Environmental Contamination and Marine Mammals – with special focus on Grey Seals (Halichoerus grypus)

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Environmental Contamination and Marine Mammals – with special focus on Grey Seals 
(*Hariocherus grypus*)

Masterthesis
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June 1, 2009
Abstract
This thesis concerns the effect of marine pollutants on marine mammals, with special focus given Bergmans studies on the Baltic grey seal. Environmental pollutions have gained increasing attention the last few decades, and the alarming levels of certain pollutant compounds found in the biota caused by the worldwide spread of anthropogenic pollutants like PCBs and DDTs, has led to numerous studies on the subjects. Potential ways in which chemical compounds may manifest themselves include direct mortality, reproductive impairment, and increased susceptibility to disease. It is often assumed that sublethal toxic effects of persistent contaminants will ultimately alter population size. Unfortunately, field studies are hampered by many confounding variables, obscuring the nature of various associations and possible cause- and effect relationships. According to Tanabe (1988) the ocean serves as sink for most pollutants, and due to their lipophilic characteristics, the levels of several pollutants the levels found has stagnated and an exposure to these contaminants can be expected in the coming decades.
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### Abbreviations

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<th>Abbreviation</th>
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<tbody>
<tr>
<td>ATP</td>
<td>Adenosine-5'-triphosphate</td>
</tr>
<tr>
<td>BSDC</td>
<td>Baltic Seal Disease Complex</td>
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<tr>
<td>DDE</td>
<td>dichlorodiphenyldichloroethylene</td>
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<tr>
<td>DDT</td>
<td>dichlorodiphenyltrichloroethane</td>
</tr>
<tr>
<td>DF</td>
<td>dibenzofuran</td>
</tr>
<tr>
<td>EPA</td>
<td>Environmental Protection Agency</td>
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<tr>
<td>FDA</td>
<td>Food and Drug Administration</td>
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<tr>
<td>HCH</td>
<td>Hexachlorocyclohexane</td>
</tr>
<tr>
<td>Hg</td>
<td>Mercury</td>
</tr>
<tr>
<td>Lb</td>
<td>Lead</td>
</tr>
<tr>
<td>MT</td>
<td>Metallothioneins</td>
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<tr>
<td>OC</td>
<td>Organochlorine</td>
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<tr>
<td>PAH</td>
<td>Polyaromatic hydrocarbon</td>
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<tr>
<td>PCB</td>
<td>Polychlorinated biphenyl</td>
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<tr>
<td>PBDE</td>
<td>Polychlorinated dibenzoether</td>
</tr>
<tr>
<td>PCDF</td>
<td>Polychlorinated dibenzofuran</td>
</tr>
<tr>
<td>PDV</td>
<td>Phocine distemper virus</td>
</tr>
<tr>
<td>SWISP</td>
<td>Swedish investigation on seal pathology</td>
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<td>WHO</td>
<td>World Health Organization</td>
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1. Introduction

Marine mammals (Fig 1) include whales (Cetaceans), seals (Pinnipeds), manatees, polar bears and sea otters, representing three different orders: the Carnivora, Cetacea and Sirenia.

Figure 1. A small selection of marine mammals Clockwise from the upper left: Beluga whales, Narwhal, Ringed seal, Walrus, Bowhead whale, Bearded Seal, and center, Polar Bear.
Source: https://www.arctic.noaa.gov/reportcard/marine-mammals.html

Environmental contaminants have gained increasing attention as potential threats on marine mammals. A principal objective in studies of the interaction between pollution and marine mammals is to understand the long term biological effects on behaviour and physiology. In addition to documentation of anomalies and residue levels in animal tissue, it is important to also have a focus on population effects.

The basic questions regarding the ecological influence of pollution are:

1. What are the global spatial and temporal trends of levels of pollutants observed in marine mammal tissues?
2. What is known about effects in marine mammals?

3. What is the lack of knowledge required to fully assess the impact of pollution on marine mammals?

Potential ways in which chemical compounds may manifest themselves include direct mortality, reproductive impairment, and increased susceptibility to disease. It is often assumed that sublethal toxic effects of persistent contaminants will ultimately alter population size. Unfortunately, field studies are hampered by many confounding variables, obscuring the nature of various associations and possible cause and effect relationships. Marine mammal populations with high exposure to for example organochlorines are also likely to have been subject to numerous other forms of human induced stress, such as other contaminants, noise pollution or changes in food quantity and quality. McBee and Bickham (1990) therefore emphasized that it is very difficult to demonstrate that changes in population parameters (natural mortality, fertility, growth) are attributable to sublethal effects of contaminants.

O’Shea and Brownell (1994) reviewed the literature on reproductive effects of dietary exposure to PCBs in carnivores (mustelids), primates, lagomorphs, bats and rodents, and noted considerable differences in sensitivity of reproduction to PCB exposure, even among species in the same genus. Experimental studies have also revealed a high individual variability in sensitivity to the same chemical. It is therefore not recommended to make generalization to the whole group of marine mammals.

Although numerous investigations have been conducted in ecotoxicology, the inherent difficulties mentioned above implies that our knowledge about the effects of many contaminants on marine mammals remain very incomplete. Occasionally the results of investigations have been interpreted to be of significance in ways that are beyond the bounds that can actually be supported by existing data.

The objective of this thesis is to look at the effect of environmental pollution on marine mammals. Based on reviewed articles concerning the different topics in this thesis I will try to answer the questions asked above, as well as give a description of the various effects observed in the Baltic grey seal by Bergman (2007).
2. Biology of the Grey seal (*Halichoerus gypsi*)

Grey seals only occur along the coasts of North east America and Northern Europe. They are subdivided into more or less isolated colonies and can roughly be subdivided into three main stocks: (1) The Northeast Atlantic stock, (2) The North-Western Atlantic stock, and (3) The Baltic Sea (Figure 2).

Adult males reach their maximum length at about 11 years and are then 2.2 m and weigh 220 kg. Adult females are nearly 15 years when they reach their maximum size of 1.8 m and 150 kg. The coat color varies and all shades of dark and light grey, brown and silver may be found. Females produce their first pup when four - five years old. Males are sexually mature at six years but do not normally appear on the breeding grounds till they are eight years old. Males are monogamous if there is ample breeding space, as on Sable Island in Canada, but on most colonies there is a certain degree of crowding, and the males are then polygynous (Mansfield 1966). Most of the breeding bulls are between 12 and 18 years old.

Grey seals usually produce their pups in the coldest months of the year. Round the coast of Britain the pupping season is between the beginning of September and the middle of December. In Norway pupping occur in October – December, and in Canada December – January. The Baltic Grey seals pup in early spring, between middle of February and middle of March.

Grey seals are mainly fish eater and show no preference for any particular species. Although pelagic and midwater fish are most frequently eaten, bottom living fish at 70 meters or more are also taken. Crustaceans and molluscs are also included in the diet.
According to Ziryanov and Mishin (2007) the grey seals annual cycle consist of 5 periods; first the pre-breeding period, which takes place close to breeding site, second is the breeding period, including parturition and copulation, third is the post-breeding period, when seals are dispersed close to moulting sites, forth is the moulting period, where animals keep close to islands and go on shore for moulting (spring concentrations), and the fifth period is that of summer migration.

2.1 The Baltic grey seal
In Northern Europe, bounded by the Scandinavian Penninsula, European mainland and the Danish islands, we find the semi-enclosed, brackish Baltic Sea (fig 3). Influenced by the saline water from the North Sea in the West and with several of North Europe's major rivers flowing in and out of the Baltic, a gradient in salinity can be expected, with decreasing salinity towards the North and East. With 9 countries bordering the Baltic, inhabited by 85 million people, the input of anthropogenic waste and contaminants from both industry and agriculture will influence the quality of the water, often one will experience an increase in eutrophication. These factors will again affect the species composition and diversity in the area. Another factor is the annual oscillation ice cover, with a long-term maximum average about 45% (Thulin and Andrushaitis 2003).
The Grey seal has had a dramatic history throughout its range with severe declines in population size. Despite this, its population has shown an increase in number was expected, but the population continued to decrease. Sweden and Finland have strict hunting regulations to protect the Baltic Sea. The main reason for decline is habitat loss and fragmentation. In the Baltic Sea, the habitat loss is due to human activities and pollution.

Figure 3: Map showing the Baltic Sea with neighboring countries. Source: [Map image]
seal population was estimated to 88 000 – 100 000 animals and intensive hunting led to severe decline in seal populations in the area. After World War II, even though hunting pressure decreased, there were no signs of recovery of Swedish seal. Reproductive failure was believed to be the cause, and PCB was suspected to be associated with the interrupted pregnancies. In the 1970s the number of grey seals was down to around 4000 animals (di Gleria 2006, Bergman 2007).

According to Bergman (2007) the period of birth lactation and mating last for 2-3 weeks and occur during the last 3 weeks of February and the 1st week of April. Most birth take place the first two weeks of March and mating occur during the last week of lactation. Further, the time between fertilisation and blastocyst implantation is about 100 days in grey seals and this period is referred to as a period of delayed or suspended implantation of the blastocyst in the uterus. This period is followed by active gestation (embryonic and foetal growth to parturition) and last for about 240 days. Confirmation of pregnancy can occur after blastocyst implantation, usually during mid June – mid August. Grey seal pups have a weight gain at 1.2-1.5 kg/day during lactation due to the fact that the mother’s milk contains ca 52% fat. They are born with their embryonic fur which they begin to shed after 3 weeks and is completely moulted within 4-5 days.

