DL-PCB congeners correlate well with differences in their binding affinity for the AhR (Safe, 1990). Toxicity depends on exposure of the organs, which is influenced by the absorption, distribution, and transfer of contaminants along with transporter proteins. The sufficient dose is required, which is a result of metabolism, detoxification, and excretion processes. Toxicity may need an activation of compound via metabolism, and toxicokinetics, tissue structure, and regulation by nerves and hormones may be essential for the manifestation of toxicity (Vähäkangas, 2011). Many times, toxicity of dioxin-like compounds has been associated with alterations of growth-regulatory genes or drug-metabolizing enzymes (Nebert, 2000) and is dependent on body burdens (DeVito & Birnbaum, 1995)

Considerable species-, strain-, age-, tissue-, and dose-specific differences occur in sensitivity to specific POPs, as well as differences in response (Pohjanvirta & Tuomisto, 1994; Van den Berg *et al*., 1994). It is challenging to extrapolate the effects seen in laboratory animals, e.g., rodents, to the effects of wild animals, because in most laboratory experiments animals are exposed to single compounds, often at acutely toxic doses, and wild animals are exposed to a mixture of compounds, often at low doses. The nature of interactions between the chemicals and also between the chemicals and physical factors varies. Interactions may include synergism, potentiation, and antagonism (Assmuth & Louekari, 2001; Vähäkangas, 2011). POPs may cause acute effects in high doses as well as long-term chronic effects at low doses. Long-term chronic exposure is the major concern in the northern ecosystems (AMAP, 2004).

Exposure to PCDD/Fs has been associated with a wasting syndrome, lymphoid involution, pancytopenia, chloracne, hyperkeratosis, gastric lesions, urinary tract hyperplasia, edema, tumor promotion, embryotoxicity, and decreased spermatogenesis, among other things. The most sensitive manifestation of 2,3,7,8-TCDD exposure is a decreased immunocompetence observed almost universally among the species in which it has been evaluated (Holsapple *et al*., 1991). Many toxic effects of TCDD, e.g., carcinogenicity, teratogenicity, hepatotoxicity, immunosuppression, and reproduction toxicity, are hypothesized to mediate through the AhR (Safe, 2001; Reen *et al*., 2002).

Immunosuppression effects caused by dioxin-like PCBs have been associated with the mass die-off of Baltic seals in 1988 (Ross *et al*., 1995, 2000). PCBs and their metabolites have been shown to interfere with the adrenal function *in vitro*, (Brandt *et al*., 1992; Lund, 1994; Johansson *et al*., 1998). A strong correlation has been found between the egg mortality of double-crested cormorants (*Phalacrocorax auritus*) and the levels of dioxin-like compounds found in bird eggs in the Great Lakes (Tillitt *et al*., 1992). POPs have been supposed to be a reason for the poor reproduction of lesser black-backed gulls (*Larus fuscus*) in the Gulf of Finland (Hario *et al*., 1999, 2004).

PBDEs have been established to affect hormonal regulation of organisms, and they are suggested as the environmental endocrine disruptors (EEDs) (Mikula & Svobodová, 2006). Adverse effects of PBDEs on hormonal mechanisms include thyroid hormone homeostasis disruption (Zhou *et al*., 2001; Zhou *et al*., 2002; Hallgren *et al*., 2001; Fowles *et al*., 1994) and sex steroid hormone disruption (Meerts *et al*., 2001; Nakari & Pesala, 2005). Other harmful effects of PBDEs are behavioral disturbances (Branchi *et al*., 2002; Kuriyama *et al*., 2005; Viberg *et al*., 2002), and spermatogenesis disorders in males (Kuriyama *et al*., 2005). PBDEs have been noticed to cause lymphoid depletion and increase oxidative stress. In addition, they have both anti-androgenic and anti-estrogenic effects (Birnbaum, 2013; Casals-Casas & Desvergne, 2011). Endocrine disruption has a particular ecological significance, because it is connected with long-term, possibly trans-generational effects and functions such as reproduction, development, metabolism, and stress endurance (Assmuth & Louekari, 2001).

There is a wide range of effects seen after exposure to PCDD/Fs and PCBs, e.g., effects on reproduction and development (WHO, 1996; Ringer *et al*., 1972; Rogan, 1982), the immune system (Vos & Luster, 1989; Tryphonas, 1994; Wong *et al*., 1992; Kerkvliet, 1995), the adrenals, thyroid gland, thyroid hormone levels, and vitamin A levels (Rolland, 2000; Simms & Ross, 2000). Also, visible changes in the liver, including hypertrophy, lesions, and tumors, have been seen (AMAP, 2004). Cancer, birth defects, decreased fertility, altered sex hormone balance, endometriosis, neurological effects and behavioral abnormalities are all linked to POPs exposure (Carpenter *et al*., 1998; Webster & Commoner, 2003; Birnbaum, 2013).

There is toxicological and mechanical knowledge of the effects of dioxins to humans; in particular they affect reproduction, hormonal balance, and the development of the nervous system. The most vulnerable are fetuses and newborn babies, because they are affected straight from the contaminant burden of the mother (EU, 2001).

2.1.5 Bioaccumulation

Many POPs can enter food chains and accumulate in wildlife. The northern ecosystem is believed to act as a sink for persistent compounds (Macdonald *et al*., 2000) because they have several characteristics which influence the extent and manner in which POPs bioaccumulate. In addition to the lipophilic nature of POPs, the most important characteristics that relate to bioaccumulation in biota are the following: 1) *Cold conditions and sunlight*, which influence the physical characteristics of the abiotic environment, the physical-chemical characteristics of contaminants, the rates of biological processes, and ecological and physiological adaptations of biota to cold and natural light; 2) *high trophic-level species*, which consume animal-originated food and have a long life span, during which the pollutants significantly accumulate; 3) *low species diversity*, which leads to simple food chains, in which the individual species adjust their feeding habits, growth rates, migration patterns, and reproductive characteristics in response to climatic factors and the availability of food resources; 4) *low productivity*, which may lead to slow growth and long life of species, and would be exposed to pollutants for a long period before being consumed in the next trophic level; 5) *cyclic annual productivity*, in connection with dispersation, migration, and utilization of food resources, all of which influence the bioaccumulation of contaminants; and 6) *physical stressors*, e.g., habitat destruction and harassment of animals (AMAP, 1998).

The vast majority of contaminants remain in the abiotic environment. Although the total quantities of persistent contaminants in biota are very small compared to the quantities in the abiotic environment, significant bioaccumulation could occur in some parts of the food web resulting in elevated concentrations in higher trophic levels.

Persistent compounds bioaccumulate along the food chain, reaching their highest levels in organisms at the top of the chain. The accumulation is affected by the properties of the organism, e.g., species, age, sex, feeding habits, reproductive status, and metabolic capacity. In addition, distribution and migration of species affect levels seen in biota (Colabuono *et al*., 2012). The extent of the metabolism of individual components becomes important in determining the levels of bioaccumulation. Different congeners are dominant at different levels of the food chain, and their concentrations depend on metabolism and physical characteristics (AMAP, 1998, 2004).

Concentrations are generally higher in adult males and non-reproductive females, while reproductively active and immature animals have lower contaminant levels (O'Shea, 1999). Freshwater and marine ecosystems usually contain higher POP levels than terrestrial ecosystems due to longer and more complex food chains. Bioaccumulation of POPs is especially significant in food chains dominated by organisms with high fat contents. Many carnivores at upper trophic levels are long-lived and may transfer POPs to offspring during gestation and lactation (UNEP, 2002). Carnivorous species of aquatic food webs, such as seals feeding at high trophic levels, may reach very high contaminant concentrations (de Wit & Muir, 2010).

The levels of contaminants in the organisms are dependent on the availability and degradability of the contaminant (O'Shea, 1999). The resistance to biodegradation gives a long half-life to a contaminant (Kelly & Gobas, 2001). It is found that bioaccumulation factors for individual animals can differ noticeably between herbivores and carnivores; in addition, the accumulation of organic chemicals also depends on aspects of physiology, time, food web characteristics, and predator-prey interactions in the trophic levels. In general, investigations of the bioaccumulation of pollutants in terrestrial environment food chains have been less extensive than in aquatic freshwater and marine environment food chains (Kelly & Gobas, 2001, 2003).

The pollutants in biological transport media are more easily available for bioaccumulation than those in abiotic media. Indeed, the POPs with properties preventing long-range transport in atmosphere and water may have a transport mode via biological media (Rigét *et al.*, 2010).

2.2 Study objects

2.2.1 Reindeer

Semi-domesticated reindeer (*Rangifer tarandus tarandus*) have an important economic and cultural meaning for thousands of people in the Finnish reindeer herding region. The number of reindeer is about 270 000 to 300 000 in Finland. One-third of the reindeer are slaughtered in autumn and early winter, when the animals have gained the highest body weight. Of the slaughtered animals, 75% are calves. The production of reindeer meat is 2.5–2.8 million kg/year, and consumption on average is 0.5 kg/person/year in Finland (Finfood). The fat content of reindeer meat is approximately 4.5%. Reindeer meat and liver contain high levels of vitamins A, B9, and B12, and iron, zinc, calcium, selenium, and essential fatty acids (Hassan, 2012).

Reindeer calves are born in spring. The nutrition of the calves during their first weeks of life is based on milk that is rich in protein and lipids (Holand *et al.*, 2002). Calves grow very quickly during the peak suckling period and their first few months of life (Timisjärvi *et al.*, 1982). The digestion of newborn calves is monogastric until their forestomachs become used to the fermentation of green vegetation. Gathering a supply of fat during summer and autumn is crucial for survival in winter, and for the reproduction of reindeer in spring (Soppela, 2004). During summer and autumn, reindeer eat various highly nutritious plant species, mostly vascular plants and mushrooms (Nieminen & Heiskari, 1989), accumulate body protein and fat, and increase their body weight.

Calves usually accumulate little fat during their first summer and are typically lean (Ringberg *et al.*, 1981). It will lose about 20% of its weight during winter (Nieminen *et al.*, 1984). The main winter feed of semi-domesticated reindeer in many areas are ground lichens (Nieminen & Heiskari, 1989). Reindeer that have received supplementary food are breeding individuals, which are kept over winter and are normally fed between December and March (Soppela, 2004).

Reindeer are well adapted to the extreme seasonal changes of temperature and nutritional supplies in the North; the changes in body conditions reflect the changes of nutritional supplies. In reindeer the deposited fat is located mainly under the skin, between the muscles, around the kidneys and the intestines, and in the bone marrow (Soppela, 2004). It is also the main supplier of energy production. Pregnant hinds primarily use their body fat stores for fetal growth (White & Luick, 1984; Tyler, 1987).

2.2.2 Moose

Wild moose (*Alces alces*) is the largest species in the deer family and a significant game animal in Finland. The distribution area of moose covers the entire country. The moose population in autumn is about 130 000, of which 45 000–70 000 are hunted yearly. Consumption of moose meat is 1.2 kg/person/year in Finland on average (Finfood, 2013). Moose meat is generally not available in the market in Finland. Its meat and liver are consumed mostly by hunters and people connected to them. Hence, the exposure of hunters to POPs via moose meat and liver is most likely higher than in the average population in Finland. There is also evidence that the intake of cadmium is higher in moose hunters than in other people (Vahteristo *et al*., 2003). The fat content of moose meat is low, approximately 3%. Moose calves are born in spring, and their first food is an energy-rich hindmilk. Moose go through significant changes in food quality, quantity, and availability throughout the year; their diet changes from deciduous leaves with high digestibility in summer to twigs of both hardwood and conifer species with low digestibility in winter (Saether & Anderson, 1990). Summer food of moose consists of green plants, leaves, and shrubs of the blueberry (*Vaccinium myrtillus*) and lingonberry (*Vaccinium vitis-idaea*). In winter their main food is the bark of aspen (*Populus tremula*) and sprouts of pine (*Pinus sylvestris*), willow (*Salix* sp.), birch (*Betula* sp.), and rowan (*Sorbus* sp.) (Siivonen & Sulkava, 2002).

2.2.3 Related species

In the Russian Arctic the sum concentrations and TEQs of PCDDs, PCDFs, and non-*ortho*-PCBs have been measured in the muscle and liver of reindeer collected in 2000–2001. Reindeer livers (n=60) contained on average 4.2–105 pg/g total WHO-TEQ lipid weight (lw), and muscle samples (n=60) 0.75–20 pg/g total WHO-TEQ lw (RAIPON/AMAP/GEF Project, 2001). In Sweden, WHO-PCDD/F-PCB-TEQ in moose muscle has been on average 8.1 pg/g lw. In reindeer muscle the mean WHO-TEQ level of PCDD/Fs and DL-PCBs has been 2.3 pg/g lw (Danielsson *et al*., 2008).

In Finland, the PCB level in the liver of moose has been below 1 μg/g wet weight (Hirvi, 2004). Considering PCBs in reindeer, the Swedish study reports a declining ΣPCB concentration in reindeer muscle in 1987–2006 (Bignert *et al*., 2008).

Concentrations of PBDEs are noticed to be relatively low in the muscle tissue of mammals from terrestrial ecosystems. ∑PBDE concentrations were from 0.5 to 1.7 μg/kg lw in reindeer and moose in Sweden (Sellström, 1999). However, PBDE levels have been observed to be high in reindeer serum/plasma samples from Norway (up to 1 270 ng/g lw). On the other hand, PBDEs (BDE-47, -100, -99, -85, and -209) were not detected in moose in that study (Polder *et al*., 2009). Another Norwegian study showed the PBDE concentration in the range of LOD to 9.4 ng/g lw in moose liver. In the same study the pooled samples of European roe deer (*Capreolus capreolus*) liver showed similar PBDE levels as found in the moose samples (Mariussen *et al*., 2008).

In Germany a study had observed that the liver of roe deer was highly contaminated with PCDD/Fs and DL-PCBs in natural surroundings (WHO-PCDD/F-PCB-TEQ 61 pg/g lw) (Schröter-Kermani *et al*., 2011). Generally, little is known about POP contamination of the terrestrial ecosystem in the Arctic, especially BFR (de Wit & Muir, 2010).

3 Aims of the study

This thesis investigated the occurrence, levels, and distribution of PCDD/Fs, PCBs, and PBDEs in a terrestrial environment, where indicator species were semi-domesticated reindeer and wild moose. The aims of the study are the following:

1. The study finds whether there are differences in PCDD/F and PCB concentrations between adult reindeer and moose and calves. Differences between reindeer with a natural diet and supplementary feedings are investigated. The study assesses whether eating reindeer and moose meat is safe or not because of PCDD/F and PCB accumulation.
2. The study investigates the PCDD/F, PCB, and PBDE compounds' concentrations in the reindeer food chain. Stillborn calves' muscle and brown adipose tissue and reindeer milk are used in assessing the transplacental and lactational transfer of compounds.
3. The study examines congener profiles of PCDD/Fs and dioxin-like PCBs (DL-PCBs) in reindeer and moose tissue. It demonstrates if there are inter-individual or inter-species differences in the accumulation and distribution of PCDD/F and DL-PCB compounds.
4. The study examines the transplacental and lactational transfer and accumulation of PCDD/F, PCB, and PBDE compounds in reindeer fed on different diets (lichen diet or concentrates diet). The accumulation of compounds in muscle and liver tissue of reindeer hinds, calves, and fetuses are studied.
5. The study investigates congener-specific PCDD/F, PCB, and PBDE concentrations in the internal organs of reindeer and moose to elucidate the contaminant profiles of different parts of the animals' bodies.

The study hypotheses are the following:

1. There are differences in PCDD/F, PCB, and PBDE concentrations between reindeer and moose.
2. Reindeer and moose have specific congener concentrations of PCDD/Fs, PCBs, and PBDEs.
3. There are spatial differences in contaminant concentrations. It is assumed that reindeer in natural pastures have more contaminants than reindeer fed with supplementary feed.

