THE RAFFLES BULLETIN OF ZOOLOGY 2002 Supplement No. **10**: 161-171

MORTALITY AND MORBIDITY OF HONG KONG FINLESS PORPOISES, WITH EMPHASIS ON THE ROLE OF ENVIRONMENTAL CONTAMINANTS

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ABSTRACT. – Data on causes of death for finless porpoise (*Neophocaena phocaenoides*) specimens stranded in Hong Kong between 1995 and 2000 were evaluated. We diagnosed several causes of death, including fishing net entanglement (44%), vessel collision (17%), reproductive abnormalities (17%), parasitic infections (17%), and shark attack (5%). The incidence of net entanglements may have increased in recent years. We also examined the levels of five types of environmental contaminants of particular concern (DDTs, PCBs, HCHs, BTs, and Hg). Organochlorine concentrations generally increased with age in males, but decreased with age in females, the latter likely due to the reproductive transfer of these lipophilic compounds to the offspring during gestation and lactation. Mercury levels increased exponentially with age in both sexes. Organochlorine pesticide (DDT), and to a lesser extent, polychlorinated biphenyl (PCB) concentrations were extremely high in some individuals. There was some evidence for higher (but not statistically significant) DDT and HCH concentrations in specimens classified as in 'poor' health. This suggests that these organochlorines may indeed be affecting the health of Hong Kong finless porpoises. Mercury levels were also very high, and were in the range of concentrations previously found to be associated with liver disease in bottlenose dolphins. However, the levels of mercury were similar in specimens classified both as in 'good' and 'poor' health. Although much further work needs to be done to clarify the factors related to mortality and morbidity of finless porpoises in Hong Kong, this preliminary study suggests that fishing net entanglement may be the primary human-related cause of death. Further, there is now sufficient evidence to point to the damaging effects of some environmental contaminants on these animals. We suggest that efforts to mitigate these effects be implemented immediately to ensure the conservation of the porpoise population.

KEY WORDS. – Finless porpoise, *Neophocaena phocaenoides*, Hong Kong, mortality, morbidity, disease, pathology, cause of death, necropsy, pollutants, contaminants.

INTRODUCTION

There has been much published on the concentrations of various pollutants[1] in the tissues of different species of cetaceans, including porpoises (see bibliography in Aguilar & Borrell, 1996; reviews in O'Shea & Geraci, 1999; O'Shea et al., 1999; Reijnders et al., 1999). Less work has been done to examine variability in contaminant levels with age and sex, or to detect temporal trends in contaminant levels (see chapters in Reijnders et al., 1999). Little research has been done to examine the effects of pollutants on the health of cetaceans, although this is clearly the most important aspect of ecotoxicology for cetacean conservation and management (but see Kuiken et al., 1993, 1994; Jepson et al., 1999; O'Shea et al., 1999; Siebert et al., 1999; Bennett et al., 2001). This is because the effects of contaminants are difficult to assess and quantify in cetaceans, largely due to the high cost of keeping captive cetaceans and ethical and legal impediments to manipulative experimentation with them (Reijnders, 1996).

Environmental contaminants probably do not often kill animals outright, but instead work interactively with other factors to degrade the health of individuals and to reduce

1 Although technically there are some distinctions, in this paper we use the terms pollutant and contaminant interchangeably.

Table 1. Summary of the finless porpoise specimens used in this study.

		Sample Size (n)				
Sex	Total n	ΣDDTs	ΣPCBs	ΣHCHs	ΣBTs	Hg
Males	23	23	20	8	10	18
Females	21	18	15	8	4	19
Unknown	7	5	4	2	3	3
Total	**51**	**46**	**39**	**18**	**17**	**40**

their resistance to disease and parasitic infestations (Bowles, 1999; Busbee et al., 1999). Therefore, although the direct cause of death may be rightly attributed to disease or parasitic infection, pollutants could well have played an indirect role in the animal's demise (e.g., through immune system disruption by organochlorines).

Finless porpoises (*Neophocaena phocaenoides*) in Hong Kong have been studied since 1993 (Parsons, 1997, 1998a; Parsons & Jefferson, 2000). Our research team has been studying them since September 1995, and our major goals have been to assess their population status and factors that threaten their survival, so that effective conservation measures can be implemented (Jefferson & Braulik, 1999). Although investigating the role of environmental contaminants has been a priority since 1998, the badly decomposed condition of the majority of specimens has hampered our ability to investigate contaminant concentrations and effects. Nevertheless, some progress has been made, and here we report information on causes of mortality, as well as contaminant levels and the factors that influence them. Also, through a crude classification of the health status of specimens, we have attempted to make some preliminary observations of associations between contaminant levels and the animals' health.

MATERIALS AND METHODS

Sample and Data Collection. – Stranded finless porpoise carcasses were examined in Hong Kong between 1993 and 2000 (see Parsons & Jefferson, 2000). Over the course of the present study (September 1995 to July 2000), we confirmed a total of 54 finless porpoise mortalities. Necropsies were performed either in the laboratory (for fresh specimens) or in the field (for those that were badly decomposed or in relatively inaccessible locations). Basic biological data and samples were collected (see Parsons & Jefferson, 2000 for a detailed discussion of the stranding program and sampling procedures). Specimens were classified as to their level of decomposition, using the codes outlined in Geraci & Lounsbury (1993).

