

# The Potential Impacts of Pollution on Humpback Dolphins, with a Case Study on the Hong Kong Population

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## Abstract

Indo-Pacific humpback dolphins (*Sousa chinensis*) inhabit coastal and estuarine waters close to anthropogenic sources of pollution. Detailed studies of contaminant levels on Hong Kong's population of humpback dolphins showed high levels of organochlorines, in particular DDT, and mercury contamination. These contaminant levels are high enough to impact humpback dolphin health. It is likely that several other populations of humpback dolphins are contaminated to a similar degree, and urgent research is warranted. In addition to organochlorine and trace element pollution, the impacts of butyltin (BT), polyaromatic hydrocarbons (PAHs), tris(4-chlorophenyl)methane (TCPMe), tris(4-chlorophenyl)methanol (TCPMeOH), and sewage pollution on humpback dolphin populations should be investigated as a matter of urgency.

**Key Words:** Indo-Pacific humpback dolphin, *Sousa chinensis*, pollution, organochlorine, trace element, butyltin, PAH, TCPMe, TCPMeOH, sewage, Hong Kong

## Introduction

Humpback dolphins (genus *Sousa*) are coastal and estuarine-dwelling species, which range throughout the coastal waters of Africa, Asia, and Australia, from northern Australia to Shanghai in the west, to West Africa in the east (Jefferson & Karczmarski, 2001; Ross et al., 1994). Concerns over the status of these dolphins have been voiced at an international level; for example, they have been highlighted by the International Whaling Commission (IWC) (2003) and International Union for the Conservation of Nature and Natural Resources (IUCN) as species at risk due to their distribution, which puts them in proximity to areas of high human density and potentially threatening activities (Perrin, 1989; Reeves & Leatherwood, 1994; Reeves et al., 2003).

One threatening activity is the input of anthropogenic pollutants into the marine environment. This paper reviews this threat for *Sousa* through the use of a case-study population in Hong Kong as an indicator of the potential scale of impacts that pollution has on humpback dolphins.

## Organochlorines

The presence in marine mammals of toxic organochlorines, in particular polychlorinated biphenyls (PCBs) and the pesticide dichlorodiphenyltrichloroethane (DDT), has received much scientific attention (Aguilar & Raga, 1990, 1993; Johnston et al., 1996; O'Shea & Brownell, 1994; Reijnders, 1996; Simmonds, 1992). Organochlorines are a significant threat to wildlife for several reasons. They act as immuno-suppressants, increasing the likelihood of mortality from infection (Friend & Trainer, 1974; Koller & Thigpen, 1973; Lahvis et al., 1995; Loose et al., 1978), and they also can disrupt mammalian reproduction (Allen & Barsotti, 1976; Kihlström et al., 1992; Platnow & Karstad, 1973). In marine mammals, organochlorine contamination has been linked with physical deformities (Zakharov & Yablokov, 1990), reproductive abnormalities and failure (Baker, 1989; Duinker et al., 1979; Reijnders, 1980, 1986), disruption of the immune system (DeGuise et al., 1998; Lahvis et al., 1995; Ross et al., 1995; de Swart et al., 1994), testosterone deficiencies in Dall's porpoise (*Phocoenoides dalli*) (Subramanian et al., 1987b), lipid metabolism abnormalities in striped dolphins (*Stenella coeruleoalba*) (Kawai et al., 1988), and the decline of beluga whales (*Delphinapterus leucas*) in the St. Lawrence Estuary (Martineau et al., 1987, 1988, 1994).

Organochlorines also can cause mortality directly. In particular, DDT can kill by direct action on the mammalian nervous system. Contamination by DDT in the brain has resulted in the death of various species of small mammal at relatively low concentrations: 17-34  $\mu\text{g}\cdot\text{g}^{-1}$  in shrews (*Blarina brevicauda*); 25  $\mu\text{g}\cdot\text{g}^{-1}$ , in bats

(*Myotis lucifugus*), and 45-50  $\mu\text{g}\cdot\text{g}^{-1}$  in laboratory rats (*Rattus norvegicus*) and mice (*Mus musculus*) (Blus, 1978; Clark, 1981; Clark et al., 1978; Dale et al., 1963; Gingell & Wallcave, 1974; Hayes, 1965; Henderson & Woolley, 1969). Although knowledge of toxic concentrations to larger mammals is scant, they appear to be of a similar level as these aforementioned small mammals (Osweiler et al., 1985).

Marine mammals occupy a high trophic level and, so, are especially susceptible to organochlorine toxicity, accumulating high levels through eating contaminated prey (e.g., Reijnders, 1986; Vos, 2000). Moreover, cetaceans are deficient in certain vital enzymes for metabolising and detoxifying organochlorines, thus increasing their vulnerability to this class of contaminants. In mammals, mono-ortho and non-ortho PCB congeners are metabolised by 3-methylcholanthrene (MC), a cytochrome P450 enzyme (sub-family CYP1A), with phenobarbital (PB); cytochrome P450 sub-family CYP2B) also being involved in the metabolisation of mono-ortho PCBs (Boon et al., 1997). Cetaceans are deficient in PB enzymes, however, and display low activities of MC enzymes, which reduce their ability to detoxify toxic PCBs (Tanabe et al., 1988; Watanabe et al., 1989).

In addition, large quantities of lipophilic organochlorines can be stored in the blubber layer of cetaceans. Although when compartmentalised in the blubber, organochlorine contaminants do not exert toxic effects when animals become debilitated or lactate, lipids become mobilised from the blubber layer into the cetacean body taking with them organochlorines, where they impact the toxicological effects on body systems (Cockcroft et al., 1989; Morris et al., 1989). As a result, organochlorines have been implicated in the mass mortalities of several species of cetacean (Aguilar & Raga, 1990, 1993; Borrell & Aguilar, 1991; Simmonds, 1992); debilitated cetaceans mobilised contaminated lipids and these contaminants severely disrupted immune systems, resulting in large-scale mortalities from pathogens that the animals would have normally been able to combat.

#### *Organochlorine Contamination in Hong Kong:*

##### *A Case Study*

Humpback dolphins inhabit several estuaries immediately adjacent to large industrial cities and centers of population (e.g., Shanghai, Hong Kong, and Singapore). Studies on organochlorine pollutants in humpback dolphins have largely been limited to studies on the Indo-Pacific humpback dolphin (*S. chinensis*) in Hong Kong (Minh et al., 1999; Parsons & Chan, 1998). Therefore, the

Hong Kong dolphin population is a good model to assess the potential impacts of contamination in humpback dolphin populations from other highly industrialized areas.

Prior to the investigations on humpback dolphins in Hong Kong, several studies on various matrices indicated that organochlorines in the region were a cause for concern. For example, Ip (1983) examined PCB and pesticide levels in human breast milk taken from Hong Kong patients and discovered unusually high levels of DDT (1.21-10.27  $\mu\text{g}\cdot\text{g}^{-1}$  lipid wt), DDE (3.36-28.57  $\mu\text{g}\cdot\text{g}^{-1}$  lipid wt), and HCH derivatives (3-47.04  $\mu\text{g}\cdot\text{g}^{-1}$  lipid wt). These concentrations are, incidentally, some of the highest levels found in the world (Ip & Phillips, 1989). Phillips (1985) determined organochlorine levels in green-lipped mussels (*Perna viridis*) in Hong Kong and discovered that there was notable DDT contamination ( $\Sigma\text{DDT}$  50-2,043  $\text{ng}\cdot\text{g}^{-1}$  dry wt), localized pockets of PCB pollution (up to 1,904  $\text{ng}\cdot\text{g}^{-1}$  dry wt), but low levels of HCH. No HCB was detected, however. A later paper (Phillips, 1990) on the same test species noted even higher levels of organochlorines ( $\Sigma\text{DDT}$  248-2,613  $\text{ng}\cdot\text{g}^{-1}$  dry wt;  $\Sigma\text{PCB}$  245-1,667  $\text{ng}\cdot\text{g}^{-1}$  dry wt;  $\Sigma\text{HCH}$  263-567  $\text{ng}\cdot\text{g}^{-1}$  dry wt). Liao (1983) recorded organochlorine levels in seabed sediments from the Pearl River, adjacent to Hong Kong and reported values of HCHs ranging from 41.9 to 101.4  $\text{ng}\cdot\text{g}^{-1}$  dry weight and  $\Sigma\text{DDT}$  from 6.5 to 14.5  $\text{ng}\cdot\text{g}^{-1}$  dry weight. The proportion of the total DDT burden that was present as DDT, as opposed to its derivatives (DDD and DDE), was high in the Liao (1983) study (e.g., DDE concentrations of 0.6-1.4  $\text{ng}\cdot\text{g}^{-1}$  dry wt versus DDT concentrations of 4.8-9.9  $\text{ng}\cdot\text{g}^{-1}$  dry wt), indicating recent inputs of DDT into the Pearl River Estuary. DDT use was not regulated in China until after the Liao (1983) study (Wolfe et al., 1984), however. As for contaminant levels in vertebrates, Chiu et al. (1991) reported PCB levels in fish (Mozambique tilapia, *Tilapia mossambica*) collected from the Shing Mun River and noted  $\Sigma\text{SPCB}$  concentrations of 12.9 to 181.6  $\text{ng}\cdot\text{g}^{-1}$  wet weight. The relatively high level of contamination suggested that local industries were using PCBs and illegally dumping these chemicals or products containing them.

