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Accumulation pattern of organochlorine pesticides and polychlorinated biphenyls in southern sea otters (*Enhydra lutris nereis*) found stranded along coastal California, USA

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Abstract

Concentrations of PCBs, DDTs (*p,p'*-DDE, *p,p'*-DDD and *p,p'*-DDT), HCHs (α-, β-, γ-isomers), chlordanes (*trans*-chlordane, *cis*-chlordane, *trans*-nonachlor, *cis*-nonachlor and oxychlordane) and HCB (hexachlorobenzene were measured in liver, kidney and brain tissues of adult southern sea otters (*Enhydra lutris nereis*) found stranded along coastal California, USA, during 1992–96. The contamination pattern of organochlorines in sea otters from several locations was in the order of DDTs > PCBs > > CHLs > HCHs > > HCB, whereas those from Monterey Harbor contained greater concentrations of PCBs than of DDTs. Hepatic concentrations of PCBs and DDTs were in the ranges of 58–8700 and 280–5900 ng/g, wet weight, respectively, which varied depending on the geographic location. Sea otters collected from Monterey Harbor contained the greatest concentrations of PCBs and DDTs. In general, accumulation of DDTs, CHLs and PCBs was greater in kidney than in liver, whereas that of HCHs was similar in both the tissues. The gender difference in organochlorine concentrations was less than those reported in cetaceans. The composition of DDTs, HCHs and CHLs compounds in sea otter tissues indicated no recent inputs of these compounds in coastal California. Sea otters that died from infectious diseases, neoplasia and emaciation contained higher concentrations of DDTs than those that died from trauma.

Keywords: Sea otter; PCBs; DDTs; California; Organochlorines;

1. Introduction

Organochlorine pesticides such as 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane (DDT), hexachlorocyclohexanes (HCHs) and chlordanes (CHLs) and polychlorinated biphenyls (PCBs) are ubiquitous anthro pogenic environmental contaminants. Although the production and use of these chemicals have been restricted or banned in most industrialized nations, considerable amounts of these persistent compounds are still cycling in the ecosphere (Tanabe, 1988; Loganathan and Kannan, 1994). The lipophilicity and persistence of these compounds contribute to their bioaccumulation and biomagnification in the food chain. In particular, marine mammals, which are top predators of the marine food web, accumulate significant amounts of organochlorines due to their long life-span, to their low biodegradation capacity and to the presence of lipid-rich blubber (Tanabe et al., 1988; Kannan et al., 1993a, b, 1994; Colborn and Smolen, 1996). Exposure to great concentrations of organochlorines has been associated with epizootics in marine mammals that began in the late 1980s (Addison, 1989). Elevated concentrations of organochlorines in the diets or bodies have been related to reproductive or immunologic dysfunctions in harbor seals (*Phoca vitulina*) in the North, Baltic and Wadden Seas (Helle et al., 1976; Reijnders, 1986; Hall et al., 1992; Heide-Jorgensen et al., 1992), beluga whales (*Delphinapterus leucas*) in the St. Lawrence Estuary (Martineau et al., 1987), striped dolphins (*Stenella coeruleoalba*) in the Mediterranean Sea (Kannan et al., 1993a; Aguilar and Borrell, 1994), California sea lions (*Zalophus californianus*) in coastal California, USA (DeLong et al., 1973) and bottlenose dolphins (*Tursiops truncatus*) in the US Atlantic coast (Kuehl et al., 1994).