4. Contaminants are transferred via the food chain.
5. Placenta and milk are important vectors in transferring the contaminants.
6. Reindeer and moose meat are safe as human food.

4 Materials and methods

4.1 Study area and sampling

The samples were mainly taken in Finnish Lapland, the most northern part of Finland. The majority of the samples were gathered above the Polar circle (66º33’39”N). In addition, some of the samples were taken in the more southern areas in the boreal zone. Sampling of reindeer and moose was conducted in the years 2006, 2010, and 2011. The sampling sites can be seen in Fig. 1. In addition, lichen (2 kg of the crop from 2007, Oulu, Finland) and food concentrate (1 kg Poro-Elo 2 plus, manufacturer Suomen Rehu, Hankkija-Maatalous Oy, P.O. Box 390, 05801, Hyvinkää, Finland) samples were studied in 2010.

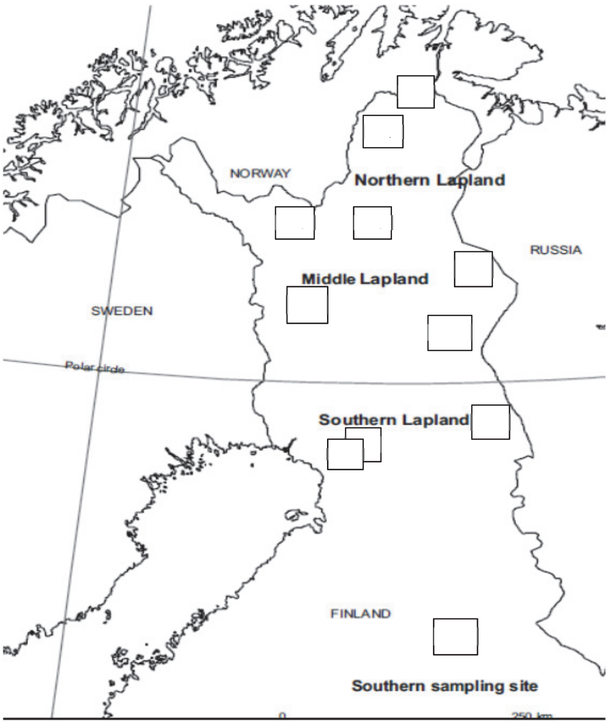

Fig. 2. Reindeer and moose samples were gathered in Northern Finland, from Northern, Middle, and Southern Lapland. In addition, some moose samples were taken from a more southern sampling site.

4.2 Animal samples and ethical considerations

Reindeer and moose samples were taken from animals slaughtered for human consumption so there was no need for animal testing permissions. In addition, there are some samples from pregnant reindeer hinds (e.g., fetuses), which are not used as food. Normally there are always some pregnant hinds among the slaughtered reindeer, so it is ethically acceptable to take these samples and use them in research purposes instead of wasting them.

4.2.1 Reindeer and moose samples

The number and date of the samples are seen in Table 2.

Table 2. The number and year of the samples.

	Sample	n (sub-n)	Year
Northern Lapland	Reindeer calf meat (individual)	10	2006
	Reindeer calf meat (individual)	2	2010
	Reindeer calf meat (pooled)	1(6)	2006
	Reindeer fetus meat	2	2010
	Reindeer adult meat	4	2006
	Reindeer adult meat	4	2010
	Reindeer stillborn calf meat	1	2006
	Reindeer calf liver	3	2006
	Reindeer calf liver	2	2010
	Reindeer fetus liver	2	2010
	Reindeer adult liver	1	2006
	Reindeer adult liver	4	2010
	Reindeer milk summer	7	2006
	Reindeer milk autumn	7	2006
	Reindeer milk control	2	2010
	Reindeer milk summer	2	2010
	Reindeer milk autumn	2	2010
	Reindeer blood	2	2010
	Reindeer placenta	2	2010
	Moose calf meat	2	2006
	Moose adult meat	2	2006
	Moose calf liver	4(1–3)	2011
	Moose adult liver	2(2)	2011
Middle Lapland	Reindeer calf meat (individual)	9	2006
	Reindeer calf meat (pooled)	1(6)	2006
	Reindeer adult meat	3	2006

	Sample	n (sub-n)	Year
	Reindeer stillborn calf meat	6	2006
9999	Reindeer stillborn calf brown adipose tissue (BAT)	2	2006
	Reindeer calf liver	3	2006
	Reindeer adult liver	2	2006
	Moose calf meat	2	2006
	Moose adult meat	2	2006
Southern Lapland	Reindeer calf meat (individual)	10	2006
	Reindeer calf meat (pooled)	1(6)	2006
	Reindeer fetus meat	1	2008
	Reindeer adult meat	4	2006
	Reindeer adult meat	1	2008
	Reindeer adult kidney	1	2008
	Reindeer adult lymphatic nodes	1	2008
	Reindeer adult abdominal fat	1	2008
	Reindeer adult bone marrow	1	2008
	Reindeer stillborn calf meat	4	2006
	Reindeer stillborn calf BAT	1	2006
	Reindeer calf liver	4	2006
	Reindeer adult liver	1	2006
	Reindeer adult liver	1	2008
	Reindeer placenta and uterus	1	2008
	Moose calf meat	2	2006
	Moose adult meat	2	2006
Southern Finland	Moose adult liver	1	2011

The method of meat sampling was standardized, allowing a comparison between the different regions. Meat samples represented the parts consumers eat. The samples were built up in a ratio of carcass meat consumption consisting of 200 g of rump, 200 g of rib and fore back, and 100 g of shoulder muscle.

The brown adipose tissue (BAT) samples were gathered from the specific locations of the calves' bodies where they existed: around the shoulders, sternum, trachea and spine, and in the abdominal and thoracic cavities. The samples weighed 20 g on average.

Milk samples in 2006 were collected from the Kaamanen experimental reindeer station in Inari (N 69° 3.9694`, E 27° 6.1877`), situated in Northern Lapland. Milk samples were collected twice from seven hinds (aged 7–9 years) for comparison of PCDD/F, PCB, and PBDE concentrations. The first samples were milked in early summer, just after calving season, and the second in autumn, 14–18 weeks after calving. The sample collection (30 ml in each) was performed by hand milking into pre-cleaned glass bottles and using nitrile gloves. The milk collection was facilitated by using Oxytocin (Partoxin vet® 17 mg [10 IU]/ml. Pharmaxim) on each hind.

Two pregnant reindeer hinds from the northern herd were captured and kept in pounds for a period of 4.5 months (from April to August 2010) in an experimental zoo in the University of Oulu, Finland. About two weeks after calving, the control milk samples were taken from both hinds. After that, the hind-calf pairs were separated into the distinct pounds for the different diet treatments. Early summer milk samples were taken in June, about six weeks from calving. At the end of August 2010, milk samples were taken again to characterize changes in milk concentration of POPs during lactation. After that, hinds and their calves were slaughtered.

Two randomly assigned pregnant reindeer hinds from the northern herd were slaughtered in the beginning of May 2010. These animals were fed with lichen and reindeer food according to normal herding practice. Unborn fetuses of these hinds were cut out with the placentas for sampling. Placentas were taken and stored at a temperature of -20°C until the preparation before the analyses. The placentomes were separated for the analyses.

4.3 Chemical analysis

Analyses were performed at the Chemical Exposure Unit at the National Institute for Health and Welfare (THL), Finland (P.O. Box 95, 70701, Kuopio, Finland). The laboratory is an accredited testing laboratory (No T077) in Finland according to the requirements of the standard EN ISO/IEC 17025, and the scope of accreditation includes PCDD/Fs, PCBs, and PBDEs from foodstuffs. The measured PCDD/F congeners included 17 2,3,7,8-chlorine substituted congeners. The 37 PCB congeners included 12 dioxin-like PCB (DL-PCBs) congeners, and the PBDEs included 15 congeners (Table 3).

Table 3. Studied PCDD/F, PCB, and PBDE congeners.

PCDD/Fs	PCBs	DL-PCBs	PBDEs
2378-TCDD	PCB-18	PCB-77	BDE-28
12378-PeCDD	PCB-28/31	PCB-81	BDE-47
123478-HxCDD	PCB-33	PCB-126	BDE-66
123678-HxCDD	PCB-47	PCB-169	BDE-71
123789-HxCDD	PCB-49	PCB-105	BDE-75
1234678-HpCDD	PCB-51	PCB-114	BDE-77
OCDD	PCB-52	PCB-118	BDE-85
2378-TCDF	PCB-60	PCB-123	BDE-99
12378-PeCDF	PCB-66	PCB-156	BDE-100
23478-PeCDF	PCB-74	PCB-157	BDE-119
123478-HxCDF	PCB-99	PCB-167	BDE-138
123678-HxCDF	PCB-101	PCB-189	BDE-153
234678-HxCDF	PCB-110		BDE-154
123789-HxCDF	PCB-122		BDE-183
1234678-HpCDF	PCB-128		BDE-209
1234789-HpCDF	PCB-138		
OCDF	PCB-141		
	PCB-153		
	PCB-170		
	PCB-180		
	PCB-183		
	PCB-187		
	PCB-194		
	PCB-206		
	PCB-209		

The meat samples were freeze-dried after homogenization, and the fat was extracted with ethanol-toluene (30/70 v/v) using Accelerated Solvent Extractor (ASE 300). The fat content of the samples was determined gravimetrically. After defatting the samples on an acidic silica column, the PCDD/Fs and non-*ortho*-PCBs were fractionated from the rest of the PCBs on a carbon column, and both fractions were further purified on an alumina column.

Studied compounds were analyzed with HRGC/HRMS (VG 70-250SE), using a selected ion monitoring mode (SIR) with a resolution of 10 000. Toxic equivalents (TEQs) were calculated for PCDD/Fs and DL-PCBs (WHO-PCDD/F-PCB-TEQs) using TEF values defined by WHO in 1998 (Van den Berg *et al.*, 1998) and 2005 (Van den Berg *et al.*, 2006).

Depending on the samples discussed, the contaminant concentrations are reported as upper or lower bound concentrations. In the upper bound method, the results of congeners with concentrations below the limit of quantification are considered as LOQ. In the lower bound method, the results of congeners with concentrations below LOQ are considered undetected.

4.4 Statistical analysis

In Sub Study 1 the statistical analysis was conducted using the SPSS 15.0 software. The means of WHO-PCDD/F-PCB-TEQ concentrations of individual reindeer calves were compared with analysis of variance (ANOVA) to reveal differences between the northern, the middle, and the southern regions. *P*-values less than 0.05 were considered statistically significant.

In Sub Study 2, one sample t-test, a univariate analysis of variance, and a Pearson correlation were used in the statistical analyses. A *P*-value less than 0.05 was considered to have a statistical significance.

In Sub Study 3, the statistical analysis was conducted using the SPSS 16.0 software. ANOVA was used to detect significant differences among the data set, when data were normally distributed. The Kruskal-Wallis test was used if homogeneity of variances did not realize. The criterion for significance was $P<0.05$.

5 Results

The total WHO-TEQs and PBDE sum concentrations in reindeer and moose muscle and liver samples are shown in Table 4.

Table 4. Total reindeer and moose WHO-TEQs (pg/g fat) and PBDE (ng/g fat) concentrations.

Sample	Area	Year	WHO-TEQ	PBDE
Reindeer muscle	North-Lapland	2006	2.51	1.47
Reindeer liver	North-Lapland	2006	47.6	0.73
Reindeer muscle	Mid-Lapland	2006	3.53	1.92
Reindeer liver	Mid-Lapland	2006	52.6	0.77
Reindeer muscle	Mid-Lapland	2010	1.03	3.2
Reindeer liver	Mid-Lapland	2010	46.8	0.48
Reindeer muscle	South-Lapland	2006	2.90	2.84
Reindeer liver	South-Lapland	2006	108.6	25.3
Reindeer muscle	South-Lapland	2008	2.24	2.1
Reindeer liver	South-Lapland	2008	165	4.1
Moose muscle	North-Lapland	2006	2.20	53.3
Moose muscle	Mid-Lapland	2006	1.89	5.0
Moose liver	Mid-Lapland	2012	2.38	0.60
Moose muscle	South-Lapland	2006	2.1	13.6
Moose liver	South-Lapland	2012	6.4	0.36
Moose liver	Mid-Finland	2012	8.37	0.47

5.1 PCDD/Fs and PCBs in reindeer and moose

In reindeer liver samples (taken in 2006) there were much more WHO-PCDD/F-TEQs than in moose liver samples (taken in 2011). Also WHO-PCB-TEQs in reindeer livers were higher than in moose livers. However, PCDD/Fs and PCBs (as WHO-TEQs) were quite equal in the muscle samples of reindeer and moose (samples from years 2003–2010) (V).

Considering the liver samples of reindeer calves and adults in 2010, it was observed that calves had higher WHO-PCB-TEQs than adults, but WHO-PCDD/F-TEQs were higher in adult reindeer than in calves. In 2006, reindeer calves had higher WHO-PCDD/F-TEQ and WHO-PCB-TEQ concentrations than adult reindeer. The proportion of WHO-PCB-TEQ from the total TEQ was greater than the WHO-PCDD/F-TEQ in the liver samples of reindeer calves in 2010, an opposite result than in 2006. In adult reindeer liver, WHO-PCDD/F-TEQ had a bigger contribution to the total TEQ than WHO-PCB-TEQ both in 2006 and 2010 (IV).

In the muscle samples of individual reindeer in 2006, the average fat-based WHO-PCDD/F-PCB-TEQ concentration was 3.2 pg/g fat in calves and 2.3 pg/g fat in adult reindeer. The highest fat-based mean level of PCDD/Fs and PCBs was found in the middle sampling region in 2006. There were statistically significant differences between the WHO-PCDD/F-PCB-TEQ concentrations in different sampling areas (P=0.01). In 2010, the total WHO-TEQs were lower in reindeer calves (1.7 pg/g fat on average) and in adult reindeer (1.1 pg/g fat on average) than in 2006. The highest PCDD/F sum in the 2010 sampling was detected in reindeer hinds from Southern Lapland (IV).

In 2006, pooled reindeer calves from the western parts of three sampling sectors (the northern, the middle and the southern) contained equal levels of contaminants, 2.4 pg WHO-PCDD/F-PCB-TEQ/g fat on average. The contributions of PCDD/Fs and PCBs to the total WHO-TEQ were similar in all samples of pooled reindeer calves regardless of the sampling area, 46% and 54%, respectively (I). In most of the reindeer muscle samples in 2010, PCBs were dominating compounds of the total WHO-TEQ (IV). This is consistent with the results of the 2006 study (I). In 2006, the calves from the western parts of the sampling sectors clearly had lower PCDD/F and PCB sum concentrations than the calves from the eastern parts of the sampling sectors. Fat contents of the calves were also lower in the western parts (II).

Contrary to reindeer, the lowest PCDD/F and PCB concentrations of moose muscle samples were found in the middle sampling region in 2006. The average lipid-based WHO-TEQ concentration was lower in moose calves (1.9 pg/g fat) than in reindeer calves. The fat-based concentration in adult moose was equal to adult reindeer (I). In the 2011 sampling, an adult moose from Northern Finland had lower PCDD/F and PCB sum concentrations in the liver than moose calves from the same area and young adult moose from Central Finland. However, one female calf from the northern area also had quite a low PCDD/F level compared to other moose calves from there. A young adult moose from the southern sampling site had the highest WHO-PCDD-TEQ of the all moose liver samples studied (V).

The fat content of moose muscle in 2006 was generally lower than the fat content of reindeer muscle. In moose calves, the mean wet weight concentration of WHO-PCDD/F-PCB-TEQ was lower than in reindeer calves, 0.05 pg/g ww versus 0.16 pg/g ww, respectively. The concentration of the wet weight-based

WHO-TEQ in adult moose muscle was equal to the concentration in adult reindeer from the northern and southern sampling regions (I).