More recent studies also indicated significant levels of contamination in the region. Hong et al. (1995) reported PCB concentrations of 3.2 to 16  $\text{ng}\cdot\text{g}^{-1}$ , HCHs from nondetectable concentrations to 2.3  $\text{ng}\cdot\text{g}^{-1}$  and  $\Sigma\text{DDT}$  from 1.38 to 25.4  $\text{ng}\cdot\text{g}^{-1}$  in sediments from Victoria Harbor, Hong Kong. Connell et al. (1998) also reported PCBs to be widely distributed in marine sediments in Hong Kong waters, with concentrations of 5 to 9.8  $\text{ng}\cdot\text{g}^{-1}$ , and elevated concentrations in some areas

such as typhoon shelters and harbors. Data on seabed sediment contamination in the Pearl River, adjacent to Hong Kong, were provided by Hong et al. (1999) ( $\Sigma$ DDT 1.36-8.99 ng.g<sup>-1</sup>;  $\Sigma$ PCB 0.18-1.8 ng.g<sup>-1</sup>;  $\Sigma$ HCH 0.28-1.23 ng.g<sup>-1</sup>). In this study, the highest sediment concentrations were noted in Shenzhen Bay, close to Castle Peak, Sha Chau, and Neilingding Island, all of which are peak areas of humpback dolphin abundance (Jefferson, 2000). Mai et al. (2002) noted even higher concentrations ( $\Sigma$ DDT 2.6-115.6 ng.g<sup>-1</sup>;  $\Sigma$ PCB 10.2-12.5 ng.g<sup>-1</sup>;  $\Sigma$ BCH up to 2.6 ng.g<sup>-1</sup>) in the Pearl River Estuary, with extremely elevated concentrations in Macau ( $\Sigma$ DDT 1629 ng.g<sup>-1</sup>;  $\Sigma$ PCB 339 ng.g<sup>-1</sup>), on the opposite side of the Estuary from Hong Kong. Hong et al. (1999) also examined organochlorine levels associated with suspended particles in the Pearl River water column and found higher levels of organochlorine contamination than in sediments ( $\Sigma$ DDT 0.67-257 ng.g<sup>-1</sup>;  $\Sigma$ PCB up to 58.2 ng.g<sup>-1</sup>;  $\Sigma$ HCH 1.28-125 ng.g<sup>-1</sup>), with the highest concentrations reported in Shenzhen Bay, off Hengmen on the eastern side of the Pearl River Estuary and upriver at Humen. Although Hong et al. (1999) stated that the ratio of DDE and of DDD to DDT indicated that input of this class of chemical into the environment was not recent, when looking at the makeup of total DDT concentrations in suspended particulates samples collected at the upper reaches of the estuary (Humen), 77% was present as DDT, with only 9% of the total in the form of DDE, which supports the argument that the input of DDT contaminations into the estuary is relatively recent. From DDT profiles in sediment, Mai et al. (2002) also considered the DDT contamination in the Pearl River Estuary to be relatively recent.

Commercially important fish collected at sea or purchased in Hong Kong markets were analysed for organochlorines by Dickman & Leung (1998). Low concentrations of DDT and its metabolites were detected in their study (1.28-16.17 ng DDE.g<sup>-1</sup> wet wt; 1.56-27.41 ng DDD.g<sup>-1</sup> wet wt; 2.83-32.81 ng DDT.g<sup>-1</sup> wet wt). In individual specimens, low concentrations of other organochlorines also were discovered, including three PCB congeners (1.11 ng PnCB-110.g<sup>-1</sup> wet wt; 1.38 ng PnCB-119.g<sup>-1</sup> wet wt; 0.32 ng HxCB-153.g<sup>-1</sup> wet wt), HCBs (3.92 ng.g<sup>-1</sup> wet wt), and Heptachlor (6.60 ng.g<sup>-1</sup> wet wt). Generally, however, Dickman and Leung found concentrations of most non-DDT organochlorine contaminants were below detection limits (i.e., <0.01 ng.g<sup>-1</sup> wet wt).

Chan et al. (1999) also reported organochlorine levels in fish purchased in Hong Kong markets ( $\Sigma$ DDT 3.3-76 ng.g<sup>-1</sup> wet wt;  $\Sigma$ PCB <0.01-94 ng.g<sup>-1</sup> wet wt; Lindane <0.1-15 ng.g<sup>-1</sup> wet wt; Dieldrin 0.29-1.1 ng.g<sup>-1</sup> wet wt; Chlordane 0.83-

7.6 ng.g<sup>-1</sup> wet wt), although it should be noted that these market fish may not necessarily have been caught in Hong Kong waters, and in riverine fish (Mozambique tilapia, *Tilapia mossambica*) from Tai Wai and Fo Tan in Hong Kong ( $\Sigma$ DDT 27-71 ng.g<sup>-1</sup> wet wt;  $\Sigma$ PCB 12-43 ng.g<sup>-1</sup> wet wt;  $\Sigma$ Chlorobenzene 2.6-4.7 ng.g<sup>-1</sup> wet wt; Chlordane 6.3-48 ng.g<sup>-1</sup> wet wt; Lindane 5.2-28 ng.g<sup>-1</sup> wet wt; Dieldrin <0.1 ng.g<sup>-1</sup> wet wt; and Mirex 0.07-0.63 ng.g<sup>-1</sup> wet wt). Despite being higher than the concentrations reported in Dickman & Leung (1998), Chan et al. (1999) noted that organochlorine levels in the fish examined in their study generally were low, and they further suggested that there may have been a decrease in environmental PCB levels between the Chiu et al. (1991) study referred to above and the Chan et al. (1999) study; however, the ratio of DDE to total DDT in the Chan et al. (1999) samples varied greatly (from 75% DDE to 15% DDE). In the Great Lakes in Canada, the percentage of DDE varied from 50% to 70% of the total DDT burden two decades after DDT use was banned in the country (Muir et al., 1990; Newsome & Andrews, 1993), and these ratios also should be the case in Hong Kong because DDT was banned for a similar period of time. This again suggests that in some areas of Hong Kong and the surrounding region, DDT may have been released recently into the marine environment (Chan et al., 1999).

Pollution studies in Hong Kong, therefore, indicate that, on one hand, PCB contamination is relatively low, although there may be local hotspots of contamination; however, the level of DDT contamination is much greater and in some areas this contamination has entered the environment recently.

To date, two studies have investigated organochlorine contamination in Hong Kong humpback dolphins (Minh et al., 1999; Parsons & Chan, 1998), and the organochlorine contaminant levels reported from these studies are summarized in Table 1. There were relatively low levels of most pesticides, including HCHs (0.01-6.88  $\mu$ g.g<sup>-1</sup> lipid wt), although PCB concentrations were slightly higher (0.19-124.98  $\mu$ g.g<sup>-1</sup> lipid wt). These results, and the profile of contaminant concentrations, generally echoed the data reported for sediments and fish species. Very high concentrations of DDT were recorded in Hong Kong humpback dolphins (1.00-380.86  $\mu$ g.g<sup>-1</sup> lipid wt), however. To put this level of contamination into context, in a comparison between ten species of coastal small cetaceans from the North Pacific, South China Sea, and Indian Ocean, Minh et al. (2000a, 2000b, 2000c) noted that animals from Hong Kong (including humpback dolphins) were the most highly DDT contaminated animals in the study.

**Table 1.** Concentrations of organochlorine compounds detected in humpback dolphins from Hong Kong; all concentrations are expressed in  $\mu\text{g}\cdot\text{g}^{-1}$  lipid weight. Data are from Parsons & Chan (1998) and italicised data are from Minh et al. (1999). Specimens SC93-25/05, SC94-28/04, and SC95-11/02 also were examined by Minh et al. (1999), but the Parsons & Chan (1998) data have been used in preference because the samples were not as degenerated as those analysed in the latter study). The notation "n.d." indicates that the contaminants were not detectable.