A total of 51 specimens were necropsied, and most were also sampled for environmental contaminants (Table 1). Blubber samples, for organic contaminant analyses, were collected from the dorsal thoracic region, and were wrapped in aluminum foil and frozen. Samples of liver and kidney were taken for heavy metal and trace element analyses; samples were placed in plastic ziplock bags and stored in a freezer. We also collected 2-4 teeth from the middle of the lower left jaw of most specimens for age determination. Gross pathology was noted during necropsies, and some samples were examined histopathologically (however, this was done opportunistically). Blubber thickness was often measured, but this was also done on an opportunistic basis.

Analyses. – We examined five classes of contaminants in two types of tissue. These were total DDT pesticide residues (ΣDDTs = DDE + DDT), total polychlorinated biphenyls (ΣPCBs = monochlorobiphenyl + dichlorobiphenyl + trichlorobiphenyl + tetra-chlorobiphenyl + pentachlorobiphenyl + hexachlorobiphenyl + hepta-chlorobiphenyl + octachlorobiphenyl + nonachlorobiphenyl + decachlorobiphenyl), total hexa-chlorocyclohexanes (ΣHCHs = alpha-HCH + beta-HCH + gamma-HCH), and total butyltins (ΣBTs) in the blubber[2]; and concentrations of the heavy metal mercury (Hg) in the liver. This selection was based on indications from earlier studies that these contaminants were the most critical, due to high levels in Hong Kong cetaceans and in some cases high known toxicity (Parsons & Chan, 1998; Parsons, 1999; Minh et al., 1999; Tanabe, 1999). Contaminants in kidney tissues were not analyzed for this study, but the samples were archived for future analysis.

Frozen tissue samples were sent to a commercial ecotoxicology laboratory in Hong Kong (ALS Technichem [HK] Pty, Ltd.) for chemical analyses. For the determination of mercury levels, samples were dissected with titanium tools. They were then digested by a close vessel microwave digestion unit by nitric acid and hydrogen peroxide mixture prior to Inductively-Coupled Plasma Mass Spectroscopy (ICPMS) and Flow Injection Mercury Analyzer (FIMS) testing. For trace organic analysis, samples were extracted with a dichloromethane/acetone mixture and pre-cleaned by passing through a GPC column prior to analysis by GC systems.

2 When the analyses for this study were done, we assumed that blubber was the tissue in which butyltins concentrate at the highest levels, but we now know that liver is the organ with the highest concentrations (Takahashi et al., 2000).

Routine quality-control (QC) checks were run with each batch of 20 samples processed. For a QC check to have been judged acceptable, 80% of target analytes must have passed all three of the following criteria: (1) average recovery of Single Control Sample (SCS) and Duplicate Control Sample (DCS) must have fallen within the recovery control limits, (2) Relative Percent Difference for the SCS and DCS must have been $\leq$ 20%, and (3) blank concentrations must have been less than the limit of reporting.

Age was estimated by decalcifying and sectioning 1-2 teeth from each specimen on a sledge-type microtome, followed by staining, and counting of growth layer groups (GLGs) in the postnatal dentine and cementum. One GLG was assumed to represent 1 year. Age data were not available for a few specimens; for these an estimate of age was made from the total length using the growth curves presented in Jefferson et al. (2002a). For more details on aging techniques, see Jefferson et al. (2002a).

Fresh carcasses (decomposition codes 2 and early 3) were qualitatively classified into one of two categories according to their overall long-term health status (i.e., in terms of chronic, as opposed to acute, conditions). Those specimens that showed evidence of a poor body condition (e.g., as evidenced by thinner than normal blubber layer, heavy parasite loads, and/or serious chronic pathological conditions), were classified as in 'poor' health. Those that showed typical body condition, normal blubber layer thickness, and no significant pathology (such as tumors) were categorized as in 'good' health. Acute conditions or trauma (such as evidence of vessel collisions or net entanglement) were not considered evidence of 'poor' long-term health. Admittedly, this classification system was crude and highly subjective, but it was intended to be the starting point for developing a more sophisticated approach in the future.

RESULTS

Levels and Causes of Mortality. – The yearly number of strandings of the two resident species of cetaceans in Hong Kong ranged from 8-15 for the finless porpoise, and from 6-11 for the humpback dolphin[3] (Fig. 1). There was no obvious annual trend in the number of finless porpoise or humpback dolphin strandings in Hong Kong between 1996 and 2000 (Fig. 1). The first year with complete data (1996) was apparently a year of high finless porpoise strandings, with 15 cases. Since then, the annual number of strandings has dropped to 11 or less.

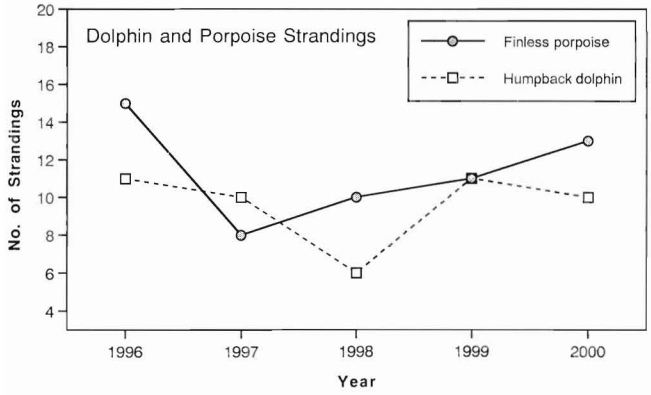

Fig. 1. Number of confirmed strandings of finless porpoises and humpback dolphins in Hong Kong, 1996-2000.