3. Materials

In this thesis I have collected data on effects of various pollutants, as well as on geographical and temporal variation of organochlorine levels in marine mammals. I have surveyed more than 120 references obtained from cross-referencing in papers and from searches in BIBSYS Ask, ISI Web of Knowledge, Biological Abstract, Pubmed and Zoological Abstract from 1965 to date. For data on metals in marine mammals the articles by Koeman et al. 1973, 1975, Cole and Deeley 1998, Harada et al 1976; Harada 1996; Gilbertson 2004, Denton and Breck 1981; Byrne et al 1985; Honda et al 1986 a,b, 1987; Dietz 1990; Sanpera et al 1993, 1996 have been very helpful, as have Jenssen 1996, Ross et al. 2004, Maugh 1975, Béland et al (1993) and Martineau et al (1994) been in the review of persistent organic pollutants. Mainly referring to Neff 1990a,b in the chapter about persistent oil pollution. Bergman (2009) is basically used in the description of POS, particular PCB on Baltic grey seal.
4. Shift in thinking about toxic effects of chemicals
Over the last 30 to 50 years the environment has been subject to pollution, and the extensive use of halogenated organic chemicals, such as DDT and hexachlorocyclohexane (HCH) used as pesticide in agriculture, and industrial chemicals like polychlorinated biphenyls (PCBs) and polybrominated diphenyl ethers (PBDEs) has led to widespread emissions (Vorkamp et al. 2008).

While modern agriculture has become dependent on pesticides, herbicides and fertilizers, chemical and mining industries have expanded their interest across the globe. The early approach in most modern societies was ‘industrial production at any cost’. Before the 1960s there were few warnings of the dangerous toxicological effects induced by human technologies. Before Rachel Carson, whose book Silent Spring warned that our song birds were wiped out by DDT, few people thought plastics or pesticides and the chemicals that comprise them were dangerous. Perhaps the first disaster to raise public awareness of the danger from industrial chemicals occurred in Japan, where methylmercury was found to be the cause of neurological deformities in nearly 10% of the children near the area of Minamata Bay. The whole story is given by Harada (1972).

In the years after World War II, the Minamata factory produced numerous organic chemicals, including acetaldehyde which is an intermediate in the manufacture of consumer products from plastics to perfumes. By 1951 the plant was producing more than 6000 tons of acetaldehyde a year. In this process mercuric sulfate was used to oxidize acetylene into acetaldehyde. The company simply dumped the by-products with all its mercury into Minamata Bay. Microbes in the water converted the mercury into an organic form, methylmercury. Fish and shellfish then started to consume and concentrate the methylmercury.

As the 1950s progressed, cats in the bay area began dying amidst fits of convulsions, crows were seen falling from the sky and fish floated dead on the bay’s surface. Some villagers began having problems with speaking and walking. Eventually, these people got problems with seeing, hearing and swallowing. By October 1956, 40 patients suffering from these symptoms had been documented and 14 of these died of convulsions. A research team from Kumamoto University concluded that the symptoms were due not to contagious microorganisms, but to a heavy metal. When mercury levels in the area were measured, the
results were shocking. The wash-water channel of the factory contained more than 2 kg mercury in every ton of sludge – so much that it was profitable to mine it!

One of the most important findings was that children born to unaffected mothers could be severely damaged by methylmercury. Simply by eating their normal diet, pregnant women were inadvertently exposing their fetuses to high doses of this compound. Brain and eye deficiencies could be caused both by transmission of mercury across the placenta and after birth by transmission through the mother’s milk. It was once thought that the placenta protected the fetus from such substances, but the fact is that the placenta concentrates methylmercury and presents it to the fetus (Eto 2000). Mercury is selectively absorbed by regions of the developing cerebral cortex (Kondo 2000), and when pregnant mice are given mercury on day 9 of gestation, nearly 50% are born with small brains (O’Hara et al 2002).

Other companies have placed heavy metal effluents into rivers where the local people have little power. Minamata syndrome has been documented in indigenous people in Ontario (Canada), and in Brazil’s Amazon Basin where new chemical plants have been established (Harada et al 1976; Harada 1996; Gilbertson 2004). High levels of mercury have been found in fish from several parts of North America, primarily in areas where chlor-alkali plants are discharging mercury-laden wastewater. For example, fish taken from large river systems in the 1970’s frequently had mercury levels in the range 10 – 30 ppm Hg (Smith & Smith 1975). It is noteworthy that the mercury levels in the fish sampled from these two systems were comparable to the levels of mercury found in fish from Minamata (5 – 40 ppm) in 1959. Nevertheless, no widespread mercury poisoning of the Minamata dimension has occurred in the United States or Canada, presumably because most people in North America eat fish less frequently than the Japanese.

The increasing knowledge of damage caused by mercury poisoning forced a shift in thinking about the food webs, bioaccumulation and protection of the fetus against environmental chemicals. A change in attitude also led to new legislations holding corporations legally responsible for their actions.

5. Contaminants – Input and Dispersal in the Marine Environment

The major groups of anthropogenic contaminants, heavy metals, acidifying gases (SO₂) and persistent organic pollutants (POPs), can all be transported from sources at low- and mid-
latitudes atmospherically to the Arctic, and to less degree Antarctica. Heavy metals and POPs are absorbed at the base of the food chain by plankton and biomagnified to significant levels at higher trophic levels, while the acidifying gases, in the form of sulphur aerosols, cause acid rain and can lead to climate changes (Bard 1999).

5.1 Metals
Metals and halogenated hydrocarbons are conservative pollutants, and unlike organic waste, these pollutants are not subject to bacterial degradation, in other words they are considered permanent additions to the marine environment. Metals are introduced to the aquatic systems as a result of the weathering of soils and rocks, from volcanic eruptions, and from a variety of human activities involving the mining, processing and use of substances that contain metal contaminants, as well as direct discharge or dumping. In certain areas dredging of contaminated harbours and estuaries are also considered important sources. Some metals are considered essential for many living organisms, like for example iron found in the vertebrate/invertebrate respiratory pigment haemoglobin, haemocyanin found in higher crustaceans and molluscs contains copper, and zinc are contained in many enzymes, others include cobalt, vanadium and manganese (Clark 2001). Apart from this, virtually all metals, including the essential metal micronutrients, are toxic to aquatic organisms if exposure levels are sufficiently high. There are definite similarities in the toxicology of many metals. For example, most heavy metals have a great affinity for sulfur, so mercury, lead and cadmium appear to exert toxic effects largely by combining with sulphur-containing amino acids in proteins. This induces interference with enzyme-mediated processes and/or disruption of cellular structure.

Ions of heavy metals (mercury, cadmium, lead), also called metalloids, can be toxic to cells at rather low concentrations and are usually not required for metabolic activity, transition metals (copper, iron) on the other hand are essential at low concentration, but toxic at high levels, and a third group of metals that are of particularly biological concern is the light metals (sodium, potassium and calcium) which are transported in aqueous solutions as mobile cations (Clark 2001). Their toxicity can ensue from (1) interfering with enzyme activity, as when mercury or cadmium bind to a protein (often to a critical cysteine residue on the protein), (2) preventing closure of a calcium ion channel, so toxic levels of calcium can enter the cell, or (3) generating reactive oxidative intermediates. Some metals can be toxic through all the above mechanisms.
The main protection against toxic metals is carried out by enzymes with specialized peptides and proteins that shackle the metal and then escort it out of the cell. These defenses are based on sequestering the metal through binding to sulfhydryl groups (-SH) on specific defense molecules. The sulfhydryl groups bind avidly to the metals, thereby preventing them from doing damage. Sulfhydryl groups do double duty in cell protection, since they are also antioxidants (they reduce oxidant molecules).

One of the major defense molecules against heavy metals is the tripeptide glutathione, composed of glutamate-cysteine-glycine. With the sulfhydryl containing cysteine group, glutathione binds with high affinity to heavy metals such as mercury and cadmium. It is very abundant in cells and also provides critical defense against oxidant damage. The cells get rid of the metal-glutathione conjugate by pumping it out of the cytoplasm using special efflux transporters (Cole and Deeley 1998).

The second defense against heavy metal toxicity is a special class of metal binding proteins called metallothioneins (MT). These proteins are cysteine-rich (at least 30% of their amino acids are cysteines), and as in glutathione, the –SH group binds to the metals (Palmiter 1998). This class of protein also has antioxidant activity. If the organism is exposed to heavy metals, its cells respond by increasing MT gene transcription and translation, which provides further protection.

In 2003 Kakuschke et al. (2006) studied one 7 months old grey seal observed around the island Helgolander Dune, Germany, looking for indications of metal sensitivity in marine mammals. They used three different approaches to allow a more detailed investigation of the grey seal. Looking at a combination of various T-cell reactions to metals, including antigen-specific sensitivity and resulting cytokine pattern, as well as metal concentrations in blood the results showed that in this case the grey seal was hypersensitive to Ni and Be. Due to small sample size and lack of a healthy control, no extensive conclusions were made, on the other hand, these data demonstrate the value of using several different approaches when investigating metal-induced immunological dysfunction and indicates that marine mammals can be considered good candidates for developing metal sensitivities.
5.1.1 Mercury

Mercury has long been recognized for its poisonous effects. It is one of few nonessential elements that shows appreciable biomagnification in marine food webs and has a relatively low threshold for toxicity. Marine mammals have evolved biochemical mechanisms to tolerate seemingly high exposure to mercury in the food chain. Mercury enters the environment as a contaminant resulting from a number of processes, including mining, manufacturing of paper and chlor-alkali plants. It is intentionally used as a fungicide, particularly as a seed dressing.