Specimen	SC93-25/05	SC94-28/04	SC94-23/12	SC95-28/05	SC95-11/02	SC95-02/04	SC95-22/06	SC95-14/09	SC95-25/05	SC96-29/08	SC96-31/08	SC96-26/05	SC97-31/05A	SC97-31/05B	SC96-09/08	SC97-27/03
Sex	M	M	M	M	M	M	M	M	F	M	M	F	M	F	F	F
Pentachlorobenzene	n.d.	n.d.	-	0.02	n.d.	n.d.	0.03	n.d.	0.01	-	-	-	-	-	-	n.d.-0.03
Hexachlorobenzene	0.08	1.84	-	0.13	0.02	n.d.	0.14	0.06	0.23	-	-	-	-	-	-	n.d.-1.84
$\Sigma$ Chlorobenzene	0.01	1.84	-	0.15	0.03	n.d.	0.16	0.06	0.24	-	-	-	-	-	-	n.d.-1.84
$\alpha$ -HCH	0.01	0.18	-	0.15	0.02	n.d.	0.09	0.03	0.06	-	-	-	-	-	-	n.d.-0.18
$\beta$ -HCH	0.06	0.56	-	5.52	0.02	0.04	0.17	0.01	0.14	-	-	-	-	-	-	0.01-5.52
$\gamma$ -HCH	0.01	n.d.	-	0.08	n.d.	n.d.	0.03	n.d.	n.d.	-	-	-	-	-	-	n.d.-0.08
$\Sigma$ HCH	0.08	0.74	0.88	5.76	0.22	0.04	0.28	0.04	0.20	4.67	6.88	0.10	0.67	0.52	1.67	0.01-6.88
HCB	0.52	0.13	0.12	-	0.05	-	-	-	-	0.20	0.04	0.06	0.32	1.08	0.10	0.04-0.52
Aldrin	0.01	0.80	-	0.17	n.d.	n.d.	0.06	0.36	0.09	-	-	-	-	-	-	n.d.-0.80
Dieldrin	0.03	1.09	-	0.59	0.11	1.02	0.07	n.d.	0.16	-	-	-	-	-	-	n.d.-1.09
$\Sigma$ Dieldrin	0.04	1.89	-	0.76	0.11	1.02	0.13	0.36	0.25	-	-	-	-	-	-	0.04-1.89
Oxychlorodane	0.02	1.30	-	0.14	0.08	n.d.	n.d.	n.d.	n.d.	-	-	-	-	-	-	n.d.-1.30
Hept Epox	0.03	0.45	-	0.07	0.03	0.06	0.01	0.01	0.01	-	-	-	-	-	-	0.01-0.45
t-Chlordane	0.01	0.54	-	0.01	n.d.	n.d.	0.01	0.02	0.02	-	-	-	-	-	-	n.d.-0.54
c-Chlordane	0.02	3.11	-	0.05	0.02	n.d.	0.02	0.07	0.03	-	-	-	-	-	-	n.d.-3.11
t-Nonachlor	0.07	16.25	-	0.81	0.30	0.01	0.05	0.12	0.06	-	-	-	-	-	-	0.01-16.25
c-Nonachlor	0.03	3.70	-	0.09	0.03	n.d.	0.02	0.06	0.03	-	-	-	-	-	-	n.d.-3.70
$\Sigma$ Chlordane	0.18	25.35	1.71	1.17	0.45	0.08	0.12	0.28	0.16	0.87	1.28	0.08	0.67	1.18	0.67	0.08-25.35
Mirex	0.01	2.01	-	0.18	0.05	n.d.	0.03	0.32	0.03	-	-	-	-	-	-	n.d.-2.01
TCPMe	0.39	0.53	0.19	-	0.14	-	-	-	-	0.14	0.23	0.01	0.12	0.41	0.19	0.02-0.53
TCPMeOH	0.87	0.44	0.5	-	0.09	-	-	-	-	0.53	0.53	0.02	0.29	0.15	0.28	0.02-0.87
$\Sigma$ PCB	0.59	124.98	91.17	15.17	22.42	0.19	2.46	6.02	2.32	43.33	46.88	6.11	65.79	42.25	43.33	0.19-124.98
p,p'-DDE	5.71	283.82	-	257.62	44.89	0.43	6.29	10.16	6.45	-	-	-	-	-	-	0.43-283.82
p,p'-DDD	3.83	51.98	-	56.93	26.03	0.41	9.07	21.96	11.51	-	-	-	-	-	-	0.41-56.93
p,p'-DDT	4.14	45.06	-	13.56	24.01	0.17	5.96	0.31	8.78	-	-	-	-	-	-	0.17-45.06
$\Sigma$ DDT	13.68	380.86	97.05	328.11	95.09	1.00	21.32	32.43	26.74	203.33	193.75	9.44	105.26	77.46	146.67	1.00-380.86

When comparing the concentrations of DDT to its metabolites in humpback dolphins, the mean ratio of DDE:DDD:DDT was 35%:40%:25%. In comparison, the ratio in coastal-dwelling Hong Kong finless porpoises (*Neophocaena phocaenoides*) was 46%:35%:19% (Parsons & Chan, 1998), and for offshore bottlenose dolphins (*Tursiops truncatus*) from the South China Sea, stranded in Hong Kong, the ratio was 77%:15%:8% (Parsons & Chan, 2001). The relatively high proportions of unmetabolised DDT in the two coastal species suggest a closer proximity to the source of DDT—spatially, temporally, and trophically (Parsons & Chan, 2001). The slightly higher proportion of underived DDT in humpback dolphins versus finless porpoises suggests that humpback dolphins may be closer to the source of the DDT than the latter species, which makes sense because, in Hong Kong, humpback dolphins inhabit the estuarine waters of the Pearl River Estuary, whereas finless porpoises inhabit more oceanic waters (Jefferson et al., 2002; Parsons, 1998a; Parsons et al., 1995).

Zhang et al. (1999) investigated organochlorine levels in sediment cores from Macau (which is situated on the opposite side of the Pearl River Estuary to Hong Kong) and noted that high DDT concentrations were found in sediments dating from 1976 to 1981, then concentrations dropped significantly between 1982 and 1991, but the highest concentrations were recorded between 1992 and 1994, after which levels dropped again. The highest DDT concentrations were recorded in the 1993 sediment layer ( $\Sigma\text{DDT}=79.0 \text{ ng.g}^{-1}$ ; DDE:DDD:DDT=38.5%:18.8%:42.7%). There also was a peak in benzenehexachloride (BHC) concentrations recorded in these sediments ( $82.3 \text{ ng.g}^{-1}$ ). This 1993 contaminant peak occurred a decade after DDT (and BHCs) theoretically was banned in China, but the authors put forward the case that the DDT peak coincided with a period of significant development and construction in the urban areas adjacent to the Pearl River, with a significant decrease in the area of cultivated land. A significant amount of DDT residues (and other pesticides such as BHCs) in agricultural soils possibly was mobilised and transported by surface runoff into the Pearl River Estuary. Such an input of DDT into the Pearl River Estuary would help explain the apparent recent input of DDT into Hong Kong's cetacean population. Due to this recent input of DDT, the implication is that over time concentrations in Hong Kong cetaceans will increase progressively to even higher levels because of organochlorine bioaccumulation.

Thus, humpback dolphins in Hong Kong display high levels of organochlorine, notably DDT,

contamination. What is the implication of this contamination on the health of this population? Kannan et al. (2000) suggested a PCB threshold level of  $17 \mu\text{g.g}^{-1}$  lipid weight, above which toxic effects occur in marine mammals; half of the Hong Kong humpback dolphin samples exceed this threshold level (Table 1). In UK cetaceans, Jepson et al. (1998) recorded increased prevalence of infectious disease in animals with blubber PCB concentrations of greater than  $29.4 \mu\text{g.g}^{-1}$  (wet wt); at least seven (44%) of the Hong Kong humpback dolphins exceeded this level. Few studies have produced health effect threshold levels for DDT in marine mammal blubber; however, Lahvis et al. (1995) recorded immune system dysfunction in bottlenose dolphins with DDE levels in the blood of  $0.0127\text{--}0.5363 \mu\text{g.g}^{-1}$ . Although blood DDE levels have yet to be recorded from Hong Kong cetaceans, high tissue levels of DDT would indicate that high blood levels of DDT are likely.

Subramanian et al. (1987b) discovered reproductive system dysfunction (i.e., reduced testosterone levels) in porpoises with blubber concentrations of PCBs ranging from  $5.62\text{--}17.8 \mu\text{g.g}^{-1}$  (wet wt) and DDT ranging from  $7.61$  to  $16.5 \mu\text{g.g}^{-1}$  (wet wt). Thirteen (81%) of the Hong Kong cetaceans exceeded this threshold level at which hormone depletion would occur.

