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Causes of Live Strandings of Northern Elephant Seals (*Mirounga angustirostris*) and Pacific Harbor Seals (*Phoca vitulina*) Along the Central California Coast, 1992-2001

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Abstract

Between January 1992 and December 2001 a total of 1,277 northern elephant seals (*Mirounga angustirostris*) and 940 Pacific harbor seals (*Phoca vitulina*) were stranded live along the central California coast. By examining medical records for each of the stranded seals, the primary cause of stranding and the secondary factors contributing to stranding were determined. The majority (88.3%) of animals were young born that year. The most common causes of live strandings for elephant seals included malnutrition (56.6%), *Otostrongylus circumlitus* infection (12.1%), and northern elephant seal skin disease (9.8%). The most common causes of strandings for harbor seals were malnutrition (51.8%), respiratory disease (9.6%), and trauma (8.0%). Common secondary factors for strandings for both elephant and harbor seals included human interaction, trauma, and ocular disease. For both species of phocid, the highest prevalence of *O. circumlitus* infection occurred in 1992 and 1993, years of a moderate El Niño-Southern Oscillation event. Phocine herpesvirus-1 infection was a common primary and secondary factor in harbor seal strandings, and the highest prevalence occurred between 1994 and 1998. Human interference in stranding events increased in the past ten years and is most commonly reported on beaches near heavily populated areas.

Key Words: northern elephant seal, *Mirounga angustirostris*, Pacific harbor seal, *Phoca vitulina*, stranding, malnutrition, *Otostrongylus circumlitus*, phocine herpesvirus-1, El Niño-Southern Oscillation, ENSO, California

Introduction

Much of the current knowledge on marine mammal diseases comes from investigations of stranded animals, as these animals provide an opportunity to study diseases in relatively inaccessible wild populations (Gulland, 1999). Examination of these animals historically led to the identification of new diseases that affect wild populations. The identification of phocine distemper virus in seals, morbillivirus in cetaceans, and, most recently, domoic acid toxicity in California sea lions (*Zalophus californianus*) all stemmed from examinations of stranded animals (Kennedy, 1990; Lipscomb et al., 1994; Scholin et al., 2000). Live stranded marine mammals are also an important source of information on the health of the marine environment (Reddy et al., 2001). Contaminants can readily be sampled from stranded animals, and diseases that may be associated with ocean pollution can be further assessed (Rowles et al., 2001). The high prevalence of neoplasia in stranded beluga whales (*Delphinapterus leucas*) of the St. Lawrence River led to theories on the relationship between disease and contaminant levels (De Guise et al., 1994; Martineau et al., 2003).

Although stranded animals represent an inherently skewed sample of the free-living population and the prevalence of diseases in live stranded animals is not the same as in the entire population, assessing temporal trends in causes of strandings and disease can aid in the evaluation of risk factors for disease in wild populations. Quantifying the causes of pinniped strandings also provides information to rehabilitation facilities that can be used to plan appropriate veterinary care. Tracking human interactions in stranding events over time can aid in assessing the impact of human encroachment on the habitat of pinnipeds.

Gerber et al. (1993) reported on diseases of pinnipeds that live stranded along the California coast between 1984-1990. Since this study, clinical diagnostic methods have improved and knowledge of diseases that affect phocids has increased tremendously (Dierauf & Gulland, 2001). In the past ten years, two El Niño-Southern Oscillation (ENSO) events have occurred, a mild event from 1992-1994 and a severe event that lasted

from the fall of 1997 through the spring of 1998 (McPhaden, 1999; Trenberth & Hoar, 1996). Although much has been written about the biological consequences of ENSO on pinniped populations (Trillmich & Ono, 1991), there is little information on the effects of ENSO events on disease prevalence. This retrospective study was undertaken to identify the causes of live stranding in two common phocid species—northern elephant seals (*Mirounga angustirostris*) and Pacific harbor seals (*Phoca vitulina*)—along the central California coast over a ten-year period spanning two ENSO events.

Materials and Methods

Medical records from The Marine Mammal Center (TMMC) in Sausalito, California, a rehabilitation center, were reviewed for all northern elephant seals and Pacific harbor seals that stranded live between 1 January 1992 and 31 December 2001 along the central California coast (37° 42' N, 123° 05' W to 35° 59' N, 121° 30' W). Seals were considered stranded based on the criteria of Gerber et al. (1993). At admission, age was determined for each animal using date, weight, pelage, stage of tooth development, and umbilical regression (Dierauf et al., 1986). Sex was determined through examination of genital morphology. Routine diagnostic procedures included physical examination, lung auscultation, complete blood count, serum chemistry profile, and fecal flotation (Bossart et al., 2001). Other diagnostics such as radiography, ultrasound, computed tomography (CT), magnetic resonance imaging (MRI), bacterial and fungal cultures, and tissue biopsies were performed as needed. A gross necropsy was performed on all animals that died during rehabilitation or that were dead on arrival (DOA) to the hospital (Rowles et al., 2001). Routine tissue samples were collected during gross necropsy from a sample of animals and fixed in 10% neutral buffered formalin. Tissues were submitted to the Anatomic Pathology Service, Veterinary Medical Teaching Hospital, University of California, Davis, California (USA); the Armed Forces Institute of Pathology, Bethesda, Maryland (USA); or to Colorado State University, Fort Collins, Colorado (USA) for histopathologic evaluation.

