Aquatic Mammals 2012, *38*(3), 301-310, DOI 10.1578/AM.38.3.2012.301

An Unusual Mortality Event of Harbor Porpoises (*Phocoena phocoena*) Off Central California: Increase in Blunt Trauma Rather Than an Epizootic

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Abstract

In 2007, the apparent increase in the number of harbor porpoises (*Phocoena phocoena*) stranding along the central California coast compared to the number of strandings the previous year resulted in the declaration of an *Unusual Mortality Event* by the National Marine Fisheries Service. A statistically significant increase in strandings occurred in 2008 and 2009, with more than twice the mean annual number of strandings documented per year in the previous decade occurring each year, but then strandings decreased in 2010. No single cause of mortality explained all the strandings, and there were no significant changes in age class or sex of strandings in 2008 and 2009. Trauma, including interspecific aggression and fisheries interactions, was the most common cause of death, and blunt force trauma increased significantly in August through October of 2008 and 2009. Domoic acid toxicosis was documented for the first time in this species. Although the cause of death for many strandings was unidentified, the increase in strandings in 2008-2009 reflects an increase in blunt trauma rather than an epizootic of disease.

Key Words: mortality, trauma, harbor porpoise, *Phocoena phocoena*, infanticide, domoic acid, stranding, UME

Introduction

Harbor porpoises (*Phocoena phocoena*) are relatively abundant in the temperate near-shore waters of Europe, Asia, and North America (Gaskin et al., 1974), although many populations are declining due to a variety of factors, including fisheries interactions, climate change, and infectious diseases (Read et al., 1993; Hammond et al., 2002; MacLeod et al., 2007; Härkönen et al., 2008). Along the west coast of the United States, the population is not uniformly distributed, with considerable spatial and temporal variability in density and population structure (Barlow, 1988; Forney, 1999; Chivers et al., 2002). In ship-based surveys in 1995, significantly more porpoises than expected by surveyors occurred in water depths of 20 to 60 m, whereas fewer porpoises than expected occurred at water depths > 60 m (Carretta et al., 2001). Furthermore, densities have been highest on surveys in northern California and Oregon, and lowest off the Big Sur coast, while population abundances in Monterey Bay (Figure 1) were higher in September than in February (Barlow, 1988; Barlow & Forney, 1994). Four stocks are now recognized off California for management purposes, from north to south: a northern California stock, a San Francisco Bay-Russian River stock, a Monterey Bay stock, and a Morro Bay stock (Figure 1). These stocks were designated based on the distribution of animals, variability in mitochondrial DNA (mtDNA) control region sequences (Rosel et al., 1995), contaminant concentrations (Calambokidis & Barlow, 1991), and more recent mitochondrial and microsatellite DNA data (Chivers et al., 2002; Carretta et al., 2009). Population sizes of these stocks were recently estimated at 11,135 for the inshore stratum of the northern California stock, 8,521 for the San Francisco Bay-Russian River

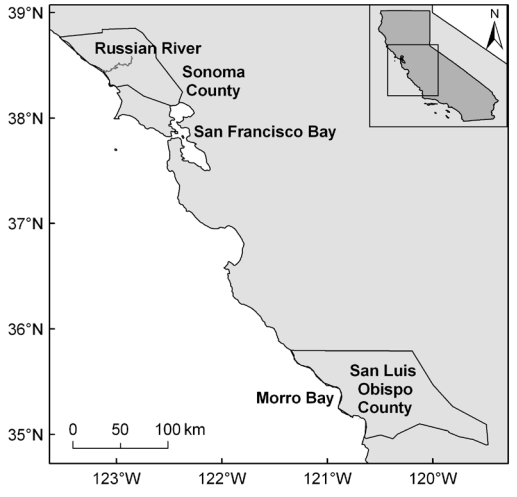

Figure 1. Map of California showing the stranding response area in this study

stock, 1,613 for the Monterey Bay stock, and 1,656 for the Morro Bay stock.

Worldwide, the most common cause of death of harbor porpoises is capture in gill nets (Read et al., 1993; Hammond et al., 2002). In a recent survey, 63% of determinable causes of harbor porpoise mortality in the mid-Atlantic region of the U.S. were interactions with fisheries (Read et al., 2006). Off California over the last 20 y, there has been high mortality in nets, with 200 to 300 animals taken annually 37 to 370 km off shore (Hanan et al., 1986; Julian & Beeson, 1998). In 1994, gill and trammel nets were banned in the Southern California Bight (SCB) within 3 nmi of the mainland coastline and 1 nmi of islands; and in September 2002, set gill nets were banned north of Point Arguello to Point Reyes in waters up to 60 fathoms (Carretta et al., 2001).

