Marine Mammals as Sentinels in Ecological Risk Assessment

Peter S. Ross
Institute of Ocean Sciences, Sidney, BC V8L 4B2, Canada*

ABSTRACT

As high trophic level organisms in the marine environment, fish-eating seals, dolphins and whales are often exposed to very high levels of fat-soluble environmental contaminants. Assessing the sources, levels and patterns of contaminants found in the tissues of marine mammals, and the biological effects of these contaminants on individuals, is essential to determining any population- or species-level impacts. While the number of contaminants to which marine mammals are exposed is staggering, designing strategies to assess the effects of complex mixtures represent a challenging yet vital part of an understanding of the "real world". At present, an accumulated "weight of evidence" suggests that ambient levels of lipophilic contaminants have adversely affected aspects of reproduction, immune function and endocrine function in marine mammals inhabiting a number of industrial coastal regions. This body of evidence is drawn from a combination of (1) epidemiological or descriptive studies of effects observed in free-ranging populations of marine mammals inhabiting contaminated areas; (2) mechanistic, cause-and-effect, laboratory rodent studies, using single- or multiple- chemical exposures in acute or chronic designs; (3) semi-field or captive studies of marine mammals fed fish from contaminated areas; and (4) laboratory studies where rodent species are used as surrogates for marine mammals, and are exposed to extracts of complex contaminant mixtures found in fish. While these approaches have been used to delineate the effects of historically introduced environmental contaminants such as PCBs and DDT on wildlife, they may serve to identify the ecological risks presented by (1) the continued leaking of stored, discontinued, chemical supplies that have not yet been destroyed (e.g., PCBs); (2) new chemicals that may have lipophilic or persistent characteristics similar to those found currently in marine mammals; and (3) diet selection as a source of contaminants for humans, since certain human groups share the same food chain with marine mammals. Contaminant mixtures to which marine
mammals are exposed differ greatly from the original industrial mixtures as a result of differing rates of accumulation and capacities to metabolize certain chemical types by the various trophic levels (e.g., invertebrates, fish, and marine mammals) of the food chain. Marine mammals ultimately provide information on the chemicals which present the greatest risk to consumers at the top of the food chain, something that cannot be adequately described or predicted in laboratory models.

**Key Words:** marine mammals, contaminants, risk assessments, food chain, wildlife, toxicology.

**INTRODUCTION**

Many marine mammal species occupy elevated trophic levels in the oceanic environment and have been found to accumulate high concentrations of numerous fat-soluble environmental chemicals, including the polychlorinated biphenyls (PCBs), -dibenzo-α-dioxins (PCDDs or dioxins), and -dibenzofurans (PCDFs or furans), as well as pesticides, including the DDT family (Addison et al., 1986; Tanabe et al., 1993; Norstrom and Muir, 1994). While it is difficult to directly assess the risks associated with the exposure of wildlife to complex environmental mixtures of chemicals, marine mammals can serve as useful indicators of ecosystem contamination. Studies of contaminants and contaminant-related effects in marine mammals are important for (1) wildlife managers and conservationists concerned with the well-being of viable populations of these animals in our coastal waters and oceans; (2) managers and policy makers concerned with the degree of contamination of the marine food chain, since marine mammals bioaccumulate high levels of fat-soluble contaminants and thereby “integrate” contaminant information in the environment (i.e., marine ecosystem health); and (3) health experts and managers concerned with the health of certain human consumer groups that rely heavily on fish consumption, including aboriginal groups and fishers, who share the same food chain with marine mammals, or people who consume marine mammals.

In addition, because of the differential bioaccumulation potentials and excretion capabilities for the various contaminants at different levels of the aquatic food chain, marine mammals and other high trophic level consumers are exposed to mixtures that often differ greatly from the original industrial product. Laboratory-based toxicological studies examining the effects of particular chemicals therefore may not accurately gauge the risks associated with real world exposures. Studies of wildlife, including fish-eating birds and marine mammals, can provide an indication of more realistic environmental exposures.