Reindeer calves from the middle sampling region contained 0.22 and adult reindeer 0.21 pg WHO-PCDD/F-PCB-TEQ g/ww. These concentrations were two times higher than the concentrations found from leaner reindeer calves and adults from other areas (I).

5.1.1 Fat-based concentrations

The PCDD/F sum of reindeer organs and tissues (excluding meat and liver) were quite equal (4.3–5.9 pg/g fat), although the fat contents varied strongly, ranging from 7.9% in the kidneys to 33% in the bone marrow. WHO-PCDD/F-TEQ varied from 1.3 pg/g fat in the bone marrow to 1.6 pg/g fat in the lymph nodes. With the PCB sum, the highest concentration detected in the bone marrow sample was 9.8 ng/g fat, followed by abdominal fat at 8.7 ng/g fat. Indicator-PCB concentrations correlated well to PCB sums, the highest level being 5.4 ng/g fat in the bone marrow followed by 4.7 ng/g fat in abdominal fat. WHO-PCB-TEQ varied from 1.5 pg/g fat in the lymph nodes to 2.1 pg/g fat in the bone marrow. However, the highest PCDD/F concentration was detected in reindeer liver samples (PCDD/F sum 462 pg/g fat) (V).

5.2 Congener-specific PCDD/Fs and PCBs in reindeer and moose

5.2.1 PCDD/Fs

The most prominent PCDD/F congener in reindeer muscle samples in the 2006 sampling (III) was OCDD, followed by 23478-PeCDF and 123678-HxCDD. In the moose muscle samples, the most dominating PCDD/F congeners were OCDD, 23478-PeCDF, 1234678-HpCDF, and 1234678-HpCDD (III). Also, 1234678-HpCDD showed an elevated concentration in the northern area's male moose calf. A similar phenomenon was observed in reindeer calves from the northern area in 2006. In the 2010 sampling the most dominating PCDD/F congeners in reindeer muscle were 2378-TCDD, 12378-PeCDD, 123678-HxCDD, 1234678-HpCDD, OCDD, and 23478-PeCDF.

In the reindeer liver samples in 2006 the most dominating PCDD/F congeners were OCDD, 23478-PeCDF, 123478-HxCDF, 123678-HxCDF, 234678-HxCDF, and 1234678-HpCDD. The congener 23478-PeCDF concentrations seemed to be high in the southern area's calves and adult reindeer, especially in their livers. That congener also showed a high contribution to stillborn calves' muscle tissue in the same area, and also in the middle area's stillborn calves' muscle.

Considering stillborn calves' brown adipose tissue (BAT) it was noticed that the mean 23478-PeCDF concentration (2.4 pg/g lipid weight) was overwhelmingly the highest of any congeners. However, many other PCDD/F congeners also existed in BAT and three of them, namely 2378-TCDD, 123478-HxCDD, and 123678-HxCDD, were lacking in muscle samples but were represented in BAT.

In reindeer liver samples in 2010 the most prominent PCDD/F congeners were 1234678-HpCDD, OCDD, 23478-PeCDF, 123478-HxCDF, 123678-HxCDF, and 1234678-HpCDF. Hence, the congener profiles in reindeer muscle and liver were partly different (IV), as they were in 2006.

When comparing the individual reindeer-fetus pairs in 2010, it was observed that 23478-PeCDF levels were highest in hind-fetus pair #5, especially in the muscle tissue of fetus #5 (0.04 pg/g ww) and the liver of hind #5 (6.6 pg/g ww). Also, the liver of calf #1 contained quite a lot of 23478-PeCDF (2.5 pg/g ww), and also 1234678-HpCDF (2.1 pg/g ww). The TEF-value (toxic equivalent factor) for 23478-PeCDF is 0.5, so its contribution to the WHO-TEQ is relatively big.

The most general PCDD/F congeners in reindeer internal organs were 23478-PeCDF and 2378-TCDF. The most conspicuous PCDD/F congeners found in moose liver samples were 23478-PeCDF and 2378-TCDF. Also, 123478-HxCDF, 123678-HxCDF, and 234678-HxCDF were well represented in some of the moose liver samples.

5.2.2 PCBs

In reindeer muscle samples in 2006, the most dominating DL-PCB congener was PCB-126 (III), with its highest concentration (39 pg/g fat) detected in the middle area's stillborn calf. That individual also contained quite a high concentration of PCB-77 (18 pg/g fat), which was the other very frequent congener in the studied population. Other DL-PCBs, which had a strong contribution to the total TEQ were PCB-81 and PCB-169 (III).

In 2010 the most conspicuous DL-PCBs in reindeer muscle were PCB-126 and PCB-77 (IV). In addition, the PCB-126 concentration was overwhelmingly highest in reindeer liver (on average 9.4 pg/g ww). PCB-126 is the most toxic congener of dioxin-like PCBs, having a TEF-value of 0.1. Other DL-PCBs detected in reindeer liver and muscle samples were PCB-118, PCB-105, and PCB-156. The most dominating PCBs other than dioxin-like ones were PCB-138, PCB-153, PCB-170, and PCB-180 (IV, Fig. 2.). The concentrations of other PCBs were very similar in reindeer liver and muscle, but slightly higher in moose muscle than in moose liver tissue.

In moose muscle samples in 2006 the most dominating DL-PCB congener was PCB-77, followed by PCB-126 and PCB-81. Also, PCB-118 was among the most detected ones. There were no differences between DL-PCB concentrations in muscle samples of adult moose and moose calves. However, PCB-77 levels in the adult moose females were lower than in the males. In moose liver, PCB-126 was the most dominating DL-PCB congener. There were similar congener profiles of other PCBs in moose muscle and liver. The dominating congeners were PCB-138, PCB-153, and PCB-180. Only PCB-28/31 seemed to be at a slightly higher level in moose liver than in muscle tissue.

PCB-126 was the most dominating DL-PCB congener in reindeer liver samples in 2006 (III). Other frequent PCB congeners were PCB-77, PCB-81, and PCB-169, although their concentrations were much lower than that of PCB-126. The highest PCB-126 level (400 pg/g fat) was detected in female reindeer calves from the middle areas.

In 2006 the overall DL-PCB profile in reindeer liver fitted well to reindeer muscle samples. In general, the concentrations of DL-PCBs were higher in the reindeer calf liver samples than in the adult samples.

Of the non-*ortho* dioxin-like PCBs in reindeer organs, the most dominating congener was PCB-126. However, PCB-77 also contributed well to the total non-*ortho*-PCB sum, especially in the lymph nodes of reindeer. The most conspicuous/abundant PCB congeners were PCB-153, PCB-180, and PCB-118. Of the non-*ortho* dioxin-like PCBs in moose liver, the most dominating congener was clearly PCB-126, when PCB-28/31, PCB-118, PCB-138, PCB-153, and PCB-180 were the most general congeners of the non-dioxin-like PCBs.

5.3 PBDEs in reindeer and moose

PBDE concentrations were higher on average in the livers of moose calves than in the livers of adults. In addition, PBDE levels were very low in general, below 0.5 ng/g fat in all moose liver samples (V). PBDE concentrations in reindeer livers were somewhat higher, up to 8 ng/g fat, and were higher in adults than in calves

(II, V). When comparing the PBDE concentrations in different organs of reindeer, it was overwhelmingly highest (29 ng/g fat) in the lymph nodes. Other tissues (abdominal fat, kidneys, and bone marrow) showed lower PBDE levels, ranging from 0.18 to 0.59 ng/g fat. Of PBDEs, the dominating congener was BDE-209, which contributed very intensively, especially in the lymph nodes (V).

The most abundant PBDE congeners in reindeer muscle samples in 2010 were BDE-209, BDE-153, BDE-99, and BDE-47 (IV). The set was similar in reindeer liver samples. The proportion of BDE-209 was clearly the biggest of all the samples; in muscle samples its contribution was over 90% of the sum of PBDEs, and in liver samples its share was 58%. In moose liver samples, the most dominating PBDE congeners were BDE-209 and BDE-47 (V).

5.4 Contaminant transfer in the food chain of reindeer

When comparing WHO-PCDD/F-TEQ and WHO-PCB-TEQ concentrations in reindeer feed concentrates and lichen, both WHO-PCDD/F-TEQ and WHO-PCB-TEQ ww-based levels were noticeably higher in lichen than in reindeer feed concentrates (IV). The contributions of WHO-PCDD/F-TEQs to total WHO-TEQs were bigger than WHO-PCB-TEQs in both matrices (78% of total mass on average).

Reindeer hind-calf pair #1, which had gone through the lichen diet, had a higher total of TEQ concentrations on average than pair #2, fed on the diet of feed concentrates. When comparing WHO-PCDD/F-TEQs in reindeer feed concentrates and in reindeer muscle tissue, it is obvious that they did not bioaccumulate into reindeer hind muscle from feed concentrates. However, when considering reindeer liver, bioaccumulation of PCDD/Fs from reindeer feed concentrates can be seen. WHO-PCB-TEQs accumulated from reindeer feed concentrates to reindeer liver, and deviated from PCDD/Fs to muscle. WHO-PCDD/F-TEQs and WHO-PCB-TEQs accumulated from lichen to the muscle and liver of reindeer calves and hinds.

WHO-PCDD/F-TEQs did not accumulate from reindeer feed concentrates to milk, but WHO-PCB-TEQs did. WHO-PCDD/F-TEQs in the muscles of reindeer calves #1 and #2 did not deviate from their hinds; it was especially DL-PCBs which made the difference. However, there were higher WHO-PCDD/F-TEQs in the liver of calves than in their corresponding hinds' livers.

WHO-PCDD/F-TEQ and WHO-PCB-TEQ results in reindeer hind-calf pair #1 (lichen diet) showed that the compounds accumulated effectively from lichen to reindeer hind liver tissue, resulting in high WHO-PCDD/F-TEQ and WHO-PCB-TEQ concentrations in calf muscle and liver tissues. Bioaccumulation of WHO-PCDD/F-TEQs and WHO-PCB-TEQs from lichen to reindeer hind muscle was not as effective as to liver (IV).

5.5 Transplacental and lactational transfer of the compounds

5.5.1 Reindeer milk

In autumn of 2006, the mean wet weight-based PCB sum concentration of milk samples, 1.20 ng/g ww, was higher than the corresponding concentration of 0.50 ng/g ww in summer. The mean sum concentration of PCDD/Fs (0.70 pg/g ww) was also higher in autumn than in the summer sampling (0.20 pg/g ww). However, a mean PBDE of 0.006 ng/g ww in autumn was 80% less than 0.03 ng/g ww in summer. Non-*ortho*-DL-PCBs increased from 1.42 pg/g ww in summer to 2.78 pg/g ww in autumn (II).

The mean fat content in the autumn milk samples (26%) in 2006 was higher than in the summer milk samples (10%). The studied milk samples (III) also showed that the PCDD/F profile was generally similar in muscle and liver samples. The most dominating congeners were 23478-PeCDF, 12378-PeCDD, 123478-HxCDF, 1234678-HpCdd, and OCDD. There was a decreasing trend of most of the congener concentrations in reindeer milk samples from summer to autumn. However, 2378-TCDF, 1234678-HpCDD, and OCDD showed higher levels in autumn than in summer.

In the same year, the most prominent non-*ortho*-DL-PCB congener in reindeer milk (III) was PCB-126; this was true for both summer and autumn samples, when the mean concentrations were 5 and 2.7 pg/g fat, respectively. A clear decrease in the concentration of PCB-126 was observed from summer to autumn. A similar decreasing trend was also observed with PCB-169. However, PCB-81 increased significantly from summer to autumn, as was the case with PCB-77, which increased from not detected in summer to 0.97 pg/g fat in autumn. There was one exceptionally high PCB-77 concentration (6.79 pg/g fat) among the autumn samples. PCB-118 remained steady from summer to autumn; concentrations of PCB-114, PCB-156, and PCB-157 decreased, and PCB-123 increased during this time, the concentrations nevertheless being very low.

The congener profile of DL-PCBs in reindeer milk in 2006 fitted well to reindeer muscle and liver samples. the most toxic, DL-PCB-126 (TEF-value 0.1), was especially well represented.

In 2010 the most important PBDE congeners in reindeer milk were BDE-47, BDE-99, and BDE-153, reflecting the profile of analyzed reindeer tissue (muscle and liver). Some differences between the milk of hinds were observed; for instance, BDE-47 was well seen in the milk samples of hind #1 (lichen diet) but not as well in hind #2's (feed concentrates diet) milk samples (IV).

A proportion of BDE-209 was generally small in milk samples in 2010, except in sample #2 in early summer, when it represented 81% of the total PBDE sum (concentration 22.5 ng/liter).

The PBDE sum in samples in 2010 increased both in hinds #1 and #2 from control milk to early summer milk, but decreased then until the late summer sampling. Especially in hind #2, the change in the PBDE sum in milk was noticeable (IV).

5.5.2 Placentas, fetuses, and stillborn reindeer calves

Stillborn reindeer calves (sampled in 2006) represented the individuals who had gotten their contaminant load only via the placenta. The order of the wet weight-based mean sum concentrations in stillborn reindeer calves' muscle tissue was 1.2 ng/g ww (PCB), 0.1 ng/g ww (PBDE), and 0.8 pg/g ww (PCDD/F). Contaminant concentrations did not indicate dependency on muscle fat content in stillborn calves' muscle.

In brown adipose tissue (BAT) there was a similar proportion of substances to that of the other tissues: PCBs (ranging from 1.9 to 13 ng/g ww), PBDEs (ranging from 0.8 to 6.4 ng/g ww), and PCDD/Fs (ranging from 3 to 3.4 pg/g ww). The fat content of BAT in the middle sampling area (40%) was higher than in the southern sampling area, where it was 26%. Concentrations of compounds in BAT showed no correlations to the fat content. Accumulation of lipophilic PCDD/Fs, PCBs, and PBDEs via placenta to muscle and BAT of reindeer fetuses was clearly seen (II).

There was a high contribution of 23478-PeCDF in stillborn calves' muscle tissue from the middle and southern sampling areas in 2006. Of the studied BAT samples, it was seen that the mean 23478-PeCDF concentration (2.4 pg/g fat) was overwhelmingly highest of any congeners. However, other PCDD/F congeners also existed in BAT. Interestingly, some congeners including the most toxic one, 2378-TCDD, were lacking in muscle samples but represented in the brown adipose tissue (III).

PCB-77 was the only non-*ortho*-DL-PCB congener higher in stillborn calves' muscle than in BAT as well as other studied reindeer samples. From non-*ortho*-DL-PCBs, PCB-126 was the most dominating one in BAT, followed by PCB-169 (III). Non-*ortho*-DL-PCB sums were higher in hinds than in fetuses in the 2010 sampling. In the 2010 placenta samples, the most dominating DL-PCB congener was PCB-126 (IV).

In placenta and uterus sample #5, and also in fetus #5, PCDD/Fs were the most apparent compounds as WHO-TEQs in 2010. PCDD/Fs were also the dominating compounds in the blood samples of hinds #3 and #4, and in the placenta of hind #4. In placenta samples the most important PCDD/F congeners were 123678-HxCDD, 2378-TCDF, and 23478-PeCDF. Sample #5 (placenta and uterus) contained more existing PCDD/F congeners than the #3 and #4 placenta samples (IV).

WHO-TEQs (as upper bound values) were higher in the blood and placentas of hinds than in their muscle. TEQ concentrations of hinds #3 and #4 were higher in the blood than in the placentas. Hind-fetus pair #5 deviated from pairs #3 and #4, when the total WHO-TEQ was higher in the fetus than in the hind muscle.
In the livers of fetuses #3 and #4, WHO-PCDD/F-TEQs were dominating, and in the liver of hind #5, WHO-PCDD/F-TEQ was clearly dominating from the total TEQ. Also, fetus #5 (analyzed as complete) had more WHO-PCDD/F-TEQ than WHO-PCB-TEQ (IV).

