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Review

Is Marine Mammal Health Deteriorating? Trends in the Global Reporting of Marine Mammal Disease

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Abstract: A recent rise in the reporting of diseases in marine organisms has raised concerns that ocean health is deteriorating. The goal of this study was to determine whether or not there has been a recent deterioration in marine mammal health by investigating the trends in disease reports over the past 40 years (categorized by the method of study, the species affected, and the etiology of the disease) and by exploring the changes in frequency of mass mortality events among marine mammals reported in the United States since 1978. The number of papers on marine mammal disease published each year has increased since 1966, although the annual publication rate appears to have stabilized since ~1992. Those published in the 1960s and 1970s were largely about helminth and bacterial disease, those investigating viruses emerged in the late 1970s and increased in the 1980s and 1990s, whereas protozoal diseases and harmful algal toxins were largely not reported until the 1990s. The annual number of mass mortality events in the U.S. approximately doubled between 1980 and 1990 but since 2000 has been between seven and eight events per year. Causes of mass mortality events have included biotoxins, viruses, bacteria, parasites, human interactions, oil spills, and changes in oceanographic conditions. Events due to biotoxins appear to be increasing, but the change in the frequency of mass mortality events from other causes over the past 40 years cannot be determined from the available published literature due to changes in marine mammal abundance, inconsistencies in effort and extent of resources for pathological investigation, and advances in technology that have allowed improved detection of pathogens and toxins in more recent years. To ensure future information on the true incidence of marine diseases and their underlying causes is more reliable, specific and directed marine health monitoring programs, well-equipped stranding networks, and dedicated diagnostic laboratories are needed.

Keywords: disease, epidemiology, marine mammal, mortality, morbidity

INTRODUCTION

A recent rise in the reporting of diseases in marine organisms has raised concerns among scientists, politicians, managers, and the public that ocean health is deteriorating (Epstein, 1996; Geraci et al., 1999; Harvell et al., 1999; Gardner et al., 2003). Marine diseases, particularly those that cause large-scale die-offs, can alter population distribution and abundance, and cause major regime changes within marine communities (Harvell et al., 2004; Kim et al., 2005). Furthermore, threats to ocean health can directly

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and indirectly impact human health, and many emerging diseases of wildlife are zoonotic (National Research Council, 1999; Knap et al., 2002; Knowlton, 2004; Cunningham, 2005). However, whether the increase in reports represents a real and widespread degeneration in the health of marine life is unclear (Harvell et al., 1999; Lafferty et al., 2004; Ward and Lafferty, 2004). The lack of information on the true incidence of marine mammal diseases (i.e., the rate at which new cases of the disease are occurring during a specified period) and their underlying causes are largely due to the lack of specific and directed marine health monitoring (Harvell et al., 2002; Kim et al., 2005). This is probably a consequence of the historical focus on domestic animal health rather than wildlife disease, and the emphasis on farm animal economy rather than conservation.

Marine mammals are particularly difficult marine organisms to study due to their aquatic, often pelagic, and non-sedentary life styles, long generation times, large body size and protected status. However, when marine mammals do wash ashore (strand) sick or dying, they command considerable attention and concern. There is, thus, an interest from the public, as well as from scientists and managers, to determine the state of marine mammal health and to identify the anthropogenic influences (such as exposure to pollutants and terrestrial pathogens, increased ocean noise and boat harassment) on the well-being of these animals. The stranding of beaked whales in association with navy exercises has raised awareness of direct effects of human activities on marine mammal health (Fernández et al., 2004; Cox et al., 2006). Indirect effects are less obvious, but there is no doubt that overt morbidity and mortality among marine mammals have resulted from terrestrial pathogens spreading to the ocean, as well as from harmful algal blooms and epidemics of virulent viruses and bacteria (Geraci et al., 1999; Miller et al., 2002). Although the role of anthropogenic factors and climate change in these die-offs is uncertain, there is a compelling body of evidence indicating that persistent organic pollutants can affect marine mammal immunity (DeSwart et al., 1994; Ross et al., 1995; Hammond et al., 2005) and the exposure of individuals to these compounds may have played a role in some viral mass mortality events (Hall et al., 1992; Aguilar and Borrell, 1994). Furthermore pathogens of terrestrial origin, such as *Toxoplasma gondii* and antibiotic-resistant bacteria are causing disease in marine mammals, and appear to originate in fresh water run-off (Miller et al., 2002; Stoddard et al., 2005). Thus anthropogenic effects on the ocean may be adversely impacting marine mammal health, causing an increase in disease outbreaks and deterioration in health.

Direct data on conventional indices used to quantify health in humans and domestic animals (hematology, serum biochemistry, and immune function markers) are relatively sparse for marine mammals, and there are insufficient published data over several decades to assess changes in health using these reports. Instead, we use frequency of disease outbreaks as indicators of health status. Data were used from two sources. Firstly, we investigate the trends in disease reports published in the scientific literature over the past 40 years, categorized by the method of study, the species affected, and the type of organism or toxin involved; secondly, we explore the changes in frequency of mass mortality events among marine mammals reported both in the United States (U.S.) since 1978, and worldwide. Data on mass marine mammal mortality events in the U.S. are considered separately not because this country is representative of the global condition, but it has a very long coastline, a wide variety of well-studied Atlantic and Pacific marine mammal species, and, under the auspices of the Marine Mammal Protection Act, has a formal and structured response to mass marine mammal mortality events, which means that effort at reporting these larger events has been relatively consistent since 1978.