While the complex nature of the contaminant mixture to which marine mammals are exposed in the environment has precluded any conclusive mechanistic understanding of a possible toxicity (Addison, 1989), there is an increasing “weight of evidence” that implicates anthropogenic contaminants with adverse effects in free-ranging marine mammals. This evidence is based largely on four lines of toxicological research, including (1) epidemiological (correlative) or descriptive (associative) studies of free-ranging populations of marine mammals inhabiting contaminated areas; (2) mechanistic, cause-and-effect, laboratory rodent studies, using single- or multiple-exposures in acute or chronic designs that support obser-
vations in (1); (3) semi-field or captive studies of marine mammals (usually harbor seals) fed fish from contaminated areas; and (4) laboratory studies where rodent species are used as surrogates for marine mammals and are exposed to the complex contaminant mixtures found in fish.

THE WEIGHT OF EVIDENCE

The first of these approaches helped to draw attention to the issue of contaminants in marine mammals in the earlier days of wildlife toxicology, as aberrant or unusual phenomena were recorded in marine mammals that were highly contaminated with a number of not-yet regulated industrial chemicals, including PCBs and DDT. Numerous reports provided indications that contaminants might be affecting free-ranging marine mammals, including observations of pathogen-associated abortions in California Sea lions, *Zalophus californianus* (Delong *et al.*, 1973), tumors, and low recruitment in St. Lawrence Beluga whales, *Delphinapterus leucas* (Martineau *et al.*, 1994; De Guise *et al.*, 1995), skeletal malformations in Baltic Sea seals (Bergman *et al.*, 1992; Mortensen *et al.*, 1992), and impaired reproduction in European seals (Helle *et al.*, 1976). However, it was difficult to conclusively link these observed effects with contaminant exposure. Studies of fish-eating birds provided more direct evidence that contaminants were affecting wildlife, as DDT was implicated in eggshell thinning and the consequent extirpation of many species of fish-eating birds from large parts of North America and Europe (Jensen, 1966; Hickey and Anderson, 1968; Wiemeyer and Porter, 1970). Not only were piscivorous animals in the environment heavily contaminated with numerous chemicals, but they were also being affected by these. Subsequently, correlative approaches were used to link PCBs to diminished T-cell function in free-ranging bottlenose dolphins, *Tursiops truncatus*, live-captured in the Gulf of Mexico (Lahvis *et al.*, 1995), and total DDT to decreased testosterone levels in Dall's porpoises, *Phocoenoides dalli*, caught incidentally in North Pacific fisheries (Subramanian *et al.*, 1987), although small sample size and a lack of age information makes it difficult to assess the ecological significance of these results.

Secondly, mechanistic studies carried out using laboratory animals provided evidence that could explain the observations in wildlife species. Eggshell thinning was observed following DDT exposure in egg-laying birds (Fry, 1995), immunotoxicity in laboratory animals exposed to PCBs and dioxins (Vos and Van Driel-Grootenhuis, 1972; Vos and Luster, 1989), and reproductive impairment in mink and ferrets exposed to PCBs (Bleavins *et al.*, 1980). Carefully controlled laboratory studies over the last 25 years have resulted in a vast database that has served not only to identify many contaminants of concern (Luster *et al.*, 1992; Luster *et al.*, 1993), but has also provided a preliminary means of assessing the relative risks that these chemicals might present to marine mammals and other wildlife. Such extrapolations involve many of the same assumptions implicit in the use of rodent models to assess the potential risk of chemicals to human health (Luster *et al.*, 1994), and are based on interspecies similarities in physiology and in the mechanism of toxic action for the chemical in question. The identification of the Aryl hydrocarbon (Ah)-receptor in the tissues of all mammals studied to date represents an example of a means of assessing the dioxin-like toxic risk of PCBs, PCDDs, and PCDFs to different mamma-
vations in (1); (3) semi-field or captive studies of marine mammals (usually harbor seals) fed fish from contaminated areas; and (4) laboratory studies where rodent species are used as surrogates for marine mammals and are exposed to the complex contaminant mixtures found in fish.

