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Environmental Contaminants and Marine Mammals

Environmental contaminants have gained increasing attention as potential threats to marine mammals. In this chapter I present an overview of the current state of knowledge on marine mammals and the two classes of environmental contaminants that have received the most attention: organochlorine compounds and toxic elements. A brief introduction to other classes of contaminants and toxins is included. The scope of the chapter does not include marine mammals and oil, which has been treated in depth in other sources (Geraci and St. Aubin 1990, Loughlin 1994). The approach is to provide a general introducrion and overview with examples of case studies. However, appendices with detailed tabulations of specific information are also provided as a key to the primary literature. This will allow the interested reader to pursue these topics in greater depth. General reference works should also be consulted (e.g., Peterle 1991, Hoffman et al. 1994, Klaassen 1996). Although numerous investigations have been conducted in this field, the state of knowledge about effects of many contaminants on marine mammals remaitis very incomplete. In addition, results of studies have occasionally been interpreted to be of significance in ways that are beyond the bounds that can actually be supported by existing data. Nevertheless, prudent interpretations of some of the findings in this field have implications that are profound and call for continued research into the future.

Organochlorine Compounds

One of humankind's major technological advances has been the capability to synthesize and manufacture chemicals on a massive scale for applications in industry, agriculture, and health. Remarkable changes in society have been enabled by these developments, but, like many technological advances, seemingly unpredictable environmental consequences have also occurred. Pollution of the planet by highly persistent organochlorine compounds is a classic example.

The organochlorines are a diverse group of industrial and agricultural compounds synthesized for various properties, including chemical stability. Many of the organochlorines are highly fat soluble (lipophilic) but have low water solubility (hydrophobic), and differentially accumulate in lipids of animals. Millions of tons of these chemicals have been produced and released into the environment, mostly during the latter half of this century (Hoffman et al. 1994). Designed for chemical stability, some of the organochlorines are extremely persistent in the environment and resistant to metabolic degradation, thereby increasing in concentrations

through food webs. Because the ultimate sinks for many of these persistent compounds are the oceans of the world, where organochlorines are rapidly adsorbed to organic matter and taken up by plankton, marine mammals have been an end point in the food web accumulation of these compounds. Numerous studies have documented the presence of organochlorines in marine mammal tissues (Appendices 1 to 4). References cited in the appendices to this chapter indicate that organochlorines have been reported in tissues of at least 23 species of pinnipeds, 44 species of odontocetes, and 11 species of baleen whales, as well as sea otters, sirenians, and polar bears. Analyses have been conducted on samples from more than 7000 individuals (Appendices 1 to 4). Many of these analyses have focused on the organochlorines, although other halogenated compounds such as the polybrominated biphenyls have occasionally also been reported. Many of the organochlorines reported in marine mammal tissues were originally introduced to the environment as pesticides targeting the nervous systems of insects. However, the reader should bear in mind that many insecticides now in use, particularly in developed nations, are not organochlorines but organophosphates, carbamates, and other forms that are not persistent in tissues and do not appreciably accumulate in marine food chains. Thus, the common use of the generic term pesticides for organochlorine insecticide residues is misleading. Similarly, the term pesticide also encompasses the numerous synthetic herbicides (some of which include halogens in their structure), which are designed to affect plant physiological systems and have never been determined to be a serious contaminant issue for marine mammals.

Expression of Results of Organochlorine Residue Analyses

The reporting of concentrations of organochlorines in tissues can be bewildering to those people who are unfamiliar with the field. Most studies report contaminant residues on the basis of mass of chemical per unit mass of tissue. The latter, however, may be expressed on the basis of fresh weight (or "wet weight") of the tissue sample, on the basis of weight of the sample with water removed ("dry weight"), or on the basis of the extractable lipid components ("lipid weight"), which for all practical purposes will contain all of the organochlorine contaminants. The most typical expression of concentrations in the literature are given as parts per million (ppm) wet weight, which on an unit of mass basis may also be expressed as μg/g, or mg/kg. Lower concentrations may be expressed as parts per billion (ppb) or by the units ng/g or μg/kg. It is important to be certain of the units in comparing findings among various studies. Publications that most thoroughly document results give values of concentrations of contaminants, but will further provide percent lipid (which can be quite variable even in blubber) and percent water, thereby permitting the reader to recalculate values for comparison with other findings. Many factors can cause variation in organochlorine concentrations in marine mammals and these are discussed in other sections of this chapter. A few studies attempt to estimate total amounts of organochlorines in bodies and organ systems of marine mammals, expressing amounts in units of mass and total body burdens. Patterns of variation in total body burdens of organochlorines can differ from patterns in concentrations in blubber.

Concentrations of contaminants in tissues are also described using different summary statistics. Often results are expressed as means, ranges, and standard deviations or standard errors. However, in many samples the actual distribution of contaminant concentration data points is nonnormal, and what is often found is a skewed array with many relatively low values and a few very high values. Arithmetic means or parametric statistical tests of hypotheses on these data can be inappropriate and misleading. Instead investigators often transform the data to a logarithmic scale, and compute a geometric mean and confidence intervals to help adjust for the non-normality of distributions. As with units of expression, it is important to be certain of the statistical basis of the summary data provided when interpreting results of various studies. Thorough presentations of results often include tables with original data in addition to summary statistics. This allows readers to make detailed comparisons and to perform their own statistical tests of hypotheses.

Major Compounds

The following section provides an overview of the characteristics of the various organochlorines frequently reported from marine mammals (Appendices 1 to 4). In subsequent sections I explore patterns of variation in organochlorine concentrations in marine mammal tissues, review evidence for impacts of these contaminants on marine mammal health and population dynamics, and summarize recent studies of organochlorine metabolism and biochemical toxicity in marine mammals. Over the years the analytical methodology used by chemistry laboratories to quantify organochlorine residues in tissues has become increasingly sophisticated. Several methods have been used, but modern, thorough studies typically use high-resolution capillary gas chromatography with electron capture detection, combined with confirmation by mass spectrometry, subsequent to use of standardized extraction and clean-up procedures using highest purity grade solvents. High-performance liquid

chromatography is sometimes used in the process of isolating certain individual compounds (for example, see Varanasi et al. 1992, Wells and Echarri 1992). Quality assurance procedures also must be followed and well documented to ensure scientific credibility. These can entail determination and reporting of error in calibrated or "spiked" samples, multiple analyses of the same sample within the same laboratory, interlaboratory comparison studies, and use of standard reference materials. Systematic procedures for collecting tissues to ensure avoidance of spurious contamination must also be followed (see Geraci and Lounsbury 1993). Steps toward developing standardized protocols for analysis of marine mammal tissues have been taken (Lillestolen et al. 1993). However, an internationally standardized set of analytical methods and quality assurance procedures has not yet been developed and formally agreed on for determination of contaminants in marine mammals, and therefore methodology can vary among studies.

DDT and Metabolites

DDT (2,2-*bis*-(*p*-chloropbenyl)-1,1,1-trichloroethane or dichlorodiphenyltrichloroethane) and its metabolites (Fig. 10-1) are the best known and most widely reported organochlorines found in marine mammal tissues and as such, deserve treatment from a historic perspective. They are arguably one of the most notorious groups of compounds to be recognized as environmental contaminants, and indeed provided significant impetus to the tremendous surge in societal awareness of the potential dangers of persistent environmental contaminants. These compounds were at the forefront in sparking growth in both the science of wildlife toxicology and government regulation of contaminants (the latter, however, was not without much debate). DDT and metabolites were first reported in marine mammal tissues in the 1960s, at about the time it became evident from field studies that they were associated with eggshell thinning and declines in populations of some species of birds (Ratcliffe 1967, Hickey and Anderson 1968, Stickel 1973). DDT was also implicated in direct mortality of some species of wildlife. Contamination of the global marine ecosystem by organochlorines was exemplified by the discovery of residues of DDT and metabolites in Antarctic seals, far from significant sources of direct exposure, reported in the journal *Nature* more than three decades ago by investigators at the Patuxent Wildlife Research Center and Johns Hopkins University (Sladen et al. 1966). Much of the controversy surrounding the regulation of DDT, however, was on the possible human health impact, primarily carcinogenic potential, rather than impacts on wildlife or possible impairment of reproduction. The evidence for its carcinogenicity continues to cause debate and, although use of DDT has been banned in some nations (particularly the developed countries of the northern hemisphere), the chemical is still used in many parts of the world. Transport from these areas to some of the more "pristine" reaches of the globe has been well documented (for example, see Simonich and Hites 1995).

Today DDT has notoriety among marine mammalogists chiefly as a contaminant and environmental "villain." However, as pointed out by Metcalf (1973), this chemical ". . . has had an influence on human ecology perhaps unmatched by any other synthetic substance. Through its effectiveness in the conquest of malaria, typhus, and other insect-borne diseases it has played a decisive role in the population explosion.

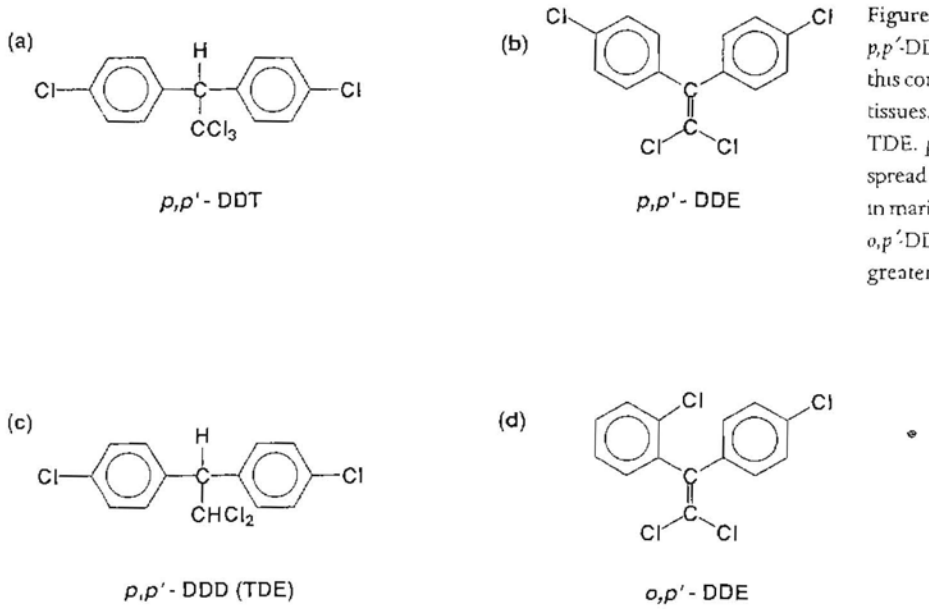

Figure 10-1. Chemical structures of (a) *p,p'*-DDT and the principal metabolites of this compound found in marine mammal tissues, (b) *p,p'*-DDE, and (c) *p,p'*-DDD or TDE. *p,p'*-DDE is usually the most widespread and abundant metabolite found in marine mammal blubber. The isomer *o,p'*-DDE (d) is reported less often but has greater estrogenic activity.

It has also become the classic example of an environmental micropollutant." DDT (and, to a lesser extent, its metabolites) is primarily neurotoxic but can also cause pathological changes to the liver and reproductive system in laboratory mammals.

The synthesis of DDT was first reported in 1874 by the German chemist Othmar Zeidler. However, the insecticidal properties of the chemical were not discovered until it was resynthesized in 1939 by Paul Müller. The magnitude of the technological impact of this discovery on humankind can be gauged by the awarding of a Nobel Prize to Müller in 1948 for "discovery of the strong action of DDT against a wide variety of arthropods" (Metcalf 1973). In India alone, the World Health Organization credited DDT spraying with a reduction in malaria from 100 million cases in 1 year in the 1930s to 150,000 per year by 1966 (Metcalf 1973). DDT was sprayed not only for control of disease vectors, but against crop and garden insects, and over entire landscapes of forested ecosystems in efforts aimed at eradication of timber pests. During World War II, soldiers were dusted with DDT to control body lice (which as vectors of typhus played a larger role than military tactics in turning the tide of many previous wars). The compound was also sprayed to protect troops from malaria and other diseases. As pointed out by Metcalf (1973), one can only speculate how history might have been changed had the insecticidal properties of DDT been recognized at the time of its discovery by Zeidler, rather than after it had rested on the shelf for some 65 years.

Unfortunately, history has a pattern whereby strides forward in technology are often accompanied by large scale setbacks to the natural functioning of ecosystems (Ehrenfeld 1981). Among such unforeseen ecosystem ramifications is the widespread occurrence of DDT and its metabolites in tissues of biota throughout the world, including marine mammals. Extremely high concentrations of these compounds, higher than found in tissues of most terrestrial mammals, have been reported in odontocete cetaceans and pinnipeds in some parts of the world. The presence of metabolites of DDT in marine mammals throughout the globe is fact. The significance of these metabolites for marine mammal populations is subject to difficulty in interpretation, as explored in further sections.

Most analyses of tissues of marine mammals for DDT and other organochlorines have focused on the blubber, which because of its high lipid content is the major storage compartment for these contaminants. The breakdown product DDE (2,2-*bis*-(*p*-chlorophenyl)-1,1-dichloroethylene or dichlorodiphenyldichloroethylene) is the most abundant metabolite found in blubber. DDE concentrations are usually far higher than of DDT or of TDE (2,2-*bis*-(*p*-chlorophenyl)-1,1-dichloroethane or tetrachlorodiphenylethane), also referred to as DDD (dichlorodiphenyldichloroethane) (Fig. 10-1). DDE is much less toxic (in terms of lethality to laboratory animals) than DDT. A small portion of the technical grade mixtures of DDT also contain the *o,p'* isomers (Fig. 10-1), which are not frequently reported in environmental samples. The *o,p'* isomers have also been reported at lower concentrations in some studies of marine mammal tissues. Some studies have also reported additional metabolites of DDT, including methyl sulfone compounds (Appendices 1 to 4). It is common for publications on concentrations of DDT and metabolites in tissues to provide an arithmetic summation of these measurements in each tissue sample. This sum of the individual components is typically referred to as "total DDT" and denoted by notations such as $\sum$DDT, DDTR, or DDTs. Some of the earlier studies (generally before the 1970s) on concentrations of DDT and metabolites in marine mammals were carried out before the recognition of polychlorinated biphenyls (PCBs) as environmental contaminants (and therefore their separation during chemical analysis), making accuracy in quantification of values reported less reliable.

The dynamics of storage and metabolism of DDE and related compounds in marine mammal tissues are complex, and although studied in some detail as described in further sections, knowledge of these dynamics and their possible ramifications to health and population stability of marine mammals remains incomplete. These compounds can be expected to be found at some level in most biota on Earth, and certainly in every living marine mammal. However, great variation exists in concentrations of these and other organochlorines in tissues of marine mammals, making it difficult to desctibe "typical" or background levels of contamination. As a rough reference point, during the 1960s and early 1970s mean concentrations of $\sum$DDT in human adipose tissue in the United States ranged from 5 to 10 ppm, and concentrations in human adipose tissues in other nations ranged from 2 ppm (Australia, Netherlands) to 28 ppm (India) (presumed lipid weight basis; Matsumura 1985; see also Jensen 1983). More current values for $\sum$DDT in human adipose tissue obtained in Mexico during the late 1980s to early 1990s range from 1.0 to 90.0 ppm (lipid basis) in fat (Waliszewski et al. 1996), and recent determinations of average $\sum$DDT in mother's milk in African countries range from 3.0 to 20 ppm (lipid basis) (Ejobi et al. 1996). Extreme cases of $\sum$DDT contamination of marine mammals have resulted in concentrations of 1000 to 2000 ppm or more in blubber. However, typical concentrations range much less than 100 ppm, with many samples at 10 ppm or less, particularly in the baleen whales or other species from the open oceans or high latitudes. Residue concentrations of total DDT in milk of marine mammals have been sel-

domly reported (Appendices 1 to 4), but are typically less than or equivalent to those found in some contemporary African mothers.

The Cyclodienes

Aldrin, dieldrin (Fig. 10-2), and endrin are cyclodienes synthesized by a chemical process referred to as the Diels-Alder reaction (hence the etymology). They are insecticides that are generally much more acutely toxic than DDT; they also were used in large quantities before restrictions in some countries during the 1970s. The cyclodienes are highly neurotoxic, with likely different mechanisms of action than DDT at the cellular and biochemical levels, and can cause reproductive defects in laboratory mammals at high dosage levels. Dieldrin is an insecticide in its own right but is also a metabolite of aldrin, which breaks down in the environment much more quickly than dieldrin. Dieldrin is commonly found in blubber of marine mammals, whereas the less persistent aldrin and more toxic endrin are seldom reported (Appendices 1 to 4). Concentrations of these chemicals in marine mammal tissues are generally lower than those of DDT and its metabolites or the PCBs. However, dieldrin in particular has been found in marine mammals throughout the world. According to Matsumura (1985:58) dieldrin "is one of the most persistent chemicals ever known." Some publications refer to dieldrin by the alternate chemical abbreviation of HEOD.

The technical grade of the cyclodiene insecticide chlordane is a mixture of compounds and isomers, about 60% of which is chlordane. Chlordane occurs as a mixture of *cis*- and *trans*-isomers (Fig. 10-2). Other persistent components of the mixture include heptachlor, nonachlor, and oxychlordane. Heptachlor is also prepared from chlordane as an insecticide in its own right and generally is of higher acute toxicity. The more toxic heptachlor epoxide (Fig. 10-2) is the principal metabolite of heptachlor found in marine mammal tissues; isomers of chlordane, oxychlordane, and nonachlors have also been reported (Appendices 1 to 4). These compounds have been found in marine mammals throughout the global marine ecosystem, and include complex mixtures and metabolites from seemingly pristine reaches such as the Arctic and Antarctic (Norstrom and Muir 1994), but at concentrations that can generally be considered low.

Toxaphene

The literature on organochlorines in marine mammal tissues occasionally reports the presence of toxaphene (Appendices 1 to 4). Technical grade toxaphene released to the environment is not a compound but a mixture of compounds and isomers used as an agricultural pesticide, particularly on

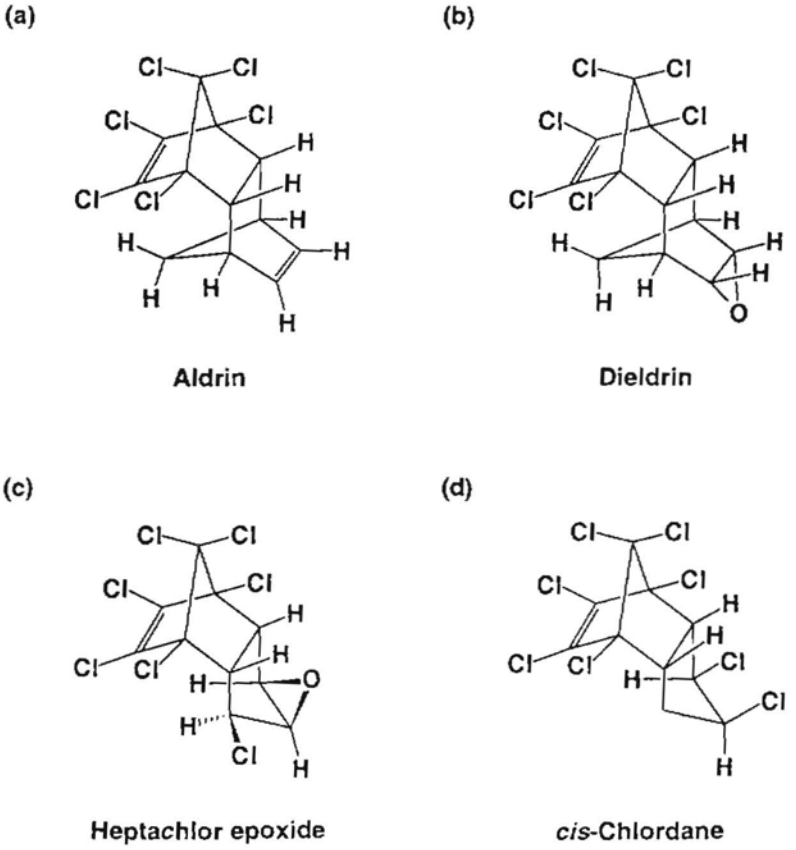

Figure 10-2. Cage diagrams of chemical structures of representative cyclodiene compounds. The cyclodienes are organochlorine insecticides or metabolites.

cotton. It consists of many chlorinated terpenes (also referred to as chlorinated camphenes, polychlorocamphenes [PCCs], and as chlorinated norbornane derivatives). The theoretical number of isomers of toxaphene compounds and metabolites is in the thousands, with estimates based on empirical analytical chemistry numbering in the hundreds. Some congeners can be masked by PCBs during chemical analysis.

Toxaphene was widely used before restrictions imposed in some nations, including a ban on insecticidal use in the United States in 1982. Restrictions were based primarily on suspected carcinogenicity and mutagenicity. Toxaphene is intermediate in acute toxicity between dieldrin and DDT. Individual components vary widely in toxicity. It was the most widely used insecticide in the United States before restrictions and continues to be used in many nations. Like other organochlorines, it can be spread through the environment by atmospheric transport and is found in low concentrations in tissues of marine mammals in remote areas such as the Arctic (Zhu and Norstrom 1993). Cumulative world use during the period 1946 to 1974 was more than 409,000 metric tons (mt). Saleh (1991) provides an excellent literature review on these and other aspects of the chemistry, toxicology, and environmental kinetics of toxaphene.

Polychlorinated Biphenyls

The polychlorinated biphenyls (PCBs, also referred to as chlorobiphenyls or CBs) are a mixture of compounds (Figs. 10-3 and 10-4). They are produced by the chlorination of biphenyls and theoretically can include a mixture of 209 different isomers and congeners, although a smaller number have actually been reported as contaminants in biological samples. They have had a wide variety of industrial applications, including use as dielectric fluids in electrical transformers; in heat transfer systems, capacitors, and plastics; as inks, additives, and lubricants; in carbonless copy paper; and in hydraulics. Publications on PCBs in marine mammals sometimes refer to them by trade names such as the Aroclors (produced in the United States) in which the last two digits refer to the weight percentage chlorine of the mixture, e.g., Aroclor 1254 is 54% chlorine by weight; other trade names of mixtures produced in different countries and sometimes referred to in the marine mammal literature include the Kanechlors (Japan), Phenoclors (France), and Clophens (Germany). These commercial mixtures contained large numbers of individual PCB congeners (e.g., Aroclor 1254 typically contains some 50 to 70 PCB compounds). The total amount of PCBs produced since 1929 has been estimated at 1.5 million mt (de Voogt and Brinkman 1989). Individual PCB congeners may also be synthesized for specialized uses. PCBs were first discovered as environmental contaminants

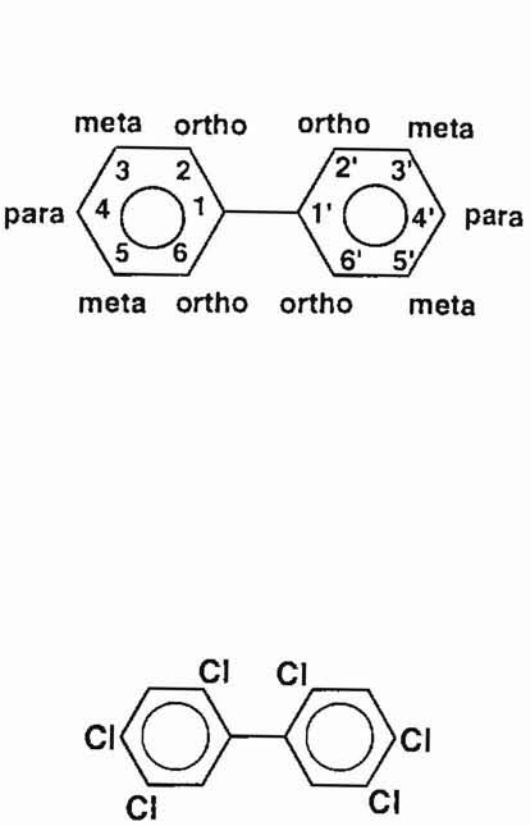

Figure 10-3. (a) Generalized structural formula for polychlorinated biphenyls (PCBs) with numbering of the carbon atoms in rings and potential positions of chlorine and vicinal H atoms. An example (b) is the metabolically resistant PCB 153 (2,2′,4,4′,5,5′-hexachlorobiphenyl).

in the late 1960s and subsequently were determined to be widespread throughout the biota of the globe.

During the 1970s and 1980s the manufacturing of PCBs was terminated in most industrialized nations with the recognition of their widespread distribution as ecological contaminants and the potential for detrimental health effects. This recognition was heightened by mass human exposure due to accidental mixing of PCBs with rice oils ("Yusho disease") in Japan in 1968 and Taiwan in 1979, resulting in a number of related ailments (although subsequent research suggests some of the resulting effects may have been attributable to unusually high concentrations of polychlorinated dibenzodioxins [PCDDs] and polychlorinated dibenzofurans [PCDFs] (Fig. 10-5) in the mixtures as well). Despite the marked cessation in their production and sale, most PCBs are still contained in systems for which they were originally designed (such as transformers and other machinery) and have not yet reached the environment. As these systems leak, degrade, and are disposed of,

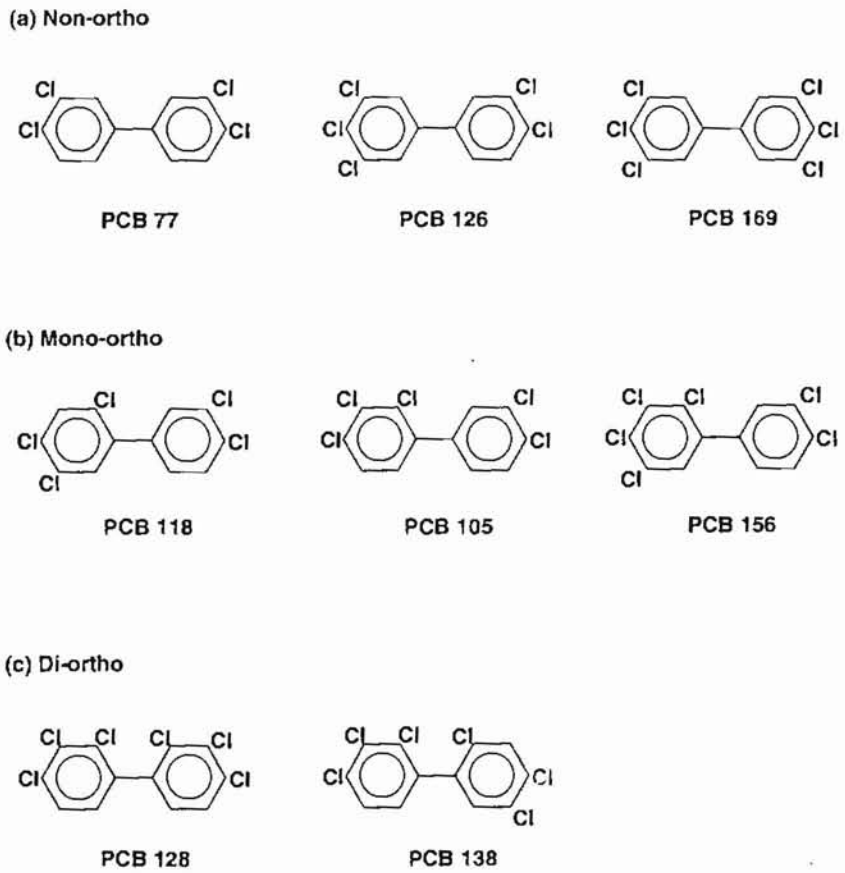

Figure 10-4. Examples of coplanar PCBs with chemical names and IUPAC numbers. Non-ortho: 3,3′,4,4′-tetrachlorobiphenyl (PCB 77); 3,3′,4,4′,5-pentachlorobiphenyl (PCB 126); 3,3′,4,4′,5,5′-hexachlorobiphenyl (PCB 169). Mono-ortho: 2,3′,4,4′,5-pentachlorobiphenyl (PCB 118); 2,3,3′,4,4′-pentachlorobiphenyl (PCB 105); 2,3,3′,4,4′,5-hexachlorobiphenyl (PCB 156). Di-ortho: 2,2′,3,3′,4,4′-hexachlorobiphenyl (PCB 128); 2,2′,3,4,4′,5′-hexachlorobiphenyl (PCB 138).

Figure 10-5. Examples of polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). The dioxin (2,3,7,8-terrachlorodibenzo-*p*-dioxin or TCDD) is a potent enzyme inducer and one of the most toxic compounds known. Although not generally reported at elevated concentrations in marine mammals, it is the basis for calculation of toxic equivalencies. Some PCBs are considered to be approximate isostereomers of TCDD.

the quantities released to the environment will increase. The ultimate repository for these compounds will be the oceans. PCB concentrations in the environment, and in the tissues of marine mammals in particular, are projected to increase for many years to come (Tanabe 1988). Like other organochlorines, PCBs are lipophilic and therefore, are readily sequestered in marine food webs. However, there is wide variance among individual congeners in their toxicity and persistence in various metabolic systems, as detailed elsewhere in this chapter in discussions of PCB metabolism.

The analytical methodology used to quantify the presence of PCBs in marine mammal tissues has matured during the past two decades. Research into the presence of organochlorines in tissues before the 1970s generally did not include determination of PCBs; this caused some inaccura-

cies in the expression of amounts of other compounds. Subsequently, the practice was to compare the amounts of PCBs present in the sample with a standard mixture such as Aroclor 1254 or 1260. However, choice of standard and analytical methodology affects the estimated concentrations, with results from the same individuals showing concentration estimates with three- to fourfold differences using different procedures and Aroclor standards. Modern analytical procedures now can provide concentrations of individual congeners, which is of great importance because of wide differences among congeners in relative toxicity, and total PCBs can be estimated as the sum of congener concentrations. Because of the wide number of individual congeners, a systematic numeric system of nomenclature for individual PCBs has been adopted in addition to standard names based on structural formulae (Figs. 10-3 and 10-4). These are referred to in the literature as IUPAC, Ballschmiter, or PCB numbers (for example, 2,3,3′,4,4′,5-hexachlorobiphenyl (Fig. 10-4b) is simply referred to as PCB 156) and are listed in detail by Ballschmiter and Zell (1980) and Ballschmiter et al. (1989). For point of reference, PCBs generally range from 1 to 10 ppm (lipid basis) in human adipose tissue and 1 to 2 ppm (lipid basis) in human milk, typically quantified in comparison with an Aroclor 1254 or 1260 standard (Ballschmiter et al. 1989). Additional discussion on the patterns of occurrence and potential effects of PCBs in marine mammals are treated elsewhere in this chapter.

Other Organochlorines

PCDFs, PCDDs, polychloroquaterphenyls (PCQs), and polychlorinated napthalenes (PCNs) have been detected in commercial PCB mixtures where they have been formed in low quantities as by-products of the PCB manufacturing process. PCDDs and PCDFs are also formed as impurities in a variety of other industrial and combustion processes (Safe 1986, 1991). The PCDD compound 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD; Fig. 10-5) is among the most toxic compounds known and is the most intensively studied. It was an impurity present in the defoliant Agent Orange used during the Vietnam War. A few reports indicate the presence of these compounds in marine mammal tissues (Appendices 1 to 4), although with few exceptions (Jarman et al. 1996) investigations have not been extensive.

Isomers of hexachlorocyclohexane (HCH), sometimes also referred to as benzene hexachloride (BHC), often appear in low concentrations in blubber of marine mammals (Appendices 1 to 4). It is an insecticide, the pure γ isomer of which is also named lindane. Lindane is neurotoxic, and some isomers of HCH have caused tumors in liver cells of laboratory mammals. Ratios and metabolism of various isomers of HCH were quantified and discussed for 10 odontocete species by Tanabe et al. (1996). Other organochlorine insecticide residues reported infrequently in marine mammals include the structurally related compounds kepone and mirex, and endosulfan (which is also used as an ascaricide). Hexachlorobenzene (HCB) is a fungicide that appears as a widespread environmental contaminant and has been detected in a variety of marine mammals (Appendices 1 to 4). Some related compounds with different numbers of chlorines are sometimes reported as total chlorobenzenes (CBz). The methyl sulfone metabolites of DDE and PCBs, which are toxic themselves, have also been quantified in marine mammal tissues (Appendices 1 to 4; Bergman et al. 1994). One recent study has also revealed the presence of hexachlorinated anthracenes in seals from the Baltic Sea (Koistinen 1990). In addition to the organochlorines, other organohalogens reported in marine mammal tissues include the polybrominated biphenyls (PBBs), manufactured as flame retardants, octachlorostyrene (OCS), and polybrominated diphenyl ethers (Kuehl et al. 1991, Kuehl and Haebler 1995). The substance *tris* (4-chlorophenyl) methanol (TCP) has been found in several species of pinnipeds and cetaceans, and may stem from synthetic dyes or DDT (Jarman et al. 1992, de Boer et al. 1996, Muir et al. 1996a).

Distribution and Kinetics of Organochlorines in Body Compartments of Marine Mammals

In marine mammals, blubber is the major repository for organochlorines, and most of the emphasis on determination of residue concentrations has focused on this tissue (Appendices 1 to 4). Tanabe et al. (1981) demonstrated that in an adult male striped dolphin (*Stenella coeruleoalba*) most of the body weight is contributed by muscle (56%), followed by blubber (17%) and bone (12%). Striped dolphin blubber, however, contained 95% of the total body burden of ∑DDT and PCBs and more than 90% of the body burden of HCHs and HCB. This is because of the higher lipid composition of blubber (about 75% of blubber weight was lipid and 91% of all lipids in the body were found in blubber, whereas muscle tissue is comprised of 1% to 4% lipid). The total amounts of organochlorines found in bone and other organs were negligible, leading these researchers to suggest that combined amounts in blubber and muscle (with its larger total mass) could be used to represent the total amounts of these contaminants in individual animals. Most organs had similar proportions of various organochlorines, except the brain and liver. Lower relative concentrations of DDT were found in the liver (implying active metabolism). The brain had lower concentrations of organochlorines on a lipid weight basis, as well as apparent differences in amounts of lower chlorinated PCBs and isomers of HCH. Lower relative con-

centrations of organochlorines in brains have also been reported for sperm whales (*Physeter macrocephalus*) (Aguilar 1983), harp seals (*Phoca groenlandica*) (Frank et al. 1973), small cetaceans (O'Shea et al. 1980), and a variety of other species (Aguilar 1985; see Appendices 1 to 4 for additional references).

Very few studies have attempted to estimate the absolute body load of organochlorines in marine mammals other than in the striped dolphin. One exception, however, is the detailed study of fin whales (*Balaenoptera physalus*) conducted by Aguilar and Borrell (1994b), who calculated tissue masses for five complete whales by dissection and determined organochlorine concentrations in samples of blubber, muscle, bone, liver, and kidney for 26 individuals, extrapolating results to a total sample of 169 whales. (For extrapolations, they estimated total body loads based on organochlorine concentrations in blubber samples and empirical relationships established in the smaller groups. Anatomical studies of reproductive tracts provided details on sexual maturity, and growth layer counts in ear plugs established age estimates.) Using this information, they were able to estimate quantities of organochlorines transferred to young by females during reproduction by differences in estimated total body loads of males and females of the same age. Body loads averaged 6.5 g for $\sum$DDT and 7.8 g for PCBs in the sample of 26, with estimates as high as 23.5 g $\sum$DDT and 20.1 g PCBs in males. These amounts are one to two orders of magnitude higher than estimates for some small cetaceans, which is attributable to the huge differences in body size and amounts of lipid.