Methods

Papers on Disease Published in Peer Reviewed Journals

To investigate and assess the trends in marine mammal disease, we reviewed 772 papers published in peer reviewed journals since 1966. Databases searched included PubMed, Medline, CAB abstracts, Google scholar, and ISI Web of Knowledge; search terms included marine mammal, pinniped, cetacean, disease, health, virus, bacteria, toxin, parasite, epidemic. A list of the papers (with their abstracts) included can be found at http://www.smru.st-and.ac.uk. Papers were categorized by both authors according to disease agent investigated, method used to investigate disease in marine mammals, and by species affected. Papers described diseases in individual marine mammals, as well as mass mortality events. Papers were published 1–5 years after a disease was identified, so these data reflect trends in disease reporting over time and not the absolute changes in the true dates that each disease was identified.

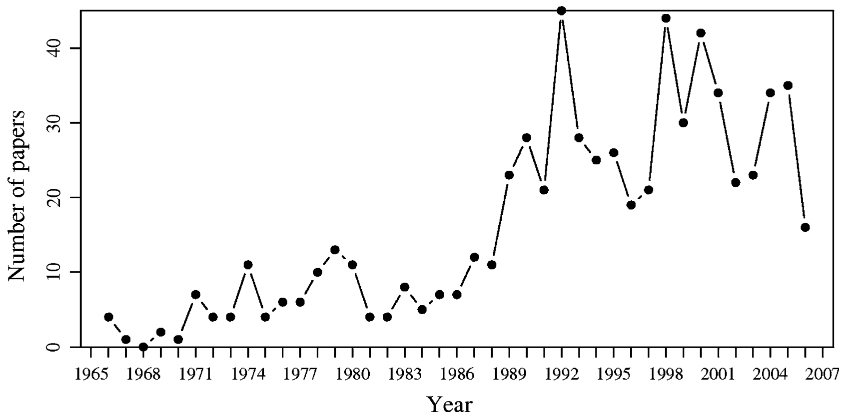

Figure 1. Number of papers on disease in marine mammals published in peer reviewed journals, 1960–2006. Note: 2006 includes papers to July only.

Mass Mortality Events

Reports of mass mortality events worldwide were obtained from the published literature, and reports of unusual mortality events in the United States were also obtained from the administrative records of the National Marine Fisheries Service (NMFS) Marine Mammal Health and Stranding Program (Silver Spring, Maryland, U.S.). These records result from stranding network members detecting sick or dead marine mammals on the beach and submitting data on each animal (including date, species, length, age class, sex, and location) to the regional stranding coordinators of the NMFS. In the U.S., large mortality events involving marine mammals are referred to in two different ways. A stranding event, in which animals usually beach alive, involving more than two marine mammals (other than mother–calf pairs) are defined as "mass strandings" (Geraci et al., 1999). These mass strandings are most common on the eastern coastline, typically involve odontocetes, and the cause is generally not identified (Geraci et al., 1999). Other mass mortalities are referred to as "unusual" mortality events as unusually large numbers of animals die. Furthermore, in an effort to improve response and investigation of these unusual mortality events, a formal federal designation of "Unusual Mortality Event" (UME), also exists, the definition being applied to an unusual event following the vote of a federally appointed group of experts that use seven criteria upon which to base their decisions. These criteria include: (1) a marked increase in the magnitude or a marked change in the nature of morbidity, mortality, or strandings when compared with prior records; (2) a temporal change in morbidity, mortality, or strandings is occurring; (3) a spatial change in morbidity, mortality, or strandings is occurring; (4) the species, age, or sex composition of the affected animals is different than that of animals that are normally affected; (5) affected animals exhibit similar or unusual pathologic findings, behavior patterns, clinical signs, or general physical condition (e.g., blubber thickness); (6) potentially significant morbidity, mortality, or stranding is observed in species, stocks, or populations that are particularly vulnerable (e.g., listed as depleted, threatened, or endangered or declining); (7) morbidity is observed concurrent with or as part of an unexplained continual decline of a marine mammal population, stock, or species. Since 1978, the administrative record of unusual mortality events has been relatively consistent, although the formal designation of the term "unusual" has varied. Thus, for this article, we examine all records of unusual mortality events, to minimize the effect of changes in nomenclature on our ability to detect changes in frequency of disease outbreaks. Mass strandings are not considered here, as these typically have not been determined to result from disease outbreaks (Geraci et al., 1999).

Results

Papers on Disease Published in Peer Reviewed Journals

The number of papers on marine mammal disease published in the scientific literature each year has increased (Fig. 1), although the annual publication rate appears to have stabilized since ~1992. In an attempt to determine any trends in the etiologies of marine mammal disease, we categorized the published studies by the agents for the disease being investigated (viral, bacterial, helminth, fungal, protozoal, and harmful algal toxins). The trend in the different disease agents studied (as a proportion of the total number reviewed, by 3-year categories) is shown in Figure 2a and b. Those papers published in the 1960s and

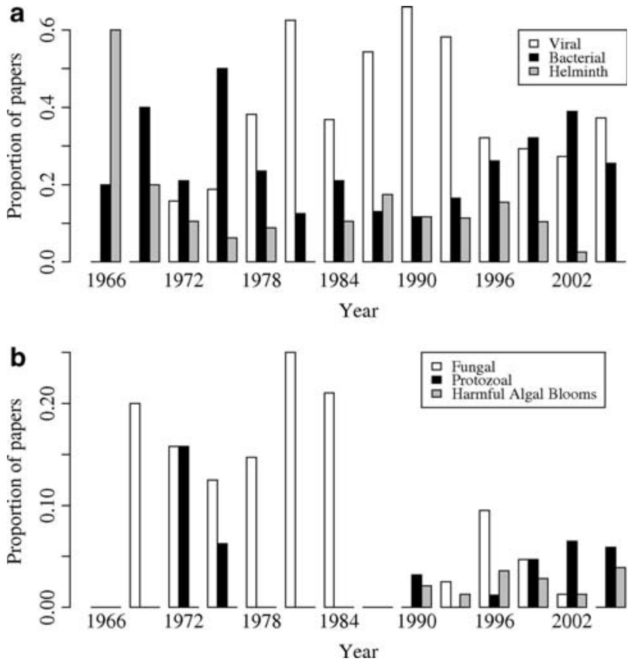

Figure 2. Proportion of the total number of papers by 3-year categories, relating to **(a)** viral, bacterial, or helminth disease; **(b)** fungal, protozoal disease, or harmful algal blooms.