Much of the mercury found in the environment is released through natural processes, and certain regions have higher mercury levels than others originating from geological sources. Mercury can also be highly volatile. Natural mercury brought to the sea surface from cold upwelling taking place in the equatorial Pacific may volatize to the atmosphere in quantities approximately equal to all global anthropogenic emissions (Kim and Fitzgerald 1986). Atmospheric mercury can return to the earth as fallout in particulate matter.

Both aerobic and anaerobic microorganisms in sediments and soil convert various organic and inorganic forms of mercury to dimethyl (\(\text{CH}_3\text{-Hg-CH}_3\)) or methyl forms. The highly toxic methyl mercury ion (\(\text{CH}_3\text{-Hg}^+\) or MeHg) is soluble in water, taken up by organisms, and can biomagnify several orders of magnitude in the food chain. Particularly, the methyl form accumulates in the kidneys, liver and brain.

5.1.2 Mercury in marine mammals

One of the most outstanding cases of interaction between toxic elements is the apparent protective effect of selenium against mercury toxicity. Mercury-selenium correlations have been documented in numerous marine mammals, and these observations are consistent with a role for selenium in protection against mercury toxicity. Laboratory studies of a number of species show that various toxic effects of mercury were prevented or reduced in severity by simultaneous or prior exposure to selenium (Cuvin, Aralar and Furness 1991). In marine mammals tissue concentrations of mercury that would indicate toxicity in other species are often exceeded with no evidence of harm. These concentrations are typically accompanied by increased selenium in the liver in a 1:1 molar ratio (Koeman et al. 1973, 1975). In the absence of selenium, mercury is bound to metallothionein proteins, which detoxify mercury, but may also cause long-term mercury retention. Selenium apparently diverts binding of mercury away
from metallothionein to higher molecular weight processes. Thus, very little of the mercury in sea lion livers, for example, was bound to metallothionein (Lee et al 1977).

In marine mammals mercury reaches the highest tissue concentration in the liver, and they have a high metabolic capacity to guard against mercury toxicity. Although most of the mercury in fish prey is in the highly toxic methylated form, the proportion of total mercury in marine mammal livers that is actually methylated is usually very low, with the methyl mercury fraction highest in other tissues like muscle, which are less active as sites of metabolic detoxification. In humans poisoned at Mimata Bay, in contrast, mercury in livers ranged from 22 to 70 ppm, but unlike in marine mammals, nearly all was methyl mercury (Britt and Howard 1983). Areas where mercury of geologic origin is naturally high, such as the Mediterranean Sea, produce very high mercury concentrations in marine mammals (André et al 1991 a,b; Leonzio et al 1992).

Baleen whales that tend to feed lower in the food chain, have very low mercury concentrations in liver in comparison with the piscivorous toothed whales and pinnipeds (Denton and Breck 1981; Byrne et al 1985; Honda et al 1986 a,b, 1987; Dietz 1990; Sanpera et al 1993, 1996). Unlike organochlorines in blubber, which are usually highest in adult males, mercury concentrations in livers of marine mammals generally show no differences between sexes. Concentrations of mercury in meat and blubber of some marine mammals could pose human health risks (Botta et al 1983; Andersen et al 1987; Simmonds et al 1994), the potential risk is related to what extent the mercury is methylated (Eaton et al 1980).

5.1.3 Lead

Lead (Pb) is widely distributed in the rocks and soils of the earth's crust. The major natural source of emissions is windblown and volcanic dust. It is mined primarily from deposits of the mineral galena (lead sulfide PbS). Since metallic lead can be separated from PbS by heating at low temperatures, easily achieved by burning wood and charcoal, it was not difficult for earlier civilizations to extract lead. As far back as 1500 BC, lead was used by the Egyptians. The Romans used lead extensively to line their aqueducts and water mains. They also used Pb to line cooking vessels, since bronze pots tend to give food a bitter taste. Gilfillan (1965) suggested that endemic lead poisoning caused by the consumption of contaminated food and water contributed significantly to the fall of the Roman Empire. After
the ninth century the use of lead salts to sweeten wine was common in Europe, and associated outbreaks of lead poisoning were not infrequent.

In modern times the lead-acid battery accounts for about 4/5 of total lead consumption. Other uses of significance from the standpoint of environmental pollution and human health are lead in ammunition, paint pigments and gasoline additives (tetraethyl lead was introduced in 1923 as an antiknock additive). Lead concentrations in Greenland ice sheets indicate that Pb concentrations of precipitation increased by several orders of magnitude between 1000 BC and 1950 (Murozumi et al 1969; Boutron et al 1991). Much of the increase occurred after 1750 and accelerated greatly after 1940. The increase between 1750 and 1940 reflects the effect of the industrial revolution and associated emissions of lead to the atmosphere, largely from lead smelters, whereas the more rapid increase after 1940 reflects the introduction of lead alkyls from vehicle exhaust emissions. The roughly seven fold decline in the lead concentration of precipitation over Greenland from 1970 to 1990 presumably reflects the restrictions imposed on lead in gasoline.

Lead is a major airborne contaminant. Once absorbed into the bloodstream, it is transported to all parts of the body primarily by red blood cells (Waldron and Stöfen 1974). Most of the inorganic lead is deposited in the bones where it replaces calcium. Once incorporated into the bone structure, lead is released back to the bloodstream at a steady, but very slow rate of about 0.1% per day (Waldron and Stöfen 1974). In contrast to inorganic lead, organic lead (lead alkyls) shows no special affinity for the bones, but instead tends to concentrate in lipid tissues, including those of the central nervous system. The highest concentrations of organic lead are found in the brain and liver.

Lead is a nonessential element that is well known for its toxicity in mammals. The pathological effects are varied, but in general reflect the tendency to interact with proteins and thus interfere with the proper functioning of enzymes (Waldron and Stöfen 1974). For example, lead is known to inhibit active transport mechanisms involving ATP and to suppress cellular oxidation-reduction reactions. Lead poisoning is also associated with serious problems related to anemia (disrupting several enzymes involved in the production of heme and causes a shortening of the life of red blood cells by disruption of its cell membrane), central nervous system (degeneration of nerve fibres) and kidney damage (atrophy of renal tubulus). The inhibition of enzymes in the biosynthesis of heme provides a well known
biochemical marker of lead exposure (Peakall 1992). From the standpoint of human health, the greatest danger of lead poisoning is undoubtedly to young children. Neurological damage at early stage may be permanent and can result in impaired physical, as well as mental development.

5.1.4 Lead in marine mammals
Lead has generally not been found in marine mammal tissues at levels that are cause for concern. The liver and kidneys of marine mammals tend to have higher concentrations than muscle and blubber. Early reports of high lead concentrations in livers of grey seals and harbour seals (12-17 ppm wet weight) around Great Britain (Holden 1975) have not been repeated in more recent sampling (Law et al 1991).

Bone is the prominent depot for long-term storage of lead in vertebrates. The highest observed lead concentration in marine mammals was 62 ppm wet weight in bone of a young bottlenose dolphin stranded on the edge of Spencer Gulf, Australia, an area subject for emissions from a lead smelter (Kemper et al 1994).

5.2 Persistent organic pollutants
According to Jenssen (1996) special focus has been given to two types of pollution: persistent organochlorine pollution and petroleum oil pollution. Persistent organic pollutants (POPs) are a structurally diverse group of compounds with several physiochemical properties and structural features in common (Borgå et al. 2004). In most industrialized nations discharges of POPs is restricted by law (Ross et al. 2004). The use of most persistent chlorinated compounds in agriculture and industrial production were stopped in the 1970s due to the increased awareness of the environmental problems (Vorkamp et al. 2008). But despite restrictions and the banning of certain compounds, contaminations of the global environment with pollutant compounds continue to present a risk to human health and other wildlife (Ross et al. 2004).

Birds and other fish-eating species, such as marine mammals, are especially vulnerable to pollutants first of all because of their long life span, secondly, the high trophic level at which these animals are found, often at the top, thirdly most of these animals have poor metabolizing abilities for many POPs, and not to mention that the pollutant compounds bioaccumulate through the food chain (Ross et al. 2004).
5.2.1 Pesticides

DDT (dichlorodiphenyltrichloroethane) was first synthesized in 1877, but did not come into use as a pesticide until 1942. It was used on a broad scale during World War II by the US Army, both to stop typhus fever epidemics that had broken out in Italy, and to eradicate malaria in the Mediterranean basin. DDT (Fig 4) was later used extensively by the World Health Organization (WHO) to control diseases like malaria and typhus fever as mentioned above, but also yellow fever, sleeping sickness and river blindness (Jukes 1974). DDT has been used to control agricultural pests on many crops throughout the world, and in forestry management to control pests such as the spruce budworm and the Dutch Elm disease vector (a beetle). In spite of the tremendous initial success with the application of pesticides, it appeared after some decades that there were two serious problems associated with extensive usage. First, pesticides often kill or seriously affect nontarget species. Secondly, many target species, particular insects, have shown a remarkable genetic adaptability to develop resistance and become immune to the pesticide’s effects. Therefore, use of DDT is banned in most countries today, except in some areas in need of controlling malaria.