Since the 2002 restriction on gill nets in California, there has been little documentation about causes of mortality of harbor porpoises off the California coast. Between 1997 and 2002, three of 11 live stranded porpoises died of pneumonia, one died of trauma, and five were neonates that died following maternal separation (Zagzebski et al., 2006). Further north along the west coast, harbor porpoises have died around Vancouver Island in a novel epizootic due to *Cryptococcus gatti*, a fungus also affecting humans in the area (Datta et al., 2009). In other parts of the world, apart from fisheries interactions, harbor porpoise mortality has resulted from parasitic pneumonia and interspecies aggression (Ross & Wilson, 1996; Siebert et al., 2006).

In 2007, an apparent increase in the number of dead harbor porpoises stranded along the central California coast compared to the previous year triggered the declaration of an *Unusual Mortality Event* (UME) by the National Marine Fisheries Service (NMFS). Herein, we investigate the changes in stranding rates associated with this UME, and we document several causes of mortality in harbor porpoises off California for the first time.

Materials and Methods

Harbor porpoises that stranded dead along the central California coast from San Luis Obispo County in the south to Sonoma County in the north (Figure 1) were recovered by the California Marine Mammal Stranding Network and examined *postmortem*. The extent of the examination varied, depending upon the state of decomposition of the carcass, experience of the prosector, and availability of funds for analyses of samples collected. Date and location of stranding, sex, and age class were recorded, and a cause for stranding was assigned to each animal. Sex determination was based on genital morphology, and age class was defined based on standard lengths as follows: neonate (< 90 cm, presence of fetal folds and hairs, and an umbilical stump), juvenile (90 to 145 cm), and adult (> 145 cm) (Read, 1990). Cause of stranding was determined based on review of results of external examination, detailed gross necropsy examination, histopathology when tissues were fresh enough for histological examination, microbiology, and results of biotoxin analyses (Rowles et al., 2001). Histopathology was performed on tissues from 81 carcasses, and fluids were tested for presence of biotoxins from 27 animals. Histopathology was performed by examination of hematoxylin and eosin stained sections of tissues fixed in 10% neutral buffered formalin. Lesions noted upon gross necropsy in four animals were swabbed, and swabs were placed in Cary-Blair transport medium (BD Diagnostics, Franklin Lakes, NJ, USA) and held at 4º C until culture within 48 h of collection. All cultures were performed at the Microbiology Laboratory, William R. Pritchard Veterinary Medical Teaching Hospital, School of Veterinary Medicine, University of California at Davis.

Samples of urine, serum, amniotic fluid, stomach contents, and feces were frozen at -70º C prior to testing for domoic acid (DA) and saxitoxin (STX) at the National Ocean Service laboratory in Charleston, South Carolina. Urine, serum, and amniotic fluid samples were centrifuged at 13,000 × g and filtered using 0.22 µm centrifugal filter devices (Nanosep MF; Pall Life Sciences, East Hills, NJ, USA) in preparation for DA and STX analyses. Stomach contents and feces were extracted for DA testing by adding four volumes of 50% aqueous methanol to the sample volume, homogenizing, and then probe sonicating on ice for 2 min. These extracts were then centrifuged at 3,400 × g, and the supernatant

was collected and syringe-filtered prior to analysis by LC-MS as described in Fire et al. (2009). The limit of quantification of this method was 1 ng DA/mL for urine, serum, and amniotic fluid, and 4 ng DA/g for stomach contents and feces. Stomach contents and feces were extracted for STX testing by combining 2 g of sample with 2 mL of 0.1 N hydrochloric acid, and homogenizing and sonicating for 2 min using a Branson 450 probe sonicator (Branson Ultrasonics Corp., Danbury, CT, USA) at 30% power. The pH of the homogenate was adjusted to fall between 3.0 and 4.0. Homogenized samples were boiled for 5 min, cooled to room temperature, and centrifuged for 10 min at 3,400 × g. The supernatants were filtered through 0.2 µm hydrophilic polypropylene (GHP/GxF) syringe-driven filter discs (Acrodisc; Pall Life Sciences). The resulting extracts were analyzed for STX-like activity using receptor-binding assay (RBA) methods described in Van Dolah et al. (1994). The limit of quantification of this method was 18 ng STX-equivalents/mL for urine, serum, and amniotic fluid, and 35 ng STX-equivalents/g for stomach contents and feces.