Between 1 September 1995 and 29 July 2000, a total of 54 finless porpoise deaths were confirmed for Hong Kong. Of this total, we were able to diagnose a cause in only 18 (33%) (Fig. 2). Most of the remaining 36 specimens (67%) were badly decomposed, although some were fresh with no evidence to indicate the cause of death. Generally, we could only determine a cause of death in freshly dead specimens - 72.2% of the diagnosed cases were code 2, while only 27.8% of the strandings as a whole were code 2. The most commonly identified cause of death was net entanglement, which was confirmed, or strongly suspected, in 8 of the 18 diagnosed cases (44%). The remaining causes of death were vessel collision (3 cases, 17%), uterine prolapse (3 cases,

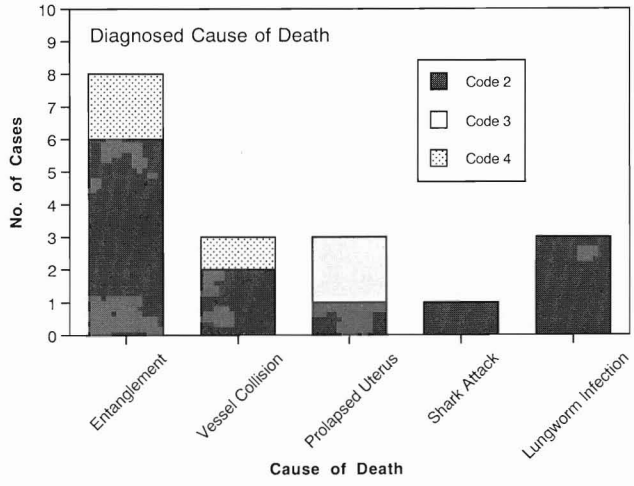

Fig. 2. Diagnosed causes of death for finless porpoises stranded in Hong Kong.

Fig. 3. Stranded finless porpoise specimen that was apparently attacked a large shark (NP97-12/04).

3 Humpback dolphin mortality data are presented here for comparison with the finless porpoise data, and to assist in determining overall trends in mortality rates of "resident" small cetaceans.

17%), lungworm infection (3 cases, 17%), and shark attack (1 case, 5%). The specimen that died from shark attack was half-eaten by a large shark - the first such record for Hong Kong (Fig. 3). We believe that the porpoise was bitten while it was alive, due to the presence of blood in the blubber layer surrounding the tooth marks.

There was a clear temporal trend in the finless porpoise cause-of-death data over the 5 years. None of the diagnosed causes of death until September 1997 were attributed to net entanglements. Since October 1997, however, all diagnosed causes of death were from fishing nets. This suggests that the incidence of net entanglement for finless porpoises in Hong Kong may have increased in recent years. Another possibility is that our ability to identify deaths from net entanglement has improved, but this would not explain the lack of other identified causes since October 1997.

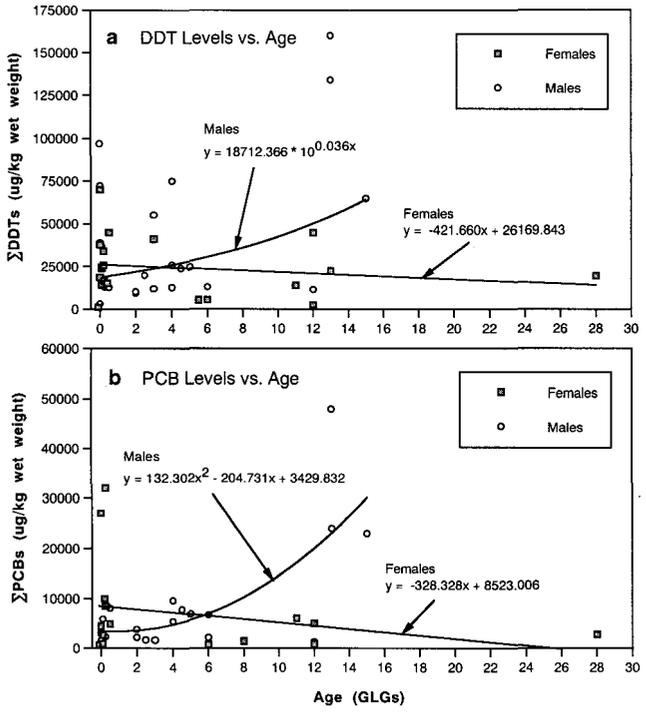

Fig. 4. Relationship between age and concentrations of organochlorines in Hong Kong finless porpoises: ΣDDTs (a) and ΣPCBs (b).