![Chemical structure of DDT](http://en.wikipedia.org/wiki/DDT)

Litterally thousands of different pesticides have been produced since 1945. When classified according to target species, they generally fall into one of the following three groups: insecticides, herbicides and fungicides. The chlorinated organics consist of chlorine atoms attached to organic moieties. Many are stable compounds that accumulate in lipid tissue. Because of their persistence, organochlorine pesticides tend to be associated with biomagnification and food chain transfer problems. Some, such as DDT and toxaphene, are highly toxic to fish; some are also highly toxic to birds.
5.2.1.1 Use of DDT to control malaria

Anopheles mosquitoes carry the protozoan parasite (frequently *Plasmodium*) that causes malaria. The parasite finds its way into the mosquito’s salvary gland, from which it is injected into the victim bitten by the mosquito. Once inside a human victim, the parasite reproduces in enormous numbers in the red blood cells, and in the case of the most malignant genus, often attacks the brain.

Around 1900 there were about 250 million cases of malaria each year worldwide, with about 1% of the cases being fatal (WHO 1988). Although most persons who become infected survive, the effects of the disease, ranging from lethargy to total incapacitation, are so widespread in the tropics that malaria has been blamed for impeding the development of entire nations (Marshall 1990). Survivors, often debilitated severely, are subject to reinfection. Most persons who die from malaria are young children. In Sri Lanka, for example, there were about 2.8 million cases of malaria in 1946. DDT spraying began in 1947 and the incidence of malaria rapidly dropped to less than 40 000 clinical cases in 1954. By 1963 there were only 17 reported cases of malaria in Sri Lanka (Edirisinghe 1988).

Euphora occurring in Sri Lanka in the early 1960’s when it was believed that malaria had been eradicated effectively from the country. Perhaps also partially in response to the publication of Rachel Carson’s *Silent Spring* in 1962, the government in Sri Lanka terminated the DDT spraying program in 1964. The result was a rapid increase in the incidence of malaria until an epidemic of 2.5 million cases occurred during 1968 and 1969 (Jukes 1974). A new spraying program reduced the incidence of malaria to less than 200 000 cases in 1972. However, in 1974 investigations revealed that resistance within the mosquito population had increased to an alarming level, and there were over 300 000 clinical cases of malaria that year. In response to this development and the impact on nontarget species, and the U.S. ban on DDT in 1972, the government began substituting malathion for DDT which was phased out entirely. Since 1978 the incidence of clinical malaria cases in Sri Lanka has been on the order of 100 000 per year, which is roughly a 10-fold improvement over the million or more cases reported in 1946 and 1968 when there was no systematic spraying program. However, it is sufficiently high to make malaria a significant public health problem in Sri Lanka.
5.2.1.2 Pesticide effects on nontarget species

The adverse effects of pesticides on nontarget species have been a major cause of both the public’s and governments’ disenchantment with the wide-scale use of pesticides: human pesticide poisonings, poisoning of agricultural livestock, the reduction of natural pest enemies, reduced pollination due to the poisoning of bees, loss of crops and trees, and fishery and wildlife losses. It was estimated that during the 1980’s the cost of these adverse effects was roughly 650 million dollars per year in the United States (Pimentel et al 1991).

As an example we can look at forest spraying with DDT to control spruce budworms in New Brunswick Canada. Beginning in 1952, large areas of forests in northern New Brunswick were sprayed each spring with DDT in an effort to control an outbreak of spruce budworms. The DDT was applied at about 50 kg/km², a typical dosage for control of many forest pests. Despite these efforts, the outbreak continued to spread, and by 1957 over 200 000 km² were spread, much for the second or third time. The Fishery Research Board of Canada had been studying coho salmon populations in the major streams of the effected watershed for several years prior to the initiation of spraying. Therefore, a reliable estimate of salmon populations before 1952 could be made. Continued monitoring of salmon populations after 1952 led to the following conclusion (Rudd 1964): (1) the 0-group (yearling) was reduced by 90%, (2) juveniles over 1 year (parr) was reduced by 70%, and (3) all aquatic insects disappeared.

Although aquatic insects began to reappear in affected streams about 3 weeks after spraying, the composition of the insect population was changed radically. Very small insects dominated and they were too small to be satisfactory utilized as food by the young salmon. Therefore the fingerlings were forced to turn to alternate sources of food and fed largely on snails (Ide 1956). The community of insects did not return to normal for 5-6 years after spraying.

5.2.1.3 Pesticide effects on birds

Field studies (Hickey and Anderson 1968; Blus et al 1971, 1972 a,b; Anderson et al 1975; Spitzer et al 1978) have revealed a negative correlation between DDE residues in eggs and eggshell thickness. It is possible that dieldrin is more effective than DDE in interfering with bird reproduction. Peakall (1970a) demonstrated convincingly that DDT can depress estrogen levels in the bird’s blood and hence cause a delay in reproduction and a reduction in the amount of calcium stored in the bone marrow. Dieldrin was found to be more potent than DDT in this respect.
Laboratory experiments (Heath et al 1969) involving controlled feeding of DDE to birds has demonstrated that DDE has a significant effect on reproduction and eggshell thinning. These effects were evident at DDE concentrations of 3 ppm wet weight, a level that was easily encountered in natural food webs. The recovery of raptorial birds and pelicans following restrictions on the use of dieldrin and DDT strongly supports the implications of the laboratory studies.

5.2.1.4 Persistence and food chain magnification

Most pesticides are stored primarily in fatty tissues where they are metabolized or excreted at a rather slow rate. These pesticides may therefore become greatly concentrated at higher trophic levels in a food chain. The high concentrations of pesticides found in the bodies of carnivorous fish and birds support this argument.

Some ideas about the persistence of DDT and its metabolites in the marine environment may be gained from an examination of the historical record of total DDT residues in anchovies taken from southern California coastal waters. Anderson et al (1975) measured the following concentrations of total DDT residues (DDT+DDE+DDD) in anchovies (ppm fresh weight) for the six years 1969–1974: 4.3, 1.4, 1.3, 1.1, 0.3, 0.15. The drop from 4.3 (1969) to 1.4 (1970) was due to the elimination of DDT discharges from the Montrose Chemical Company outfall in 1970. The second sharp decline from 1.1 (1972) to 0.3 (1973) is attributed to the ban of DDT use in the United States, which went into effect in January 1973.

Since in both cases effects were evident in the anchovies within 1 year from the associated discharge reductions, it seems clear that the effective half-life of DDT and its metabolites in this system must be of the order of some months.

5.2.2 Polychlorinated Biphenyls

Polychlorinated biphenyls (PCB) are complex mixtures of chlorine substituted biphenyls. When benzene is heated to 800 °C, in the presence of lead as a catalyst, biphenyl is formed. This is a molecule in which two benzene rings are linked by a single bond between two carbons that have lost their hydrogen bond. If biphenyls and Cl₂ are brought together at 200 °C, in the presence of a ferric chloride catalyst, some of the hydrogen atoms become replaced
by chlorine. The longer the reaction is allowed to proceed, the greater the extent of chlorination of the biphenyl molecule. Theoretically there are 209 different isomers (congeners) of polychlorinated biphenyls (fig5), and about 130 have been generated in commercial products (common trade names are Askarel and Aroclor). Commercial PCBs are produced to physical, not chemical, specifications and will therefore contain a mixture of 130 isomers. Although PCBs are not pesticides, they are similar in many ways to chlorinated hydrocarbon pesticides.

![Chemical structure of PCB](http://en.wikipedia.org/wiki/Polychlorinated_biphenyl)

**Figure 5.** Chemical structure of PCB source: [http://en.wikipedia.org/wiki/Polychlorinated_biphenyl](http://en.wikipedia.org/wiki/Polychlorinated_biphenyl)

PCBs are chemically inert liquids and excellent electrical insulators. These stable compounds are non-flammable and highly resistant to strong reagents and heat. Destruction by burning requires a temperature of over 1300 °C. PCB has therefore been used in large quantities as insulator and coolant fluids in power transformers and capacitors, and hydraulic fluids. In addition there are a variety of other industrial applications including heat transfer fluids in machinery, plasticizers (as agent used to keep other materials such as PVC products more flexible), and small ingredients in painting, inks, lubricants and coatings. PCBs were manufactured on large scale between 1930 and 1980.

Previously, PCB-containing materials, such as power transformers, were just dumped into landfills, and their PCB content was allowed to leak into the ground. Significant quantities have also been released into the environment by leakage from machineries, like hydraulic systems, fall-off of ship painting, garbage burning at low temperatures and degradation of materials containing PCB, like plastic. It was not until 1966, i.e. 36 years with large scale production, when it was discovered that living organisms contained PCB. During an environmental investigation of DDT, a Swedish group found some ‘unknown compounds’
which was identified as PCB isomers. This discovery marked the start of a world wide research on the dispersal and biomagnification of PCB.

The initial extensive usage and inattentive disposal practices, has made PCBs to a widespread and persistent environmental contaminant. It is found in living organisms from the Arctic to the Antarctic at most depth. PCBs are practically insoluble in water, but are soluble in hydrophobic media, such as fatty or oily substances. Because of their stability and solubility in fatty tissue, PCB undergoes biomagnification in food chains. For example, the PCB concentration in eggs of herring gulls in the Great Lakes (USA-Canada) is 50 000 times that in the phytoplankton.