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Mustelids such as otters and mink are aquatic mammals that are particularly sensitive to chemical contamination. Although a great deal of controversy exists in relating contaminant exposures to population decline (Kruuk et al., 1991), PCBs have been implicated in the decline in populations of European otters (*Lutra lutra*; Smit et al., 1994). Southern sea otters (*Enhydra lutris nereis*) are the largest of mustelids and distributed throughout the central coast of California with concentrations in Monterey Bay. Sea otters are carnivores and close relatives of river otters. The population growth rate of sea otters is ~5% per year in California, compared with 17–20% per year in southeast Alaska and the Aleutian Islands (Estes, 1990). The reduced rate of increase in the California sea otter population has been attributed to emigration, habitat destruction and increased mortality due to diseases, possibly caused by exposure to toxic contaminants (Estes, 1990). Pre- and post-weaning mortality has been considered as an important cause for the reduced population growth (Riedman et al., 1994). Prevalence of infectious diseases in southern sea otters found dead along the coastal waters of California has raised concern about immune suppression caused by exposure to chemical contaminants (Thomas and Cole, 1996). Although the concentrations of PCBs and DDTs have been reported in various marine mammal species since the 1960s, limited data is available regarding organochlorine concentrations in sea otters from coastal California, USA (Shaw, 1971; Jarman et al., 1996; Estes et al., 1997). Sea otters and their principal prey items (benthic invertebrates) are relatively sedentary and, therefore, are good indicators of local contamination by organochlorines. In this study, PCBs, DDTs (*p,p'*-DDE, *p,p'*-DDD, *p,p'*-DDT), HCHs (α-, β- and γ-isomers), CHLs (*trans*-chlordane, *cis*-chlordane, *trans*-nonachlor, *cis*-nonachlor and oxychlordane) and HCB were measured in liver, kidney and brain tissues of sea otters found stranded along the coastal waters of California during 1992–96. Accumulation of organochlorines by sea otters and its relationship to their health was also examined.

2. Materials and methods

2.1. Samples

Adult sea otters that died along the coast of California were collected through a stranding network coordinated by the US Fish and Wildlife Service and California Department of Fish and Game with the cooperation of other federal agencies, academic and private institutions. The sampling locations are shown in Fig. 1. Among these locations, Half Moon Bay, Moss Landing, Monterey Harbor, and Morro Bay have sheltered fishing and pleasure boat moorings. The four

Fig. 1. Map of California showing sampling locations of sea otters.

otters listed as Monterey Harbor (Table 1) were found within or near the mouth of Monterey Harbor. Estero Bay has oil tanker traffic and off-shore moorings. San Simeon is a relatively less polluted location than the other sites and Diablo Canyon is a more open sea.

Otter carcasses in good postmortem condition were rapidly chilled or frozen and shipped overnight to the National Wildlife Health Center, Madison, WI, for necropsy. Causes of death, collection date, location, sex, length and weight of animals analyzed are presented in Table 1. The otters in this group died from infectious diseases ($n=8$), trauma ($n=5$), unknown causes ($n=4$) or miscellaneous problems ($n=3$) such as neoplasia, emaciation and esophageal impaction. The otters died from trauma due to shark bite or gun shots. The diseased otters were fatally infected by bacteria (e.g. pneumonia), protozoa (e.g. encephalitis), acanthocephalan parasites and fungi (e.g. coccidioidomycosis). All the animals were adults. The adult age designation was based on dentition and total length measurements and based on cementum annuli counts (Morejohn et al., 1975). Generally, adults had no deciduous teeth and indications of at least early wear on their permanent teeth. Adult females were those >105 cm in total length (about 4–5 years of age or more) and males >115 cm (about 5 years or more). The brain, liver and kidney were collected from the carcasses at the time of necropsy, wrapped in aluminum foil or whirlpac bags and stored frozen at −20°C until analysis. Additional details about samples and sampling locations have been reported elsewhere (Kannan et al., 1998).

2.2. Chemical analysis

Organochlorine pesticides and PCBs were analyzed according to methods described elsewhere (Kannan et al., 1995; Nakata et al., 1995). The method consists of

Table 1
Concentrations of organochlorine pesticides and polychlorinated biphenyls (ng/g, wet wt) in liver, kidney and brain tissues of sea otters found stranded along coastal California