Pertinent information collected from the medical records included date and location of stranding; age class; sex; observations at stranding; final disposition; cause of death; diseases diagnosed; and laboratory, necropsy, and histopathology results. Observations at stranding included weight, activity level, presence of a mother seal, signs of illness or wounds, presence of large numbers of people near the animal, and harassment of the seal by people and animals. The primary cause of a stranding was defined as the disease or condition that was the most likely cause of the animal's stranding based on all information recorded. Up to three secondary factors also were determined for each animal. Secondary factors were defined as conditions that likely contributed to the animal's stranding. Some animals developed diseases during rehabilitation that were unrelated to the initial cause of stranding. Diseases diagnosed clinically or via necropsy after the first week of hospitalization were only tabulated as a stranding cause if they could have been an initial factor in the animal's stranding.

Clinical signs that were considered indicative of *Otostrongylus circumlitus* infection in elephant seals included anorexia, harsh lung sounds, depression, neutrophilia, and disseminated intravascular coagulation (Gulland et al., 1997b). *O. circumlitus* infection was considered a secondary cause of stranding if an animal died from a separate condition, however, infestation by the nematodes was found incidentally at necropsy. Elephant seals and harbor seals that had clinical signs of respiratory infection, yet the cause of the respiratory disease could not be determined, were characterized as having "respiratory disease" as the primary cause of stranding.

Injuries such as bite wounds, abscesses, lacerations, and musculoskeletal injuries were grouped under the category of "trauma." Injuries that occurred due to fisheries interactions, such as net entanglement or fishhook ingestion, were grouped under the "fisheries" category. Animals that stranded with evidence of ingestion of foreign material, or with injuries from human-made debris other than fisheries-related debris, were categorized under "debris entanglement/ingestion." Animals that stranded with tarring of the fur coat were categorized as "oiled fur." "Human interaction" was defined as any type of unauthorized public involvement in the stranding and included illegal pickup of animals, harassment by people or dogs, animals hauling out on crowded beaches, interactions with animals prior to rescue, and direct physical injury. Seals that stranded in highly populated or inappropriate areas that were subsequently relocated to more appropriate haulout beaches were also included in this category. Animals with gunshot injuries were grouped in a separate category. An animal with evidence of trauma from a boat strike was classified as "trauma" for the primary stranding factor, with a secondary factor of "human interaction."

Statistical analysis was completed using *Microsoft Excel 2000* and *SPSS Version 9.0*. Associations among stranding causes, number of strandings, and year were examined using a chi-square goodness-of-fit test. Associations between

strandings by sex and by year also were examined using a chi-square goodness-of-fit test. Linear regression was performed to examine changes in frequency of human interactions over time.

Results

Elephant Seals

Of the 1,277 stranded northern elephant seals examined during the ten-year study period, 706 were male and 560 were female. The gender of 11 animals was not determined. There was no significant difference in the number of male or female seals stranded in any year during the study period. One-hundred fifty-nine were pre-weaned pups, 901 were molted weanlings, 206 were yearlings, six were subadults, and only five were adults. A total of 420 (32.8%) of the 1,277 stranded seals died or were euthanized. Histopathology was performed on 133 of the 420 deceased animals. The highest number of strandings occurred in 1998 (195), 1993 (174), and 1992 (172)—all years of ENSO events.

The major primary causes of stranding for elephant seals were malnutrition (56.6%), *Otostrongylus circumlitus* infection (12.1%), and northern elephant seal skin disease (9.8%). The most common secondary causes were trauma, human interaction, and ocular lesions (Table 1).