Contingency table analysis was used to test whether frequency of strandings varied across variables such as month, age class, sex, cause of death, and county. Fisher's exact test was used when expected values in cells were low. When significant lack of fit was found with Fisher's exact test, subsequent Chi-squared tests were performed so that standardized residuals could be examined to determine where lack of fit occurred. A time series of the number of strandings per month was constructed for 1998 through 2010. This was seasonally adjusted to remove a pattern of variability by month by subtracting the mean number of strandings for each month (all years combined) from the monthly stranding time series. Subsequently, the changepoint package in *Program R* (Killick & Eckley, 2011) was used to determine when significant changes in mean seasonally adjusted strandings occurred. We were interested in detecting relatively large changes, so we initially limited the maximum number of change points to four. Change point analysis results are influenced by the analysis method, type of penalty applied, and the potential number of change points allowed (Killick & Eckley, 2011). We used the Binary Segmentation method (Scott & Knott, 1974; Sen & Srivastava, 1975) which allowed for specification of the maximum number of change points. We varied the number of allowed change points from two to six in order to evaluate the sensitivity of results to this parameter.

Results

Stranding Numbers

The NMFS declared an Unusual Mortality Event (UME) of harbor porpoise strandings in California in 2007 due to the apparent increase compared to previous years. A UME is a term for an increase in marine mammal strandings used by federal agencies in the U.S. to describe an event that is unexpected, involves a significant die-off of any marine mammal population, and demands an immediate response; it does not necessarily refer to an epizootic (Dierauf & Gulland, 2001). This declaration made funds available to the stranding network for extensive carcass examination. The UME was formally declared over in December 2010.

Between 1 January 1998 and 31 December 2010, 495 harbor porpoises stranded along the central California coast (Table 1). Using the 495 stranding records for all years, we found that there was significant differences in the frequency of strandings by month ($\chi^2 = 301.29$, df = 11, $p < 0.001$). In order to better discern the timing of any changes in mean stranding frequency between 1998 and 2010, we constructed a seasonally adjusted monthly time series. Change point analysis allowing for as many as four changes in mean seasonally adjusted strandings in fact detected four changes. These included an increase in June 2008, a decrease in November 2008, another increase in June 2009, and another decrease in November 2009, following which there was no more change through 2010. Thus, there were two periods of heightened stranding, accounting for season, and they occurred in June through October of both 2008 and 2009 (Figures 2 & 3). We ran change point analyses, allowing from two to six changes in mean. The results always detected the increase in June 2008, which we consider the onset of the significant increases in strandings (in contrast to the declared UME in fall 2007).

The difference in frequency and timing of strandings during June through October 2008-2009 are evident in Figure 2. In most years, there was a peak in strandings in summer. Notably, in 2008, the peak occurred later and was broader (sustained through October). In 2009, there was also a broader peak than average, but this was shifted back toward the typical timing compared to 2008.

To further evaluate the characteristics of strandings during the period of increased strandings, we tested whether the frequency of strandings during June through October of 2008 and 2009 differed from the same months in all other years with respect to month, age class, sex, cause of death, and location (county). There was no significant difference in the sex distribution of stranded porpoises during the period of increased strandings vs "normal" periods (Fisher's exact test, $p = 0.41$). There was a significant difference (Fisher's exact test, $p = 0.04$) in the age class distribution (calf, juvenile, adult, unknown), but this was due to a higher proportion of unknown-aged porpoises in the periods of increased strandings. When the

Table 1. Number of stranded harbor porpoises (*Phocoena phocoena*) along the central California coast by year and county, 1998-2008; C = calves (standard length [SL] < 90 cm), J = Juvenile (SL 90 to 145 cm), and A = Adults (SL > 145 cm).