The general framework for understanding the kinetics of organochlorines in marine mammals is similar to that established for other homeotherms, in which the blood is considered a central "compartment" through which various organs maintain a dynamic relationship with contaminant exposure (Aguilar 1985). As noted for striped dolphins, the organochlorine residues found in other organs are generally distributed according to their fat content, with concentrations expressed on a lipid weight basis being highly similar among all organs. The brain tends to have lower concentrations of organochlorines than would be predicted on the basis of its fat content. This is not thought to be attributable to a blood–brain barrier, which is unlikely to be effective for liposoluble organochlorines, but rather to the kinds of lipids present in the brain. The brain has a higher amount of phospholipid in comparison with other organs, and organochlorines tend to have a lower affinity for phospholipids than for other lipids. In general, organs with higher amounts of the less polar triglycerides and nonesterified fatty acids have higher organochlorine concentrations (Aguilar 1985). Characteristics of compounds present will vary among organs as well, with highly polar forms occurring at higher concentrations in phospholipids, and greater proportions of degradation products present in the liver because of its active metabolic function.

Although most of the organochlorines in the bodies of marine mammals can be found in blubber, the dynamics of this relationship are complex. Blubber is not metabolically inert. Many marine mammal species undergo extensive annual and seasonal changes in the amounts of fat stored in blubber. These changes correlate with cycles in breeding and lactation, and migrations off feeding grounds. Residue concentrations in blubber can be diluted with rapid expansion of the lipid component during seasonal fattening periods or growth. In contrast, marine mammals sampled as stranded carcasses may have depleted lipid reserves from disease or starvation, with consequent elevations in organochlorine residue concentrations in blubber. Although in marine mammals the rates at which organochlorines are either passed into the blood with lipid mobilization or are concentrated in the remaining fat are poorly known, it is likely that both processes take place (Aguilar 1985, 1987). It has also been suggested that organochlorine concentrations in blubber of different whale species with similar prey may be inversely related to body size, owing to both overall metabolic rate and the size of the lipid compartment available for dilution of residues (Aguilar 1989, cited in Borrell 1993).

A number of variables affecting organochlorine content of blubber can be controlled during sampling. Blubber fat content can vary by topographic location on the body and by structural stratification within areas. Fatty acids are more highly saturated in inner deposits, and changes to the inner deposits may contribute more greatly to variations in blubber thickness. These differences require consistency in field sampling protocols (see Geraci and Lounsbury 1993). Lipid composition of blubber can be dissimilar to other fat-rich areas such as visceral fat deposits and the melon area of cetaceans. In addition, lipid composition of the blubber itself can vary among species. Time since death may also alter residue concentrations in tissues. Borrell and Aguilar (1990) repeatedly sampled blubber of a stranded striped dolphin carcass for organochlorines over a 55-day period and found changes in concentrations (both declines and increases, depending on the compound), beginning 2 weeks after initial sampling.

Patterns of Organochlorine Concentrations in Marine Mammals

Age, Sex, and Reproductive Status

Age, sex, and reproductive status have significant effects on organochlorine residue concentrations in blubber of marine

Table 10-1. Summary of Selected Investigations on the Relationships among Sex, Age, and Organochlorine Concentrations in Blubber of Marine Mammals

Group and Species	Region	General Findings	References
Pinnipeds			
Callorhinus ursinus	Japan	PCBs and ΣDDT in females increased to 6 years of age, then decreased sharply, increasing again after age 20. No trend observed in HCHs.	Tanabe et al. 1994b
Eumetopias jubatus	Alaska	Concentrations of ΣDDT, PCBs, and chlordanes were higher in males than females. Age-related increases occurred in males; females increased in organochlorines until age 5, then decreased until about age 20–25, when increases coincident with likely reproductive senescence occurred.	Lee et al. 1996
Halichoerus grypus	Nova Scotia	Higher concentrations of PCBs in mature males; concentrations in mature females 30% of males; newborn to 3 weeks no sex difference, concentrations 10% of adult males.	Addison et al. 1988
H. grypus	North Sea (England)	Total organochlorine concentrations higher in mature males than immature males, and higher in males than females. Significant variation in total organochlorine concentrations with both age and blubber thickness.	Donkin et al. 1981
Odobenus rosmarus	Arctic (Canada)	Concentrations of multiple organochlorines higher in males than females.	Muir et al. 1995
O. rosmarus	Arctic (Greenland)	Concentrations of ΣDDT and PCBs increase with age in males, but not females. PCB concentrations decrease with age in females and are significantly higher in immature than mature females. No correlations existed between age and ΣDDT in females.	Born et al. 1981
Phoca groenlandica	North Atlantic (Canada)	ΣDDT, PCBs, and dieldrin increase with age in young females, reaching a plateau at sexual maturity.	Frank et al. 1973
P. groenlandica	Greenland Sea	Concentrations of PCBs, DDE, and HCH in blubber increased with age in males. PCDDs and PCDFs did not.	Oehme et al. 1995b
P. groenlandica	Gulf of St. Lawrence (Canada)	Concentrations of ΣDDT, DDE, and PCBs increased with age, but TDE, DDT, and dieldrin did not.	Addison et al. 1973
P. groenlandica	Gulf of St. Lawrence and Hudson Strait (Canada)	Concentrations of ΣDDT, PCBs, and other organochlorines higher in males than females.	Beck et al. 1994
P. groenlandica	Northwest Atlantic and Arctic oceans (Canada, Greenland)	ΣDDT, PCBs, heptachlor epoxide, dieldrin concentrations generally higher in adult males than in adult females, increase with age in males but not females. Variations in patterns occurred among areas.	Ronald et al. 1984a
P. hispida	Baltic Sea	ΣDDT and PCB residue concentrations significantly higher in males in comparison with reproductive females; concentrations increase with age in males but not in females.	Helle et al. 1976a,b
P. hispida	Arctic (Canada)	Concentrations of DDE, DDT, and PCBs in blubber lipids increased with age in males, but not in females, and were inversely proportional to blubber thickness. Concentrations in males were higher than in females.	Addison and Smith 1974; Addison et al. 1986a
P. hispida	Norway	Higher concentrations of PCBs and DDE in males than females.	Daelemans et al. 1993
P. hispida	Lake Saimaa (Finland)	Concentrations of ΣDDT and PCBs increase with age in males but not females.	Helle et al. 1983
P. hispida	Arctic Ocean (Canada)	PCB congeners, ΣDDT, and chlordane concentrations increased with age in males, but not in females. Concentrations were significantly higher in males. Age and sex differences also occurred among groups in PCB congeners and homologs.	Muir et al. 1988b
P. vitulina	North Sea (U.K.)	No significant variation in concentrations of ΣDDT, PCBs, dieldrin with sex; increase with age for PCBs only.	Hall et al. 1992
P. vitulina	Northern Ireland	Higher concentrations of DDE in males than females; no differences between sexes in other organochlorines.	Mitchell and Kennedy 1992
P. vitulina	North Sea (Germany, Iceland)	Higher concentrations of PCBs in males, but no tests of statistical significance.	Heidmann et al. 1992
Odontocete cetaceans			
Berardius bairdii	Western North Pacific (Japan)	DDE and PCB residue concentrations higher in males than females.	Subramanian et al. 1988a
Cephalorhynchus commersonii	Southern Indian Ocean (Kergulean Islands)	Males with higher concentrations of ΣDDT and PCBs than females. Concentrations decline with age in females, increase in males.	Abarnou et al. 1986
Delphinapterus leucas	St. Lawrence estuary (Canada)	PCBs, ΣDDT concentrations higher in adult males than females, increase with age in both sexes.	Martineau et al. 1987

Table 10-1 continued

Group and Species	Region	General Findings	References
D. leucas	St. Lawrence estuary (Canada)	PCBs, ΣDDT, mirex, others positively correlated with age of adult females but not adult males. Perhaps due to shifts in diets by older males, lack of reproduction and age structure in females.	Muir et al 1996a
D. leucas	Canada (several regions)	PCB concentrations higher in males than females, variable results with other organochlorines Negative correlations between age and concentrations of some organochlorines in females and positive correlations in males in some stocks	Muir et al 1990
Delphinus delphis	Indian Ocean (South Africa)	Increased concentrations of PCBs and ΣDDT with age in males, rapid decline in females at the age of sexual maturity Mature males with significantly higher concentrations than all age and sex classes except nonreproductive females	Cockcroft et al. 1990
Globicephala macrorhynchus	Western North Pacific (Japan)	DDE and PCB residue concentrations increase with age in females to about 10 years, then decrease sharply with reproductive maturity, remain low until reproductive senescence.	Tanabe et al. 1987b
G. melaena	Faroe Islands	Higher ΣDDT and PCB concentrations in mature males than mature females No differences between sexes in immature animals. ΣDDT and PCB concentrations decline significantly with age in mature females, but show no trend with age in mature males.	Borrell 1993, Borrell and Aguilar 1993; Borrell et al 1995
Lagenorhynchus acutus	Faroe Islands	Higher ΣDDT and PCB concentrations in males.	Borrell 1993
L. albirostris	Northwestern Atlantic (Newfoundland)	Concentrations of ΣDDT, PCBs, chlordane, and PCB congeners increase with age in males but not in females.	Muir et al. 1988a
Phocoena phocoena	Faroe Islands	Higher ΣDDT and PCB concentrations in males.	Borrell 1993
P. phocaena	Northwestern Atlantic (Bay of Fundy)	Males with higher concentrations of ΣDDT, PCBs, and dieldrin than females. Concentrations in reproductive females lower than in immature and resting females Concentrations increase with age in males, but decrease in females	Gaskin et al 1982, 1983
P. phocoena	North Sea (Scotland)	Significant increase in ΣDDT, PCBs, chlordanes, and dieldrin with age in males but not females No changes in HCB concentrations.	Wells et al. 1994
P. phocoena	North Sea (Netherlands)	Higher PCB concentrations in blubber of males, increasing with age	van Scheppingen et al. 1996
P. phocoena	Northeastern Atlantic	Concentrations of multiple organochlorines increased with age in blubber of males, except HCB, HCHs.	Kleivane et al. 1995
P. phocoena	Great Britain	Higher organochlorine concentrations with age.	Kuiken et al. 1994
Phocoenoides dalli	Bering Sea and Northwestern Pacific	Higher concentrations of PCBs and DDE in males than females; concentrations in females decrease with increasing age after 2 years until age 6 or 7 Relative concentrations of certain PCB congeners shift with age in females but not males	Subramanian et al. 1988b
Physeter macrocephalus	North Atlantic (Spain)	Males with lower concentrations of PCBs and ΣDDT than females, perhaps due to differences in feeding habits and habitat	Aguilar 1983
P. macrocephalus	North Sea	Positive correlations between body length and concentrations of HCB, dieldrin, TDE, *o,p'*-DDT, chlordanes, several PCB congeners	Law et al 1996
Stenella coeruleoalba	Mediterranean Sea	DDE and PCB concentrations higher in males than females.	Aguilar and Borrell 1994a
Tursiops truncatus	Indian Ocean (South Africa)	Mature males with significantly higher concentrations of PCB and ΣDDT than females or immatures, concentrations lowest in reproductively active females Concentrations increase with age in males.	Cockcroft et al. 1989
T. truncatus	Western North Atlantic (U.S.A.)	DDE, PCB, and *t*-nonachlor concentrations higher in blubber and liver of adult males than adult females.	Geraci 1989
T. truncatus	Atlantic Coast (U.S.A.)	Higher organochlorine concentrations in males	Kuehl et al. 1991
T. truncatus	Gulf of Mexico (U.S.A.)	Higher PCB and ΣDDT concentrations in males, other organochlorines variable ΣDDT increased with age in males, but not in females.	Salata et al. 1995
T. truncatus	Gulf of Mexico (U.S.A.)	Higher organochlorines in adult males than females, immatures	Knehl and Haebler 1995
Mysticete cetaceans			
Balaenoptera acutorostrata	Antarctic	DDE and PCB concentrations increase with age in males but not females, and are generally higher in mature males than in mature females	Tanabe et al. 1986

Continued on next page

Table 10-1 continued

Group and Species	Region	General Findings	References
B. borealis	Eastern North Atlantic	Higher organochlorine concentrations in males.	Borrell 1993
B. physalus	Eastern North Atlantic	No differences in concentrations of PCBs or ΣDDT in immature males and females. Concentrations increased with age and body size in males but decreased in females, tending to reach a plateau in both sexes. Patterns of total body loads were also similar. Relative abundance of degraded compounds increased with age in males, decreased in females. Amounts transferred with reproduction estimated.	Aguilar and Borrell 1988, 1994b
B. physalus	Eastern North Atlantic	Higher organochlorine concentrations in males.	Borrell 1993
Eschrichtius robustus	Pacific Coast North America	Higher concentrations of mirex, DDE, and ΣDDT in males.	Varanasi et al. 1994

DDE = 2,2,-*bis*-(*p*-chlorophenyl)-1,1 dichloroethylene; DDT = 2,2,-*bis*-(*p*-chlorophenyl)-1,1-trichloroethane, ΣDDT = arithmetic summation of concentrations of isomers and metabolites of DDT. HCB = hexachlorobenzene. HCH = hexachlorocyclohexane; PCBs = polychlorinated biphenyls; PCDDs = polychlorinated dibenzo-*p*-dioxins; PCDFs = polychlorinated dibenzofurans, TDE = 2,2,-*bis*-(*p*-chlorophenyl)-1,1-dichloroethane.

mammals (Table 10-1). The general pattern observed extends to most species examined thus far and is based primarily on studies of DDT and metabolites and PCBs. Immature males and females often show no differences in organochlorine concentrations in blubber. Organochlorine concentrations then increase with age in males but decrease with age in mature females. Mature animals often show significant differences between sexes (males with higher concentrations) and in ratios among compounds. These differences are attributable to transfer of organochlorines from females to young during gestation and lactation, and the lack of these avenues for excretion in males. Organochlorines are accumulated by the fetus transplacentally, but reproductive transfer in the lipid-rich milk is especially pronounced. Addison and Stobo (1993) reported that 98% of the organochlorine burden of gray seal pups at weaning is obtained through maternal milk; 30% of the total body burden of ΣDDT and 15% of PCBs in adult females are transferred to a single lactating young (Addison and Brodie 1977). Calculations indicate that a primiparous female Steller sea lion transfers about 80% of its PCBs and ΣDDT to its first-born pup through lactation (Lee et al. 1996). Among cetaceans, it has been estimated that more than 90% of the PCBs of adult female striped dolphins is transferred to the calf through lactation (Tanabe 1988); about 80% of the PCBs and ΣDDT of a female bottlenose dolphin is transferred to the first-born calf (Cockcroft et al. 1989); and young adult pilot whales transfer about 68% of the ΣDDT and 100% of the PCBs through lactation to their calves (Borrell et al. 1995). Therefore, these contaminants are physically passed from generation to generation. The proportion of the body burden of organochlorines transferred to young by females may be less in mysticetes than in odontocetes (Tanabe et al. 1986; Aguilar and Borrell 1988, 1994b). Female fin whales were estimated to transfer about 26% of the total body load of ΣDDT and 14% of PCBs to the first-born calf, with amounts transferred decreasing substantially with subsequent reproduction (Aguilar and Borrell 1994b). These percentages for fin whales are lower than some of the estimates of reproductive transfer in other marine mammals (Table 10-2), but the lower proportions may be attributable to shorter lactation periods in fin whales. Total amounts transferred to young "are not expected to have significant toxicological effects on the population" (Aguilar and Borrell 1994b), a conclusion that should not necessarily be extended to other species because fin whales are among the least contaminated cetaceans.

Differences between sexes in proportions and ratios of DDT metabolites and PCB congeners in North Atlantic fin whales and sei whales (*Balaenoptera borealis*) may be attributable to corresponding modifications in enzymatic activity of the liver produced by differences in organochlorine burdens within sexes, as well as by reproductive transfer of certain compounds by females to offspring (Aguilar and Borrell 1988, Borrell 1993). The repeated findings of differences in organochlorine residue concentrations among age and sex categories (Table 10-1) point out both the need to account for these factors in reporting research results and the difficulties inherent in interpretation of data based on small sample sizes. The only notable exception to this pattern occurs in belugas (*Delphinapterus leucas*) from the St. Lawrence River estuary (Canada), in which correlations with age and certain organochlorines exist in females but not males (Muir et al. 1996a). This may be attributable to an unusual age distribution of the sample population, lack of reproduction in older

Table 10-2. Studies Pertinent to Reproductive Transfer of Organochlorine Compounds between Females and Offspring in Marine Mammals

Group and Species	General Findings	References
Pinnipeds		
Arctocephalus gazella *Callorhinus ursinus* *Mirounga angustirostris* *Neophoca cinerea* *Zalophus californianus*	Determined organochlorine concentrations in milk, including specific PCB congeners. DDE and PCBs found in milk of pinnipeds in all areas, including Antarctica, but one to two orders of magnitude higher in northern hemisphere samples	Bacon et al. 1992
Callorhinus ursinus	Determined HCH concentrations and ratios in milk, and in tissues of neonates	Mossner et al. 1992
Eumetopias jubatus	Estimated lactational transfer based on changes in organochlorine concentrations in blubber of females at age of reproductive maturity in contrast to males. Calculations suggest that about 80% of PCBs and ΣDDT are transferred to the first-born pup.	Lee et al. 1996
Halichoerus grypus	Determined concentrations of DDE, DDT, ΣDDT, PCBs, and PCB congeners in maternal milk, blubber, blood and pup blubber and blood. Demonstrated selectivity in transfer of some organochlorines from blood to milk.	Addison and Brodie 1987
H. grypus	Determined organochlorine concentrations (ΣDDT, PCBs, and trans-nonachlor, HCB, α-HCH) in blubber of neonates, and changes in concentrations to weaning and 1 year of age. Total burdens increased sharply from birth to weaning, then did not change, except for α-HCH, which declined significantly.	Addison and Stobo 1993
H. grypus	Determined concentrations of ΣDDT, PCBs, and dieldrin in mothers and fetuses. Estimated that the near-term fetus contained less than 1% of the organochlorine burden of the mother, obtained through transplacental transfer.	Donkin et al. 1981
H. grypus	Determined concentration of ΣDDT and PCBs in maternal blubber and milk and pup blubber. Estimated that about 30% of ΣDDT and 15% of PCBs in females are transferred to pup through annual lactation.	Addison and Brodie 1977
Phoca hispida	Concentrations of ΣDDT and PCBs present in blubber of neonates, increased rapidly during suckling period.	Helle et al. 1983
Odontocete cetaceans		
Delphinapterus leucas	DDE, ΣDDT, PCBs, and PCB congeners reported in milk sample from one female.	Massé et al. 1986
Delphinus delphis	Marked decline in PCB and ΣDDT concentrations in blubber of females after age of sexual maturity; calculations of amounts transferred to first-born calves.	Cockcroft et al. 1990
Globicephala melaena	ΣDDT transfers to the fetus more readily than PCBs, but transfer rates are comparable in lactation. Reproductive transfer from females declines with age, with young females transferring three times that of older females by lactation and six times by gestation.	Borrell et al. 1995
Phocoena phocoena	Higher dieldrin and ΣDDT concentrations in immature and resting female blubber than in reproductive females	Gaskin et al. 1971
P. phocoena	Determined presence of dieldrin, chlordane compounds, HCB, PCBs and PCB congeners in fetal blubber, demonstrating transplacental transfer	Wells et al. 1994
P. phocoena	Determined presence of DDE, DDT, TDE, o,p'-TDE, PCBs, dieldrin, HCB, HCHs in tissues of a fetus, demonstrating transplacental transfer.	Duinker and Hillebrand 1979
Phocoenoides dalli	Decline in PCB and DDE concentrations in female blubber at sexual maturity. Greater transfer of lower chlorinated biphenyls in lactation than gestation.	Subramanian et al. 1988b
Physeter macrocephalus	PCB and ΣDDT concentrations reported in milk samples.	Aguilar 1983
Stenella coeruleoalba	Reported concentrations of ΣDDT, PCBs, and HCHs in milk	Kawai et al. 1988
S. coeruleoalba	Determined organochlorine burdens in a pregnant female and near-term fetus, and estimated transplacental transfer rates from 4.0% to 9.7% for PCBs, ΣDDT, HCHs, and HCB	Tanabe et al. 1982
Tursiops truncatus	Significant decline in PCB and ΣDDT concentrations in blubber of females after age of sexual maturity. Calculations that 80% of body load of female is passed to first-born calf	Cockcroft et al. 1989
T. truncatus	Determined concentrations of DDE, PCBs, and other organochlorines in milk of captive females. Highest concentrations in 34-year old first lactation, lowest in youngest female	Ridgway and Reddy 1995
Mysticete cetaceans		
Balaenoptera physalus	Estimated total body loads of ΣDDT and PCBs in males and females, and determined age-related trends in each sex. Differences at similar ages were considered due to reproductive transfer. About 26% of total body load of ΣDDT and 14% of PCBs transferred to first calf, with decreasing amounts transferred in subsequent reproduction	Aguilar and Borrell 1994b
Other		
Ursus maritimus	Determined changes in concentrations of ΣDDT, PCBs, chlordane, HCHs, and CBz in adipose tissue and milk of females and cubs, and mean daily ingestion rates through lactation.	Polischuk et al. 1995

See Table 10-1 for summaries of additional studies on changes in organochlorine concentrations in females at reproductive maturity

CBz = chlorobenzenes, DDE = 2,2-bis-(p-chlorophenyl) 1,1-dichloroethylene, DDT = 2,2,-bis-(p-chlorophenyl)-1,1-trichloroethane, ΣDDT = arithmetic summation of concentrations of isomers and metabolites of DDT, HCB = hexachlorobenzene; HCH = hexachlorocyclohexane; PCBs = polychlorinated biphenyls, TDE = 2,2,-bis-(p-chlorophenyl)-1,1-dichloroethane

females, and age-related shifts in food habits (Muir et al. 1996a).

Regional, Ecological, and Temporal Variation

In addition to variation attributable to age and sex, major differences in observed concentrations of organochlorine residues in marine mammals occur according to geographic location and feeding ecology. Marine mammals in areas with generally high organochlorine inputs, such as near-shore waters close to coastal industrial and agricultural centers, tend to have higher concentrations of organochlorines in tissues. Similarly, species that tend to feed higher in marine food webs also tend to have increased concentrations in tissues. Baleen whales tend to have much lower organochlorine residue concentrations in blubber than other marine mammals, attributable to their generally more pelagic habits (resulting in reduced exposure to near-shore contamination) and low position in the food chain (O'Shea and Brownell 1994). Members of inshore populations of piscivorous species tend to have greatest concentrations of organochlorines in tissues, particularly where subject to industrial contamination. Extremely high concentrations of ΣDDT and PCBs have been reported in odontocete cetaceans from the eastern North Pacific off southern California, the mid-Atlantic coast of the United States, the Mediterranean Sea, and the St. Lawrence River estuary. Very high contamination of pinnipeds has been reported from the Baltic Sea and coastal southern California. Odontocetes and pinnipeds from such areas can have concentrations of PCBs and ΣDDT that are two to three orders of magnitude higher than in those from the open ocean or coastal reaches of more pristine latitudes. Greater numbers of kinds of organochlorines are also usually detected in these contaminated populations. However, even in remote reaches of the Arctic, a wide array of organochlorines has been demonstrated in marine mammal tissues (Muir et al. 1990, 1992a; Norstrom and Muir 1994). Marine mammals in the northern oceans tend to be more contaminated with organochlorines than those in southern oceans, but this trend may shift as usage of some organochlorines declines in the north and increases in developing nations of the southern tropics (O'Shea and Brownell 1994, Tanabe et al. 1994a, Oehme et al. 1995a). Southern oceans currently have less contamination than northern oceans because of less industrialization and less intensive agriculture, but contamination with DDT has already increased; in addition, much of the organochlorine deposition in tropical countries is removed to the atmosphere by high temperatures and heavy rainfall, with subsequent global redistribution (Hidaka et al. 1983; Tanabe et al. 1983, 1984b; Kawano et al. 1986, 1988; Tanabe 1988). However, little attention has focused on residue surveys in southern hemisphere marine mammals. More than 90% of the individuals sampled in the literature summarized in Appendices 1 to 4 have been in the northern hemisphere (more than 75% of these from European and Canadian waters).

Organochlorine contamination of marine mammals on a broad regional basis has been intensively studied in the north polar regions (particularly the Canadian Arctic and subarctic; see Appendix), where marine food chains include plankton, fish, seals, polar bears (*Ursus maritimus*), and humans. Excellent reviews on this topic have been provided by Muir et al. (1992a) and Norstrom and Muir (1994). Higher levels of more volatile organochlorines (such as HCHs and components of chlordane and toxaphene) relative to PCBs and ΣDDT are found in marine mammals of high latitudes, and these more volatile organochlorines are relatively more uniformly distributed across these regions. This is attributable to atmospheric transport from lower, warmer latitudes and subsequent condensation in cool regions—a process referred to as the global distillation effect (Simonich and Hites 1995). Whereas compounds such as the PCBs and the DDT group are the predominant organochlorine residues in tissues of marine mammals in temperate and tropical zones, concentrations of HCHs and components of chlordane and toxaphene are found at nearly equivalent concentrations in marine mammals of the polar regions. Although relative proportions change with increasing latitude, actual concentrations are low. PCBs in Arctic ringed seals (*Phoca hispida*) occur in concentrations some 10 to 50 times lower than in Scandinavian and Baltic ringed seals, and PCBs and ΣDDT concentrations are 25 to 30 times lower in Arctic belugas than in belugas from the St. Lawrence River (this difference is not apparent in the more volatile HCH and chlordane components in beluga tissues) (Muir et al. 1992a). Concentrations of organochlorines in marine mammals of the Arctic are, as a rule, higher than in those from the Antarctic. PCDDs are found at greater concentrations in marine mammals of the high Arctic than in the subarctic, possibly because of deposition by the particulate-laden "Arctic haze" transported over the pole from sources in Europe and Asia, "sometimes as thick as smog in major cities" (Norstrom et al. 1990). PCDDs and PCDFs do not biomagnify in seals or polar bears (Norstrom et al. 1990, de Wit et al. 1992). Polar bears have a capacity to metabolize certain organochlorines that is unique among marine mammals, and perhaps among all animals (Norstrom et al. 1988, Norstrom and Muir 1994). Biomagnification of ΣDDT from seal prey does not occur, and chlordane components are also much more readily metabolized by polar bears.

Variation in population structure and dynamics, particularly age at first reproduction, can also result in seeming differences in contamination among populations, species, and

regions because of varying patterns of organochlorine accumulation with age and sex as noted previously (also see Wells et al. 1994). In addition, Aguilar et al. (1993) noted that after adjustment for age, females from different pods of long-finned pilot whales (*Globicephala melaena*) from the fishery of the Faroe Islands could be distinguished by aspects of their organochlorine residue concentrations, particularly by ratios of *p, p'*-DDE/$\sum$DDT. These researchers suggested that observed differences in organochlorine profiles likely corresponded to ecological segregation in use of food resources. Contaminant concentrations and ratios have also been used as possible indicators of populations that may segregate geographically (Aguilar 1987, Kleivane et al. 1995). Populations of walrus (*Odobenus rosmarus*) in the Canadian Arctic differ in concentrations and characteristics of organochlorines in blubber, and these differences suggest significant seal eating by walrus in some areas (Muir et al. 1995).

A few studies have examined changes in contamination of marine mammals with organochlorines over time. For example, Aguilar (1984) investigated changes in the ratio of DDE to $\sum$DDT in several species of marine mammals from the North Atlantic over the period 1964 to 1981. These ratios showed a significant increase with time, indicating continual transformation of DDT to DDE in marine systems and also indicating that such ratios may be used to assess the recency of chronology of DDT inputs to marine ecosystems. Tanabe et al. (1994b) examined trends in organochlorine contaminants in northern fur seals (*Callorhinus ursinus*) from Japan from 1971 to 1988. $\sum$DDT increased until the mid-1970s, then declined. The proportion of $\sum$DDT composed of DDT dropped off sharply after the early 1970s. PCBs also increased until the mid-1970s, then dropped off to a constant level during the 1980s. HCHs showed a slight decline. In the Baltic Sea, $\sum$DDT and PCBs decreased considerably from the 1969 to 1973 period to the 1980s in ringed seals, whereas $\sum$DDT (but not PCBs) declined similarly in gray seals (*Halichoerus grypus*). The differences in trends have been attributed to differences in feeding ecology between the two species (Blomkvist et al. 1992). In male harp seals from the St. Lawrence River estuary, concentrations of PCBs decreased, but $\sum$DDT did not decline with time (1982–1989); however, the percentage of $\sum$DDT composed of DDE increased, indicating continued decreases in input of the more readily metabolized DDT (Beck et al. 1994). Seasonal changes were also apparent, with winter and summer areas differing in exposure through the food. However, interpretations of temporal differences must be approached with caution because of potential differences in sample attributes and in analytical procedures. Prevalence of chlordane and toxaphene components in Hudson Bay beluga whales increased from the 1960s to the 1980s, consistent with the hypothesis of increased atmospheric deposition from the lower latitudes (Muir et al. 1990). St. Lawrence River belugas showed declines in $\sum$DDT, PCBs, and some other organochlorines in males (but not females) sampled in 1993 to 1994 in comparison with the early and mid-1980s (Muir et al. 1996b).

Impacts of Organochlorines on Marine Mammal Populations

Numerous studies have suggested that exposure to organochlorines could have impacts on marine mammal populations. Such impacts would most likely be manifested through mortality or reproductive impairment. To date only a few studies have been carried out that firmly support this possibility, but findings have confounding factors that complicate establishment of possible cause-and-effect relationships or prevent pinpointing specific compounds. However, mounting evidence suggests that organochlorines may be detrimental to marine mammal populations. Three potential ways in which organochlorine effects may manifest themselves are direct mortality, reproductive impairment, and increased susceptibility to disease. Studies that relate to these possibilities and the general link to population declines are examined below.

Direct Mortality

There is no evidence for direct mortality of marine mammals caused by organochlorine compounds. This topic was reviewed for the baleen whales by O'Shea and Brownell (1994), who noted that the brain is the only tissue in mammals and birds in which concentrations of organochlorines can be considered diagnostic of cause of death. Organochlorine concentrations reported in brains of baleen whales were far lower than diagnostic levels in other mammals. Concentrations reported in brains of small cetaceans, pinnipeds, or other marine mammals noted in studies summarized in Appendices 1 to 4 also do not approach those consistent with lethality in other species. However, such levels have not been accurately established specifically for marine mammals, brain tissue is not routinely obtained from marine mammals for organochlorine analysis, and sample sizes worldwide are generally small.

Reproductive Impairment

Organochlorines (largely PCBs) have been *experimentally* verified to be responsible for impaired reproduction only in the harbor seal (*Phoca vitulina*) (Reijnders 1986), although it has been suggested that even in the relatively controlled experimental studies of harbor seals, confounding effects may have occurred (Addison 1989). The harbor seal feeding experiment was conducted to follow up field surveys that re-

vealed high concentrations of organochlorines in a declining population in the Wadden Sea (Reijnders 1980). Control and experimental groups of 12 female harbor seals each were fed diets low in organochlorines (mackerel from the eastern North Atlantic) or high in organochlorines (other fish species from the Dutch Wadden Sea), primarily PCBs and DDE, over a 2-year period. Blood samples were periodically monitored for progesterone and estradiol-17β, and breeding males were introduced to both groups. Reproductive success was significantly lower in females fed fish from the Wadden Sea, and failure was thought to occur at the implantation stage of pregnancy.

Association of elevated organochlorine concentrations with impaired reproduction (but not direct experimental evidence) has been noted in three other pinniped species from two other contaminated coastal areas: ringed and gray seals from the Baltic Sea (Helle et al. 1976a, 1976b) and California sea lions (*Zalophus californianus*) (DeLong et al. 1973). However, interpretation of these data is not simple. Female marine mammals with impaired reproduction for reasons other than contaminant exposure will also have higher organochlorine concentrations in tissues because they are not able to excrete these chemicals through lactation. Hence, in contrast to experimental feeding studies (Reijnders 1986), higher organochlorine concentrations found in field surveys of tissues of females with impaired versus nonimpaired reproduction does not constitute unequivocal evidence of cause-and-effect relationships. This was apparent in investigations of stillbirths and premature pupping in California sea lions. Initial studies associated this phenomenon with high organochlorine residues (DeLong et al. 1973), but later investigations showed that disease agents (*Leptospirosis*) and other factors may have been at least partly responsible (Smith et al. 1974, Gilmartin et al. 1976, Martin et al. 1976). Furthermore, although contaminated with organochlorines, California sea lion populations have not declined but have generally increased in recent decades (Le Boeuf and Bonnell 1980, Lowry et al. 1992, O'Shea and Brownell 1998). In the case of seals from the Baltic, Helle et al. (1976a,b) found higher concentrations of organochlorines in female ringed seals with uterine occlusions or stenosis, but Blomkvist et al. (1992) later reported no relation between organochlorine concentrations and uterine pathology in female gray seals from the Baltic. Reijnders (1984) and Addison (1989) provide further review of the evidence for effects of contaminants on pinniped reproduction. Without additional experimental studies such as those with harbor seals, links between organochlorine exposure and reproductive anomalies are unlikely to be entirely conclusive.

Evidence for impaired reproduction in cetaceans attributable to organochlorine exposure is very limited. Martineau et al. (1987) suggested that elevated PCBs in belugas in the St. Lawrence River affected their reproduction. Béland et al. (1991) provided additional data on a small sample of stranded adult female belugas from the St. Lawrence that included observations of reproductive pathology, but the nonspecific nature of the lesions and the representativeness of the sample are not clear enough to draw firm conclusions of cause-and-effect relationships. Addison (1989) pointed out that residue concentrations may not be markedly higher in St. Lawrence River belugas than in other populations when other factors are considered. For example, body condition may have influenced results, and other forms of habitat deterioration may also have affected this population (Addison 1989). Indirect evidence for potential impacts of organochlorines on cetacean reproduction is provided by biochemical lesions. In Dall's porpoises (*Phocoenoides dalli*), Subramanian et al. (1987) obtained a weak correlation between testosterone concentrations in blood and DDE concentrations in blubber of 12 males collected in May to June 1984 in the northwestern North Pacific. No significant correlations existed among testosterone levels and other organochlorines. This finding is of interest but should be followed with expanded studies that partition variation among other possible confounding factors such as nutritional status and age.

No evidence exists for organochlorine impacts on reproduction in sirenians, where contamination is low because of their position in the food chain (O'Shea et al. 1984). There are no published studies demonstrating impacts of organochlorines on reproduction in sea otters (*Enhydra lutris*), but this is a topic of interest for future study because of the high susceptibility of some other mustelids to reproductive impairment by PCBs (for review, see O'Shea and Brownell 1994). Although they are at the top of the Arctic food chain, it is "unlikely that organochlorines are currently having a significant effect on the polar bear reproduction," due both to exposure levels and the remarkable detoxification capabilities of this species (Norstrom et al. 1988).

Susceptibility to Disease

Bergman and Olsson (1985) examined 19 gray 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, nail deformations, and other lesions. The nature of the pathology suggested the existence of a disease complex involving organochlorine interference with the endocrine system, resulting in hyperadrenocorticism. PCBs were especially suspect, in part because effects on adrenal function have been demonstrated in laboratory studies of other mammals (Fuller and Hobson 1986). In addition, evidence that Aroclor 1254 alters the synthesis of steroids after in vitro exposure of

adrenals from gray 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 gray 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 harbor seals (Mortensen et al. 1992), and both species have been found to have chromosomal aberrations possibly related to contaminant exposure (Hongell 1996). 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 (*Phocoena phocoena*) stranded singly in various areas of Great Britain. They used quantitative methods of histopathology and chemistry with detailed statistical analyses to investigate this hypothesized relationship. Adrenal hyperplasia was found, but not in association with concentrations of seven organochlorine pesticides and metabolites or 25 PCB congeners. It 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 concentrations in blubber of harbor 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). Statistical analyses were adjusted for age class, sex, and location. Higher concentrations of organochlorines were observed in the seals that succumbed to the epizootic. However, as pointed out by Hall et al. (1992), seasonal differences in blubber thickness could also have contributed to the lower concentrations of organochlorines in survivors, and age-related effects may have been obscured. These investigators decided that "data are not sufficient to conclude that there was a direct link between mortality from PDV infection and OC [organochlorine] contamination."