Sample ID	Sex	Date collected	Location	Cause of death	Age (years)	Length (cm)	Weight (kg)	Fat (%)	PCBs	DDTs	HCHs	CHLs	HCB
Liver													
13318-001	m	11 Jan 95	Half Moon Bay	trauma	ND	127	23	3.5	140	470	17	14	4.6
13677-001	m	15 Apr 95	Moss Landing	trauma	ND	ND	25.3	3	880	3800	10	53	1.1
11538-001	f	15 May 93	Monterey Harbor	undetermined	ND	122	13.1	3.5	2100	2900	130	91	2.4
12526-001	f	12 Jan 94	Monterey Harbor	disease	13	126	14.8	2.4	8700	2600	50	500	8
13316-001	f	8 Jan 95	Monterey Harbor	disease	ND	116	13.7	2.7	7300	4700	35	280	1.7
11248-001	f	14 Jan 93	Monterey Harbor	neoplasia	11	118	18.8	3.5	7000	5900	15	370	1.4
13712-001	m	26 Jun 95	San Simeon	trauma	10	128	29.3	4.7	120	860	18	8	1.6
14381-001	m	20 Jul 96	Estero Bay	undetermined	2	125	23.1	2.6	360	840	18	24	2.2
14387-001	m	25 Jul 96	Estero Bay	undetermined	7	125	25.5	4	130	540	4.6	5.1	4.5
11937-001	m	18 Nov 93	Estero Bay	trauma	3	126	18	5.2	1400	1600	27	53	1.9
11740-001	m	16 Aug 93	Estero Bay	trauma	9	124	30.5	6.8	58	280	5.3	4.1	0.94
11336-001	f	11 Feb 93	Estero Bay	disease	11	120	14.8	3.9	1200	2500	76	260	1.4
11631-001	f	27 Jun 93	Estero Bay	disease	ND	117	19.7	3.7	470	290	8.7	10	1.1
11510-001	f	5 May 93	Estero Bay	undetermined	7	123	16.4	8.6	79	360	24	7.3	1.7
11019-001	f	5 Sep 92	Estero Bay	emaciation	ND	108	15	3.4	510	1200	65	38	2
14373-001	m	11 Jul 96	Morro Bay	disease	10	123	20.3	8.2	300	1200	5.8	21	1.3
13227-001	m	3 Dec 94	Morro Bay	esophageal impaction	2	130	21.7	7.2	320	1200	11	33	2.3
13791-001	m	30 Jul 95	Morro Bay	disease	12	139	29.4	3.4	280	1800	68	28	2.5
13479-001	f	20 Mar 95	Diablo Canyon	disease	8	124	16.6	2.6	430	1900	26	28	2.1
14063-001	f	21 Dec 95	Diablo Canyon	disease	10	123	23.5	5.4	270	1100	17	23	0.74
Mean ± SD						123 ± 6.1	21 ± 5.3	4.4 ± 1.8	1600 ± 2600	1800 ± 1500	32 ± 31	93 ± 140	2.3 ± 1.6
Kidney													
13318-001	m	11 Jan 95	Half Moon Bay	trauma	ND	127	23	3.9	180	1400	15	30	1.9
13677-001	m	15 Apr 95	Moss Landing	trauma	ND	ND	25.3	3.3	1600	7800	6.4	110	0.48
12526-001	f	12 Jan 94	Monterey Harbor	disease	13	126	14.8	1.7	4600	2300	18	190	1.6
13712-001	m	26 Jun 95	San Simeon	trauma	10	128	29.3	16	140	2800	65	40	2.6
14387-001	m	25 Jul 96	Estero Bay	undetermined	7	125	25.5	4.7	50	700	6.7	8.5	0.88
11019-001	f	5 Sep 92	Estero Bay	emaciation	ND	108	15	1.9	150	500	17	10	0.93
14373-001	m	11 Jul 96	Morro Bay	disease	10	123	20.3	11	1400	8600	19	130	1.9
13791-001	m	30 Jul 95	Morro Bay	disease	12	139	29.4	3.3	560	2500	7.2	23	0.5
10901-001	m	29 Mar 92	Morro Bay	intestinal perforation	ND	129	31.5	3.9	120	290	3.8	4.4	0.28
Mean ± SD						130 ± 8	24 ± 5.7	5.5 ± 4.5	980 ± 3400	3000 ± 2900	18 ± 18	61 ± 63	1.2 ± 0.8
Brain													
13318-001	m	11 Jan 95	Half Moon Bay	trauma	ND	127	23	8.8	69	300	3.3	2.6	0.64
13677-001	m	15 Apr 95	Moss Landing	trauma	ND	ND	25.3	8.7	330	1400	8.1	39	0.5
13712-001	m	26 Jun 95	San Simeon	trauma	10	128	29.3	7.3	40	120	6.5	2.4	0.72
14387-001	m	25 Jul 96	Estero Bay	undetermined	7	125	25.5	11	35	160	4.7	2.1	0.56
13791-001	m	30 Jul 95	Morro Bay	disease	12	139	29.4	7.1	89	350	5.3	4.4	0.28
Mean ± SD						130 ± 5.4	27 ± 2.5	8.6 ± 1.4	110 ± 110	470 ± 480	5.6 ± 1.6	10 ± 14	0.5 ± 0.1