	Contra Costa				Monterey				San Luis Obispo				Santa Clara				Sonoma			
Year	C	J	A	U	C	J	A	U	C	J	A	U	C	J	A	U	C	J	A	U
1998	1					6	4	1			1						1		1	
1999						4	2		1		1									
2000						2	4		1			1								
2001						1		1												
2002						1	1	1										1		
2003					1															
2004						5	3	1	3	2	2								1	
2005					2	5	6			2	1									
2006						4	2												1	
2007					3	4	5			2	1	1						1		
2008						8	4	1	3	9	7	9							1	
2009				1	1	6	4	1		4	4			1				1		
2010			1			4	1			2	1									
Total	1	0	1	1	7	50	36	6	8	21	18	11	0	1	0	0	1	3	4	0
County Total	3				99				58				1				8			

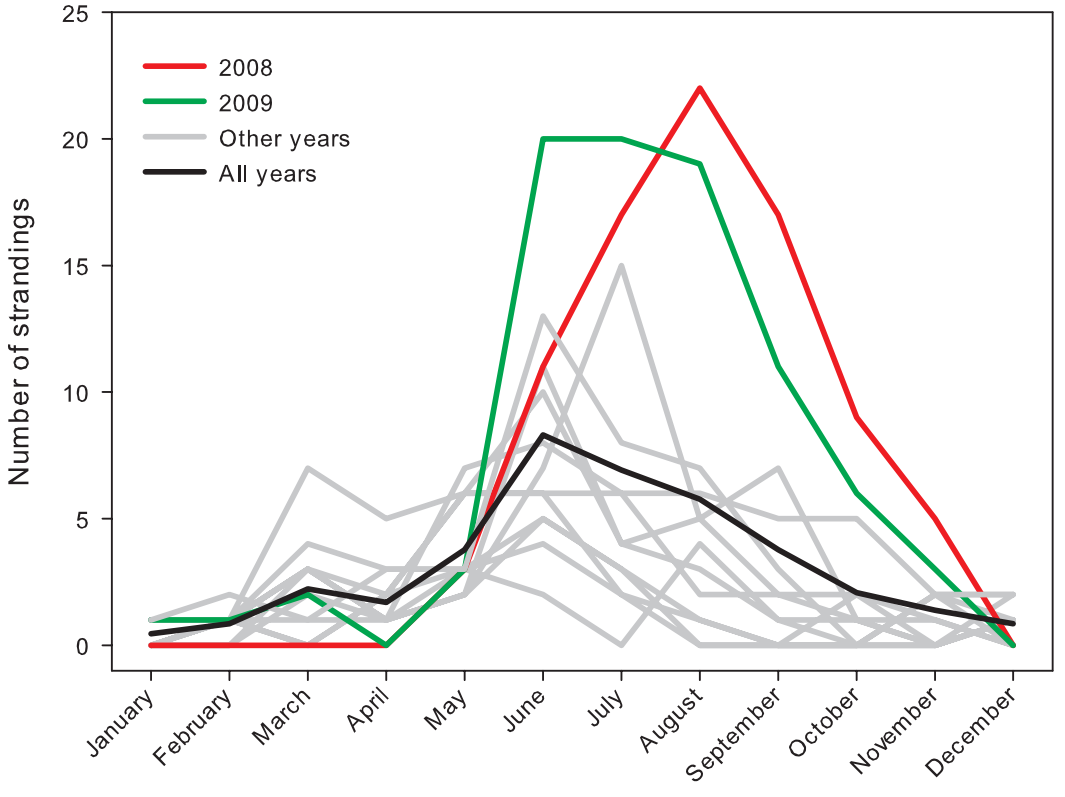

Figure 2. Seasonal changes in strandings that took place in 2008-2009 compared to other years

analysis was limited to known age class animals, there was no difference (Fisher's exact test, p = 0.40). As indicated by the shift to strandings later in the year (Figure 2), we detected significant differences in strandings across months during increased stranding periods (Fisher's exact test, $p <$ 0.001). Evaluation of Chi-squared test standardized residuals indicated that during periods of increased strandings, fewer strandings occurred during June but more occurred during August and September.

To evaluate changes in the location of stranded porpoises during the period of increased strandings, we combined relatively rare strandings from Alameda, Contra Costa, Santa Clara, and Sonoma Counties with nearby Marin County. In addition, we examined Monterey, San Francisco, San Luis Obispo, San Mateo, and Santa Cruz Counties individually. There were significant differences in the distribution of stranded porpoises by county (Fisher's exact test, p = 0.01). Based upon standardized Chi-squared residuals, this was due to a greater proportion of strandings in the relatively southern San Luis Obispo County and fewer in the more northerly San Mateo County during increased vs non-increased stranding periods.