Kuiken et al. (1994) used another approach to test the hypothesis that exposure to organochlorines causes immunosuppression in marine mammals. They examined 94 harbor porpoise carcasses found stranded in Great Britain from 1989 to 1992 and analyzed the blubber for a variety of organochlorines, including 25 individual PCB congeners. Each case was classified as having died from physical trauma (principally accidental deaths in fisheries) or from infectious or parasitic disease. (Death attributable to trauma was considered to be likely independent of organochlorine concentrations, whereas marine mammals found dead due to disease could have relatively higher organochlorine concentrations if the latter produced immunosuppression that rendered them more susceptible to disease.) Diseased porpoises had higher PCB concentrations in blubber than those found dead from trauma. However, body condition was also poorer in diseased porpoises, and the region of origin had a major effect on PCB concentrations in blubber. When the analyses were adjusted for region by limiting the data set to 69 individuals from a more well-defined area, there were no significant differences in concentrations of organochlorines between the diseased and physical trauma groups. Similarly, Blomkvist et al. (1992) found no significant differences in PCB and DDT concentrations in juvenile harbor seals from Sweden collected during and before a PDV outbreak in 1988. Schumacher et al. (1993) examined thyroid glands of harbor seals that died during a PDV epizootic as well as harbor seals and harbor porpoises that did not. They distinguished histological changes consistent with impacts of PCBs on laboratory animals but could not demonstrate conclusively that PCBs were responsible for the conditions observed in the seals. Jenssen et al. (1994) did not find a correlation between PCBs and thyroid hormone levels in blood of gray seal pups.

Kendall et al. (1992) examined the relationships among concentrations of organochlorines in blubber and plasma thymulin concentrations of harbor seals and gray 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, influences development of T cells, and can be stimulated by adrenocorticotrophic hormone. The thymus is sensitive to some contaminants, such as dioxin.) Thymulin levels were negatively correlated with titers to morbillivirus in gray seals, but this relationship was not affected by organochlorine concentrations. Similarly, no relationships were detected between thymulin levels and organochlorine concentrations in blubber of harbor seals. Kendall et al. (1992) suggested the immunosuppressive effect of the morbillivirus itself may have obscured relationships between organochlorine concentrations and thymulin levels, and that additional study should further examine these relationships based on organochlorine concentrations in blood. Significant relationships between thymulin levels and time since exposure

combined with levels of PCB 153 and PCB 180 in multiple regression analysis suggested such an effect, although these investigators stated that "these results should not be interpreted as implying that seals with high OC levels were therefore more vulnerable to mortality from PDV." This caution is reinforced by experimental dosing studies. Harder et al. (1992) exposed six harbor seals to dietary PCBs, followed by dosing with cell-cultured phocine distemper virus. Four control seals that had minimal PCB exposure were also dosed with PDV. All seals developed severe clinical signs of PDV, and both PCB exposed and unexposed groups suffered mortality and showed no differences in antibody production. Therefore, PCB dosing in this experiment had no influence on suscepubility to the morbillivirus, although concentrations of PCBs reached in the exposed animals were at levels exceeded by those measured in some field samples.

In contrast, Ross et al. (1995) fed two groups of 11 young harbor seals relatively uncontaminated Atlantic herring or herring from the Baltic Sea for 2 years. The latter group had higher concentrations of PCBs, dioxins, and furans. The seals fed Baltic Sea herring had lower in vivo immunological responses to ovalbumin injection. The researchers suggested that these findings support the contention that organochlotines played a role in the European PDV outbreaks through immunosuppression, but noted that "it is difficult to extrapolate from the immunological responses using ovalbumin as an antigen to a seal's ability to mount a specific immune response against a pathogen in the natural environment."

Considerable analytical efforts have gone into investigations of the potential connection between organochlorine contamination and the striped dolphin morbillivirus epizootic in the Mediterranean Sea during the early 1990s. Kannan et al. (1993c) and Aguilar and Borrell (1994a) observed concentrations of PCBs (particularly the coplanar PCB congeners) in blubber of some of these individuals that were much higher than reported in other marine mammals elsewhere in the world (reaching as much as 1000 ppm on a lipid weight basis). They speculate that PCBs and DDT may have played a role in immunosuppression and susceptibility to morbillivirus. Blubber from bow-riding striped dolphins sampled by biopsy dart (for a review of this technique, see Aguilar and Borrell 1994c) in the same region in years before and after the epizootic had PCB concentrations that were significantly lower than in blubber of those found dead during the epizootic. PCB concentrations in liver were higher than in blubber in those found dead during the epizootic, suggesting mobilization of lipids and release of PCBs to the bloodstream. Aguilar and Borrell (1994a) noted three possible hypotheses to explain the potential relationship between increased PCB concenttations and susceptibility to the morbillivirus epizootic: (1) PCBs could have caused immune system depression, perhaps through effects on the thymus. Individuals with higher PCBs in tissues may therefore have been more susceptible to the disease and suffered higher mortality; (2) liver lesions that were common in striped dolphins that succumbed to the infection may have been caused by mobilized PCBs, and interacted with the disease to cause higher mortality in individuals with higher PCB burdens; and (3) the liver lesions could have been caused by a previous condition, resulting in higher PCB concentrations in tissues of affected dolphins because of a reduced capacity of the liver to metabolize PCBs.

A very detailed field study was carried out by Hall et al. (1997) to address possible relationships between PCB exposure and susceptibility to disease in gray seal pups. During a 3-year period, wild gray seal mother–pup pairs were captured and sampled for individual PCB congener analysis in milk and for determination of various hematological and blood chemistry properties. Pups were also challenged with morbillivirus vaccines and stimulated with mitogens to determine immunocompetence. No relationships were found between the prevalence of infection in pups (as an indicator of possible immunosuppression) and cumulative exposure to PCBs in mother's milk. Pups born to females with high levels of PCBs in the milk did not show any biochemical, hematological, or immunological abnormaliues in comparisons to pups suckling from females with lower concentrauions. In a different approach, attempting to test the hypothesis that organochlorines suppress the immune system of dolphins, Lahvis et al. (1995) determined concentrations of PCBs, DDT, DDE, and *o,p'*-DDE in blood samples of five free-ranging male bottlenose dolphins. They compared these concentrations with in vitro mitogen-induced proliferation responses of lymphocyte cultures from these same individuals. Linear regression analyses indicated correlations between reduced immune responses and higher organochlorine concentrations. This was considered consistent with the hypothesis and with findings in studies of laboratory animals, although sample sizes were very small and spurious effects could possibly exist (for example, age of dolphins in this study also appeared to be correlated with organochlorine concentrations).

Controlled experimental studies related to immune function and organochlorine exposure were carried out on harbor seals in the Netherlands (de Swart et al. 1993, 1994, 1996; Ross et al. 1996). Patterned after similar studies investigating reproductive effects (discussed previously), two groups of captive seals were captured as newly weaned pups in a relatively uncontaminated area of Scotland and held for a 1-year acclimation period. They were then matched by weight and sex and fed either herring from the Atlantic (relatively low in

organochlorine contamination) or more highly contaminated herring from the Baltic Sea over a 2.5-year period. Daily intakes of organochlorines were estimated based on chemical analysis of samples (including determination of specific PCB congeners) and calculations of toxic equivalents (TEQ, see definition in section on metabolism in this chapter). The Baltic herring diet was about five times as high in PCBs and ∑DDT and 10 times higher in toxic equivalents. As in previous studies (Brouwer et al. 1989), vitamin A levels were lower in the group of seals fed this diet. In addition, counts of total white blood cells and granulocytes (but not lymphocytes or monocytes) were higher. Immune function differed after stimulation with certain mitogens: natural killer cell activity and lymphocyte function assays were significantly lower in seals with greater organochlorine exposure (de Swart et al. 1994). Responses were inversely correlated with TEQ summations (predominantly PCBs) in blubber biopsies. Cellular rather than humoral immunity appeared affected, consistent with expectations from laboratory animal studies. Seals were then subjected to a 15-day fasting period at the end of the study. Although concentrations of organochlorines in blood were increased during mobilization of lipids as energy reserves, differences were not observed in either group after the 15-day period. These studies support the possibility that organochlorine exposure could have led to an increased susceptibility to the morbillivirus infections seen in wild populations of marine mammals. As pointed out by Kennedy (1995), however, immunosuppression also occurs as a direct result of morbillivirus damage to lymphoid tissues, resulting in the numerous secondary infections observed in recent morbillivirus-caused epizootics. Morbilliviruses are known for their very high virulence and have historically produced very high mortality in immunologically naive populations of terrestrial mammals, even before the widespread synthesis of organochlorines by humans. It is likely that morbillivirus infections alone were the primary cause of high mortality in recent marine mammal die-offs, but the possibility of organochlorines contributing to susceptibility cannot be fully excluded.

The St. Lawrence River estuary in Quebec, Canada, is highly contaminated with a wide variety of pollutants, including organochlorines, toxic elements, and polyaromatic hydrocarbons. Although it is not possible to separate the individual effects of these contaminants on disease processes, Béland et al. (1993) and Martineau 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. Reproductive abnormalities were also observed, along with high levels of numerous contaminants in comparison with tissues from other populations. Much interest centers around the role of organochlorines in the disease processes observed in these animals (Béland et al. 1993, Martineau et al. 1994). A high prevalence of carcinomas has also been reported in California sea lions, with a hypothesized link to possible contaminant exposure (Gulland et al. 1996).

Links between Organochlorine Levels and Marine Mammal Population Declines

It is often assumed that sublethal toxic effects of persistent contaminants will ultimately alter population size, survival, recruitment, and species composition of mammalian communities. However, as pointed out by McBee and Bickham (1990), changes in such parameters attributable to sublethal effects of contaminants have rarely been demonstrated in any wild mammalian populations, terrestrial or marine (see also Heinz 1989). 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 organochlorines are also likely to have been subjected to numerous other forms of human-induced stress, such as other contaminants, noise pollution and disturbance, habitat deterioration, or changes in food quantity and quality. The cause-and-effect relationships between contaminants and population declines of marine mammals may be further obscured by overhunting. In the cases of seals in the Baltic and belugas in the St. Lawrence, previous overhunting is well documented and was probably the primary cause of population declines, with contaminants thought to play a role in preventing population growth after hunting ceased (Helle 1980, Reijnders 1985, Sergeant and Hoek 1988). Very recently, more detailed assessments of trends in St. Lawrence River belugas showed that the population has not declined since the early 1970s, and that hunting in the 1970s was the major factor impacting population growth potential (Kingsley 1998). During recent years, in some parts of the Baltic, seal populations have increased or remained stable as concentrations of some organochlorines in blubber have decreased (Olsson et al. 1992). Sea lions in California, in contrast, have had elevated organochlorine concentrations, but populations continue to increase (O'Shea and Brownell 1998).

Although evidence for the impacts of organochlorines on reproduction in marine mammals is limited, it is supported by studies in which some of these compounds have been determined to affect reproduction in laboratory animals. In particular, dietary PCBs can profoundly impair reproduction of females in some mammals. The susceptibility of mammalian species to PCBs is variable, however, even among closely related taxa. Although the literature on contaminants in marine mammals often generalizes from studies of other mammals that show dramatic effects (such as

mink), some species have very low sensitivity. 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. This wide variance in sensitivity makes generalization to marine mammal species difficult. It is likely that concentrations of PCBs in food of the baleen whales and sirenians are typically lower than those demonstrated to cause effects in most other mammals. However, this may not be the case for piscivorous species of cetaceans and pinnipeds that feed near shore in contaminated regions.

Metabolism, Biotransformation, and Biochemical Toxicity

The metabolism, biotransformation, and excretion of organochlorines involves processes that convert these hydrophobic compounds to more polar metabolites. Details of the precise metabolic pathways are complex and incomplete, but models of these processes implicate certain organochlorines as inducers of enzymes that could also lead to endocrine imbalances, a critical biochemical link to reproductive impairment. A general overview of this rapidly advancing field is provided in this section. I encourage the reader to consult other sources, including texts or reviews in biochemical toxicology or biotransformation (e.g., Klaassen 1996), for more in-depth treatments. Reviews of the metabolism of organochlorines in relation to enzyme induction and possible mechanisms of toxicity have been prepared by a number of investigators, including Borlakoglu and Haegele (1991), Goldstein and Safe (1989), Hodgson et al. (1980), Matsumura (1985), Paasivirta (1991), Peakall (1992), Rice and O'Keefe (1995), and Safe (1984, 1990, 1994). De Voogt et al. (1990), Boon et al. (1992, 1994), and Reijnders (1994) have specifically reviewed this topic for marine mammals. This overview condenses information provided in these papers and references therein. This is a field that is growing rapidly.

Mixed Function Oxidase Induction

Research into the biochemical pathways of organochlorine metabolism in laboratory mammals has shown that initial steps take place on membranes of the endoplasmic reticulum of the microsomes of liver cells (hepatocytes). This is the site where the cytochrome P-450-dependent monoxygenase enzyme systems function, and an understanding of their role is critical for interpreting recent literature on contaminants in marine mammals. Cytochrome P-450 is actually a family of hemoproteins that gives a characteristic absorption spectrum of 450 nm, hence the derivation of the name. There are many types of cytochrome P-450 monoxygenases associated with the endoplasmic reticulum of the liver, any one or more of which may be induced by a particular foreign compound (xenobiotic). Each form in turn catalyzes the oxidative metabolism of a relatively specific group of lipophilic substrates. Referred to as the mixed function oxidase (MFO) system, these biochemical pathways initially evolved to allow animals to detoxify poisonous natural compounds (such as plant defensive chemicals) at a higher rate of metabolism, thus removing them from the body more quickly. [It has been suggested that natural selection may have resulted in carnivores being less able to metabolize xenobiotics than herbivores because of the wider number of plant toxins normally ingested by the latter (Peterle 1991).] The MFO system is also capable of enhancing the metabolism of anthropogenic chemicals such as the organochlorines, but during some metabolic transformations can actually render some compounds to forms that are more toxic. Many hundreds of xenobiotics from numerous sources are now known to cause induction of enzymes of the MFO system. All of these MFO-inducing xenobiotics are organic and lipophilic, and can only be excreted after metabolic conversion to polat products. Typically, the initial step involves oxygenation in the MFO system, which is characterized by the requirement of the reduced form of nicotinamide-adenine dinucleotide phosphate (NADPH), microsomes, and oxygen fot degradation of the foreign compound. Induction of MFO activity is a true induction, resulting in synthesis of new enzymes (rather than activation of previously synthesized enzymes). In some cases the metabolites produced from synthetic organochlorines can have greater toxicity than the parent compounds.

Cytochrome P-450 is the component that binds with oxygen and the substrates, thereby converting them into compounds that are more polar and subject to enhanced excretion. The significance of the presence of many cytochrome P450s is that each can exhibit a different substrate preference, providing the system with an overall ability to oxidize many different xenobiotics. The number of known genes that encode these various cytochtome P-450 enzymes is large and continues to grow. Nebert et al. (1991) described more than 150 forms of cytochrome P-450, each belonging to one of 27 gene families. A gene family may respond to a characteristic group of xenobiotics as well as endogenous compounds. The literature includes an evolving nomenclature for these families. Boon et al. (1992) provide a summary as follows: the gene is indicated by an italicized root symbol CYP (cytochrome P-450), followed by an Arabic numeral indicating the family, a letter designating the subfamily, and an Arabic numeral tepresenting the individual gene within the subfamily (e.g., CYP1A1).

Biochemists have tentatively grouped inducers of MFO

activity into two primary classes on the basis of drug metabolism studies. One class is characterized by induction by phenobarbital (PB) and includes a wide variety of anthropogenic compounds. The other class is induced by 3-methylcholanthrene (3-MC). PB-type inducers induce the CYP2B subfamily and result in a proliferation of the smooth endoplasmic reticulum and induction of cytochrome P-450, as well as a number of other oxidative activities, some of which have been measured in marine mammals (Table 10-3). There is little or no increase in arylhydrocarbonhydroxylase (AHH) activity. The 3-MC class of compounds, in contrast, does not cause a marked increase in the smooth endoplasmic reticulum, and induces cytochrome P-448. The 3-MC category of compounds induces oxidative activity involving AHH and *O*-deethylation of 7-ethoxycoumarin. The 3-MC class induces the CYP1A subfamily and includes the most potent enzyme inducer known, TCDD (Fig. 10-5). In addition to these two primary classes of enzyme inducers, some xenobiotic compounds induce both kinds of activities and are referred to as mixed-type inducers.

The Ah Receptor, PCBs, and the Toxic Equivalency Concept

Xenobiotics can function as enzyme inhibitors, inducers, or substrates, and the many interactions between different compounds and enzymes can be extremely complex. The mechanism of induction of cytochrome P-450 is incompletely known. Some of the observations can be accounted for by the Ah receptor model (Nebert and Gonzalez 1987, Safe 1994), in which highly hydrophobic xenobiotics enter cells through typical uptake processes and then possibly compete successfully with a hypothetical normal cellular ligand for a receptor protein, designated the arylhydrocarbon or Ah receptor. According to this model (which is very generalized and is rapidly being revised as new work develops), this results in the formation of an inducer–receptor complex in the cytosol. This complex is then translocated to an unknown site, leading to the transcription of specific mRNAs that, after translation on ribosomes, ultimately induce forms of cytochrome P-450 that are incorporated into the endoplasmic reticulum. This model may explain the enhanced metabolism of pollutants and the pattern of metabolites formed from xenobiotics present in the cytosol. However, metabolism may lead to the formation of reactive intermediates that are capable of binding to critical molecules in the cytosol or the nucleus, thereby initiating toxicity in the cell. A wide variety of toxic responses in laboratory animals have been correlated with P-450 induction. Most of the supporting information for the model has come from laboratory rats and inbred strains of laboratory mice, where it has been shown that strict molecular structure is required to bind successfully to the Ah receptor (Nebert and Gonzales 1987, Safe, 1984). The Ah receptor is thought to play a role in controlling portions of what biochemists refer to as Phase I and Phase II drug metabolism, which coordinate the oxidative metabolism and conjugation of xenobiotics. Phase I drug metabolism reactions include induction of aldrin epoxidation, AHH, ethoxycoumarin-*O*-deethylation (ECOD), and ethoxyresorufin-*O*-deethylation (EROD). Induction of enzymes involved in Phase II drug metabolism reactions include epoxide hydrolases, glucuronyl transferases, and glutathione-*S*-transferases. Patterns of enzyme induction can be used to distinguish among the various classes of inducers, and have been measured in some marine mammal species (Table 10-3; discussed later).

The toxic equivalency concept is based on the likely structure–activity relationships (SAR) of xenobiotics with receptors. TCDD has a planar aromatic structure that fits closely to the Ah receptors. Certain PCB congeners have a structure that can also fit this Ah or "dioxin receptor." Their toxicity depends on the location of the chlorine atoms. For example, when only one chlorine atom at the 4 position of PCB 126 (Fig. 10-4) is changed to 5′ (PCB 127), AHH induction decreases to 1/50,000 of that of PCB 126 (Paasivirta 1991). The measurement of AHH or EROD induction has been used as a screening method for possible TCDD-like toxicity of PCBs, with degree of induction showing correlation with other toxic effects in laboratory mammals. This has resulted in the "toxic equivalency factor (TEF)" concept. The concept has been most extensively applied to PCBs where it was first developed for marine mammals by Tanabe and colleagues, particularly with reference to cetaceans (see below). After careful scrutiny, a number of caveats to the use of TEFs have been recently raised (Reijnders 1994, Safe 1994). Nevertheless, a body of literature has developed that uses the TEF approach in studies of contaminants in marine mammals, requiring a brief overview.

A TEF can be calculated for a specific PCB congener based on the potency of a biological response in relation to the response to TCDD, the most toxic of the PCDDs and PCDFs (Safe 1990, Ahlborg et al. 1994). The coplanar and mono-orthoplanar PCBs (Fig. 10-4) give the most similar responses to TCDD, but activities are generally much less pronounced than in PCDDs and PCDFs. TEFs are calculated as the ratio of the magnitude of a biological response to the PCB to the same response to TCDD. TEFs are commonly based on the ability to induce components of the MFO system (an Ah receptor-mediated response) but can also be expressed in terms of immunotoxicity, carcinogenicity, or other responses. The PCB congener with the highest TEF is PCB 126 (0.1 TEF).

The TEF concept has been extended to include an overall estimate of the TCDD-like toxicity of the entire complex

Table 10-3. Summaries of Studies Reporting Enzyme Induction in Marine Mammals

Group and Species	General Findings	References
Pinnipeds		
Cystophora cristata	Hepatic cytochrome P-450 activities measured in 18 adults and 7 pups, and in response to phenobarbitol treatment in 1 pup. Enzyme activities determined for NADPH (cytochrome P-450), NADH (cytochrome b_5), EROD, PROD, ECOD, MCOD, E_2-OHase, UDP-GT. No contaminant concentrations determined.	Goksøyr et al. 1992
Halichoerus grypus	Hepatic cytochrome P-450 levels increased with age and were correlated with concentrations of PCBs in blubber.	Addison et al. 1988
H. grypus	EROD activity determined in 8 adults and 12 pups. Activity in liver samples was higher than in kidney, and higher in adults than pups. EROD activity did not differ by sex or age in adults. Concentrations of contaminants not reported.	Addison and Brodie 1984
Phoca groenlandica	Hepatic cytochrome P-450 activities measured in 10 adult females and 11 pups, and in response to phenobarbital treatment in 1 pup. Enzyme activities determined for NADPH (cytochrome P-450), NADH (cytochrome b_5), EROD, PROD, ECOD, MCOD, E_2-OHase, UDP-GT. No contaminant concentrations determined.	Goksøyr et al. 1992
P. vitulina	Hepatic EROD and BPOH activities similar to other mammals, whereas cytochrome P-450 and b_5 concentrations were slightly lower. MFO activities in newborns lower than in adult females. No data provided on contaminant concentrations.	Addison et al. 1986b
P. vitulina	Determined in vitro hepatic microsomal metabolism of PCB 127 and EROD activity in one seal. EROD activity was comparable to the rat, metabolite production was comparable to a harbor porpoise.	Murk et al. 1994
Odontocete cetaceans		
Delphinapterus leucas	Extensive biochemical and molecular characterization of hepatic microsomal enzymes from 8 males and 5 females from the Canadian Arctic. Determined activities of cytochrome P450, CYP1A, cytochrome b_5, EROD, PROD, E_2-OH, AHH. CYP1A activity verified and correlated with PCB concentrations in tissues. Activities consistently higher in males. Immunochemical similarities of other P-450 forms related to CYP2B and CYP2E1 observed.	White et al. 1994
Globicephala macrorhynchus	Hepatic cytochrome P-450 activities determined for 2 fetuses, and 33 immature and mature individuals. Enzyme activities determined for NADPH-cytochrome c reductase, ALDE, AH, AHH, EROD. No significant difference in monooxygenase activities between sexes or among age groups. Concentrations of contaminants not reported.	Watanabe et al. 1989
Orcinus orca	Hepatic cytochrome P-450 activities determined for 3 individuals. Enzyme activities determined for NADPH-cytochrome c reductase, ALDE, AH, AHH, EROD. Concentrations of contaminants not reported.	Watanabe et al. 1989
Phocoena phocoena	Determined in vitro hepatic microsomal metabolism of PCB 127 and EROD activity in one porpoise. EROD activity was less than in a harbor seal, but PCB metabolite formation was comparable.	Murk et al. 1994
Stenella coeruleoalba	BPMO activity in biopsy skin samples of 7 individuals determined in relation to organochlorine concentrations in blubber. Enzyme activities and organochlorine concentrations were much higher than in fin whales from the same area.	Fossi et al. 1992
S. coeruleoalba	Hepatic cytochrome P-450 activities determined for 5 individuals. Enzyme activities determined for NADPH-cytochrome c reductase, BPOH, ALDE, AH, EROD. Concentrations of contaminants not reported.	Watanabe et al. 1989
Mysticete cetaceans		
Balaenoptera acutorostrata	Determined concentrations of hepatic cytochrome P-450, cytochrome b_5, EROD, ECOD, NADPH-cytochrome P-450 reductase, AHH, APDM in four females, one male, and two fetuses. Intrahepatic differences observed only in EROD. Concentrations of contaminants not reported.	Goksøyr et al. 1985, 1986
B. acutorostrata	Determined concentrations of hepatic and renal cytochrome P-450, cytochrome b_5, NADPH-cytochrome P-450 reductase, EROD, ECOD, biph.-40H, AHH, E-20H, UDP-GT, GSH-T in four fetuses and 10 adults.	Goksøyr et al. 1988, 1989
B. physalus	BPMO activity in biopsy skin samples of 9 individuals determined in relation to organochlorine concentrations in blubber. Enzyme activities and organochlorine concentrations were much lower than in striped dolphins from the same area.	Fossi et al. 1992

Table 10-3 continued

Group and Species	General Findings	References
Other		
Ursus maritimus	Detailed analysis of CYP1A, CYP2B protein content, EROD, PROD, BROD in hepatic microsomes together with determination of concentrations of congeners of PCBs, PCDDs, PCDFs, other organochlorines. High correlations between CYP1A activity and EROD, PROD, TEQs, PCBs, PCDDs, PCDFs. Low BROD correlations. CYP2B correlations highest with chlordanes, o-PCBs.	Letcher et al. 1996

Source: See also Fossi and Marsili 1997.

AH = aniline hydroxylase; AHH = aryl (benzo[a]pyrene) hydrocarbon hydroxylase; ALDE = aldrin epoxidase; APDM = aminopyrine *N*-demethylase; biph.-4OH = biphenyl 4-hydroxylase; BPMO = benzo[a]pyrene monooxygenase; BOPH = benzo[a]pyrene hydroxylase. CYP1A = subfamily of monoxygenases (see text); CYP2B = subfamily of monoxygenases (see text); CYP2E = subfamily of monoxygenases (see text); E_2-OH = estradiol 2-hydroxylase; ECOD = 7-ethoxycoumarin *o*-deethylase; EROD = 7-ethoxyresorufin *o*-deethylase; GSH-T = glutathione *S*-transferase; MFO = mixed function oxygenase; NADH = nicotinamide adenine dinucleotide (reduced); NADPH = nicotinamide adenine dinucleotide phosphate (reduced); PCBs = polychlorinated biphenyls. PROD = pentoxyresorufin *o*-depentylase. UDP-GT = uridine diphosphate glucuronyl transferase.

mixture of PCBs, PCDDs, and PCDFs present in environmental and tissue samples. This is commonly referred to as the total "toxic equivalents" or TEQ of a mixture, calculated as the sum of the concentration of each PCB, PCDD, or PCDF times its TEF for the entire measured sample. Although individual PCBs have much lower TEFs than the dioxins and dibenzofurans, they are nearly always present at much higher concentrations, and the TEQ of a mixture is thought to better reflect the overall potential toxic impact. However, a number of investigators have pointed out that toxic responses may not be additive (although this may be the case for Ah receptor-mediated toxicity) but can be nonadditive with antagonistic effects. This is clearly seen in some response systems such as immunotoxicity, where the measured responses can be considerably less than predicted on the basis of the sum of TEFs (Safe 1994). Furthermore, as pointed out by Reijnders (1994), even when TEQs are calculated on the basis of Ah receptor-mediated responses, differences in induction of P-450-based enzymes exist among species and organs, and the toxicities of PCB metabolites are not included in calculations. This is important because some phenolic metabolites of PCBs have greater potencies than the parent compounds.

Metabolism and Possible Toxicity of PCB Congeners

The quantification of PCBs in marine mammal tissues was initially limited to comparisons with standard mixtures such as Aroclor 1254. However, Tanabe and coworkers at Ehime University in Japan (see references below) made groundbreaking advances in isomer-specific quantification of PCBs in cetacean tissues. Their initial findings on residue concentrations led to some interesting hypotheses suggesting possible links among PCB congener profiles, MFO activity, and potential for toxicity. Although there are in theory 209 possible isomers and congeners of PCBs (only about 120 are known from industrial mixtures), the investigations focused on a subset of the 20 coplanar PCBs with non-*ortho* chlorine substitution in the biphenyl rings, in particular those with four or more chlorine atoms in the *para* and *meta* positions (Fig. 10-4). These were chosen because of structural similarity to TCDD and their similar toxic responses (but requiring higher doses), that include induction of MFO activity.

Tanabe et al. (1987a, 1988) revealed the presence of trace amounts of coplanar PCB 77, PCB 126, and PCB 169 (Fig. 10-4) in small sample sizes of fish, cetaceans (finless porpoise [*Neophocaena phocaenoides*], Dall's porpoise, Baird's beaked whale [*Berardius bairdii*], Pacific white-sided dolphins [*Lagenorhynchus obliquidens*], killer whales [*Orcinus orca*]), humans, dogs, and cats. Samples in remote marine areas contained coplanar PCBs, and their concentrations in animal tissues were highly correlated with total PCB concentrations (although three to five orders of magnitude lower). Considered together with the proportions of coplanar PCBs determined in commercial PCB preparations, these findings constituted evidence that coplanar PCBs were widely distributed as contaminants of the global environment as a direct result of general industrial PCB pollution. The initial study (Tanabe et al. 1987a) revealed that the amounts of these coplanar PCBs in the environmental samples, although low, were much higher than those of PCDDs and PCDFs, both in terms of absolute concentrations and on the basis of toxic equivalency as related to enzyme induction in laboratory animals (discussed previously). On the basis of the pattern of relative concentrations of these coplanar PCBs in commercial mixtures and biota, these investigators further suggested that cetaceans may have a lower capacity to metabolize these congeners. (However, this suggestion was based on small sample sizes of a limited number of other taxa.) Tanabe et al. (1987a:158) further cautioned that, although coplanar PCBs may have a greater potential for harm

than dioxins or dibenzofurans when viewed from a toxic equivalency standpoint, "the toxic effects of trace levels of coplanar PCBs, PCDDs, PCDFs on a long-term basis to humans and environmental animals are neither fully understood nor clearly demonstrated."

Additional investigations on this topic ensued. On the basis of calculations derived from the residue concentrations of coplanar PCBs (Fig. 10-4) in cetaceans relative to those in potential food organisms and a small number of other mammalian species, it was hypothesized that cetaceans lacked the capacity to metabolize these congeners, and, as a correlate, were likely to have no PB-type MFO systems and a relatively small capacity for induction of MC-type enzymes (Tanabe 1988, Tanabe et al. 1988). It was also suggested that low enzyme induction capacity could cause reproductive toxicity by these chemicals (Tanabe 1988), but this speculation was not based on direct measurements of enzyme induction capacity or strong evidence for reproductive impairment in cetaceans. Furthermore, the relationship between MFO activity and reproductive effects in mammals currently remains an area of considerable debate and uncertainty (Stone 1994).

Kannan et al. (1989) expanded on the earlier work and determined concentrations of mono- and di-*ortho* analogs of the coplanar PCBs (Fig. 10-4) as well as these non-*ortho* chlorine substituted congeners in the same or a similar series of samples. Investigations into the occurrence of these analogs were called for by their potentially comparable toxicity with the non-*ortho* chlorine substituted PCBs, as suggested by their structure and enzyme-inducing capability in laboratory mammals. The mono- and di-*ortho* coplanar PCBs were also found in all samples at low concentrations but in greater amounts than dioxins or dibenzofurans. Interestingly, however, the concentrations of all coplanar PCBs relative to total PCBs did not vary between humans and cetaceans, an observation inconsistent with the hypothesis that cetaceans lack an ability to metabolize these compounds (Kannan et al. 1989). Data on concentrations of specific PCB congeners in harbor porpoises from near the Netherlands, and other cetaceans, agreed with earlier findings of metabolism of congeners with vicinal H atoms in the *ortho*, *meta* position and a maximum of one *ortho*-Cl, but also suggested an ability for cetaceans to metabolize congeners with vicinal H atoms in *meta* and *para* positions, although at a lesser capacity than in seals (Duinker et al. 1989). PCB 153 is especially resistant to metabolism in the harbor porpoise (van Scheppingen et al. 1996).

The hypothesis that cetaceans lack an ability to metabolize coplanar PCBs was further tested by Watanabe et al. (1989) by the direct determination of MFO activity in liver microsomal samples from 33 short-finned pilot whales (*Globicephala macrorhynchus*) (and two fetuses), five striped dolphins, one killer whale, and one laboratory rat. MFO activity of both MC and PB types was measured (Table 10-3) and found in all cetaceans, with some significant differences among species. Levels of cytochrome P-450 in cetacean liver microsomes were comparable to those from other mammals. Activities of EROD and AHH in cetaceans were comparable to those in the rat, whereas activities of aldrin epoxidase (ALDE) and AH were lower (Watanabe et al. 1989). Although both MC and PB-type induction was demonstrated to occur, results were interpreted to support the hypothesis that PB-type induction (low ALDE and AH activity) is low in cetaceans and could account for the pattern of coplanar congener accumulation in tissues demonstrated by other studies. NADPH cytochrome c reductase, cytochrome P-450, and MFO activities were generally lower in the fetal pilot whales than in mature or immature individuals, but did not differ between sex or age groups (Kannan et al. 1989). This study was a milestone in providing measurements of MFO in cetaceans. However, it tempers earlier conclusions about a complete inability of cetaceans to metabolize certain coplanar PCBs because of a lack of enzyme induction capability. The work also underscores the need to better understand the variability in MFO activity levels in wild populations, exacerbated by very little data on relationships between organochlorine concentrations in tissues or food and MFO activity levels in marine mammals (Table 10-3). Additional studies, such as those by Letcher et al. (1996), in which MFO activities, CYP protein contents, and organochlorine concentrations in polar bear livers were determined and correlations established, will help fill these information gaps. The recent establishment of cell culture lines and verification of the existence of an Ah receptor in bottlenose dolphins should also allow more thorough investigation of various aspects of P-450 induction in the future (Carvan et al. 1994).

As progress in understanding details of PCB metabolism in cetaceans was advancing based on analytical chemistry studies in Japan, researchers in Europe also began congener-specific determinations in marine mammals, emphasizing seals. Boon et al. (1987) determined concentrations of individual congeners in the diet, blood, and feces of captive female harbor seals fed organochlorine-contaminated, as well as less-contaminated control fish in experimental studies in the Netherlands. The patterns of PCBs within fish and seal blood samples were nearly constant, but differed substantially between the two; differences in congener composition between seal groups were related solely to diet (Storr-Hansen et al. 1995). Molecular structure of congeners that persisted and accumulated in seal blood differed from that of congeners that were metabolized. Congeners that were me-

tabolized by the seals were those with 3 to 6 chlorine atoms and penta- and hexa-chlorinated biphenyls with vicinal H atoms at *meta–para* positions, or vicinal H atoms at *ortho–meta* positions with single *ortho*-chlorine atoms. Congeners with 5 to 10 chlorine atoms and with either 2 or 3 *ortho*-chlorines were not metabolized. Boon et al. (1987) noted that congener persistence was likely related to enzymatic metabolism as it is influenced by molecular structure. Congeners with vicinal H atoms at *meta–para* positions were metabolized in "globular configurations" (rings perpendicular); such metabolism is likely to be carried out by the P-450 enzymes. Congeners with *ortho–meta* vicinal H atoms reach a planar configuration that is more likely to be metabolized by P-448 enzyme systems. Structurally, the mono-*ortho* chlorine-containing congeners can reach both globular and planar configurations, and are known to be mixed-type MFO inducers (Boon et al. 1987). The capacity of harbor seals to metabolize these two classes of PCB congeners is shared with some small cetaceans and the polar bear, but may be diminished in ringed seals (Boon et al. 1989). Ratios of the more easily metabolized PCB 52 to the resistant PCB 153 also are similar among porpoises and dolphins from the North Sea of Scotland but are much lower in harbor seals from the same region (Wells et al. 1994).