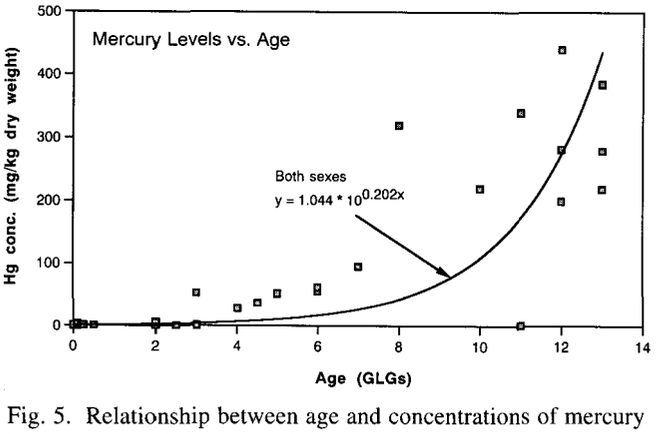

Fig. 5. Relationship between age and concentrations of mercury in Hong Kong finless porpoises.

Contaminant Concentrations. – Concentrations of total DDT ranged from 1,121-160,000 µg/kg wet weight (mean = 32,762.8 ± s.d. 32,838.94, n = 46). Some neonates showed very high concentrations (up to 96,960 µg/kg), but the highest level (160,000 µg/kg) was found in a 13-year old male (Fig. 4a). In females, there was a slight decreasing trend with age. Males showed more scatter, but in general there was a tendency toward increasing levels in older animals. Specimens classified as in 'poor' health (mean = 42,212.2 ± s.d. 44,356.77 µg/kg, n = 13) had levels of DDTs higher than did those in 'good' health (mean = 27,145.0 ± s.d. 14,021.05 µg/kg, n = 6). However, this difference was not significant (Mann-Whitney U = 38.0, df = 17, $p > 0.05$).

The range for total PCBs was 610-48,000 µg/kg wet weight (mean = 8,190.3 ± s.d. 10,591.58, n = 39). PCBs showed more clear cut patterns of change with age (Fig. 4b). Levels in females decreased with increasing age and the highest concentrations were in neonates. All adult females showed relatively low levels (< 5,000 µg/kg). Levels in males generally increased with age, and the 3 highest values were found in the 3 oldest male specimens. Levels of PCBs were slightly higher in 'poor' health specimens (mean = 8,188.9 ± s.d. 15,113.70 µg/kg, n = 9) than in 'good' health specimens (mean = 5,346.0 ± s.d. 2,451.65 µg/kg, n = 5), albeit a non-significant difference (Mann-Whitney U = 15.0, df = 12, $p > 0.05$).

Total HCHs ranged from 20-1,100 µg/kg wet weight (mean = 266.6 ± s.d. 315.12, n = 18). We did not plot age vs. HCH concentration by sex, due to the small overall sample size. However, there appeared to be a general tendency for reduced levels in older females - the three lowest concentrations were for three of the oldest females in the sample. Males presented much higher levels - 7 of the 8 highest concentrations were in males ranging from 0-13 years of age. Porpoises classified as in 'poor' health (mean = 344.1 ± s.d. 374.76 µg/kg wet weight, n = 10) had much higher levels than those considered to be in 'good' health (mean = 53.0 µg/kg wet weight, n = 2), although the sample size was too small to test for statistical significance. The range of total BT values was 0-160 µg/kg wet weight (mean = 21.9 ± s.d. 38.10, n = 17). We did not conduct further analyses of BT levels, due to the small sample size and paucity of data for female specimens (n = 4).

Mercury ranged from 0.49-440 mg/kg dry weight (mean = 86.5 ± s.d. 130.42, n = 40). Levels increased exponentially with age in a relatively predictable pattern, with no apparent difference between the sexes (Fig. 5). All young specimens (< 3 years of age) displayed levels near zero. Levels for 3-6 year-olds were intermediate, those for older specimens (10-16 years) were all very high (Fig. 4). Animals classified as in 'good' health (mean = 125.9 ± s.d. 184.14 mg/kg, n = 6) showed higher mercury concentrations than did those classified as in 'poor' health (mean = 61.4 ± s.d. 114.59 mg/kg, n = 14), although the difference was not significant (Mann-Whitney U = 29.0, df = 18, $p > 0.05$).

DISCUSSION

Levels and Impacts of Mortality. – Although Parsons (1998a) found a general increase in the annual number of strandings of finless porpoises in Hong Kong from 1973 to 1996, he was unable to determine if this increase resulted from increasing levels of mortality or from a general increase in the efficiency of the stranding recovery program. During the course of the present study (1996 to 2000), we found no evidence of an increasing trend in annual mortality. This suggests either that the increase reported by Parsons (1998a) was not the result of increasing mortality, or that such an increase has now generally levelled-off.

As previously reported for Hong Kong (Parsons, 1998a; Jefferson & Braulik, 1999), neonates comprised a high proportion of the total finless porpoise strandings (18 of 51 necropsied, or 35.3%). Of these, six were determined to have died as a result of trauma (and therefore we can assume that pollutants were not a factor). This leaves 12 of 51 (29.4%) being neonates with a cause of death potentially related to environmental pollutants. This is a seemingly high proportion, but it is known that first year mortality tends to be high in most mammals and cetaceans appear to be no exception (see Caughley, 1966; Barlow & Boveng, 1991). However, taken in association with the very high concentrations of DDT and other organochlorines recorded in the tissues of some neonates, it is suggestive of problems associated with high doses of potentially-toxic organochlorines being transferred from mother to offspring (as has been suggested for humpback dolphin calves in Hong Kong waters - Parsons, 1997).