Northern elephant seal skin disease was a primary stranding factor in 125 animals and a secondary stranding factor in 15 animals. Prevalence of skin disease varied by year (Figure 1) and was significantly higher in 1992 ($X^2 = 15.9$, DF = 1, $p < 0.01$) and 1994 ($X^2 = 21.9$, DF = 1, $p < 0.01$). The

Table 1. Primary and secondary causes of stranding for northern elephant seals along the central California coast from 1992-2001

Primary causes of stranding	Total number of stranded animals
Malnutrition	724
Otostrongylus circumlitus infection	155
Northern elephant seal skin disease	125
Respiratory disease	104
Trauma	43
Human interaction	21
Septicemia	18
Ocular disease	18
Unknown	18
Congenital disease	14
Gastrointestinal disease	12
Miscellaneous	7
Debris entanglement/ingestion	5
Neurologic disease	5
Gunshot	4
Fisheries interactions	4
Secondary causes of stranding	
Human interaction	117
Trauma	116
Ocular disease	57
Oiled fur	25
O. circumlitus infection	17
Northern elephant seal skin disease	15
Respiratory disease	11
Miscellaneous	8
Gastric impaction	6

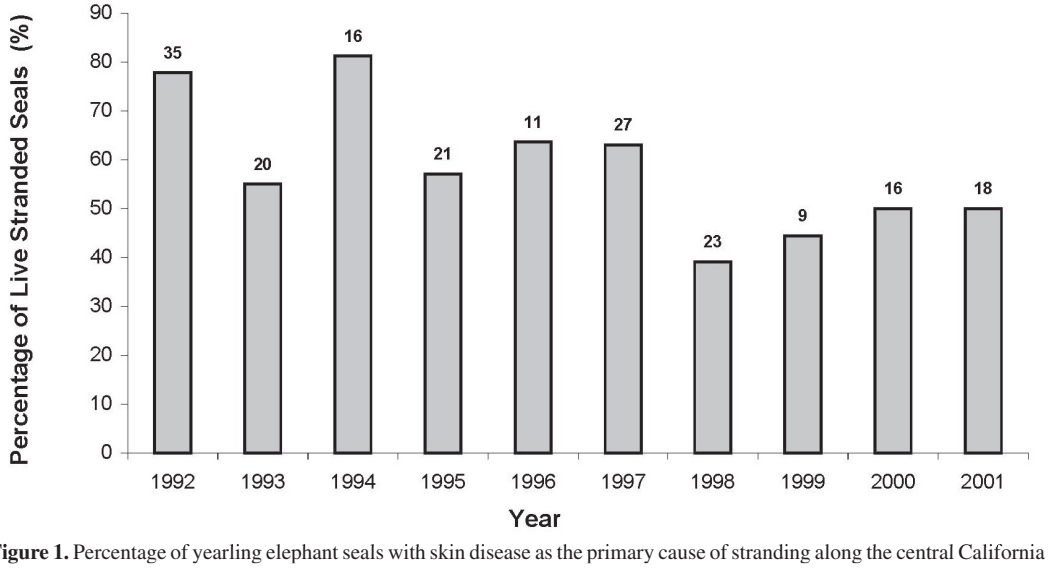

Figure 1. Percentage of yearling elephant seals with skin disease as the primary cause of stranding along the central California coast; the numbers over the bars indicate the total number of yearling seals stranded that year.

disease was characterized by alopecia, hyperpigmentation, and epidermal ulceration and necrosis, as reported by Beckmen et al. (1997).

O. circumlitus infection, with arteritis, pneumonia, and disseminated intravascular coagulation, as described by Gulland et al. (1997b), was a primary stranding factor for 155 elephant seals and a secondary stranding factor in 17 elephant seals. Prevalence of *O. circumlitus* infection (Figure 2) was significantly higher in 1992 ($X^2 = 7.29$, DF = 1, $p < 0.01$) and 1993 ($X^2 = 7.65$, DF = 1, $p < 0.01$). Other separate conditions often were diagnosed in conjunction with nematode infection. These conditions included, in order of decreasing frequency, septicemia, northern elephant seal skin disease, enterocolitis, hepatitis, endocarditis, endometritis, epicarditis, meningitis, gastritis, and aspergillosis.

Six elephant seals had histologic evidence of leptospirosis in 1995. Leptospirosis was determined to be the primary cause of stranding in two seals. In the remaining four seals, leptospirosis developed during rehabilitation and, therefore, was not considered a cause of stranding.

Ocular disease was the primary cause of stranding for 18 elephant seals (1.4%) and was a secondary factor for 57 animals. Ocular disorders included keratitis, corneal ulcers, conjunctivitis, lens luxation, cataracts, hyphema, and prolapse of the third eyelid. Fourteen animals had congenital defects, including hydrocephalus (5/14), inguinal hernia (2/14), diaphragmatic hernia (2/14), shoulder joint malformation (1/14), or overriding aorta with pulmonary artery aplasia (1/14). One animal had both a ventricular septal defect and hydrocephalus, while another animal had a ventricular septal defect and a persistent ductus arteriosus. One elephant seal had pulmonary artery stenosis and a persistent ductus arteriosus. Full descriptions