Causes of Death

A number of different causes of death were observed (Figure 4), although for the majority of the porpoises (279/495), the cause of death was undetermined. Generally, this was due to the poor condition of the carcass when it was examined, either due to decomposition or scavenging, which precluded diagnostic sample collection for histology, microbiology, or toxicology. Because of their low frequencies of occurrence, we combined deaths due to domoic acid toxicosis, trauma (other), and "Other" categories when comparing frequencies of causes of death. There was a highly significant difference in the distribution of causes of death (Fisher's exact test, $p < 0.001$). Evaluation of standardized Chi-squared residuals revealed that this was because in increased stranding periods, a far higher proportion of blunt force trauma, but a lesser proportion of maternal separation and human interaction trauma, occurred compared to in other periods.

Neonate porpoises stranded more commonly between June and August than in other months in all years (Figure 3). The majority of neonates showed no specific lesions, and in 10 of these animals that were examined histologically, amniotic fluid or meconium was observed within the lungs indicating

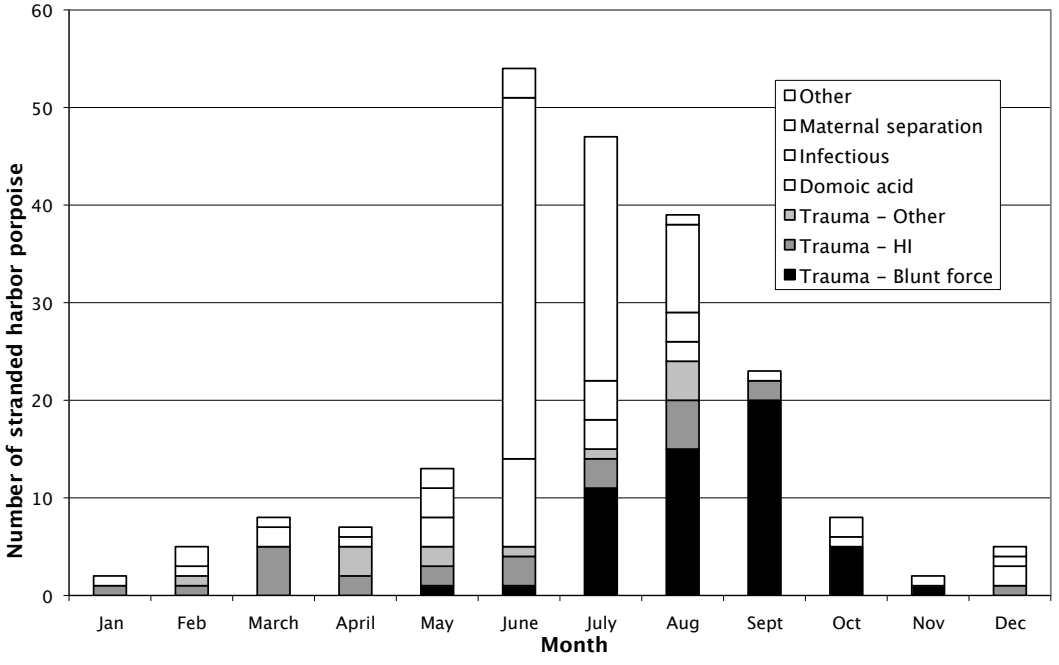

Figure 3. Seasonality of harbor porpoise mortality from different identified causes; HI = human interaction.

fetal distress before death; these deaths were classified as due to maternal separation, but the ultimate cause of death could not be determined. One male neonate (TMMC-C276) was severely bruised, with hemorrhages within the skeletal muscle of the dorsum and around the genital opening. Cause of bruising was unknown and could have resulted from dystocia or trauma from another animal or human.

While juvenile and adult porpoises stranded in all months of the year (Figure 3), all late-pregnant females stranded in June and July. Two periparturient female porpoises appeared to have died due to complications of pregnancy resulting in septicemia. One (TMMC-C235) had a large fetus in the vagina that could not be easily manually removed, a rupture of the uterine wall, and a septic peritonitis. Two other females (TMMC-C236 and TMMC-C259) had late term fetuses *in utero,* endometritis, and septicemia. Four periparturient females (TMMC-C153, TMMC-C187, TMMC-C257, and TMMC-C260) had mild to moderate meningoencephalitis. The significance of this inflammatory brain lesion was unclear.