PCBs have been found to display a degree of toxicity comparable to that displayed by pesticides. They interfere with the reproduction of a variety of species, including fish, birds and mammals (Maugh 1975). The reproduction of mink, for example, is completely halted if the PCB concentration in their food exceeds 5 ppm (Maugh 1972). In high doses, PCB causes cancer in test animals.

It is generally agreed that PCBs are not very acutely toxic to humans when ingested in small quantities. Workers exposed to PCBs in production of capacitors and testing dielectric fluids were reported to suffer from allergic dermatitis, eye irritation, asthmatic bronchitis, gastrointestinal disturbance and headaches (Maugh 1975; EPA 1980). Of these symptoms, the most common reaction to exposure is chloracne; a persistent disfiguring and painful analog to common acne. This is a biological response by humans to exposure to many types of organochlorine compounds. However, one of the problems in evaluating the health effects of PCBs is that commercial PCB mixtures often contain small amounts of polychlorinated dibenzofurans which is several orders of magnitude more toxic than PCBs (EPA 1980). On a gram for gram basis, polychlorinated dibenzofurans are more than 500 times as toxic. With respect to human health, PCBs rank third in toxicity, behind dioxins and furans, when the most toxic isomer of each group is considered (Sun 1983). In Yusho, Japan, more than thousand persons became ill in 1968 after consumption of PCBs that accidentally had been mixed with cooking oil (Sun 1983). The PCBs had been used as a heat exchanger fluid in the deodorization process for the oil. Since the PCBs had been heated, the level of PCDF contamination was much greater than occurs in freshly prepared commercial PCB. The people suffered health effects far worse than did workers at PCB manufacturing and handling plants,
even though the resulting PCB levels in their bodies were about the same. Within two months 29 died and many developed chloracne. The PCB concentration in the rice oil averaged 2500 ppm, and the affected persons were estimated to have ingested about 2 g of PCBs that contained 2 – 5 ppm of polychlorinated dibenzofurans. It has been concluded that PCDF was responsible for ¾ of the effects with PCB themselves responsible for the rest.

Strong heating of PCBs in presence of an oxygen source generally results in the production of small amounts of dibenzofurans. The basic furan ring contains five atoms, one of which is oxygen and the other four of which are carbon atoms that participate in double bonds. The dibenzofurans (DFs) have a benzene ring fused at opposite side of the furan ring. As is the case for dioxins, all chlorinated dibenzofuran congeners are planar; that is, all C, O, H and Cl atoms lie in the same plane. When they are formed from PCB most of the chlorine in the original PCB molecule is still present in the dibenzofuran. Polychlorinated dibenzofurans are denoted PCDF. Like dioxins, polychlorinated dibenzofurans are also produced in small quantities by a number of processes including the bleaching of pulp and the incineration of garbage.

Realizing the potential seriousness of the PCB contamination problem, USA and Japan stopped production of PCBs at large scale in 1978; i.e. 12 years after the Swedish documentation of uptake in the food web. The Environmental Protection Agency (EPA) in USA, under authorization of the Toxic Substances Control Act, banned the manufacture, processing, distribution in commerce and use of PCB in 1979. The EPA ruling, however, allowed the continued use of PCB in existing enclosed electrical equipment under controlled conditions. The life time of such equipment is 20 – 30 years. At the time it was estimated that there were 20 – 30 000 PCB-containing transformers in the United States. By 1985 PCBs were also banned in most European countries.

It is estimated that 1.2 million tons of PCB have been produced since 1930, and that about 370 000 tons have been released into the environment. About 780 000 tons are still in use or placed on land deposits. Only 50 000 tons, i.e. 4% of the total production is broken down or degraded. It is therefore expected that PCBs will represent a significant environmental contaminant for some decades into the 21 century.
From the extensive sampling by Harvey et al (1973, 1974) it appears that PCBs are 50-100 times more persistent than DDT residues in the environment. As is the case for chlorinated hydrocarbon pesticides, PCB concentration in the ocean is quite low, whereas the concentrations are much higher in the lipid tissue of aquatic organisms. However, PCB levels in the muscle of cod, haddock and halibut sampled by Harvey and coworkers ranged from 2 to 190 ppb, well below the present Food and Drug Administration (FDA in USA) guideline for edible fish flesh. Ultimately most PCBs discharged to aquatic systems are presumably either degraded in the water column or buried in the sediment. For example, sinking zooplankton fecal pellets is a major mechanism for transporting PCBs to the sediments of the oceans (Elder and Fowler 1977).

It is noteworthy that in 1985 several Beluga whales died in the St. Lawrence River, and autopsies revealed PCB levels that were utterly fantastic – 575 ppm in the lipid tissue and as high as 1750 ppm in the milk (IJC 1985). Such events underscore dramatically the persistence of PCBs and the tendency of such stable compounds to be concentrated through biological processes.

5.2.2.1 Organochlorines in marine mammals

The St. Lawrence River estuary in Quebec, Canada, is highly contaminated with a wide variety of pollutants, including organochlorines, toxic elements, and polyaromatic hydrocarbons (PAHs). Although it is not possible to separate the individual effects of these contaminants on disease processes, Béland et al (1993) and Martinan et al (1994) reported a high prevalence of tumors, digestive tract and mammary gland lesions, and other abnormalities (including true hermaphroditism) in belugas found dead and examined at necropsy. However, Addison (1989) pointed out that body condition may have influenced the level of measured concentrations, and that habitat destruction may also have influenced the population. Reproductive abnormalities were also observed along with high levels of numerous contaminants in comparison with tissues from other populations. Much interest centred round the role of organochlorines in the disease processes observed in these animals.

The ultimate sinks for many of these persistent compounds are considered to be the world oceans (Tanabe et al 1994), therefore marine mammals have been an end point in the food web accumulation of organochlorines. Aquatic organisms are almost always exposed to a variety of interacting (additive, synergistic or antagonistic) contaminants in natural circumstances (MacFarland & Clarke 1989). According to Borgå et al. (2004) there are
several factors, like lipid content, body size, gender, reproduction, habitat use, migration, biotransformation, seasonal changes in habitat condition, feeding ecology and trophic position that influence OC concentration and bioaccumulation in marine biota. These factors and their importance will vary between organisms and OCs.

Evidence for direct mortality caused by organochlorine compounds was reviewed for baleen whales by O'Shea and Brownell (1994). They noted that the brain is the only tissue in mammals and birds in which concentrations of organochlorines can be considered diagnostic of death. However, organochlorine concentrations reported in brains of baleen whales were far lower than diagnostic levels in other mammals. Concentrations reported in brains of small cetaceans and pinnipeds also do not approach those consistent with lethality in other species.

Surveys of the declining population of harbour seals in the Wadden Sea revealed unusual high concentrations of organochlorines (Reijnders 1980). Later PCB was experimentally verified to be responsible for impaired reproduction in harbour seals (Reijnders 1986).

To further investigate the hypothesis that the adrenal cortex of marine mammals is enlarged as a result of exposure to organochlorines, Kuiken et al. (1993) examined 28 harbor porpoises stranded in various areas of Great Britain. They concluded that adrenal hyperplasia was generally related to chronic causes of death such as disease and starvation, and was thought to be a general indicator of stress rather than organochlorine exposure.

Because of the findings of immunosuppression by organochlorines in studies of laboratory animals, a number of investigators have considered that organochlorine exposure may have played a role in the recent mass die-offs of marine mammals caused by morbilliviruses. This is an area of much active research, conflicting results, and some contention. Hall et al. (1992) tested the relationship between organochlorine concentration in blubber of harbour seals that died in a morbillivirus (phocine distemper virus, PDV) outbreak in Great Britain in 1988 and those that survived (sampled alive by capture and biopsy in 1989). Higher concentrations of organochlorines were observed in the seals that succumbed to the epizootic. However, Hall et al. (1992) pointed out that seasonal differences in blubber thickness and age effects could also have contributed to the lower concentrations of organochlorines in survivors. They therefore concluded that their data were not sufficient to conclude that there was a distinct link between mortality from PDV infection and OC concentration.
Blomkvist et al. (1992) found no significant differences in PCB and DDT concentrations in juvenile harbour seals from Sweden collected during and before a PDV outbreak in 1988. And Jenssen et al. (1994) did not find a correlation between PCBs and thyroid hormone levels in blood of grey seal pups.

Kendall et al. (1992) examined the relationships among concentrations of organochlorines in blubber and plasma thymulin concentrations of harbour seals and grey seals during and after an epizootic of PDV at coastal Scotland and Northern Ireland. Studies in other laboratories indicated that thymulin concentrations are low in mammals with immunodeficiency. Thymulin is produced in the thymus and influences development of T-cells, and can be stimulated by adrenocorticotropic hormone. The thymus is sensitive to some contaminants, for example dioxin. Thymulin levels were negatively correlated with titers to morbillivirus in grey seals, but this relationship was not affected by organochlorine concentrations. Similarly, no relationships were found between thymulin levels and organochlorine concentrations in blubber of harbour seals (Kendall et al. 1992).

Ross et al. (1995) fed two groups of 11 young harbour seals relatively uncontaminated Atlantic herring and herring from the Baltic Sea for two years. The latter group had higher concentrations of PCBs, dioxins and furans. The seals fed Baltic herring had lower in vivo immunological responses to ovalbumin injection. This suggests that the OC played a role in the European PDV outbreaks through immunosuppression.