We cannot evaluate the impact of the observed levels of mortality on the finless porpoise population, because of uncertainty about the overall population size of the stock and the actual level of mortality. However, the average number of strandings from 1996 to 1999 (mean = 11.0) represents 7.0% of the peak estimated abundance for finless porpoises in Hong Kong, and 4.5% of the peak estimated abundance for Hong Kong plus the surrounding areas that have been surveyed (Jefferson et al., 2002b). For dolphins and porpoises, annual mortality rates greater than 4-5% are considered to be unsustainable (Reilly & Barlow, 1986; Slooten & Lad, 1991). Thus, despite the many sources of uncertainty, a conservative approach to finless porpoise management in Hong Kong would view current levels of mortality as unsustainable.

Variation of Contaminant Levels with Age and Sex. – We found that organochlorine concentrations generally increased with age in male finless porpoises, which is in agreement with the literature on other species (Aguilar et al., 1999). Apparently the enzymatic mechanisms for metabolizing organochlorines are deficient in small cetaceans, and these substances therefore bioaccumulate in lipophilic tissues (Tanabe et al., 1988; 1994). In females, on the other hand, there was an overall decrease in levels with age; older adults generally had much lower levels than neonates (unfortunately, we had few specimens between 1 and 5 years of age). This is also in line with what has been found for other small cetaceans, and is apparently related to the reproductive transfer of lipophilic compounds to offspring through gestation and lactation in cetaceans (Tanabe et al., 1982; Tanabe & Tatsukawa, 1991; Aguilar et al., 1999).

Mercury concentrations showed a predictable exponential increase with age in Hong Kong finless porpoises. This is again consistent with what has been found in previous studies (see Bowles, 1999), and it apparently results from a relative inability to "offload" mercury, once it gets into a small cetacean's tissues. However, this may be tempered somewhat by a tendency for selenium to have a detoxifying effect on the body mercury stores (Bowles, 1999; O'Shea & Geraci, 1999). Parsons (1999) reported a significant correlation between selenium and mercury concentrations in Hong Kong finless porpoises, so this mechanism does appear to be in effect. In this study, we did not test samples to determine selenium levels, but we plan to do so in the future.

We were not able to test butyltin levels for variation with age and sex, but Takahashi et al. (2000) found that in general levels tend to increase in subadult marine mammals and then to level-off around the age of adulthood. The same basic trend was found for total tin (organic plus inorganic tin).

Geographic Differences in Contaminant Levels. – Pesticide (DDT) concentrations of finless porpoises from Hong Kong were exceptionally high compared to those from other parts of China (Yangtze River and the Yellow and Bohai seas) (Table 2). Levels from Japanese animals were also high, although not as high as those from Hong Kong. Minh et al. (2000a) compared concentrations of DDTs among nine odontocete species from 11 geographic regions, and Hong Kong finless porpoises showed the highest levels (followed by Hong Kong humpback dolphins). Parsons and Chan (2001) compared the ratio of DDT to its metabolites, DDD and DDE, between finless porpoises and bottlenose dolphins (*Tursiops truncatus*) occupying a more offshore habitat in the South China Sea. The ratio of DDT/DDD/DDE was 25%/40%/35% for finless porpoises and 7%/15%/77% for bottlenose dolphins. This suggests that finless porpoises are temporally and/or spatially closer to the source of DDT contamination. This is to be expected, as finless porpoises are more coastal than bottlenose dolphins in this area.

Few studies have reported levels of PCBs for finless porpoise populations (Table 2). The only area with comparative data is Japan, and in general the concentrations of animals from Hong Kong appear to be lower than those from Japanese waters. Thus, while PCBs still may be a concern, they are probably of less concern than is DDT. Levels of HCH in finless porpoises have not been commonly reported, and we could not find studies for comparison to our Hong Kong data. However, Parsons and Chan (1998) reported total HCH concentrations of 14.5-1,279 μg/kg wet weight, which supports the findings of this study. In the comparison of

Minh et al. (2000a), Hong Kong finless porpoises exhibited concentrations of PCBs and HCHs in the mid- to lower end of the range for nine species from 11 areas.

Butyltin concentrations have only been reported in the literature for finless porpoises from Japan and the East China Sea (Table 2). The Hong Kong specimens had BT levels in general much lower than those from these other areas. However, it should be remembered that these comparisons were made based on concentrations in the blubber, and it is now known that the liver contains the highest concentrations of these residues (Iwata et al., 1995; Takahashi et al., 2000). Takahashi et al. (2000) compared hepatic butyltin levels for cetaceans from 11 different geographical areas, and the Hong Kong specimens (finless porpoises and humpback dolphins) showed the highest levels. This suggests that butyltin contamination may be a serious issue for Hong Kong cetaceans, and additional Hong Kong specimens are currently being tested to further examine hepatic BT levels.

Finally, among the potentially toxic trace elements, only mercury has been analyzed in the current study. While there is a wide range, the maximum value for Hong Kong specimens was much higher than for specimens from Japan or other parts of China (Table 2). While the implications of this are unknown, this may be cause for concern.