1970s were largely about helminths and bacterial disease; those investigating viruses emerged in the late 1970s and increased in the 1980s and 1990s. Fungal disease, particularly lobomycosis in dolphins, seems to have been particularly studied in the late 1970s and early 1980s, whereas protozoal diseases and the effects of harmful algal toxins on marine mammals were largely not reported until the 1990s (Fig. 2b). Although the studies must be interpreted in the light of the type of investigation and may, to some extent, be biased by any delay in publication, they appear to reflect our increased ability to detect and identify more fastidious pathogens, such as small bacteria, protozoa, and algal toxins in more recent years. As novel laboratory techniques have been introduced, the number of publications on the agents that the techniques can detect increased. For example, the introduction of polymerase chain reaction (PCR) as a routine diagnostic tool for detecting viruses in the late 1980s was associated with an increase in reports of virus infections in marine mammals, and use of high performance liquid chromatography for the detection of biotoxins was not common until the 1990s.

The scientific approaches used to study the occurrence of disease have also changed over time. Most studies are investigations on individuals and 12% of the publications were case studies, reporting disease on one animal. Few studies have included sufficient individuals to be able to make inferences about impacts of disease at the population level, with the exception of the effect of mass mortalities. Figure 3a and b shows how the proportion of each sampling method reported (of the total number of papers) has changed since 1966. The number of studies using harvested and captive animals has steadily declined (Fig 3a). Studies where only animals by-caught in fishing nets were included are not seen until the late 1990s, but studies on stranded animals will include by-caught animals as well as those found washed ashore. Studies on stranded animals have increased, and constitute approximately 57% of the papers reviewed (Fig. 3b). Studies from live stranded and rehabilitated marine mammals have also increased, with few studies on these animals prior to 1985. Papers on mass mortalities, compared with studies on individually stranded animals, peaked in the early 1990s. This peak reflects reports from the high profile morbillivirus outbreaks in the U.S. and Europe in the late 1980s. A second peak occurs in the 2005–present year group, largely as a result of publications on the 2002 European phocine distemper virus (PDV) outbreak.

The vast majority of published studies have been carried out on the harbor seal *(Phoca vitulina)*, with the bottlenose dolphin *(Tursiops truncatus)* the most commonly studied cetacean. This probably reflects the relative abundance and near-shore distribution of these two species and the fact that bottlenose dolphins have often been kept in captive display facilities where case studies (particularly on lobomycosis) have been carried out. Harbor seals have also been subject to mass mortalities in recent years and harbor porpoises *(Phocoena phocoena)*, which also feature high on the list, are often stranded or by-caught. Interestingly, the distribution is about equal between seals and cetaceans, but it is clear that almost nothing has been reported about diseases in some species.

Temporal and Spatial Trends in Unusual Mortality Events

The total number of unusual mortality events reported over time in the U.S. is shown in Figure 4a. The annual number of events approximately doubled between 1980 and 1990 but, since 2000, it has remained at between seven and eight events per year. Causes of mortality events have included biotoxins, viruses, bacteria, parasites, human interactions, oil spills, and changes in oceanographic conditions

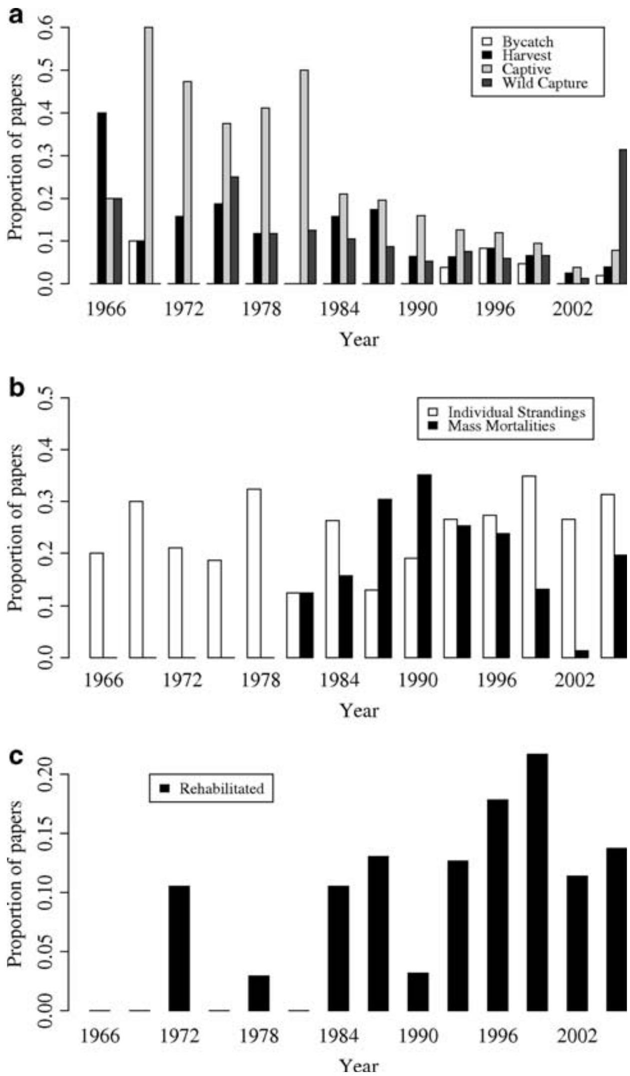

Figure 3. Proportion of the total number of papers by 3-year categories, using (**a**) by-caught, harvested, captive, or wild captured; (**b**) individual strandings and mass mortalities; (**c**) rehabilitated marine mammals to study the occurrence of disease.