Fetuses #3 and #4 had lower PCDD/F sum concentrations and WHO-PCDD/F-TEQs in their muscle than their hinds. The placentas of hinds #3 and #4 contained higher PCDD/F sums than the corresponding fetuses' muscle. However, when considering the total body burden of PCDD/Fs in fetuses (muscle and liver) there were no differences between WHO-PCDD/F-TEQ concentrations in fetuses and placentas (Figure 2).

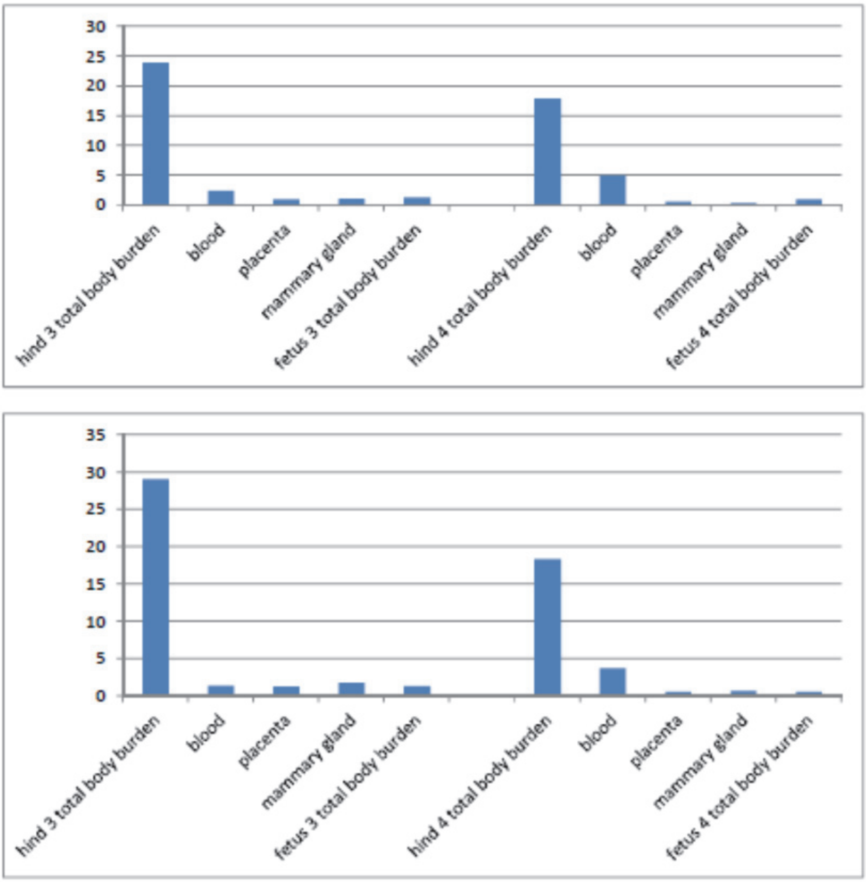

Fig. 3. WHO-PCDD/F-TEQs (toxic equivalencies of dioxins) and WHO-PCB-TEQs (toxic equivalencies of PCBs) (pg/g fat) in reindeer hinds, blood, placentas, and fetuses. Total body burden=muscle + liver.

Fetus #5 had a higher PCDD/F sum than what was found from its hind's muscle. However, hind #5's liver clearly contained the highest sum of PCDD/F (about 460 pg/g fat), so the total body burden was bigger in that reindeer hind. Fetuses #3 and #4 had much lower (over 100-fold) levels of PCDD/Fs in their liver than their hinds had (IV).

The PBDE sum (23 ng/g fat) was clearly highest in fetus #5. It had more PBDEs than the corresponding placenta and uterus, and the concentration was also higher than its hind's muscle and liver. Also, fetus #4 contained a higher PBDE level than what was its hind's total body burden. However, hind #3 and fetus #3 had similar levels of PBDEs in their bodies. In addition, PBDE concentrations were higher in fetuses #3 and #4 than in their corresponding placentas (Figure 3).

In reindeer liver samples in 2010, the highest PBDE sum concentration was detected in hind #5 (4.1 ng/g fat). In that sample the proportion of BDE-209 was as big as 96%.

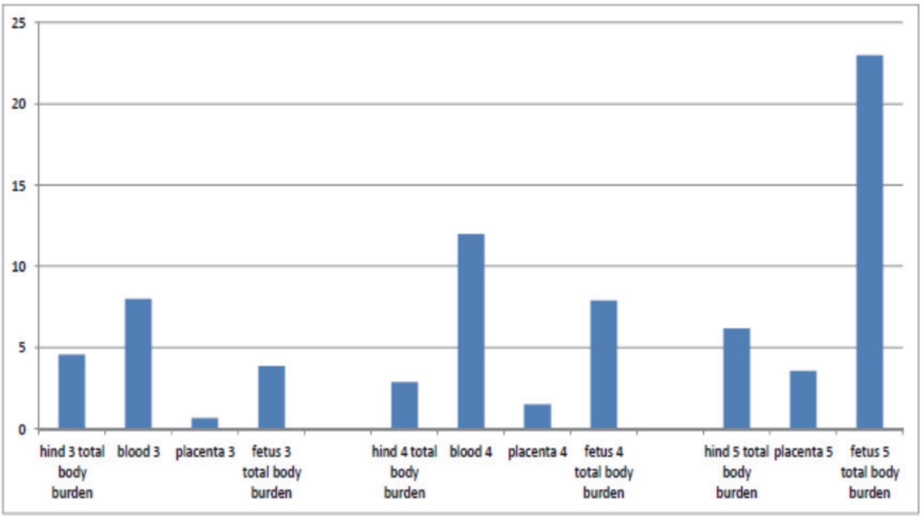

Fig. 4. PBDEs (Sum of 15 polybrominated diphenyl ether congeners) (ng/g fat) in reindeer hinds, blood, placentas, and fetuses. Total body burden=muscle + liver.

5.6 Safety of reindeer and moose meat as human food

In 2006 none of the measured WHO-TEQ concentrations exceeded the maximum limit value for WHO-PCDD/F-TEQ (3.0 pg/g fat in the meat of bovine animals), and in only 3 samples out of 29 individual reindeer calves, a level above the maximum limit of 4.5 pg/g fat for WHO-PCDD/F-PCB-TEQ was exceeded (I).

Of the average daily consumption of meat (126 g/day) (Männistö *et al.*, 2003), the consumption of reindeer meat is only 1.37 g/day. The average daily exposure via reindeer meat can be calculated as 0.003 pg WHO-PCDD/F-PCB-

TEQ/kg bw where the average body weight is 76 kg. For very high consumption of reindeer meat (178 g/day, where all the meat consumed is reindeer meat), a daily exposure of 0.4 pg WHO-PCDD/F-PCB-TEQ/kg bw can be calculated. This contributes to 25% of the total intake of PCDD/Fs and PCBs in the general Finnish population (1.5 pg WHO-PCDD/F-PCB-TEQ/kg bw per day; Hallikainen *et al*., 2006).

The high-consumption scenario for eating moose meat (178 g/day, where all the meat consumed is moose meat) gives a daily intake of 0.12 pg WHO-PCDD/F-PCB-TEQ/kg bw. The latter calculation is based on the average 0.05 pg/g WHO-PCDD/F-PCB-TEQ ww of moose meat. The human PCDD/F and PCB exposure via reindeer and moose meat is typically very modest, and even for the high consumption of reindeer meat the contribution exceeds 25% of the total intake (I).

The WHO-PCDD/F-TEQ concentration was higher in reindeer calves' livers in 2006 than in 2010; it was also higher than WHO-PCDD/F-TEQ in adult reindeer livers. However, WHO-PCB-TEQ has been increased from 2006 to 2010 in reindeer calf livers, as it has been in the adult livers. In addition, PCDD/Fs (as WHO-TEQs) were higher in the adult reindeer livers in 2010 than in 2006 (II, IV). The maximum limit of WHO-PCDD/F-PCB-TEQ for the liver of terrestrial animals is 10.0 pg/g fat in EU legislation (EU, 1881/2006). This is clearly exceeded in reindeer livers (V).

5.6.1 Lipid contents

In 2006, the adult reindeer had a higher fat content on average (4.9%) in the muscle samples than reindeer calves (4.7%); The liver samples of adult reindeer also showed a higher fat content on average (6.3%) than the reindeer calves' samples (6.1%). Differences between the fat contents of individual reindeer calves were statistically significant (P=0.013) (I). In 2010, reindeer calves had a higher fat percentage in their muscle (4%) and liver (5.4%) than hinds (2.9% and 4.5%, respectively) (IV, Table 1). Fat contents of fetuses' muscle (2.2%) and liver (1.8%) were lower than calves' and hinds'. The blood fat content (0.2% on average) was low (IV).

In the milk sample of hind #1 (lichen diet), the fat content increased from 12% in the control to 16.7% in early summer, but decreased again until late summer (10.5%). In the milk of hind #2 (feed concentrates diet), there was an increasing trend from control (10.3%) and early summer (12.9%) to late summer

(19.6%) sampling (IV). In 2006 the fat content of autumn milk was significantly higher than in the summer sampling (*P*=0.001) (II).

6 Discussion

6.1 Reindeer and moose samples 2006

The results showed a lower mean level of WHO–PCDD/F–PCB–TEQ in the muscle meat of reindeer calves than food monitoring conducted earlier (Kiviranta *et al*., 2006). Both an earlier study and the study in 2006 indicate that reindeer calves have higher PCDD/F and PCB concentrations on average than adult reindeer. A similar phenomenon has been observed as well in other animals, such as pigs and sheep (Fernandes *et al*., 2011), although also opposite results are seen, which indicate that young and immature animals have generally lower POP levels in their body than adult animals (O'Shea, 1999). In the middle sampling region, where the highest WHO-TEQ concentrations were found, reindeer hinds had grazed only in natural pastures. In the northern and southern regions, the pregnant female reindeer had received supplementary feed during winter. Dietary differences may affect the milk's chemical composition. In addition, in all three sampling regions, the reindeer calves born in spring had grazed only on natural pastures in the summer before they were slaughtered in autumn.

The adult reindeer from the southern sampling site, which had been given supplementary feed, had a somewhat lower WHO–PCDD/F–PCB–TEQ level compared to reindeer from the middle region. The lowest concentration was in the adult reindeer from the northern region, where reindeer had supplementary feed in winter. However, reindeer in the northern site received much less supplementary feed than reindeer in the southern region.

In individual reindeer calves from the northern and southern regions, and in adult reindeer from the northern region, the proportion of PCDD/Fs and PCBs of the total TEQ was 44% and 56%, respectively (I). In the middle sampling area, however, reindeer calves and adult reindeer had a higher contribution of DL-PCBs to the total TEQ than to the corresponding contribution in other sampling regions, namely 68% (I). This higher proportion of WHO–PCB–TEQ found from the individual reindeer from the middle region, could be due to differences in the feeding habits among the regions. Different fall-outs of PCDD/Fs and PCBs in the regions from where the supplementary feed had been imported to the southern and northern reindeer herding regions may be the other reason. The proportion of DL-PCBs of WHO-PCDD/F-PCB-TEQ was the smallest in the adult reindeer in the southern region, at 39% (I).

The reason for the different amount of contaminants remained unclear. A concentration of WHO-PCDD/F-PCB-TEQ was highest in calves from the middle sampling region. However, dietary differences most probably had effects to the levels observed. Animals which got their food straight from nature are exposed more to environmental contaminants than animals which got supplementary feed concentrates. The samples of pooled reindeer were collected from more western parts of the sampling regions than the samples from the individual reindeer. Differences in feeding habits (e.g., for the ratio of hay and feed concentrates) between the western and eastern parts of the regions might explain the differences in the contributions of PCDD/Fs and PCBs to total WHO-TEQ.

The fat-based WHO-PCDD/F-PCB-TEQ concentration in moose calves was lower than in reindeer calves, while in adult moose it was equal to adult reindeer (I). The number of moose samples was quite limited, however, and conclusions drawn from the concentration levels can only be indicative. The WHO-TEQ level in moose in 2006 was higher than in the earlier study of 2003–2005; however, the samples of moose meat in that earlier study were collected from the southeast part of Finland.

In addition to lower levels of contaminants in moose, some distinctive differences between the results from reindeer and moose could be detected. Firstly, the proportion of WHO-PCDD/F-TEQ compared to WHO-PCB-TEQ was higher at 63% in moose than in a majority of reindeer samples (I). This could be explained by the different feeding habits of reindeer and moose, and/or by the individual variations in metabolic activity and detoxification processes. Secondly, the concentrations of PCDD/Fs and PCBs in moose calves were lower than in adult moose, which was the opposite in reindeer samples (I, Fig. 2). Intake of contaminants by reindeer and moose calves occurs mainly via nursing in the early life stage, and reindeer calves are more exposed to PCDD/Fs and PCBs via milk (Ruokojärvi *et al*., 2007).

It is not known if there are differences in physiology and feeding habits between reindeer and moose that could have an effect on the levels of PCDD/Fs and PCBs in calves. Specific interest is focused on the lipid contents of animals. However, there were no differences between the lipid contents of reindeer and moose muscle meat. In the fat-based results, the positive correlation between fat content and contaminant levels was weak. The samples were collected in autumn when the animals have stored body fat during summer, and this might affect the fat–fatty-based contaminant correlation.

It is possible that feeding rhythm and food quality have effects on contaminant accumulation in reindeer and moose. Moose eat small amounts of food several times a day and intestinal content may not have so many lipid particles adhered with contaminants than reindeer have. A fast pass through the digestive tract may eliminate organic pollutants, such as PCDD/Fs and PCBs.

The differences in WHO-TEQ levels of reindeer and moose muscle samples in 2006 were mainly due to the differences in the levels of DL-PCBs. The concentrations of individual PCDD/F congeners were often below the limit of quantification (LOQ). A substantial portion of the WHO-PCDD/F-TEQ originates from LOQ alone, since results are reported as upper bound concentrations.

Comparing the fat contents of reindeer and moose meat (about 5%) to domestic animals in Finland, it is seen that they have quite a low fat percentage. Only beef meat has an equally low fat content; chicken meat, sheep meat, and especially pork meat have higher fat contents (ranging from 8.5% to 17%) (Fineli, 2013).

6.2 PCDD/Fs, PCBs, and PBDEs in reindeer tissue in 2006

There may be properties in the bioaccumulation of compounds resulting in elevated PCDD/F and PCB levels in reindeer calves compared to adult reindeer. Lactation as a physiological phenomenon may multiply concentrations in this particular section of the food chain, where the relative big burden of compounds is transported to organisms with advanced transport of dietary lipids (i.e., milk fat) and enter calves' growing body volume.

However, reindeer calves' growth is the most intense in their first summer (Nieminen, 1994), when the high chemical exposure may be partly hidden by dilution of contaminants in the growing tissue volume. If equilibrium partitioning of compounds according to a fat content of animal tissue is occurring, the highest contaminant levels should be in the fattest animals. The correlation between the wet weight-based concentrations and the fat content of reindeer muscle was the most obvious with PCBs, while PCDD/Fs showed only poor dependency on fat.

Colabuono *et al*. (2012) have reported higher PCB levels in the livers than in adipose tissue of migratory birds with poor body condition. Birds in good body condition had lower PCB levels in the liver and stored it to fat deposits, respectively. Generally, reindeer liver contained more PCDD/Fs than PCBs. Similar results have been observed in sheep samples, too (Fernandes *et al*., 2011).