THE WEIGHT OF EVIDENCE

The first of these approaches helped to draw attention to the issue of contaminants in marine mammals in the earlier days of wildlife toxicology, as aberrant or unusual phenomena were recorded in marine mammals that were highly contaminated with a number of not-yet regulated industrial chemicals, including PCBs and DDT. Numerous reports provided indications that contaminants might be affecting free-ranging marine mammals, including observations of pathogen-associated abortions in California Sea lions, Zalophus californianus (Delong et al., 1975), tumors, and low recruitment in St. Lawrence Beluga whales, Delphinapterus leucas (Martineau et al., 1994; De Guise et al., 1995), skeletal malformations in Baltic Sea seals (Bergman et al., 1992; Mortensen et al., 1992), and impaired reproduction in European seals (Helle et al., 1976). However, it was difficult to conclusively link these observed effects with contaminant exposure. Studies of fish-eating birds provided more direct evidence that contaminants were affecting wildlife, as DDT was implicated in eggshell thinning and the consequent extirpation of many species of fish-eating birds from large parts of North America and Europe (Jensen, 1966; Hickey and Anderson, 1968; Wiemeyer and Porter, 1970). Not only were piscivorous animals in the environment heavily contaminated with numerous chemicals, but they were also being affected by these. Subsequently, correlative approaches were used to link PCBs to diminished T-cell function in free-ranging bottlenose dolphins, Tursiops truncatus, live-captured in the Gulf of Mexico (Lahvis et al., 1995), and total DDT to decreased testosterone levels in Dall's porpoises, Phocoenoides dalli, caught incidentally in North Pacific fisheries (Subramanian et al., 1987), although small sample size and a lack of age information makes it difficult to assess the ecological significance of these results.

Secondly, mechanistic studies carried out using laboratory animals provided evidence that could explain the observations in wildlife species. Eggshell thinning was observed following DDT exposure in egg-laying birds (Fry, 1995), immunotoxicity in laboratory animals exposed to PCBs and dioxins (Vos and Van Driel-Grootenhuis, 1972; Vos and Luster, 1989), and reproductive impairment in mink and ferrets exposed to PCBs (Bleavins et al., 1980). Carefully controlled laboratory studies over the last 25 years have resulted in a vast database that has served not only to identify many contaminants of concern (Luster et al., 1992; Luster et al., 1993), but has also provided a preliminary means of assessing the relative risks that these chemicals might present to marine mammals and other wildlife. Such extrapolations involve many of the same assumptions implicit in the use of rodent models to assess the potential risk of chemicals to human health (Luster et al., 1994), and are based on interspecies similarities in physiology and in the mechanism of toxic action for the chemical in question. The identification of the Aryl hydrocarbon (Ah)-receptor in the tissues of all mammals studied to date represents an example of a means of assessing the dioxin-like toxic risk of PCBs, PCDDs, and PCDFs to different mamma-
Table 1. Summary of effects observed in captive harbor seal study and the subsequent parallel study of rats exposed perinatally to a lipid extract of the same herring from the contaminated Baltic Sea fed to the seals (adapted from (Ross et al., 1996) and (Ross et al., 1997)). Effects are relative to the respective control groups fed Atlantic Ocean herring (seals) or lipid extracts from the herring (pregnant and nursing rats). The sensitivity of T-cell function and the thymus, but not B-cell function, to the immunotoxic actions of complex environmental mixtures is characteristic of dioxin-like effects in single-chemical, laboratory rodent studies.

<table>
<thead>
<tr>
<th>Effect</th>
<th>&quot;Baltic group&quot; of captive seals</th>
<th>&quot;Baltic group&quot; of rats exposed perinatally</th>
<th>&quot;TCDD group&quot; of rats exposed perinatally</th>
</tr>
</thead>
<tbody>
<tr>
<td>T-cell function, <em>in vitro</em></td>
<td>Diminished</td>
<td>Diminished</td>
<td>Diminished</td>
</tr>
<tr>
<td>Delayed-type hypersensitivity</td>
<td>Diminished</td>
<td>Unaffected</td>
<td>Unaffected</td>
</tr>
<tr>
<td>Thymus cellularity</td>
<td>Not done</td>
<td>Diminished</td>
<td>Diminished</td>
</tr>
<tr>
<td>B-cell function</td>
<td>Unaffected</td>
<td>Unaffected</td>
<td>Diminished</td>
</tr>
<tr>
<td>Baseline or virus-associated natural killer (NK) cell activity</td>
<td>Diminished</td>
<td>Diminished</td>
<td>Diminished</td>
</tr>
</tbody>
</table>

more invasive experiments and host resistance (virus challenge) assays represented important advantages in these largely immunotoxicological studies.