PCBs, polychlorinated biphenyls; DDTs, 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethanes; HCHs, hexachlorocyclohexanes; CHLs, chlordanes; ND, not determined.

extraction of sample tissues (2–6 g) with mixed solvents of diethyl ether (300 ml) and hexane (100 ml) using Soxhlet apparatus for 7 h. Fat content was determined from the K-D (Kuderna-Danish) concentrated aliquots of these extracts. The remaining extracts were then transferred to a glass column packed with 20 g Florisil, followed by elution with a mixture of 150 ml of 80% acetonitrile and 20% hexane-washed water. The eluate from the Florisil column was collected in a separatory funnel containing 100 ml of hexane and 600 ml of hexane-washed water. After partitioning, the hexane layer was concentrated, purified with sulfuric acid, and passed through a 12-g Florisil packed glass column for separation. The first fraction eluted with hexane contained HCB, PCBs, *p,p'*-DDE and *trans*-nonachlor. The second fraction eluted with 20% dichloromethane in hexane contained HCH isomers (α-, β- and γ-HCH), CHLs (*trans*-chlordane, *cis*-chlordane, *cis*-nonachlor and oxychlordane) and *p,p'*-DDD and *p,p'*-DDT.

Each fraction was concentrated and injected into a Hewlett Packard 5890 Series II high resolution gas chromatograph equipped with a ^{63}Ni electron capture detector. A fused silica capillary column (30 m×0.25 mm i.d.) coated with DB-1 (100% dimethyl polysiloxane, J&W Scientific, Folsom, CA) at 0.25 μm film thickness was used for the quantification. The column oven was programmed from an initial temperature of 60°C (1-min hold) to 160°C at a rate of 20°C/min, held for 10 min, and then ramped at a rate of 2°C/min to 260°C with a final hold time of 20 min. The injector and detector temperatures were maintained at 260 and 280°C, respectively. Helium and nitrogen were the carrier and the make up gases, respectively. Concentrations of individually resolved peaks were summed to obtain the total PCB concentration. An equivalent mixture of Kanechlors 300, 400, 500 and 600 with known PCB composition and content was used as the standard. Identification and quantification of individual PCB isomers and congeners based on Kanechlor mixtures have been reported earlier (Tanabe et al., 1987). Organochlorine pesticides were quantified from individually resolved peak areas with corresponding peak areas of standards. Recoveries of PCB congeners and organochlorine pesticides were determined in fortified samples of corn oil carried through the analytical procedure were between 95 and 103%. Detection limits for PCBs and organochlorine pesticides were 0.1 ng/g, wet weight. Concentrations are presented on a wet weight basis, unless specified otherwise. Data were analyzed by Mann–Whitney *U* test and Student's *t*-test to calculate the differences in concentrations. For statistical analysis, all the comparisons were based on lipid normalized concentrations of organochlorines.

3. Results and discussion

3.1. Concentrations

By far, most studies of organochlorines in marine mammals are related to their concentrations in blubber. Other tissues have been examined in relatively few cases and, therefore, distribution of organochlorines within an animal is studied infrequently. Unlike cetaceans and pinnipeds, sea otters do not possess blubber and therefore liver, kidney and brain tissues were used in this analysis.

Organochlorine compounds were detected in all the sea otters collected from various locations in coastal California. Among organochlorines analyzed, DDTs (sum of *p,p'*-DDE, *p,p'*-DDD and *p,p'*-DDT) were the most predominant compounds (1800 ± 1500 ng/g, wet wt, in liver) in sea otters from various locations in coastal California, except those from Monterey Harbor, in which PCBs were more abundant (Table 1). Concentrations of CHLs and HCHs in livers were less than those of PCBs and DDTs and were in the range of 4.1–500 ng/g, wet weight (mean: 93), and 4.6–130 ng/g, wet weight, (mean: 32), respectively. HCB contamination in sea otter tissues was minimal.