Domoic Acid Toxicosis

Strandings attributed to domoic acid exposure were observed in 2002, 2005, 2007, and 2010 (Figure 4). Amniotic fluid from one pregnant female (TMMC-C153/LML-PP-2007May4) was positive for the biotoxin domoic acid at 1,177 ng/mL, and gastric fluid from another adult female (MLML-MM-1527) was positive for DA at 7.5 ng/g. Domoic acid toxicosis was also considered to be the cause of death of three male porpoises (TMMC-C210, LML-PP-2007May11, and TMMC-C240) with urine levels of 21,314, 10,712, and 30 ng/mL DA, respectively, as well as a subadult female porpoise with fecal and intestinal levels exceeding 25,000 ng/g DA. Two additional adult female porpoises (TMMC-C153 and TMMC-C328) had detectable levels of DA in feces (> 4 ng/g) and stomach contents (3 ng/g), respectively. Interestingly, the porpoises with high levels of domoic acid in urine did not have obvious lesions in the hippocampus. One (TMMC-C210) had a focal granuloma in the right temporal lobe and a moderate, chronic active, lymphoplasmocytic and neutrophilic meningoencephalitis.

Trauma

Trauma was the most common diagnosed cause of death, with 91 animals having hemorrhages and skeletal fractures. In 54 of these porpoises, the trauma was clearly multidirectional blunt trauma, with skull and bilateral rib fractures and hemorrhages in the blubber and skeletal muscle, occasionally accompanied by rake marks on the skin. Thirty of these cases were juveniles (17 males, 12 females, 1 unknown sex), and 24 were adults (11 males, 13 females), in addition to the one neonate described above. One juvenile male (TMMC-C240) with severe traumatic lesions, including fractured ribs and rake marks over 50%

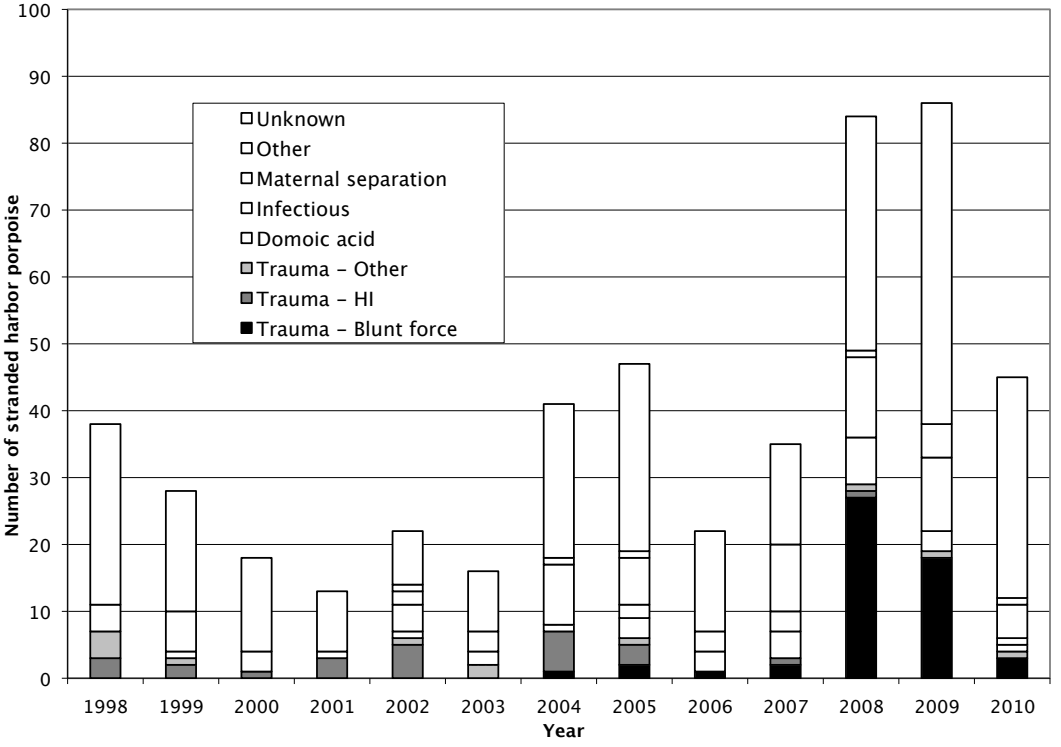

Figure 4. Number of harbor porpoises stranding between 1998 and 2010 from different causes of death; HI = human interaction.

of the body, had a neutrophilic and lymphoplasmocytic meningoencephalitis on histology. One juvenile male porpoise (TMMC-C264) appeared to have died due to asphyxia as it had a sculpin wedged in the blowhole and pink foam in the trachea, bronchi, and bronchioles, with no significant lesions on histology or domoic acid in body fluids.