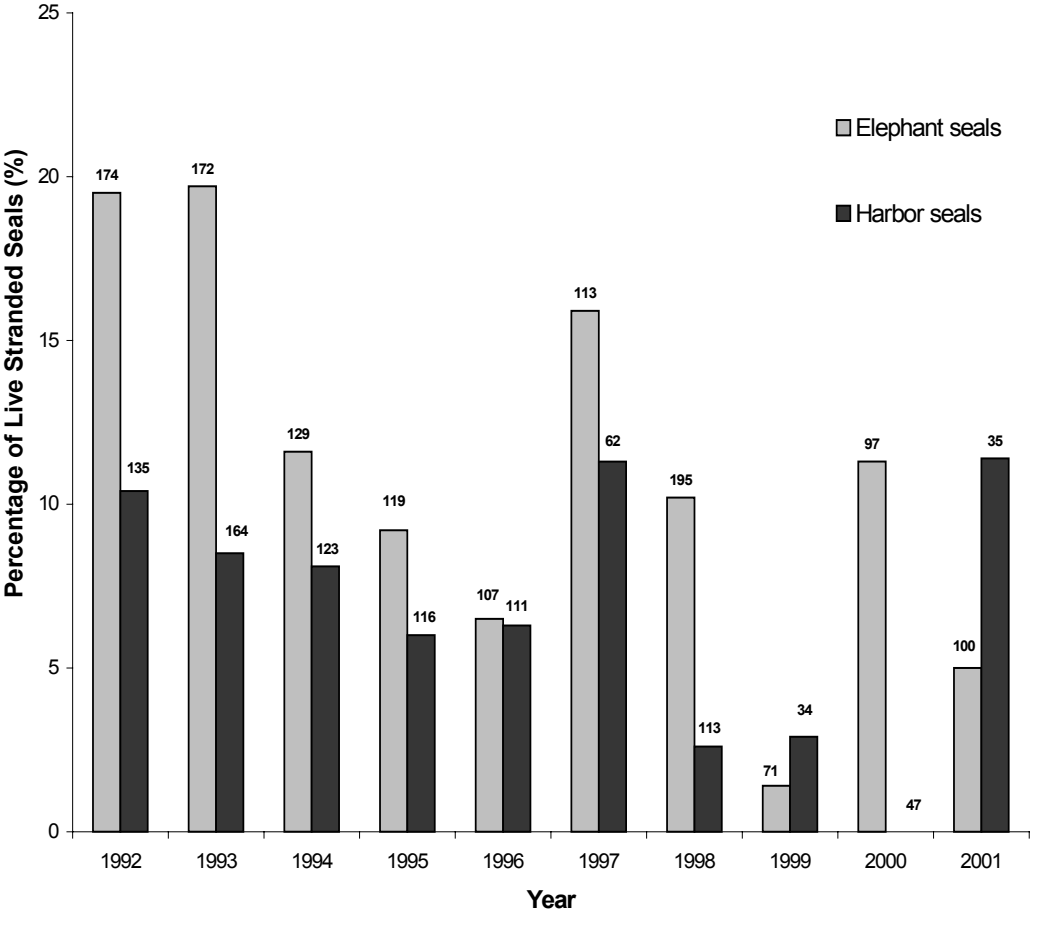

Figure 2. The percentage of elephant and harbor seals with *Otostrongylus circumlitis* infection and related disease as the primary cause of stranding along the central California coast; numbers over the bars equal the total number of seals stranded that year.