The pattern of congeners found in polar bears is especially unique. Many of the congeners prominent in their seal prey are absent or occur at relatively low concentrations in polar bear tissues. Identities of specific congeners indicate an ability of polar bears to metabolize PCBs with nonchlorinated *para* positions, adjacent nonchlorinated *ortho–meta* positions, or both *ortho* positions chlorinated in one ring (Norstrom et al. 1988). Differences in metabolism of congeners among marine mammal species, as well as differences between marine mammals and laboratory species, point "to the dangers of extrapolation between species" (Boon et al. 1992:152). Many recent studies have included congener-specific determinations in marine mammals and will help further elucidate patterns of PCB metabolism (Appendices 1 to 4). Such recent work with belugas of the St. Lawrence River estuary, for example, implies high activity of both CYP1A and CYP2B enzyme systems, perhaps through heightened induction from heavy PCB exposure (Muir et al. 1996a).

The use of enzyme induction information is growing in the study of contaminants in marine mammals. These are sensitive bioassays, but they do not firmly establish the existence of harmful impacts to individual animal health or population status. Much like the simple presence of contaminants in tissues, consequences can only be inferred based on results of laboratory studies of other species, which unfortunately sometimes show significant variation in responses. In addition to the caveats and research needs noted above, Peakall (1992:99) recently pointed out that for wildlife species in general, "it has not been established whether induction of cytochrome-mediated enzyme activities leads either directly or indirectly to toxicity or whether enzyme induction and toxicity are independent aspects of the responses." Clearly, this is an area of much needed additional, coordinated research.

Marine Mammals and the "Gender Benders"

Much recent attention has focused on the role of organochlorine contaminants as disrupters of the endocrine system in humans and wildlife. The existence of chemicals with endocrine-disrupting capacity ("gender benders") is well known, as exemplified by the drug diethylstilbestrol (DES), used to prevent miscarriages in women in the 1950s and 1960s. Laboratory animals and humans exposed to DES prenatally can exhibit a number of anomalies of the reproductive system as adults. Similar changes have been seen in wildlife species in areas very heavily contaminated with organochlorines, including altered hormone levels and dysfunctional manifestations of reproductive behavior (Colborn et al. 1993, Raloff 1994). Endocrine disrupters can include any xenobiotics that interfere with the normal function of hormone receptor proteins (which mediate the effects of endogenous hormones on gene activation). These chemicals can either mimic the hormone (Fig. 10-6) by binding to the receptor and activating a hormonelike response, or can act as an antagonist by binding to the receptor protein, making it unavailable to be activated by the natural hormone. In the case of the estrogen receptor (ER), the former action is termed estrogenic, whereas the latter is antiestrogenic.

Organochlorines exhibit a wide and mixed array of estrogenic activity in studies of laboratory animals. For example, *p,p′*-DDE is not estrogenic, whereas *o,p′*-DDT is estrogenic; Ah receptor agonists, however, such as TCDDs and PCDFs, are antiestrogenic. The presence of complex mixtures of both estrogenic and antiestrogenic organochlorines in marine mammals makes it difficult to predict ultimate biological effects. The organochlorines may be present at lower concentrations than natural hormones and may have much lower activities; hormonelike natural chemicals (such as those found in the human diet) often have much stronger endocrine effects than organochlorines in laboratory tests. The question of whether organochlorines at typical background concentrations exert any meaningful endocrine effects at all is a topic of much debate, particularly as applied to humans (Colborn and Clement 1992, Colborn et al. 1993, Stone 1994, Safe 1995). There has been little intensive study of this topic in marine mammals although the experimental

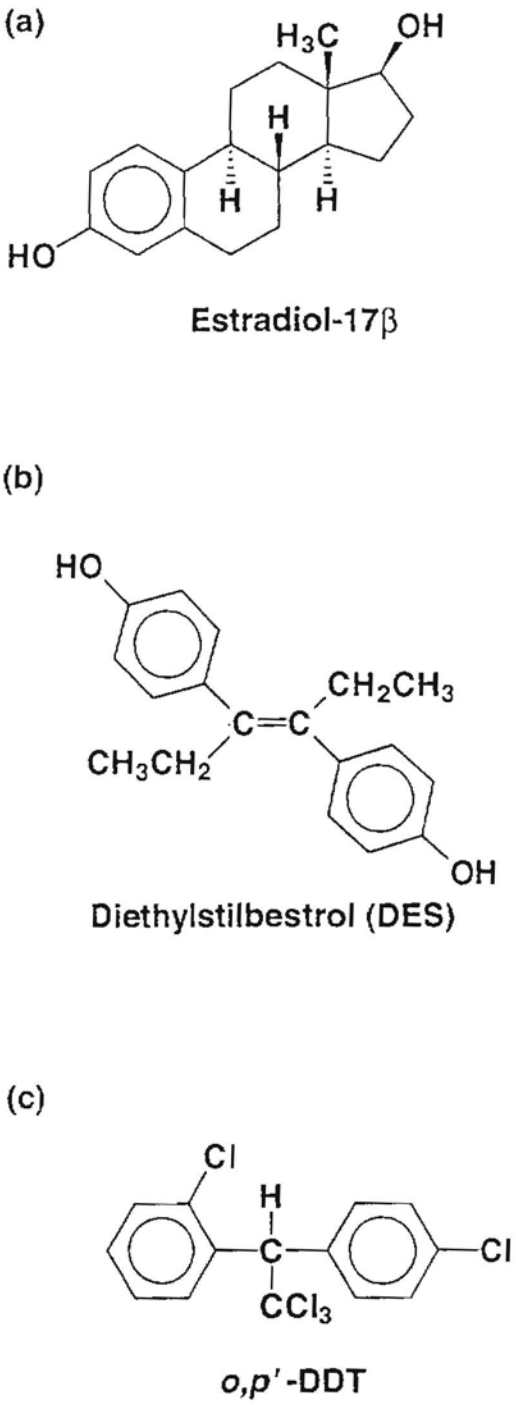

Figure 10-6. Structural similarities of some estrogenic compounds. Estradiol-17β is a "natural" ovarian estrogen, diethylstilbestrol (DES) is a synthetic pharmaceutical, and *o,p'*-DDT is an isomer of the insecticide DDT. Controversy surrounds the importance of synthetic organochlorines as estrogenic or antiestrogenic disrupters of endocrine metabolism at typical background levels.

studies of harbor seals support such a possibility. Not only do captive seals fed diets high in organochlorines fail to reproduce normally, they also show biochemical lesions (lowered retinol and thyroid hormones) compatible with an endocrine disruption hypothesis (Brouwer et al. 1989).

Toxic Elements

A long-standing postulate of toxicology, first attributed to the Renaissance scientist Paracelsus in the 1500s, is that "all substances are poisons; there is none which is not a poison. The right dose differentiates a poison from a remedy" (Gallo 1996). No better example of this can be found than in the study of toxic elements. Unlike organochlorines, elements are naturally occurring substances, many of which are essential to normal metabolic function and sustenance of life. However, at certain exposure levels, these elements can have toxic effects, and some elements have no known essential role in normal biological processes. About 80 of the elements on the periodic chart can be considered metals. Goyer (1996) provides an overview of toxicology of these metallic elements. A few elements have been a major focus in studies of marine mammals, primarily because of their known danger and toxicity to humans and other animals. Significant attention has thus been given to investigating cadmium, lead, and mercury in tissues of marine mammals. These metals are treated in depth in this chapter and an overview of some of the other elements is also provided. Original studies have reported results of analyses of up to 40 trace elements in tissues of nearly 6000 individuals in more than 60 species of marine mammals. Many of these studies are summarized in Appendices 5 to 8. The methods used to quantify residues of toxic elements in tissues can vary among studies. These should include systematic procedures for tissue collection and storage that prevent spurious contamination; up-to-date methods of sample preparation, including tissue homogenization and digestion; and the use of quality assurance procedures such as comparisons with certified standard reference materials, use of procedural blanks, multiple analyses of the same samples within the same laboratories, and interlaboratory calibration and comparison studies. Zeisler et al. (1993) provide an example of excellent quality assurance procedures for determination of elemental concentrations in marine mammal tissues. Actual chemical quantification procedures can vary with the laboratory, elements chosen for analysis, desired sensitivity, and overall objectives. Such techniques may involve atomic absorption spectrometry (flame, flameless, or cold vapor may be appropriate depending on the element), instrumental neutron activation analysis with gamma ray spectrometry (e.g., Mackey et al. 1996), inductively coupled argon plasma emission/mass spectrometry, x-ray fluores-

cence, prompt gamma activation analysis, and differential pulse and square wave stripping voltammetry (e.g., Zeisler et al. 1993).

Cadmium

Cadmium is a naturally occurring, nonessential element. It can become an environmental contaminant as a by-product of various industrial processes, such as mining and smelting, petroleum production, and manufacturing of a number of products. It has been used in items as diverse as storage batteries, paints, and nuclear reactors, and is also released into the environment through motor vehicle exhausts and the breakdown of automobile tires. Cadmium concentrations can increase in sediments of aquatic systems subject to industrial effluents. In mammals, toxic effects of cadmium exposure are mitigated by protective binding with endogenously produced metallothionein, a low molecular weight protein. Cadmium typically reaches its highest concentrations in the kidneys, although greater amounts can be found in the liver in cases of unusually excessive exposure. Humans chronically exposed to cadmium ingestion from polluted mine waste water used for crop irrigation near Toyama Bay in Japan suffered itai-itai ("ouch-ouch") disease. This syndrome, which included leg and back pain, skeletal deformities, and susceptibility to bone fractures, was attributable to loss of calcium through cadmium-damaged kidneys (Goyer 1996). A variety of other toxic effects has also been produced in laboratory mammals subjected to excessive cadmium exposure, including disorders of the circulatory system, nervous system, and reproductive system (particularly in males), as well as renal system pathology. However, in some cases relatively high amounts are required to produce these effects, some of which may fail to occur in the presence of adequate dietary zinc and selenium. Cadmium is only slowly excreted from the body and has a half-life of 30 years in humans.

Cadmium concentrations have been determined in organs of numerous marine mammal species (Appendices 5 to 8). As in other mammals, almost without exception the highest cadmium concentrations in organs of marine mammals occur in kidneys, with lesser concentrations in liver, followed by relatively lower amounts in muscle and most other organs and tissues, including bone. The relatively high accumulation of cadmium in kidneys has been demonstrated for at least 28 species of marine mammals in all major taxonomic groups (see references in Appendices 5 to 8). Great variation in cadmium concentrations in kidneys of marine mammals has been noted among individuals, with much of the variation attributable to increased accumulation with age (Table 10-4). Unusually high concentrations of hundreds of parts per million of cadmium have been reported in kidneys of a variety of species (Table 10-5).

Despite seemingly high concentrations in kidneys of some individuals (linked to age in many), I am unaware of any published observations demonstrating cadmium-induced pathology in marine mammals. Some of the high cadmium concentrations reported in marine mammals are undoubtedly a result of naturally high cadmium concentrations in prey species rather than anthropogenic contamination. High cadmium concentrations in organs of squid, for example, are a known source of elevated cadmium in marine mammals (Hamanaka et al. 1977; McClurg 1984; Leonzio et al. 1992; Szefer et al. 1993, 1994; Malcolm et al. 1994; Caurant and Amiard-Triquet 1995). Certain molluscs have also been suggested as a dietary source of cadmium in walruses (Miles and Hills 1994).

Marine mammals appear to share the protective action of metallothionein proteins against cadmium toxicity, first reported in terrestrial mammals. Metallothioneins are induced by the presence of divalent cations such as Hg^{++}, Cd^{++}, Cu^{++}, and Zn^{++} and have a high affinity for binding such cations. Forms of metallothioneins have been reported in an array of organisms ranging from blue-green algae to mammals. Metallothionein has been isolated from tissues of gray seals and northern fur seals (Olafson and Thompson 1974), California sea lions (Lee et al. 1977), ribbon seals (*Phoca fasciata*) (Mochizuki et al. 1985), harbor seals (Mochizuki et al. 1985, Tohyama et al. 1986), and narwhals (*Monodon monoceros*) (Wagemann et al. 1984). Other metal-binding proteins have been isolated from tissues of sperm whales and California sea lions (Ridlington et al. 1981). Most of the cadmium in the cytosol of sea lion liver and kidney is in association with metallothionein (Lee et al. 1977). In the harbor seal, metallothionein levels in livers and kidneys increased positively with age and with cadmium and zinc concentrations, indicating sequestration of these metals by this protein. The thiol groups of metallothionein proteins are thought to act as soft Lewis bases that bind readily with soft (easily polarizable) Lewis acids such as Zn^{++}, Cd^{++}, and Hg^{++} (Perttilä et al. 1986). In addition to zinc, some studies have shown positive correlations between cadmium and selenium concentrations in organs of marine mammals, indicating participation of these elements in formation of the biochemical complexes that reduce toxicity.

There appear to be no notable differences between the sexes in cadmium concentrations in tissues of marine mammals. Little cadmium seems to be transferred to the fetus (Honda and Tatsukawa 1983, Roberts et al. 1976, Wagemann et al. 1988), although minor amounts of such transfer have been documented (Meador et al. 1993). In striped dolphins, cadmium in bones increased primarily in the late suckling

Table 10-4. Summary of Studies Reporting Relationships between Age, Sex, and Metal Concentrations in Marine Mammals

Group and Species	Organ	Relation with Age and Sex	References
Pinnipeds			
Arctocephalus gazella	L	Cd increase with age. No relationship with age and concentrations of Rb, Mg, Sr, Mo, Pb, Cu, Zn, Cr, Hg.	Malcolm et al 1994
A. pusillus	Multiple organs	Hg increase with age in liver, spleen, brain, and hair.	Bacher 1985
Callorhinus ursinus	Bone, K, L, M	Cd increase with age in liver, muscle, bone, but not kidney. Hg increase with age in liver. No relationships with age and Pb, Ni, Zn in kidney, liver, muscle, or bone.	Goldblatt and Anthony 1983
C. ursinus	K, L, M	Age positively correlated with Hg in muscle, liver, kidney, Fe in muscle and liver, Cd in kidney. Negative correlation with age and Mn in muscle, kidney, Cu in kidney.	Noda et al. 1995
C. ursinus	K, L, M	Concentrations of Cd, Pb in liver and kidney did not correlate with age. Hg in liver correlated with age. Hg in kidney and muscle did not.	Anas 1974a
Erignathus barbatus	L, M	Hg, Se increase with age.	Smith and Armstrong 1975, 1978
Eumetopias jubatus	K, L, M, O	Cd increase with age in kidney, liver.	Hamanaka et al. 1982
Halichoerus grypus	K, L	Positive correlation with Se, Hg, and age.	Perttilä et al. 1986
H. grypus	B, K, L	Positive correlations between age and Cd, Cr, V, Hg, and Se in either kidney, liver, or both. Negative correlation of Co with age. No correlations with age for Al, As, Pb. Relationships with other elements also determined	Frank et al. 1992
H. grypus	Teeth	Age and Cd, Cr, Pb show negative correlation, Zn positive correlation, Cu no correlation.	Heppleston and French 1973
H. grypus	Br, K, L	Cd increase with age in kidneys, Hg increased with age in livers. Hg in brain did not increase with age.	Heppleston and French 1973
Odobenus rosmarus	K, L	Cd, As increase with age in liver and kidney, Zn in kidneys. No correlations with age and Pb, Hg, or Se. No metals varied with sex except higher Se in livers of females and higher As in livers and kidneys of males.	Warburton and Seagars 1993
O. rosmarus	K, L, M	Hg increase with age in liver.	Born et al. 1981
Phoca groenlandica	Br, K, L, M	Cd, Hg, Se increase with age in kidney, liver, muscle, dependent on location. No consistent relation with age and Cu.	Ronald et al. 1984b
P. groenlandica	K, L, M, O	Cu and Zn higher in pups than mothers, higher proportion MeHg in livers of pups. MeHg correlated with age in muscle only.	Wagemann et al. 1988
P. groenlandica	L, M	Hg increase with age class.	Botta et al. 1983
P. hispida	K, L, M	Cd, Hg increase with age in kidney; Cu decrease with age in liver, kidney, muscle. As increase with age in liver; Pb independent of age; other relations also examined.	Wagemann 1989
P. hispida	K, L	No correlation with age and Se or Hg.	Perttilä et al. 1986
P. hispida	Hair	No differences in concentrations of Cd, Cr, Hg, Ni, Pb in hair of still-born, pups, adults, except Ni higher in still-borns than pups, yearlings; Hg lower in subadults to age 2 than adults or pups.	Hyvärinen and Sipilä 1984
P. hispida	K, L, M	Cd in kidney and Ag, Hg in liver increased with age; Cu in three tissues and Zn in muscle decreased with age. No age effects noted with Pb, Se.	Wagemann 1989
P. hispida	L, M	Hg, Se increase with age.	Smith and Armstrong 1975, 1978
P. vitulina	K, L	Cd, Hg in liver and kidney increase with body length.	Tohyama et al. 1986
P. vitulina	K, L, M, O	Cd increase with age in kidney, liver. No increase in Pb with age. Hg increase with age in liver, rate of increase varies with location.	Roberts et al. 1976
P. vitulina	B, K, L	Positive correlations between age and Cd, Cr, V, Hg, and Se in either kidney, liver, or both. Negative correlation of Co with age. No correlations with age for Al, As, Pb. Relationships with other elements also determined.	Frank et al 1992
P. vitulina	K, L	Cd increase with age in kidneys and Pb and Hg in livers of males but not females. No correlation with age and Pb in kidney, As or Se in liver.	Miles et al. 1992
P. vitulina	Br, K, L	Apparent increases with age in Cd and Pb in liver and kidney, Hg in liver. No age-related patterns in Zn, Cu concentrations in kidney, liver, or brain.	Drescher et al. 1977
P. vitulina	Hair, skin	Pb, Cd increase with age in hair of males, not females. Cd higher in males. No differences or effects noted for Hg.	Wenzel et al. 1993
P. vitulina	Br, K, L	Cd increase with age in kidneys, Hg increase with age in livers. Hg in brain did not increase with age.	Heppleston and French 1973
P. vitulina	L	Hg increase with age in livers.	Koeman et al. 1972
P. vitulina	Br, K, L	Hg, Se increase with age in liver.	Reijnders 1980

Table 10-4 continued

Group and Species	Organ	Relation with Age and Sex	References
P. vitulina	K, L	Hg increase with body length in liver, kidney, Se increase in liver.	Himeno et al 1989
Zalophus californianus	Br, K, L, M, O	No apparent Hg increase with age.	Buhler et al 1975
Odontocete cetaceans			
Delphinapterus leucas	L	Hg increase in liver with age.	Béland et al. 1991
D. leucas	K, L, M	Hg increase with age in liver and kidney, Cd increase with age in kidney, Cu decrease with age in liver, muscle, and kidney	Wagemann et al. 1990
D. leucas	K, L, M	Positive correlations with age and Cd, Se, Hg in liver and Hg in kidneys	Hansen et al. 1990
D. leucas	L	Ag, Hg, Se increase with age.	Becker et al. 1995
Globicephala melaena	L	Hg, Pb, Se increase with body length.	Meador et al 1993
G. melaena	B, K, L, M	Hg increase with age in all organs, depending on location Cd increase with age or length in kidneys, liver. No correlation with age and Cu, Zn, Pb. Other relationships noted.	Muir et al. 1988a
G. melaena	K, L, O	Hg, Se, Pb increase with body length in livers. Only Pb in brain differed by sex.	Meador et al. 1993
G. melaena	B, K, L, M	Correlations with body size investigated for Ag, Cd, Cu, Hg, Se, Zn. Hg in muscle, Se and Hg in liver, and Cd, Se and Zn in kidneys were positively correlated with size No differences between sexes in total Hg, which was lower in immatures.	Julshamn et al 1987
G. melaena	K, L, M, O	Cd, Hg, Se, in liver and kidney correlated with age and length. Other patterns depended on pod of origin Metals in liver higher in females than males within age groups.	Caurant et al. 1993
G. melaena	L	Ag, Hg, Se increase with age.	Becker et al 1995
G. melaena	L	Ag, Hg, Se increase with body length, eight other elements show no correlation with length.	Mackey et al. 1995
Lagenorhynchus albirostris	B, K, L, M	Cd increase with length in liver, kidney, with age in muscle No correlation with age or length for Hg, Se, Cu, Zn, Pb.	Muir et al. 1988a
Monodon monoceros	B, K, L, M	Complex relationships among effects of size and sex on concentrations of Hg, Se, Cd, Cu, Zn, and Pb in four tissues. Only 7 of 26 relationships involved sex differences, with no obvious pattern Cd increase with size in kidneys, but decrease in livers; Hg increase with size in livers of males but decrease in females, increased with size in kidneys of both sexes. Pb decrease with length in livers. Other relationships noted.	Wagemann et al. 1983
M. monoceros	K, L, M	Positive correlations with Cd and age in livers of males and kidneys of females, Hg and Se in all organs, Zn in kidneys of females.	Hansen et al 1990
Phocoena phocoena	K, L, O	Hg and Cd, but not Cu or Zn increase with body length in livers and kidneys.	Falconer et al. 1983
P. phocoena	Br, K, L, M	Hg increase with age.	Gaskin et al 1979
P. phocoena	K, L	Increase in total Hg, Se with age. No differences between sexes within age classes.	Teigen et al 1993
P. phocoena	K, L, M	Total Hg increase with body length, with MeHg decreasing as a proportion of total. No differences between sexes.	Joiris et al. 1991
Physeter macrocephalus	M	Hg lower in smaller, nonreproductive females than reproductive females No correlation between Hg and body length in females, negative correlation in males.	Cannella and Kitchener 1992
Stenella attenuata	K, L, M, O	Cd increase with age in multiple organs. Hg increase with age, higher in females.	André et al 1990, 1991b
S coeruleoalba	K, L, M	Increases in concentration with age for Fe, Pb, Ni, Cd, Hg in muscle, Pb, Ni, Cd, Hg in liver, Hg in kidney; Mn, Zn and Cu decreased with age in liver, Mn and Cu decreased with age in kidney. Complex changes seen at weaning and between calves and older animals.	Honda et al. 1983
S. coeruleoalba	Bone	Complex relations with various metals and age and developmental state. Total Hg increased with age.	Honda et al. 1986a
S. coeruleoalba	K, L, M, O	Hg increased with total length in liver and muscle. No difference between sexes.	Andre et al. 1991a, b
S. coeruleoalba	K, L, M, O	Cd concentrations increase markedly from birth to 1.5 yr in most organs, remaining constant to age 15, then increasing with age in kidney, liver, muscle. Zn concentrations in various organs did not change with age in adults; Cu decrease with age in liver, kidney. Pb, Ni increase with age in muscle, liver, Other changes noted	Honda and Tatsukawa 1983, Honda et al. 1983
S. coeruleoalba	Bone	Complex changes from fetal to old age noted in several elements. Cd, Hg, Pb, Zn increase with age in mature animals; MeHg constant. Pb in liver highest in mature males	Honda et al. 1986a
S. coeruleoalba	Muscle	Hg in muscle increase with age.	Arima and Nagakura 1979

Continued on next page

Table 10-4 continued

Group and Species	Organ	Relation with Age and Sex	References
S. coeruleoalba	Br, K, L, M	Hg increase with length.	Leonzio et al. 1992
Tursiops truncatus	K, L, M	Cd concentrations in kidneys positively correlated with total length. Cu in livers highest in neonates, decreasing with age.	Wood and Van Vleet 1996
Mysticete cetaceans			
Balaenoptera acutorostrata	L	Correlations between age and Cd, Fe, and Hg in livers. No relationships with Mn, Zn, Cu, Pb, Ni, Co. Only Fe differed between sexes, higher in females.	Honda et al. 1987
B. acutorostrata	K, L, M	Correlations between age and Cd in muscle, Hg, Se in liver.	Hansen et al. 1990
B. physalus	K, L, M	Total Hg and organic Hg in liver, total Hg in muscle increase with age. No differences between sexes.	Sanpera et al. 1993
B. physalus	K, L, M	Complex relationships that vary with location and sex for Cd, Cu, Zn in three tissues.	Sanpera et al. 1996
Polar bears, sea otters, and sirenians			
Dugong dugon	K, L, M	Complex relationships and interactions. Cu, Mn in liver and kidney negatively correlated with age. Zn, Cd increase with age in liver and kidney; Fe increase with age in liver and muscle, Ag with age in kidney, Co in liver. No relationships with age and Ag in liver, Fe and Co in kidney, Zn, Cu, Mn in muscle.	Denton et al. 1980
Trichechus manatus	K, L	Size and Cn in livers varied negatively, Cd in kidneys increase, whereas Fe in livers and Pb in livers and kidneys showed no relationship. Metal concentrations did not vary by sex.	O'Shea et al. 1984
Ursus maritimus	L	Effect of age and location on Cd, Hg, and Se concentrations. No effect of age on Ag, As, Ca, Cu, Fe, K, Mg, Mn, Na, P, Sr, Zn.	Norstrom et al. 1986
U. maritimus	L	Cd, Hg, Se increase with age in livers; K, Mn, Mg, and P decrease with age; Ag, Ca, Na no trend with age.	Braune et al. 1991
U. maritimus	Hair	Total Hg in hair not related to age or sex.	Born et al. 1991
U. maritimus	L, M	Hg in livers of adults higher than young in some areas. No differences by sex.	Lentfer and Galster 1987

B = blubber, Br = brain, K = kidney, L = liver, M = muscle; MeHg = methyl mercury; O = other.

stage, probably because of a higher absorption efficiency from milk and rapid bone growth phases in comparison to later ages (Honda et al. 1986a). Premature and normal pups of ringed seals do not differ in cadmium concentrations in hair (Hyvärinen and Sipilä 1984). Broad regional trends in cadmium concentrations in livers of polar bears have been noted across the Arctic (Norstrom et al. 1986).

Lead

Lead is a nonessential element that is well known for its toxicity in mammals. It is a major airborne contaminant. Some primary sources of introduction of lead to the environment are from production of storage batteries, automotive exhaust, combustion of fuel with lead additives, smelting, pigments in paints, water pipes (particularly in older plumbing systems), and lead arsenate used as an insecticide. Pathology of lead poisoning has been well documented for domestic animals and humans, with evidence of the latter extending back to the ancient Romans. Modern causes of lead poisoning include ingestion of lead paint fragments by children and consumption of lead-tainted illegal moonshine whiskey. A variety of sublethal behavioral effects of lead are known from laboratory studies, and chronic exposure can cause pathological disorders of the nervous system, gastrointestinal tract, renal system (correlated histopathologically by the presence of lead-based intranuclear renal inclusion bodies), and immunotoxicity. Lead exposure also interferes with the production of hemoglobin and red blood cells. The inhibition of enzymes (delta aminolevulinic acid dehydratase) in the biosynthesis of heme provides a well-known biochemical marker of lead exposure (Peakall 1992).

Lead has generally not been found in marine mammal tissues at levels that are cause for concern. Most reports are of concentrations in soft tissues such as liver, kidney, and muscle (Appendices 5 to 8). The liver and kidneys of marine mammals tend to have higher lead residue concentrations than muscle, blubber, or other soft tissues. There is no clear pattern across studies that examine the relative amounts of lead in liver versus kidney of marine mammals. In most studies, concentrations in these tissues are less than 1 ppm (wet weight) and in nearly all cases are within the normal ranges

Table 10-5. Examples of Unusually High Concentrations (ppm) of Cadmium, Mercury, and Lead Reported in Soft Tissue of Marine Mammals

Group and Species	Cadmium	Mercury	Lead	References
Pinnipeds				
Arctocephalus gazella	684 (K, D)			Malcolm et al. 1994
Callorhinus ursinus	568 (K, D)		1.8 (K, W) 14.8 (L, D)	Anas 1974a, Goldblatt and Anthony 1983
Erignathus barbatus		420 (L, W)		Smith and Armstrong 1975
Halichoerus grypus		1097 (L, W)	7.0 (L, W)	Law et al. 1991, Simmonds et al. 1993
Odobenus rosmarus	458 (K, D)		11.6 (K, D)	Warburton and Seagars 1993
Phoca hispida	608 (K, D)			Wagemann 1989
P. vitulina		>700 (L, W)	2.3 (L, W)	Duinker et al. 1979, Reijnders 1980, Roberts et al. 1976
Zalophus californianus	569 (K, D)	240 (L, W)	3.0 (K, W)	Braham 1973, Buhler et al. 1975, Martin et al. 1976
Odontocete cetaceans				
Delphinus delphis			3.5 (L, W)	Kuehl et al. 1994
Delphinapterus leucas	275 (K, D)	756 (L, D)	2.1 (L, D)	Wagemann et al. 1990
Globicephala melanea	425 (K, D)	626 (L, D)		Meador et al. 1993
Kogia breviceps	412 (K, W)			Marcovecchio et al. 1990
Monodon monoceros	800 (K, D)			Wagemann et al. 1983
Phocoena phocoena			4.3 (L, W)	Law et al. 1991
Pseudorca crassidens	106 (K, W)	728 (L, W)		Baird et al. 1989
Stenella coeruleoalba		1544 (K, W)	12.4 (K, D)	André et al. 1991a, Leonzio et al. 1992
Tursiops truncatus		13,150 (L, D)		Leonzio et al. 1992
Mysticete cetaceans				
Balaenoptera acutorostrata	115 (K, D)		2.6 (L, D)	Honda et al. 1987
B. physalus	209 (K, D)			Sanpera et al. 1996
Other				
Dugong dugon	308 (K, D)			Denton et al. 1980
Trichechus monatus	190 (K, D)		7.1 (K, D)	O'Shea et al. 1984
Ursus maritimus			1.6 (L, W)	Norheim et al. 1992

K = kidney; L = liver; D = dry weight basis; W = wet weight basis

seen in other mammals, including humans. Some of the higher concentrations reported in soft tissues of various species are provided in Table 10-5. Comparatively high concentrations reported in livers of a small number of harbor porpoises and one white-beaked dolphin (*Lagenorhynchus albirostris*) from Danish waters (to 5.3 ppm and 4.5 ppm wet weight, respectively) more than 20 years ago (Andersen and Rebsdorff 1976) have not been replicated. Similarly, early reports of high lead concentrations in livers of gray seals and harbor seals (to 17 and 12 ppm wet weight, respectively) around Great Britain (Holden 1975) have not been repeated in more recent sampling (Law et al. 1991).

Most studies of concentrations of lead in soft tissues of marine mammals show no consistent trend with age or sex (see references in Table 10-4). However, a few studies have correlated concentrations of lead in soft tissues with age: higher concentrations in liver and kidney of older harbor seals were reported by Drescher et al. (1977); decreases in lead concentrations with age were found in teeth of gray seals (Hepplestón and French 1973); lead concentrations in livers of pilot whales increased with body length (Meador et al. 1993) but not age (Muir et al. 1988a); and lead in livers of harbor seals increased with age in males, but not in females or in kidneys of either sex (Miles et al. 1992). The most detailed study to show a trend in lead concentrations in soft tissues with age was conducted in striped dolphins. Lead concentrations in muscle, liver, and kidney increased with age until about 1 year, leveled off up to 18 years, then increased in older age and were highest in the oldest individuals (Honda et al. 1983).

Bone is the prominent depot for long-term storage of lead in vertebrates, and the highest concentration of lead known in tissues of marine mammals is 61.6 ppm (wet weight) in bone of a young bottlenose dolphin stranded on the edge of Spencer Gulf, Australia, an area known for emissions from a lead smelter (Kemper et al. 1994). A maximum of 62.8 ppm (dry weight) reported for the humerus of a California sea lion was considered comparable to values in normal human bone (Braham 1973). Bones of most other marine mammals exam-

ined have had far less lead and, although generally negligible concentrations are reported in marine mammal bone, the number of studies on this topic has been limited. Amounts present are usually higher than those in soft tissues (Braham 1973, Roberts et al. 1976, Goldblatt and Anthony 1983, Kemper et al. 1994). Honda et al. (1984) noted that, although bone of striped dolphin represented only 4% of the body weight, nearly 13% of the body burden of lead was in the skeleton, with certain bones having higher concentrations than others. Lead in female striped dolphin bone occurred at lower concentrations than in males, and lead accumulated most rapidly during the suckling period (Honda et al. 1986a). There are no published studies indicating histopathological damage or biochemical lesions caused by lead exposure in marine mammals. One account of a possible case of lead poisoning in an unspecified marine mammal (but without documentation of lead concentrations in tissues) was suggested by Britt and Howard (1983). Smith et al. (1990, 1992) used isotopic ratios to show that, although lead concentrations in teeth of sea otters have not changed between modern and preindustrial eras, the source of lead has shifted from naturally derived lead to anthropogenic aerosol-dominated forms.

Mercury

Long recognized for its poisonous effects, mercury is one of the few nonessential elements that shows appreciable biomagnification in marine food webs and has a relatively low threshold for toxicity. Despite the latter, 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, combustion of fossil fuels, manufacturing of paper, and chlor-alkali plants. It is intentionally used as a fungicide, particularly as a seed dressing. Much mercury in the environment is also released through natural processes, and certain regions have higher mercury levels from geological sources. Mercury can also be highly volatile. Natural mercury brought to the sea surface from cold upwellings 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 soils convert various organic and inorganic forms of mercury to dimethyl (CH_3-Hg-CH_3) or methyl forms. The highly toxic methyl mercury ion (CH_3-Hg^+ or MeHg) is soluble in water, taken up by organisms, and can biomagnify several orders of magnitude in the food chain.

Mercury is best known for its neurotoxic effects and was once used in the production of felt hats, which inspired the character of the "Mad Hatter" in Lewis Carroll's *Alice in Wonderland*. It is also nephrotoxic, immunotoxic, mutagenic, crosses the placenta, and is found in milk. In recent history there have been notable cases of human mercury poisoning. In the 1950s and 1960s, people in the area of Minamata Bay and Niigata Bay in Japan suffered an epidemic of paralysis and mortality that was traced to seafood contaminated with mercury from effluents of chlor-alkali plants. In these instances, mercury concentrations in fish were up to 2000 times that of mercury concentrations in the surrounding water. Mass poisonings also occurred in recent history in Iraq, where 459 deaths were attributed to ingesting bread in which flour was made from mercury-treated seed. In comparison to other forms of mercury, methyl mercury is readily absorbed by the gastrointestinal tract. Mercury accumulates in the kidneys, liver, and brain (particularly the methyl form), but the organ distribution and toxicity also varies with the chemical form. In marine mammals, most chemical analyses have determined the concentrations of total mercury in tissues rather than the more toxic methyl mercury fraction (Appendices 5 to 8). A few studies report "organic mercury" (not strictly equivalent to methyl mercury) as a fraction of the total mercury.