There is evidence of increasing accumulation of PCDD/Fs to the liver of rats due to increased exposure of these compounds. Accumulation occurs in the microsomal fraction of the liver (Bell *et al*., 2007). The most important accumulation factor is CYP1A2 enzyme which is induced by dioxins. If induction occurs, dioxins accumulate in the liver (Diliberto *et al*., 1995); if this applies in reindeer, too, the higher PCDD/F (and possibly PCB) concentrations in calves may be explained by a temporal and high (in proportion to body volume) exposure via lactation in a stage in which there is a high number of free acceptors (Ah-receptors) in the liver.

Calves may have liver functions in a state where the developing detoxification system occurs with a high receptor protein synthesis that gathers toxicants in the liver. However, calf liver may have weaker detoxification functions than the adult that result in high concentrations of non-metabolized compounds. This study does not completely support a conception that PCDD/Fs, PCBs, and PBDEs accumulate in animal tissue via lipid partitioning only (Patterson *et al*., 1987). Instead, there could be a prominent function of other than lipid molecules, specifically CYP enzymes, in special tissues such as liver.

The effect of soil to contaminant concentrations in reindeer calves was not studied in this research, but there is a phenomenon, which may increase the POP load in calves. Newborn calves eat soil during their first three weeks of life to achieve a suitable microbial environment in their stomachs. This could enhance the contamination of calves.

The study of roe deer has shown that there is no correlation between contaminated soil and the contaminant load in liver. The roe deer's liver was highly loaded by WHO-PCDD/F-PCB-TEQ even if there was no contamination in the grazing area soil. Also, sheep livers in Germany have been highly contaminated with PCDD/Fs and DL-PCBs, and no correlation could be found between liver contamination and grassing areas or husbandry. It is postulated that a specific metabolism of sheep causes this enrichment of PCDD/Fs and PCBs in liver, and this is concluded to be the case with roe deer, too (Schröter-Kermani *et al*., 2011).

6.2.1 PCDD/Fs, PCBs, and PBDEs in reindeer milk

In 2006, sampling the levels of PCDD/Fs and PCBs in reindeer milk did not decrease in a comparable rate of fat content along with a course of lactation, and it constitutes a basic difference between human beings and reindeer. With human mothers, the contaminant levels have been observed to decrease as lactation continues (Kiviranta *et al*., 1999). In suckling lambs and pigs, mother's milk is considered an important source of dioxin contaminants, but it is also stated that this process is particularly marked at the onset of lactation and then gradually declines when maternal body burden decreases (Fernandes *et al*., 2011). It is possible that lactating reindeer get more contaminant load via green summer plants. However, it is assumed that fast-growing green plants, except the cucumber family, do not accumulate PCDD/Fs and PCBs effectively.

Reindeer calves have been estimated to be exposed to PCDD/Fs and PCBs considerably via milk (Ruokojärvi *et al*., 2007). In humans, a background exposure of breast-fed infants has been estimated to be up to two orders of magnitude higher than in adults, and the nursing infants absorb more than 90% of ingested PCDD/Fs and PCBs. The yield of reindeer milk may vary depending on environmental factors, and milk composition also varies naturally due to the lactation cycle. Lactation normally lasts from 24 to 26 weeks, and at that time the total milk production is approximately 100 kg (Gjøstein *et al*., 2004).

Opposite of PCDD/Fs and PCBs, PBDE concentration decreased from summer to autumn. This indicates that reindeer hind get rid of PBDEs effectively by lactation. On the other hand, when the fat content of reindeer milk increased from summer to autumn, it could be so that PBDEs were diluted in the bigger mass of fat and the dilution effect was seen as decreased concentrations. However, PCDD/Fs and PCBs did not show any dilution effect when their concentrations increased in milk from summer to autumn. There seem to be different kinds of behavior of dioxin-like and PBDE compounds.

6.2.2 PCDD/Fs, PCBs, and PBDEs in stillborn calves' muscle and BAT

Reindeer calves' brown adipose tissue develops during prenatal development and has a significant impact on calves' survival in spring time, when it is frequently cold. BAT is an energy store containing high vascularized adipocytes, which have a specific energy-producing protein system in their mitochondria (Cannon & Nedergaard, 2004). Contaminants are supposed to accumulate in the developing calf when the female's lipid stores are used for energy production and tissue forming in the calf. Reindeer hinds lose a part of their fat stores during winter (Nieminen *et al*., 1984) and if lipid partitioning of compounds occurs, it is obvious that they accumulate in offspring, who have brown adipose tissue with high blood vessel density and a live changing of particles and molecules transmitted by blood. Lipophilic contaminants are assumed to pass easily through the cell membranes and achieve BAT. The space that a fetus comprises acts as a potential recipient of partitioning contaminants. Since calves lose their BAT in the first month postpartum, the toxic effects of POPs may manifest due to mobilization and subsequent distribution of contaminants in the tissues, in muscle, for example.

6.3 Congener-specific PCDD/Fs and DL-PCBs in reindeer and moose in 2006

Octachlorodibenzo-*p*-dioxin (OCDD), 2378-TCDF, and 1234678-HpCDD seemed to exist in the muscle samples, but not in the brown adipose tissue, of stillborn reindeer calves. Highly chlorinated congeners such as 1234678-HpCDD and OCDD are generally considered to accumulate well in lipid-rich tissue, such as brown adipose (where the fat content on average is 30%), so the lack of these congeners is interesting.

OCDD concentration was noticeably high in male calf samples from the northern zone. That was a parallel result to the reindeer calf muscle and liver samples from the same sampling zone, and indicated high OCDD exposure in the northern part of Finnish Lapland. Very similar concentrations of 23478-PeCDF, 123478-HxCDF, 123678-HxCDF, and 234678-HxCDF in the livers of the southern zone's reindeer calves and adults could indicate an equal amount of binding sites for dioxins in the liver.

For comparison, some of these congeners found in reindeer liver, namely 23478-PeCDF, 1234678-HpCDD, and OCDD, have been the most general congeners in roe deer liver samples in Germany (Schröter-Kermani *et al.*, 2011). Roe deer's feeding behavior is very similar to that of reindeer: It uses a variety of grasses, lichens, mushrooms, berries, twigs, branches, buds, and shoots. In this study, OCDD had a tendency to exist in high concentrations in reindeer calf muscle and liver samples from the northern area. This may indicate a high local exposure to OCDD in Northern Finland, considering that adult reindeer in the same area also had proportionately high OCDD concentrations, especially in their livers.

Lower OCDD and 1234678-HpCDD concentrations in Northern Lapland's reindeer and moose samples may indicate higher metabolic activity and elimination potential of these congeners in adult animals. In Middle and Southern Lapland, exposure, especially to OCDD, may be lower, which is reflected by lower contamination levels in both calves and adults. Concentration of 2378-TCDF was higher in the adult moose than in moose calves, an opposite result than that of reindeer.

DL-PCB concentrations were generally higher in calf liver samples than in adult reindeer, indicating that the calf liver functions in a state of effective accumulation of toxicants and weak detoxification; this results in high concentrations of non-metabolized compounds in calf livers. However, concentrations of PCB-77 were higher in adult reindeer livers than in calves' livers.

PCB-169 existed in many of the samples, the highest level shown in the stillborn calf of the middle area. Overall, the non-*ortho*-DL-PCB profile in reindeer liver fitted well to reindeer muscle samples. PCB-77 concentrations in moose samples were significantly lower in females than in males in every zone, indicating excretion of compounds via lactation.

6.4 Congener-specific PCDD/Fs and DL-PCBs in reindeer milk in 2006

PCDD/Fs in reindeer milk samples (III) showed a similar profile to muscle and liver samples. OCDD, which showed the highest total contribution of PCDD/Fs, interestingly increased from not detected in summer to 0.6 pg/g fat on average in autumn milk samples. Despite the increasing fat content of milk during the lactation period, the levels of some particular congeners were increased, indicating no dilution effect.

The most prominent non-*ortho*-DL-PCB congener in reindeer milk (III) was PCB-126; this is true of both summer and autumn samples. A clear decrease in the concentration of PCB-126 was seen from summer to autumn. The congener profile of DL-PCBs in reindeer milk was again fitted well to reindeer muscle and liver samples. Especially the most toxic, DL-PCB-126 (TEF-value 0.1), was well represented. However, even emphasizing the importance of the lactational transfer of persistent organic compounds it is worth noting that the highest concentrations of PCB-126 and PCB-169 were found from a stillborn calf that had gotten its body burden only via the placenta.

The mean fat content in autumn milk samples (26%) was higher than in summer milk samples (10%) which may influence the lipid-based concentrations detected. The fat content of reindeer milk normally varies from 13% to as much as 30% during the lactation process (Luhtala *et al.*, 1968). The concentrations of PCDD/Fs and DL-PCBs are shown in Table 5.

Table 5. PCDD/F and DL-PCB congeners in reindeer milk samples.

Congener	Summer milk	Autumn milk
2378TCDF	0.011	0.076
2378TCDD	0.010	0.000
12378PeCDF	0.033	0.000
23478PeCDF	0.260	0.143
12378PeCDD	0.143	0.016
123478HxCDF	0.160	0.044
123678HxCDF	0.063	0.024
234678HxCDF	0.046	0.000
123789HxCDF	0.000	0.000
123478HxCDD	0.035	0.000
123678HxCDD	0.124	0.023
123789HxCDD	0.000	0.000
1234678HpCDF	0.000	0.000
1234789HpCDF	0.000	0.000
1234678HpCDD	0.098	0.126
OCDF	0.000	0.000
OCDD	0.000	0.589
PCB-77	0.000	0.970
PCB-81	0.062	0.364
PCB-126	5.070	2.653
PCB-169	0.798	0.511
PCB-105	0.176	0.122
PCB-114	0.012	0.008
PCB-118	0.404	0.390
PCB-123	0.000	0.005
PCB-156	0.091	0.060
PCB-157	0.015	0.008
PCB-167	0.025	0.022
PCB-189	0.009	0.009

6.5 Reindeer samples 2010

Total WHO-TEQs were higher in the muscle tissue of reindeer calves than in hinds (1.1 pg/g fat on average). This is probably the consequence of effective intake and distribution of PCDD/Fs, especially dioxin-like PCBs, in a calf after the hind had excreted and transferred compounds into milk. The concentrations in milk samples, (especially of hind #1) being higher than the concentrations in hinds' muscle, support this idea (IV).

Hind-fetus pairs #3 and #4 showed that total TEQs were lower in the fetuses' muscle and liver than in their corresponding hinds (Table 2). This probably indicates the effective barrier of placentas to toxic compounds. Hind-fetus pair #5 deviated from pairs #3 and #4, when the total TEQ was higher in the fetus (analyzed as a complete body) than in hinds' muscle, although the sampled fetus was younger (about 100 days) than fetuses #3 and #4 (about 200 days). That may indicate some effective POP collector tissue in the fetus. However, TEQs in the placenta of hind #5 were similarly higher than in hind muscle, as it was in pairs #3 and #4.

Calves #1 and #2 had higher WHO-PCDD/F-TEQs and WHO-PCB-TEQs than their corresponding hinds, but fetuses #3 and #4 had lower WHO-TEQs than hinds. The undeveloped liver functions of fetuses probably reflect as low contaminant burdens in their livers. In the liver of hind #5 WHO-PCDD/F-TEQ was clearly dominating from the total TEQ. Also as complete analyzed fetus #5 had more WHO-PCDD/F-TEQ than WHO-PCB-TEQ. This may be a consequence of sampling procedure: Hind-fetus pair #5 were sampled earlier and from a different location than other samples.

WHO-PCDD/F-TEQ did not accumulate in milk from reindeer feed concentrates, but WHO-PCB-TEQ did. This may be the result of rumen's ability to produce fatty acids containing DL-PCBs, transport them to the blood circulation, and finally to the excretive route of lactation. The overall consequence is higher WHO-PCB-TEQ levels in reindeer calf muscle and liver than corresponding concentrations in reindeer hind tissue.

Bioaccumulation of WHO-PCDD/F-TEQs and WHO-PCB-TEQs from lichen to reindeer hind muscle is not so effective as to liver. This may be the result of effective fatty acids and contaminants adhered, gathering functions of liver in lichen digestion from the rumen in adult reindeer. The overall observation was that WHO-PCDD/F-TEQs and WHO-PCB-TEQs were accumulated in reindeer calves on both diets, but it was especially noticeable in the lichen diet. However, it remained open if there were some other routes (such as atmospheric deposition or water supply) for PCDD/Fs and PCBs getting to the reindeer food chain.

Placentas of hinds #3 and #4 contained higher PCDD/F sums (about 5 times higher than the PCDD/F sum in fetuses' muscle) than the corresponding fetuses, which may indicate an effective barrier to PCDD/F transport from hind to fetus, at least in specific cases.

Fetuses #3 and #4 had significantly lower (over 100-fold) levels of PCDD/Fs in their livers than their hinds had. This observation may mean that liver activity in relation to the accumulation of toxic contaminants increases significantly after birth and during the first living months of reindeer calves.

In reindeer liver samples, the highest PBDE sum concentration was detected in hind #5. In that sample, a proportion of BDE-209 was as high as 96%. It is worth noting that PBDE sum concentrations were generally lower in reindeer liver samples than in other samples, including in the muscle tissue of fetuses. An exception was hind #5, whose liver contained more PBDEs than its corresponding muscle sample. It seems that PBDEs are transported to muscle tissue to a greater extent than in liver tissue, and that may be the result of transporting them via lymphatic fluid. On the other hand, there may not be receptors to PBDEs in the liver and hence they do not accumulate at the same rate than dioxin-like compounds.

In 2010, reindeer milk sampling WHO-PCDD/F-TEQ and PCB-TEQ decreased in hind #2 (feed concentrates diet) from control to late summer. With hind #1 (lichen diet) there was an increasing trend with WHO-PCB-TEQ from control to late summer (IV).

Hind #2 especially showed changes in the PBDE sum in milk, indicating an effective excretion of PBDEs via milk in reindeer. In milk #2 the fat content increased from early to late summer, which may affect the PBDE levels detected in the samples.

Interestingly, in the milk of reindeer hind #2, the quantity of congeners decreased dramatically during the lactation period, while in the milk of hind #1 the congener pattern remained very stable. That may be the result of the different diets of these hinds (hind #1 on the lichen diet and hind #2 on the reindeer food diet).

The most important PCDD/F congeners in lichen were OCDD, 1234678-HpCDD, 1234678-HpCDF, and OCDF, and in reindeer feed concentrates OCDD and 1234678-HpCDD. It was interesting that OCDD concentrations in milk samples were very low and did not accumulate in milk. However, in the reindeer muscle and liver samples OCDD was found, which indicates another possible exposure route, such as water supply, than lactation. In an earlier study (Suutari *et al.*, 2012), OCDD was found to exist in a rather high concentration in reindeer milk (0.6 pg/g fat) even if it was not detected in summer milk. That indicates an exposure peak to OCDD, which was observed to be one of the major congeners in deposition in Northern Finland (Kiviranta, 2005).

PCB-126 is the most toxic congener of dioxin-like PCBs having a TEF-value of 0.1. Its proportionally high level in the liver tissue may indicate liver function as a main organ in the intoxication process. Hence, a high concentration of the most toxic compound is seen when adherence to the dioxin-receptor is effective.

PCB-52 and PCB-101 were at a high level in reindeer feed concentrates. PCB-99, which was at a high level in the milk samples of hind #1 (lichen diet), was at a somewhat lower level in lichen which may indicate some other exposure route (e.g., hay) for this congener than lichen.

A proportion of BDE-209 was generally minor in milk samples except in #2 in early summer, when its share of the total sum was 81%. That indicates quite a strong exposure of hind #2 to BDE-209 in the experimental zoo in early summer, because the BDE-209 in the control milk of hind #2 was not detected. Considering the reindeer feed concentrates given to hind #2, it was observed that it contained 94% of BDE-209 from the total PBDE sum. Comparing that to the PBDE sum in lichen it was quite high and may explain the high BDE-209 in early summer milk.