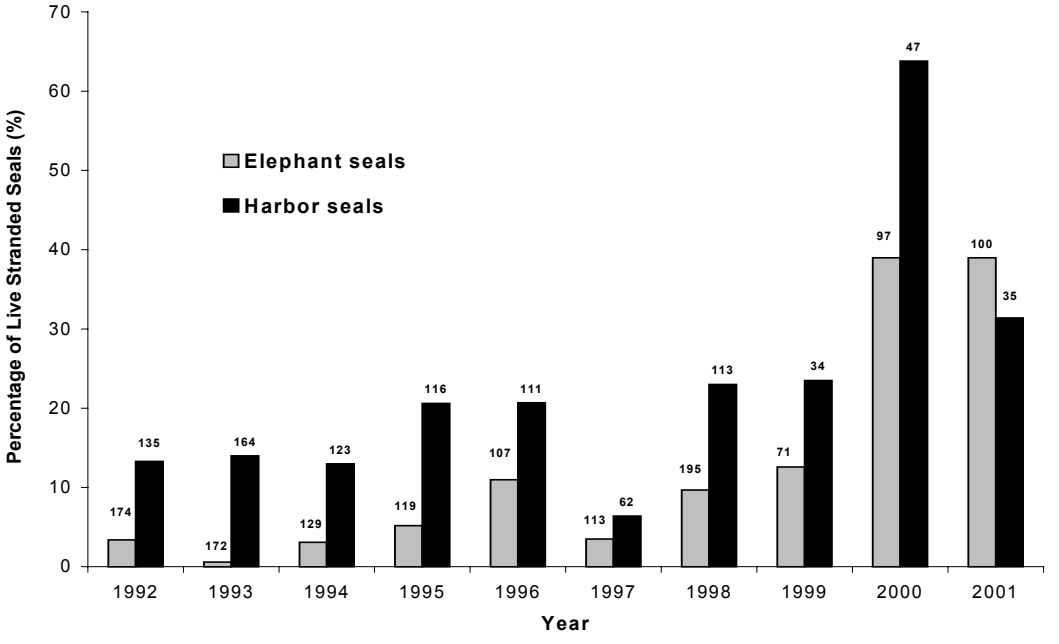

Figure 3. The percentage of stranded seals with human interaction as a primary or secondary stranding factor along the central California coast; numbers over the bars equal the total number of seals stranded that year.

of these congenital abnormalities have been previously reported (Trupkiewicz et al., 1997).

Twelve animals stranded due to gastrointestinal disorders, which included gastritis, enteritis, and colitis. Neurologic disease (5 animals) included both meningitis and encephalitis. Conditions tabulated under the category of "miscellaneous" for primary causes of stranding (n = 7) included *Leptospira* sp. infection (2), ruptured bladder (1), ruptured stomach (1), nephritis (1), biliary adenocarcinoma (1) (Fauquier et al., 2003), and fungal dermatitis (1). Miscellaneous conditions (n = 8) considered secondary stranding factors included vaginitis (3), colitis (2), congenital muscular defect (1), gun shot (1), and pox virus infection (1).

Human interaction was the primary cause of stranding in 21 elephant seals and was the most common secondary cause of stranding (117 seals). The prevalence of human interactions in stranded events (Figure 3) increased with time ($R^2 = 0.62$, DF = 9, $p < 0.01$). Human interactions were more common on beaches in highly populated areas (Figure 4).

Harbor Seals

A total of 940 harbor seals stranded during the ten-year study period; 456 were males and 479 were females. The sex of five animals was not determined. Significantly more female seals stranded than males in 2000 ($X^2 = 10.6$, DF = 1,

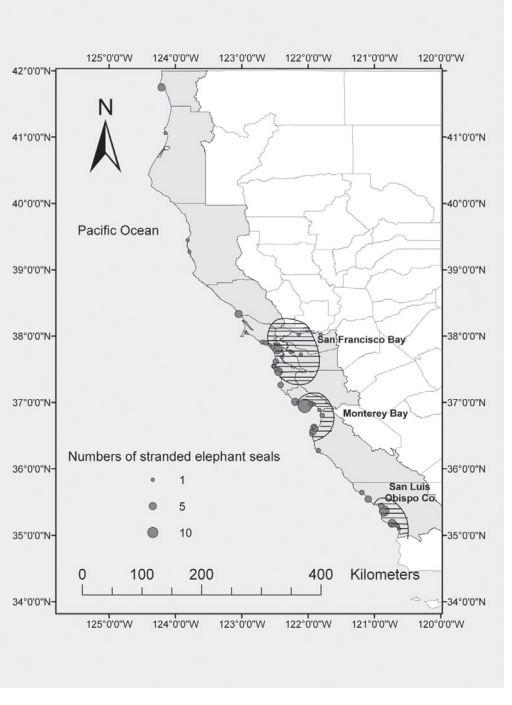

Figure 4. Map of the central California coast illustrating the incidence of human interactions in elephant seal stranding by location