Three animals (TMMC-C141, TMMC-C145, and TMMC-C190) died acutely with stomachs distended with partially digested fish, *perimortem* linear cutaneous injuries, and foam in the trachea and bronchi. They were therefore assumed to have died due to fishery interactions. An additional 22 animals were reported with signs of trauma consistent with human interaction (including net marks, cuts, and puncture wounds consistent with anthropogenic causes). Twelve animals died due to trauma from other or unclassified sources, including predator encounters (shark bites). Another juvenile female (TMMC-C159) had an intussusception, with a 4-cm-long section of jejunum telescoped through another, and a severe peritonitis. One adult female (LML-PP-2010JUN23) had a traumatic pyothorax associated with a *Chimaera* spine.

Infectious Disease

One juvenile female (TMMC-C164) had a severe necrotizing pyogranulomatous bronchopneumonia with intralesional bacteria typical of *Nocardia* sp., although bacteria were not isolated from lung cultures. One porpoise (LML-PP-24APR2006) had an encephalitis associated with the protozoa *Sarcocystis neurona* (Rejmanek et al., 2010). On histology, a number of lesions were observed in these porpoises that were not considered to have contributed to death. Verminous pneumonia characterized by a broncho-interstitial inflammation with eosinophilic granulomata around nematodes was observed to some degree in all adult animals examined histologically. In each animal with verminous pneumonia, varying severity of a membranous glomerulopathy with glomerulosclerosis was observed. Mild myocarditis and myocardial fibrosis with no visible etiological agent was observed in six adult animals (TMMC-C235, TMMC-C236, TMMC-C256, TMMC-C259, TMMC-C262, and TMMC-C268), and thymic cysts were observed in two adult female porpoises (TMMC-C268 and TMMC-C235). Interestingly, the fetus within one of these females (TMMC-C235F) had multinucleated giant cells within the thymus. Metastatic neoplasia suggestive of a mammary or urogenital carcinoma with metastasis to the spleen and regional lymph nodes was observed in one pregnant adult female porpoise (TMMC-C280). Cultures of the cervix and amniotic fluid for *Brucella* were negative.

Bacterial cultures yielded mixed cultures of a variety of organisms in most cases. *Klebsiella pneumonia* HMV type was cultured from the abdominal fluid of a fetus from a pregnant female (TMMC-C257). Other isolates from this mother-fetus pair included *Pseudomonas* sp. and *Psychrobacter phenylpyruvica*. *Actinobacillus scotiae* was isolated from the vagina of a septicemic pregnant female (TMMC-C259) in a mixed culture with *Protobacterium damsela*.

Discussion

These data demonstrate that the UME of harbor porpoises off the central California coast that was declared in 2007, with significantly elevated observed stranding rates in 2008 and 2009, was the result of an increase in mortality from several different causes, with the increase in strandings due to blunt trauma being the most significant. The number of strandings detected in each year is influenced by search effort and public reporting of strandings, as well as through changes in porpoise mortality, so it is likely that after 2007, a greater number of porpoise strandings were reported to the stranding network due to publicity surrounding the declaration of a UME. However, this change in effort cannot be quantified. The stranding of periparturient females and neonates in June and July rather than year-round suggests that seasonality in calving occurs. Mortality associated with domoic acid toxicosis is reported here for the first time in this species, and it contributed to the localized increase in mortality.

Examination of a dead porpoise cannot always conclusively determine the cause of death. In many cases in this study, investigations were limited by the state of decomposition of the carcass, precluding useful histology or microbiology. Lack of histology in many cases will result in under-diagnosis of diseases detectable only histologically such as encephalitis. In contrast, conditions easily diagnosed on gross examination, such as bone fractures following trauma, are likely well-represented in this dataset, although these cases could have had undiagnosed histological lesions, predisposing to trauma. In some cases that were examined histologically, lesions were observed that could not explain mortality but may have contributed to ill health. Thus, although in some cases gross lesions clearly indicated blunt force trauma as an immediate cause of death, in one of these cases examined histologically (TMMC-C240), meningoencephalitis likely predisposed the animal to attack. As not all cases were examined histologically, it is possible that further underlying diseases could exist, confounding results of the observations reported here.