6.6 PCDD/Fs, PCBs, and PBDEs in the organs of reindeer and moose

Of the studied moose samples, a young adult moose from Central Finland had the highest WHO-PCDD-TEQ in its liver. That may indicate a higher exposure to PCDD/Fs in more southern regions in Finland (V).

Low concentrations of WHO-PCB-TEQs of the calves and adult moose correlated with WHO-PCDD/F-TEQs: One moose calf and one adult had lower WHO-PCB-TEQ concentrations than the other samples studied from Northern Finland. Lower WHO-PCB-TEQs of these samples may indicate different exposure conditions to PCBs. The adult moose from Northern Finland had the lowest WHO-PCB-TEQ from the all samples studied, as well as the lowest WHO-PCDD/F-TEQ (V). It has been found in earlier studies that Finnish reindeer calves' livers contained more PCDD/Fs and PCBs than adult reindeer livers, which could be the case for moose as well (Ruokojärvi *et al.*, 2011).

The fat contents of the moose liver samples were very similar (on average 5.3%), so they do not explain variation in contamination levels. The conception that PCDD/Fs, PCBs, and PBDEs accumulate in liver tissue via lipid partitioning only is not supported by this study: Instead, there may be other functionally important molecules such as CYP enzymes in a special tissue such as liver. In this study we do not have information of the effectiveness of moose liver enzymes in relation to contaminant accumulation. It is known that CYP1A2 enzyme is a high-affinity binding protein to dioxin-like compounds, and binding to this protein restricts the metabolic processes by other enzymes (e.g., CYP1A1). Hence, the compounds are stored in the liver (Diliberto *et al*., 1995).

The highest PCDD/F sum was detected in a young adult moose from Central Finland and the highest toxicity (as WHO-PCDD/F-TEQ) was found in the same individual. The highest PCB sum and also the highest indicator-PCB sum were, for their part, found in a female calf from Northern Finland. Adult moose from Northern Finland seemed to have somewhat lower PCB concentrations than calves from the same area, but generally it seems that there is stronger exposure to PCBs there than in Southern Finland. That is supported also by the observation that WHO-PCB-TEQs were generally higher in Northern than in Southern Finland. On the other hand, there is an interesting variation between the calf samples (#1, #2, #3, and #4) from the north, when they had almost equal PCB sums but toxic load (as WHO-PCB-TEQ) was clearly lower in sample #2. That may indicate exposure to varying PCB congeners in that region.

In the previous study it was observed that PBDE concentrations were similar in calf and adult reindeer livers, but slightly higher in the livers of fetuses than their corresponding hinds (Ruokojärvi *et al*., 2011). In this study, PBDE concentrations were higher on average in the livers of moose calves than in adults. In addition, PBDE concentrations were very low in general, below 0.5 ng/g fat in all moose liver samples. Very similar PBDE concentrations have also been found from Finnish semi-domesticated reindeer liver samples, although occasional higher concentrations have been found (Ruokojärvi *et al*., 2011).

WHO-PCDD/F-PCB-TEQ in moose muscle samples in Sweden (8.1 pg/g fat on average) (Danielsson *et al*., 2008) was shown at a higher level than moose muscle (I) and also moose liver samples in Finland. That may indicate that Finnish moose are less exposed to PCDD/Fs and PCBs. However, contaminations of Finnish reindeer liver do not support the idea of minimal environmental pollution in Finland. There can also be some individual metabolic differences, which influence the contamination levels in moose. Considering PBDEs, it seems that they accumulate as high concentrations in moose muscle but less in liver (V, Fig. 1). Nutritional factors may also have a role in the accumulation of PBDEs in moose muscle. It is known that moose prefer willow in their diet, and it has been found to be an ideal atmospheric PBDE passive sampler (Hu *et al*., 2011). PBDE accumulation in moose muscle may occur when they are transported with chylomicrons in lymph-to-muscle tissue. It may also be so those PBDEs pass the liver without binding to CYP-enzymes and further accumulate in muscle tissue.

There may be a higher expression rate for CYP1A2 in reindeer liver than in moose liver, which can have an effect on concentrations of dioxin-like compounds seen in the livers. The amount of CYP enzymes varies between the species naturally and animals vary widely in their capacities to biotransform absorbed xenobiotics (Oser, 1981; Caldwell, 1989). There can also be differences between the contaminant levels in the diets of reindeer and moose. Reindeer's important winter food supply, lichen, effectively gathers toxicants which are deposited in the ground.

Comparing PBDE concentrations in different organs of reindeer, it was observed that the highest concentration (29 ng/g fat) was in the lymph nodes. Abdominal fat, kidneys, and bone marrow showed lower PBDE levels (ranging from 0.18 to 0.59 ng/g fat). Of the PBDEs, the dominating congener was BDE-209, which contributed very intensively, especially in the lymph nodes (V). It may be so that PBDEs are transported well in the lymphatic fluid of reindeer and its accumulation occurs in the lymph nodes. It may also be possible that PBDE accumulation disturbs the normal functions of lymph nodes and affects the resistance of diseases.

6.7 Food safety

In this study it was observed that food safety of reindeer and moose meat is good. In 2006, none of the measured WHO-TEQ concentrations exceeded the maximum limit value for WHO-PCDD/F-TEQ and in only 3 samples out of 29 individual reindeer calves, a level above the maximum limit of 4.5 pg/g fat for WHO-PCDD/F-PCB-TEQ was exceeded (I). However, considering reindeer livers it can be noticed that the maximum level of WHO-PCDD/F-PCB-TEQ for the liver of terrestrial animals, 10.0 pg/g fat, is exceeded clearly in reindeer livers (V).

6.8 Biomonitoring

It was shown in this study that Finnish semi-domesticated reindeer and wild moose are good indicator species of POP contamination in the terrestrial environment, reindeer describing the situation in northern parts of the country especially. This is particularly true when considering the areal differences between reindeer samples; the results showed that the highest dioxin concentrations were in reindeer in the middle sampling area, where animals were not given any supplementary feed.

7 Conclusions and future objectives

The study hypotheses were answered as the following:

1. There are differences between the contaminant concentration in reindeer and moose. Reindeer calves had on average higher PCDD/F and PCB concentrations than adult reindeer, moose, and moose calves. PBDE concentrations were higher on average in adult reindeer than in reindeer calves. The highest PBDE concentration was detected in the muscle of moose calves.
2. There are individual and interspecies differences in accumulation and congener-specific distribution of PCDD/Fs, PCBs, and PBDEs. The content of contaminants affects the distribution, too. General PCDD/F congeners in reindeer muscle and liver were 23478-PeCDF, 123678-HxCDD, and OCDD. OCDD was common also in moose muscle samples. The most dominating DL-PCB congener in reindeer muscle and liver and moose liver was PCB-126. In moose muscle samples, PCB-77 was the most abundant congener. Of PBDEs, BDE-47 and BDE-209 were the most conspicuous congeners in moose samples, and BDE-47, BDE-99, BDE-153, and BDE-209 in reindeer muscle samples.
3. There are spatial differences in contaminant concentrations. Both moose and reindeer are indicators of environmental pollution. Moose represent the whole country and reindeer represent Lapland, where in different areas, supplement feed may lower the exposure. Persistent organic pollutants accumulated in the reindeer food chain more with the natural lichen diet and had higher contaminant concentration in their bodies than supplementary-fed reindeer. That was obvious, especially with reindeer calves in Middle Lapland.
4. Persistent organic pollutants transfer via the reindeer food chain. This was clearly seen, especially in reindeer with the lichen diet: The compounds accumulate effectively from lichen to reindeer hind liver tissue and also to milk, and further to calf muscle and liver tissues.
5. PCDD/Fs and PCBs accumulated in reindeer calves by milk and PBDEs to fetuses by the placenta. Lactational transfer seemed to be more important in transferring PCDD/Fs and DL-PCBs, when PBDEs were effectively transported by the placenta.
6. According to this study it is safe to eat reindeer and moose meat. Reindeer liver should be avoided, because of the high levels of dioxin-like compounds.

7. In the future there could be an important task to investigate more properly the health outcomes linked to observing POP concentrations in reindeer and moose, and also in humans who consume various organs of reindeer and moose as food. In addition, it is reasonable to continue POP monitoring with reindeer and moose, as they have been proved to be excellent indicators of environmental contaminants.

References

Abraham K, Wiesmüller T, Hagenmaier H, Neubert D (1990) Distribution of PCDDs and PCDFs in various tissues of marmoset monkeys. Chemosphere 20: 1071-1078.

Ahlborg UG, Becking GC, Birnbaum LS, Brouwer A, Derks HJGM, Feeley M, Color G, Hanberg A, Larsen JC, Liem AKD, Safe SH, Schlatter C, Wærn F, Younes M, Yrjänheikki E (1994) Toxic equivalency factors for dioxin-like PCB's. Chemosphere 28: 1049-1067.

Alcock RE & Jones KC (1996) Dioxins in the environment: A review of trend data. Environ Sci & Technol 30: 3133-3143.

AMAP (1998) Hansen JC, Gilman A, Klopov V, Odland JO (eds.) Pollution and Human Health. In: AMAP Assessment Report: Arctic Pollution Issues, p. 775-844. Arctic Monitoring and Assessment Programme, Oslo, Norway.

AMAP (2003) AMAP Assessment 2002: Human Health in the Arctic. Arctic Monitoring and Assessment Programme (AMAP) Oslo, 137 p.

AMAP (2004) AMAP Assessment 2002: Persistent Organic Pollutants in the Arctic. Arctic Monitoring and Assessment Programme (AMAP) Oslo, 309 p.

Anderson HA &Wolff MS (2000) Environmental contaminants in human milk. J Expo Anal Environ Epidemiol 10: 755-760.

Assmuth T & Louekari K (2001) Research for management of environmental risks from endocrine disrupters-contexts, knowledge base, methodologies and strategies. Finnish Environment Institute. Oy Edita Ab, Helsinki.

Bell D, Clode S, Fan M, Fernandes A, Foster P, Jiang T, Loizou G, MacNicoll A, Miller B, Rose M, Tran L (2007) Toxicity of 2,3,7,8- tetrachlorodibenzo-p-dioxin (TCDD) in the developing male Wistar (Han) Rat II: Chronic dosing causes developmental delay. Toxicol Sci 99: 214-233.

Bergman Å, Ryden A, Law RJ, de Boer J, Covaci A, Alaee M, Birnbaum L, Petreas M, Rose M, Sakai S, Van den Eede N, van der Veen I (2012) A novel abbreviation standard for organobromine, organochlorine and organophosphorus flame retardants and some characteristics of the chemicals. Environ Int 49: 57-82.

Bignert A, Danielsson S, Odsjö T, Remberger M (2008) Organic contaminants in moose (Alces alces) and reindeer (Rangifer tarandus) in Sweden from the past twenty years. Swedish monitoring programme in terrestrial biota. Rapport till Naturvårdsverket 43: 09-25.

Birnbaum LS (2013) Health effects of BFR: cause for concern! Sixth international symposium on flame retardants, San Francisco, California. Presentation.

Branchi I, Alleva E, Costa LG (2002) Effects of perinatal exposure to a polybrominated diphenyl ether (PBDE 99) on mouse neurobehavioral development. Neurotoxicology 23: 375-384.

Brandt I, Jönsson CJ, Lund BO (1992) Comparative studies on adrenocorticolytic DDT-metabolites. Ambio 21: 602-605.

Breivik K, Sweetman A, Pacyna JM, Jones KC (2002) Towards a global historical emission inventory for selected PCB congeners – a mass balance approach. 1. Global production and consumption. Sci Total Environ 290: 181-198.

Brzuzy LP & Hites RA (1996) Global mass balance for polychlorinated dibenzo- p-dioxins and dibenzofurans. Environ Sci Technol 30: 1797-1804.

Caldwell J, Weil A, Tanaka Y (1989) Species differences in xenobiotic conjugation, p. 217-224. In: Kato R, Estabrook RW and Cayen MN (Eds.), Xenobiotic metabolism and disposition (Proc. 2nd Int. ISSX Meeting, Kobe; 1998). Taylor and Francis, New York.

Cannon B & Nedergaard J (2004) Brown Adipose Tissue: Function and Physiological Significance. Physiol Rev 84: 278-337.

Carpenter DO, Arcaro KF, Bush B, Niemi WD, Pang S, Vakharia DD (1998) Human health and chemical mixtures: an overview. Environ Health Perspect, 106: 1263-1270.

Casals-Casas C, Desvergne B (2011) Endocrine disruptors: From endocrine to metabolic disruption. Annu Rev Physiol 73: 135-162.

Chen G & Bunce NJ (2003) Polybrominated diphenyl ethers as ah receptor agonists and antagonists. Toxicol Sci 76: 310-320.

Colabuono FI, Taniguchi S, Montone RC (2012) Organochlorine contaminants in albatrosses and petrels during migration in south Atlantic Ocean. Chemosphere 86: 701-708.

Cuthill S, Wilhelmsson A, Mason GGF, Gillner M, Poellinger L, Gustafsson J-A (1988) The dioxin receptor: A comparison with the glucocorticoid receptor. J Steroid Biochem 30: 277-280.

Dallaire F, Dewailly É, Laliberte C, Muckle G, Ayotte P (2002) Temporal trends of organochlorine concentrations in umbilical cord blood of newborns from the lower north shore of the St. Lawrence river (Québec, Canada). Environ Health Perspect 110: 835-838.

Danielsson S, Odsjö T, Bignert A, Remberger M (2008) Organic contaminants in moose (Alces alces) and reindeer (Rangifer tarandus) in Sweden from the past twenty years. Comments concerning the national Swedish contaminant monitoring programme in terrestrial biota. Report nr 7:2008. Swedish Museum of Natural History. 43 p.

Darnerud PO, Eriksen GS, Johannesson T, Larsen PB, Viluksela M (2001) Polybrominated diphenyl ethers: occurrence, dietary exposure, and toxicology. Environ Health Perspect 109: 49-68.

de March BGE, de Wit CA, Muir DCG, Braune BM, Gregor DJ, Norstrom RJ, Olsson M, Skaare JU, Stange K (1998) Persistent organic pollutants. In: AMAP Assessment Report. Arctic Pollution Issues, Chapter 6, p. 183-372. Arctic monitoring and assessment programme, Oslo, Norway.

DeVito MJ & Birnbaum LS (1995) The importance of pharmacokinetics in determining the relative potency of 2,3,7,8-tetrachlorodibenzo-p-dioxin and 2,3,7,8-tetrachloro-dibenzofuran. Fundam Appl Toxicol 24: 145-148.

de Wit C A, Muir D (2010) Levels and trends of new contaminants, temporal trends of legacy contaminants and effects of contaminants in the Arctic: Preface. Sci Total Environ 408: 2852-2853.

de Wit CA, Alaee M, Muir DCG (2006) Levels and trends of brominated flame retardants in the Arctic. Chemosphere 64: 209-233.

de Wit CA (2002) An overview of brominated flame retardants in the environment. Chemosphere 46: 583-624. Diliberto JJ, Akubue PI, Luebke RW, Birnbaum LS (1995) Dose-response relationships of tissue distribution and induction of CYP1A1 and CYP1A2 enzymatic activities following acute exposure to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in mice. Toxicol Appl Pharmacol 130: 197-208.

EU (2001) Commission Regulation 2001/C 322/02.

EU (2006) Commission Directive 2006/13/EY.

EU (2006) Commission Regulation (EU) No 1881/2006.

EU (2012) Commission Regulation (EU) No 277/2012.

Evira (2007) Finnish Food Safety Authority.

Fernandes AR, Foxall C, Lovett A, Rose M, Dowding A (2011) The assimilation of dioxins and PCBs in conventionally reared farm animals: Occurrence and biotransfer factors. Chemosphere 83: 815-822.