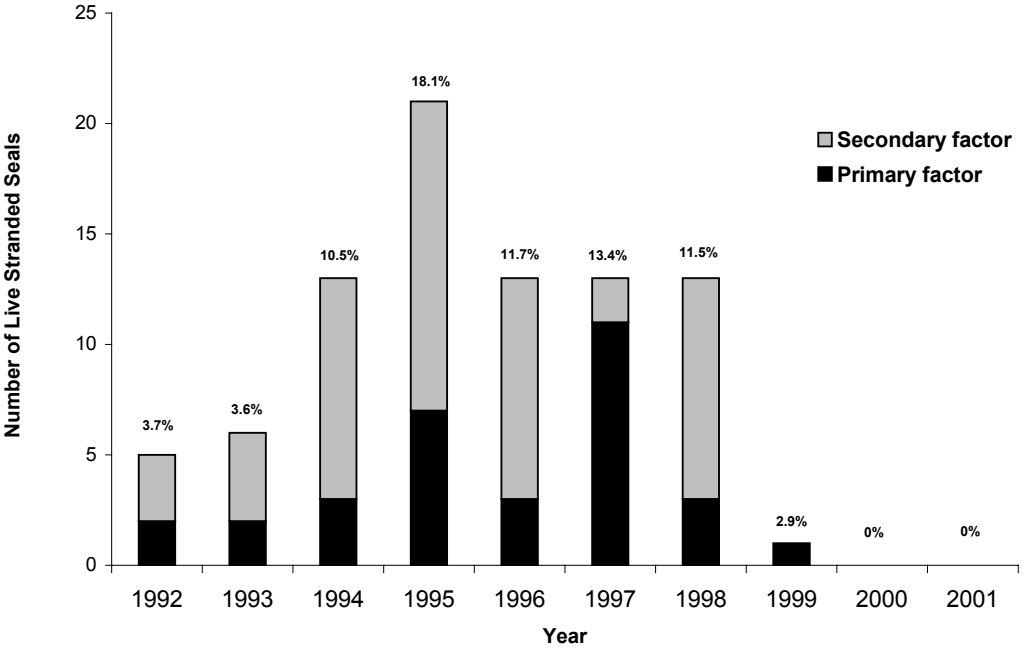

Figure 5. The number of harbor seals with herpesvirus infection as either a primary or secondary stranding factor along the central California coast; numbers over bars indicate the percentage of seals with herpesvirus as a stranding factor per year.

$p < 0.01$), but not in other years. Of the total animals stranded, 737 were pre-weaned pups, 161 were weanlings, 11 were yearlings, six were subadults, and 25 were adults. Five-hundred ninety-nine seals died or were euthanized, and histopathology was performed on 286 of these animals. The highest number of strandings occurred in 1992 (135) and 1993 (164).

The most common causes of stranding for harbor seals were malnutrition (51.8%), respiratory disease (9.6%), and trauma (8.1%). Human interference, trauma, and phocine herpesvirus–1 (PhHV-1) infection were frequent secondary causes of stranding (Table 2).

Herpesvirus infections were only observed in pups and weanlings. Between 1994 and 1998, there was an increase in the number of strandings in which PhHV-1 infection was either a primary or a secondary cause of stranding (Figure 5) and the prevalence of herpesvirus infection as a stranding factor was significantly higher in 1995 ($X^2 = 15.9$, DF = 1, $p < 0.01$). After 1998, the prevalence of herpesvirus infection decreased significantly. Other diseases often were diagnosed in conjunction with herpesvirus infection via histopathology. These diseases included, in order of decreasing frequency, omphalophlebitis, meningoencephalitis, septicemia, pneumonia, peritonitis, gastroenteritis, hepatitis, *O. circumlitus* related disease, pleuritis, cystitis, pancreatitis, and endocarditis.

O. circumlitus associated bronchopneumonia, as described by Gulland et al. (1997), was a primary stranding factor in 67 seals and a secondary factor in 13 seals. The highest prevalences of *O. circumlitis* associated disease occurred in 1992, 1993, and 1997; however, the annual difference was not significant (Figure 3).

Ocular diseases were a primary cause of stranding in six animals and a secondary factor in 34 animals. The types of ocular disorders diagnosed were similar to those observed in elephant seals. Twelve animals were diagnosed with congenital defects that were determined to be the primary cause of stranding. These included diaphragmatic hernias (11/12) (five of which were hiatal hernias) and a conjoined fetal twin (1/12).

Gastrointestinal disorders in stranded harbor seals included enteritis, colitis, and gastric rupture. Neurologic disease included meningitis, encephalitis, and vestibular disease. Protozoa associated meningoencephalitis was considered in a separate category and included eight strandings due to *Sarcocystis* sp. infection and one stranding due to an unclassified protozoal organism (Lapointe et al., 2003). Miscellaneous conditions ($n = 8$) included lymphosarcoma (1), pregnancy toxemia (1), *Leptospira* sp. infection (1), nephritis (1), intestinal volvulus (1), gunshot (1), hepatitis (1), and abortion (1). Miscellaneous secondary stranding factors ($n = 8$) included fisheries

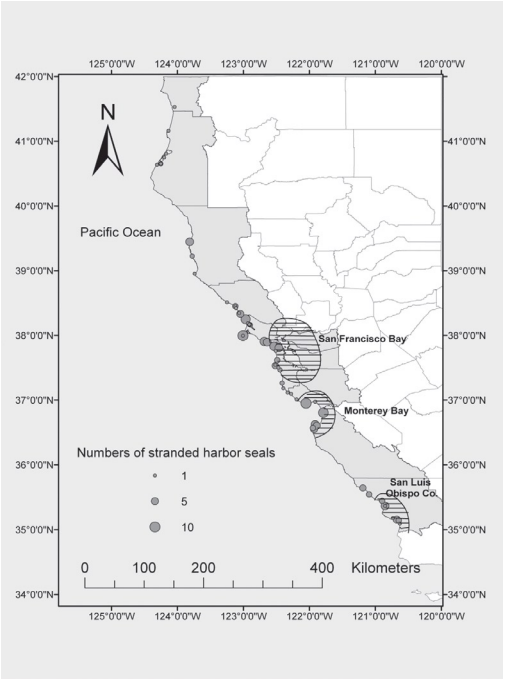

Figure 6. Map of the central California coast illustrating the incidence of human interaction in harbor seal stranding by location

interactions (3), otitis (3), gunshot (1), and gastroenteritis (1).