Contaminant levels in stranded animals do not necessarily reflect contaminant burdens in the entire population; they may be biased towards higher pollutant loads (because heavily contaminated animals may be more likely to suffer morbidity or mortality). Nonetheless, there are indications that organochlorine contamination could disrupt the immune and reproductive systems in at least a proportion of the humpback dolphin population in Hong Kong.

Parsons (1997a) estimated the highest body burden for Hong Kong's humpback dolphins was  $20.6 \text{ mg.kg}^{-1}$  body weight  $\Sigma\text{PCB}$  and  $62.8 \text{ mg.kg}^{-1}$  body weight  $\Sigma\text{DDT}$  (using contaminant data from Parsons & Chan, 1998). The  $\text{LD}_{50}$  level for DDT in mammals (i.e., the level of contamination at which 50% of the sample animals die as a result of a lethal dose of chemicals) is  $>60 \text{ mg.kg}^{-1}$  body weight (Pimentel, 1971); however, as mentioned above, much of the organochlorine body burden in cetaceans is compartmentalised in the blubber and relatively, toxicologically, inert. If these lipids were mobilised (in the event of a debilitating illness or decrease in prey availability, for example), and if the above  $\text{LD}_{50}$  value is applicable to cetaceans (which could be debatable), the DDT body burdens in Hong Kong humpback dolphins could, theoretically, be high enough to cause mortality.

Another toxicological issue is the potential for organochlorine poisoning in neonatal animals because adult females pass on their organochlorine burden to their offspring. Organochlorines are lipophilic and, therefore, can be passed on to neonatal animals via lactation (Cockcroft et al., 1989; Morris et al., 1989; Subramanian et al., 1987a). This is particularly pertinent because cetacean milk has a high lipid content and is consumed in large quantities during the initial, rapid, development and growth of neonates. Cockcroft et al. (1989) estimated that 80% of an adult female's PCB and DDT burden is transferred to her firstborn calf.

Parsons & Chan (1998) recorded organochlorines in milk extracted from the stomach of a humpback dolphin calf (Table 2). These contaminant levels showed that organochlorine transfer via lactation definitely occurs and, moreover, that the amount of organochlorines ingested by calves was substantial. To put the milk organochlorine levels in context, the World Health Organization's tolerable daily intake limits for humans for  $\Sigma$ PCB and  $\Sigma$ DDT are  $1 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$  and  $20 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$ , respectively. The limits for a *S. chinensis* calf weighing approximately 10 kg would, theo-

retically, be  $10 \mu\text{g}\cdot\text{day}^{-1}$   $\Sigma$ PCB and  $200 \mu\text{g}\cdot\text{day}^{-1}$   $\Sigma$ DDT, respectively (although it should be noted that guidelines developed as the result of testing laboratory mammal species, and devised for humans, may not necessarily be applicable to dolphins). Dolphin milk has a high lipid content of approximately 20-40% and, extrapolating from the concentrations found in this study, the mother's milk had an approximate contaminant level of 500-1,000  $\mu\text{g}\cdot\text{l}^{-1}$   $\Sigma$ PCB or 3,740-7,500  $\mu\text{g}\cdot\text{l}^{-1}$   $\Sigma$ DDT. The daily recommended intake of these two organochlorines could, in a worse case scenario, and assuming that health guidelines for humans are applicable to dolphins, be exceeded if the calf consumed more milk per day than a mere 27 ml for  $\Sigma$ DDT or 10 ml for  $\Sigma$ PCB. Cockcroft & Ross (1990) estimated that a bottlenose dolphin calf (approximately equal to a humpback dolphin calf in size) ingests approximately 4.3 l of milk daily. Hence, the daily intake of organochlorines through suckled milk would be up to 159 times the recommended daily limit for humans for DDT and up to 430 times the recommended daily limit for humans for PCBs.

A high proportion of stranded cetaceans in Hong Kong are neonatal animals (Parsons, 1998b). This apparently high rate of observed neonatal mortality could be attributed to organochlorine poisoning (Parsons, 1998b; Parsons et al., 1999a). In addition, immune system abnormalities also have been observed in neonatal Hong Kong cetaceans (Parsons et al., 1999b), and it was suggested that organochlorines could be a contributing factor as well.

Ingesting contaminated food also could impact adult humpback dolphins. Chan et al. (1999) estimated the possible toxicological impacts on the human population of ingesting contaminated market fish in Hong Kong. The researchers estimated that, based on organochlorine concentrations in market fish, a 60-kg human would have a tolerable daily intake of the contaminated fish of 375 g per day. Extrapolating the Chan et al. (1999) data for a 180-kg humpback dolphin, and assuming that humpback dolphins ingest 9.6-11.8 kg of fish per day (Parsons, 1998c), humpback dolphins would exceed the tolerable daily intake of organochlorines for humans by between 8.5-10.5 times; however, it should be stressed that not all of the fish species analysed in Chan et al. (1999) were from Hong Kong waters, and species analysed were not necessarily species consumed by humpback dolphins. Even so, the figures above indicate that consumption of organochlorines by Hong Kong humpback dolphins through prey species could be substantial and warrants more detailed studies, looking specifically at contaminant levels in dolphin prey species in quantities proportional

**Table 2.** Organochlorine concentrations recorded in milk extracted from the stomach of a humpback dolphin calf ( $\mu\text{g}\cdot\text{g}^{-1}$  lipid wt); values are from Parsons & Chan (1998); n.d. indicates that contaminants were not detectable.

Organochlorine	Concentration
Pentachlorobenzene	n.d.
Hexachlorobenzene	0.12
$\Sigma$ Chlorobenzene	0.12
$\alpha$ -HCH	0.21
$\beta$ -HCH	0.74
$\gamma$ -HCH	0.21
$\Sigma$ HCH	1.16
Aldrin	n.d.
Dieldrin	0.69
$\Sigma$ Dieldrin	0.69
Oxychlorodane	n.d.
Hept Epox	n.d.
t-Chlordane	0.06
c-Chlordane	0.06
t-Nonachlor	0.06
c-Nonachlor	n.d.
$\Sigma$ Chlordane	0.19
Mirex	n.d.
$\Sigma$ PCB	2.49
p,p'-DDE	4.83
p,p'-DDD	5.93
p,p'-DDT	2.96
$\Sigma$ DDT	13.72

to the actual dietary intake of these dolphins. Such a study was proposed, and a potential methodology outlined by Clarke et al. (2000), but, as yet, results have not been published.

One means of measuring the toxicological impacts of organochlorines has been highlighted by several authors in recent years (e.g., Minh et al., 1999, 2000a, 2000b; Simmonds et al., 2000), that is, TCDD Toxic Equivalents (TEQs). The concept of TEQs was developed to provide a value of toxicity for mixtures of organochlorines, such as dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs), and dioxin-like PCBs, and where different congeners in a mixture may have different toxicity levels. The various chemicals are given a toxicity value relative to one particular dioxin (i.e., 2, 3, 7, 8, -tetrachlorodibenzo-p-dioxin [TCDD]), and the various values are summed to give a total TEQ rating. A series of TEQs were calculated by Minh et al. (1999) for Hong Kong cetaceans, based on dioxin-like PCBs; they are summarized in Table 3. The summed mean TEQ for dioxin-like PCBs from this study was 509.8 pg.g<sup>-1</sup> which, to put this in context, was nearly double the blubber TEQ recorded for Baltic seals (286 pg.g<sup>-1</sup>), which were exhibiting symptoms of organochlorine-induced, immune system suppression (Ross et al., 1995; Vos, 2000). Moreover, one feature of TCDD (and TCDD-like PCB) toxicity is atrophy of the thymus gland (Vos, 2000), and such symptoms have been noted in a neonatal finless porpoise from Hong Kong waters (Parsons et al., 1999b).