Fiedler H (1996) Sources of PCDD/PCDF and impact on the environment. Chemosphere 32: 55-64.

Fineli (2013) Finnish Food Composition Database, Release 16 (December 9).

Finfood. www.lihatiedotus.fi/www/fi/tilastot/lihan_kulutus_suomessa.php. 11.8.2014.

Fisk AT, de Wit CA, Wayland M, Kuzyk ZZ, Burgess N, Letcher R et al (2005) An assessment of the toxicological significance of anthropogenic contaminants in Canadian arctic wildlife. Sci Total Environ 351-352: 57-93.

Foster W, Chan S, Platt L, Hughes C (2000) Detection of endocrine disrupting chemicals in samples of second trimester human amniotic fluid. J Clin Endocrinol Metab 85: 2954-2957.

Fowles JR, Fairbrother A, Baecher-Steppan L, Kerkvliet NI (1994) Immunologic and endocrine effects of the flame-retardant pentabromodiphenyl ether (DE-71) in C57BL/6J mice. Toxicol 86: 49-61.

Franklin J, Atkinson R, Howard PH, Orlando JJ, Seigneur C, Wallington TJ, Zetzsch C (2000) Quantitative determination of persistence in air. *In: Evaluation of persistence and long-range transport of organic chemicals in the environment.* SETAC Special Publication Series, pp. 7-62. SETAC Books, Pensacola, FL, USA.

Geyer HJ, Schramm K-W, Feicht EA, Behechti A, Steinberg C, Bruggemann R, Poiger H, Henkelmann B, Kettrup A (2002) Half-lives of tetra-, penta-, hexa-, hepta-, and octachlorodibenzo-*p*-dioxin in rats, monkeys, and humans-a critical review. Chemosphere 48: 631-644.

Gjøstein H, Holand Ø, Weladji R (2004) Milk production and composition in reindeer (Rangifer tarandus tarandus): Effect of lactational stage. Comp Biochem Physiol 137: 649-656.

Hallgren S, Sinjari T, Hakansson H, Darnerud PO (2001) Effects of polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) on thyroid hormone and vitamin A levels in rats and mice. Arch Toxicol 75: 200-208. Hallikainen A, Parmanne R, Kiviranta H, Vartiainen T (2006) Voiko silakkaa edelleen syödä? Dioksiinien saanti elintarvikkeista arvioitu uudelleen. Duodecim 122: 801-4.

Hallikainen A, Kiviranta H, Isosaari P, Vartiainen T, Parmanne R, Vuorinen PJ (2004) Kotimaisen järvi- ja merikalan dioksiinien, furaanien, dioksiinien kaltaisten PCB-yhdisteiden ja polybromattujen difenyylieettereiden pitoisuudet. Elintarvikeviraston julkaisuja 1/2004. Edita Express, Pasila, Helsinki.

Hansen JR, Hansson R, Norris S (1996) The state of the European arctic environment. EEA environmental monograph No. 3, Oslo, Norway. 136 p.

Hansen JC (2006) Environmental medicine in an arctic perspective: “The arctic dilemma” Revisited. Int J Circumpolar Health 4: 365-368.

Hario M, Himberg K, Hollmen T, Rudbäck E (1999) Polychlorinated biphenyls in diseased lesser blackbacked gull (Larus fuscus fuscus) chicks from the Gulf of Finland. Environ Pollut 107: 5360.

Hario M, Hirvi JP, Hollmén T, Rudbäck E (2004) Organochlorine concentrations in diseased vs. healthy gull chicks from the northern Baltic. Environ Pollut 127: 411-423.

Hassan AEA (2012) Nutrients and toxic elements in semi-domesticated reindeer in Norway. PhD dissertation. University of Tromso. 62 p.

Hirako M (2007) Distribution of PCDDs, PCDFs and dioxin-like PCBs in the blood, testis and adipose tissue of suckling beef calves. Chemosphere 71: 219-226.

Hirvi JP (2004) Haitallisten aineiden esiintyminen metsämaan humuksessa. Abstract: Content of harmful substances in forest humus layer in Finland. Seppälä, J. & Idman, H. (Eds.). Maaperänsuojelu: Geologian tutkimuskeskuksen ja Suomen ympäristökeskuksen tutkimusseminaari 5.11.2004. Helsinki, Suomen ympäristökeskus. Suomen ympäristö 726. S. 7076. ISBN 952111830X, ISSN 12387312. http://www.ymparisto.fi/default.asp?contentid=105943&lan=fi Hlimi T (2012) Traditional food consumption behavior and concern with environmental contaminants among Cree schoolchildren of the Mushkegowuk territory . Int J Circumpolar Health 71: 17344.

Holand Ø, Gjøstein H, Aikio P, Nieminen M, White RG (2002) Traditional reindeer milking and prospects of developing reindeer dairy farming as a niche based production. In: Roginski H, Fuquay JW, Fox PF. (Eds.) Encyclopedia of dairy science, vol 3 Academic, London, p. 637-643.

Ikonomou MG, Rayne S, Addison RF (2002) Exponential increase of the brominated flame retardants, polybrominated diphenyl ethers, in the Canadian Arctic from 1981 to 2000. Environ Sci Technol 36: 1886-1892.

IPCS (1993) Environmental health criteria 140, polychlorinated biphenyls and terphenyls, 2nd ed. international programme on chemical safety, World Health Organization, Geneva.

IPCS (1994) Brominated diphenyl ethers. In: Environmental health criteria 162. International programme on chemical safety, World Health Organization, Geneva.

Jensen J, Adare K, Shearer R (Eds.) (1997) Canadian arctic contaminants assessment report, northern contaminants program, minister of Indian affairs and northern development, Ottawa, 460 p.

Jenssen BM (2006) Endocrine disrupting chemicals and climate change: a worst case combination for Arctic marine mammals and seabirds? Environ Health Perspect 114: 76-80.

Johansson M, Larsson C, Bergman Å, Lund O-B (1998) Structure-activity relationship for inhibition of CYP11B1-dependent glucorticoid synthesis in Y1 cells by aryl methyl sulfones. Pharmacol Toxicol 83: 225-230.

Jones KC & de Voogt P (1999) Persistent organic pollutants (POPs): state of the science. Environ Pollut 100: 209-221. Kelly BC & Gobas FAPC (2003) An arctic terrestrial food-chain bioaccumulation model for persistent organic pollutants. Environ Sci Technol 37: 2966-2974.

Kelly BC & Gobas FAPC (2001) Bioaccumulation of persistent organic pollutants in lichen-caribou- wolf food chains of Canada's central and western arctic. Environ Sci Technol 35: 325-334.

Kerkvliet NI (1995) Immunological effects of chlorinated dibenzo-p-dioxins. Environ Health Perspect 103: 47-53. Kiviranta H, Purkunen R, Vartiainen T (1999) Levels and trends of PCDD/Fs and PCBs in human milk in Finland. Chemosphere 38: 311-323.

Kiviranta H (2005) Exposure and human PCDD/F and PCB body burden in Finland (PhD thesis) National Public Health Institute, Department of Environmental Health.

Kiviranta H, Hallikainen A, Ruokojärvi P, Rantakokko P, Vartiainen T (2006) PCDD/F, PCB and PBDE levels in finnish foodstuffs in 2003-2005. Organohalogen Compd 68: 1898-1901.

Kostyniak PJ, Hansen, LG, Widholm JJ, Fitzpatrick RD, Olson JR, Helferich JL, Kim KH, Sable HJK, Seegal RF, Pessah IN, Schantz SL (2005) Formulation and characterization of an experimental PCB mixture designed to mimic human exposure from contaminated fish. Toxicol Sci 88: 400-411.

Krowke R, Abraham K, Wiesmüller T, Hagenmaier H, Neubert D (1990) Transfer of various PCDDs and PCDFs via placenta and mother's milk to marmoset offspring. Chemosphere 20: 1065-1070.

Kuriyama SN, Talsness CE, Grote K, Chahoud I (2005) Developmental exposure to low-dose PBDE- 99: effects on male fertility and neurobehavior in rat offspring. Environ Health Perspect 113: 149-154.

Lakshman MR, Campbell BS, Chirtel SJ, Ekarohita N, Ezekiel M (1986) Studies on the mechanism of absorption and distribution of 2,3,7,8- tetrachlorodibenzo- p-dioxin in the rat. J Pharmacol Exp Ther 239: 673-677.

Laurent C, Marchand P, Feidt C, Le Bizec B, Rychen G (2005) Tissue distribution and bioconcentration factors of PCDD/Fs in the liver and adipose tissue following chronic ingestion of contaminated milk in rats. Chemosphere 60: 929-938.

Law R J, Allchin CR, de Boer J, Covaci A, Herzke D, Lepom P, Morris S, Tronczynski J, de Eit CA (2006) Levels and trends of brominated flame retardants in the European environment. Chemosphere 64: 187-208.

Law RJ, Alaee M, Allchin CR, Boon JP, Lebeuf M, Lepom P, Stern GA (2003) Levels and trends of polybrominated diphenylethers and other brominated flame retardants in wildlife. Environ Int 29: 757-770.

Letcher RJ, Bustnes JO, Dietz R, Jenssen BM, Jørgensen EH, Sonne C, Verreault J, Vijayan MM, Gabrielsen GW (2010) Exposure and effects assessment of persistent organohalogen contaminants in arctic wildlife and fish. Sci Total Environ 408: 2995-3043.

Letcher RJ (1996) The ecological and analytical chemistry of chlorinated hydrocarbon contaminants and methyl sulfonyl-containing metabolites in the polar bear (Ursus maritimus) food chain. (PhD Thesis, Carleton University, Canada).

Li Y-F, Harner T, Liu L, Zhang Z, Ren N-Q, Jia H, Ma J, Sverko E (2010) Polychlorinated biphenyls in global air and surface soil: distributions, air-soil exchange, and fractionation effect. Environ Sci Technol 44: 2784-2790.

Luhtala A, Rautiainen A, Antila M (1968) Die zusammensetzung der finnischen renntiermilch. Suomen Kemistilehti B41: 6-9.

Lund B-O (1994) In vitro bioactivation and effects on steroid metabolism of DDT, PCBs and their metabolites in the grey seal (Halichoerus grypus). Environ Toxicol Chem 13: 911-917.

Lydersen C, Wolkers H, Severinsen T, Kleivane L, Nordǿy ES, Skaare JU (2002) Blood is a poor substrate for monitoring pollution burdens in phocid seals. Sci Total Environ 292: 193-203.

Macdonald RW, Barrie LA, Bidleman TF, Diamond ML, Gregor DJ, Semkin RG, Stachan WMJ, Li YF, Wania F, Alaee M, Alexeeva LB, Backus SM, Bailey R, Bewers JM, Gobeil C, Halsall CJ, HammerT, Hoff JT, Jantunen LMM, Lockhart WL, Mackay D, Muir DCG, Pudykiewicz J, Reimer KJ, Smith JN, Stern GA, Schroeder WH, Wagemann R, Yunker MB (2000) Contaminants in the Canadian arctic: 5 years of progress in understanding sources occurrence and pathways. Sci Total Environ 254: 93-234.

Mackay D, Shiu WY, Ma KC (1992) *Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals*. Volume II. Polynuclear aromatic hydrocarbons, polychlorinated dioxins and dibenzofurans. Lewis Publishers, Boca Raton, FL. 597 p.

Main KM, Kiviranta H, Virtanen HE, Sundqvist E, Tuomisto JT, Tuomisto J, Vartiainen T, Skakkebaek NE, Toppari J (2007) Flame retardants in placenta and breast milk and cryptorchidism in newborn boys. Environ Health Perspect 115: 1519-1526.

Malcolm HM, Osborn D, Wright J, Wienburg CL, Sparks TH (2003) Polychlorinated biphenyl (PCB) congener concentrations in seabirds found dead in mortality incidents around the British coast. Environ Contam Toxicol 45: 136-147.

Marchand P, Vénisseau A, Brosseau A, Gadé-Hildevert C, Ramdin F, Le Bizec B (2007) Study of a relationship between PCB concentrations in bovine serum, fat and muscle samples. Organohalogen Compd 69: 2380-2383. Marinovich M, Sirtori CR, Galli CL, Paoletti R (1983) The binding of 2,3,7,8- tetrachlorodibenzodioxin to plasma lipoproteins may delay toxicity in experimental hyperlipidemia. Chem Biol Interact 45: 393-399.

Mariussen E, Steinnes E, Breivik K, Nygård T, Schlabach M, Kålås JA (2008) Spatial patterns of polybrominated diphenyl ethers (PBDEs) in mosses, herbivores and carnivore from the Norwegian terrestrial biota. Sci Total Environ 404: 162-170.

Martinez JM, DeVito MJ, Birnbaum LS, Walker NJ (2003) Toxicology of dioxins and dioxin-like compounds. In: Schecter A & Gasiewicz TA (Eds.) Dioxins and health (Second Edition). John Wiley & Sons, Inc. 952 p.

McLachlan MS & Hutzinger O (1990) Concentrations of PCDD/F in air and particulate at a rural site in West Germany. Organohalogen Compd 1: 479-484.

Meerts IATM, Van Zanden JJ, Luijks EAC, Van Leeuwen-Bol I, Marsh G, Jakobsson E, Bergman A, Brouwer A (2000) Potent competitive interactions of some brominated flame retardants and related compounds with human transthyretin in vitro. Toxicol Sci 56: 95-104.

Meerts IATM, Letcher RJ, Hoving S, Marsh G, Bergman A, Lemmen JG, Van Der Burg B, Brouwer A (2001) In vitro estrogenicity of polybrominated diphenyl ethers, hydroxylated PBDEs, and polybrominated bisphenol A compounds. Environ Health Perspect 109: 399-407.

Mikula P & Svobodová Z (2006) Brominated flame retardants in the environment: their sources and effects (review). Acta Vet Brno 75: 587- 599.

Moser GA & McLachlan MS (2001) The influence of dietary concentration on the absorption and excretion of persistent lipophilic organic pollutants in the human intestinal tract. Chemosphere 45: 201-211.

Männistö S, Ovaskainen M-L, Valsta L (Eds.) (2003) The National FINDIET 2002 Study, Department of Nutrition, National Public Health Institute, B3/2003, Helsinki, Finland.

Nair A, Mandapati R, Dureja P, Pillai MK (1996) DDT and HCH load in mothers and their infants in Delhi, India. Bull Environ Contam Toxicol 56: 58-64.

Nakari T, Pesala P (2005) In vitro estrogenicity of polybrominated flame retardants. Aquat Toxicol 74: 272-279.

Nebert DW, Gonzalez FJ (1987) P450 genes: structure, evolution, and regulation. Annu Rev Biochem 56: 945-993. Nebert DW (2000) Drug-metabolizing enzymes, polymorphisms and interindividual response to environmental toxicants. Clin Chem Lab Med 38: 857-861.

Nieminen M & Heiskari U (1989) Diets of freely grazing and captive reindeer during summer and winter. Rangifer 9: 17-34.

Nieminen M, Ojutkangas V, Timisjärvi J, Hissa R (1984) Serum lipids, thyroxine and catecholamine levels in the reindeer with reference to the annual climatic cycle. Comp Biochem Physiol 79A: 87-92.

Nieminen M (1994) Poro, ruumiinrakenne ja elintoiminnat. Pohjolan Sanomat Oy, Kemi. 169 p.

NIP (National Implementation Plan) (2006) Pysyviä orgaanisia yhdisteitä koskevan Tukholman yleissopimuksen velvoitteiden kansallinen täytäntöönpanosuunnitelma. http://www.ymparisto.fi/pop. 26.11.2013.

Nyholm JR, Lundberg C, Andersson PL (2010) Biodegradation kinetics of selected brominated flame retardants in aerobic and anaerobic soil. Environ Pollut 158: 2235-2240.