Human interaction was the most common secondary stranding factor (164 animals), and 19 harbor seals had human interaction as a primary cause of stranding. Although there were a higher number of interactions in some of the later years of the study period there was not a significant increase over time ($R^2 = 0.36$, DF = 9, $p = 0.065$). Human interactions were more common on beaches near known harbor seal rookeries and in more highly populated areas (Figure 6).

Table 2. Primary and secondary stranding causes for Pacific harbor seals along the central California coast from 1992-2001

Primary causes of stranding	Total number of stranded animals
Malnutrition	487
Respiratory disease	90
Trauma	76
Otostrongylus circumlitus related disease	67
Unknown	38
Omphalophlebitis	33
PhHV-1 infection	32
Septicemia	20
Human interference	19
Gastrointestinal disease	14
Neurologic disease	13
Congenital disease	12
Fisheries interaction	10
Protozoal infection	9
Miscellaneous	8
Ocular disease	6
Peritonitis	4
Debris entanglement/ingestion	2
Secondary causes of stranding	
Human interference	164
Trauma	117
PhHV-1 infection	53
Omphalophlebitis	48
Ocular disease	34
Respiratory disease	20
Oiled fur	19
O. circumlitus related disease	13
Gastric impaction	10
Miscellaneous	8

Discussion

The most common primary cause of live strandings in both northern elephant seals and Pacific harbor seals was malnutrition of young seals under one year old. Most malnourished elephant seals were weaned, whereas harbor seals were pre-weaned. Much of this age bias is likely due to the life history of the species. Northern elephant seals give birth on legally protected rookeries between January and March and wean at four weeks of age, after which they leave the rookeries to forage. At this time, they are vulnerable to malnutrition and human interactions (Le Boeuf et al., 1994). Harbor seals in California give birth from mid-March to mid-May, and pups are weaned at four weeks of age (Reeves et al., 1992). Occasionally, they give birth on beaches frequented by humans where unknowing citizens sometimes "rescue" apparently abandoned seals when in fact the mother seal was foraging nearby (Allen et al.,1984). Thus, some of the young harbor seal pups brought into rehabilitation were healthy newborn seals. Both elephant seals and harbor seals give birth during a time of year when periodic winter storms hit the California coast. Some young animals are separated from their mothers during these storms, which tend to be more common in El Niño years. Strandings and malnourishment of young seals are, therefore, more common in El Niño years. Malnutrition was also the most common disease finding in both elephant seals and harbor seals in the previous decade (Gerber et al., 1993).

Otostrongylus circumlitus nematodes are transmitted to seals through ingestion of fish intermediate hosts, and death from the infection can occur as long as one month post infection (Bergeron et al., 1997). Because death due to *O. circumlitus* in elephant seals often occurs during the prepatent period, antemortem diagnosis of infection sometimes was difficult and the number of elephant seals categorized as having *O. circumlitus* infection as the primary cause of stranding was likely underestimated. *O. circumlitus* infection was the second and third most common stranding cause in elephant seals and harbor seals, respectively, but the role of *O. circumlitus* infection in the population dynamics of elephant seals has yet to be determined. The severe reaction to immature parasite stages in young elephant seals has been postulated to be due to a recent host-parasite association and may be associated with the low genetic diversity found in elephant seal populations (Gulland et al., 1997b). The cause for the higher prevalence of infections in both species in 1992 and 1993 is not known. An ENSO event occurred during these years and may have increased susceptibility to infection, as poor nutritional conditions resulting from altered food sources can reduce host immunity (Gershwin et al., 2000). Alternatively, infective parasite load of the prey may have increased during these periods or phocid prey shifts may have occurred secondary to altered oceanic conditions brought on by ENSO. No increase in prevalence of *O. circumlitus* was observed during the severe ENSO of 1998, however. In the decade prior to this study (1984 to 1990), 0.9% of the stranded elephant seal pups and 0.5% of stranded older seals were diagnosed with verminous pneumonia, and pneumonia of unknown etiology was diagnosed in 2.8% of the stranded elephant seals (Gerber et al., 1993). In our study, 12.1% of the stranded elephant seals had *O. circumlitus* infection as the primary cause of stranding, and 8.1% of them had respiratory disease as the primary cause of stranding, suggesting prevalence of this infection has increased over time. In contrast, the prevalence of *O. circumlitus* related disease in harbor seals was similar between the two decades. The difference in prevalence of *O. circumlitus* morbidity in elephant seals between the two studies may reflect improved diagnostic capability or may indicate a true increase in prevalence of this nematode infestation in the California northern elephant seal population.