One of the most outstanding cases of interactions between toxic elements is the apparent protective effect of selenium against mercury toxicity. Laboratory studies of a number of organisms 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, but these concentrations are typically accompanied by increased selenium in the liver in a 1:1 molar ratio (Koeman et al. 1973, 1975). The interactions and molecular level complexities of this protective effect are not well understood (Lee et al. 1977, van de Ven et al. 1979, Cuvin-Aralar and Furness 1991), but may include redistribution of mercury away from sensitive organs (such as the kidney) to muscle and other tissue. In the absence of selenium, mercury is bound to metallothionein proteins (see section on cadmium), which detoxify mercury, but also may cause long-term mercury retention. Selenium apparently diverts binding of mercury away from metallothionein to higher molecular weight proteins. Very little of the mercury in sea lion livers, for example, was bound to metallothionein (Lee et al. 1977), and no significant correlation exists between metallothionein and mercury in livers in harbor seals (Tohyama et al. 1986). Some of the proposed mechanisms for the protective effect of selenium against mercury have been reviewed by Cuvin-Aralar and Furness (1991), and include redistribution of mercury, competition for binding sites, formation of a mercury–selenium complex (described in cetacean livers

by Martoja and Viale 1977), conversion of toxic forms of mercury to more benign forms, and prevention of oxidative damage.

Mercury determinations in tissues of marine mammals typically focus on the liver, although orher tissues and organs sometimes examined include kidney, muscle, blubber, and hair (Appendices 5 to 8). Concentrations of total mercury are usually higher in liver than in kidney, muscle, or other tissues and have often been shown to increase significantly in liver with age (although the proportion that is methyl mercury decreases with age) (Table 10-4). Some of the concentrations of total mercury found in livers of marine mammals are far in excess of those rhat would be toxic to other mammals, but lethal effects have generally not been observed. This is apparently because of the metabolic capacity of marine mammals to protectively 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. Examples of extraordinarily high concentrations of total mercury in livers of marine mammals without evidence of accompanying toxicity include cases where mercury reached hundreds and sometimes thousands of parts per million (Table 10-5). In humans poisoned at Minamata Bay, in contrast, mercury in livers ranged from 22 to 70 ppm, but unlike in marine mammals, nearly all was methyl mercury (Britr 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. 1991a, 1991b; Leonzio et al. 1992; Kemper et al. 1994). Marine mammals that typically feed lower in the food chain, such as baleen whales and sirenians, have very low mercury concentrations in liver in comparison with piscivorous species (Denton and Breck 1981; O'Shea et al. 1984; Byrne et al. 1985; Honda et al. 1986b, 1987; Dietz er al. 1990; Sanpera et al. 1993). Unlike organochlorines in blubber, which are usually highest in adult males, mercury concentrations in livers of marine mammals generally show no differences between sexes or are higher in females (Table 10-4). Concentrations of mercury in tissues of some marine mammals could pose human health risks in areas where people consume organs from these animals (for example, see Botta et al. 1983, Andersen et al. 1987, Simmonds et al. 1994), but the potential risk relares to the extent to which mercury is methylated (Eaton et al. 1980).

Mercury–selenium correlarions have been derermined in tissues of numerous marine mammals, and results are consistent with a role for selenium in protection against mercury toxicity (Koeman et al. 1973, 1975; Cuvin-Aralar and Furness 1991). Such positive correlations have been noted, for example, in muscle, bone, livers, kidneys, and brains of striped dolphins (Arima and Nagakura 1979, Honda et al. 1986a, Leonzio et al. 1992); in kidneys, livers, and muscle of belugas (Wagemann et al. 1990); in livers and kidneys of harbor, harp, gray, ringed, and bearded seals (*Erignathus barbatus*) (Smith and Armstrong 1978, van de Ven et al. 1979, Reijnders 1980, Ronald et al. 1984b, Perttilä et al. 1986, Wagemann et al. 1988, Frank et al. 1992); in livers of bottlenose dolphins (Kuehl et al. 1994); in livers and kidneys of pilot whales (Julshamn et al. 1987, Muir et al. 1988a, Caurant et al. 1993, Meador et al. 1993); in livers and kidneys of white-beaked dolphins (Muir et al. 1988a); in livers of polar bears (Norstrom et al. 1986, Braune et al. 1991, Norheim et al. 1992); in livers and kidneys of walrus (Taylor et al. 1989, Warburton and Seagars 1993); and in livers and kidneys of narwhals (Wagemann et al. 1983). Many of these studies also verify the approximately 1:1 molar ratio of mercury to selenium in liver, suggestive of a protective effect of selenium (Koeman et al. 1975). The mercury-to-selenium ratios in fish (as prey species) differ dramatically from those seen in marine mammals (Koeman et al. 1973, 1975; Kari and Kauranen 1978). Numerous other studies have conducted analyses on relations between mercury and various other elements in organs of marine mammals (Appendices 5 to 8). The gross distribution of mercury within different parts of the liver has been determined for a few species and appears homogenous (Nielsen and Dietz 1990, Stein et al. 1992).

A detailed example of investigations on the distribution of mercury, methyl mercury, and selenium in marine mammal body compartments was conducted on striped dolphins (Itano et al. 1984a,b,c). Similar patterns revealed by these analyses have also been observed in other marine mammal species (see references in Appendices 5 to 8). Samples of 15 organs and tissues were analyzed from 55 individuals. Highest total mercury was found in the liver, but liver had the lowest proportion of methyl mercury. Selenium was also highest in the liver. Total mercury concentrations in muscle and liver increased with age, bur leveled off at age 20 to 25 years (total body burdens reached a constant level at age 16 years). No differences in total mercury concentrations were apparent in tissues or whole bodies of males and females. Selenium and mercury concentrations were significantly correlated in nearly all tissues and organs. In the striped dolphins, 90% of the entire body burden of methyl mercury was located in muscle. In feruses and calves of striped dolphins, most of the mercury was methylared, and in the term fetuses was about 1% of that in the pregnant female. Total mercury (most of which was merhylated) in milk was much lower than that in any other tissue. Transfer of mercury across the placenta, relatively low concentrations in milk, and high proportions

of methyl mercury in pup liver have also been reported in pinnipeds (Wagemann et al. 1988).

Unlike investigations on other metals in marine mammals, a few experimental studies have been conducted on toxicology of mercury in seals. Tillander et al. (1972) dosed a captive female ringed seal with radioactively labeled methyl mercury and determined a two-phased excretion rate. One component involved rapid excretion of about 55% of the mercury with a half-time of about 3 weeks, whereas the remainder was excreted with a half-time of 500 days. Ramprashad and Ronald (1977) administered methyl mercuric chloride at two dosage levels (0.25 and 25 mg/kg) to four harp seals and determined an effect of mercury exposure on sensory epithelium in the cochlea. Ronald et al. (1975, reported in Holden 1978) found that at the higher dosage level death occurred in 20 to 26 days due to renal failure in these seals, which showed increases in mercury concentrations in the brain and liver, as well as hepatitis and renal failure. Freeman et al. (1975) reported that in harp seals given 0.25 mg/kg methyl metcuric chloride for 61 days, more than 70% of the mercury in the liver was inorganic, demonstrating substantial demethylation. Muscle tissue had higher proportions of methyl mercury. In vitro synthesis of steroids in gonads and adrenals of harp and gray seals due to mercury exposure has also been reported (Freeman et al. 1975, Freeman and Sangalang 1977). Van de Ven et al. (1979) administered methyl mercuric chloride to captive gray seals and found that both mercury and selenium increased in livers and kidneys, but only mercury increased in other tissues. In vitro tests for several enzymatic demethylation mechanisms were negative, and no indication of demethylation processes by the microflora of the gut was found. Administration of methyl mercury also did not stimulate the P-450 enzyme system. Results suggested that the role of selenium in ameliorating mercury toxicity primarily involves tissue distribution and accumulation. However, Himeno et al. (1989) reported higher in vitro demethylation activity in liver and kidneys of harbor seals in comparison with laboratory rats and mice. Few studies of mercury contamination in marine mammals have included associated investigations of histopathology. Rawson et al. (1993, 1995) noted mercury-associated pigment granules and liver disease in bottlenose dolphins with relatively high concentrations of mercury in comparison to those without such conditions.

Other Elements

Various studies have provided data oti concentrations of up to 40 trace elements in tissues of marine mammals (Appendices 5 to 8). Most of these elements are currently of lesser concern as toxic contaminants in marine mammals than cadmium, lead, or mercury, chiefly because of their relatively low concentrations, their necessity as essential dietary elements, or because of an absence of information suggesting harmful effects at reported levels of exposure. Arsenic has been reported in numerous species of marine mammals, but at levels not considered toxic. Copper is an essential element that typically decreases with age in livers of marine mammals (Table 10-4). It has not been implicated as a potential threat in marine mammals except for a specific localized case where it was applied as an aquatic herbicide in a winter feeding ground of Florida manatees (O'Shea et al. 1984). In this case, elevated hepatic copper concentrations were found to correspond with geographic patterns of copper herbicide use (after adjusting for age), with maximum concentrations in liver equivalent to those associated with toxic effects in some sensitive terrestrial mammals. Unusually high concentrations of silver have been documented in the livers of Alaska beluga whales, positively correlated with selenium and age, but the toxicological significance of these findings is unknown (Becker et al. 1995). Similarly, livers of some Alaskan marine mammals appear to have elevated vanadium concentrations relative to marine mammals from other areas (Mackey et al. 1996). Most of the work on selenium in marine mammals has focused on its protective association with mercury, but this essential elemetit can be toxic to other mammals in its own right. Very little is known about interactions among other elements in marine mammals, but imbalances, particularly involving bromine, have been suggested as playing a role in premature parturition in California sea lions (Mattin et al. 1976). Certainly, much remains to be learned about the roles of potentially toxic elements in the health of marine mammals. Recent work, for example, has revealed the presence of organotins (butyltins) in blubber of eight species of marine mammals, with higher concentrations in individuals from coastal areas (Iwata et al. 1994). Unlike organochlorines, the butyltins have a greater affinity for tissues with a higher protein-binding capacity, such as liver and hair, than lipid-rich tissues (Kannan et al. 1996, Kim et al. 1996). Butyltins have been used in a variety of applications, including marine antifouling paints, pesticides, and wood preservatives. The possible toxic significance of their recently discovered (Iwata et al. 1994, Kim et al. 1996, Kannan et al. 1998) accumulation in tissues of marine mammals is undetermined.

Other Contaminants and Toxins

Air Pollutants

Marine mammals inhabiting urbanized coastal areas are subject to contamination from various air pollutants at levels similar to those experienced by large fractions of the human

population. However, little research has been conducted on potential impacts of air pollution on marine mammals. Rawson et al. (1991) noted the presence of carbon deposits in macrophages in mediastinal lymph nodes and lung tissue of Atlantic bottlenose dolphins from the west coast of Florida. Such deposits are also typical in humans and domestic animals in urban areas, and stem from inhaled carbon particles that enter the alveoli and are ingested by macrophages that pass to the mediastinal lymph nodes of the pulmonary lymph systems. No pathology was associated with these deposits in the limited number of cases observed in Florida. Mercuric selenide (tiemannite) granules in tissues of bottlenose dolphins and pilot whales from this area may possibly result from inhalation of atmospheric mercury (Rawson et al. 1995).

Aromatic and Polycyclic Aromatic Hydrocarbons

The aromatic and polycyclic aromatic hydrocarbons (AHs and PAHs) are often topics of investigation in marine pollution studies, but have not been an extensive focus of inquiry in marine mammals. These compounds can stem from numerous natural and anthropogenic sources, but as contaminants they are chiefly associated with components of petroleum. AHs have carbon atoms arranged in ring structures (1 to 6 rings with 6 carbon atoms per ring) and PAHs are AHs with up to 7 fused carbon rings that can have substitutions attached (Albers 1995). They include some well-known carcinogens such as benzo[*a*]pyrene. The PAHs and AHs do not show great biomagnification in food chains and are readily metabolized by many organisms. Hellou et al. (1991) found relatively low concentrations of PAHs in muscle tissue of 28 harp seals from the northwest Atlantic, and no accumulation with age. Low concentrations were also reported in muscle samples from smaller numbers of a wider range of species from the same region, including harbor, harp, hooded (*Cystophora cristata*), and ringed seals, and single beluga, sperm whale, and minke whales, common and white-sided dolphins (Hellou et al. 1990). Law and Whinnett (1992) also reported similarly low concentrations of PAHs in muscle tissue of 26 harbor porpoises from the coast of the United Kingdom. PAHs have been hypothesized to be responsible for tumors in belugas of the St. Lawrence River estuary through the formation of DNA adducts (e.g., Martineau et al. 1988), but this view is not fully accepted (Geraci et al. 1987), and DNA adducts have also been reported at similar levels from beluga whale livers in remote locations with negligible PAH contamination (Ray et al. 1992).

Dinoflagellate Toxins

Irruptions of toxin-producing marine dinoflagellates of various species occur throughout the world. Although not considered anthropogenic contamination, a seeming increase in irruptions worldwide is suspected to be associated with nutrient enrichment and other human activities (Anderson 1994). Some dinoflagellate blooms have been associated with mortality of fish, birds, and mammals, as well as human mortality and illness (particularly from ingesting shellfish and other seafood that have concentrated dinoflagellate toxins). The characteristics of poisoning vary with species of dinoflagellate, mode of exposure, and chemical characteristics of the toxins produced. The resulting toxic syndromes (known best from human exposure) fall into four categories: neurotoxic shellfish poisoning, paralytic shellfish poisoning (PSP), diarrhetic shellfish poisoning, and ciguatera poisoning (Baden 1983, Steidinger and Baden 1984). Most symptoms are neurological or gastrointestinal. Mortality of bottlenose dolphins and manatees has been associated with blooms of the dinoflagellate *Ptychodiscus brevis* (also referred to as *Gymnodinium breve*) in Florida (Gunter et al. 1948, Layne 1965, O'Shea et al. 1991), which were also held responsible for the widely publicized manatee die-off of 1996 (Bossart et al. 1998). In the latter case, manatees may have been exposed to lethal amounts of brevetoxin through ingestion of contaminated food or water or perhaps through inhalation of aerosols released during lysis of cells by wind and wave action at the water surface (severe lesions were present in the lungs of many manatees). Deaths attributable to toxins produced by other dinoflagellate species have also been observed in humpback whales (Geraci et al. 1989) and sea otters (De Gange and Vacca 1989). In the case of the humpback whales, the agent was saxitoxin produced by the dinoflagellate *Alexandrium tamarense*, a substance that can accumulate in food chains and had built up in mackerel ingested by the humpbacks. Experimental feeding studies have demonstrated that sea otters will selectively avoid the most toxic portions of prey clams with high quantities of PSP toxins (Kvitek et al. 1991).

Radionuclides

Ionizing radiation can produce a broad spectrum of injuries to mammals at the molecular, cellular, organ, and organismal levels, including effects on behavior, growth and development, and mutagenicity and carcinogenicity (for a review, see Eisler 1994). Anthropogenic radionuclides that contaminate today's ecosystems come primarily from fallout from nuclear weapons testing (which peaked 30 to 50 years ago), the Chernobyl accident in 1986, nuclear reactor operations, nuclear fuel processing and disposal, and applications in medicine, industry, agriculture, and research. Only a few studies have examined marine mammals to determine the extent of their contamination by radionuclides, and none has

reported any associated effects. Anderson et al. (1990) examined milk and tissues of gray seals collected in 1987 from the North Sea and North Atlantic for cesium-137 (^{137}Cs) and the actinides plutonium and americium. Levels of ^{137}Cs were low in milk and tissues and about 70% was attributed to the nuclear reprocessing industry in England, with the remainder from the Chernobyl accident. Actinides were barely detectable. Anderson et al. (1990) concluded that there was no evidence for extensive concentration of radionuclides from fish prey and that the radiation doses received by seals were below the limit for human radiation workers, but likely higher than limits set for the general public. Calmet et al. (1992) collected muscle and liver tissues from spotted, spinner, and common dolphins from the eastern Pacific and determined ^{137}Cs concentrations as well as ^{40}K and ^{210}Pb. These investigators concluded that the concentration factor from seawater was about the same as in fish, and that the radiation doses measured are unlikely to have effects on dolphin populations. Other marine mammals in which radionuclides have been measured include fin whales (Osterberg et al. 1964, Samuels et al. 1970), harp seals (Samuels et al. 1970), sperm whales, spotted seals, and bearded seals (Holtzman 1969, cited in Eisler 1994).

Conclusions and Future Research Needs

Interpreting the significance of the presence of contaminants in marine mammal tissues is a difficult and sometimes controversial area. However, the reader should bear in mind that the published literature summarized in the appendices to this chapter includes results of organochlorine and toxic element residue surveys from more than 13,000 samples of marine mammals. Laboratory analyses for determination of contaminants are expensive; these results represent a cumulative investment by society of many millions of dollars. An enormous amount of information has been learned from these surveys about fundamental patterns of variation in contaminant residues in marine mammals. Some of the organochlorines, for example, are known to accumulate differentially in the most lipid-rich tissues, and to increase with age in males but not in reproductive females who largely shunt these substances to their young through lactation. DDE and PCBs are the most commonly reported organochlorines. PCBs vary widely in individual persistence and resistance to metabolism. Global transport of organochlorines has been verified, and these substances show up in the bodies of marine mammals, albeit at low concentrations, in some of the most remote areas of the world's oceans. In more inshore species in some of the most heavily contaminated environments, an alphabet soup of different organochlorines can be detected, some at incredibly high concentrations, particularly in species highest in the food web.

The study of organochlorines is complex, however, and analyses that focus on residues alone, although expensive, leave much to be desired for interpretation of significance to the health and dynamics of exposed populations. In addition, many studies have small sample sizes. Despite a considerable investment in research, no marine mammal deaths in the wild have conclusively been shown to be a direct result of organochlorine or toxic element exposure. Indirect effects have been difficult to pinpoint. Reproductive impairment and gross lesions *associated with* organochlorine contaminants have been convincingly demonstrated in a few highly polluted areas, and experimental cause-and-effect studies have been carried out in only one species. There is mixed evidence for linkages with increased susceptibility to disease, and the complex study of biochemical and physiological effects in marine mammals is in its infancy. Although there is a lack of absolute scientific certainty in linking the presence of specific organochlorine contaminants to detrimental impacts on marine mammal populations, the body of indirect, circumstantial evidence reviewed in this chapter continues to grow. Organochlorine pollution of the seas will continue, particularly from PCBs, and efforts to abate contamination without waiting for more "smoking guns" can only benefit marine mammal conservation in the long run.

The study of toxic elements has included determination of residue concentrations from thousands of individuals in numerous species. This work has revealed patterns of variation that include relationships among metals, such as the protective linkage between selenium and mercury, organ-specific sites of accumulation for certain metals, and age-dependent accumulation (cadmium in kidneys) or loss (copper in livers) of some elements. Some elements can be found in very high concentrations in marine mammal tissues because of high, naturally occurring geologic sources (for example, mercury in the Mediterranean Sea) or relatively high amounts in prey (for example, cadmium in squid). However, most of the mercury found in marine mammal livers is not in the toxic methylated form, and although mercury may sometimes occur at concentrations in livers of marine mammals that exceed toxic amounts in other species, it appears to be detoxified and no harmful effects have been conclusively demonstrated. Similarly, higher concentrations of cadmium in tissues of marine mammals are protectively bound by metallothionein proteins, and no associated pathological effects of cadmium are known from these animals. Very little is known about sources of variation, interactions and effects of most of the other potentially toxic elements found in tissues of marine mammals. Unlike organochlorines, most research has not gone beyond the study of residue concentra-

tions, and little detail is known about relationships among metals and marine mammal physiology, disease, or population dynamics. Knowledge of other contaminants and toxins in marine mammals is even less comprehensive although, unlike anthropogenic chemicals, dinoflagellate biotoxins have been more firmly established as sources of direct mortality and morbidity of marine mammals.

To have a maximum impact on knowledge, future research on the presence of contamiuant residues in marine mammals needs to emphasize obtaining sufficiently large sample sizes to partition variation in residue concentrations among such potentially significant sources as age, sex, location, reproductive status, feeding habits, and uutritional status. In addition, ro allow for adequate iuterpretation, contaminant studies should include corollary research such as gross pathology and histopathology, analysis of reproductive tracts, and evidence of biochemical lesions such as MFO activity, circulating levels of hormones, and orher biomarkers. These data should be collected from the same individuals for which contaminant concentrations are determined. Knowledge of overall habitat quality and the status of the population (increasing or declining, harvested or unharvested) from which samples are obtained is also useful for interpretation of contaminant data. These requirements are a tall order and would have greatest chances of success in establishing associations between contaminant exposure and biological effects if carried out simultaneously in areas of contrasting contamination. Because of sample size requirements, advantage should be taken of obtaining fresh samples from by-catches of fisheries or from harvested populations. As an alternative approach, utilization of biopsy sampling techniques (Aguilar and Borrell 1994c) would be of particular value if combined with studies that emphasize longitudinal record keeping on reproductive histories of some of the same individuals (see Wells, Boness, and Rathbun, Chapter 8, this volume) or where detailed observational follow-up is possible, as in the case of pinniped breeding colonies. These suggestions notwithstanding, impacts of contaminants on marine mammals would most conclusively be determined through carefully controlled captive feeding experiments. Thus far, expense and ethical–political considerations have prevented such research in all but a very few cases.

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Appendices

The lirerature on environmental contaminants in marine mammals spans numerous disciplines, making access difficult to those beginning work in this field. Sources include journals in the fields of chemistry, biochemistry, environmental sciences, physiology, toxicology, wildlife biology, marine mammalogy, and other areas of study. For ease of entry into this literature, I have tabulated information from most of the published references in this field that have appeared during the past 30 years (through most of 1996). Tables can be used to find information on various species, regions of the world, and types of contaminants, organs, and numbers of individuals investigated. I hope these tabulations prove useful in planning future studies as well as in making comparisons with completed work. Original sources should be obtained for more detailed observations and interpretation. Species names are as used in the orignal publications and may differ from those provided in Chapter 1.

Appendix 10-1. Summary of Selected Organochlorine Residue Surveys in Pinnipeds

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
Arctocephalus forsteri	New Zealand (captive)	—	L	5	PCBs, DDE, DDT, TDE, ∑DDT	Solly and Shanks 1976; Lock and Solly 1976
A. gazella	Antarctica	1984–85	Mi	3	PCB congeners, DDE, *o,p'*-DDE, DDT, *o,p'*-DDT, TDE, *o,p'*-TDE, chlordanes, heptachlor epoxide, dieldrin	Bacon et al. 1992
A. gazella	Antarctica	1987	B	11	PCB congeners, PCDDs, PCDFs	Oehme et al. 1995b
A. pusillus	Southeastern Australia	—	B, L, M, O	11	DDT, TDE, DDE, ∑DDT, PCB, HCB, HCH	Smillie and Waid 1987
Collorhinus ursinus	Alaska	1981	Mi	7	PCB congeners, DDE, *o,p'*-DDE, DDT, *o,p'*-DDT, TDE, *o,p'*-TDE, chlordanes, heptachlor epoxide, dieldrin	Bacon et al. 1992
C. ursinus	Alaska	1990	B, L	2	∑DDT, PCBs	Varanasi et al. 1992, 1993b
C. ursinus	Alaska (Pribilof Islands)	1990	B, Br, L, Lu, Mi	4	HCHs	Mösstier et al. 1992
C. ursinus	Bering Sea (Alaska)	1987	B, K, L, M	2	DDT, *o,p'*-DDT, TDE, DDE, *o,p'*-DDE, ∑DDT, PCBs, PCB congeners, HCB, HCHs, heptachlor epoxide, chlordane, *t*-nonachlor, dieldrin	Schantz et al. 1993

Appendix 10-1 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
C. ursinus	Eastern North Pacific (Alaska, Washington)	1968–69	Br, L	30	DDE, TDE, DDT, dieldrin	Anas and Wilson 1970
C. ursinus	Eastern North Pacific (Southern California)	1979–80	L	4	DDE, TDE, DDT, ∑DDT, PCBs	Britt and Howard 1983
C. ursinus	Eastern North Pacific (Washington)	1972	B	12	∑DDT plus PCBs combined	Anas and Worlund 1975
C. ursinus	Japan	1971–88	B	105	DDE, DDT, TDE, ∑DDT, PCBs, PCB congeners, HCHs	Tanabe et al 1994b
C. ursinus	North Pacific (Pribilof Islands; Washington)	1968–69	B, Br, L, M, Mi	37	DDE, TDE, DDT, dieldrin	Anas 1971
C. ursinus	Pribilof Islands (Alaska)	1972	B, O	7	DDE, TDE, DDT, *o,p′*-DDT, *o,p′*-TDE, ∑DDT, dieldrin, PCBs	Kurtz and Kim 1976
Cystophora cristata	Greenland	1972	B	5	DDE, PCBs, heptachlor epoxide, aldrin, lindane	Clausen et al. 1974; Clausen and Berg 1975
C. cristata	Gulf of St. Lawrence (Canada)	1960s	B	1	DDE, TDE, DDT, PCBs, dieldrin	Holden 1970
Erignathus barbatus	Arctic (Canada)	1971–72	Fat	2	DDE, TDE, DDT, PCBs	Bowes and Jonkel 1975; Bowes and Lewis 1974
E. barbatus	Greenland	1972	B	5	DDE, PCBs, heptachlor, heptachlor epoxide, aldrin, lindane	Clausen et al. 1974; Clausen and Berg 1975
Eumetopias jubatus	Japan	1990	B	4	DDE, DDT, TDE, ∑DDT, PCBs, PCB congeners, HCHs	Tanabe et al. 1994b
E. jubatus	Alaska	1985–90	B, L	8	∑DDT, PCBs	Varanasi et al. 1992, 1993b
E. jubatus	Alaska, Bering Sea	1976–81	B, L	43	DDE, TDE, DDT, ∑DDT, HCHs, HCB, chlordanes, nonachlors, oxychlordane, PCBs	Lee et al. 1996
E. jubatus	Japan	1994	B, K, L, M, O	1	∑DDT, PCBs, HCHs, chlordanes	Kim et al. 1996
Halichoerus grypus	Baltic Sea	1969–73	B	60	∑DDT, PCBs	Olsson et al. 1975
H. grypus	Baltic Sea	1991	B, L	1	∑DDT, PCBs, methyl sulfones of PCB congeners and DDE	Bergman et al. 1994
H. grypus	Baltic Sea (Finland)	1981–87	B	7	PCB congeners, PCDFs, PCDDs, PCNs, hexachlorinated anthracenes	Koistinen 1990
H. grypus	Baltic Sea (Finland)	1985–89	B	1	∑DDT, HCH, lindane, HCB, chlordane, toxaphene, PCN, PCBs, PCB congeners, dibenzofurans	Paasivirta and Rantio 1991
H. grypus	Baltic Sea (Gulf of Finland)	1976–82	B, K, L, M	9	DDT, TDE, DDE, ∑DDT, PCBs, chlordanes, trans-nonachlor, oxychlordane	Perttilä et al. 1986
H. grypus	Baltic Sea (Sweden)	1968	B, L, Mi	8	DDT, ∑DDT, PCBs	Jensen et al. 1969
H. grypus	Baltic Sea (Sweden)	1979–87	B	—	PCDDs, PCDFs	deWit et al. 1992
H. grypus	Baltic Sea (Sweden)	1976	Fat	3	PCBs, PCTs	Renberg et al. 1978
H. grypus	Baltic Sea (Sweden)	1974–77	B	5	∑DDT, PCBs, chlordanes, HCHs, toxaphene	Jansson et al. 1979
H. grypus	Baltic Sea (Sweden)	—	B, K, L, other	5	DDE, PCBs, phenolic metabolites	Jansson et al. 1975
H. grypus	Baltic Sea (Sweden)	1979–90	B	37	∑DDT, PCBs, PCB congeners, methyl sulfones of PCBs and DDE	Blomkvist et al. 1992; Haraguchi et al. 1992
H. grypus	Britain	—	B, Br, K, L, M, O	25	∑DDT, PCBs, dieldrin	Heppleston 1973
H. grypus	Eastern North Atlantic (Norway)	1977–79	B, L	12	∑DDT, PCBs, HCB, HCHs	Ofstad and Martinsen 1983

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Appendix 10-1 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
H. grypus	England	1960's	B, L	13	DDE, TDE, DDT, PCBs, dieldrin	Holden 1970
H. grypus	England	1988–89	B, K, L	8	DDE, TDE, DDT, dieldrin, HCH, HCB, PCBs	Simmonds et al. 1993
H. grypus	Gulf of St. Lawrence (Canada)	1960's	B	7	DDE, TDE, DDT, PCBs, dieldrin	Holden 1970
H. grypus	North Atlantic (Wales)	1988	B, K, L, M	2	HCB, HCHs, dieldrin, DDE, DDT, TDE, PCBs, PCB congeners	Morris et al. 1989
H. grypus	North Sea (England)	1965	L	1	DDE, dieldrin	Robinson et al. 1967
H. grypus	North Sea (England)	1972	B, L	189	DDE, TDE, DDT, ∑DDT, PCBs, dieldrin, total organohalogens	Donkin et al. 1981
H. grypus	North Sea (England)	1988	B	3	DDE, TDE, DDT, PCBs, PCB congeners, dieldrin, HCHs, HCB	Law et al. 1989
H. grypus	North Sea (Scotland)	1988	B	7	∑DDT, PCBs	Blomkvist et al. 1992
H. grypus	Northeast Atlantic (France)	—	B, K, L, M	4	∑DDT, PCBs	Alzieu and Duguy 1979
H. grypus	Northwest Atlantic (Nova Scotia)	1987	B, Mi	25	PCBs	Schweigert and Stobo 1994
H. grypus	Norway	1991	Blood	17	DDE, DDT, ∑DDT, PCBs, PCB congeners	Jenssen et al. 1994
H. grypus	Nova Scotia	—	B	38	PCBs	Addison et al. 1988
H. grypus	Scotland	1960s	B	31	DDE, TDE, DDT, PCBs, dieldrin	Holden 1970
H. grypus	Scotland	1965–67	B, Br, K, L, M, O	—	DDE, TDE, DDT, ∑DDT, dieldrin	Holden and Marsden 1967
H. grypus	Western North Atlantic (Canada)	1967	B	—	DDE, TDE, DDT, dieldrin	Holden and Marsden 1967
H. grypus	Western North Atlantic (Nova Scotia)	1982	B	16	DDE, TDE, DDT, ∑DDT, PCBs	Addison et al. 1984
H. grypus	Western North Atlantic (Nova Scotia)	1984–85	B, O	30	DDE, DDT, ∑DDT, PCBs, PCB congeners	Addison and Brodie 1987
Hydrurga leptonyx	Australia	—	B	1	ODE, DDT, PCBs, oxychlordane, heptachlor epoxide, HCB	Kemper et al. 1994
H. leptonyx	New Zealand (captive)	—	L	1	PCBs	Solly and Shanks 1976
Leptonychotes weddelli	Antarctic	1981	B	1	∑DDT, PCBs, HCHs	Tanabe et al. 1983
L. weddelli	Antarctic	1981	B	3	∑DDT, PCBs, *cis*-chlordane, *cis*-nonachlor, *t*-nonachlor, oxychlordane	Kawano et al. 1984
L. weddelli	Antarctica	1980–82	B, Br, K, L, M, O	5	∑DDT, PCBs, PCB congeners, *cis*-chlordane, *t*-chlordane, *cis*-nonachlor, *t*-nonachlor, oxychlordane	Kawano et al. 1986, 1988; Hidaka et al. 1983
L. weddellii	Antarctica	—	B	1	toxaphene	Vetter et al. 1992
L weddellii	Antarctica (McMurdo Sound)	1965–67	Fat	20	DDE, DDT	Brewerton 1969
L weddellii	Antarctica (Ross Island)	1964	Br, fat, K, L, M, O	16	DDE, DDT	George and Frear 1966
L. weddellii	Antarctic (Weddell Sea)	—	B	4	HCBs, HCHs, DDE, DDT, TDE, ∑DDT, PCBs, PCB congeners, toxaphene, chlordane	Luckas et al. 1990
L. weddellii	Ross Sea Antarctica	1965–67	Fat	20	DDT, DDE	Brewerton 1969
Lobodon carcinophagus	Ross Sea (Antartica)	1964	B, L	1	DDE, TDE, DDT, ∑DDT	Sladen et al. 1966

Appendix 10-1 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
Mirounga angustirostris	California	—	B, blood	—	PCB congeners, HCB, mirex, *t*-nonachlor, oxychlordane, heptachlor epoxide	Newman et al. 1994
M. angustirostris	California	1974–81	L	7	DDE, TDE, DDT, ΣDDT, PCBs	Britt and Howard 1983
M. angustirostris	California	1983–84	B, K, L, M	4	ΣDDT, PCBs	Schafer et al. 1984
M. angustirostris	California	1986–87	Mi	4	PCB congeners, DDE, *o,p'*-DDE, DDT, *o,p'*-DDT, TDE, *o,p'*-TDE, chlordanes, heptachlor epoxide, dieldrin	Bacon et al. 1992
Monachus monachus	Mediterranean (Greece)	1990	B, Br, K, L, M, O	1	PCBs, ΣDDT, HCHs	Cebrian Menchero et al. 1994
M. schauinslandi	Hawaii	—	B, K, L, Lu, O	1	DDE, PCBs, PCP	Takei and Leong 1981
Neophoca cinerea	Australia	1987	Mi	5	PCB congeners, DDE, *o,p'*-DDE, DDT, *o,p'*-DDT, TDE, *o,p'*-TDE, chlordanes, heptachlor epoxide, dieldrin	Bacon et al. 1992
Odobenus rosmarus	Arctic (Greenland)	1975, 1977	B	28	ΣDDT, DDE, DDT, PCBs	Born et al. 1981
O. rosmarus	Arctic (Baffin Bay, Canada)	—	B	4	PCBs, PCB congener ratios	Norstrom et al. 1992
O. rosmarus	Arctic (Canada)	1985–92	B	53	DDE, ΣDDT, PCBs, PCB congeners, dieldrin, mirex, oxychlordane, chlordanes, toxaphene, HCHs, CBz, PCDDs, PCDFs	Muir et al. 1995
O. rosmarus	Bering Sea	1981–84	B	53	Dieldrin, oxychlordane, 12 others analyzed but not found	Taylor et al. 1989
Ommatophoca rossi	Antarctica	1981–82	B	20	DDT, DDE, TDE, PCBs, dieldrin	McClurg 1984
Phoca groenlandica	Arctic (Greenland Sea)	1991	B, Br	11	DDE, TDE, DDT, ΣDDT, HCHs, HCB, PCBs, PCB congeners, PCDDs, PCDFs	Oehme et al. 1995b
P. groenlandica	Gulf of St. Lawrence (Canada)	1968	Mi	1	DDE, DDT	Cook and Baker 1969
P. groenlandica	Gulf of St. Lawrence (Canada)	1971	B	18	DDE, TDE, ODT, ΣDDT, PCBs, dieldrin	Addison et al. 1973
P. groenlandica	Gulf of St. Lawrence (Canada)	1971–73	B, Br, K, L, M, O	31	DDE, TDE, DDT, ΣDDT, PCBs, dieldrin, HCBs	Rosewell et al. 1979
P. groenlandica	Gulf of St. Lawrence (Canada)	1972–73	B, Br, K, L	10	DDE, TDE, DDT, ΣDDT, dieldrin, PCBs	Jones et al. 1976
P. groenlandica	Gulf of St. Lawrence (Canada)	1982	B	22	DDE, TDE, DDT, ΣDDT, PCBs	Addison et al. 1984
P. groenlandica	Gulf of St. Lawrence and Hudson Strait (Canada)	1988–89	B	50	ΣDDT, *p,p'*-DDE, PCBs, PCB congeners	Beck et al. 1994
P. groenlandica	North Atlantic (Canada)	1969–71	B, Br, L, M	78	ΣDDT, PCBs, dieldrin	Frank et al. 1973
P. groenlandica	Northwest Atlantic and Arctic oceans (Canada, Greenland)	1976–78	B, Br, K, L, M, O	248	ΣDDT, PCBs, dieldrin, chlordane, heptachlor epoxide, HCB	Ronald et al. 1984a
P. groenlandica	Western North Atlantic	1954–62	commercial oils	—	ΣDDT, DDE, TDE, DDT, dieldrin, PCBs	Addison et al. 1972
P. hispida	Arctic (Canada)	—	B	8	PCBs, PCB congeners	Ford et al. 1993
P. hispida	Arctic (Canada)	1960s	B	3	DDE, TDE, DDT, PCBs, dieldrin	Holden 1970