The only other species of cetacean, besides the finless porpoise, that occurs regularly in Hong Kong waters is the Indo-Pacific humpback dolphin (Parsons et al., 1995). It may be instructive to compare levels of various contaminants between these two species, and we plan such a comparison when we have more data on humpback dolphin pollutant concentrations. However, in preliminary studies of organochlorine levels in these two species from strandings in Hong Kong, Parsons & Chan (1998) and Minh et al. (1999) found similar mean DDT concentrations between the two species, but levels of PCBs (Parsons & Chan, 1998; Minh et al., 1999) and HCHs (Minh et al., 1999) were more than twice as high in humpback dolphins.

It must be mentioned here that there is a potential bias in using stranded specimens to measure contaminant concentrations in cetacean populations. We know that organochlorine levels, at least, can be altered in an unpredictable manner by the actions of tissue decomposition, and that stranded specimens may not be representative of the population as a whole (see Aguilar & Borrell, 1994). Therefore, although there are legal and ethical issues to be considered, the use of skin and blubber biopsies for examining especially organochlorine levels is becoming increasingly more common (Ross et al., 2000).

The Effects of Environmental Contaminants. – Trace elements and heavy metals are common in the marine environment, especially in heavily industrialized regions. Cetaceans appear to accumulate these chemicals in their tissues, primarily through ingestion of prey, in proportion to their representation in the local environment (Johnston et al., 1996). There have been almost no specific studies of the toxicological effects of trace elements and metals on cetaceans, and most of what is known comes from inferences made from studies on humans (Johnston et al., 1996; Bowles, 1999). One notable exception was a recent study on wild bottlenose dolphins in the Atlantic Ocean, in which liver disease was found to be associated with chronic mercury contamination (Rawson et al., 1993). Animals in the current study had liver mercury concentrations quite similar to those of the specimens in the Rawson et al. (1993) study displaying liver disease. In this study, we did not find higher levels of mercury in 'poor' health specimens. However, it should be cautioned that the present study suffers from a number of limitations in its ability to detect evidence of health impacts. Mercury is known to have damaging effects on the nervous, excretory, and immune systems, and can cause mutations (O'Shea & Geraci, 1999). In view of this, we suggest that mercury should continue to be viewed as a pollutant of concern, and further study is clearly needed. Also, owing to selenium's protective effect against mercury toxicity, we plan to measure levels of selenium in the future

Compared to metals, more work has been done on organochlorines and their effects on cetaceans. Organic chemicals (including PCBs, hydrocarbons, and pesticides such as DDT) are known to be a potential threat to cetaceans, because they bioaccumulate in top predators, and are passed from generation to generation. Also, due to the absence or reduction of certain enzymes, cetaceans have a low capacity to metabolize (and thus detoxify) these compounds (Tanabe et al., 1994).

Organochlorines have been reported to interfere with reproductive capacity, cause immunosuppression (lowered resistance to disease), and have carcinogenic (cancer-causing) and teratogenic (development) effects (Tanabe & Tatsukawa, 1991; Busbee et al., 1999). Exposure during early development can affect the endocrine, reproductive, immune, and nervous systems, sometimes not expressing its effects until adulthood. For instance, high concentrations of PCBs and DDE were found to be correlated with lowered testosterone levels in the blood of Dall's porpoises (*Phocoenoides dalli*) in the North Pacific (Subramanian et al., 1987). In another study, Martineau et al. (1988) found that industrial contaminants were associated with lesions and cancer-like tumours in beluga whales (*Delphinapterus leucas*) in the St. Lawrence Estuary. Many of these were implicated in the deaths of the animals. Clear evidence showed that high levels of organochlorines suppressed the immune response of bottlenose dolphins in the southeastern USA (Lahvis et al., 1995). Finally, high concentrations of organochlorines are suspected to have been a causal factor in the die-offs of dolphins in the Mediterranean Sea and northeastern USA in recent years (Kannan et al., 1993; Reijnders et al., 1999; Aguilar, 2000). While the link between organochlorines and marine mammal die-offs has not yet been unequivocally proven, there is good reason to be concerned about such factors (Kennedy, 1999).

In this study, PCB levels were similar for 'good' and 'poor' health specimens. Concentrations of DDTs and HCHs were

Table 2. Levels of environmental contaminants found in finless porpoise tissues from different areas. Data are expressed in the following units of measure: µg/kg wet weight for DDTs, PCBs, and BTs; mg/kg dry weight for mercury (Hg) (except as noted). DDTs, PCBs, and BTs were analyzed in the blubber; mercury was analyzed in the liver (except as noted). Numbers in parentheses are sample sizes.

Area	ΣDDTs	ΣPCBs	ΣBTs	Mercury	Reference(s)
Japan	12,000-137,000 (6)	18,000-320,000 (7)	98-2,600 (4)	160[a] (1)	O'Shea et al., 1980; Kannan et al., 1989; Iwata et al. 1994,, 1995; Arima & Nagakura, 1979; Le et al., 1999
Yangtze River	1,480-131,800 (12+)	—	—	0.48-0.87[b] (2)	K. Zhou et al., 1993; Lui et al., 1983; Yang et al., 1988
Yellow Sea	2,270-77,690 (18)	—	—	0.24-34.8 (21)	R. Zhou et al., 1993a,b; Zhang et al., 1996
Bohai Sea	365-2,732 (10)	—	—	0.43-76.05 (13)	Zhang et al., 1993, 1995, 1996
East China Sea	—	—	350-1,200 (8)	31.8-105.8[c] (34)	K. Zhou et al., 1994; Tanabe et al., 1998
Hong Kong	5,464-260,349[d] (11)	339-14,652[d] (11)	—	<0.37-385 (15)	Parsons & Chan, 1998; Parsons, 1999
Hong Kong	1,121-160,000 (43)	610-48,000 (36)	0-159 (17)	0.49-440 (40)	Minh et al., 1999; This study

a The single specimen was a 72-cm fetus, and mercury was analyzed from muscle tissue.
b Mercury levels analyzed from muscle tissue.
c Presented on a dry weight basis.
d Converted from lipid weight basis using data in Parsons and Chan (1998).