A large number of elephant seals stranded due to northern elephant seal skin disease in both 1992 and 1994, but the prevalence in this study was lower than the 36.5% reported between 1984 and 1990 (Gerber et al., 1993). The reasons for these annual changes in prevalence of skin disease cases are unclear, as is the etiology of the disease. A detailed analysis of the affected animals that stranded in 1992 has been reported previously, and hypothyroidism and polychlorinated biphenyl (PCB) toxicosis were postulated as potential etiologies (Beckmen et al., 1997).

The pathophysiology and epidemiology of PhHV-1 infections in harbor seals are complex. Although infection can be associated with morbidity and mortality in young harbor seals (Gulland et al., 1997a), PhHV-1 is endemic in North American harbor seal populations and there is an increased prevalence of exposure with age (Goldstein et al., 2003). Natural infection occurs in young seals due to lateral and vertical transmission (Goldstein, 2003). Herpesviruses can cause immunosuppression and render animals more susceptible to bacterial infections. Alternatively, primary bacterial infections can cause latent herpesvirus infections to recrudescence (Roizman et al., 1992). Consequently, PhHV-1 was only listed as a primary cause of stranding if herpesvirus associated lesions were present in animals that died within one week of stranding. If an animal died after one week of rehabilitation with herpesvirus-related lesions, PhHV-1 was listed as a secondary factor.

PhHV-1 was a primary cause of stranding in 32 of the 940 stranded harbor seals and a secondary stranding factor in 53 animals. In all years, the cases tended to be temporally clustered, with increased numbers of cases occurring after the first case of the year. This pattern also has been reported in other studies (Gulland et al., 1997a) and was shown to be partially due to lateral transmission during rehabilitation (Goldstein, 2003). The number of harbor seals with PhHV-1 infection as a primary or secondary stranding factor was the highest between 1994 and 1998. This may have been due to a common environmental stressor occurring in these years that activated latent infection in the seals, or to increased lateral transmission during rehabilitation. The dramatic decrease in herpesvirus-related strandings after 1998 is striking. Major renovations of facilities at The Marine Mammal Center were completed by 1999, which allowed for decreased handling of animals, and changes were made to the diet of harbor seals (Gulland, pers. comm.). These changes likely improved rehabilitation conditions, decreased stress, and reduced both the lateral transmission of virus and the prevalence of bacterial infections that could predispose phocids to recrudescence of viral infection.

Neoplastic disorders were only documented in one elephant seal and one harbor seal, both of which were adult animals. In comparison,

sympatric California sea lions (*Zalophus californianus*) have a high prevalence of urogenital carcinomas (Gulland et al., 1996) that accounted for 3% of strandings during the same ten-year period (Greig & Gulland, personal communication).

The increase in human interactions over the ten-year study period is likely due to several factors. As the number of people inhabiting coastal areas increases, the possibility of interaction between people and stranded animals also increases. This is supported by the majority of human interactions occurring in highly populated coastal areas. Public awareness of stranded marine mammals also has increased in recent years. During the study period, documentation of human interactions in the medical records became more diligent in the later years; therefore, some of the earlier incidences of interactions may not have been recorded. Indirect human interactions were included in the categories of gunshot, fisheries interactions, marine debris, and oiled fur. The higher prevalence of human interactions with harbor seals compared to elephant seals is likely due to the location of the rookeries. Harbor seals give birth on a large number of beaches in developed areas, while elephant seal rookeries, such as the rookeries of Año Nuevo and Point Reyes National Seashore, are more isolated. The prevalence of human-related injuries in phocid seals was much less than those previously reported for California sea lions along the California coast (Goldstein et al., 1999).

During the study period, medical assessment of animals varied considerably, depending on the availability of diagnostic tools and the experience of the hospital staff. Because knowledge of pinniped diseases and diagnostic capabilities have increased in the last decade, health assessment of animals improved over the study period. The information documented in the medical records was highly variable, which occasionally confounded the determination of the cause of stranding. Discrimination between conditions present at stranding from diseases developed during hospitalization was essential since the initial cause of stranding was sometimes different from the actual cause of death. An understanding of this difference has important management implications because the impacts of rehabilitation on disease prevalence needs to be understood to prevent transmission of diseases from rehabilitated animals to the wild population.

Although there are inherent biases (e.g., in age, health) in the disease data from live stranded animals, analysis of causes of stranding from long-term datasets can help assess disease trends in wild populations and may aid in future management. This study of the causes of phocid strandings revealed annual variability in the prevalences of a number of diseases, as well as changes in prevalence of disease over subsequent decades. Further data on factors contributing to these conditions are needed to understand these changes in prevalence and their relationship to prevalence of disease in the free-ranging population.

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