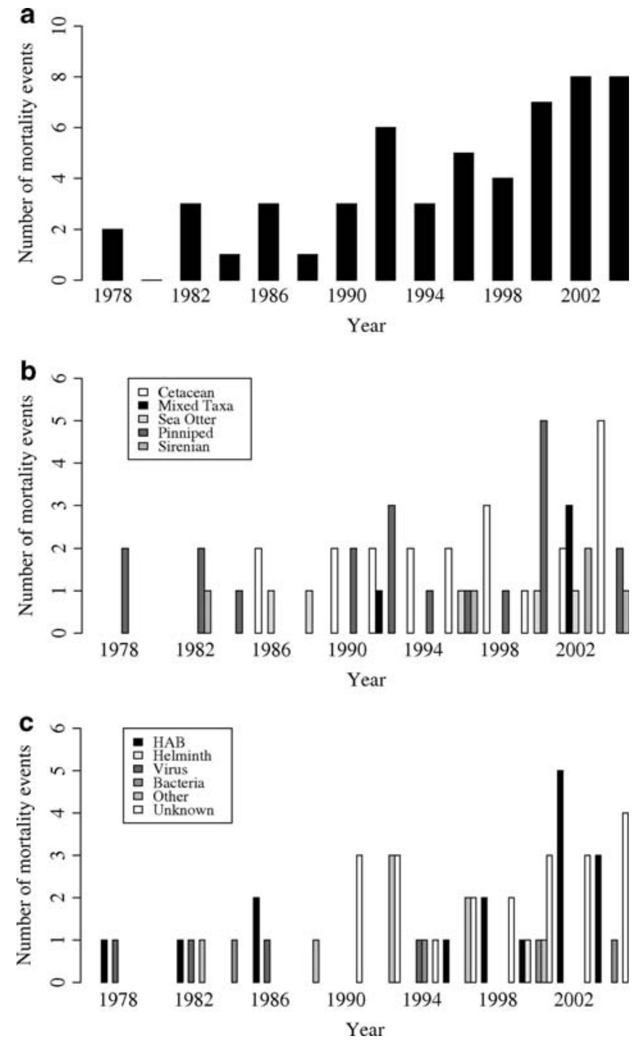

Figure 4. Marine mammal unusual mortality events in the U.S. (data from National Marine Fisheries Service database as listed in Table 1) by 2-year categories in (**a**) total number of marine mammal mortality events in the U.S. by year; (**b**) by identified causes, 1978–2005 (HAB = harmful algal blooms; other = human interaction, oil spills, and changes in oceanographic conditions); (**c**) in different taxa, 1978–2005.

(Fig. 4b). Prior to 1996, when a manatee die-off was caused by brevetoxin, only five mass mortality events were associated with exposure to biotoxins from harmful algal blooms. Since then, 12 of 31 events in the U.S. have been associated with exposure to biotoxins (Fig 4c), and events caused by domoic acid on the West coast of the U.S. and brevetoxin on the East coast are increasing in frequency. This reflects a true increase in the frequency of harmful algal blooms around the U.S., especially over the last decade (Van Dolah, 2000).

Frequency of unusual mortality events from other causes has not obviously changed in recent years. Interestingly, influenza virus was the cause of two mortality events in 1979 and 1982, but in more recent years, influenza epidemics in seals have not been detected and morbillivirus epidemics have been the more common causes of virus-associated mass mortality events.

Some other types of mortality events, such as those along the California coast associated with El Niño and leptospirosis, also occur at regular intervals. Mortality events in the U.S. have been most frequent in Florida and California, and have been most frequently reported in bottlenose dolphins, California sea lions *(Zalophus californianus)*, and manatees *(Trichechus manatus latirostris)*.

Table 1. Summary of Marine Mammal Unusual Mass Mortality Events Reported 1978–2005[a]