somewhat higher in 'poor'-health animals than in those that were considered to be in 'good' health. The results must be interpreted with caution, however, because of the limitations described above and since differences were not statistically significant (probably due to small sample size and large variance effects). In fact, Minh et al., (2000b) found that toxic equivalents (TEQs) of PCBs in Hong Kong finless porpoises exceeded levels known to be associated with immune system dysfunction in harbour seals (*Phoca vitulina*). Suggestions that this could be a problem are supported by Parsons et al. (1999), who discovered evidence of immune system dysfunction in a finless porpoise calf stranded in Hong Kong. Further research is needed to determine whether these environmental contaminants are indeed affecting the health of finless porpoises in Hong Kong. In particular, DDTs appear to be a serious concern, due to their very high levels in some animals.

Recently, two newly identified organic contaminants, *tris*(4-chlorophenyl)methane and *tris*(4-chlorophenyl)methanol, have been found in significant concentrations in the blubber of finless porpoises from Hong Kong (as well as in Hong Kong humpback dolphins) (Minh et al., 2000c). The effects of these poorly known contaminants have yet to be investigated.

Butyltins (organotins) were only recently recognized as serious threats to marine mammal health. These compounds, most commonly used in anti-fouling paints applied to ship hulls in dry docks, are among the most toxic substances known to occur in the oceans. Although their serious effects on lower animals have been well documented, it is only in the last few years that researchers have even begun searching for them in cetaceans (see Tanabe et al., 1998; Tanabe, 1999). Finless porpoises in Japan were reported to have high levels of these compounds, likely representing a serious health risk (Iwata et al., 1995, 1997).

We did not examine sewage-borne bacteria in this study. The effects of sewage on wild cetaceans remain largely unknown (Johnston et al., 1996). However, it is known that sewage can introduce pathogenic bacteria (such as *Salmonella* sp. and *Vibrio cholera*) and viruses, which in turn can cause diseases such as hepatitis. Mycotic and fungal infections have also been linked to exposure to sewage in some other species, and high fecal bacterial levels have already been found in some waters of Hong Kong (Parsons, 1998b). There are many case studies in the veterinary literature of captive cetaceans succumbing to pathogenic bacteria (most of which are known to be associated with sewage discharges - see review in Parsons, 1998b), but apparently there have been no detailed studies on what effects sewage discharges have on wild populations of cetaceans. Although Hong Kong's proposed Strategic Sewage Disposal Scheme (SSDS) is intended to result in better treatment of sewage, finless porpoises may be at greater risk from sewage-related problems in the future if the SSDS outfall brings them into increased contact with human sewage (Parsons, 1998b). The impact of sewage pollution on finless porpoises and other cetaceans should be investigated further.

CONCLUSIONS

Although there has been a great deal of work examining concentrations of environmental contaminants in wild cetaceans, there has been very little work examining their effects on wild populations. The major exception appears to be for some of the organochlorines, where links between pollutant concentrations and health factors have been established. However, the overall paucity of such studies should not be taken to indicate that there is no reason to believe that these contaminants are problematic. For most of the above chemicals, studies on humans and other types of mammals have demonstrated the toxicity that can be expected (see Busbee et al., 1999). Unfortunately, there are few or no guidelines from elsewhere in the world for setting safe or acceptable water quality standards to protect coastal cetacean populations.

Dolphins and porpoises appear to be vulnerable to the toxic effects of various forms of environmental contamination, at least in the extremely high levels that they are currently being subjected to. The insidious ways in which these compounds often work - by compromising the immune system, inhibiting hormones, and causing reproductive and other organ system abnormalities - ensures that any impacts on cetacean health will be difficult to detect and prove. Therefore, we should not wait for incontrovertible proof, but instead begin to work on solutions now. This will be a challenging task, and will likely involve the development of new research techniques, and cross-disciplinary approaches. A first step should be to develop better methods of quantifying assessments of general body condition, pathology, and parasite loads. The largely qualitative nature of most veterinary assessments has continually limited our ability to look for links between contaminant levels and animal health. In addition to these research-related approaches, steps should be taken immediately to reduce the levels of known toxic substances that are being released into the environment.

ACKNOWLEDGEMENTS

We would like to thank all those who were involved in the necropsies and in collection and analysis of samples from the specimens. In particular, E. C. M. Parsons, I. Beasley, M. Torey, S. Hung, and L. Law were heavily involved at various stages. The marine mammal stranding program in Hong Kong is coordinated and funded by the Agriculture, Fisheries, and Conservation Department (AFCD), and we thank especially D. Choi, S. P. Lau, F. Y. Wong, W. W. Cheng, and J. Lun for their help over the last few years. Thanks to E. C. M. Parsons, R. R. Reeves, and B. D. Smith for reviewing earlier drafts of this paper.