#### *The Impacts of Organochlorine Contamination on Other Humpback Dolphin Populations*

In addition to the Pearl River (and Hong Kong), Jefferson & Hung (2004) suggested eight *S. chinensis* populations occurred in Chinese estuaries, including the Min, Jiulong, and Yangtze River estuaries, all of which are situated in proximity to major population centers and, as such, high levels of pollution would be expected. In the Min River, ΣHCH concentrations of 4.2-9.4 ng.g<sup>-1</sup> (dry wt) and ΣDDT concentrations of 6.9-13.1 ng.g<sup>-1</sup> (dry wt) were recorded (Hu et al., 1996), which demonstrated a much higher level of contamination than the Yangtze River (ΣHCH 0.4-0.7 ng.g<sup>-1</sup> dry wt; ΣDDT 0.1-0.2 ng.g<sup>-1</sup> dry wt; ΣPCB 3.0-9.5 ng.g<sup>-1</sup> dry wt) (Wu et al., 1999), but similar levels of contamination, for PCBs and DDTs as in the Pearl River, as noted above (Liao, 1983). The Jiulong River was slightly less contaminated than the Min or Pearl Rivers (ΣHCH 0.29-0.69 ng.g<sup>-1</sup> dry wt; ΣDDT 4.1-6.1 ng.g<sup>-1</sup> dry wt; ΣPCB 0.45-1.15 ng.g<sup>-1</sup> dry wt) (Zhang et al., 1996), but more so than the Yangtze. Moreover, in the Jiulong River, like the Pearl River, a substantial

**Table 3.** Concentrations of non-ortho and mono-ortho coplanar PCBs and their 2, 3, 7, 8 - TCDD toxic equivalents (TEQs); concentrations of PCB congeners are expressed as ng/g wet weight, and TEQs are expressed as pg/g. Values are from Minh et al. (1999).

Non-ortho	Mean	Range
IUAPC-77	9.1	3.6-19
TEQ	4.6	0.36-1.90
IUAPC-126	2.1	1.1-2.9
TEQ	210	110-290
IUAPC-169	0.62	0.19-0.99
TEQ	6.2	1.9-9.9
Mono-ortho		
IUAPC-105	440	290-590
TEQ	44	29-59
IUAPC-118	1500	1,100-2,000
TEQ	150	110-120
IUAPC-156	190	90-290
TEQ	95	45-145

proportion of total concentration was as DDT, rather than DDD or DDE (Wu et al., 1999), demonstrating a relatively recent input of this contaminant. In comparison, the majority of DDT present in the Yangtze River samples was present as the derivative DDE. This suggests that the degree of organochlorine contamination reported for humpback dolphins in the Pearl River also could be similar for the humpback dolphins in the Min River, with the potential for contamination in the Jiulong River population as well.

Aerial contamination of Southeast Asia by organochlorines continues, due to the volatilisation of historical organochlorine residues and the recent applications of pesticides (Phillips & Tanabe, 1989). Water bodies in the tropics act as a sink for these air-borne pollutants, which ultimately contaminate the marine biota (Tanabe et al., 1994). It is expected that there will be a significant increase in organochlorine contamination of coastal cetaceans in Southeast Asia (such as the Indo-Pacific humpback dolphin) during the next decade as a result of their accumulation of both historical and recent DDT.

Organochlorine pesticides, including DDT, also are used widely in India (Shailaja & Singbal, 1994) since 1948 (Mehrotra, 1985a, 1985b) and considerable quantities of DDT, Aldrin, and HCHs have been recorded from coastal sediments in the Bay of Bengal (Sarkar & Sen Gupta, 1988). These contaminated sediments are known to be ingested by coastal fish, which in turn become contaminated (Aldrin up to 1.03 ng.g<sup>-1</sup>; ΣDDT up to 19.34 ng.g<sup>-1</sup>) (Rajendran et al., 1992; Shailaja

& Singbal, 1994). From an analysis of these contaminated fish, primary DDT was a major component of the total DDT concentration (Shailaja & Singbal, 1994), suggesting, as in Hong Kong, a recent input of the toxin into the biota.

Two papers documented organochlorine levels in humpback dolphins from the Bay of Bengal, and it was noted that the dolphins possessed relatively low levels of contamination in comparison to humpback dolphins in Southeast Asia (HCB 0.5-4.6  $\mu\text{g.g}^{-1}$  wet wt;  $\Sigma\text{HCH}$  0.22-1.1  $\mu\text{g.g}^{-1}$  wet wt;  $\Sigma\text{DDT}$  11-14  $\mu\text{g.g}^{-1}$  wet wt;  $\Sigma\text{PCB}$  0.92-1.8  $\mu\text{g.g}^{-1}$  wet wt [Tanabe et al., 1993]; and HCB 0.01  $\mu\text{g.g}^{-1}$  wet wt;  $\Sigma\text{HCH}$  0.14-1.5  $\mu\text{g.g}^{-1}$  wet wt;  $\Sigma\text{PCB}$  4.0-5.0  $\mu\text{g.g}^{-1}$  wet wt [Prudente et al., 1997]). Tanabe et al. (1996) reiterated this showing that HCH concentrations in these humpback dolphins were extremely low compared to animals sampled in other regions; however, Prudente et al. (1997) reported relatively high levels of DDT in the animals that they analysed ( $\Sigma\text{DDT}$  41-63  $\mu\text{g.g}^{-1}$  wet wt) and, moreover, these levels were among some of the highest recorded for cetaceans from the Indian Ocean and North Pacific.

In the three humpback dolphins analysed by Tanabe et al. (1993) from the Bay of Bengal, the percentage of total DDT present as underived DDT was 5.8%, 22%, and 34% as compared to 25% in Hong Kong animals. Since two of the animals showed relatively high ratios of this un-metabolised contaminant, it could indicate recent DDT inputs into the environment; however, Prudente et al. (1997) reported that DDE contributed nearly 95% of the total DDT concentrations for the three animals examined in their study.

The sample size of humpback dolphins examined in both the Tanabe et al. (1993) and Prudente et al. (1997) papers were small ( $n=3$  in both), however, and the samples were collected more than a decade ago (between 1988 and 1992). To get a clearer idea of current organochlorine levels in humpback dolphins, more research needs to be conducted on contaminant levels in this area.

Humpback dolphins also occur on the west coast of India, including in the Mandovi River, Goa (Parsons, 1998d). Elevated concentrations of organochlorines have been recorded in sediments from this river system ( $\Sigma\text{HCH}$  3.8  $\text{ng.g}^{-1}$  dry wt;  $\Sigma\text{DDT}$  73  $\text{ng.g}^{-1}$  dry wt;  $\Sigma\text{PCB}$  170  $\text{ng.g}^{-1}$  dry wt) (Iwata et al., 1994) and in nearby coastal sediments ( $\Sigma\text{DDT}$  10.8-179.1  $\text{ng.g}^{-1}$  dry wt) (Sarkar & Sen Gupta, 1987). These sediment contaminant concentrations were considerably higher than those recorded from the Pearl River (Liao, 1983). Fish from the midwestern coast of India were not highly contaminated ( $\Sigma\text{DDT}$  8.1-54.3  $\text{ng.g}^{-1}$  wet wt), however, and did not reveal any primary DDT in their tissues when analysed (Shailaja &

Sen Gupta, 1989). More pollution-oriented studies are required around the coasts of the subcontinent to determine the exact threat organochlorine compounds pose to coastal cetaceans.

Organochlorine contaminant levels in humpback dolphins are unknown for the rest of their range, although Cockcroft (1999) mentioned that South African humpback dolphins from Kwazulu-Natal display the highest organochlorine concentrations in any marine mammal from South African waters, by virtue of their close proximity to agricultural and industrial areas. This was quantified by Cockcroft (2002), who noted that organochlorine concentrations of up to 131  $\mu\text{g.g}^{-1}$  wet weight and 50  $\mu\text{g.g}^{-1}$  wet weight have been recorded for  $\Sigma\text{DDT}$  and PCB congener 1260, respectively, in South African humpback dolphins, but more detailed information on humpback dolphin contamination from this areas is, as yet, unpublished.

Although DDT use was discontinued in South Africa in 1976, Van Dyk et al. (1982) reported that it was still used in state-controlled anti-malaria actions to quell mosquitoes, so there could be more recent inputs of this pesticide into the South African marine environment. Generally, organochlorine contaminant levels are relatively low in South African cetaceans (e.g.,  $\Sigma\text{DDT}$  <26.25  $\mu\text{g.g}^{-1}$  wet wt) (Cockcroft, 1999), although (non-*S. chinensis*) cetacean contaminant levels in Kwazulu-Natal, where there is a well-studied coastal population of humpback dolphins (Saayman & Tayler, 1979), were slightly higher ( $\Sigma\text{PCB}$  up to 48.3  $\mu\text{g.g}^{-1}$  wet wt and  $\Sigma\text{DDT}$  up to 65.2  $\mu\text{g.g}^{-1}$  wet wt). Nonetheless, levels of organochlorines in South African cetaceans were sufficient to cause disruption to immune and reproductive systems and warrant concern from researchers in that country (Cockcroft, 1999).