Concentrations of DDT in the liver of sea otters from Monterey Harbor and Morro Bay were within the range of those reported for animals collected in 1970 (Shaw, 1971). However, hepatic DDT concentrations of up to 15 000 ng/g, wet weight, were reported in sea otters from Monterey Bay in 1970 (Shaw, 1971), which is 3-fold greater than the maximum concentration found in this study. Mean concentrations of PCBs and DDTs in sea otter livers were 10- and 2-times greater than those reported in 1988–91 (Jarman et al., 1996) and 1991–92 (Estes et al., 1997). Specific reasons for the greater concentrations of PCBs and DDTs measured in this study than those reported by Jarman et al. (1996) and Estes et al. (1997) are not known. Differences in sample biological parameters such as age, sex and sampling locations and the selection of congeners for PCB quantification may account for some of the observed differences. In this study, all the PCB congeners found in otter tissues were quantified, while the number of congeners quantified by Jarman et al. (1996) and Estes et al. (1997) was not indicated. In any case, these results suggest continuing exposure of sea otters to PCBs in coastal California, particularly in Monterey Harbor.

3.2. Tissue distribution and composition

Concentrations of organochlorines in liver, kidney and brain of individual sea otters were compared. Only the animals for which all three of these tissues had been analyzed were used in this comparison. Among these tissues, kidney contained greater concentrations of DDTs, CHLs and PCBs than liver (Table 2). In contrast, the concentrations of HCHs were similar between liver and kidney whereas those of HCB were greater in liver than in kidney. Concentrations of organochlorines were least in the brain. DDT, HCH, CHL and HCB concentrations were significantly higher in kidney ($p < 0.05$) than those in brain. Despite great fat content in brain, concentrations of organochlorines were 3- to 6-fold less than in liver (Table 2). Lipid-normalized ratios of concentrations for organochlorines between liver and brain and kidney and brain were about 2-fold greater than the concentration ratios calculated on wet weight basis (Table 2). While lesser concentrations of organochlorines in brain could be explained by the hematoencephalic barrier, which has been assumed to be responsible for this phenomenon, differences in the lipid-normalized concentrations of organochlorines among tissues suggest differences in the composition of lipids between brain and other tissues. The brain lipid is composed mainly of phosphoglycerides, sphingolipids and

Table 2
Organochlorine concentrations (ng/g, wet wt) and their ratios between liver/kidney, liver/brain and kidney/brain, in sea otter tissues[a]

Tissue/concentration ratio	Fat (%)	PCBs	DDTs	HCHs	CHLs	HCB
Liver	3.7 ± 0.7	310 ± 325	790 ± 610	23 ± 25	22 ± 20	2.9 ± 1.6
Kidney	6.2 ± 5.5	510 ± 640	3000 ± 2800	20 ± 25	42 ± 40	1.3 ± 0.9
Brain	8.6 ± 1.6	110 ± 120	470 ± 530	5.6 ± 1.8	12 ± 16	0.5 ± 0.2
Concentration ratios						
Wet weight basis						
Liver/kidney		1.1 ± 0.9	0.43 ± 0.3	2.6 ± 3.8	0.5 ± 0.4	3.1 ± 1.9
Liver/brain		2.9 ± 0.6	3.5 ± 2.8	4.6 ± 4.9	2.9 ± 1.5	5.7 ± 3.2
Kidney/brain		3.7 ± 1.9	9.0 ± 8.1	3.6 ± 3.8	7.4 ± 6.5	2.2 ± 1.1
Lipid weight basis						
Liver/kidney		1.6 ± 1.3	0.6 ± 0.4	2.8 ± 3.6	0.7 ± 0.3	3.6 ± 1.7
Liver/brain		6.8 ± 2.3	7.1 ± 4.7	10 ± 10	6.7 ± 3.9	14 ± 8.3
Kidney/brain		7.4 ± 5.5	12 ± 2.5	4.6 ± 3.1	11 ± 8.9	3.7 ± 1.9

PCBs, polychlorinated biphenyls; DDTs, 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethanes; HCHs, hexachlorocyclohexanes; CHLs, chlordanes. Values are mean ± SD.

