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NEONATAL MORTALITY IN NEW ZEALAND SEA LIONS (*PHOCARCTOS HOOKERI*) AT SANDY BAY, ENDERBY ISLAND, AUCKLAND ISLANDS FROM 1998 TO 2005

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ABSTRACT: As part of a health survey of New Zealand sea lions (*Phocarctos hookeri*) on Enderby Island, Auckland Islands (50°30′S, 166°17′E), neonatal mortality was closely monitored at the Sandy Bay colony for seven consecutive years. Throughout the breeding seasons 1998–99 to 2004–05, more than 400 postmortem examinations were performed on pups found dead at this site. The primary causes of death were categorized as trauma (35%), bacterial infections (24%), hookworm infection (13%), starvation (13%), and stillbirth (4%). For most pups, more than one diagnosis was recorded. Every year, two distinct peaks of trauma were observed: the first associated with mature bulls fighting within the harem and the second with subadult males abducting pups. In 2001–02 and 2002–03, epidemics caused by *Klebsiella pneumoniae* increased mortality by three times the mean in nonepidemic years (10.2%). The increased mortality was attributed directly to acute suppurative infection due to the bacterium and also to an increase in traumatic deaths of debilitated pups. Parasitic infection with the hookworm *Uncinaria* spp. was a common finding in all pups older than three weeks of age and debilitation by the parasite may have contributed to increased susceptibility to other pathogens such as *Klebsiella* sp. or *Salmonella* sp. This study provides valuable quantitative data on the natural causes of neonatal mortality in New Zealand sea lions that can be used in demographic models for management of threatened species.

Key words: Bacterial infection, health, hookworm infection, mortality, New Zealand sea lion, pathology, *Phocarctos hookeri*, pups.

INTRODUCTION

The New Zealand sea lion, *Phocarctos hookeri* (formerly known as Hooker's sea lion) is one of the rarest and most locally endemic members of the otariid family. Because of its restricted breeding distribution and limited population growth, the species was declared threatened in 1997 under provisions of the New Zealand Marine Mammals Protection Act 1978 (Molloy and Davis, 1994) and was listed as vulnerable by the World Conservation Union–IUCN (Reijnders et al., 1993). New Zealand sea lions breed on New Zealand's sub-Antarctic islands between latitudes 48°S and 53°S (Gales and Mattlin, 1997). Their population size is estimated at between 10,000 and 12,000 animals, approximately 5,000 of which are of mature age (Chilvers, unpubl. data). This is one of the smallest population sizes reported for an otariid species, and available data suggest that this population is declining despite having been protected since the late 1890s (Taylor, 1971; Wilkinson et al., 2003). Ninety percent of all breeding is concentrated on Dundas and Enderby Islands within the Auckland Islands group.

A highly localized distribution increases the vulnerability of the New Zealand sea lion to anthropogenic threats, such as fishing on the Aucklands Shelf, where 17 to 140 sea lions are accidentally caught each season (Baird, 1996; Wilkinson et al., 2003). The full impact of fisheries on the sea lion population is unknown, but several models suggest that this level of take may limit the species capacity to increase in number, and under some scenarios, this results in

population decline (Doonan and Cawthorn, 1984; Woodley and Lavigne, 1993). This uncertainty about anthropogenic impacts on the population is amplified when other natural regulators on population growth are considered, such as disease. From 1997 to 2005, three epidemics (1997–98, 2001–02 and 2002–03) occurred among New Zealand sea lions on the Auckland Islands (Baker, 1999; Wilkinson et al., 2006). The January 1998 event resulted in the death of 53% of that season's pups as well as an unknown number of adult animals (Baker, 1999). That event was the first evidence that disease plays a role in the demography of the New Zealand sea lion. It also highlighted the lack of baseline health data on the species and provided the impetus for a directed survey of neonatal mortality at the Sandy Bay colony, the second largest and most intensively studied breeding site for New Zealand sea lion. In this study, we determined causes of New Zealand sea lion pup mortality from 1998 to 2005, and here we discuss their importance and temporal variations within and between the breeding seasons.

MATERIALS AND METHODS

Over the survey period from 1998 to 2005, daily counts of live and dead pups were undertaken at the Sandy Bay rookery on Enderby Island, Auckland Islands (50°30′S, 166°17′E), and accurate pup production estimates were conducted using direct counts and mark-recapture techniques (Gales and Fletcher, 1999; Wilkinson et al., 2003; Chilvers, unpubl. data). Pups were born between mid-December and mid-January with a mean parturition date at about 26 December (Cawthorn et al., 1985; Chilvers, unpubl. data).

For every breeding season from 1998, pups on the beach were observed at least twice daily from early December through to the middle of February. Thus, all carcasses were retrieved within <12 hr of death. Carcasses from which all or most of the internal organs had been scavenged were not included in the study unless the cause of death was apparent. The animals were identified by sex, weighed (to nearest 100 g), measured (body length and girth at umbilicus), and examined externally for wounds, scars, and ectoparasites before being necropsied using a standard postmortem procedure (Duignan et al., 2003). Body condition was determined based on ventral abdominal blubber depth and liver weight. In fresh specimens, whole blood was collected from the heart and centrifuged to harvest serum. A sample of blubber was stored in liquid nitrogen for fatty acid analysis as part of associated diet and foraging studies on this species. Both normal and abnormal tissues were fixed in 10% buffered formalin to be processed later at Massey University (Palmerston North, New Zealand) using routine procedures and stains for veterinary histopathology (Luna, 1993).