In the first of these, juvenile rats were fed freeze-dried herring from the same two batches used in the captive seal study (Ross et al., 1996b). While laboratory testing revealed no overt effect on immune function in these animals, diminished thymocyte numbers, lower CD4:CD8 ratios, and a higher virus titer following challenge with rat cytomegalovirus suggested immunotoxicity and a diminished host resistance to infectious disease (see Table 1).

In the second of these studies, rat pups born to mothers exposed orally on a daily basis during pregnancy and lactation to oil extracts from either the Atlantic or the Baltic herring, or the Atlantic herring plus a low level of 2,3,7,8-TCDD, were examined for immunotoxicity (Ross et al., 1997). Rat pups in both the Baltic and TCDD groups exhibited diminished T-cell function, consistent with observations in the seal study (see Table 1) and immunotoxicological studies of dioxin-like compounds in laboratory mammals (Vos and Luster, 1989). In addition, transient effects on thymus cellularity and diminished specific antibody responses following virus
challenge provided additional information that was used to extrapolate to the seal study. While precise comparisons are difficult, the exposure levels (i.e., daily intakes) for dioxin-like contaminants were similar between the Baltic group of rats and the Baltic group of seals, strengthening the implications and the relevance of the effects observed in both species.

While marine mammal research may represent the best “real world” approach among these lines of research, the diminished confidence about mechanisms of action, and cause-and-effect, in any form of wildlife toxicology necessitates a multi-tiered strategy. A “weight of evidence” is accumulated by an extrapolation across these approaches, with a robust ecological risk assessment process helping to evaluate the strengths and weaknesses of the extrapolations (see Figure 1). For example, the risk of adverse effects in free-ranging marine mammals may be predicted on the

---

**Figure 1.** Extrapolations are required in assessing the risk of environmental contaminants to marine mammals. Since uncertainties and a low degree of confidence exist for studies of free-ranging marine mammals, risk assessment is best served by an accumulation of a “weight of evidence” from a combination of these approaches. A wealth of toxicological information has been generated for many chemical products using laboratory rodent studies, but similar approaches are neither practical nor ethically acceptable in marine mammals. The alternative would involve a risk assessment based on a combination of investigative approaches. In this manner, ecologically relevant information can be generated (i.e., free-ranging marine mammals) that has a mechanistic basis in toxicology (i.e., laboratory rodent studies), and uncertainties can be minimized by appropriate and critical extrapolations among each of these lines of research. A comparison of exposure levels or burdens in different species or experimental approaches could then be used in predicting risk of toxicity.
basis of (1) the levels and types of chemical contaminants found in samples obtained from a population in question; (2) a comparison of these levels to those which caused adverse effects in captive harbour seals; and (3) a comparison of levels, types, and effects of these chemicals in both single-chemical or complex mixture design in laboratory animals. Interspecies similarities and differences must be addressed during the extrapolations. Similarities in immunotoxic effects observed in fish, birds, and marine mammals exposed to environmental contaminants through their diets underline the utility of studying free-ranging organisms (Luebke et al., 1997).

**COMPLEX MIXTURES IN THE “REAL WORLD”**

While the weight of evidence to date suggests that certain marine mammal populations exposed to ambient environmental levels of a complex mixture of lipophilic chemical contaminants have been adversely affected, it has been impossible to identify any one particular chemical or chemical class responsible for these effects. Practical, legal, and ethical considerations have precluded mechanistic, cause-and-effect studies in marine mammals. However, the combination of approaches described above have helped to shed some light on chemical classes that may present the greatest risk to marine mammals today, and that may have caused some of the bioeffects described in earlier studies.