[a] Five animals for which liver, kidney and brain concentrations available were used for calculation.

cholesterol, while lipids in the liver and kidney are usually composed of triacylglycerols in addition to phosphoglycerides, sphingolipids and cholesterol (Long, 1961). Phosphoglycerides, sphingolipids and cholesterol are polar lipids to which organochlorines, particularly PCBs, have less affinity than to triacylglycerols. Relatively lower PCB concentrations in brain than in other tissues have also been found in cetaceans (Tanabe et al., 1981; Duinker et al., 1989; Bernhoft and Skaare, 1994).

p,p'-DDE accounted for greater than 95% of the total DDT concentrations in liver and kidney (Fig. 2). Brain contained a higher percentage of *p,p'*-DDD and *p,p'*-DDT compared to those of liver and kidney. Among HCHs, β-HCH constituted greater than 90% of total HCHs in liver and kidney. Particularly, the proportion of β-HCH was greater in females than in males, which is different from those reported in other marine mammals. This may indicate that the lactational transfer of these compounds to pups has been lower in sea otters. Considerable enrichment of α-HCH (22%) was also found in brain. Occurrence of a noticeable proportion of α-HCH in brain has been reported in birds and marine mammals (Tanabe et al., 1981; Everaarts et al., 1991). This has been suggested to be due to increased polarity of α-HCH enabling it to cross the blood-brain barrier. Among CHLs, *trans*-nonachlor was the major compound, followed by oxychlordane and *cis*-nonachlor. The proportions of *trans*- and *cis*-chlordanes in brain were greater than those in other tissues. Especially, *cis*-chlordane and *cis*-nonachlor were greater in brain than in other tissues, which may be due to the specific affinity of brain lipids to these compounds. The compositions of DDT, HCH and CHL compounds in sea otter tissues are indicative of the lack of fresh inputs of these compounds in coastal California. A greater biotransformation capacity could also lead to the predominance of metabolites rather than the parent compounds in tissues.

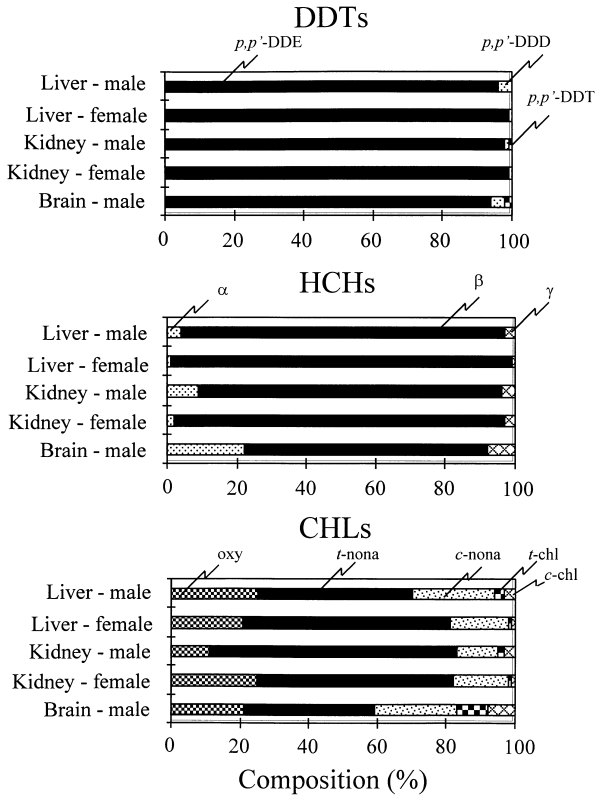

Fig. 2. Composition (%) of 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)-ethane (DDT), hexachlorocyclohexane (HCH) and chlordane (CHL) compounds in liver, kidney and brain tissues of sea otters.

3.3. Geographical variation

Comparison of organochlorines in sea otters from different sampling locations revealed the presence of significantly greater concentrations of PCBs and DDTs ($p < 0.05$) in Monterey Harbor animals (Table 3). Concentrations of PCBs in livers and kidneys of sea otters collected from Monterey Harbor were 4-fold greater than the overall mean (1600 ± 2600 ng/g in liver and 980 ± 1400 ng/g in kidney). The concentrations of DDTs were great in several locations including Monterey Harbor, Moss Landing, Morro Bay and Estero Bay, where the input of DDT from agricultural and urban drainages could have contributed to high concentrations. HCHs and CHLs concentrations were greater in sea otters from Monterey Harbor ($p < 0.05$) than those from other locations. These results indicate the presence of local sources of PCBs in Monterey Harbor.