5.2.2.2 Effects of organochlorines on the Baltic Grey seal
Severe decline in certain seal populations, especially in the Baltic Sea in the 1970s led to speculations about environmental pollution being the cause. It turned out that the contamination came from agriculture and industry, and organochlorine like PCBs and DDTs were found in high concentration in the biota. The discovery of high contamination levels in the biota led to further investigations and studies to look at effects that could help explain the decline in the grey seal, the ringed seal (Phoca hispida bottica) and the harbour seal (Phoca vitulina) populations.

Bergman and Olsson (1985) examined 19 grey seals and 10 ringed seals from the polluted Baltic Sea and reported the occurrence of uterine stenoses and occlusions, benign uterine
tumors, adrenocortical hyperplasia, hyperkeratosis and nail deformations. The nature of the pathology suggested the existence of a disease complex involving organochlorine interference with the endocrine system, resulting in hyperadrenocorticism. PCB was especially suspected, in part because effects on adrenal function have been demonstrated in laboratory studies of other mammals (Feller and Hobson 1986). In addition, evidence that Aroclor 1254 alters the synthesis of steroids after in vitro exposure of adrenals from grey seals has been reported (Freeman and Sangalang 1977). Changes in symmetry of the skull and frequencies of bone lesions, indicating possible developmental and hyperadrenocortical effects of organochlorines, have also been noted in museum specimens of grey seals collected from the Baltic Sea after 1960, when pollution was substantial, in comparison with specimens collected in previous years (Zakharov and Yablokov 1990; Bergman et al. 1992; Olsson et al. 1994). Similar, but less pronounced, increases in skull lesions in recent years in comparison with historic specimens have also been reported for Baltic Sea harbour seals (Mortensen et al. 1992). Both these coastal seal species have been found to have chromosomal aberrations possibly related to contaminant exposure (Hengell 1996).

Bergman (2007) looked at the tendencies of pathological changes in seals found in Swedish waters and the relation to environmental pollution over a 25 year (1977-2002) period. As part of the SWISP project he conducted post mortem examinations of whole body or organ samples. Necropsy of a total of 729 Baltic Grey seals, 376 whole bodies and 353 organ samples. Animals in this study were either found dead on shore, in fishing gear or shot due to severe illness. One of the important factors behind the declines in population size was reproduction failure in females (Bergman 2007). Interrupted pregnancies are caused by occlusions and stenoses of the uterus (Bergman 1999). Effects on Baltic grey seals are described below.

5.2.2.2.1 Integuments
During the period 1977-1983 investigations performed on 13 adult grey seal females revealed three cases of regional skin changes. Localised in the ventral thoracic and abdominal region extending to medial surface of the fore and hind flippers, these can be seen as more or less advanced hypertrichosis. Thin epidermis, hyperkeratosis, dilation of hair follicles and chloracne-like changes were also observed in one female aged 30 years. Changes were also observed around the supercilia of both eyes. After 1983, no similar changes were observed.
Furthermore, unspecific chronic inflammation of claw folds and dyskeratosis of the horn were revealed by microscopic examination. The effects of PCBs and DDTs mentioned above is also shown for other animals, like European ferrets and American mink, fed PCB during experimental investigation according to Bergman.

5.2.2.2 Regional intestinal ulcers
A common feature of the BSDC was ulcers of the intestinal mucosa. Bergman found these in the ileocaeco-colonic region, mostly in the anterior part of the colon. Hypertrophy of the muscular tunic was almost always found in combination with ulcers in the affected region. The severity of the lesions (mucosal extension and depth in the intestinal wall) was graded three ways: slight, moderate and severe. Lesions classified as severe were deep and large ulcerations (fig 6-9). These also affected the muscular tunic involving the serosa, showing fibrinous or fibrous adhesion to adjacent organs. Ulceration extending through the ileum, caecum and abdomen was also observed in some cases, and open communication to the abdomen is considered fatal.

Figure 6 (left) and 7 (right). Severe colonic ulcer in a grey seal female yearling. A, ileum. B, caecum. C, colon. The ulcer is most commonly found in the anterior colon (fig 5, arrows). Arrowheads indicate hookworms, magnification (fig 6). Source: Bergman 2007, photo by Anna Roos

Bergman found that fatal intestinal ulcers occurred in all age classes, with the highest number of moderate to severe colonic ulcers found in the group of 4-15 year old grey seal, see graph in fig 10. The appearance of hookworms, mostly found in or in the vicinity of large wound, could sometimes be difficult to detect because in some cases there would be only one specimen.
Figure 8 (above) and 9 (below). Severe colonic ulcer in grey seal female, aged 30 years. In fig.7 chronic adherence (arrows) of mesentery to caecum are shown and the black area shows sign of older haemorrhages beneath the serosa of anterior colon. In fig. 8 a large and deep ulcer with loss of of mucous membrane of ileum, caecum and small part of anterior colon is shown. P indicates the naked Peyer’s patch of ileum near the ileo-colonic orifice (IOC). Source: Bergman 2007, photo by Anna Roos.

Colonic ulcers in Baltic grey seals

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Legend:
- Orange: 1-3 years
- Orange: 4-15 years (born 1980 or 21980)
- Blue: 16-25 years (only females in the first ten-year-period)
- Black: >25 years (only females)
Figure 10. Graph showing number and prevalence (%) of grey seals collected during 1977-1996, with no or slightly advanced (left) or moderate to severe (right) colonic ulcers.

5.2.2.2.3 Vascular lesions
Changes found in the main arteries of grey seal were restricted to the aorta and its bifurcations. Here the changes were most evident in the distal part of the aorta. Slight changes were presence of focal minor whitish spots and streaks with intimal thickening. Moderate changes were larger confluent areas of intimal thickening containing sub-intimal lipid deposits, while severe changes found were lesions of the above character occupying most of the vessels. These were extending around the whole lumen and had lost its elasticity.

5.2.2.2.4 Adrenocortical hyperplasia
Hyperplasia was exclusively found in the cortex, and occur later than in colonic ulcers, but both prevalence and severity of this lesions were found to agree well with those of colonic ulcers. No significant differences between the prevalence of this changes was seen between early and late birth periods. Signs of adenomatous proliferations in the hypophysis have not been observe by Bergman at macro- and micro scopic examinations of Baltic seals. It seems improbable that the adrenocortical hyperplasia, which was observed in the seals was du to hyperstimulation from the hypophysis. Signs of effects on the hypophysis exerted by a possible adrenal hyperfunction are also of great interest.

5.2.2.2.5 Lesions of female reproductive organs
Stenosis (narrowing) and occlusion (fig 11) of the uterine lumen are most frequently located in the middle part of the horn, and at the site of obstruction the uterine wall is usually thickened. In the grey seal the character of stenosis, obstructions are usually less developed, than in ringed seals. In both cases such obstructions cause accumulation of fluid. Large prevalence of uterine leiomyomas was also observed by Bergman, all tumors showing the same gross appearance. The consistency was firm, white-brown to white-yellow on cut surface, with whirled pattern and preferably located in the wall of the uterine corpus. Large in size and often multiple tumors were seen, and in some cases the central area showed necrosis (dead tissue).

Usually uterine leiomyomas do not occur before the age of 30 in Baltic grey seals, but two cases aged 15 and 22 years respectively, were observed.
5.2.2.6 Skull-bone lesions and bone mineral density
Lesions most often found around the canine teeth and the incisive part of the masticatory bones, but also around the premolars and molar both in the upper and lower jaws. Lesions were aged related with highest prevalence in the early period.

The prevalence of trabecular bone mineral density was highest in the latter period, while the cortical bone mineral density show highest prevalence for specimens collected in the period 1850-1955 and a continuous decline after that.

The inflammatory processes seen in seal are the same as seen in other animals an man. The most probable cause explaining these effects are osteoporosis – loss of bony support for the teeth and further aggravation of the lesions. While the prevalence was high in the beginning of the study, and a decreasing trend was observed in recent years by Bergman. Any certain conclusions could not be made, but much indicates that changes in composition and levels of contaminants the last 3-4 decades can be the cause.

5.2.2.3 Global spatial trends
Concentration levels of various contaminants have had a tendency to decrease during the last decades in regions where it initially was high, and increase in areas far from the source. Redistribution and atmospheric transportation are the best explanations for the shift seen in contamination levels worldwide. Due to lack of long-term surveys, limited or non-existant surveys in Africa and the southern hemisphere, information on OCs is neither in time or space homogeneously distributed (Aguilar et al. 2002). Observed concentration levels in different species vary due to several factors, e.g. species-specific accumulation rates, differences in
analytical techniques and individual-specific accumulation rates related to age and sex. Therefore geographical comparison of pollution burdens should only be carried out in the same tissues, from the same species, and from animals of the same sex and age (intra-specific variations).

Uptake of trace elements is predominantly dietary. The general pattern is that trace element concentrations in marine mammals appear to be related to their feeding habits and area of exposure (Wagemann & Muir 1984; O’Shea & Brownell 1994; Hansen et al 1990; Skaare et al 1990; Skaare et al 1994). Cadmium, copper and zinc levels are higher in species that feed more on squid, compared to species that feed primarily on fish. This is due to the relative higher levels of these elements in squid, which is a general phenomenon in the marine environment (Förstner & Wittmann 1979).