In summary, although for many humpback dolphin populations in much of the species range, information on organochlorine contamination is scarce at best, studies in Hong Kong demonstrated that organochlorine pollution is sufficient to cause considerable concern for at least one population of humpback dolphins. At the very least, other populations of humpback dolphins in Chinese waters probably are facing similar problems in terms of organochlorine contamination. Work should be conducted to identify which other humpback dolphin populations may also be at risk from this class of anthropogenic pollutants. Moreover, to quantify the impacts of pollution on humpback dolphin populations, detailed investigations should be instigated to assess the impacts of organochlorine contamination on humpback dolphin bodily systems and health. Such studies should involve histopathology



studies on the tissues of the reproductive and immune systems of freshly deceased animals as well as haematological studies, cross-referenced with data on the contaminant burden of the stranded animals. Because analysing stranded animals for contaminants can give a biased representation of the contaminant burden of the population at large, studies such as biopsy sampling may be required to look at potential health biomarkers and give a more accurate representation of contaminant burdens in living population members. It should be emphasised, however, that invasive techniques should be used with caution and should not be conducted on animals from populations that are already under stress.

### Trace Elements

Trace elements are byproducts of many industrial processes. They enter the marine environment through atmospheric and land-based effluent sources. Once in the marine environment, they are taken into the marine biota, particularly in demersal fish, which may inhabit and ingest contaminated sediments. In vertebrates, trace elements typically concentrate in protein-rich tissues, such as in the liver and muscle, although cadmium is notable for concentrating in kidney tissues.

The toxicological properties of trace elements were brought to the world's attention with the outbreak of Minamata disease (mercury poisoning) in Japan during the 1960s. Mercury toxicity, for example, causes neurological damage, immunosuppression, and can cause fetal abnormalities (Clarkson, 1987; Von Burgh & Greenwood, 1991). Trace element contamination has not been restricted to the human population, however; levels of trace element contamination have been reported for a variety of cetacean species (Law, 1996). Some species possess extremely high concentrations of toxic elements; for example, small cetaceans in Japan had liver mercury concentrations of up to 485  $\mu\text{g.g}^{-1}$  wet weight or approximately 1,600  $\mu\text{g.g}^{-1}$  dry weight (Honda et al., 1983). The Mediterranean is inhabited by some of the most highly trace element-contaminated cetaceans, with levels of mercury up to 13,156  $\mu\text{g.g}^{-1}$  dry weight (Leonzio et al., 1992) recorded in a bottlenose dolphin (*Tursiops truncatus*) from the Tyrrhenian Sea. In recent years, there have been several mass mortalities of cetaceans in the Mediterranean, and it was suggested that anthropogenic contaminants may be the underlying cause (Simmonds, 1992).

High trace element burdens in cetaceans have been associated with a variety of biological and pathological responses. These include lesions

and fatty degeneration in bottlenose dolphins (Rawson et al., 1993), decreasing nutritional state (Siebert et al., 1995), and increased severity of parasitic lesions (Siebert et al., 1999). Of particular concern is the build up of mercury, which marine mammals tend to accumulate in the liver to higher levels than other marine organisms (Law et al., 1991). Mercury can build up over time in cetaceans from the consumption of prey species, which are a major source of mercury, especially methylmercury (Siebert et al., 1999). Moreover, this methylated form of mercury can pass across the placental barrier, causing high mercury burdens in young individuals (Andre et al., 1990).

To date, studies into trace element contamination of humpback dolphins have been limited to China, with an emphasis on Hong Kong. The sediments in the coastal waters of Hong Kong display high levels of trace element contamination (Wong, 1996), with extremely elevated concentrations of toxic elements in some areas, such as typhoon shelters and Victoria Harbor (Cr: > 80  $\mu\text{g.g}^{-1}$  dry wt; Pb: > 50  $\mu\text{g.g}^{-1}$  dry wt) (Morton & Blackmore, 2001), and some areas in Hong Kong are becoming increasingly contaminated (Blackmore, 1998). Studies indicated that trace element concentrations in marine species also may be high; for example, Chan (1995) reported elevated levels of trace metals in fish caught in Victoria Harbor, including high concentrations of cadmium (3.3  $\mu\text{g.g}^{-1}$  dry wt), copper (5.7  $\mu\text{g.g}^{-1}$  dry wt), lead (19.1  $\mu\text{g.g}^{-1}$  dry wt), and zinc (66.6  $\mu\text{g.g}^{-1}$  dry wt) in rabbitfish (*Siganus oramin*) collected from this area.

Moreover, Dickman & Leung (1998) analysed mercury concentrations in various species of store-bought fish caught in Hong Kong waters (mean concentration=0.12  $\mu\text{g Hg.g}^{-1}$  wet wt), as well as commercially exploited species of fish (*Argyrosomonus argentus*, *Cynoglossus melapterus*, *Arnoglossus tenuis*, *Saurida elongata*, *S. tumbil*, and *Platycephalus indicus*) collected from the eastern and western waters of Hong Kong. The fish collected from the eastern waters had a mean mercury concentration of 0.156  $\mu\text{g Hg.g}^{-1}$  wet weight, which was lower than the mean mercury concentration recorded for western water fish (0.215  $\mu\text{g Hg.g}^{-1}$  wet wt), which were collected from areas frequented by humpback dolphins.

Within the western waters of Hong Kong, one of the areas most used by Hong Kong humpback dolphins coincides with a dumping site for contaminated sediments, in which trace element concentrations are extremely high (Jefferson, 2000) (Table 4). Moreover, in this part of Hong Kong, the bioavailability of trace elements, in particular toxic cadmium, is increased as the result of lower

salinities caused by the freshwater influx of the Pearl River (Morton & Blackmore, 2001).

Parsons (1997a, 1999a) analysed levels of trace element contamination in whole fish caught from this area of high humpback dolphin abundance, which is adjacent to the aforementioned contaminated mud pits, and discovered that concentrations of trace metals were relatively high. There were considerable amounts of selenium (up to 109.8  $\mu\text{g.g}^{-1}$  dry wt), cadmium, copper, mercury, and zinc (up to 56.9, 53.4, 380, and 91.6  $\mu\text{g.g}^{-1}$  dry wt, respectively) in whole, homogenized fish. Concentrations of arsenic and lead were consistently very high in the specimens sampled (up 417 and 511  $\mu\text{g.g}^{-1}$  dry wt, respectively). Bottom-dwelling fish were, in general, more heavily contaminated by trace metals than were more pelagic species, which was unsurprising because these species are obviously in closer contact with contaminated sediments.

Concerns over the toxicological impact of ingesting trace element contaminated fish have been voiced for the human population of Hong Kong. Dickman et al. (1999) demonstrated that members of the human population, consuming four or more meals of fish per week, displayed elevated concentrations of mercury in their hair (4.07  $\mu\text{g Hg.g}^{-1}$  dry wt) and, moreover, these elevated human concentrations of mercury were linked to decreased fertility in males.

Therefore, Parsons (1998c) estimated the toxicological impact of the consumption of these contaminated fish by dolphins and estimated daily intakes of trace elements (Table 5). A high daily intake by humpback dolphins of the toxic trace metals cadmium, mercury, and lead was suggested. To put the values in Table 5 into context, the World Health Organization's tolerable daily

intakes in humans for four of the most toxic of the trace elements were 2.0  $\mu\text{g.kg}^{-1}$  body weight.  $\text{day}^{-1}$  for inorganic arsenic, 1.0  $\mu\text{g.kg}^{-1}$  body weight.  $\text{day}^{-1}$  for cadmium, 0.71  $\mu\text{g.kg}^{-1}$  body weight.  $\text{day}^{-1}$  for mercury, and 3.57  $\mu\text{g.kg}^{-1}$  body weight.  $\text{day}^{-1}$  for lead. The daily intakes of trace elements were clearly higher than these tolerable intakes, more than 90 times higher in the case of lead. Clarke et al. (2000) discussed taking this methodology further and suggested ways to develop risk assessment models for humpback dolphins in Hong Kong based on the ingestion of trace element (and organic pollutant) contaminated prey items. Results from this proposed study have not yet been published.

Actual trace element contamination levels in Hong Kong humpback dolphin tissues were summarized in Parsons (1999b), and these values are presented in Table 6. In addition, Kubota et al. (2001) and Takahashi et al. (2000) presented information on arsenic and tin concentrations, respectively, in dolphin liver tissue from this population, that were of a similar scale as those presented in Table 6 (0.2-5.34  $\mu\text{g As.g}^{-1}$  dry wt and 7.2-26  $\mu\text{g Sn.g}^{-1}$  dry wt). Surprisingly, despite theoretically high dietary intakes of the trace elements arsenic, chromium, lead, molybdenum, and nickel, levels of these trace elements in actual humpback dolphin tissues were an order of magnitude lower than in the prey species, suggesting that dolphins can eliminate a large proportion of these trace elements from their bodies (Parsons, 1998c). Concentrations of zinc and, in particular, mercury were observed to biomagnify, however; that is to say, concentrations in humpback dolphin tissues (Parsons, 1999b) were considerably higher than in consumable food items (Parsons, 1998c, 1999a).