3.4. Gender variation

Gender differences in organochlorine concentrations (lipid weight basis) were examined based on animals collected from Estero Bay in order to avoid the location-specific influence on this parameter. No gender differences in organochlorine concentrations were found in Estero Bay animals ($p < 0.05$; Table 4). This is different from the observations in cetaceans and pinnipeds, in which adult females usually contain significantly lower concentrations than males, probably due to the lactational transfer of these compounds from mothers to offspring (Tanabe et al., 1994; Nakata et al., 1995; Salata et al., 1995). Even when the overall mean concentrations of organochlorines were compared between sexes, females contained significantly greater ($p < 0.05$) concentrations of PCBs, DDTs and CHLs than males. Greater concentrations of organochlorines in females could be due to the contribution of samples from Monterey Harbor, which were all females. Exclusion of Monterey Harbor females from this comparison resulted in similar concentrations in females and males. Greater concentrations of organochlorines in females or less gender equality in organochlorine concentrations may be due to less transfer of organochlorines through lactation or lower lipid content of milk in sea otters than in cetaceans. Fat content in four milk samples

Table 3
Concentrations of organochlorines (ng/g, wet wt; mean and range) in livers of sea otters from various locations along coastal California

Location	Sex	*n*	Fat (%)[a]	PCBs	DDTs	HCHs	CHLs	HCB
Half Moon Bay	m	1	3.5	140	470	17	14	4.6
Moss Landing	m	1	3	880	3800	10	53	1.1
Monterey Harbor	f	4	3.0	6300	4000	58	310	3.4
				(2100–8700)	(2600–5900)	(15–130)	(91–500)	(1.4–8)
San Simeon	m	1	4.7	120	860	18	8	1.6
Estero Bay	m	4	4.7	490	820	14	22	2.4
				(58–1400)	(280–1600)	(4.6–27)	(4.1–53)	(0.94–4.5)
	f	4	4.9	570	1100	43	79	1.6
				(79–1200)	(290–2500)	(8.7–76)	(7.3–260)	(1.1–2)
Morro Bay	m	3	6.3	300	1400	28	27	2.0
				(280–320)	(1200–1800)	(5.8–68)	(21–33)	(1.3–2.5)
Diablo Canyon	f	2	4.0	350	1500	22	26	1.4
				(270–430)	(1100–1900)	(17–26)	(23–28)	(0.74–2.1)

PCBs, polychlorinated biphenyls; DDTs, 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethanes; HCHs, hexachlorocyclohexanes; CHLs, chlordanes.
[a] Mean.

Table 4
Gender differences in the concentrations of organochlorines (ng/g; mean and range) in the livers of sea otters

Sex	Fat (%)[a]	PCBs	DDTs	HCHs	CHLs	HCB
Overall (wet wt basis)						
Male ($n = 10$)	4.86	400	1300	18	24	2.3
		(58–1400)	(280–3800)	(4.6–68)	(4.1–53)	(0.94–4.6)
Female ($n = 10$)	3.97	2800	2400	45	160	2.3
		(79–8700)	(290–5900)	(8.7–130)	(7.3–500)	(0.74–8)
Estero Bay animals (lipid wt basis)						
Male ($n = 4$)	4.7	11000 ± 12000	20000 ± 14000	350 ± 300	540 ± 510	62 ± 45
Female ($n = 4$)	4.9	15000 ± 12000	28000 ± 28000	1100 ± 970	2000 ± 3100	36 ± 17

PCBs, polychlorinated biphenyls; DDTs, 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethanes; HCHs, hexachlorocyclohexanes; CHLs, chlordanes.
[a] Mean.

taken from California sea otter ranged from 21 to 26% (Jenness et al., 1981). Many phocid seals and cetaceans have higher average concentrations of milk fat than sea otters, exceeding 50% in some stages of lactation (Riedman, 1990). However, further studies with larger numbers of samples are needed to examine this specific pattern of less gender differences in organochlorine concentrations in sea otters.