The most common identified cause of death during the increase in strandings in 2007 through 2009 was blunt force trauma. The lesions observed in many of these porpoises were markedly similar to those described in porpoises and dolphins in other coastal waters assumed to have been attacked by bottlenose dolphins (*Tursiops truncatus*) (Ross & Wilson, 1996; Patterson et al., 1998; Dunn et al., 2002). Interspecific aggression by bottlenose dolphins on harbor porpoises is thought to be a consequence of the similarity in size between harbor porpoises and young dolphins, resulting in a misdirected infanticide (Patterson et al., 1998). The harbor porpoises observed with blunt force trauma during the summer and fall of 2008, however, were not all small animals: some ranged from 140 to 150 cm, with one female measuring 163 cm in length. During the increased stranding of porpoises with lesions typical of blunt force trauma in 2008, direct evidence of bottlenose dolphins attacking porpoises was obtained (Cotter et al., 2011); therefore, interspecies aggression from bottlenose dolphins was considered the most likely cause of trauma in these cases.

The domoic acid levels detected in several animals are among the highest values reported from a marine mammal, and they were about ten times higher than levels detected in California sea lions (*Zalophus californianus*) dying from domoic acid toxicosis in 1998 (Scholin et al., 2000). The lack of lesions in the hippocampus of these porpoises with high levels of domoic acid in urine is consistent with observations in sea lions that indicate lesions are only present in animals that have survived several days post intoxication, and not in animals dying peracutely (Silvagni et al., 2005). Domoic acid is a biotoxin produced by diatoms, especially *Pseudo-nitzschia australis,* that appear to be increasing off the central California coast in recent years, and blooms were observed during the summer mortality of porpoises in 2007 and 2008 (Bejarano et al., 2008). Domoic acid toxicosis thus contributed to the elevated stranding rates observed during this UME (particularly in 2007) but was not the sole cause of the increase in strandings.

Microscopic observations included conditions that likely represent incidental findings. The presence of endoparasites in the liver and lung or remnant inflammation are not uncommon in harbor porpoises (Raga et al., 2002). *Halocercus* sp. is the likely causative agent of the verminous pneumonia, and *Campulla* sp. are causative agents of the verminous cholangitis/periportal hepatitis and biliary hyperplasia. The parasite-induced inflammatory response varied from moderate to severe; and while this could have resulted in a degree of debilitation, it is unlikely to be the cause of stranding.

Membranous glomerulopathy was observed in adult females. Membranous glomerulopathy results from deposition of immune complexes (antigen + antibody) on glomeruli and can result in renal dysfunction. Membranous glomerulopathy can be primary or secondary; in the latter, systemic infection, chronic endoparasitism, cancer, and drug reactions can result in the formation of immune

complexes on the glomerular foot processes (podocytes) (Abramowsky et al., 1981). The porpoises with membranous glomerulopathy had chronic verminous pneumonia, which could have been the source of the immune complexes.

The meningoencephalitis observed in one juvenile harbor porpoise was a suppurative meningoencephalitis that was likely of bacterial origin. The source of bacterial infection was not determined, but it likely emanated from the sites of trauma. Nonsuppurative meningoencephalitis observed in the adult animals is not specific for a pathogen and could reflect chronic antigenic stimulation post-infection. The inflammation was mild and unlikely to have resulted in neurologic dysfunction. Possible causes would include viral (e.g., morbillivirus, herpesvirus), *Brucella* sp., protozoal (*Toxoplasma* sp.), or immune mediated; however, a causative agent was not observed in these cases. Immunohistochemistry and polymerase chain reaction should be used on future cases to further investigate the cause of the encephalitis observed, but these could not be performed retrospectively in this study due to lack of available tissues.

Other findings were considered to be incidental and did not contribute to the demise of the animals. Thymic cysts have been described previously in harbor porpoises (Siebert et al., 2006) and were observed in adult animals in which this lymphopoietic organ undergoes regression. Verminous and granulomatous lymphadenitis was present in many harbor porpoises and reflects sites of current or prior parasitic migration. Other causes of granulomas would include fungi and high order bacteria.

In conclusion, a variety of causes of mortality were observed in stranded harbor porpoises along the central California coast from 1998 through 2010. In many cases, cause of death was undiagnosed; and due to limited necropsies of some cases, mortality due to infectious disease and toxicoses may have been underdiagnosed. Trauma was the most common cause of death, with trauma from interspecific aggression (characterized by blunt force trauma) increasing significantly in August through October 2008 and 2009 and being documented for the first time in California.

Acknowledgments

We thank Tanja Zabka, David Rotstein, the Armed Forces Institute of Pathology, and Melissa Miller for necropsy examination of porpoises; Ray Bandar, Sarah Allen, and David Jessup for logistic support; Michelle Barbieri and Tenaya Norris for manuscript preparation; and all members of the California Marine Mammal Stranding Network for assistance with carcass recovery and examination.

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