Among the group of trace elements, there is only a large data set for mercury. Considering the geographical distribution there is no clear latitudinal gradient in mercury levels in marine mammals. This may be explained by the fact that mercury enters the environment via anthropogenic as well as geological sources. This leads to a rather patchy distribution where some regions have levels of an order of magnitude higher than in others. Regions where high mercury levels are observed include the Arctic, the Mediterranean, the Oslofjord and the Wadden Sea (Muir et al 1992; Wagemann & Muir 1984; Leonzio et al 1992; Skaare et al 1990; Skaare et al 1994; Reijnders 1980).

A long term study by Pereira et al. (2009) on mercury and PCB congener concentrations in gannet (Morus bassanus) eggs in Britain (from Ailsa Craig and Bass Rock) showed that the half-lives of congeners have increased, this is not unique for gannets, several other studies also confirm that PCB rates have decreased the last decade. Differences in initial environmental inputs and remobilization processes can be reflected by variances in the temporal trends of half-lives for congeners. Gannet eggs were expected to reflect local environmental contamination, and the results indicated that females assimilate contaminants from their prey and mobilize it into their eggs, differences between the two areas can be seen. Absolute concentrations and temporal trends may reflect differences between areas in contaminated inputs from coastal sources. Differences observed between colonies can also be explained by variation in prey selection.
5.2.2.4 Temporal trends

Temporal trends for different OCs in marine biota have been established for several oceans and seas (Tanabe et al. 1994, Loganathan 1991). From a global point of view, Tanabe (1988) concluded that PCB levels in marine biota are unlikely to decline in the near future, due to the fact that only 30% of all PCBs produced have so far dispersed into the environment. Of 20 million tons of PCB produced, more than 30% is still in use and therefore under control. It is essential that a stringent policy is developed to collect and adequately destroy those amounts. From the rest, only 1% has reached the oceans and 30% has accumulated in dump sites and sediments of lakes, coastal zones and estuaries. Their future dispersal into oceans further strengthens Tanabe’s conclusion about the unlikeliness of future decline.

It should be noted that in areas close to the source of pollutants, levels have declined due to restricted use and regulated disposal. However, studies in the Arctic (Norström 1988), the North Sea (De Boer 1988), the Baltic Sea (Bingert et al. 1993) and the Pacific (Tanabe et al. 1994), clearly indicate that the decline leveled off between 1980 and 1985. Particularly Arctic waters and adjacent seas become the major sink for OC.

5.3 Petroleum Oil Pollution

Complex mixtures of hydrocarbons, named crude oil, have molecules consisting of 4-26 carbon atoms or more. The arrangement of atoms can either be straight, branched or cyclic chains, including aromatic compounds. 25% of the oil may be represented by non-hydrocarbons, and vanadium and sulphur compounds are also included. Differences in composition of crude oil will vary from one oilfield to another. The same classes of compounds are found in most crude oils, but there are variations in relative amounts of each constituent (Neff 1990a). Before the crude oil can be used, a distillation process taking different fractions or cuts at different boiling ranges, called refining, is essential. Input of petroleum hydrocarbons to the sea comes from a variety of source, transportation by tankers, tanker accidents, dry docking or from fixed installations (costal refineries, offshore production, marine terminals). Other sources, like urban run-off, industrial and municipal waste, and atmospheric fall-out, as well the occurrence of natural inputs from oil seeps several places in the world also contribute to the total input in marine environments. Oil spills attract greatest public attention due to humans first hand encounters or through media (Clark 2001).
5.3.1 Effects of oil pollution
The effect of oil is ultimately determined by its chemical properties. Understanding the properties of petroleum, its composition, how it enters the marine environment and what happens to it there after are very important when evaluating the consequences of oil exposure in marine mammals (Neff 1990a).
According to Scheffer (1990) there are three main reasons why the effects on marine mammals are not particularly well known. First, even though they share certain adaptations to life in the sea, they comprise five distinct zoological groups with different life cycles, patterns of habitat use and they possess different responses to oil. Second, due to habitats in remote regions, studies of certain species can be very costly in terms of research vessels (ships, aircraft), diving gear or other underwater electronic devices. Handling and keeping animals in captivity can cause problems due to large body sizes. Respiratory system modified for diving are another challenge researchers face in terms of anaesthetizing or immobilizing the marine animals studied. Third, protection by law shield marine mammals from direct approach by researchers. Research per se is not banned, but often impeded by legal requirements to obtain scientific permits. Public resistance is also something researchers will encounter when experimenting on animals using invasive methods.

5.3.2 Toxicity of petroleum compounds
The composition governs crude or refined petroleum oil’s behaviour and fate when spilled at sea. A distinction can be made between acute (immediate) oil pollution, accidental spills leading to release of large volumes of oil on one hand, and chronic (residual) pollution caused by continuous low-level discharge on the other (Clark 2001). Acute toxicity tends to increase with molecular weight.

The impact of an oil spill will vary significantly with the time of the year, with potentially higher risk in the summer time compared to the winter-time. While how close a population is to the environmental carrying capacity at the time of a catastrophic event can influence the population’s rate of recovery (Neff 1990b).
Due to variation in the quality of earlier reports, often being vague, brief or less objective, predicting the consequence of oil exposure in pinniped species can be challenging. According to the latest studies, stress caused by exposure to oil is believed to have an additive effect along with possible association of organochlorine residues and premature births with infectious organisms (St. Aubin 1990).
5.3.3 Effects and recovery in marine mammals

Recovery time depends on percentage of females killed, status of the population in relation to its carrying capacity at the time, and the vicissitudes of the environment (Neff 1990b).

Density-dependent mechanisms, other than effect of crowding in colonies in highly polygynous species, like regulation of pinniped populations are poorly understood. Solid substrate with ready access to the sea, were pinnipeds are born, are boundaries were oil is likely to be spilled and accumulated (McLaren 1990).

Disruptive effects on individuals or pinniped populations caused by oil spill interfere with normal behavior patterns, and the effect on maternal behavior is of particular concern. The mucus membrane surrounding the eyes, lining the oral cavity, respiratory surfaces and anal and urogenital orifices are considered the most sensitive tissues exposed. Seals covered in oil loose their insulating effect, and ingested oil, depending on amount and composition, can cause subtle, as well as progressive organ damage, even acute death. Ingestion of hydrocarbons can cause irritation or destruction- irritated or destroyed epithelial cells lining the stomach and intestine which again, will affect mortality, digestion and absorption (St. Aubin 1990).

According to Wursig (1990) population estimates can be crude, this will reflect difficulties in censusing dispersed pelagic animals. Advanced technologies can improve our knowledge of cetacean abundance. In addition to population size, direct exploitation to discrete subpopulation, habitat deterioration, fishery-related mortality or increased competition for fisheries’ resource will improve our knowledge of cetacean abundance.

He also states that “Human induced mortality is shifting from the purposeful whaling of recent decades to incidental kills related to fishing activities. The role of coastal pollution has yet to be defined.”

Some Cetaceans have monotypic diets, and in cases of oil spill the occurrence and magnitude of nutritional effects in their diet might change. These changes will depend on the intensity and geographical coverage of oil and the amount of detrimental impact on alternative prey species. Feeding at several trophic levels occur among Cetaceans, and since petroleum hydrocarbons do not biomagnify, species feeding on benthic organisms are therefore more likely to ingest contaminated food compared to species feeding higher in the food chain. Most species are capable of making adjustments if they are affected by destruction of local stocks.
Group structure and social behavior vary between the different species of Cetaceans, and also diurnally and seasonally. Diurnal and seasonal variations in behavior can change the risk of exposure to oil considerably. For species where schooling is part of their social behavior the effect of a spill can be catastrophic, and the number of affected animals can be very high.

It is important to state that the only marine mammal actually affected by oil pollution is the sea otter (*Enhydra lutris*). In March 1989 the Exxon Valdez oil spill posed a great threat to the sea otters mainly because they rely on pelage rather than blubber for insulation, oil residues will drastically reduce the insulative value of the fur. 871 animals were found dead, in addition 123 died in rehabilitation centers (Ballachey *et al.* 1994).

5.3.4 Fate of petroleum oils in the marine environment

The fate of oil differ with each category of discharge and petroleum product, and are affected by the timing and relative importance of biological, physical and chemical processes. The properties (physical and chemical) of spilled oil are altered by weathering, this again will influence the toxicity to marine organisms. The most important weathering processes includes microbial degradation, formation of emulsions, spreading, evaporation, dissolution, absorption to suspended particulate matter, dispersion into the water column, stranding on shore, sedimentation to the seafloor or photochemical oxidation (Neff 1990a).

Type of oil, location of the event and climatic conditions determines the physical impact and fate of a spill. Most environments recover rather quickly after a spill, on the other hand, the Arctic, as well as estuaries and salt marshes are highly sensitive. The Arctic due to low temperatures causing slower breakdown of petroleum and prolonged recovery, the latter because trapped oil followed by delayed or prevented weathering. According to Neff (1990b) no direct relation can be seen between marine mammal trophic level and the concentration of residues.

6. Risk assessment

In both cetaceans and pinnipeds the dominant system in metabolizing OCs is the P450 enzyme system. However, there is a clear difference in metabolic capacity between cetaceans and seals, between species of both groups, and even between individuals (Tanabe *et al.* 1988). There are good indications that pollutants can have a significant impact on marine mammals.
However, the exact threats are difficult to quantify. This is mainly due to lack of knowledge of actual physiological effects of known contaminants and unknown threats by scarcely studied contaminants. We also have little knowledge of future trends in global pollution and contaminant disposal. These questions have been addressed by designing a conceptual framework for prediction of pollution impact on marine mammals, based on multiple response assessment as elaborated by Reijnders (1994).