3.5. Impact on the population

Studies have examined the association between elevated accumulation of organochlorines and impairment of immune function, disease development or adverse health effects in marine mammals (Reijnders, 1986; Martineau et al., 1987; Kannan et al., 1993a; Kuehl et al., 1994; Kuiken et al., 1994; Swart et al., 1994; Corsolini et al., 1995; Colborn and Smolen, 1996). Sea otters may be sensitive to chemical contamination since studies with closely related mustelids such as otters and mink have shown that these animals are sensitive to PCB contamination (Smit et al., 1994). A critical body residue (EC_{50}) for PCBs of 1.2 μg/g, wet weight, has been proposed for effects on litter size in mink (Leonards et al., 1995). Hepatic concentrations of PCBs in adult sea otters collected from Monterey Harbor were 2- to 3-fold greater than the critical residues estimated for mink.

Measured concentrations of organochlorines in livers of sea otters were compared with those reported for marine mammals from other locations in North America. Mean concentration of PCBs in liver was 1.6-fold less than those found in the liver of beluga whales (*D. leucas*) from the St. Lawrence Estuary, Canada (Martineau et al., 1987). The concentrations of PCBs in Monterey Harbor animals were 2- to 3-fold greater than those of beluga whales. Mean concentration of DDTs in the livers of sea otters was similar to that reported for beluga whales (1.3 μg/g, wet wt; Martineau et al., 1987). DDT concentrations in sea otter livers were 3- to 10-fold less than those reported for California sea lions (*Z. californianus*) with premature parturition (DeLong et al., 1973). Similarly, the concentrations of PCBs and DDTs in the liver of sea otters were about 2 to 3-fold less than those reported for harbor seals (*P. vitulina*) collected in 1990–92 from the northeastern USA (Lake et al., 1995).

3.6. Health status

In our previous study, we examined the relationship between butyltin concentrations in tissues and the health of sea otters and found that otters that died from diseases contained greater concentrations than those that died rapidly from trauma and were otherwise relatively healthy (Kannan et al., 1998). Although the number of samples analyzed is small, the concentrations of organochlorines measured in sea otters were compared in relation to their health status (Table 5). Otters that died from infectious diseases and miscellaneous causes such as neoplasia, emaciation and esophageal impaction contained greater concentrations of PCBs and DDTs (Table 5). The ranges of concentrations observed in different mortality groups were wide and there were no significant differences ($p > 0.05$) in the PCB concentrations among different groups based on the health status. Nevertheless, DDT concentrations were significantly greater ($p < 0.05$) in sea otters that died of infectious diseases than those that died from trauma and unknown causes. Examination of a larger number of samples is necessary to evaluate the cause–effect linkage with the slow growth of the California sea otter population as compared with the northern populations.

Few studies have reported the concentrations of organochlorines in sea otters and this study shows the presence of noticeable concentrations of organochlorines in sea otter tissues. Since sea otters lack blubber, organochlorines tend to accumulate in target organs such as liver and kidney, which deserves attention from a health stand point. Concentrations of organochlorines, especially PCBs and DDTs, in Monterey Harbor animals are similar or greater than those of beluga whales with poor recruitment in St. Lawrence

Table 5
Concentrations of organochlorines (ng/g, wet wt; mean and range) in livers of sea otters in relation to their health status

Cause of death	*n*	Fat (%)[a]	PCBs	DDTs	HCHs	CHLs	HCB
Infectious disease	8	4.0	2400 (270–8700)	2000 (290–4700)	36 (5.8–76)	140 (10–500)	2.4 (0.74–8)
Trauma	5	4.6	520 (58–1400)	1400 (280–3800)	15 (5.3–27)	26 (4.1–53)	2.0 (0.94–4.6)
Unknown causes	4	4.7	670 (79–2100)	1200 (360–2900)	44 (4.6–130)	32 (5.1–91)	2.7 (1.7–4.5)
Miscellaneous	3	4.7	2600 (320–7000)	2800 (1200–5900)	30 (11–65)	150 (33–370)	1.9 (1.4–2.3)

PCBs, polychlorinated biphenyls; DDTs, 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethanes; HCHs, hexachlorocyclohexanes; CHLs, chlordanes.
[a] Mean.

Estuary, Canada (Martineau et al., 1987). Examination of a larger number of animals would provide information on the effect of organochlorines on recruitment of sea otters in California.

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