Olson JR (2003) Pharmacokinetics of dioxins and related chemicals. In: Schecter A & Gasiewicz TA. Dioxins and health (Second Edition). John Wiley & Sons, Inc. 952 p.

Opperhuizen A & Sijm DTHM (1990) Bioaccumulation and biotransformation of polychlorinated dibenzo-pdioxins and dibenzofurans in fish. Environ Toxicol Chem 9: 175-186.

Oser BL (1981) The rat as a model for human toxicological evaluation. J Toxicol Environ Health 8: 521-542. O'Shea T (1999) Environmental contaminants and marine mammals. In: Biology of marine mammals. Reynolds III JE and Rommel SA (Eds.). (Smithsonian Institution Press, USA) p. 485–536.

Patterson DG, Hampton L, Lapeza CR, Belser WT, Green V, Alexander L, Needham LL (1987) High-resolution gas chromatographic/high-resolution mass spectrometric analysis of human serum on a whole-weight and lipid basis for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. Anal Chem 59: 2000-2005.

Pohjanvirta R & Tuomisto J (1994) Short-term toxicity of 2,3,7,8- tetrachlorodibenzo-p-dioxin in laboratory animals: effects, mechanisms, and animal models, Pharmacol Rev 46: 483-549.

Polder A, Skåre JU, Tryland M, Ropstad E, Gabrielsen GW, VikørenT, Arnemo JM, Mørk, T, Killengreen S, Leonards P, Lie E (2009) Screening of halogenated organic compounds (HOCs) in wild living terrestrial mammals in Svalbard, Norway and Northern Sweden. SPFO-report: 1064/2009. p. 53.

RAIPON/AMAP/GEF Project (2001) Persistent toxic substances (PTS), food security and indigenous peoples of the Russian North. Contact: AMAP Secretariat, Oslo, Norway.

Rahman F, Langford KH, Scrimshaw MD, Lester JN (2001) Polybrominated diphenyl ether (PBDE) flame retardants. Sci Total Environ 275: 1-17.

Reen RK, Cadwallader A, Perdew GH (2002) The subdomains of the transactivation domain of the aryl hydrocarbon receptor (AhR) inhibit AhR and estrogen receptor transcriptional activity. Arch Biochem Biophys 408: 93-102.

Renner R (2000) What fate for brominated fire retardants? Environ Sci & Techn 34: 222A-226A.

Rigét F, Bignert A, Braune B, Stow J, Wilson S (2010) Temporal trends of legacy POPs in arctic biota, an update. Sci Tot Environ 408: 2874-2884.

Ringberg T, White RG, Holleman DF, Luick JR (1981) Body growth and carcass composition of lean reindeer (Rangifer tarandus tarandus L.) from birth to sexual maturity. Can J Zool 59: 1040-1044.

Rolland RM (2000). A review of chemically-induced alterations in thyroid and vitamin A status from field studies of wildlife and fish. J Wildl Dis 36: 615-635.

Ross PS, De Swart RL, Reijnders PJ, Van Loveren H, Vos JG, Osterhaus AD (1995) Contaminant-related suppression of delayed-type hypersensitivity and antibody responses in harbor seals fed herring from the Baltic Sea, Environ Health Perspect 103: 162-167.

Ross PS, Vos JG, Birnbaum LS, Osterhaus ADME (2000) PCBs are a health risk for humans and wildlife, Science 289: 1878-1879.

Ruokojärvi P, Laaksonen S, Suutari A, Kiviranta H, Vartiainen T (2007) Levels of PCDD/Fs and PCBs in reindeer- first results of the Finnish study on POPs in reindeer food chain. Organohalogen Compd 69: 2319-22. Ruokojärvi P, Suutari A, Hallikainen A, Laaksonen S, Nieminen M, Kiviranta H (2011) Distribution of PCDD/Fs, PCBs and PBDEs in semidomesticated reindeer (Rangifer tarandus tarandus L.) meat and liver. Organohalogen Compd 73: 1321-4.

Saether BE & Andersen R (1990) Resource limitation in a generalist herbivore, the moose Alces alces: Ecological constraints on behavioural decisions. Can J Zool/68: 993-999.

Safe S (1990) Polychlorinated biphenyls (PCBs), dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs), and related compounds: environmental and mechanistic considerations which support the development of toxic equivalency factors (TEFs). Crit Rev Toxicol 21: 51-88.

Safe S (1994) Polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs): Biochemistry, toxicology, and mechanisms of action. CRC Crit Rev Toxicol 13: 319-393.

Safe S (2001) Molecular biology of the Ah receptor and its role in carcinogenesis. Toxicol Lett 120: 1-7.

Schlabach M & Skotvold T (1996a) Undersøkelse av PCDD/PCDF i omgivelsene rundt pelletsverket ved aktieselskapet sydvaranger. Norwegian Institute for Air Research, Lillestrøm, NILU-rapport OR 11/96, 111p.

Schlabach M & Skotvold T (1996b) Undersøkelse av PCDD/PCDF i næringsmidler i Sørvaranger. Norwegian Institute for Air Research, Lillestrøm, NILU-rapport OR 29/96, 74p.

Schröter-Kermani C, Rappolder M, Neugebauer F, Päpke O (2011) PCDD, PCDF, and DL-PCB in terrestrial ecosystem: Are there correlations of levels or patterns in soil and roe deer liver? Organohalogen Compd 73: 1325-1328. Sellström U (1999) Determination of some polybrominated flame retardants in biota, sediment and sewage sludge. Doctoral dissertation, University of Stockholm.

Siivonen L ja Sulkava S (2002) Pohjolan nisäkkäät, 5. painos. Otavan Kirjapaino Oy, Keuruu, 224 p.

Simms W & Ross PS (2000) Developmental changes in circulatory vitamin A (retinol) and its transport proteins in free-ranging harbor seal (Phoca vitulina) pups. Can J Zool, 78: 1862-1868.

Sinkkonen S, Paasivirta J (2000) Degradation half-life times of PCDDs, PCDFs and PCBs for environmental fate modeling. Chemosphere 40: 943-949.

Soppela P, (2004) Body condition, the fatty acid composition of different tissue fats and carcass fat quality. Survey for the implementation of the research programs results. Arctic Centre Reports. 50 p.

Suutari A, Ruokojärvi P, Kiviranta H, Verta M, Korhonen M, Nieminen M, Laaksonen S (2011) Polychlorinated dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs), polychlorinated biphenyls (PCBs), and polybrominated diphenylethers (PBDEs) in Finnish semi-domesticated reindeer (Rangifer tarandus tarandus L.) Environ Int 37: 335-41.

Söderström G, Sellström U, de Wit CA, Tyskling M (2004) Photolytic debromination of decabromodiphenyl ether (BDE 209). Environ Sci Technol 38: 127-132.

Tanabe S (1988) PCB problems in the future: foresight from current knowledge. Environ Pollut 50: 5-28.

Tanabe S, Iwata H, Tatsukawa R (1994) Global contamination by persistent organochlorines and their ecotoxicological impact on marine mammals. Sci Total Environ 154: 163-177.

Tillitt DE, Ankley GT, Giesy JP, Ludwig JP, Kurita-Matsuba H, Weseloh DV, Ross PS, Bishop CA, Sileo L, Stromborg KL, Larson Kubiak TJ (1992) Polychlorinated biphenyl residues and egg mortality in double-crested cormorants from the Great Lakes. Environ Toxicol Chem 11: 1281-1288.

Timisjärvi J, Nieminen M, Roine K, Koskinen M, Laaksonen H (1982) Growth in the reindeer. Acta Vet Scand 23: 603-618.

Tryphonas H (1994) Immunotoxicity of polychlorinated biphenyls: present status and future considerations. Exp and Clin Immunogen 11: 149- 162.

Tuomisto J, Vartiainen T, Tuomisto JT (2011) Synopsis on dioxins and PCBs. National Institute for Health and Welfare. www.thl.fi/dioxin. 19.08.2014.

Tyler NJC (1987) Body composition and energy balance of pregnant and non- pregnant Svalbard reindeer during winter. Zool Symp Lond 57: 203-229.

UNEP (United Nations Environment Programme) (2002) Arctic regional report. Regionally based assessment of persistent toxic substances.

UNEP (United Nations Environment Programmme) (2001) Final act of the conference of plenipotentiaries on the Stockholm convention on persistent organic pollutants. UNEP, Geneva, Switzerland. 44 p.

USEPA (2000) Exposure and human health reassessment of 2,3,7,8- tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Part I. Estimating exposure to dioxin-like compounds, Vol. 2, sources of dioxin-like compounds in the United States, draft final report, EPA/600/P-00/001Bb, Sept.

Vahteristo L, Lyytikäinen T, Venäläinen ER, Eskola M, Lindfors E, Pohjanvirta R, Maijala R (2003) Cadmium intake of moose hunters in Finland from consumption of moose meat, liver and kidney. Food Addit Contam 20: 453-463.

van den Berg M et al (1994) The toxicokinetics and metabolism of polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs) and their relevance for toxicity. Crit Rev Toxicol 24: 1-74.

van der Berg M, Birnbaum L, Bosveld ATC, Brunström B, Cook P, Feeley M, Giesy JP, Hanberg A, Hasegawa R, Kennedy SW, Kubiak T, Larsen JC, van Leeuwen FXR, Liem AKD, Nolt C, Peterson RE, Poellinger L, Safe S, Schrenk D, Tillitt D,Tysklind M, Younes M, Wærn F, Zacharewski T (1998) Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. Environ Health Pespect 106:12.

van den Berg M, Birnbaum LS, Denison M, De Vito M, Farland W, Feeley M, Fiedler H, Hakansson H, Hanberg A, Haws L, Rose M, Safe S, Schrenk D, Tohyama C, Trischer A, Tuomisto J, Tysklind M, Walker N, Peterson RE (2006) Toxicol Sci 93: 223.

van den Heuvel JP & Lucier G (1993) Environmental toxicology of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans. Environ Health Perspect 100: 189-200.

van der Veen I & de Boer J (2012) Phosphorus flame retardants: Properties, production, environmental occurrence, toxicity and analysis. Chemosphere 88: 1119-1153.

van Esch GJ (1994) Environmental Health Criteria 162: Brominated diphenyl ethers. WHO, Geneva.

Van Oostdam J, Gilman A, Dewailly E, Usher P, Wheatly B, Kuhnlein HV, Neve S, Walker J, Tracy B, Feely M, Jerome V, Kwavnick B (1999) Human health implications of environmental contaminants in arctic Canada: A review. Sci Total Environ 230: 1-82.

Van Oostdam J, Donaldson S G, Feeley M, Arnold D, Ayotte P, Bondy G, Chan L, Dewaily E, Furgal CM, Kuhnlein H, Loring E, Muckle G, Myles E, Receveur O, Tracy B, Gill U, Kalhok S (2005) Human health implications of environmental contaminants in arctic Canada: a review. Sci Total Environ 351-352: 165-246.

Vartiainen T, Kiviranta H, Hallikainen A, Strandman T (2001) Elintarvikkeiden orgaaniset ympäristömyrkyt ja niiden siirtyminen ihmiseen. Duodecim 117: 91-7.

Viberg H, Fredriksson A, Eriksson P (2002) Neonatal exposure to the brominated flame Retardant, 2,2',4,4',5-pentabromodiphenyl ether causes altered susceptibility in the cholinergic transmitter system in the adult mouse. Toxicol Sci 67: 104-107. VNp 842/(1997) Valtioneuvoston asetus jätteen polttamisesta. Ympäristöministeriö 20.02.2013. Vähäkangas K (2011) Introduction to toxicology of contaminants. Contaminants and human health 2011. Presentation. Summer institute of circumpolar health research 13-17 June 2011. Pohto, Oulu, Finland.

Waliszewski SM, Aguirre AA, Infanzon RM, Siliceo J (2000) Carry-over of persistent organochlorine pesticides through placenta to fetus. Salud Publica Mex 42: 384-390.

Wania F & Mackay D (1995) A global distribution model for persistent organic chemicals. Sci of the Tot Environ 160/161: 211-232.

Wania F & Mackay D (1996) Tracking the distribution of persistent organic pollutants. Environ Sci Technol 30: 390A-396A.

Webster TF & Commoner B (2003) Overview: The dioxin depate. In: Schecter A & Gasiewicz TA. Dioxins and health (Second Edition). John Wiley & Sons, Inc. 952 p.

White RG & Luick JR (1984) Plasticity and constraints in the lactational strategy of reindeer and caribou. Symp Zool Lond 51: 215-232.

Whitlock JP (1990) Genetic and molecular aspects of 2,3,7,8-tetrachlorodibenzo- p-dioxin action. Annu Rev Pharmacol Toxicol 30: 251-277.

WHO (World Health Organization) (1996) Environmental Health Criteria 181: Chlorinated paraffins. World Health Organization, Geneva, Switzerland.

Wong S, Fournier M,Coderre D, Banska W, Krzystyniak K (1992) Environmental immunotoxicology. In: Peakall DB (Eds.). Animal biomarkers as pollution indicators, 8-189. Chapman and Hall, London, UK.

Zhou T, Taylor MM, Devito MJ, Crofton KM (2002) Developmental exposure to brominated diphenyl ethers results in thyroid hormone disruption. Toxicol Sci 66: 105-116.

Zhou T, Ross DG, Devito MJ, Crofton KM (2001) Effects of short-term in vivo exposure to polybrominated diphenyl ethers on thyroid hormones and hepatic enzyme activities in weanling rats. Toxicol Sci 61: 76-82.

Öberg M, Sjödin A, Casabona H, Nordgren I, Klasson-Wehler E, Håkansson H (2002) Tissue distribution and half-lives of individual polychlorinated biphenyls and serum levels of 4-hydroxy-2,3,3`,4`,5-pentachlorobiphenyl in the rat. Toxicol Sci 70: 171-182.

Original papers

I Suutari, A., Ruokojärvi, P., Kiviranta, H., Hallikainen, A., Laaksonen, S. (2009). Polychlorinated dibenzo-*p*-dioxins, dibenzofurans, and polychlorinated biphenyls in semi-domesticated reindeer (*Rangifer tarandus tarandus*) and wild moose (*Alces alces*) meat in Finland. Chemosphere 75, 617-622.

II Suutari, A., Ruokojärvi, P., Kiviranta, H., Verta, M., Korhonen, M., Nieminen, M., Laaksonen, S. (2011). Polychlorinated dibenzo-*p*-dioxins (PCDDs), dibenzofurans (PCDFs), polychlorinated biphenyls (PCBs), and polybrominated diphenyl ethers (PBDEs) in Finnish semi-domesticated reindeer (*Rangifer tarandus tarandus* L.). Environment International 37, 335-341.

III Suutari, A., Hallikainen, A., Ruokojärvi, P., Kiviranta, H., Nieminen, M. Laaksonen, S. (2012). Persistent Organic Pollutants in Finnish reindeer (*Rangifer tarandus tarandus* L.) and moose (*Alces alces*). Acta Veterinaria Scandinavica 54 (Suppl 1): S11.

IV Holma-Suutari, A., Ruokojärvi, P., Laaksonen, S., Kiviranta, H., Nieminen, M., Viluksela, M., Hallikainen, A. (2014). Persistent organic pollutant levels in semi-domesticated reindeer (*Rangifer tarandus tarandus* L.), feed, lichen, blood, milk, placenta, foetus and calf. Science of the Total Environment 476-477, 125-135.

V Holma-Suutari, A., Ruokojärvi, P., Kiviranta, H., Laaksonen, S., Nieminen, M., Viluksela, M., Hallikainen, A. (2014). Polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs), polychlorinated biphenyls (PCBs) and polybrominated diphenyl ethers (PBDEs) in tissues of Finnish semi-domesticated reindeer (*Rangifer tarandus tarandus* L.) and wild moose (*Alces alces*). Manuscript.

Original publications are not included in the electronic version of the dissertation.

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