The dioxin-like chemicals, including some members of the PCBs, PCDDs, and PCDFs, have been implicated in thymus atrophy and immunotoxicity in laboratory-based rodent studies (Vos and Luster, 1989), while both PCB hydroxy-metabolites and dioxin-like chemicals have been implicated in lower plasma levels of vitamin A and thyroid hormone (Brouwer et al., 1986; Brouwer et al., 1998). Many of the observations in free-ranging marine mammals and captive harbor seals are consistent with these effects of the dioxin-like, Ah-receptor binding, chemicals and the PCB metabolites. In the parallel perinatal rodent experiment aimed at mimicking the seal feeding study (Ross et al., 1997), not only were the effects in the Baltic group consistent with the immunotoxic actions of dioxin-like chemicals, but similar, yet more pronounced, effects were observed in the third, positive control, 2,3,7,8-TCDD-spiked, Atlantic herring oil-exposed group. While such studies do not conclusively establish the dioxin-like chemicals as responsible for the effects described in the immunotoxicological harbour seal study, they are highly suggestive and highlight the relative risks that such chemicals present to free-ranging marine mammals.

Similarity in chemical structures and the identification of the Ah-receptor provide a means of simplifying the complexity of the 419 possible PCB, PCDD, and PCDF congeners, thereby allowing for a ranking of the relative dioxin-like risks presented by each of these chemical classes. The development of TEFs on the basis of the relative toxicities of the dioxin-like PCB, PCDD and PCDF congeners to the most toxic of these, 2,3,7,8-TCDD (Van Zorge et al., 1989; Safe, 1992; Ahlborg et al., 1994), represents the best current alternative to the use of actual chemical concentrations. The simplified, mechanistically defined TEQ provides a useful summarizing approach that predicts the dioxin-like risk of each of these chemical classes to marine mammals.

When expressed as a percentage of the total TEQ, PCBs have often been found to present the greatest dioxin-like risk in studies of wildlife species (Tillitt et al.,
basis of (1) the levels and types of chemical contaminants found in samples obtained from a population in question; (2) a comparison of these levels to those which caused adverse effects in captive harbour seals; and (3) a comparison of levels, types, and effects of these chemicals in both single-chemical or complex mixture design in laboratory animals. Interspecies similarities and differences must be addressed during the extrapolations. Similarities in immunotoxic effects observed in fish, birds, and marine mammals exposed to environmental contaminants through their diets underline the utility of studying free-ranging organisms (Luebke et al., 1997).

COMPLEX MIXTURES IN THE “REAL WORLD”

While the weight of evidence to date suggests that certain marine mammal populations exposed to ambient environmental levels of a complex mixture of lipophilic chemical contaminants have been adversely affected, it has been impossible to identify any one particular chemical or chemical class responsible for these effects. Practical, legal, and ethical considerations have precluded mechanistic, cause-and-effect studies in marine mammals. However, the combination of approaches described above have helped to shed some light on chemical classes that may present the greatest risk to marine mammals today, and that may have caused some of the bioeffects described in earlier studies.

The dioxin-like chemicals, including some members of the PCBs, PCDDs, and PCDFs, have been implicated in thymus atrophy and immunotoxicity in laboratory-based rodent studies (Vos and Luster, 1989), while both PCB hydroxy-metabolites and dioxin-like chemicals have been implicated in lower plasma levels of vitamin A and thyroid hormone (Brouwer et al., 1986; Brouwer et al., 1998). Many of the observations in free-ranging marine mammals and captive harbor seals are consistent with these effects of the dioxin-like, Ah receptor binding, chemicals and the PCB metabolites. In the parallel perinatal rodent experiment aimed at mimicking the seal feeding study (Ross et al., 1997), not only were the effects in the Baltic group consistent with the immunotoxic actions of dioxin-like chemicals, but similar, yet more pronounced, effects were observed in the third, positive control, 2,3,7,8-TCDD-spiked, Atlantic herring oil-exposed group. While such studies do not conclusively establish the dioxin-like chemicals as responsible for the effects described in the immunotoxicological harbour seal study, they are highly suggestive and highlight the relative risks that such chemicals present to free-ranging marine mammals.