Year	Species and no. affected	Location	Cause and comments	References
1978	Hawaiian monk seals *Monachus schauinslandi* (50)	Northwest Hawaiian Islands, U.S.	Ciguatoxin and maitotoxin suspected	Gilmartin et al., 1980
1979–1980	Harbor seals *Phoca vitulina* (400)	Cape Cod, Massachusetts, U.S.	Influenza A (a mycoplasma was concurrently isolated from these seals)	Geraci et al., 1982
1982	Harbor seals *Phoca vitulina*	Cape Cod, Massachusetts, U.S.	Influenza A	Hinshaw et al., 1984
1982	Florida manatees *Trichechus manatus latirostris* (39)	Southwest Florida, U.S.	Brevetoxin	O'Shea et al., 1991
1983	Several pinniped species (especially California and Galapagos sea lions *Zalophus californianus);* Galapagos fur seals (thousands)	West coast of U.S.; Galapagos	El Niño	Trillmich and Ono, 1991
1984	California sea lions *Zalophus californianus* (226)	California, U.S.	Leptospirosis	Dierauf et al., 1985
1987	Sea otters *Enhydra lutris* (34)	Kodiak Island, Alaska, U.S.	Saxitoxin	DeGange and Vacca, 1989
1987	Humpback whales *Megaptera novaeangliae* (14)	Massachusetts, U.S.	Saxitoxin	Geraci et al., 1989
1987–1988	Bottlenose dolphins *Tursiops truncatus* (645)	New Jersey, Delaware, Virginia, North Carolina, South Carolina, Georgia, Florida, U.S.	Morbillivirus. Brevetoxin was detected in dolphins, its role in the event is unclear	Scott et al., 1988; Geraci, 1989; Lipscomb et al., 1994; Duignan et al., 1996; Schulman et al., 1997; Friedlaender, 2000; McLellan et al., 2002
1987–1988	Baikal seals *Phoca siberica* (80–100,000)	Lake Baikal	Canine distemper virus (CDV)	Grachev et al., 1989; Osterhaus et al., 1989
1988	Harbor seals *Phoca vitulina* (~18,000)	Northern Europe; U.K.	Phocine distemper virus (PDV)	Kennedy et al., 1988; Osterhaus et al., 1988
1989	Sea otters *Enhydra lutris* (3500–5000)	Alaska, U.S.	*Exxon Valdez* oil spill	Loughlin, 1994
1990	Striped dolphins *Stenella coeruleoalba* (550)	Mediterranean Sea	Dolphin morbillivirus (DMV)	Aguilar and Raga, 1993; Domingo et al., 1992
1990	Bottlenose dolphins *Tursiops truncatus* (146)	Texas, Louisiana, Mississippi, Alabama, U.S.	Unknown. Unusual skin lesions observed	B. Brown memo to N. Foster, March 2, 1990; Kuehl and Haebler, 1995; Hansen, 1992; Medway report to Fox, June 29, 1990

Table 1. Continued

Year	Species and no. affected	Location	Cause and comments	References
1991	Harbor seals *Phoca vitulina* (34)	New Jersey, U.S.	Unknown. *Erysipelothrix rhusiopathiae* cultured from 4 seals, poxvirus present in 1, saxitoxin negative	Informational memo J. A. Knauss, May 10, 1991
1991	California sea lions *Zalophus californianus* (160)	California, U.S.	Leptospirosis	Gulland et al., 1996a
1991	Bottlenose dolphins *Tursiops truncatus* (30)	(Sarasota) Florida, U.S.	Unknown	Working Group files, NMFS
1992	Harbor seals *Phoca vitulina* (29); Harbor porpoises *Phocoena phocoena* (5); Rough toothed dolphin *Steno bredanensis* (1)	Oregon, Washington, U.S.	Unknown	H. Braham memos to W. Aron Oct. 9, 1992; Oct. 16, 1992; and Dec. 1992
1992	Dugong *Dugong dugong* (100)	Queensland, Australia	Starvation	Preen and Marsh, 1995
1992	Common dolphins *Delphinus delphis* (118)	Southwest England, U.K.	Fisheries interaction	Kuiken et al., 1994
1992	Phocid seals (24)	Maine, Massachusetts, Connecticut, U.S.	Unknown. Morbillivirus and/or influenza suspected	Geraci et al., 1993; Callan et al., 1995; Early, 1992
1992	Bottlenose dolphins *Tursiops truncatus* (220)	(Calhoun and Aransas counties) Texas, U.S.	Carbamates suspected	Colbert et al., 1999; Duignan et al., 1996; Sweeney, 1992
1992–1993	California sea lions *Zalophus californianus* (∼1000)	California, U.S.	El Niño. 50 gunshot	Greig et al., 2005
1993	Harbor porpoises *Phocoena phocoena* (64)	Maine, Massachusetts, Connecticut, New Jersey, Delaware, Virginia, North Carolina, U.S.	Fisheries interaction	Haley and Read, 1993; Read and Murray, 2000
1993	Pinnipeds (53)(9 Steller *Eumatopias jubatus;* 15 California sea lions *Zalophus californianus;* 28 harbor seals *Phoca vitulina;* 1 unknown species sea lion)	Washington, U.S.	Gun shot. Bullets found on gross necropsy and X-ray	Norberg [personal communication]
1994	Common dolphins *Delphinus delphis*	Black Sea	Morbillivirus	Birkun et al., 1999
1994	Common dolphins *Delphinus delphis* (53)	California, U.S.	Listed in Wilkinson, 1996, as a mortality event but not formally voted a UME by the Working Group. Etiology unknown	Reidarson et al., 1998