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Appendix 10-1 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
P. hispida	Arctic (Canada)	1970–72	Fat, L, M	11	DDE, TDE, DDT, *o,p*-DDT, PCBs	Bowes and Jonkel 1975; Bowes and Lewis 1974
P. hispida	Arctic (Canada)	1972	B	28	DDE, DDT, ∑DDT, PCBs	Addison and Smith 1974
P. hispida	Arctic (Canada)	1989–91	B	11	DDE, ΣDDT, PCBs, PCB congeners, dieldrin, mirex, oxychlordane, chlordanes, toxaphene, HCHs, HCBz, PCDDs, PCDFs	Muir et al. 1995
P. hispida	Arctic (Canada)	1992	B	1	PCBs, ∑DDT, HCHs, chlordane, toxaphene, PCB congeners	Zhu et al. 1995; Zhu and Norstrom 1993
P. hispida	Arctic (Norway)	—	B	11	HCBs, HCHs, DDE, DDT, TDE, ∑DDT, PCBs, PCB congeners	Luckas et al. 1990
P. hispida	Arctic (Norway)	1960s	B	2	DDE, TDE, DDT, PCBs, dieldrin	Holden 1970
P. hispida	Arctic (Baffin Bay, Canada)	—	B	4	PCBs, PCB congener ratios, PCDDs, PCDFs	Norstrom et al. 1992
P. hispida	Arctic (Canada)	1983–84, 1975–76, 1972	B, L	67	DDE, ∑DDT, PCBs, PCB congeners, HCHs, chlordanes, toxaphene, dieldrin, CBZ	Muir et al. 1988b
P. hispida	Arctic (Canada)	1983–84, 1985–86	B	78	PCDDs, PCDFs, PCBs, HCB	Norstrom et al. 1990
P. hispida	Baltic Sea	1969–73	B	33	∑DDT, PCBs	Olsson et al. 1975
P. hispida	Baltic Sea	1969–74	B	73	∑DDT, PCBs	Helle et al. 1976a; Olsson et al. 1975
P. hispida	Baltic Sea	1975	B	109	∑DDT, PCBs	Helle et al. 1976b
P. hispida	Baltic Sea (Finland)	1981–87	B	7	PCB congeners, PCDFs, PCDDs, PCNs, hexachlorinated anthracenes	Koistinen 1990
P. hispida	Baltic Sea (Finland)	1985–89	B	1	∑DDT, HCH, lindane, HCB, chlordane, toxaphene, PCN, PCBs, PCB congeners, dibenzofurans	Paasivirta and Rantio 1991
P. hispida	Baltic Sea (Gulf of Bothnia, Sweden)	1968	B	2	DDT, ∑DDT, PCBs	Jensen et al. 1969
P. hispida	Baltic Sea (Sweden)	1960s	B	1	DDE, TDE, DDT, PCBs, dieldrin	Holden 1970
P. hispida	Baltic Sea (Sweden)	1979–87	B	—	PCDDs, PCDFs	deWit et al. 1992
P. hispida	Baltic Sea (Sweden)	1980–88	B	17	∑DDT, PCBs, PCB congeners, methyl sulfones of PCBs and DDE	Blomkvist et al. 1992; Haraguchi et al. 1992
P. hispida	Chukchi Sea, Norton Sound (Alaska)	1988	B, K, L	4	DDT, *o,p'*-DDT, TDE, DDE, *o,p'*-DDE, ∑DDT, PCBs, PCB congeners, HCB, HCHs, heptachlor epoxide, chlordane, *t*-nonachlor, dieldrin	Schantz et al. 1993
P. hispida	Greenland	1972	B	5	DDE, PCBs, heptachlor, heptachlor epoxide, aldrin, lindane	Clausen et al. 1974; Clausen and Berg 1975
P. hispida	Lake Saimaa (Finland)	1977–81	B, K, L, M, O	14	DDE, TDE, DDT, ∑DDT, PCBs, chlordane, chlorophenols	Helle et al. 1983
P. hispida	North Sea (Germany)	—	B	1	DDE, DDT, TDE, ∑DDT, PCBs, lindane, dieldrin	Harms et al. 1977/78
P. hispida	North Sea (Germany)	1975	B	1	∑DDT, PCBs, dieldrin, lindane	Drescher 1978
P. hispida	Northern Quebec, Canada	1989–90	B	41	PCBs, chlordanes, nonachlors, oxychlordane, heptachlor epoxide, photoheptachlor	Zhu et al. 1995
P. hispida	Norway	1986	B	7	PCDDs, PCDFs, HCB, HCHs, DDE, TDE, DDT, ∑DDT, PCBs	Oehme et al. 1988, 1990
P. hispida	Norway	1990	B, L, K	13	PCBs, DDE, PCB congeners	Daelemans et al. 1993

Appendix 10-1 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
P. hispida	Western Arctic (Canada)	1981	B	31	DDE, DDT, ∑DDT, PCBs	Addison et al. 1986a
P. hispida	Western North Atlantic (Canada)	1967	B	—	DDE, TDE, DDT, dieldrin	Holden and Marsden 1967
P. largha	Alaska	1975	O	—	DDE, DDT, *o,p'*-DDT	Serat et al. 1977
P. largha	Japan	1991	B	4	DDE, DDT, TDE, ∑DDT, PCBs, PCB congeners, HCHs	Tanabe et al. 1994b
P. siberica	Lake Baikal	—	B	1	∑DDT, PCBs, chlordane, HCHs, toxaphene	Kucklick et al. 1993
P. siberica	Lake Baikal	—	B	1	DDE, TDE, DDT, *o,p'*-DDT, ∑DDT, PCBs, PCB congeners, HCB, HCHs, heptachlor, chlordanes, *t*-nonachlor, toxaphene	Kucklick et al. 1994
P. vitulina	Alaska	1989–90	B, L	9	∑DDT, PCBs	Varanasi et al. 1992, 1993b
P. vitulina	Baltic Sea (Sweden)	—	B	9	HCB, HCHs, DDE, TDE, DDT, ∑DDT, PCBs, PCB congeners	Luckas et al. 1990
P. vitulina	Baltic Sea (Sweden)	1979–89	B	55	∑DDT, PCBs, PCB congeners, methyl sulfones of PCBs and DDE	Blomkvist et al. 1992; Haraguchi et al. 1992
P. vitulina	Baltic Sea (Sweden)	1979–87	B	—	PCDDs, PCDFs	de Wit et al. 1992
P. vitulina	Britain	—	B, Br, K, L, M, O	5	∑DDT, PCBs, dieldrin	Heppleston 1973
P. vitulina	California	1970	B, Br, L, M, O	2	DDE, TDE, DDT	Shaw 1971
P. vitulina	Cook Inlet (Alaska)	1976–78	B	23	DDE, DDT, oxychlordane, PCBs, others analyzed but not detected	Miles et al. 1992
P. vitulina	Eastern North Atlantic (Norway)	1977–80	B, K, L, M, O	10	∑DDT, PCBs, HCB, HCHs	Ofstad and Martinsen 1983
P. vitulina	Eastern North Pacific (Southern California)	1976–80	L	4	DDE, TDE, DDT, ∑DDT, PCBs	Britt and Howard 1983
P. vitulina	Eastern North Pacific (U.S.A.)	1971	B	13	∑DDT + PCBs	Anas 1974b
P. vitulina	England	1960s	B	15	DDE, TDE, DDT, PCBs, dieldrin	Holden 1970
P. vitulina	England	1988	B, K, L	15	DDE, TDE, DDT, dieldrin, HCH, HCB, PCBs	Simmonds et al. 1993
P. vitulina	Iceland	1988–89	B	10	PCBs	Heidmann et al. 1992
P. vitulina	Netherlands	—	B	1	PCBs, mirex	ten Noever de Brauw et al. 1973
P. vitulina	Netherlands (captive)	1981–83	Blood, feces	—	PCB congeners	Storr-Hansen et al. 1995
P. vitulina	North Atlantic (U.S.A.)	1980, 1990–92	B, L	15	DDE, mirex, HCB, α-chlordane, *trans*-nonachlor, PCBs, PCB congeners	Lake et al. 1995
P. vitulina	North Atlantic (Iceland)	—	B	7	HCB, HCHs, DDE, TDE, DDT, ∑DDT, PCBs, PCB congeners	Luckas et al. 1990
P. vitulina	North Atlantic (Bay of Fundy, Gulf of Maine)	1971	B, Br, L, M	12	DDE, TDE, DDT, *o,p'*-DDT, ∑DDT, dieldrin, PCBs	Gaskin et al. 1973
P. vitulina	North Sea (Denmark)	1988–91	B, Br, Li, M, K, O	5	DDE, HCB, PCBs, PCB congeners	Storr-Hansen and Spliid 1993b
P. vitulina	North Sea (Denmark)	1988	B	21	DDE, HCB, PCBs, PCB congeners	Storr-Hansen and Spliid 1993a
P. vitulina	North Sea (England)	1988	B	10	DDE, TDE, DDT, PCBs, PCB congeners, dieldrin, HCHs, HCB	Law et al. 1989
P. vitulina	North Sea (Germany)	—	B, Br, K, L	70	DDE, DDT, TDE, ∑DDT, PCBs, lindane, dieldrin	Drescher et al. 1977; Harms et al. 1977/78

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Appendix 10-1 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
P. vitulina	North Sea (Germany)	—	B	11	HCB, HCHs, DDE, TDE, DDT, ∑DDT, PCBs, PCB congeners	Luckas et al. 1990
P. vitulina	North Sea (Germany)	1988	B	1	toxaphene	Vetter et al. 1992
P. vitulina	North Sea (Germany)	1988	B	1	toxaphene	Kallenborn et al. 1994
P. vitulina	North Sea (Germany)	1988	B	5	DDE, TDE, DDT, HCHs, HCB, PCB congeners, PCDDs, PCDFs	Beck et al. 1990
P. vitulina	North Sea (Germany)	1988–90	B	27	PCBs	Heidmann et al. 1992
P. vitulina	North Sea (Germany, Denmark)	1975–76	B, K, L	16	DDE, TDE, DDT, ∑DDT, PCBs, dieldrin, HCHs, heptachlor epoxide, others analyzed but not detected	Reijnders 1980
P. vitulina	North Sea (Netherlands)	—	B, Br, K, L, O	8	DDE, TDE, DDT, *o,p'*-TDE, HCHs, PCBs, dieldrin, mirex	Duinker et al. 1979
P. vitulina	North Sea (Netherlands)	1960s	Fat, L	3	DDE, TDE, DDT, dieldrin	Koeman and van Genderen 1966
P. vitulina	North Sea (Netherlands)	1970–71	B	8	DDE, TDE, DDT, PCBs, dieldrin, HCB	Koeman et al. 1972
P. vitulina	North Sea (Netherlands)	1972–81	B	175	PCBs	Van der Zande and De Ruiter 1983
P. vitulina	North Sea (Netherlands)	1975–76	B, K, L	14	DDE, TDE, DDT, ∑DDT, PCBs, dieldrin, HCHs, heptachlor epoxide, others analyzed but not detected	Reijnders 1980
P. vitulina	North Sea (Netherlands)	1978	B, Br, L	3	∑DDT, PCBs, dieldrin, heptachlor epoxide, oxychlordane, *trans*-nonachlor	Kerkhoff et al. 1981; Kerkhoff and de Boer 1982
P. vitulina	North Sea (Norway)	1988	B, Br, K, L	10	PCB congeners	Bernhoft and Skaare 1994
P. vitulina	North Sea (U.K.)	1988–89	B	89	PCB congeners, DDE, TDE, DDT, ∑DDT, dieldrin	Hall et al. 1992
P. vitulina	Northern Ireland	1988	B, L, K	55	DDE, TDE, *o,p'*-DDT, DDT, ∑DDT, PCBs, HCB, HCHs, heptachlor, heptachlor epoxide, chlordanes, dieldrin	Mitchell and Kennedy 1992
P. vitulina	Puget Sound, U.S.A	1972–82	B	17	DDE, DDT, PCBs, heptachlor epoxide, HCHs, nonachlors, oxychlordane, chlordane	Walker et al. 1989
P. vitulina	Scotland	1960's	B	21	DDE, TDE, DDT, PCBs, dieldrin	Holden 1970
P. vitulina	Scotland	1965–66	B	—	DDE, TDE, DDT, dieldrin	Holden and Marsden 1967
P. vitulina	Scotland	1988	B	9	DDE, PCBs	Simmonds et al 1993
P. vitulina	Scotland	1988	B	1	PCB congeners	Wells and Echarri 1992
P. vitulina	Sweden	1969–73	B	8	∑DDT, PCBs	Olsson et al. 1975
Zalophus californianus	California	—	Fat, L	1	DDE	Theobald 1973
Z. californianus	California	1969	B, Br, L, M	11	DDE, TDE, DDT, dieldrin	Anas 1971
Z. californianus	California	1988	Mi	1	PCB congeners, DDE, *o,p'*-DDE, DDT, *o,p'*-DDT, TDE, *o,p'*-TDE, chlordanes, heptachlor epoxide, dieldrin	Bacon et al. 1992
Z. californianus	California	—	L	—	DDE	Hall et al. 1971
Z. californianus	California	1970	B, Br, L	20	∑DDT, PCBs	DeLong et al. 1973
Z. californianus	California	1970–81	L	69	DDE, TDE, DDT, ∑DDT, PCBs	Britt and Howard 1983
Z. californianus	California	1970	B, Br, L, M	25	DDE, TDE, DDT, ∑DDT	LeBouef and Bonnell 1971

Appendix 10-1 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
Z. californianus	California	1988–92	B	7	DDE, DDT	Lieberg-Clark et al. 1995
Z. californianus	Oregon	1970–73	Br, L, M, fat	19	DDE, TDE, DDT, ΣDDT, PCBs	Buhler et al. 1975

B = blubber; Br = brain; K = kidney; L = liver; Lu = lung; M = muscle; Mi = milk; O = other. Dashes appear where values were not available in original source.

CBZ = chlorobenzenes; DDE = 2,2,-*bis* (p-chlorophenyl)-1, 1-dichloroethylene; DDT = 2,2,-*bis*-(*p*-chlorophenyl)-1, 1-trichloroethane; ΣDDT = arithmetic summation of concentrations of isomers and metabolites of DDT; HCB = hexachlorobenzene; HCH = hexachlorocyclohexane; PCBs = polychlorinated biphenyls; PCDDs = polychlorinated dibenzo-*p*-dioxins; PCDFs = polychlorinated dibenzofurans; PCNs = polychlorinated napthalenes; PCP = pentachlorophenol; PCTs = polychlorinated terphenyls; TDE = 2,2,-*bis*-(*p*-chlorophenyl)-1, 1-dichloroethane.

Appendix 10-2. Summary of Selected Organochlorine Residue Surveys in Odontocete Cetaceans

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
Berardius bairdii	Western North Pacific (Japan)	1985	B	3	PCBs, PCB congeners, PCDFs, PCDDs	Kannan et al. 1989
B. bairdii	Western North Pacific (Japan)	1985	B	37	DDE, PCBs	Subramanian et al. 1988a
B. bairdii	Western North Pacific (Japan)	1985–89	B	3	HCHs	Tanabe et al. 1996
Cephalorhynchus commersonii	Southern Indian Ocean (Kerguelen Island)	1983	Melon	11	DDE, DDT, ΣDDT, PCBs, HCH, ratios, PCB congeners	Abarnou et al. 1986
C. heavisidii	Southwestern Indian Ocean (South Africa)	1977–87	B	9	ΣDDT, PCBs, HCB	de Kock et al. 1994
C. hectori	New Zealand	—	B	1	ΣDDT, PCBs, lindane	Baker 1978
C. hectori	New Zealand	1985–87	B	6	PCDDs, PCDFs	Buckland et al. 1990
Delphinapterus leucas	Arctic (Canada)	—	B	6	ΣPCBs, PCB congeners	Ford et al. 1993
D. leucas	Arctic Ocean (Canada)	1972	B, L, M	14	ΣDDT, DDE, TDE, DDT, *o,p'*-DDT, PCBs analyzed but not detected	Addison and Brodie 1973
D. leucas	Arctic Ocean (Canada)	1983–84	B	29	PCDDs, PCDFs, PCBs, HCB	Norstrom et al. 1990
D. leucas	Baltic Sea (Germany)	—	B, L	1	DDE, TDE, DDT, ΣDDT, PCBs, lindane, dieldrin	Harms et al. 1977/78
D. leucas	Canada (six locations)	1966–87	B, L, M	88	DDE, ΣDDT, PCBs, PCB congeners, ratios, HCH, toxaphene, dieldrin, mirex, chlordane, *trans*-nonachlor, HCB	Muir et al. 1990
D. leucas	Chukchi Sea (Alaska)	1989–90	B	4	DDT, *o,p'*-DDT, TDE, DDE, *o,p'*-DDE, ΣDDT, PCB congeners, HCB, HCHs, heptachlor epoxide, chlordane, t-nonachlor, dieldrin	Schantz et al. 1993
D. leucas	Hudson Bay (Canada)	1966	Oils	—	DDE, TDE, DDT, ΣDDT, dieldrin, PCBs	Addison et al. 1972
D. leucas	St. Lawrence River (Canada)	1982–85	B, K, L, Mi, O	26	ΣDDT, DDT, TDE, DDE, PCBs, PCB congeners	Martineau et al. 1987; Massé et al. 1986
D. leucas	St. Lawrence River (Canada)	—	B	10	PCBs, PCB congener ratios, PCDDs, PCDFs	Norstrom et al. 1992
D. leucas	St. Lawrence River (Canada)	1988	B, L	1	ΣDDT, PCBs, methyl sulfones of PCB congeners and DDE	Bergman et al. 1994

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Appendix 10-2 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
D. leucas	St. Lawrence River (Canada)	1993–94	B	16	DDE, TDE, DDT, ΣDDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, dieldrin, mirex, toxaphene, CBz, HCHs, chlordanes, nonachlors, oxychlordane, heptachlor epoxide, TCP-methane, OCS, PCBs, PCB congeners	Muir et al. 1996b
D. leucas	St. Lawrence River (Canada), and Eastern Newfoundland	1987–90	B, L	38	ΣDDT, DDE, TDE, DDT, dieldrin, toxaphene, t-nonachlor, oxychlordane, mirex, TCP-methane, PCDD congeners, PCDF congeners, PCB congeners, CBz, HCHs	Muir et al. 1996a
Delphinus delphis	Atlantic coast (U.S.A.)	1986–88	B	4	DDE, dieldrin, HCB, mirex, chlordanes, lindane, heptachlor epoxide, PCB congeners, PCDD, PCDFs, PBBs	Kuehl et al. 1991
D. delphis	California	1974–76	B, Br, M	13	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs, dieldrin, HCB, heptachlor epoxide, *trans*-nonachlor, oxychlordane, *cis*-chlordane	O'Shea et al. 1980
D. delphis	Eastern North Atlantic (Spain)	1979	B	1	DDE, TDE, DDT, *o,p'*-DDT, ΣDDT	Aguilar 1984
D. delphis	Western North Pacific	1987	B	2	HCHs	Tanabe et al. 1996
D. delphis	Eastern North Pacific (California)	1978–84	B, Br, K, L, M	11	ΣDDT, PCBs	Schafer et al. 1984
D. delphis	France	—	B, K, L, M, O	25	ΣDDT, DDE, DDT, TDE, PCBs	Alzieu and Duguy 1979
D. delphis	Indian Ocean (South Africa)	1980–86	B, L	97	PCBs, ΣDDT	Cockcroft et al. 1990
D. delphis	Japan	1968–75	B, M	2	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs, dieldrin, HCB, toxaphene, heptachlor epoxide, *trans*-nonachlor, endrin, mirex	O'Shea et al. 1980
D. delphis	Mediterranean Sea (France)	1977	B	1	DDE, TDE, DDT, HCH, PCBs	Vicente and Chabert 1978
D. delphis	New Zealand (captive)	—	L	3	PCBs, DDE, TDE, DDT, ΣDDT	Solly and Shanks 1976; Lock and Solly 1976
D. delphis	North Sea (England)	1965	B, L	1	DDE, dieldrin	Robinson et al. 1967
D. delphis	North Sea (Netherlands)	1979	B, Br, K, L, M, O	1	DDE, TDE, DDT, dieldrin, HCB, HCHs, PCBs, PCB congeners	Duinker et al. 1989
D. delphis	North Atlantic (U.S.A.)	1971–75	B	1	PCBs, chlordane, dieldrin, DDE, TDE, DDT, ΣDDT	Taruski et al. 1975
D. delphis	Southeastern Atlantic (South Africa)	1980–87	B	—	ΣDDT, PCBs	de Kock et al. 1994
D. delphis	Southwestern Indian Ocean (South Africa)	1984–87	B	17	ΣDDT, PCBs, HCB	de Kock et al. 1994
D. delphis	Victoria, Australia	—	B	1	DDT, PCBs	Kemper et al. 1994
D. delphis	Western North Atlantic	—	B	3	PCB congeners	Kuehl et al. 1994
Feresa attenuata	Florida (Gulf and Atlantic)	1975–78	B, Br, K, L, M, O	3	ΣDDT, DDE, TDE, DDT, dieldrin, PCBs	Forrester et al. 1980
Globicephala macrorhynchus	California	1974–76	B, M	2	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs	O'Shea et al. 1980

Appendix 10-2 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
G. macrorhynchus	Caribbean Sea (St. Lucia)	1972	B, K, L, M	5	DDE, TDE, DDT, ΣDDT, PCBs, dieldrin	Gaskin et al. 1974
G. macrorhynchus	Japan	1968–75	B	6	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs, dieldrin, HCB, toxaphene	O'Shea et al. 1980
G. macrorhynchus	Eastern North Pacific (California)	1971	L	5	DDE	Hall et al. 1971
G. macrorhynchus	Western North Atlantic (U.S.A.)	—	B, L	5	ΣDDT, PCBs	Varanasi et al. 1992
G. macrorhynchus	Western North Pacific (Japan)	1985	B	29	DDE, PCBs	Tanabe et al. 1987b
G. melaena	France	—	B, K, L, M	16	ΣDDT, DDE, DDT, TDE, PCBs	Alzieu and Duguy 1979
G. melaena	North Atlantic	1962–67	Oils	—	DDE, TDE, DDT, ΣDDT, dieldrin, PCBs	Addison et al. 1972
G. melaena	North Atlantic (U.S.A.)	1971–75	B	2	PCBs, chlordane, dieldrin, DDE, TDE, DDT, ΣDDT	Taruski et al. 1975
G. melaena	Northwestern Atlantic (Newfoundland)	1980	B	41	DDE, ΣDDT, PCBs, PCB congeners, dieldrin, toxaphene, HCB, HCHs, t-nonachlor, chlordane, mirex	Muir et al. 1988a
G. melaena	Western North Atlantic	—	B, L	11	DDE, PCBs, *trans*-nonachlor	Geraci 1989
G. melanea	Western North Atlantic (U.S.A.)	1986–90	B, L	23	ΣDDT, PCBs, chlordanes	Varanasi et al. 1993b
G. melaena	Australia	—	B	9	DDE, DDT, TDE, ΣDDT, PCBs, dieldrin, HCB, HCH	Kemper et al. 1994
G. melaena	North Atlantic (Faroe Islands)	1986–88	B	130	DDE, TDE, DDT, *o,p'*-DDT, ΣDDT, PCBs	Borrell et al. 1995
G. melaena	North Atlantic (Faroe Islands)	1987	B, M	211	DDE, TDE, *o,p'*-DDT, DDT, ΣDDT, PCBs	Borrell 1993; Borrell and Aguilar 1993
G. melaena	North Atlantic (Faroe Islands)	1986	B, O	50	DDE, PCBs, dieldrin, HCH, heptachlor epoxide	Simmonds et al. 1994
Grampus griseus	British Columbia	1988	B	1	DDE, TDE, DDT, ΣDDT, CBz, HCB, HCHs, chlordanes, nonachlors, oxychlordane, heptachlor epoxide, photoheptachlor, mirex, toxaphene, OCS, non-ortho PCB congeners, PCBs, PCDD congeners, PCDF congeners	Jarman et al. 1996
G. griseus	France	—	B, K, L, M, O	6	ΣDDT, DDT, DDE, TDE, PCBs	Alzieu and Duguy 1979
G. griseus	Eastern North Atlantic (Spain)	1978	B	1	DDE, TDE, DDT, *o,p'*-DDT, ΣDDT	Aguilar 1984
G. griseus	Eastern South Atlantic (South Africa)	1970	B, L	1	ΣDDT, dieldrin	Aucamp et al. 1971
G. griseus	Southwestern Indian Ocean (South Africa)	1984	B	2	ΣDDT, PCBs, HCB	de Kock et al. 1994
Hyperoodon spp.	North Atlantic	1962	Oils	—	DDE, TDE, DDT, ΣDDT, dieldrin, PCBs	Addison et al. 1972
H. ampullatus	North Sea (Germany)	—	B	1	DDE, TDE, DDT, ΣDDT, PCBs, lindane, dieldrin	Harms et al. 1977/78
Kogia breviceps	Australia	—	B	1	DDT, dieldrin	Kemper et al. 1994
K. breviceps	Southwestern Indian Ocean (South Africa)	1978–87	B	5	ΣDDT, PCBs, HCB	de Kock et al. 1994

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Appendix 10-2 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
K. breviceps	Western Atlantic (Florida)	1974	Mi	1	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-DDT, ΣDDT, PCBs, dieldrin	Jenness and Odell 1978
K. simus	Southwestern Indian Ocean (South Africa)	1976–84	B	4	ΣDDT, PCBs, HCB	de Kock et al. 1994
Lagenodelphis hosei	Eastern Tropical Pacific	—	B, M	1	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs	O'Shea et al. 1980
L. hosei	Japan	1991	B	4	HCHs	Tanabe et al. 1996
Lagenorhynchus acutus	Faroe Islands	1987	B	13	DDE, TDE, *o,p'*-DDT, DDT, ΣDDT, PCBs	Borrell 1993
L. acutus	Atlantic Coast (U.S.A.)	1989	B	3	DDE, dieldrin, HCB, mirex, chlordanes, lindane, heptachlor epoxide, PCB congeners, PCDD, PCDFs, PBBs	Kuehl et al. 1991
L. acutus	North Atlantic (Nova Scotia)	1971–75	B	1	PCBs, chlordane, dieldrin, DDE, TDE, DDT, ΣDDT	Taruski et al. 1975
L. acutus	Western North Atlantic	—	B	2	PCB congeners	Kuehl et al. 1994
L. albirostris	North Sea	—	B	1	Toxaphene	de Boer and Wester 1993
L. albirostris	North Sea (Denmark)	1972	B, Br, L, M, O	1	DDE, TDE, DDT, ΣDDT, PCBs, dieldrin	Andersen and Rebsdorff 1976
L. albirostris	North Sea (Netherlands)	1977	B, Br, M	3	DDE, TDE, DDT, dieldrin, HCB, HCHs, PCBs, PCB congeners	Duinker et al. 1989
L. albirostris	North Sea (Scotland)	1977	B	5	ΣDDT, PCBs, dieldrin, heptachlor epoxide	Kerkhoff et al. 1981
L. albirostris	Northwestern Atlantic (Newfoundland)	1982	B	27	DDE, ΣDDT, PCBs, PCB congeners, dieldrin, toxaphene, HCB, HCHs, *t*-nonachlor, chlordane, mirex	Muir et al. 1988a
L. obliquidens	North Pacific	1991	B	3	HCHs	Tanabe et al. 1996
L. obliquidens	North Pacific (Japan)	1981	B	5	ΣDDT, PCBs, HCHs	Tanabe et al. 1983
L. obscurus	Southwestern Indian Ocean (South Africa)	1977–87	B	12	ΣDDT, PCBs, HCB	de Kock et al. 1994
L. obscurus	Western South Pacific	1980	B	1	ΣDDT, PCBs, HCHs	Tanabe et al. 1983
Lissodelphis borealis	North Pacific	1991	B	2	HCHs	Tanabe et al. 1996
Mesoplodon bidens	France	—	L, O	1	ΣDDT, PCBs	Alzieu and Duguy 1979
M. densirostris	Mediterranean (Spain)	1980	B, Br, M	1	DDE, *o,p'*-DDT, TDE, DDT, ΣDDT, PCBs	Aguilar et al. 1982
M. densirostris	North Atlantic (U.S.A.)	1971–75	B	2	PCBs, chlordane, dieldrin, DDE, TDE, DDT, ΣDDT	Taruski et al. 1975
M. densirostris	Southwestern Indian Ocean (South Africa)	1984–86	B	6	ΣDDT, PCBs, HCB	de Kock et al. 1994
M. layardi	Southwestern Indian Ocean (South Africa)	1978–85	B	2	ΣDDT, PCBs, HCB	de Kock et al. 1994
M. layardi	Australia	1989	B	1	ΣDDT, DDE, DDT, TDE, PCBs	Anderson 1991
M. mirus	Southwestern Indian Ocean (South Africa)	1986	B	2	ΣDDT, PCBs, HCB	de Kock et al. 1994
M. stejnegeri	Japan	1984	B, K, L, M	1	DDE, PCBs	Miyazaki et al. 1987
M. sp.	Australia	—	B	2	DDE, DDT, PCBs, dieldrin, oxychlordane, HCB	Kemper et al. 1994
Monodon monoceros	Arctic (Baffin Bay, Canada)	—	B	17	PCBs, PCB congener ratios	Norstrom et al. 1992
M. monoceros	Arctic (Canada)	—	B	17	ΣPCBs, PCB congeners	Ford et al. 1993

Appendix 10-2 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
M. monoceros	Arctic (Canada)	1982–83	B, L	21	Chlorobenzenes, HCH, chlordanes, DDE, ΣDDT, mirex, dieldrin, toxaphene, PCBs, PCB congeners	Muir et al. 1992b
Neophocoena phocoenoides	Seto-Inland Sea (Japan)	1985	B	1	PCBs, PCB congeners, PCDFs, PCDDs	Kannan et al. 1989
N. phocoenoides	Western North Pacific (Japan)	1968–75	B, Br, M	6	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs, dieldrin, HCB, toxaphene, heptachlor epoxide, *trans*-nonachlor, oxychlordane, mirex	O'Shea et al. 1980
Orcinus orca	British Columbia, Washington	1986–89	B	6	DDE, TDE, DDT, ΣDDT, CBz, HCB, HCHs, chlordanes, nonachlors, oxychlordane, heptachlor epoxide, photoheptachlor, mirex, toxaphene, OCS, PCBs, non-ortho PCB congeners, PCDD congeners, PCDF congeners	Jarman et al. 1996
O. orca	Western North Pacific (Japan)	1986	B	3	PCBs, PCDDs, PCDFs, PCB congeners	Ono et al. 1987; Kannan et al. 1989
O. orca	Victoria, Australia	—	B	1	ΣDDT, dieldrin, heptachlor, endrin, HCB	Kemper et al. 1994
Peponocephala electra	Japan	1982	B	5	ΣDDT, PCBs, HCHs	Tanabe et al. 1983
P. electra	Japan	1982	B	3	HCHs	Tanabe et al. 1996
Phocoena phocoena	Baltic Sea (Germany)	—	B, L	2	DDE, TDE, DDT, ΣDDT, PCBs, lindane, dieldrin	Harms et al. 1977/78
P. phocoena	Baltic Sea (Poland)	1989–90	B, L, M	3	PCB congeners	Falandysz et al. 1994
P. phocoena	Baltic Sea (Poland)	1989–90	B, L, M	3	PCBs, ΣDDT, HCHs, HCB, aldrin, dieldrin, heptachlor, heptachlor epoxide, chlordanes	Kannan et al. 1993b
P. phocoena	British Columbia	1987–89	B	7	DDE, TDE, DDT, ΣDDT, CBz, HCB, HCHs, chlordanes, nonachlors, oxychlordane, heptachlor epoxide, photoheptachlor, mirex, toxaphene, octachlorostyrene, PCBs, non-ortho PCB congeners, PCDD congeners, PCDF congeners	Jarman et al. 1996
P. phocoena	California	1974–76	B, Br, M	1	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs, dieldrin, HCB, heptachlor epoxide, trans-nonachlor	O'Shea et al. 1980
P. phocoena	California	1987–88	B	3	DDE, TDE, DDT, ΣDDT, CBz, HCB, HCHs, chlordanes, nonachlors, oxychlordane, heptachlor epoxide, photoheptachlor, mirex, toxaphene, octachlorostyrene, PCBs, PCDD congeners, PCDF congeners	Jarman et al. 1996
P. phocoena	Denmark (North Sea, Baltic), West Greenland	1986–88	B	27	HCHs, HCB, DDE, TDE, DDT, ΣDDT, PCBs, PCB congeners	Granby and Kinze 1991
P. phocoena	Eastern North Pacific (Washington)	1992	B, L	3	ΣDDT, PCBs, chlordanes	Varanasi et al. 1993b