According to Harding et al. 2007, when investigating the vulnerability of populations to different future scenarios, risk assessments can be used as a tool. It is a scientific process that can be divided into several stages, including exposure assessment, elucidation of dose response relationships and hazard identification. To achieve results in scientific risk characterizations, all these activities should be integrated. To reduce uncertainties involved in risk characterization, certain assumptions, dosimetric and mechanistic information should be incorporated in place of default positions (Birnbaum 1994).

To determine any population or species-level impacts, it is essential to assess the sources, biological effects of contaminants on individuals and levels and patterns of contaminants. The ecological risk presented by new chemicals with persistent or lipophilic characteristics like those already found in marine mammals, continued leaking of compounds stored, and diet selection as a source of contaminants for humans. When combining epidemiological or descriptive studies of contaminated areas and the effects seen in the marine mammals, the use of single- or multiple chemical exposures in acute or chronic designs in mechanistic, cause-and-effect, laboratory rodent studies, marine mammals fed fish from contaminated areas in semi-field or captive studies, and laboratory studies using rodent species as surrogates for marine mammals, levels of lipophilic contaminants effect on reproduction, immune function and endocrine function can be determined. They can also be used to identify the ecological risks presented above (Ross 2000, Fisk et al.2005). Another approach is to study the use of biomarkers, biological responses related to contaminant exposure. These are measurements of normal processes taking on abnormal values due to exposure of contaminants (Fisk et al.2005).

Assessing risks associated with exposure of wildlife to complex environmental mixtures of chemicals can be difficult, but on the other hand, can marine mammals serve as useful indicators of ecosystem contaminations. As mentioned by Ross (2000) are studies of
contaminants and contaminant-related effects in marine mammals important for several reasons (1) concerns about the well-being of viable populations of mammals in coastal waters and oceans by wildlife managers and conservationists, (2) policy makers and managers’ concern with the degree of contamination of the marine food chain (bioaccumulation of contaminants and marine ecosystem health) and (3) the health of certain human consumer groups (aboriginal groups and fishers) worries health experts and managers.

6.1 Risk assessment and oil
Several methods can be used in assessing the effects of an oil spill on marine mammals; (1) use of mathematical method which incorporates information on the behaviour of spilled oil with the data on the life history of the species in question, (2) understanding of direct and indirect effects of oil on individuals – information can be generated from field observation and experimental studies. Advantages of computer modelling always produce a result – a prediction of impact and when developing offshore oil reserve, it can be applied as part of risk assessments. On the negative side, modelling also has several limitations, including that simplified assumption must be made to integrate processes highly variable and complex, the predictions made are only as reliable as the data used to drive the model, and the propagation and perhaps magnification of errors in the first when models are linked (output from one is used as input to another). Identifying gaps in data and helping to determine where research support should be directed, is the greatest utility of a model. While some computer models have dealt with the behaviour of marine mammals and population dynamic, others have been developing and evaluating in order to predict the trajectory and fate of oil spilled in the marine environment. Sensitivity analysis showed that the time of year, timing of the stages in the life cycle of the population being modelled, and the size and trajectory of the oil spill are the most important factors determining the predicted proportion of the affected pollution. The outcome of a model run is dependent on both animals and oil occurring at the same place at the same time. (Neff 1990b). Rare or endangered, or species especially vulnerable to pollutants are most vulnerable (Neff 1990a), and the best available science should be included to improve the risk assessment process (Birnbaum 1994).

7. Discussion
Environmental pollution has caused great concern about the effects on humans and other wildlife the last few decades. Synthetic compounds, like organochlorines, are man-made and do
not occur naturally in the environment. In the 1950’s and 1960’s the discovery of persistent compounds being globally widespread led to further investigation of the subject by numerous researchers worldwide. Detectable concentrations are found even in the most remote areas of the world and virtually all organisms are exposed to these persistent compounds.

In most places where restriction and banning of compounds have been enforced, several studies report of declines in the POP residue levels in the environment (Addison et al 1986). On the other hand, substantial quantities of PCBs remain in use and are stored in landfills in large quantities. Redistribution and leakages of these compounds are worrying, and Tanabe (1988) has predicted that the PCB levels in the global environment are unlikely to decrease in the near future.

In the aquatic environment, high levels of PCB contamination tend to occur particularly near heavily industrialized areas with active emission sources. Areas with highly polluted rivers, lakes and estuaries are shown to contain much higher levels of PCB than non-polluted waters. According to WHO (1993) non-polluted waters of the Great Lakes region have been estimated to contain <5 ng/l, while heavily polluted rivers and estuaries may reach 500 ng/l.

The primary mechanisms leading to the contamination of remote environments are atmospheric transport and deposition of PCBs and similar pollutants are the primary mechanisms behind contamination of remote regions (Buckley 1982). PCBs are continuously being transported to the Arctic via the atmosphere (Hargrave et al. 1988), deposited in the aquatic and terrestrial systems (Gregor and Grummer 1989), and incorporated into Arctic food webs (Muir et al. 1988). Degradation of PCBs is generally very slow in the aquatic environment (Strachan 1988) due to the fact that most PCBs lie buried in sediments and are inaccessible to sunlight or volatisation unless disturbed (Moore and Walker 1991), the half-lives of PCBs in the sediments is therefore very long, in the order of decades.

Bioaccumulation is a critical aspect of the environmental fate and behavior of PCBs. As a result of high lipid solubility and slow rate of metabolism and elimination they tend to accumulate in almost all organisms (WHO 1993). These characteristics permit PCBs to accumulate to relatively high levels in the biota, even at low exposure rates. Exposure to sustained low levels of PCBs in the abiotic environment may result in adverse chronic effects.
in the biota. The temporal trends of PCBs and DDT were studied by Addison et al. (2005) and Muir and Kwan (2003) and these studies showed that from the early 1970’s to the late 1990’s the organochlorines declined significantly by a factor of 20 – 70% at all studied sites. A majority of the decline occurred in the 1970’s and since the mid 1980’s the concentration levels have levelled out and stayed fairly constant without any trend.

Riget et al. (2005) found that for ringed seals, the highest concentrations of mercury were at three Canadian locations: Grise fjord (Nunavut), Sachs Harbour and Holma (NWT), while the lowest levels of OCs were observed in the western and eastern Canadian Arctic archipelago. According to Muir et al. (2000) walruses feed predominantly on benthic invertebrates and due to low concentrations of pollutant accumulated at lower trophic levels, walruses therefore have lower concentrations of POPs than other other marine mammals in the Arctic. Concentrations of many organochlorines in Canadian Arctic marine biota are generally lower than in European Arctic and eastern Greenland, but are higher than in Alaska compared to other circumpolar countries (Braune et al. 2005), and in Canada the concentrations of Hg are substantially higher than elsewhere. The focus on marine mammals stems from their importance in the culture and diet of Inuits, not to mention that the levels of persistent OCs and Hg are elevated in marine mammal tissues. However, it is important to emphasized that spatial trends of persistent OCs can generally only be done qualitatively because they are based on different analytical techniques and definition of various OC groups (including the congeners) and frequently important biological data such as age, sex, blubber thickness, reproductive status, are lacking for many species (Muir et al. 2000).

Wageman et al. (1996) reported that Hg in livers of beluga increased significantly in both the western and eastern Canadian Arctic from 1980 to 1994. For the Beaufort Sea coast, Lockhart et al. (2005) reported a 4-fold increase in hepatic Hg in beluga, with most of the increase occurring in the early 1990s, but no further change between 1996 and 2002. Belugas in the eastern Canadian Arctic showed a similar trend. A closer examination of the data revealed that beluga showing the largest Hg increases were collected in areas influenced by large freshwater drainages (Mackenzie River, Nelson river, James bay rivers). This suggests that changes in Hg accumulation by the Beluga may be related to freshwater input in these waters (Lockhart et al. 2005).
In the western Arctic, Hg concentrations in modern Beaufort Sea beluga appears to be of the order 10 fold higher than 500 years ago, based on the analyses of teeth (Braune et al. 2005). There is also evidence of a several-times Hg increase in modern polar bears over the pre-industrial background, based on hair samples. The level of thyroid hormones declines with increasing concentration of PCB. In addition dioxins are known to have a negative effect on immune function.

8. Conclusion

Even though it can be difficult to point out precise cause-effect relationships between pollutants and marine mammals in many cases there is no doubt that various pollutants still pose major threats to marine ecosystems. Some species are well documented, but insufficient data supporting biological, chemical and epidemiological findings in certain species enhance the need for further investigations. Particularly in the Southern hemisphere and for species inhabiting these areas, the lack of knowledge is insufficient. On the other side, all nations are actively trying to reduce the emission of pollutants, and with all the available techniques that exist monitoring.

In case of low concentrations in the nature, reasons for concern are still there: little is known about the long-term effects of chronic exposure to low concentrations, and further investigations are needed.

9. Acknowledgement

I would like to thank my supervisor Karl Inne Ugland for all the help and guidance he has provided during these years working on my thesis. A special thank to my parents, Anne and Ervin Friseid, for their support, both in words and economically and their confidence in me. Would also like to thank my friends and co-students Silje, Elin og Maia, and my boyfriend Håvar for always being there and bearing with me during this time.
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