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Appendix 1: Summary of finless porpoise contaminant concentration data.

Specimen No.	Length (cm)	Sex	Age (GLGs)	Code	Condition	DDTs (ug/kg wet)	PCBs (ug/kg wet)	HCHs (ug/kg wet)	BTs (ug/kg wet)	Hg (mg/kg dry)
NP93-06/05	140	F	3	4	nd	nd	nd	nd	nd	<0.9
NP93-22/12	71	F	0	2	nd	70,000	27,000	580	nd	1
NP94-30/07	155	?	11	nd	nd	nd	nd	nd	nd	<0.7
NP94-04/11	137	?	4	nd	nd	34,530	nd	120	nd	nd
NP94-16/12	82	F	0.1	nd	nd	nd	nd	nd	nd	0.5
NP95-15/03	105	M	0.25	2	Poor	13,000	2,300	260	nd	1
NP95-02/11	83	F	0.1	4	Poor	24,000	2,600	64	nd	1
NP95-25/12	143	F	3	2	Poor	41,350	nd	80	nd	<0.6
NP96-08/01	138	M	3	3	nd	55,270	nd	130	nd	nd
NP96-12/01	168	M	13	3	Poor	160,000	48,000	1100	nd	280
NP96-13/01	150	F	13	3	Good	22,570	nd	60	nd	385
NP96-14/01	86	M	0	2	Poor	72,220	nd	760	nd	<0.7
NP96-15/01	87	F	0.2	2	Poor	25,830	nd	130	nd	<0.9
NP96-23/01	67	M	0	2	Poor	96,960	nd	820	nd	<0.9
NP96-08/03	137	F	5	3	Poor	nd	nd	nd	nd	51
NP96-09/03	94	F	0.4	3	nd	15,000	32,000	nd	nd	<0.9
NP96-12/03	128	M	2	3	nd	9,500	2,200	nd	nd	<0.9
NP96-25/04	84	M	0.1	2	Poor	37,000	5,900	180	nd	<0.7
NP96-23/07	144	M	4	3	nd	26,000	6,700	54	0	62
NP97-23/01	157	F	12	3	Poor	2,600	800	27	nd	200
NP97-26/01	150	F	5.5	3	Poor	5,400	1,400	20	nd	320
NP97-12/04	>>130	F	11	2	Good	14,000	6,100	46	nd	340
NP97-15/08	157	?	8	3	nd	10,000	8,100	53	38.05	220
NP97-09/09	nd	M	nd	4	nd	58,000	32,000	315	nd	315
NP97-11/10	137	M	4	2	Good	44,700	5,530	nd	nd	28
NP98-04/01	75	F	0	3	nd	69,500	8,290	nd	nd	1.1
NP98-07/01	67	M	0	2	Poor	39,000	1,500	nd	15.37	1
NP98-06/03	158	M	4.5	4	nd	24,050	7,800	nd	47.94	37
NP98-09/04	99	F	0.5	2	Good	45,000	4,900	nd	nd	0.84
NP98-23/06	130	M	3	3	nd	12,000	1,600	nd	159.92	52.6
NP98-09/25	161	?	7	3	nd	16,000	5,050	nd	0	95.4
NP98-10/10	76	F	0	3	Poor	18,600	3,100	nd	0	0.49
NP98-15/11-A	168	F	12	4	nd	30,400	4,260	nd	nd	440
NP98-15/11-B	87 (fetus)	F?	-0.1	4	nd	1,121	610	nd	nd	nd
NP98-29/12	ca. 156	F	6	4	nd	3,240	506	nd	nd	55.7
NP99-02/01	131	M	4	4	nd	12,810	5,400	nd	nd	nd
NP99-30/01	160	M	13	3	nd	134,000	24,000	nd	nd	219
NP99-17/02	ca. 159	M	6	4	nd	13,079	2,100	nd	nd	nd
NP99-18/05	ca. 99	M	0.5	2	Poor	12,800	8,100	nd	nd	0.69
NP99-31/05	166	M	15	4	nd	65,000	23,000	nd	0	nd
NP99-16/07	154	M	5	3	nd	25,000	6,900	nd	nd	52.2
NP99-02/08	119	M	2	4	nd	10,200	3,800	nd	0	5.85
NP99-31/08	89	F	0.2	4	nd	34,000	10,000	nd	16.93	nd
NP99-31/10	140	?	7	4	nd	13,970	3,900	nd	0	nd
NP99-17/11	163	M	12	4	nd	11,600	1,300	nd	27.68	282
NP99-21/11	87	F	0.1	3	nd	14,100	830	nd	0	2.96
NP00-04/01	98	F	0.25	2	Good	16,600	8,500	nd	nd	0.68
NP00-12/01	77.3	F	0	4	nd	nd	nd	nd	0	nd
NP00-12/02	124	M	2.5	2	Good	20,000	1,700	nd	nd	0.79
NP00-26/03	165	F	28	4	nd	19,700	2,700	nd	nd	nd
NP00-26/05	77	M	0	4	nd	3,200	660	nd	0	nd
NP00-23/06	135.5	M	8	4	nd	nd	nd	nd	36.45	nd
NP00-05/09	168	M	12	4	nd	nd	nd	nd	29.62	nd