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Appendix 10-2 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
P. phocoena	Faroe Islands	1987–88	B	6	DDE, TDE, *o,p'*-DDT, DDT, ΣDDT, PCBs	Borrell 1993
P. phocoena	France	—	B, L, O	3	PCBs, DDE, ΣDDT	Alzieu and Duguy 1979
P. phocoena	Great Britain	1990–91	B	28	DDE, TDE, DDT, PCBs, HCB, HCHs, dieldrin	Kuiken et al. 1993
P. phocoena	Great Britain	1989–92	B	94	DDE, TDE, DDT, dieldrin, HCB, HCH, PCBs, PCB congeners	Kuiken et al. 1994
P. phocoena	Greenland	1972	B	2	DDE, PCBs, heptachlor epoxide, aldrin, lindane	Clausen et al. 1974; Clausen and Berg 1975
P. phocoena	Greenland	1989	B, L	4	PCB congeners, PCDD congeners, PCDF congeners	van Scheppingen et al. 1996
P. phocoena	North Atlantic (Bay of Fundy)	1969–70	B, L	36	DDE, TDE, DDT, *o,p'*-DDT, ΣDDT, dieldrin	Gaskin et al. 1971
P. phocoena	North Atlantic (Wales)	1988	B, M, L, O	4	HCB, HCHs, dieldrin, DDE, DDT, TDE, PCBs, PCB congeners	Morris et al. 1989
P. phocoena	North Atlantic (U.S.A.)	1971–75	B	1	PCBs, chlordane, dieldrin, DDE, TDE, DDT, ΣDDT	Taruski et al. 1975
P. phocoena	Northeast North Atlantic (Atlantic, North Sea, Kattegat)	1987–1991	B	34	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-DDT, dieldrin, endrin, HCHs, oxychlordane, *trans*-nonachlor, heptachlor epoxide, HCB, PCB congeners	Kleivane et al. 1995
P. phocoena	North Sea	—	B	1	Toxaphene	de Boer and Wester 1993
P. phocoena	North Sea	1970–71	B	7	DDE, TDE, DDT, PCBs, dieldrin, HCB	Koeman et al. 1972
P. phocoena	North Sea (Denmark)	1972–73	B, Br, K, L, M, O	7	DDE, TDE, DDT, ΣDDT, PCBs, dieldrin	Andersen and Rebsdorff 1976
P. phocoena	North Sea (Germany)	—	B, L	1	DDE, TDE, DDT, ΣDDT, PCBs, lindane, dieldrin	Harms et al. 1977/78
P. phocoena	North Sea (Germany)	1988	B	1	DDE, TDE, DDT, HCHs, HCB, PCB congeners, PCDDs, PCDFs	Beck et al. 1990
P. phocoena	North Sea (Scotland)	1988–91	B	48	HCB, ΣDDT, PCBs, PCB congeners, endrin, dieldrin, heptachlor epoxide, chlordanes, t-nonachlor, oxychlordane	Wells et al. 1994
P. phocoena	North Sea (Netherlands)	1977–79	B, Br, K, L, O M,	11	DDE, TDE, DDT, dieldrin, HCB, HCHs, PCBs, PCB congeners	Duinker et al. 1989
P. phocoena	North Sea (Netherlands)	1978	B, K, L, M, O	4	PCBs, PCB congeners	Duinker et al. 1988
P. phocoena	North Sea (Netherlands)	1979	B, K, L	1	DDE, TDE, DDT, o,p'-TDE, PCBs, dieldrin, HCB, HCHs	Duinker and Hillebrand 1979
P. phocoena	North Sea (Netherlands)	1990–93	B, K, L	22	PCBs, PCB congeners, PCDD congeners, PCDF congeners	van Scheppingen et al. 1996
P. phocoena	Northwestern Atlantic (Bay of Fundy, Rhode Island, Maine, Newfoundland, Prince Edward Island)	1971–77	B, Br, K, L, M, Mi, O	107	PCBs, HCB, chlordanes	Gaskin et al. 1983
P. phocoena	Northwestern Atlantic (Bay of Fundy, Rhode Island, Maine, Nova Scotia, Newfoundland, Prince Edward Island)	1969–73	B	115	ΣDDT	Gaskin et al. 1982

Appendix 10-2 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
P. phocoena	Scotland	1967	B, Br, K, L, M, O	4	DDE, TDE, DDT, ΣDDT, dieldrin	Holden and Marsden 1967
P. phocoena	Scotland	1990	B	1	PCB congeners	Wells and Echarri 1992
P. phocoena	Western North Atlantic (U.S.A.)	1991	B, L	3	ΣDDT, PCBs, chlordanes	Varanasi et al. 1993b
P. phocoena	Western North Atlantic (U.S.A.)	—	B, L	9	DDE, PCBs, *trans*-nonachlor	Geraci 1989
Phocoenoides dalli	Japan	1983	B	4	DDE, DDT, TDE, ΣDDT, PCBs, PCB congeners, HCHs	Tanabe et al. 1994b
P. dalli	Northern North Pacific	1980–82	B	3	DDE, TDE, DDT, HCHs, chlordanes	Kawano et al. 1988
P. dalli	Northern North Pacific	1980, 1985	B	5	PCBs, PCB congeners	Kannan et al. 1989
P. dalli	Bering Sea and Northwestern Pacific	—	B	42	PCBs, DDE, PCB congeners	Subramanian et al. 1988b
P. dalli	British Columbia	1987–88	B	3	DDE, TDE, DDT, ΣDDT, CBz, HCB, HCHs, chlordanes, nonachlors, oxychlordane, heptachlor epoxide, photoheptachlor, mirex, toxaphene, OCS, non-ortho PCB congeners, PCBs, PCDD congeners, PCDF congeners	Jarman et al. 1996
P. dalli	North Pacific (Bering Sea, North Pacific, Japan)	1980–82	B	6	ΣDDT, PCBs, HCHs	Tanabe et al. 1983
P. dalli	North Pacific (Bering Sea, North Pacific, Japan)	1985–89	B	9	HCHs	Tanabe et al 1996
P. dalli	Northwestern Pacific	1984	B	12	PCBs, DDE	Subramanian et al. 1987
P. dalli	California	1974–76	B, M	1	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs, dieldrin, HCB, *trans*-nonachlor	O'Shea et al. 1980
P. dalli	Western North Pacific (Japan)	1968–75	B, Br, M	1	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs, dieldrin, HCB, *trans*-nonachlor	O'Shea et al. 1980
Physeter catodon	Antarctic	1962–66	Oils	—	DDE, TDE, DDT, ΣDDT, dieldrin, PCBs	Addison et al. 1972
P. catodon	Eastern North Pacific (California)	1983	B, M	1	ΣDDT, PCBs	Schafer et al 1984
P. catodon	Eastern North Pacific (California)	1968	B, Br, L	6	DDE, TDE, DDT, dieldrin, others analyzed but not detected	Wolman and Wilson 1970
P. catodon	Lesser Antilles	1971–75	B	2	PCBs, chlordane, dieldrin, DDE, TDE, DDT, ΣDDT	Taruski et al 1975
P. catodon	North Atlantic	1967	Oils	—	DDE, TDE, DDT, ΣDDT, dieldrin, PCBs	Addison et al. 1972
P. macrocephalus	Australia	1989	B	2	DDE, TDE, DDT, PCBs	Anderson 1991
P. macrocephalus	France	1976	B, L, M, O	2	ΣDDT, PCBs	Alzieu and Duguy 1979
P. macrocephalus	Iceland	1982	B	10	DDE, TDE, *o,p'*-DDT, DDT, ΣDDT, PCBs	Borrell 1993
P. macrocephalus	North Atlantic (Spain)	1979-80	B, Br, K, L, M, O	14	DDE, TDE, DDT, *o,p'*-DDT, ΣDDT, PCBs	Aguilar 1983
P. macrocephalus	North Sea	1994–95	B	7	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-DDT, *o,p'*-TDE, dieldrin, HCB, HCHs, *trans*-chlordane, *cis*-chlordane, *trans*-nonachlor, PCBs, PCB congeners	Law et al. 1996

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Appendix 10-2 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
P. macrocephalus	North Sea (Netherlands)	1979	B, Br, M, O	1	DDE, TDE, DDT, dieldrin, HCB, HCHs, PCBs, PCB congeners	Duinker et al. 1989
P. macrocephalus	Scotland	1990	B	1	PCB congeners	Wells and Echarri 1992
P. macrocephalus	South Africa	1994	B	12	DDE, TDE, DDT, ΣDDT, PCBs, dieldrin	Henry and Best 1983
P. macrocephalus	Southwestern Indian Ocean (South Africa)	1986	B	1	ΣDDT, PCBs, HCB	de Kock et al. 1994
P. macrocephalus	Victoria, Australia	—	B	1	ΣDDT, PCBs, dieldrin, endrin, HCB, BHC	Kemper et al. 1994
Platanista gangetica	India (Ganges River)	1988–92	B, K, L, M	4	PCBs, ΣDDT, HCHs, HCB, aldrin, dieldrin, heptachlor, heptachlor epoxide, chlordanes	Kannan et al. 1993a
Pantoporia blainvillei	South Atlantic (Uruguay)	1974	B, Br, M	8	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs, dieldrin, HCB, toxaphene, heptachlor expoxide, *trans*-nonachlor, oxychlordane, cis-chlordane	O'Shea et al. 1980
Pseudorca crassidens	British Columbia	1987–89	B	2	DDE, TDE, DDT, ΣDDT, CBz, HCB, HCHs, chlordanes, nonachlors, oxychlordane, heptachlor epoxide, photoheptachlor, mirex, toxaphene, OCS, non-ortho PCB congeners, PCBs, PCDD congeners, PCDF congeners	Jarman et al. 1996
P. crassidens	Eastern North Pacific (Canada)	1988	B	1	ΣDDT, PCBs, methyl sulfones of PCB congeners and DDE	Bergman et al. 1994
P. crassidens	North Pacific (Canada)	1987	B, Br, L	1	PCBs, DDE, TDE	Baird et al. 1989
Sotalia fluviatilis	Colombia	1977 (captive)	B, Br, K, L, M, O	2	DDE, TDE, DDT, dieldrin, HCB, HCHs, PCBs, PCB congeners	Duinker et al. 1989
S. fluviatilis	—	1970–71	B	1	DDE, TDE, DDT, PCBs, dieldrin	Koeman et al. 1972
Sousa chinensis	India (Bay of Bengal)	1990–91	B	3	HCHs, HCB, DDE, DDT, TDE, *o,p'*-DDT, ΣDDT, PCBs	Tanabe et al. 1993
S. chinensis	India (Bay of Bengal)	1992	B	2	HCHs	Tanabe et al. 1996
Stenella attenuata	Australia	—	B	1	DDE, TDE, DDT, ΣDDT, PCBs, heptachlor, oxychlordanes, HCB	Kemper et al. 1994
S. coeruleoalba	Eastern North Atlantic (Spain)	—	B	1	DDE, TDE, DDT, *o,p'*-DDT, PCBs	Borrell and Aguilar 1990
S. coeruleoalba	Eastern Tropical Pacific	1973–76	B, Br, M	14	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs, dieldrin, toxaphene, heptachlor expoxide, *trans*-nonachlor, endrin, *cis*-chlordane	O'Shea et al. 1980
S. coeruleoalba	France	—	B, K, L, M, O	27	ΣDDT, DDE, DDT, TDE, PCBs	Alzieu and Duguy 1979
S. coeruleoalba	Japan	1968–75	B, Br, M	5	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs, dieldrin, HCB, toxaphene, *trans*-nonachlor	O'Shea et al. 1980
S. coeruleoalba	Japan	1978–79, 1986	B	16	PCBs, ΣDDT, HCHs, HCB	Loganathan et al. 1990
S. coeruleoalba	Japan	1992	B	4	HCHs	Tanabe et al. 1996
S. coeruleoalba	Mediterranean	1991	B	7	PCBs, ΣDDT	Fossi et al. 1992
S. coeruleoalba	Mediterranean	1990	B	10	ΣDDT, PCBs, PCB congeners	Kannan et al. 1993c
S. coeruleoalba	Mediterranean	1987–91	B	181	PCBs	Aguilar and Borrell 1994a

Appendix 10-2 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
S. coeruleoalba	Mediterranean	1990–93	B	89	ΣDDT, PCBs, PCB congeners	Marsili and Focardi 1996
S. coeruleoalba	Mediterranean	1990	Br, K, L, Lu, M, O	10	ΣDDT, PCBs	Guitart et al 1996
S. coeruleoalba	North Atlantic (U.S.A.)	1971–75	B	2	PCBs, chlordane, dieldrin, DDE, TDE, DDT, ΣDDT	Taruski et al. 1975
S. coeruleoalba	North Atlantic (Wales)	1988	B, M, O	1	HCB, HCHs, dieldrin, DDE, DDT, TDE, PCBs, PCB congeners	Morris et al. 1989
S. coeruleoalba	North Pacific (Japan)	1978	B, Br, K, L, M, O	6	HCB, PCBs, ΣDDT, HCHs, DDE, DDT, TDE	Tanabe et al 1981
S. coeruleoalba	North Pacific (Japan)	1978–79	B, L, M, K, O	2	PCBs, ΣDDT, HCHs	Kawai et al 1988
S. coeruleoalba	North Pacific (Japan)	1978	B	4	ΣDDT, PCBs, HCHs	Tanabe et al 1983
S. coeruleoalba	North Pacific (Japan)	1978–79	B, Br, K, L, M, O	2	ΣDDT, PCBs, HCHs	Kawai et al. 1988
S. coeruleoalba	Southwestern Indian Ocean (Sonth Africa)	1984–86	B	2	ΣDDT, PCBs, HCB	de Kock et al. 1994
S. coeruleoalba	Western North Pacific	1978	O	4	ΣDDT, PCBs, HCHs, PCB congeners, DDE, TDE, DDT	Tanabe et al. 1984b
S. coeruleoalba	Western North Pacific (Japan)	1978	B, Br, K, L, M, O	6	DDE, TDE, DDT, ΣDDT, PCBs, HCHs, HCB	Tanabe et al. 1981
S. longirostris	Caribbean Sea (St. Lucia)	1972	B, K, L, M	2	DDE, TDE, DDT, ΣDDT, PCBs, dieldrin	Gaskin et al. 1974
S. longirostris	Eastern Tropical Pacific	1980–82	B	2	HCHs	Tanabe et al. 1996
S. longirostris	India (Bay of Bengal)	1990–91	B	5	HCHs, HCB, DDE, DDT, TDE, *o,p'*-DDT, ΣDDT, PCBs	Tanabe et al. 1993, 1996
Steno bredanensis	Hawaii	1976	B, Br, M	7	DDE, TDE, DDT, *o,p'*-TDE, *o,p'*-DDT, PCBs, dieldrin, *trans*-nonachlor	O'Shea et al. 1980
Tursiops truncatus	—	1970–71	B	2	DDE, TDE, DDT, PCBs, dieldrin, HCB	Koeman et al 1972
T. truncatus	Atlantic Coast (U.S.A.)	1987–89	B, Br, K, L	14	DDE, dieldrin, HCB, mirex, chlordanes, lindane, heptachlor epoxide, PCB congeners, PCDD, PCDFs, PBBs	Kuehl et al. 1991
T. truncatus	Atlantic Coast (U.S.A.)	1987–89	B	9	PCB congeners	Kuehl et al. 1994
T. truncatus	Australia	—	B	6	DDE, DDT, ΣDDT, PCBs, dieldrin, heptachlor, lindane	Kemper et al 1994
T. truncatus	California	1974–76	B, M	2	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs, dieldrin, HCB, heptachlor epoxide, *trans*-nonachlor, oxychlordane, *cis*-nonachlor, *cis*-chlordane	O'Shea et al. 1980
T. truncatus	Eastern North Pacific (California)	1980–84	B, Br, L, K, M	7	ΣDDT, PCBs	Schafer et al 1984
T. truncatus	France	—	K, L, M, O	5	ΣDDT, DDT, DDE, TDE	Alzieu and Duguy 1979
T. truncatus	Gulf of Mexico (Florida)	1969–70 (captive)	B	4	DDE, TDE, DDT, PCBs, dieldrin	Dudok van Heel 1972
T. truncatus	Gulf of Mexico (Texas, Florida)	—	B	33	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, ΣDDT, dieldrin, endrin, aldrin, mirex, HCB, HCHs, heptachlor, heptachlor epoxide, oxychlordane, nonachlors, chlordanes, PCBs, PCB congeners	Salata et al. 1995

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Appendix 10-2 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
T. truncatus	Gulf of Mexico (U.S.A.)	1990	B	26	DDE, DDT, dieldrin, mirex, HCB, *cis*-chlordane, oxychlordane, heptachlor epoxide, PCBs, PBBs, PBDPEs, OCS	Kuehl and Haebler 1995
T. truncatus	India (Bay of Bengal)		B	4	HCHs, HCB, DDE, DDT, TDE, *o,p'*-DDT, ΣDDT, PCBs	Tanabe et al. 1993
T. truncatus	Indian Ocean (South Africa)	1980–87	B	105	PCBs ΣDDT, dieldrin	Cockcroft et al 1989
T. truncatus	North Atlantic (Wales)	1988	B, M, O	1	HCB, HCHs, dieldrin, DDE, DDT, TDE, PCBs, PCB congeners	Morris et al 1989
T. truncatus	North Atlantic (Wales)	1989–91	B	3	DDE, TDE, DDT, dieldrin, HCB, HCHs, PCBs, PCB congeners	Law et al. 1995
T. truncatus	North Sea (Scotland)	1988–91	B	6	ΣDDT, HCB, PCBs, PCB congeners, dieldrin, endrin, heptachor epoxide, chlordanes, *t*-nonachlor, oxychlordane	Wells et al 1994
T. truncatus	Northern Gulf of Mexico (Texas)	1990	B, L	20	ΣDDT, PCBs, PCB congeners	Varanasi et al. 1992
T. truncatus	Northern Gulf of Mexico (U.S.A.)	1990–92	B, L	65	ΣDDT, PCBs, chlordanes	Varanasi et al. 1993b
T. truncatus	Scotland	—	B	1	PCB congeners	Wells and Echarri 1992
T. truncatus	Southeastern Atlantic (South Africa)	1976–87	B	—	ΣDDT, PCBs	de Kock et al. 1994
T. truncatus	Southwestern Indian Ocean (South Africa)	1976–87	B	6	ΣDDT, PCBs, HCB	de Kock et al 1994
T. truncatus	Western North Atlantic (Florida)	1965 (captive)	B, Br, M	1	DDE, TDE, DDT, dieldrin, HCB, HCHs, PCBs, PCB congeners	Duinker et al 1989
T. truncatus	Western North Atlantic (U.S.A.)	1987–88	B, L	56	DDE, PCBs, PCB congeners, *trans*-nonachlor	Geraci 1989
T. truncatus	captives	1992–93	Mi	5	DDE, TDE, DDT, dieldrin, HCB, heptachlor epoxide	Ridgway and Reddy 1995
Ziphius cavirostris	France	—	M, O	2	ΣDDT, PCBs	Alzieu and Duguy 1979
Z. cavirostris	North Atlantic (Bermuda Island)	1981	B, K, L, M, O	4	PCBs, PCB congeners	Duinker et al. 1988

B = blubber; Br = brain; K = kidney, L = liver, Lu = lung; M = muscle; Mi = milk; O = other. Dashes appear where values were not available in original source

CBZ = chlorobenzenes. DDE = 2,2 -*bis* (p-chlorophenyl)-1, 1-dichloroethylene; DDT = 2,2,-*bis*-(*p*-chlorophenyl)-1, 1-trichloroethane; ΣDDT = arithmetic summation of concentrations of isomers and metabolites of DDT; HCB = hexachlorocyclohexane, OCS = octachlorostyrene; PBDPEs = polybrominated diphenyl ethers, PCBs, polychlorinated biphenyls; PCDDs = polychlorinated dibenzo-*p*-dioxins; PCDFs = polychlorinated dibenzofurans, TCP = *tris*(4-chlorophenyl)methane/methanol, TDE = 2,2,-*bis* (p-chlorophenyl)-1, 1-dichloroethane.

Appendix 10-3. Summary of Selected Organochlorine Residue Surveys in Baleen Whales

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
Balaena mysticetus	Alaska	1992	B	2	ΣDDT, PCBs, chlordanes	Varanasi et al 1993b
B mysticetus	Arctic	1986–88	B, O	6	TDE, dieldrin, heptachlor epoxide, BHC, endrin, endosulfan sulfate, kepone	Bratton et al 1993
Balaenoptera acutorostrata	Antarctic	1980–81	L	30	ΣDDT, PCBs	Tanabe et al. 1984a
B. acutorostrata	Antarctic	1984–85	B	37	DDE, PCBs	Tanabe et al. 1986
B. acutorostrata	Arctic	1985	L	1	PCBs	Goksøyr et al 1988

Appendix 10-3 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
B. acutorostrata	Eastern North Pacific (California)	1977	B, Br, L, M	1	ΣDDT, PCBs	Schafer et al 1984
B. acutorostrata	Mediterranean (France)	1977	K, L, O	1	PCBs	Alzieu and Duguy 1979
B. acutorostrata	South Africa	1974	B	29	DDE, TDE, DDT, ΣDDT, PCBs, dieldrin	Henry and Best 1983
B. acutorostrata	Southwestern Indian Ocean (South Africa)	1984	B	1	ΣDDT, PCBs, HCB	de Kock et al. 1994
B. acutorostrata	Eastern North Pacific (Washington)	1989–90	B	2	ΣDDT, PCBs, chlordanes	Varanasi et al. 1993b
B. acutorostrata	Canada (St. Lawrence River)	1988–90	B	5	ΣDDT, PCBs, mirex	Béland et al. 1991
B. borealis	Antarctica	1950	Oils	—	DDE, TDE, DDT, ΣDDT, dieldrin, PCBs	Addison et al 1972
B. borealis	Iceland	1982–85	B	40	DDE, TDE, *o,p'*-DDT, DDT, ΣDDT, PCBs	Borrell 1993
B. borealis	North Atlantic (Iceland)	1982	B	23	DDE, ΣDDT	Borrell and Aguilar 1987
B. borealis	South Africa	1974	B	1	DDE, TDE, DDT, ΣDDT, PCBs, dieldrin	Henry and Best 1983
B. edeni	Eastern South Pacific (Chile)	1983	B, L	2	DDE, TDE, DDT	Pantoja et al. 1984, 1985
B. musculus	Antarctica	1950	Oils	—	DDE, TDE, DDT, ΣDDT, dieldrin, PCBs	Addison et al. 1972
B. musculus	Eastern Tropical Pacific	1980	L	1	DDE, TDE, DDT, ΣDDT, PCBs	Britt and Howard 1983
B. musculus	Canada (St. Lawrence River)	1988–90	B	2	ΣDDT, PCBs, mirex	Béland et al. 1991
B. physalus	Eastern North Atlantic (Spain)	1980	M, O	68	DDE, TDE, DDT, *o,p'*-DDT, ΣDDT, PCBs	Aguilar and Jover 1982
B. physalus	Eastern North Atlantic (Spain)	1980	B	1	DDE, TDE, DDT, *o,p'*-DDT, ΣDDT	Aguilar 1984
B. physalus	Eastern North Atlantic	1982–84	B	166	DDE, TDE, *o,p'*-DDT, DDT, ΣDDT, PCBs, PCB congeners	Aguilar and Borrell 1988
B. physalus	Eastern North Atlantic (Spain)	1982–84	B	137	DDE, ΣDDT	Borrell and Aguilar 1987
B. physalus	Eastern South Pacific (Chile)	1983	B, L	1	DDE, TDE, DDT, dieldrin, aldrin, lindane	Pantoja et al. 1984, 1985
B. physalus	France (Atlantic, Mediterranean)	1973, 1976	B, K, L, O	2	ΣDDT, PCBs	Alzieu and Duguy 1979
B. physalus	Iceland	1982–86	B	51	DDE, TDE, *o,p'*-DDT, DDT, ΣDDT, PCBs	Borrell 1993
B. physalus	Mediterranean (France)	—	B, Br, L	1	ΣDDT, PCBs	Viale 1981
B. physalus	Mediterranean	—	B, Br, L, K	1	PCBs, ΣDDT	Viale 1981
B. physalus	Mediterranean	1991	B	9	ΣDDT, PCBs	Fossi et al. 1992
B. physalus	Mediterranean	1990–93	B	68	ΣDDT, PCBs, PCB congeners	Marsili and Focardi 1996
B. physalus	North Atlantic	1967–70	Oils	—	DDE, TDE, DDT, ΣDDT, PCBs, dieldrin	Addison et al. 1972
B. physalus	South Africa	1974	B	6	DDE, TDE, DDT, ΣDDT, PCBs, dieldrin	Henry and Best 1983
B. physalus	Greenland	—	B	6	ΣDDT, PCBs, dieldrin	Holden 1975
B. physalus	Canada (St. Lawrence River)	1988–90	B	2	ΣDDT, PCBs, mirex	Béland et al. 1991
B. physalus	Western North Atlantic (Canada)	1970–71	B	12	DDE, TDE, DDT, *o,p'*-DDT, PCBs	Saschenbrecker 1973
Caperea marginata	Australia	1987	B	3	DDE, TDE, DDT, ΣDDT, dieldrin, HCB	Kemper et al 1994
C. marginata	Southwestern Indian Ocean (South Africa)	1987	B	1	ΣDDT, PCBs, HCB	de Kock et al. 1994
Eschrichtius robustus	Eastern North Pacific (California)	1968–69	B, Br, L	23	DDE, TDE, DDT, dieldrin, others analyzed but not detected	Wolman and Wilson 1970
E. robustus	Eastern North Pacific (California)	1976	B, Br, L, M	1	ΣDDT, PCBs	Schafer et al. 1984

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Appendix 10-3 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
E. robustus	Eastern North Pacific (British Columbia)	1987–88	B	2	PCDD congeners, PCDF congeners	Jarman et al. 1996
E. robustus	Eastern North Pacific (U.S.A.)	1988–91	B, Br, L	22	DDE, TDE, DDT, *o,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, PCBs, PCB congeners, heptachlor, heptachlor epoxide, α-chlordane, *t*-nonachlor, HCB, BHC, dieldrin, mirex, aldrin	Varanasi et al. 1993a, Varanasi et al. 1994
Eubalaena australis	Southwestern Indian Ocean (South Africa)	1984	B	2	ΣDDT, PCBs, HCB	de Kock et al. 1994
E. glacialis	Western North Atlantic	1988–89	B	35	DDE, TDE, DDT, ΣDDT, PCBs, dieldrin, heptachlor epoxide, chlordane, HCB	Woodley et al. 1991
Megaptera novaeangliae	North Atlantic (Nova Scotia)	1971–75	B	1	PCBs, chlordane, dieldrin, DDE, TDE, DDT, ΣDDT	Taruski et al. 1975
M. novaeangliae	North Atlantic (U.S.A.)	1971–75	B	1	PCBs, chlordane, dieldrin, DDE, TDE, DDT, ΣDDT	Taruski et al. 1975
M. novaeangliae	Western Atlantic (Lesser Antilles)	1971–75	B	2	PCBs, chlordane, dieldrin, DDE, TDE, DDT, ΣDDT	Taruski et al. 1975
M. novaeangliae	Wesrern North Atlantic (U.S.A.)	—	B	8	DDE, PCBs, *trans*-nonachlor	Geraci 1989

B = blubber; Br = brain; K = kidney, L = liver; M = muscle; O = other. Dashes appear where values were not available in original source.

BHC = benzene hexachloride; DDE = 2,2,-*bis*-(*p*-chlorophenyl)-1, 1-dichloroethylene, DDT = 2,2,-*bis*-(*p*-chlorophenyl)-1,1-trichloroethane; ΣDDT = arithmetic summation of concentrations of isomers and metabolites of DDT; HCB = hexachlorobenzene; PCBs = polychlorinated biphenyls; PCDDs = polychlorinated dibenzo-*p*-dioxins, PCDFs = polychlorinated dibenzofurans; TDE = 2,2,-*bis*-(*p*-chlorophenyl)-1,1-dichloroethane.

Appendix 10-4. Summary of Selected Organochlorine Residue Surveys in Sea Otters, Sirenians, and Polar Bears

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
Enhydra lutris	California	1969–70	A, Br, K, L, O	10	DDE, TDE, DDT	Shaw 1971
Trichechus manatus	Florida	1974	B, Br, L, M, O	1	DDE, PCBs, dieldrin	Forrester et al. 1975
T. manatus	Florida	1977–81	B	26	ΣDDT, dieldrin, PCBs	O'Shea et al. 1984
T. manatus	Florida	1982	B, Br	4	DDE	O'Shea et al. 1991
T. manatus	Florida	1990–93	B, K, L	19	*o,p'*-TDE, *o,p'*-DDT, HCB, lindane	Ames and Van Vleet 1996
Dugong dugon	Sulawesi (Indonesia)	1975	M	2	PCB, DDT, BHC	Miyazaki et al. 1979
Ursus maritimus	Arctic (Canada)	1992–94	L	16	PCB congeners, PCDD congeners, PCDF congeners, ΣDDT, methylsulfone PCBs, chlordane	Letcher et al. 1996
U. maritimus	Arctic (12 regions)	1989–91	A	94 (pooled)	DDE, PCBs, PCB congeners, methyl sulfones of PCBs and DDE	Letcher et al. 1995
U. maritimus	Arctic/subarctic (Canada)	1969–84	A, L	141	DDE, TDE, DDT, ΣDDT, HCBs, HCHs, dieldrin, chlordane-relared compounds, PCBs, PCB congeners	Norstrom et al. 1988
U. maritimus	Arctic Ocean (Canada)	1982–84	A, L	113	PCDDs, PCDFs, PCBs, HCB	Norstrom et al. 1990
U. maritimus	Arctic Ocean (Canada)	1982, 1984	A	20	ΣDDT, PCB congeners, heptachlor epoxide, chlordane isomers, HCHs, dieldrin, PCBs	Muir et al. 1988b
U. maritimus	Arctic Ocean (Norway)	1978–89	A, L	24	DDT, *o,p'*-DDT, DDE, PCBs, PCB congeners, HCB, HCH, oxychlordane, heptachlor, heptachlor epoxide, dieldrin	Norheim et al 1992

Appendix 10-4 continued

Species	Region	Sample Period	Tissues Sampled	Number of Individuals Sampled	Compounds Reported	References
U. maritimus	Arctic (Norway)	1990–91	Mi	6	PCB congeners, PCDDs, PCDFs	Oehme et al. 1995a
U. maritimus	Baffin Bay (Canada)	—	A	6	PCBs, PCDDs, PCDFs, PCB congeners	Norstrom et al. 1992
U. maritimus	Canada	1968–72	A, Br, L, M, Mi	40	DDE, DDT, PCBs	Bowes and Jonkel 1975; Bowes and Lewis 1974
U. maritimus	Canada (Manitoba)	1992–93	A, Mi	7	ΣDDT, PCBs, chlordanes, HCHs, HCBs	Polischuk et al. 1995
U. maritimus	Greenland	1972	A	1	DDE, PCBs, hepachlor epoxide, aldrin	Clausen et al. 1974; Clausen and Berg 1975
U. maritimus	Hudson Bay (Canada)	1985	A, L	2	ΣDDT, PCBs, methyl sulfones of PCB congeners and DDE	Bergman et al. 1994
U. maritimus	Northern Quebec (Canada)	1989–90	A	52	PCBs, chlordanes, nonachlors, oxychlordane, heptachlor epoxide, photoheptachlor	Zhu et al. 1995
U. maritimus	Repulse Bay (Canada)	1989	A	1	ΣDDT, PCBs, HCHs, chlordane, toxaphene, PCB congeners	Zhu et al. 1995; Zhu and Norstrom 1993

A = adipose tissue; B = blubber; Br = brain; K = kidney; L = liver; M = muscle; Mi = milk; O = other.

BHC = benzene hexachloride; DDE = 2,2,-*bis*(*p*-chlorophenyl)-1, 1-dichloroethylene; DDT = 2,2,-*bis*(*p*-chlorophenyl)-1,1-trichloroethane; ΣDDT = arithmetic summation of concentrations of isomers and metabolites of DDT; HCB = hexachlorobenzene; HCH = hexachlorocyclohexane; PCBs = polychlorinated biphenyls; PCDDs = polychlorinated dibenzo-*p*-dioxins; PCDFs = polychlorinated dibenzofurans; TDE = 2,2,-*bis*-(*p*-chlorophenyl)-1,1-dichloroethane.