Table 1. Continued

Year	Species and no. affected	Location	Cause and comments	References
1994	Bottlenose dolphins *Tursiops truncatus* (72)	Texas, U.S.	Morbillivirus	Lipscomb et al., 1996
1995	California sea lions *Zalophus californianus* (222)	California, U.S.	Leptospirosis	Greig et al., 2005
1996	Sea otters *Enhydra lutris* (68)	(Cordova) Alaska, U.S.	Malnutrition, parasites, cold	Ballachey et al., 2002
1996	Right whales *Eubalaena glacialis* (6)	Florida, Georgia, U.S.	Blast injury suspected	Ridgway, 1996
1996	Florida manatees *Trichechus manatus latirostris* (149)	Florida (West coast), U.S.	Brevetoxin	Bossart et al., 1998; Landsberg and Steidinger, 1998
1996	Bottlenose dolphins *Tursiops truncatus* (30)	Mississippi, U.S.	Unknown. Coincident with algal bloom	Working Group annual meeting report 1997 notes
1997	Mediterranean monk seals *Monachus monachus* (150)	Western Sahara	Harmful algal bloom/morbillivirus	Harwood, 1998; Hernandez et al., 1998; Osterhaus et al., 1998
1997	Harbor seals *Phoca vitulina* (90)	California, U.S.	*Pseudomonas aeruginosa* associated with pneumonia. Virus suspected.	Working Group files, NMFS
1998	Hooker's sea lions *Phocarctos hookeri* (60% of pups)	New Zealand	Unknown, bacteria likely	Baker, 1999
1998	California sea lions *Zalophus californianus* (70)	California, U.S.	Domoic acid	Scholin et al., 2000; Gulland, 2000; Silvagni et al., 2005
1997–1998	California sea lions *Zalophus californianus* (hundreds)	California, U.S.	El Niño	Greig et al., 2005
1999	Harbor porpoises *Phocoena phocoena* (216)	Maine, Massachusetts, Maryland, Virginia, North Carolina, U.S.	Oceanographic factors suggested [Hohn, personal communication]	Marine Mammal Commission, Annual Report to Congress
2000	Caspian seals *Phoca caspica* (10,000)	Caspian Sea	Canine distemper virus (CDV)	Kennedy et al., 2000
2000	Beaked whales	Bahamas	Anthropogenic sound	Evans and England, 2001
2000	Sea otters *Enhydra lutris* (100)	(Cordova) Alaska, U.S.	Parasites ingested at a fish processing plant with discarded waste	Minutes of UME Working Group meeting 2001
1999–2000	Bottlenose dolphins *Tursiops truncatus* (115)	Florida (Panhandle), U.S.	Brevetoxin	Mase et al., 2000
1999–2001	Gray whales *Eschrichtius robustus* (651)	California, Oregon, Washington, Alaska, U.S.; Canada; Mexico	Unknown. Starvation involved	Gulland et al., 2005; Moore et al., 2001
2000	California sea lions *Zalophus californianus* (178)	California, U.S.	Leptospirosis	Greig et al., 2005
2000	California sea lions *Zalophus californianus* (184)	California, U.S.	Domoic acid	Gulland et al., 2002
2000	Harbor seals *Phoca vitulina* (26)	California, U.S.	Unknown. Viral pneumonia suspected	Minutes of the UME Working Group meeting 2001, NMFS

Table 1. Continued

Year	Species and no. affected	Location	Cause and comments	References
2001	Bottlenose dolphins *Tursiops truncatus* (35)	Florida (Indian River Lagoon), U.S.	Unknown. Saxitoxin present in puffer fish	Barros, 2001 [unpublished]; Leighfield, 2002 [unpublished]
2001	Harp seals *Phoca groenlandica* (453)	Maine, Massachusetts, U.S.	Unknown	Harris et al., 2002
2001	Hawaiian monk seals *Monachus schauinslandi* (11)	Hawaii (Northwest Hawaiian Islands), U.S.	Malnutrition	Antonelis et al., 2001
2002	Harbor seals *Phoca vitulina* (~25,000)	Northern Europe; U.K.	Phocine distemper virus	Jensen et al., 2002
2002	Multispecies(Common dolphins *Delphinus delphis;* California sea lions *Zalophus californianus;* sea otters *Enhydra lutris;* ~500)	California, U.S.	Domoic acid	Mazet et al., 2005
2002	Hooker's sea lion *Phocarctos hookeri*	New Zealand	*Klebsiella pneumoniae*	Duignan et al., 2003
2002	Beaked whales (14)	Canary Islands, Spain	Gas embolism, sonar	Jepson et al., 2003; Fernández et al., 2004
2002	Florida manatees *Trichechus manatus latirostris* (34)	Florida (West coast), U.S.	Brevetoxin	Flewelling et al., 2005
2003	Multispecies(Common dolphins *Delphinus delphis;* California sea lions *Zalophus californianus;* sea otters *Enhydra lutris*; ~500)	California, U.S.	Domoic acid	Minutes of the UMWE Working Group annual meeting 2004
2003	Sea otters *Enhydra lutris* (69)	California, U.S.	Ecological factors	Draft report to UME Working Group, NMFS
2003	Beluga whales *Delphinapteus leucas* (20)	Cook Inlet, Alaska, U.S.	Increased detection, ecological factors	Vos and Shelden, 2005
2003	Large whales (16 humpback, 1 fin, 1 minke, 1 pilot, 2 unknown)	Maine, U.S.	Unknown. Saxitoxin and domoic acid detected in 2 and 3 humpbacks, respectively	Draft report to UME Working Group, NMFS
2003–2004	Harbor seals *Phoca vitulina;* Minke whales *Balaenoptera acutorostrata*	Gulf of Maine, U.S.; Exclusive Economic Zone (EEZ)	Unknown	Touhey [personal communication]
2003	Florida manatees *Trichechus manatus latirostris* (96)	Florida (West coast), U.S.	Brevetoxin	Minutes of the UME Working Group annual meeting 2004
2004	Bottlenose dolphins *Tursiops truncatus* (107)	Florida (Panhandle), U.S.	Brevetoxin	Draft report to UME Working Group; Gaydos [in preparation]; Flewelling et al., 2005
2004	Small cetaceans (67)	Virginia, U.S.	Unknown	Barco, report to Working Group
2004	Small cetaceans	North Carolina, U.S.	Unknown	Hohn [personal communication]
2004	California sea lions *Zalophus californianus* (405)	California, Oregon, Washington, U.S.; Canada	Leptospirosis	Raverty et al., 2005

Table 1. Continued

Year	Species and no. affected	Location	Cause and comments	References
2005	Manatees *Trichechus manatus*; Bottlenose dolphins *Tursiops truncatus* (ongoing Dec. 2005)	Florida (West coast), U.S.	Brevetoxin. Bird, turtle, and fish kills associated with the event	
2005	Harbor porpoises *Phocoena phocoena* (ongoing Dec. 2005)	North Carolina, U.S.	Unknown	Hohn [personal communication]
2005	California sea lions *Zalophus californianus*; Northern fur seals *Callorhinus ursinus* (several hundred)	California, U.S.	Domoic acid	Goldstein et al., 2005
2005	Mixed cetaceans	North Carolina, U.S.	Unknown	Hohn et al., 2006
2005	Large whales	Eastern North Atlantic, U.S.	Domoic acid suspected	Touhey [personal communication]
2005–2006	Bottlenose dolphins *Tursiops truncatus*	Florida, U.S.	Brevetoxin suspected	

[a]Data are obtained from the published literature and the National Marine Fisheries Service database.