Similarity in chemical structures and the identification of the Ah receptor provide a means of simplifying the complexity of the 419 possible PCB, PCDD, and PCDF congeners, thereby allowing for a ranking of the relative dioxin-like risks presented by each of these chemical classes. The development of TEFs on the basis of the relative toxicities of the dioxin-like PCB, PCDD and PCDF congeners to the most toxic of these, 2,3,7,8-TCDD (Van Zorge et al., 1989; Safe, 1992; Ahlborg et al., 1994), represents the best current alternative to the use of actual chemical concentrations. The simplified, mechanistically defined TEQ provides a useful summarizing approach that predicts the dioxin-like risk of each of these chemical classes to marine mammals.

When expressed as a percentage of the total TEQ, PCBs have often been found to present the greatest dioxin-like risk in studies of wildlife species (Tillitt et al.,
concentrations of lipophilic chemicals increase, while concentrations decline in females as they reproduce and transfer their contaminant burden to their offspring transplacentally and via lactation (Addison and Stobo, 1993; Borrell et al., 1995; Nakata et al., 1995). The use of "mean concentrations" in studies of free-ranging marine mammals therefore is misleading in the absence of age and sex data. In addition, contaminant burdens may differ between age class partly as a result of historical changes in environmental contamination and the long lifespan of most marine mammals. Other factors that affect contaminant levels in marine mammals include prey selection (Storr-Hansen et al., 1995; Muir et al., 1995), condition (Klevane et al., 1995), and less well understood factors, including migration. Eliminating or characterizing such confounding factors therefore should be an integral part of any study that aims to quantify contaminant levels and predict risks on this basis to a marine mammal population.

THE HARBOR SEAL AS A SENTINEL SPECIES FOR MARINE MAMMALS

Marine mammals are large organisms living in an aquatic environment that is often poorly understood. Any studies attempting to evaluate the potential risks presented by contaminants to the well-being of marine mammals face tremendous obstacles, with these being very likely insurmountable in the case of large cetaceans. It is perhaps no coincidence that the harbor seal has emerged as an important sentinel species for many European and North American toxicologists attempting to assess the potential risks of contaminant exposure to other marine mammal species.

The harbor seal is a relatively small (adults 65 to 130 kg), nonaggressive, long-lived (males live to 20 years, females to 30 years) animal that is widely distributed along most temperate coastlines in the northern hemisphere. This pinniped is largely non-migratory and inhabits both contaminated and uncontaminated coastal environments. The harbor seal was also the primary victim of the *Morbillivirus*-associated mass mortality in northern Europe, which drew attention (and scientific resources) to this species. Many carefully designed studies have been carried out that have documented aspects of harbor seal diseases, physiology, behavior, endocrinology, immunology, and toxicology. The collection of blubber samples from other marine mammal species can provide detailed contaminant information which can be compared with established "effects levels" in harbor seals (e.g., the 17 mg/kg ΣPCB or 209 ng/kg TEQ, which proved to be immunotoxic and endocrine-disrupting in the captive harbour seal study (De Swart et al., 1996; Ross et al., 1996)). While any interspecies extrapolation must be made carefully, the use of harbor seals as sentinels for other marine mammals might be likened to the use of laboratory rodents in pharmacologic and toxicologic studies assessing potential risks associated with exposure to chemicals or drugs for humans.