**Table 4.** A comparison of trace element concentrations ( $\mu\text{g.g}^{-1}$  dry wt) in marine sediments in Hong Kong and other polluted estuaries

Location	East Sha Chau, North Lantau, Hong Kong			Bristol Channel England	Mersey Estuary England
Activities	Contaminated Mud Dump Site			Industry and sewage	Industry and sewage
Reference	CES & Binnies (1994)			Bryan et al. (1985)	Langston (1986)
Element	Minimum	Maximum	Mean		
Cd	37.8	505.4	77.5	1.1	3.9
Cu	4.71	108.12	33.65	54	144
Hg	0.04	0.63	0.14	0.48	6.2
Ni	20.96	197.6	35.34	33	44
Pb	31.83	61.86	48.19	88	205
Zn	51.42	204.58	128.56	255	255

**Table 5.** Estimated trace element intake from ingesting contaminated prey items in humpback dolphins from Hong Kong (Parsons, 1998c)

Trace element	Estimated daily intake ( $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{body weight}\cdot\text{day}^{-1}$ )
Arsenic	410-518
Cadmium	34-44
Chromium	40-50
Cobalt	4.5
Copper	44-56
Lead	320-403
Mercury	51-65
Molybdenum	22-27
Nickel	19-24
Selenium	99-125
Zinc	388-490

The mercury concentrations recorded in Hong Kong cetaceans were very high (mean:  $142 \mu\text{g}\cdot\text{g}^{-1}$  dry wt  $\pm 91.9$  SD; max:  $906 \mu\text{g}\cdot\text{g}^{-1}$  dry wt/ $272 \mu\text{g}\cdot\text{g}^{-1}$  wet wt) (Parsons, 1998c, 1999b). Wagemann & Muir (1984) suggested that levels of 100-400  $\mu\text{g}\cdot\text{g}^{-1}$  wet weight of mercury in liver may present a threat to marine mammals. Siebert et al. (1999) recorded significant associations between mercury levels and severity of lesions for animals with concentrations with a maximum value of half that recorded in Hong Kong ( $<450 \mu\text{g}\cdot\text{g}^{-1}$  dry wt); however, more chronic effects, such as liver disease, occur with mercury concentrations as low as  $50\text{-}61 \mu\text{g}\cdot\text{g}^{-1}$  wet weight (Rawson et al., 1993) and other, sublethal effects, such as infertility (e.g., Dickman et al., 1999), also could be an issue. Mercury poisoning is, therefore, considered a potential threat to Hong Kong's humpback dolphin population.

Huang et al. (1999) examined trace elements in a range of tissues from three adult and five juvenile humpback dolphins from the Xiamen population and recorded levels which were broadly

similar to those recorded in Hong Kong, although maximum zinc levels were higher and cadmium levels were lower in the Xiamen samples (Table 7). Mercury concentrations were high, however. Maximum mercury concentrations in liver tissues of Xiamen humpback dolphins were identical to maximum mercury levels recorded in Hong Kong (up to  $272 \mu\text{g}\cdot\text{g}^{-1}$  wet wt). Toxic effects resulting from mercury contamination, therefore, are also a potential threat to this second Chinese humpback dolphin population.

There is insufficient contamination information for humpback dolphins and their environment outside of Hong Kong and Xiamen. Le et al. (1999) reported concentrations of tin ranging from  $0.57$  to  $1.9 \mu\text{g}\cdot\text{g}^{-1}$  dry weight in liver tissue of four humpback dolphins from the Bay of Bengal, 47% of which was present in an organic butyltin form (see below); unfortunately, other trace element data is lacking. As is the case of organochlorine contamination, estuarine locations in proximity to high levels of human and industrial activity likely have similar levels of trace element contamination.

### Butyltins

Butyltins (BTs) are a group of chemicals recently highlighted as a possible threat to cetaceans (Tanabe, 1999). Several studies have been undertaken on the absorption of BT by coastal cetaceans in Japan, where high levels have been recorded (Iwata et al., 1995; Kim et al., 1996). BTs are extremely toxic and can cause growth retardation and imposex in marine organisms in concentrations as low as  $10\text{-}20 \text{ ng}\cdot\text{L}^{-1}$  (Gibbs & Bryan, 1986; Lawler & Aldrich, 1987). They can cause fish mortality in concentrations as low as  $3\text{-}5 \text{ mg}\cdot\text{L}^{-1}$  (Ward et al., 1981) and disrupt the mammalian immune system (Seinen & Willems, 1976; Vos et al., 1984). The main sources of BTs are anti-fouling paints (primarily containing tributyltin [TBT]) applied to the hulls of marine

**Table 6.** Range of trace element concentrations reported from humpback dolphins in Hong Kong waters ( $\mu\text{g}\cdot\text{g}^{-1}$  dry wt) (Parsons, 1999b)

Tissue	Arsenic	Cadmium	Cobalt	Chromium	Copper	Mercury	Molybdenum	Nickel	Lead	Selenium	Tin	Zinc
Liver	< 0.36	< 0.36	< 0.36	< 0.36	< 0.93	< 0.36	< 0.65	< 0.65	< 0.36	< 0.78	< 0.65	24.13
	12.94	23.17	< 0.90	0.65	30.62	906	1.62	0.89	8.59	131	8.90	243
Kidney	< 0.90	< 0.70	< 0.70	< 0.70	7.03	< 0.70	< 0.70	< 0.70	< 0.87	< 0.90	< 0.90	53.4
	12.12	84.1	< 0.90	< 0.90	23.61	35.77	0.76	0.91	13.13	21.27	8.68	234
Blubber	< 0.35	< 0.30	< 0.35	< 0.35	< 0.35	< 0.30	< 0.35	< 0.30	< 0.46	< 0.46	< 0.30	2.00
	18.32	< 1.00	1.11	2.68	2.68	< 1.00	1.11	1.16	12.05	15.64	3.47	18.24

**Table 7.** Range of trace element concentrations reported from humpback dolphins in Xiamen waters ( $\mu\text{g.g}^{-1}$  wet wt) (Huang et al., 1999)

Tissue	n	Cadmium	Copper	Mercury	Lead	Zinc
Liver	3	0.10	9.74	0.231	1.07	84.4
		0.30	30.9	272	2.60	275
Muscle	8	0.013	1.72	0.068	0.55	50.7
		0.12	19.6	8.08	1.69	198
Skin	2	0.006	0.87	0.117	0.15	7.01
		0.029	0.73	11.5	0.75	40.5
Intestine	2	0.036	6.36	0.242	2.01	110
		0.042	30.6	0.552	2.30	204
Kidney	1	0.02	19.6	0.558	2.69	92.7
Heart	1	0.04	15.2	0.558	1.09	111
Uterus	1	0.045	19.7	0.482	1.75	113
Faeces	1	0.03	25.0	0.533	1.64	166

vessels and structures. Monobutyltin (MBT) and dibutyltin (DBT) also are used by industry to stabilize chlorinated polymers and in the production of silicones and polyurethane foams (Tanabe et al., 1998).

It is extremely likely that many areas populated by humpback dolphins are highly contaminated with BTs. For example, humpback dolphins inhabit the waters of several coastal ports in Asia that host a large volume of shipping and, therefore, potential BT pollution (e.g., Shanghai, Bombay, Singapore, and Hong Kong). Hong Kong is known to be heavily contaminated by BTs, with high levels of TBT in coastal waters (up to  $1,000 \text{ ng.L}^{-1}$ ) (Lau, 1991) and concentrations up to  $53,000 \text{ ngSn.g}^{-1}$  noted in marine sediments (Ko et al., 1995). Hong Kong's humpback dolphins inhabit an area with not only a high volume of shipping traffic (and, hence, leaching of TBT), but also several dry-dock facilities where ships with TBT coated hulls are frequently berthed and sometimes repainted (Parsons, 1997a). In addition, there are undoubtedly numerous factories and facilities both in Hong Kong and neighbouring areas of China, that could be utilising MBT and DBT in the production and processing of commercial products. As a result, concentrations of BTs ranging from 13 to  $31 \mu\text{g.g}^{-1}$  dry weight have been found in the liver tissue of Hong Kong dolphins (Takahashi et al., 2000); these extremely elevated levels are among the highest BT concentrations reported from cetaceans worldwide.