Discussion and Conclusions

This survey of the peer reviewed literature on marine mammal diseases and reports of marine mammal mass mortality events suggests that there has indeed been an increase in the frequency of marine mammal die-offs resulting from exposure to harmful algal blooms over the past 40 years. The evidence for changes in frequency of other types of diseases is difficult to interpret, particularly in recent years for a number of reasons. These include changes in investigation effort, improved methodology for detection of disease, biases associated with sources of animals for detection of disease, and changes in the population density of some coastal marine mammal populations that are more easily studied.

Effort aimed at identifying disease has changed due to the underlying increasing global trend in the reporting of marine mammal disease in the literature and in the documenting of mass mortalities associated with a general increase in scientific research and in the number of researchers in this field. This latter increase was previously noted by Lavigne et al. (1999) and attributed to changing attitudes to marine mammals. For example, the membership of the Society for Marine Mammalogy, since its inception in 1981 to its most recent conference in 2005, has increased approximately threefold from ~780 to ~2300.

Over the last 40 years, many marine mammal populations have increased significantly, due to changes in hunting and harvesting pressure and possibly to changes in food availability where ecosystem regime changes have occurred. This not only will influence the availability of carcasses for detection, but can allow density-dependent diseases to increase in prevalence. If a certain proportion of a population dies due to environmental changes and further selective pressures result in malnutrition, large increases in marine mammal populations may result in clusters of malnourished animals dying, increasing the likelihood of their detection by humans. Mass mortality events associated with starvation are reviewed in Geraci et al. (1999). Density-dependent diseases will increase as marine mammal populations recover. Epidemiological models of PDV suggest the population of harbor seals in Europe is not large enough to sustain the disease as endemic, epidemics only occurring when the pool of susceptible seals is over a critical threshold (Grenfell et al., 1992) and the disease will fade out between outbreaks. The first epidemic of PDV in European harbor seals in 1988–1989 was unexpected and required considerable effort to identify its etiology (Kennedy et al., 1988). However, disease modeling exercises predicted that another epidemic was likely after approximately 10 years based on knowledge of morbillivirus epidemiology and harbor seal population dynamics (Grenfell et al., 1992). Another epidemic of PDV occurred in European harbor seals in 2002, and although many details of the epidemic are still unclear (Harkonen et al., 2006), these recurring epidemics at approximately a decade interval do not suggest a change in harbor seal health over this period, but reflect changes in population immunity. A much longer dataset (i.e., 100 years) would be needed to detect changes in frequency of morbillivirus epidemics in seals at this inter-epidemic interval. Nothing is known about the diseases that may have been introduced into many marine mammal populations prior to 1960, but which did not establish themselves because populations were historically too small for the disease to propagate between individuals.

The change in methodology used to study marine mammal health and disease can also influence the frequency of novel disease detection. The change from studying harvested and by-caught animals to studying stranded and rehabilitated animals will result in a higher detection of diseased animals. An increase in the numbers of individuals stranding in a location can indicate an increase in mortality rate (Heide-Jorgensen et al., 1992; Harkonen et al., 2006), although stranding rates and numbers often do not correlate directly with mortality rates due to a variety of factors influencing carcass deposition and detection (Eguchi, 2002; Thompson et al., 2005). Documentation of stranding events has improved globally over the last 40 years, with the earliest organized attempts originating in the United Kingdom (U.K.) (Fraser,1934, 1946, 1953, 1956; Geraci, 1978; Sergeant, 1982; Dierauf and Gulland, 2001).

Stranded animals are a useful source of information on diseases occurring in marine mammals, as they are more readily available to pathologists than free-ranging animals. A number of emerging disease entities, suites of lesions and infectious agents in marine mammals were first identified in stranded animals, after which their presence in the free-ranging population was confirmed. Pathogens identified through stranding programs include phocine distemper virus (PDV) that caused the death of over 18,000 harbor *(Phoca vitulina)* seals in Europe in 1988 and over 20,000 in 2002 (Osterhaus et al., 1988; Kennedy et al., 1988; Jensen et al., 2002; Harkonen et al., 2006), phocine herpesvirus (PhHV1) isolated from stranded harbor seals in 1985 (Osterhaus et al., 1985), and *Brucella marinus* in a variety of species (Ross et al., 1996; Garner et al., 1997). Stranded animals can also alert us to diseases that are present in the more inaccessible wild animals that would be difficult to detect in random samplings of such populations. For example, 17% of sexually mature California sea lions *(Zalophus californianus)* that stranded and died along the northern coast of California showed neoplasia when examined postmortem (Gulland et al., 1996b). In comparison, only one case of neoplasia has been observed in California sea lions at rookeries on San Miguel Island (CA) where there are more than 100,000 sea lions [Spraker, personal communication]. The study of neoplasia pathogenesis is thus more readily performed on stranded sea lions than on those in rookeries, and stranded animals essentially serve as sentinels for their wild con-specifics. Animals in rehabilitation also provide a highly significant sampling method for the study of marine mammal disease. Indeed, the discovery that domoic acid produced by harmful algal blooms of *Pseudo-nitzschia australis* was causing mortality and seizures in California sea lions was entirely as a result of a comprehensive rescue and rehabilitation program for the affected region (Scholin et al., 2000).