HUMANS AT THE TOP OF THE FOOD CHAIN

While marine mammals represent some of the most contaminated organisms in the world, humans occupying similar niches have also been found to be highly contaminated with PCBs and other fat-soluble contaminants. Inuit in the Canadian
Arctic, for example, have PCB levels that are five to ten times higher than the average southern Canadian, reflecting their consumption of large quantities of fish and marine mammal tissues (Dewailly et al., 1993; Kuhnlein et al., 1995; CACAR, 1997). Certain human consumer groups, including families of Lake Michigan sportfisheirs (Pelletier et al., 1996), subsistence groups on the north shore of the St. Lawrence estuary (Ayotte et al., 1997), and consumers of Baltic Sea fish (De Wit et al., 1992), all have elevated levels of lipophilic environmental chemicals. Studies of several human cohorts are also beginning to reveal adverse effects of these dietary exposures to contaminants, including effects on thyroid hormone concentrations and neurological development in breast-fed infants in the Netherlands (Pluim et al., 1993; Koopman-Esseboom et al., 1994; Koopman-Esseboom et al., 1996), neurotoxicity in children born to Lake Michigan sportfisheir families (Jacobson and Jacobson, 1997), and possible immunotoxicity in Inuit infants (Dewailly et al., 1995).

A direct comparison of humans with marine mammals has obvious limitations, but the evaluation of exposure levels and patterns can provide an initial basis for assessing the possible risks associated with a dietary intake of a similar complex mixture of contaminants (see Table 2). The human consumer groups that are at the greatest risk of contaminant-related effects are likely to be those that are most ecologically similar to marine mammals, and despite differences in behavior, physiology, ecology, and genetic susceptibility, comparisons between these two high trophic level animals are inescapable. In this regard, the fact that exposure levels of Inuit infants are approaching those of the captive harbour seal that suffered from immunotoxicity and endocrine disruption may be cause for concern.

CONCLUSIONS

Marine mammals are exposed to myriad environmental contaminants. A combination of studies aimed at characterizing contaminant patterns, levels and possible bioeffects in populations inhabiting contaminated areas, captive feeding studies that reflect real world feeding scenarios, and the design and application of laboratory rodent experiments that parallel or mimic marine mammal studies have helped to identify marine mammal populations at risk for the adverse effects of certain chemicals. Much of the theoretical and mechanistic bases for interpreting such studies rely on the numerous laboratory rodent studies that have used simplified exposure regimes in acute or chronic designs. In this way, fat-soluble, environmental contaminants, and particularly the PCBs, have been identified as chemicals that have likely affected the well-being of several marine mammal species in the past, and will continue to present a tangible risk to free-ranging marine mammals well into the 21st century (Ross et al., 1996). While the implementation of regulatory controls for PCBs in the 1970s led to declines in PCB levels in wildlife compartments, recent evidence suggests that these have stabilized since the mid-1980s (Olsson and Reutergardh, 1986; Loganathan et al., 1990; Addison and Smith, 1998; Bignert et al., 1998).

A better understanding of the effects of PCBs and related contaminants on marine mammals may not only help in our understanding of the risks associated with the contamination of the global environment, but can and should provide “real
The estimated average daily intakes of total 2,3,7,8-TCDD-Toxic Equivalents (TEQ) for PCBs, PCDDs and PCDFs in different species and human groups. Numbers are expressed as ng/kg body weight, and an indication is given if any adverse effects were observed during the course of the studies. Effects observed in such cases included one or more of: immunotoxicity, neurotoxicity and disruption of thyroid hormones and retinoids.

<table>
<thead>
<tr>
<th>Group</th>
<th>Exposure route</th>
<th>Tissue</th>
<th>Daily intake</th>
<th>Total TEQ</th>
<th>Effects observed</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Harbour seal</td>
<td>Atlantic Ocean</td>
<td>herring</td>
<td>0.3-0.6</td>
<td>62</td>
<td>n.a.</td>
<td>(De Swart et al., 1994)</td>
</tr>
<tr>
<td></td>
<td>Perinatally to lipid extract of Atlantic herring</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(Ross et al., 1997)</td>
</tr>
<tr>
<td>Harbour seal</td>
<td>Baltic Sea herring</td>
<td>(in blubber)</td>
<td>1.2-5.6</td>
<td>209</td>
<td>Yes</td>
<td>(De Swart et al., 1996)</td>
</tr>
<tr>
<td></td>
<td>Perinatally to lipid extract of Baltic herring</td>
<td>(in blubber)</td>
<td></td>
<td></td>
<td></td>
<td>(Ross et al., 1997)</td>
</tr>
<tr>
<td>PVG rat</td>
<td>Perinatally to lipid extract of Atlantic herring</td>
<td></td>
<td>0.5</td>
<td>?</td>
<td>Yes</td>
<td>(Ross et al., 1997)</td>
</tr>
<tr>
<td>PVG rat</td>
<td>Perinatally to lipid extract of Baltic herring</td>
<td></td>
<td>2.1</td>
<td>?</td>
<td>Yes</td>
<td>(Ross et al., 1997)</td>
</tr>
<tr>
<td>PVG rat</td>
<td>Perinatally to 2,3,7,8-TCDD-spiked Atlantic herring</td>
<td></td>
<td>154</td>
<td>?</td>
<td></td>
<td>(Ross et al., 1997)</td>
</tr>
</tbody>
</table>
Table 2. The estimated average daily intakes of total 2,3,7,8-TCDD-Toxic Equivalents (TEQ) for PCBs, PCDDs and PCDFs in different species and human groups. Numbers are expressed as ng/kg body weight, and an indication is given if any adverse effects were observed during the course of the studies. Effects observed in such cases included one or more of: immunotoxicity; neurotoxicity and disruption of thyroid hormones and retinoids.