BTs also have been reported in the liver tissue of four humpback dolphins from the Bay of Bengal, although these concentrations ( $0.067\text{--}0.200 \mu\text{g.g}^{-1}$  wet wt, [Tanabe et al., 1998];  $0.27\text{--}0.90 \mu\text{g.g}^{-1}$  dry wt [Le et al., 1999]) were almost an order of magnitude lower than those reported

for Hong Kong humpback dolphins. Tanabe et al. (1998) suggested that the majority of the BT contamination in these Indian animals could have a shipping-related source, as the proportion of TBT in the samples analysed was relatively high.

Next to nothing is known at this time about the effects of BT contamination in these cetaceans. Bearing in mind the extremely elevated concentrations in Hong Kong animals, further analysis of BT contamination in the tissues of these humpback dolphins, as well as in animals from populations inhabiting other areas of high shipping traffic, should be a priority.

### Potential Threats from Other Pollutants

#### *Polynuclear Aromatic Hydrocarbons*

Polynuclear Aromatic Hydrocarbons (PAHs) are produced primarily during combustion, both natural (e.g., bush fires) and anthropogenic. They also are associated with sites where petroleum-based discharges have occurred. They can combine (especially Benzo[a]pyrene and its derivatives) with DNA to produce an extremely carcinogenic compound (Hansen & Shane, 1994), and the high rate of cancer seen in beluga whales (*Delphinapterus leucas*) in the St. Lawrence Estuary has been attributed to these chemicals (Beland & Martineau, 1988; Martineau et al., 1994).

Major anthropogenic sources of PAHs in coastal areas include vehicle exhausts, frying oil in both restaurants and homes, car exhausts, oil spills from shipping and refineries, and fossil fuel-fired power stations. PAHs, and their nitrogenic derivatives, commonly are recorded downwind of power stations (Sloss & Smith, 1993).

Connell et al. (1998) reported PAH concentrations in Hong Kong marine sediments ranging from 40-60 ng.g<sup>-1</sup> wet weight, although levels in some particularly contaminated areas were greater (e.g., 116 ng.g<sup>-1</sup> wet wt in Victoria Harbor and 1,159 ng.g<sup>-1</sup> wet wt in typhoon shelters). Mai et al. (2002) recorded even higher levels of 323 to 2,372 ng.g<sup>-1</sup> dry weight in sediments in the Pearl River Estuary. Moreover, sediment concentrations of 1,168 to 21,329 ng.g<sup>-1</sup> dry weight were noted in rivers leading into the Pearl River Estuary, and a substantial 14,812 ng.g<sup>-1</sup> dry weight was recorded from Macau Harbor (Mai et al., 2002). Mai et al. (2003) considered PAH contamination in the Pearl River region to be moderate on a global scale and to possess equivalent levels of contamination to several polluted industrialized areas such as the UK's Tamar Estuary and Australia's Brisbane River (Mai et al., 2002).

Given this sediment contamination, and the fact that there is a coal-fired power station only a few hundred metres from one of the areas of highest humpback dolphin abundance in Hong Kong, and that atmospheric sources of PAHs are one of the main contributors to PAH contamination in the area (Mai et al. 2003), there is potential for PAH contamination in these animals. Thus, PAH contamination is a potential, but unquantified, threat to the health of humpback dolphins, and an investigation of PAH concentrations both in the animals and their prey species is needed.

*Tris(4-chlorophenyl)methane (TCPMe) and Tris(4-chlorophenyl)methanol (TCPMeOH)*

Two organic pollutants that recently have been documented in cetaceans and pose a potential health risk are tris(4-chlorophenyl)methane (TCPMe) and tris(4-chlorophenyl)methanol (TCPMeOH). These organic compounds occur as the result of impurities in agrochemical mixtures and the production of synthetic dyes (Jarman et al., 1992), as well as being a byproduct in the production of DDT (Buser, 1995). In many respects, these chemicals can have similar effects on mammals as PCBs and DDT in that they may disrupt hormone systems (Aguilar et al., 1997), in particular adversely effecting reproductive systems by having an anti-androgen effect (Körner et al., 1997), as well as causing changes in the spleen and liver and altering white blood cell and lymphocyte counts (Poon et al., 1997). Both compounds bioaccumulate and biomagnify, potentially to a greater extent even than DDT and, in addition, could be more persistent in the marine environment (Falandysz et al., 1999). A variety of marine mammal species have been documented carrying TCPMe and TCPMeOH as contaminants (e.g., de Boer et al., 1996; Jarman et al., 1992;

Walker et al., 1989; Watanabe et al. 1999) including humpback dolphins from Hong Kong (Minh et al., 1999).

Concentrations of TCPMe and TCPMeOH in Hong Kong's humpback dolphins (4.1-290 ng TCPMe.g<sup>-1</sup> wet wt; 12-270 ng TCPMeOH.g<sup>-1</sup> wet wt) (Minh et al., 1999, 2000b, 2000c) were higher than concentrations reported from cetaceans in the Baltic Sea (Falandysz et al., 1999), Philippine and Indian coasts, and most coastal cetaceans from Japan (Watanabe et al., 1999), and although TCPMe levels were lower, TCPMeOH levels were higher than those reported from beluga whales from the St Lawrence Estuary, Canada (Muir et al., 1996). The toxicological effects of TCPMe and TCPMeOH on cetaceans are as yet unknown. Nonetheless, bearing in mind the toxicological effects noted for other mammalian species, and the concentrations described in Hong Kong, contamination levels of these compounds should be investigated in other humpback dolphin populations.

*Sewage Pollution*

Cetaceans are vulnerable to a number of diseases, parasites, and pathogens that can be transmitted via either human or livestock waste. For example, Bossart et al. (1990) obtained an immune response to a hepatitis B-like agent in a Pacific white-sided dolphin (*Lagenorhynchus obliquidens*), suggesting the potential for human to dolphin transmission of the virus. Considering that hepatitis A and B are prevalent in many areas of the Indo-Pacific, it is possible that these viruses could pass to coastal cetaceans, such as humpback dolphins, via sewage effluent.

Bacteria associated with sewage, including *Streptococcus faecalis*, *Escherichia coli*, and other fecal coliforms, have been cultured from several cetaceans stranded in Hong Kong (Parsons & Jefferson, 2000). This is not surprising because fecal coliforms were recorded in North Lantau waters, the area of greatest humpback dolphin abundance in Hong Kong (Jefferson, 2000), in concentrations of over 20,000 100mL<sup>-1</sup> (Parsons, 1997b).

To date, there has only been one assessment on the potential impacts of sewage pollution on humpback dolphins. This study took place in Hong Kong, a region which discharges over 2,000 million liters of sewage into its coastal waters every day. Bacteria can egress into the mammalian body by a variety of routes, and Parsons (1997b) estimated that a Hong Kong humpback dolphin's minimum daily intake of sewage bacteria through ingesting contaminated seawater alone could be up to 70,500 fecal coliforms.day<sup>-1</sup>. To put this in context, a one-off ingestion of 200-

300 coliforms is considered to be unacceptable for humans. In addition to this source of bacterial uptake, dolphins also could uptake bacteria from contaminated prey items, through dermal wounds, and exposed mucus membranes.

The majority of humpback dolphin populations exist in the coastal or estuarine waters of developing nations, countries that have little provision for sewage treatment. For some populations, the adjacent human population is substantial. Therefore, more studies should be conducted into the potential impacts of this type of pollution on coastal cetacean populations such as humpback dolphins.

The level of sewage pollution in the humpback dolphin habitat is particularly pertinent when one considers that for many populations the accumulation of organochlorines and other anthropogenic chemicals in dolphin tissues may disrupt immune systems (Parsons & Chan, 1998), making them more vulnerable to bacterial (Smith et al., 1978; Thomas & Hinsdill, 1978) and viral diseases (Friend & Trainer, 1974; Koller & Thigpen, 1973). The impacts of sewage pollution would, thus, be exacerbated if animals also are exposed to immuno-suppressive pollutants such as organochlorines, BTs, and some trace elements. These different types of pollutants could, with the action of sewage pathogens, act synergistically to debilitate cetacean populations, and this should be investigated.

### Conclusions

Although detailed studies on pollution and its effects on humpback dolphin populations largely are restricted to Hong Kong, this population can be used as a model for the probable situation for humpback dolphins inhabiting several other areas. For example, *Sousa* populations inhabiting the waters of Singapore, Bombay, Xiamen, and Shanghai are likely exposed to similar levels of contamination. The fact that such elevated concentrations of toxic DDT, mercury, and BT are found in the Hong Kong population should be an indicator that pollution is unequivocally a serious problem and threat to the health and status of humpback dolphins in this area, and by virtue of similar habitats, *Sousa* populations in many other regions of the world.

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