Stranded animals, however, do not constitute an ideal system for the study of disease, as they do not represent the entire population (Aguilar and Borrell, 1994). In addition, samples of stranded animals are rarely age and sex structured, and biological data such as individual life histories, feeding habits, reproductive success, or disease progression are not typically available. Examination of both live and dead strandings are useful for detecting disease but are limited when the objective is to assess the prevalence of a disease and its impact on a host population, since the sample will be skewed towards those likely to come ashore. Decomposition is also a problem for obtaining an accurate diagnosis and good histology among dead strandings, while human concern and intervention can interfere with research on live stranded animals.

The examination of stranded cetaceans has also allowed the detection of a novel suite of lesions, termed "gas bubble disease" (Jepson et al., 2003). Animals with these lesions stranded in association with navy exercises, but whether the frequency of mortality of cetaceans from these lesions is changing over time in association with increasing levels of ocean noise cannot be determined due to the lack of a systematic time series on pathology of stranded cetaceans worldwide. The few case studies on this syndrome are a result of the fortuitous proximity of skilled veterinary pathologists, fresh stranded cetaceans, and publicly accessible information on naval activities. To determine the extent of this problem and whether its frequency is changing, improved facilities and logistic support for examining dead marine mammals thoroughly are needed along coastlines throughout the world.

It is possible that prior to the recent interest in ocean health, some marine mammal disease outbreaks may have been missed. However, high numbers of dead animals are difficult to ignore—there have been reports of seals suffering "epizooty" in the literature from the 1700s (see Harwood and Hall, 1990)—so it is unlikely that epidemics would have been missed prior to the first of the recent events in the 1980s. Events are more likely to be documented in the peer-reviewed literature when the causes were identified and can be acknowledged retrospectively, than events with no identified causes. Thus, there may have been a number of marine mammal mortality events of unknown etiology between 1978 and 1992 that are not recorded in the published literature reviewed here. As technology advances, so have methods to detect toxins and pathogens from smaller samples of less well-preserved tissues. This has resulted in the changes in frequency that etiological agents have been described in the published literature (Fig. 2). There is also variability worldwide in the availability of more advanced laboratory techniques for examining marine mammal tissues, as well as a lack of diagnostic tests validated for marine mammals. To prevent this bias from continuing to impair our ability to detect changes in marine mammal health, dedicated laboratories for marine mammals are needed.

To determine whether marine mammal health is indeed changing, a concerted effort is needed worldwide to coordinate investigations into marine mammal die-offs, to investigate associations between disease and ecological variables, to share data on methodology used for investigations and laboratory studies, and to document the stranding response effort. Over time, we will be able to use geographic information systems (GIS) and spatial epidemiological studies to investigate temporal trends in marine mammal disease. In 1988, the first dramatic PDV epidemic that killed over 18,000 harbor seals in Europe raised awareness of the need for contingency plans to investigate marine mammal die-offs, and for long-term monitoring of strandings (Heide-Jorgensen et al., 1992; Thompson and Hall, 1993). In 1989, the Department of the Environment (now the Department for the Environment, Food and Rural Affairs) in the U.K. established a national program to investigate marine mammal mortalities, and to coordinate responses. In the U.S., three specific events triggered the development of a legal framework that addressed marine mammal unusual mortality events. The first was a stranding of 14 humpback whales *(Megaptera novaeangliae)* off Cape Cod, Massachusetts, in 1987 (Geraci et al., 1989). The second was a bottlenose dolphin die-off along the Atlantic seaboard between 1987 and 1988 (Geraci, 1989), and the third the Exxon Valdez oil spill in Prince William Sound, Alaska, in 1989 (Loughlin, 1994). As a response to the increased concerns over these die-offs and the potential for environmental degradation, the Marine Mammal Health and Stranding Response Act was established, and became Title IV of the Marine Mammal Protection Act (MMPA) with a provision for responses to marine mammal unusual mortality events. There are four aims of these responses: (1) to minimize death and suffering, (2) to determine the cause of the event, (3) to determine the effect of the event on the population, and (4) to identify the role of environmental variables in the event; only the first two of these have been significantly addressed since the UME program was established in 1992. Efforts to minimize death and suffering have improved as an increasing number of stranding network facilities employ veterinarians to advise on medical care of stranded marine mammals, and the field of marine mammal medicine has developed considerably over the last 10 years. Identification of the cause of mortality events has been achieved in just over half (56%) of the investigations, some of these resulting in the detection of novel disease syndromes in marine mammals (Geraci et al., 1989; Lipscomb et al., 1994; Bossart et al., 1998; Scholin et al., 2000).

Identification of novel diseases requires significant resources and collaboration, thus achieving this through investigations of mortality of wild mammals is a considerable accomplishment. However, trends in the frequency of these diseases, the impact of environmental change on these diseases, and the effects of these mortality events on host populations cannot be determined from the data currently available. The available data are adequate to identify an increase in frequency of marine mammal die-offs in the United States due to poisoning by harmful algal blooms, but other changes in marine mammal health worldwide cannot be determined in this study. Without this additional information, the status of marine mammal health and the impacts of anthropogenic activities and environmental factors, such as climate change, cannot be assessed. To improve our understanding of marine mammal health and determine the impacts of human activities on these animals, improved programs for disease surveillance, emergency response, laboratory diagnosis, and research into contributing factors are needed, with international collaborations to address marine mammal health in ocean ecosystems without national boundaries.

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