<table>
<thead>
<tr>
<th>Group</th>
<th>Exposure route</th>
<th>Daily intake</th>
<th>Tissue burden</th>
<th>Effects observed?</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Harbour seal</td>
<td>Atlantic Ocean herring</td>
<td>0.3–0.6</td>
<td>62</td>
<td>n.a.</td>
<td>(De Swart et al., 1994; Ross et al., 1996)</td>
</tr>
<tr>
<td>Harbour seal</td>
<td>Baltic Sea herring</td>
<td>1.2–5.6</td>
<td>209</td>
<td>Yes</td>
<td>(De Swart et al., 1994; Ross et al., 1996)</td>
</tr>
<tr>
<td>PVG rat</td>
<td>Perinatally to lipid extract of Atlantic herring</td>
<td>0.3</td>
<td>?</td>
<td>n.a.</td>
<td>(Ross et al., 1997)</td>
</tr>
<tr>
<td>PVG rat</td>
<td>Perinatally to lipid extract of Baltic herring</td>
<td>2.1</td>
<td>?</td>
<td>Yes</td>
<td>(Ross et al., 1997)</td>
</tr>
<tr>
<td>PVG rat</td>
<td>Perinatally to 2,3,7,8-TCDD-spiked Atlantic herring</td>
<td>134</td>
<td>?</td>
<td>Yes</td>
<td>(Ross et al., 1997)</td>
</tr>
</tbody>
</table>
world" examples to be considered in risk assessment studies of new industrial chemicals. Society at large will ultimately assess the value of these wildlife species and their relevance in risk assessment. Given the limited direct economic returns generally afforded by marine mammals and the difficulties in ascribing a single social value to these animals, using marine mammals for risk assessment purposes will be best justified on the basis of their sentinel role in the oceanic environment and their ability to integrate contaminant information. While laboratory-based toxicological studies can serve to identify the potential risks associated with exposure to particular chemicals, marine mammals offer an opportunity to study the effects of complex mixtures and provide valuable insight into the contamination of our marine and coastal environment.

ACKNOWLEDGMENTS

The author gratefully acknowledges the critical comments of R. F. Addison, R. W. Macdonald and two anonymous referees.

REFERENCES


world” examples to be considered in risk assessment studies of new industrial chemicals. Society at large will ultimately assess the value of these wildlife species and their relevance in risk assessment. Given the limited direct economic returns generally afforded by marine mammals and the difficulties in ascribing a single social value to these animals, using marine mammals for risk assessment purposes will be best justified on the basis of their sentinel role in the oceanic environment and their ability to integrate contaminant information. While laboratory-based toxicological studies can serve to identify the potential risks associated with exposure to particular chemicals, marine mammals offer an opportunity to study the effects of complex mixtures and provide valuable insight into the contamination of our marine and coastal environment.

ACKNOWLEDGMENTS

The author gratefully acknowledges the critical comments of R. F. Addison, R. W. Macdonald and two anonymous referees.

REFERENCES