Appendix 10-5. Summary of Selected Surveys of Metals and Trace Element Concentrations in Tissues of Pinnipeds

Species	Region	Sample Period	Tissues Sampled	Max. No. of Individuals Sampled	Metals and Trace Elements Reported	References
Arctocephalus australis	Western South Atlantic (Argentina)	—	B, K, L, M	8	Cd, Cu, Hg, Zn	Marcovecchio et al. 1994
A. gazella	Antarctic (South Georgia)	1987	L	11	Ba, Cd, Ce, Co, Cu, Cr, Hg, I, La, Mg, Mo, Pb, Rb, Sn, Sr, Zn	Malcolm et al. 1994
A. gazella	Antarctic	1987–89	B, K, M, L	4	Cd, Cu, Hg, Zn	de Moreno et al. 1997
A. pusillus	Australia (Bass Strait)	—	Br, K, L, M, O	16	Hg	Bacher 1985
A. spp.	Australia	—	K, L, M, O	16	Cd, Hg, Pb	Kemper et al. 1994
Callorhinus ursinus	Alaska	1972	Bl, Mi, hair	7	Hg	Kim et al. 1974
C. ursinus	Bering Sea	1984	K, L, M, O	—	Cd, Cr, Cu, Ni, Pb, Se, Zn	Richard and Skoch 1986
C. ursinus	Bering Sea	1987	K, L, M	2	40 trace elements, MeHg	Zeisler et al. 1993
C. ursinus	Japan, Pribilof Islands	1990–92	K, L, M	67	Cd, Cu, Fe, Hg, Mn, Zn	Noda et al. 1995
C. ursinus	Pribilof Islands	1970	Br, L, M	39	As, Cd, Hg, Pb	Anas 1973, 1974a
C. ursinus	Pribilof Islands	1975	K, L, M, bone	39	Cd, Hg, Ni, Pb, Zn	Goldblatt and Anthony 1983
C. ursinus	Eastern North Pacific (Washington)	1970–71	Br, K, L, M	39	As, Cd, Hg, Pb	Anas 1973, 1974a
Cystophora cristata	Greenland	1984	L	3	Cd, Hg, Se, Zn	Nielsen and Dietz 1990
C. cristata	Greenland	1984–87	K, L, M	4	Hg, organic Hg	Dietz et al. 1990
C. cristata	Gulf of St. Lawrence	1971	B, L, M, hair	3	Hg	Sergeant and Armstrong 1973

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Appendix 10-5 continued

Species	Region	Sample Period	Tissues Sampled	Max. No. of Individuals Sampled	Metals and Trace Elements Reported	References
Erignathus barbatus	Alaska	—	L	3	Ag, Cd, Hg, Se, V	Mackey et al. 1996
E. barbatus	Arctic (Canada)	1973	L, M	6	Hg, MeHg	Smith and Armstrong 1975
E. barbatus	Arctic Ocean (Canada)	1973–76	L, M	64	Hg, MeHg, Se	Smith and Armstrong 1978
E. barbatus	Greenland	1984–87	K, L, M	3	Hg, organic Hg	Dietz et al. 1990
E. barbatus	Northwest Atlantic (Canada)	—	Claws	9	Hg, MeHg	Freeman and Horne 1973
Eumetopias jubatus	Japan	1976–77	Br, K, L, M, O	22	Cd, Zn	Hamanaka et al. 1982
E. jubatus	Japan	1994–95	B, K, L, M, O	7	Butyltin compounds	Kim et al. 1996
Halichoerus grypus	Baltic Sea (Sweden)	1979–90	B, K, L	19	Al, As, Ca, Cd, Co, Cr, Cu, Fe, Hg, Mg, Mn, Ni, Pb, Se, V, W, Zn	Frank et al. 1992
H. grypus	England	1988–89	B, K, L	8	Hg	Simmonds et al. 1993
H. grypus	Great Britain	1968–72	Br, K, L, teeth	73	Cd, Cr, Cu, Hg, Pb, Zn	Heppleston and French 1973
H. grypus	Great Britain	1977	L, bile	15	Hg, MeHg, Se	van de Ven et al. 1979
H. grypus	Gulf of Finland	1976–82	B, K, L, M	9	Cd, Cu, Hg, Pb, Se, Zn	Pertillä et al. 1986
H. grypus	North Sea (Germany)	—	L	1	Cd, Cu, Hg, Pb, Zn	Harms et al. 1977/78
H. grypus	North Sea (Scotland)	—	B, K, L, O	1	Hg	Jones et al. 1972
H. grypus	North Sea (U.K.)	—	B, Br, K, L, M, O	13	Cd, Cu, Hg, Pb, Zn	Holden 1975
H. grypus	Nova Scotia	1971	B, K, L, M	11	Hg	Sergeant and Armstrong 1973
H. grypus	Nova Scotia	1972	B, Br, K, L, M, O	6	Hg, MeHg	Freeman and Horne 1973
H. grypus	St. Lawrence River	1983–90	L	5	Cd, Hg	Béland et al. 1991
H. grypus	U.K.	1988–89	L	14	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Law et al. 1991, 1992
H. grypus	Wales	1988	B, K, L, M	2	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Morris et al. 1989
Histriophoca fasciata	Okhotsk Sea	1975	L, M, O	28	Cd, Zn	Hamanaka et al. 1977
Hydrurga leptonyx	Antarctic	1989	K, L, M, O	3	Ca, Hg, K, Mg, Na	Szefer et al. 1993
H. leptonyx	Antarctic	1989	K, L, M, O	3	Ag, Cd, Co, Cr, Cu, Fe, Mn, Ni, Pb, Zn	Szefer et al. 1994
H. leptonyx	Australia	—	B, K, L, M	2	Cd, Hg, Pb	Kemper et al. 1994
Leptonychotes weddellii	Antarctic	—	K, O	1	Cd, Cu, Fe, Mg, Pb, Zn	Mishima et al. 1977
L. weddellii	Antarctic	—	B, Bl, K, L, M, Bone, O	3	Cd, Cu, Fe, Hg, Mn, Ni, Pb, Zn	Yamamoto et al. 1987
L. weddellii	Antarctic	1983	K, L, M	8	Cd, Cu	Steinhagen-Schneider 1986
L. weddellii	Antarctic	1989	K, L, M	2	Ca, Hg, K, Mg, Na	Szefer et al. 1993
L. weddellii	Antarctic	1989	K, L, M	2	Ag, Cd, Co, Cr, Cu, Fe, Mn, Ni, Pb, Zn	Szefer et al. 1994
Lobodon carcinophagus	Antarctic	1983	K, L, M	9	Cd, Cu	Steinhagen-Schneider 1986
L. carcinophagus	Antarctic	1989	K, L, M	27	Ca, Hg, K, Mg, Na	Szefer et al. 1993
L. carcinophagus	Antarctic	1989	K, L, M	27	Ag, Cd, Co, Cr, Cu, Fe, Mn, Ni, Pb, Zn	Szefer et al. 1994
Mirounga leonina	Antarctic	1987–89	M, B, O	2	Cd, Cu, Hg, Zn	de Moreno et al. 1997
Monachus monachus	Greece	1986–91	Hair	—	Cd, Cu, Hg, Pb, Zn	Yediler et al. 1993
Neophoca cinerea	Australia	—	K, L	5	Cd	Kemper et al. 1994

Appendix 10-5 continued

Species	Region	Sample Period	Tissues Sampled	Max. No of Individuals Sampled	Metals and Trace Elements Reported	References
Odobenus rosmarus	Alaska	1986–89	K, L	56	Ag, Al, As, B, Ba, Be, Cd, Cr, Cu, Fe, Hg, Mg, Mn, Mo, Ni, Pb, Sb, Se, Sn, Sr, Tl, V, Zn	Warburton and Seagars 1993
O. rosmarus	Bering Sea	1981–84	K, L	65	As, Cd, Hg, Pb, Se, Zn	Taylor et al 1989
O. rosmarus	Greenland	1975–77	K, L, M	69	Hg, MeHg	Born et al. 1981
O. rosmarus	Arctic Ocean (Canada)	1988	Teeth	12	Cu, Pb, Sr, Zn	Evans et al. 1995
Ommatophoca rossi	Antarctic	1981–82	L	20	Cd, Co, Cr, Cu, Fe, Hg, Mn, Ni, Pb, Zn	McClurg 1984
Otaria flavescens	Western South Atlantic (Argentina)	—	B, K, L, M	7	Cd, Hg	Marcovecchio et al. 1994
Phoca groenlandica	Greenland	1984–87	K, L, M	3	Hg, organic Hg	Dietz et al. 1990
P. groenlandica	Arctic (Greenland, Canada)	1976–78	Bl, Br, K, L, M	43	Cd, Cu, Hg, Pb, Se	Ronald et al. 1984b
P. groenlandica	Newfoundland	1980	L, M	30	Hg	Botta et al. 1983
P. groenlandica	Canada (Gulf of St. Lawrence)	1973	B, Bl, Br, K, L	20	Hg, MeHg	Jones et al. 1976
P. groenlandica	Gulf of St. Lawrence	—	B, Br, K, L, M, O	10	Hg, MeHg	Freeman and Horne 1973
P. groenlandica	Gulf of St. Lawrence	1984	K, L, M, Mi	40	Cd, Cu, Hg, MeHg, Se, Zn	Wagemann et al. 1988
P. groenlandica	Northwest Atlantic (Canada)	1976–78	Bl, Br, K, L, M	205	Cd, Cu, Hg, Pb, Se	Ronald et al. 1984b
P. groenlandica	St. Lawrence River	1971	B, L, M	20	Hg	Sergeant and Armstrong 1973
P. groenlandica	St. Lawrence River	1983–90	L	1	Cd, Hg	Béland et al 1991
P. hispida	Alaska	—	L	13	Ag, Cd, Hg, Se, V	Mackey et al. 1996
P. hispida	Alaska (Chukchi Sea, Nome)	1988–89	K, L	4	40 trace elements, MeHg	Zeisler et al. 1993
P. hispida	Arctic (Canada)	1972	L, M	80	Hg, MeHg	Smith and Armstrong 1975
P. hispida	Arctic (Canada)	1983	K, L, M	28	Ag, Cd, Cu, Hg, Pb, Se, Zn	Wagemann 1989
P. hispida	Arctic Ocean (Canada)	1972–77	L, M	390	Hg, MeHg, Se	Smith and Armstrong 1978
P. hispida	Baltic Sea (Sweden)	1979–90	B, K, L	17	Al, As, Ca, Cd, Co, Cr, Cu, Fe, Hg, Mg, Mn, Ni, Pb, Se, V, W, Zn	Frank et al. 1992
P. hispida	Finland	—	Hair	32	Cd, Cr, Hg, Ni, Pb	Hyvärnnen and Sipilä 1984
P. hispida	Finland	1967–68	L, M, O	25	Hg	Henriksson et al 1969
P. hispida	Finland (Lake Saimaa)	1967	K, L, M	7	Hg	Helminen et al. 1968
P. hispida	Finland (Saimaa, Bothnian Bay)	1974–75	B, K, L, M	15	Hg, MeHg, Se	Kari and Kauranen 1978
P. hispida	Greenland	1984–87	K, L, M	12	Hg, organic Hg	Dietz et al. 1990
P. hispida	Gulf of Finland	1976–82	B, K, L, M	11	Cd, Cu, Hg, Pb, Se, Zn	Pertillä et al. 1986
P. hispida	Netherlands (captive)	—	B, Br, K, L	1	Hg, MeHg, Se	van de Ven et al. 1979
P. hispida	North Sea (Germany)	—	L	1	Cd, Cu, Hg, Pb, Zn	Harms et al. 1977/78
P. hispida	North Sea (Germany)	1975	L	1	Cd, Cu, Hg, Pb, Zn	Drescher 1978
P. hispida	Northwest Atlantic (Canada)	—	Claws	14	Hg, MeHg	Freeman and Horne 1973
P. hispida	Okhotsk Sea	1975	O	1	Cd, Zn	Hamanaka et al. 1977
P. largha	Japan	1992	B	1	Butyltin compounds	Iwata et al. 1994
P. vitulina	Alaska	1976–78	K, L	23	As, Cd, Hg, Pb, Se	Miles et al. 1992
P. vitulina	Baltic Sea (Sweden)	1979–90	B, K, L	14	Al, As, Ca, Cd, Co, Cr, Cu, Fe, Hg, Mg, Mn, Ni, Pb, Se, V, W, Zn	Frank et al. 1992
P. vitulina	Eastern North Pacific (U.S.A.)	1970–71	L	13	Hg	Anas 1974a
P. vitulina	England	1988–89	B, K, L	14	Hg	Simmonds et al 1993

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Appendix 10-5 continued

Species	Region	Sample Period	Tissues Sampled	Max. No. of Individuals Sampled	Metals and Trace Elements Reported	References
P. vitulina	Germany	1988	Hair, skin	47	Cd, Hg, Pb	Wenzel et al. 1993
P. vitulina	Great Britain	1968–72	Br, K, L, teeth	31	Cd, Cr, Cu, Hg, Pb, Zn	Heppleston and French 1973
P. vitulina	Japan	1984	K, L	15	Cd, Cu, Hg, Zn	Tohyama et al. 1986
P. vitulina	Netherlands	1974–75	B, Br, K, L, M, O	7	Hg, MeHg, Se	van de Ven et al. 1979
P. vitulina	North Sea (England)	1969–70	Br, K, L, M, O	9	Cd, Hg, Pb	Roberts et al. 1976
P. vitulina	North Sea (Germany)	—	K, L, M	7	Cd, Cu, Hg, Pb, Zn	Harms et al. 1977/78
P. vitulina	North Sea (Germany)	1974–76	Br, K, L	63	Cd, Cu, Hg, Pb, Zn	Drescher et al. 1977
P. vitulina	North Sea (Netherlands)	—	B, Br, K, L, O	9	Cd, Cr, Cu, Fe, Mn, Pb, Zn	Duinker et al. 1979
P. vitulina	North Sea (Netherlands)	1970–71	Br, L	11	As, Cd, Hg, Se, Zn	Koeman et al. 1972
P. vitulina	North Sea (Netherlands)	1975–76	Br, K, L	14	Br, Hg, MeHg, Se	Reijnders 1980
P. vitulina	North Sea (Schleswig-Holstein and Denmark)	1975–76	Br, K, L	16	Br, Hg, MeHg, Se	Reijnders 1980
P. vitulina	North Sea (Sweden)	1979–90	B, K, L	38	Al, As, Ca, Cd, Co, Cr, Cu, Fe, Hg, Mg, Mn, Ni, Pb, Se, V, W, Zn	Frank et al. 1992
P. vitulina	North Sea (U.K.)	—	B, Br, K, L, M, O	15	Cd, Cu, Hg, Pb, Zn	Holden 1975
P. vitulina	Nova Scotia	1971	B, L, M, hair	8	Hg	Sergeant and Armstrong 1973
P. vitulina	Nova Scotia	1972	B, Br, K, L, M, O	1	Hg	Freeman and Horne 1973
P. vitulina	Okhotsk Sea	—	K, L	15	Hg, MeHg, Se	Himeno et al. 1989
P. vitulina	Okhotsk Sea	1975	L, M, O	4	Cd, Zn	Hamanaka et al. 1977
P. vitulina	Scotland	1969–70	Br, K, L, M, O	11	Cd, Hg, Pb	Roberts et al. 1976
P. vitulina	Scotland	1988	B	9	Hg	Simmonds et al. 1993
P. vitulina	St. Lawrence River	1983–90	L	10	Cd, Hg	Béland et al. 1991
P. vitulina	U.K.	1988–89	L	28	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Law et al. 1991, 1992
P. vitulina	Western North Atlantic (Bay of Fundy)	1971	B, Br, L, M	12	Hg, MeHg	Gaskin et al. 1973
P. vitulina	Western North Atlantic (U.S.A.)	1980, 1991	L	7	Hg	Lake et al. 1995
Zalophus californianus	California	1972	K, L	40	Ag, Br, Ca, Cd, Cu, Fe, Hg, K, Mg, Mn, Na, Se, Zn	Martin et al. 1976
Z. californianus	California	1971–72	Br, bone, K, L, M, O	6	Pb	Braham 1973
Z. californianus	Captive	—	L	1	Hg	Theobald 1973
Z. californianus	Oregon	1970s	K, L	7	Cd, Hg	Lee et al. 1977
Z. californianus	Oregon	1970–73	Multiple organs	20	Cd, Hg, MeHg	Buhler et al. 1975

Dashes appear where values were not available in original source.

B = blubber; Bl = blood; Br = brain; K = kidney; L = liver; M = muscle; MeHg = methyl mercury; Mi = milk; O = other

Appendix 10-6. Summary of Selected Surveys of Metals and Trace Elements in Tissues of Odontocete Cetaceans

Species	Region	Sample Period	Tissues Sampled	Max. No. of Individuals Sampled	Metals and Trace Elements Reported	References
Delphinapterus leucas	Alaska (Chukchi Sea)	1989–90	L	6	40 trace elements, MeHg	Zeisler et al. 1993
D. leucas	Alaska	1989–92	L	15	Ag, Hg, Se	Becker et al. 1995
D. leucas	Alaska	—	L	15	Ag, Cd, Hg, Se, V	Mackey et al. 1996

Appendix 10-6 continued

Species	Region	Sample Period	Tissues Sampled	Max. No. of Individuals Sampled	Metals and Trace Elements Reported	References
D. leucas	Baltic Sea (Germany)	—	K, L, M	1	Cd, Cu, Hg, Pb, Zn	Harms et al. 1977/78
D. leucas	Greenland	1984–86	K, L, M	43	Cd, Hg, Se, Zn	Hansen et al. 1990
D. leucas	Greenland	1984–87	K, L, M	6	Hg, Organic Hg	Dietz et al. 1990
D. leucas	Canadian Arctic and St. Lawrence	1981–87	K, L, M	144	Cd, Cu, Hg, Pb, Se, Zn	Wagemann et al. 1990
D. leucas	St. Lawrence River	1988–90	L	35	Cd, Hg	Béland et al. 1991
Delphinus delphis	Australia	—	B, K, L, M, O	32	Cd, Hg, Pb	Kemper et al. 1994
D. delphis	Gulf of Guinea	1975	Lung	1	Cd, Cr, Fe, Hg, Pb, Ti	Viale 1981
D. delphis	Irish Sea	1990–91	L	8	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Law et al. 1992
D. delphis	Tropical Atlantic	1975	B, K, L, M	1	Hg, Se	Martoja and Viale 1977
D. delphis	Mediterranean Sea	1973	B, L, M	1	Hg, Se	Martoja and Viale 1977
D. delphis	Mediterranean Sea (France)	1973	Lung	1	Cd, Cr, Fe, Hg, Pb, Ti	Viale 1981
D. delphis	Mediterranean Sea (France)	1977	B	1	Cd, Cu, Pb	Vicente and Chabert 1978
D. delphis	New Zealand	1970–71	L	2	As, Cd, Hg, Se, Zn	Koeman et al. 1972, 1975
D. delphis	North Pacific	1987	B	1	Butyltin compounds	Iwata et al. 1994
D. delphis	North Sea (Belgium)	1986	L, M	1	Hg, MeHg	Joiris et al. 1991
D. delphis	Wales	1990	L	1	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Law et al., 1991, 1992
D. delphis	Western North Atlantic (U.S.A.)	1987–89	L	3	Cd, Cr, Hg, Mn, Pb, Se	Kuehl et al. 1994
D. delphis	Australia	1989	K, L, M	8	Cd, Cu, Hg, Pb, Se, Zn	Anderson 1991
Globicephala macrorhynchus	California	1971	L	6	Hg	Hall et al. 1971
G. macrorhynchus	Caribbean Sea	1972	K, L, M	5	Hg, MeHg	Gaskin et al. 1974
G. macrorhynchus	Japan	1975	M	12	Hg, Se	Arima and Nagakura 1979
G. macrorhynchus	Western North Atlantic (Georgia)	—	B, L, M	1	Hg, MeHg	Windom and Kendall 1979
G. macrorhynchus	Western North Atlantic (southeastern U.S.A.)	1977	B, L, K	4	Cd, Hg, Se	Stoneburner 1978
G. melaena	Newfoundland	1980–82	B, K, L, M	41	As, Cd, Cu, Hg, Pb, Se, Zn	Muir et al. 1988a
G. melaena	North Atlantic (Faroe Islands)	1977–78	B, K, L, M	30	As, Cd, Cu, Hg, MeHg, Se, Zn	Julsbamn et al. 1987
G. melaena	North Atlantic (Faroe Islands)	1986	B, O	53	Hg	Simmonds et al. 1994
G. melaena	Northwestern Atlantic (Massachusetts)	1986–90	B, L, K, O	17	As, Cd, Cu, Hg, MeHg, Pb, Se	Meador et al. 1993
G. melaena	St. Lawrence River	1983–90	L	2	Cd, Hg	Béland et al. 1991
G. melaena	Tasmania	1981–83	M	2	Hg	Munday 1985
G. melaena	Northwestern Atlantic (Massachusetts)	1990–91	L	8	Ag, Hg, Se	Becker et al. 1995
G. melaena	Northwestern Atlantic (Massachusetts)	1990–91	L	8	20 elements	Mackey et al. 1995
G. melaena	North Atlantic (Faroe Islands)	1986–88	K, L, M, Mi, O	131	As, Cd, Cu, Hg, Se, Zn	Caurant et al. 1993
G. melaena	North Atlantic (Faroe Islands)	1986–88	Blood, urine	40	Cd	Caurant and Amiard-Triquet 1995
G. melaena	North Atlantic (Faroe Islands)	1987	L	14	Hg, organic Hg	Schintu et al. 1992
Globicephala sp.	Australia	1989	K, L	4	Cd, Cu, Hg, Pb, Se, Zn	Anderson 1991
Globicephala sp.	Australia	—	K, L, M, O	12	Cd, Hg, Pb	Kemper et al. 1994
Hyperoodon ampullatus	North Sea (Germany)	—	L, M	1	Cd, Cu, Hg, Pb, Zn	Harms et al. 1977/78

Continued on next page

Appendix 10-6 continued

Species	Region	Sample Period	Tissues Sampled	Max. No. of Individuals Sampled	Metals and Trace Elements Reported	References
H. ampullatus	St. Lawrence River	1983–90	L	1	Cd, Hg	Béland et al. 1991
H. planifrons	Australia	—	Bone	3	Pb	Kemper et al. 1994
Kogia breviceps	Australia	—	B, L, O	5	Cd, Hg, Pb	Kemper et al. 1994
K. breviceps	Western South Atlantic (Argentina)	—	B, K, L, M, O	1	Cd, Cu, Hg, Zn	Marcovecchio et al. 1990, 1994
Lagenorhynchus obscurus	New Zealand	1970–71	L	1	As, Cd, Hg, Se, Zn	Koeman et al. 1972, 1975
L. acutus	Ireland	1989	L	1	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Law et al. 1991
L. acutus	Western North Atlantic (U.S.A.)	1987–89	L	2	Cd, Cr, Hg, Mn, Pb, Se	Kuehl et al. 1994
L. acutus	Western North Atlantic (Massachussetts)	1993	L	4	20 elements	Mackey et al. 1995
L. albirostris	Irish Sea	1989	L	1	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Law et al. 1991, 1992
L. albirostris	Newfoundland	1980–82	K, L, M	27	Cd, Cu, Hg, Pb, Se, Zn	Muir et al. 1988a
L. albirostris	North Sea (Denmark)	1972	B, L, M, O	1	Cu, Hg, Pb, Zn	Andersen and Rebsdorff 1976
Mesoplodon ginkgodens	Japan	1993	B	1	Butyltin compounds	Iwata et al. 1994
Mesoplodon. sp.	Australia	—	B, K, L, M, O	18	Cd, Hg, Pb	Kemper et al. 1994
Monodon monoceros	Arctic Ocean (Canada)	1978–79	B, K, L, M	60	As, Cd, Cu, Hg, Pb, Se, Zn	Wagemann et al. 1983
M. monoceros	Greenland	1984–86	K, L, M	98	Cd, Hg, Se, Zn	Hansen et al. 1990
M. monoceros	Greenland	1984–87	K, L, M	6	Hg, organic Hg	Dietz et al. 1990
Neophocoena phocoenoides	Japan	1973	M	1	Hg, MeHg, Se	Arima and Nagakura 1979
N. phocoenoides	Japan	1981–92	B	3	Butyltin compounds	Iwata et al. 1994
N. phocoenoides	South China Sea	1990	B	1	Butyltin compounds	Iwara et al. 1994
Orcinus orca	Australia	—	K, L	1	Cd, Hg, Pb	Kemper et al. 1994
O. orca	Japan	1986	B	1	Butyltin compounds	Iwata et al. 1994
Phocoena phocoena	Baltic, North Sea (Germany)	—	K, L, M	3	Cd, Cu, Hg, Pb, Zn	Harms et al. 1977/78
P. phocoena	Irish Sea	1988–90	L	36	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Law et al. 1992
P. phocoena	Western North Atlantic (Bay of Fundy)	1969–77	Br, K, L, M, O	146	Hg, MeHg	Gaskin et al. 1972, 1979
P. phocoena	Western North Atlantic (northeastern U.S.A.)	1991	Br, K, L, O	3	As, Cd, Cu, Hg, Pb, Se	Stein et al. 1992
P. phocoena	Western North Atlantic (northeastern U.S.A.)	1990–92	L	6	20 elements	Mackey et al. 1995
P. phocoena	North Sea (Belgium, Denmark)	1987–90	K, L, M	17	Hg, MeHg	Joiris et al. 1991
P. phocoena	North Sea (Denmark)	1972–73	B, L, M, O	4	Cu, Hg, Pb, Zn	Andersen and Rebsdorff 1976
P. phocoena	North Sea (Netherlands)	1970–71	Br, L	6	As, Cd, Hg, Se, Zn	Koeman et al. 1972, 1975
P. phocoena	North Sea (Scotland)	1974	Br, K, L, O	26	Cd, Co, Cr, Cu, Hg, MeHg, Ni, Pb, Zn	Falconer et al. 1983
P. phocoena	Norway	1989–90	K, L	92	Hg, Se	Teigen et al. 1993
P. phocoena	Baltic Sea (Poland)	1989–93	K, L, M	4	Ag, Cd, Cr, Cu, Mn, Ni, Pb, Zn	Szefer et al. 1994
P. phocoena	St. Lawrence River	1983–90	L	9	Cd, Hg	Béland et al. 1991
P. phocoena	Wales	1988	B, L, M, O	4	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Morris et al. 1989
P. phocoena	U.K.	1988–90	L	20	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Law et al. 1991, 1992
Phoceonoides dalli	North Pacific	1987	B	2	Butyltin compounds	Iwata et al. 1994

Appendix 10-6 continued

Species	Region	Sample Period	Tissues Sampled	Max No. of Individuals Sampled	Metals and Trace Elements Reported	References
P. dalli	Northwestern Pacific	NR	Multiple organs	2	Cd, Cu, Fe, Hg, Mn, Ni, Pb, Zn	Fujise et al. 1988
P. dalli	Western North Pacific	—	B, Br, K, L, M, O	3	Cd, Cu, Fe, Hg, Mn, Ni, Pb, Zn	Fujise et al. 1988
P. dalli	Western North Pacific	1978	L, M, O	2	Cd, Zn	Hamanaka and Mishima 1981
Physeter catodon	—	—	M	1	Hg	Suzuki and Miyoshi 1973
P. catodon	Antarctic	1972	M	6	Hg, MeHg	Nagakura et al. 1974
P. catodon	North Pacific	1972	M	7	Hg, MeHg	Nagakura et al. 1974
P. macrocephalus	Australia	—	B, K, L, M	3	Cd, Hg, Pb	Kemper et al. 1994
P. macrocephalus	Mediterranean Sea	1974	Lung	1	Cd, Cr, Fe, Hg, Pb, Ti	Viale 1981
P. macrocephalus	North Sea (Netherlands)	1995	L	1	As, Cd, Cr, Cu, Hg, Ni, Pb, Se, Zn	Law et al. 1996
P. macrocephalus	North Sea (Belgium)	1989	L, M	1	Hg, MeHg	Joiris et al. 1991
P. macrocephalus	Southern Australia	1976	M	414	Hg	Cannella and Kitchener 1992
P. macrocephalus	Australia	1989	M	1	Hg	Anderson 1991
Platanista gangetica	India	1988–92	K, L, M	4	Cd, Cu, Fe, Mn, Ni, Pb, Zn	Kannan et al. 1993a
Pontoporia blainvillei	Western South Atlantic (Argentina)	—	B, K, L, M, O	7	Cd, Cu, Hg, Zn	Marcovecchio et al. 1990, 1994
Pseudorca crassidens	Australia	—	B, K, L, M	38	Cd, Hg, Pb	Kemper et al. 1994
P. crassidens	Eastern North Pacific (British Columbia)	1987	Br, K, L	1	As, Ca, Cd, Cu, Fe, Hg, Mg, Mn, Pb, Se, Zn	Baird et al. 1989
Sotalia guianenses	Surinam	1970–71	L	2	As, Cd, Hg, Se, Zn	Koeman et al. 1972, 1975
Stenella attenuata	Australia	—	B, L, M	2	Hg, Pb	Kemper et al. 1994
S. attenuata	Japan	1972	M	2	Hg, MeHg, Se	Arima and Nagakura 1979
S. attenuata	Eastern Tropical Pacific	1977–83	Multiple organs	27	Cd	André et al. 1990
S. attenuata	Eastern Tropical Pacific	1977–83	Multiple organs	44	Hg	André et al. 1991b
S. coeruleoalba	Eastern North Atlantic (France)	1972–80	K, L, M, O	8	Hg	André et al. 1991a
S. coeruleoalba	Irish Sea	1990–91	L	3	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Law et al. 1992
S. coeruleoalba	Japan	1973–74	M	11	Hg, MeHg, Se	Arima and Nagakura 1979
S. coeruleoalba	Japan	1978–80	Bone	40	Ca, Cd, Cu, Fe, Hg, MeHg, Mn, Ni, Pb, Se, Zn	Honda et al. 1986a
S. coeruleoalba	Japan	1977–79	Multiple organs	55	Hg, MeHg, Se	Itano et al. 1984a,b,c
S. coeruleoalba	Japan	1977–80	M, L, K	59	Cd, Cu, Fe, Hg, Mn, Ni, Pb, Zn	Honda et al. 1983
S. coeruleoalba	Japan	1977–80	Multiple organs	76	Cd, Zn	Honda and Tatsukawa 1983
S. coeruleoalba	Japan	1979	Bone	1	Ca, Cd, Cu, Fe, Hg, Mn, Ni, Pb, Se, Zn	Honda et al. 1984
S. coeruleoalba	Japan	1980	M	5	Hg, MeHg, Se	Itano et al. 1985a,b
S. coeruleoalba	Mediterranean (Italy)	1987–89	Br, K, L, M	23	Cd, Hg, Pb, Se, Zn	Leonzio et al. 1992
S. coeruleoalba	Mediterranean (France)	1972–80	K, L, M, O	27	Hg	André et al. 1991a
S. coeruleoalba	Mediterranean (France)	1973	Lung	1	Cr, Hg	Viale 1981

Continued on next page

Appendix 10-6 continued

Species	Region	Sample Period	Tissues Sampled	Max. No. of Individuals Sampled	Metals and Trace Elements Reported	References
S. caeruleoalba	Mediterranean (France)	1988–90	B, Br, K, L, M, O	13	Hg	Augier et al. 1993
S. coeruleoalba	Mediterranean Sea	1973–92	Multiple organs	6	Cd, Hg, Pb, Se	Viale 1994
S. coeruleoalba	Wales	1988	B, M, O	1	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Morris et al. 1989
S. coeruleoalba	Wales	1990	L	2	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Law et al. 1991, 1992
S. longirostris	Bay of Bengal	1990	B	1	Butyltin compounds	Iwata et al. 1994
Stenella sp.	Australia	—	B, K, L, M	3	Cd	Kemper et al. 1994
Stenella sp.	Caribbean Sea	1972	K, L, M	2	Hg, MeHg	Gaskin et al. 1974
Tursiops gephyreus	Western South Atlantic (Argentina)	—	K, L, M, O	2	Cd, Cu, Hg, Zn	Marchovecchio et al. 1990, 1994
T. gilli	Japan	1973	M	1	Hg, MeHg, Se	Arima and Nagakura 1979
T. truncatus	Australia	—	B, K, L, M, O	24	Cd, Hg, Pb	Kemper et al. 1994
T. truncatus	Belgium (captive)	1989	K, L, M	2	Hg, MeHg	Joiris et al. 1991
T. truncatus	Florida (Gulf and Atlantic coasts)	1990–94	K, L, M	39	Cd, Cu, Zn	Wood and Van Vleet 1996
T. truncatus	Gulf of Mexico (Florida)	1969–71 (captive)	Blood, L	11	As, Cd, Hg, Sb, Se, Zn	Dudok van Heel 1972
T. truncatus	Gulf of Mexico (Florida)	1987–91	L	12	Hg	Rawson et al. 1993
T. truncatus	Gulf of Mexico	1990	L	26	Cd, Cr, Hg, Mn, Pb, Se	Kuehl and Haebler 1995
T. truncatus	Irish Sea	1989	L	2	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Law et al. 1991, 1992
T. truncatus	Mediterranean Sea	1975–86	Multiple organs	3	Ca, Cd, Cr, Cu, Fe, Hg, Pb, Ti, V, Zn	Viale 1994
T. truncatus	Mediterranean Sea	1973–75	B, K, L, M	2	Hg, MeHg, Se	Martoja and Viale 1977
T. truncatus	Mediterranean Sea	1973–75	Lung	3	Cd, Cr, Fe, Hg, Pb, Ti	Viale 1981
T. truncatus	Mediterranean Sea (Corsica)	1975–86	B, K, L, M, O	3	Ca, Cd, Cr, Cu, Fe, Hg, Pb, Ti, V	Viale 1994
T. truncatus	Mediterranean Sea (Italy)	1987–89	Br, K, L, M	6	Cd, Hg, Pb, Se, Zn	Leonzio et al. 1992
T. truncatus	Mediterranean Sea (Italy)	1992	B, L	3	Butyltin compounds	Kannan et al. 1996
T. truncatus	Netherlands (captive)	1970–71	L	3	As, Cd, Hg, Se, Zn	Koeman et al. 1972, 1975
T. truncatus	Wales	1988	B, M, O	1	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Morris et al. 1989
T. truncatus	Western North Atlantic (Georgia, USA)	1971	Br, K, L, M, O	1	Hg	Stickney et al. 1972
T. truncatus	Western North Atlantic (U.S.A.)	1987–89	L	9	Cd, Cr, Hg, Mn, Pb, Se	Kuehl et al. 1994
Ziphius cavirostris	Australia	—	Bone	1	Pb	Kemper et al. 1994
Z. cavirostris	Bermuda	1981	B, K, L, M, O	4	Cd, Cu, Fe, Mn, Ni, Pb, V, Zn	Knap and Jickells 1983
Z. cavirostris	Mediterranean Sea	1974	B, K, L, M	1	Hg, MeHg, Se	Martoja and Viale 1977
Z. cavirostris	Mediterranean Sea (France)	1974	Lung	1	Cd, Cr, Fe, Hg, Pb, Ti	Viale 1981
Z. cavirostris	Western South Atlantic (Argentina)	—	B, K, L, M	1	Cd, Hg	Marcovecchio et al. 1994

Dashes appear where values were not available in original source.

B = blubber, Br = brain, K = kidney, L = liver; M = muscle, MeHg = methylmercury; Mi = milk, NR = not reported, O = other

Appendix 10-7. Summary of Selected Surveys of Metals and Trace Elements in Tissues of Baleen Whales

Species	Region	Sample Period	Tissues Sampled	Max. No. of Individuals Sampled	Metals and Trace Elements Reported	References
Balaena mysticetus	Alaska	—	L	3	Ag, Cd, Hg, Se, V	Mackey et al. 1996
B. mysticetus	Arctic Ocean (Alaska)	1979–88	B, K, L, M	12	Ag, As, Ba, Be, Cd, Cr, Cu, Hg, Fe, Ni, Pb, Sb, Se, Ti, Zn	Bratton et al. 1993; Byrne et al. 1985
Balaenoptera acutorostrata	Antarctic	1980–85	L	135	Cd, Co, Cu, Fe, Hg, Mn, Ni, Pb, Zn	Honda et al. 1986b, 1987
B. acutorostrata	Antarctic	1985	B	1	Butyltin compounds	Iwata et al. 1994
B. acutorostrata	Greenland	1980	K, L, M	24	Cd, Hg, Se, Zn	Hansen et al. 1990
B. acutorostrata	Greenland	1980	K, L, M	22	Cd, Cu, Hg, Pb, Se, Zn	Kapel 1983
B. acutorostrata	Greenland	1984–87	K, L, M	3	Hg, organic Hg	Dietz et al. 1990
B. acutorostrata	St. Lawrence River	1983–90	L	9	Cd, Hg	Béland et al. 1991
B. acutorostrata	Wales	1991	L	1	Cd, Cr, Cu, Hg, Ni, Pb, Zn	Law et al. 1992
Balaenoptera sp.	Australia	—	Bone	5	Pb	Kemper et al. 1994
B. borealis	Tasman Sea	1972	M	9	Hg, MeHg	Nagakura et al. 1974
B. physalus	Southern Ocean	1947–48	M	8	Hg, MeHg	Nagakura et al. 1974
B. physalus	Eastern North Atlantic	1983–86	K, L, M	36	Hg, organic Hg	Sanpera et al. 1993
B. physalus	North Atlantic (Iceland, Spain)	1983–86	K, L, M	118	Cd, Cu, Zn	Sanpera et al. 1996
B. physalus	Mediterranean	1975	Lung	1	Cd, Cr, Fe, Hg, Pb, Ti	Viale 1981
B. physalus	St. Lawrence River	1983–90	L	1	Cd, Hg	Béland et al. 1991
Caperea marginata	Australia	—	B, L, M, O	8	Cd, Hg, Pb	Kemper et al. 1994
C. marginata	Tasmania	1980s	M	1	Hg	Munday 1985
Eschrichtius robustus	Eastern North Pacific (U.S.A.)	1988–91	Br, K, L, O	11	Ag, Al, As, Ba, Cd, Cr, Cu, Fe, Hg, Mn, Ni, Pb, Se, Sn, Sr, Zn	Varanasi et al. 1994

B = blubber; Br = brain; K = kidney; L = liver; M = muscle; MeHg = methyl mercury, Mi = milk; O = other

Appendix 10-8. Summary of Selected Surveys of Metals and Trace Element Concentrations in Tissues of Sea Otters, Sirenians, and Polar Bears

Species	Region	Sample Period	Tissues Sampled	Max. No. of Individuals Sampled	Metals and Trace Elements Reported	References
Enhydra lutris	California	1987–89, ancient	Teeth	13	Pb	Smith et al. 1990
E. lutris	North Pacific (Amchitka Island)	1986–87, ancient	Teeth	10	Pb	Smith et al. 1990
Dugong dugon	Australia	—	K, L, M	2	Hg	Denton and Breck 1981
D. dugon	Australia	1974–78	Br, K, L, M	43	Ag, Cd, Co, Cr, Cu, Fe, Mn, Ni, Pb, Zn	Denton et al. 1980
D. dugon	Sulawesi (Indonesia)	1975	M	2	Hg, MeHg, Se	Miyazaki et al. 1979
Trichechus manatus	Florida	1977–81	K, L, M	54	Cd, Cu, Fe, Hg, Pb, Se	O'Shea et al. 1984
T. manatus	Florida	1982	Br, K, L	8	Cd, Cu, Hg, Pb	O'Shea et al. 1991
Ursus maritimus	Arctic (Greenland, Svalbard)	1978–89	Hair, K, L, M	97	Hg	Born et al. 1991
U. maritimus	Canada	1910–80	Hair	146	Hg	Eaton and Farant 1982
U. maritimus	Alaska	1972	L, M	~62	Hg	Lentfer and Galster 1987
U. maritimus	Arctic	1976–88	Hair	141	Hg	Renzoni and Norstrom 1990
U. maritimus	Canada (Northwest Territories)	1982–84	L	124	Ag, As, Ba, Be, Ca, Cd, Cu, Fe, Hg, K, Mg, Mn, Mo, Na, P, Se, Sr, Ti, V, Zn, Zr	Braune et al. 1991; Norstrom et al. 1986
U. maritimus	Greenland	1984–87	K, L, M	4	Hg, organic Hg	Dietz et al. 1990
U. maritimus	Svalbard	1978–89	K, L	22	As, Cd, Cu, Hg, Pb, Se, Zn	Norheim et al. 1992

B = blubber; Br = brain, K = kidney, L = liver; M = muscle; MeHg = methyl mercury, Mi = milk.

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