Tissues and organs routinely sampled for histopathology included skin, skeletal muscle, brain, spinal cord, tongue, tonsil, thyroid, trachea, lungs, heart, lymph nodes, diaphragm, liver, spleen, pancreas, adrenal glands, kidney, stomach, intestine, urinary bladder, and gonads. Samples for bacteriology were systematically collected from liver, lung, tonsil, thymus, lymph nodes, spleen, feces, and any lesions or exudates and were stored in liquid nitrogen. Sheep blood agar, MacConkey, and XLD (xylose-lysine desoxycholate) agar plates were used for bacterial culture of selected samples: priority was given to internal lymphoid organs and lesions of pups diagnosed with bacterial infection. Beside routine enzymatic tests (oxidase, catalase, and coagulase), Microbact™ (MedVet Science Pty. Ltd, Adelaide, Australia), API®20NE (BioMérieux, France), and Rapid ID32 STREP® (BioMérieux, France) test kits were used to identify the microorganisms. *Salmonella* isolates were sent to Environmental Science & Research (Porirua, Wellington, New Zealand) for phagetyping.

A sample of internal organs from 30 pups was selected for virology. Spleen was the organ of choice if available. However, in two animals abdominal lymph node was selected by default. The samples were homogenized and cocultured on Vero cells and incubated (37 C, 5% CO_2 incubator) for two passages (7 days for each passage) and observed for cytopathic effects characteristic for morbilliviruses and herpesviruses.

The entire intestine was fixed in 10% formalin for later recovery of endoparasites. All parasites (ecto- and endoparasites) were stored in vials containing 90% ethanol.

Cause of death was categorized as follows: stillbirth, trauma, malnutrition, infection with the hookworm *Uncinaria* spp. (Castinel et al., 2006), bacterial infections, and fatal congenital defects. Stillbirths were identified by pulmonary atelectasis, an attached fresh umbilicus,

TABLE 1. New Zealand sea lion pups born, found dead, and examined at necropsy at Sandy Bay Beach, Enderby Island, from 1998–99 to 2004–05. "Total born" corresponds to the mark-recapture estimates at 16 January. "Total dead" includes data from early December to 16 February for each breeding season.

Season	Total born	Total dead	Mortality rate to mid-February (%)	Postmortem[a]
1998–99	513	33	6.4	26
1999–00	506	44	8.7	34
2000–01	562	61	10.9	48
2001–02	403	126	31.3	126
2002–03	489	108	22.1	108
2003–04	507	67	13.2	62
2004–05	441	54	12.2	51
Overall	3,421	493	14.4	455

[a] Does not include scavenged pups.

and often some autolysis of the carcass. Trauma included death from bite wounds, drowning, or crushing causing regurgitation and aspiration. Malnourished pups were often below average birth weight (10.6 kg for males and 9.7 kg for females; Chilvers, unpubl. data), had no blubber layer, and had hepatic atrophy. Hookworm enteritis was characterized by copious bloody content in the intestine, serosal ecchymoses, high worm burdens (mean individual burden: 824 hookworms; Castinel et al., 2007a), and anemic carcasses. Bacterial infection included any or all of the following: septicemia, suppurative pleuritis, peritonitis or meningitis, abscesses, suppurative or necrotizing pneumonia, and suppurative arthritis. Any pup may have had more than one diagnosis ranked in order of significance as the cause of death. Diagnosis was regarded as open where no clear cause of death could be determined at the postmortem examination.

Statistical analysis of data was undertaken using the statistical computer package SAS (Statistical Analyses System, Version 9.1, SAS Institute Inc., Cary, North Carolina, USA). Mortality rate (modeled as the proportion of deaths in a population per annum) was analyzed using the GENMOD procedure with a logit model that considered the fixed effects of year, sex, and their interaction. Likewise, each cause of mortality was analyzed using the GENMOD procedure with a logit model including the fixed effects of year, sex, and their interactions. Results were significant for $P \leq 0.05$. Prevalences are given as mean percentages.

RESULTS

In total, 455 pups were included in the survey, which constituted 13.3% of the pups born at Sandy Bay colony and 92.3% of the total reported dead for this site over the study period (Table 1). For the entire period of study, the annual pup mortality (number of dead pups from early December to mid-February) on Sandy Bay beach ranged between 6.4% and 31.3%, with a mean of 14.4% (Table 1). However, for the 2001–02 and 2002–03 breeding seasons, the mean mortality to mid-February (respectively 31.3% and 22.1%) was significantly higher compared to the other years (10.2%; $P<0.0001$). This high death rate was attributed to two years of epidemics caused by *Klebsiella pneumoniae* (Wilkinson et al., 2006). For the 2004–05 season, the mortality rate returned to almost the same level as in the pre-*Klebsiella* epidemic seasons (Table 1). In dead pups for which the sex of animals was reported, the number of males examined at necropsy was not significantly different from the number of females over the seasons.

In nonepidemic years, 32.2% of pups died from trauma, 17.0% from starvation, and 16.4% from hookworm infection. Bacterial infections were responsible for 13.5% of pup mortality outside the *K. pneumoniae* epidemics (Fig. 1). During these epidemics, the proportion of death directly attributed to bacterial infections increased to 36.3%, which was significantly greater than in the nonepidemic years ($P<0.0001$). In general, bacterial infec-

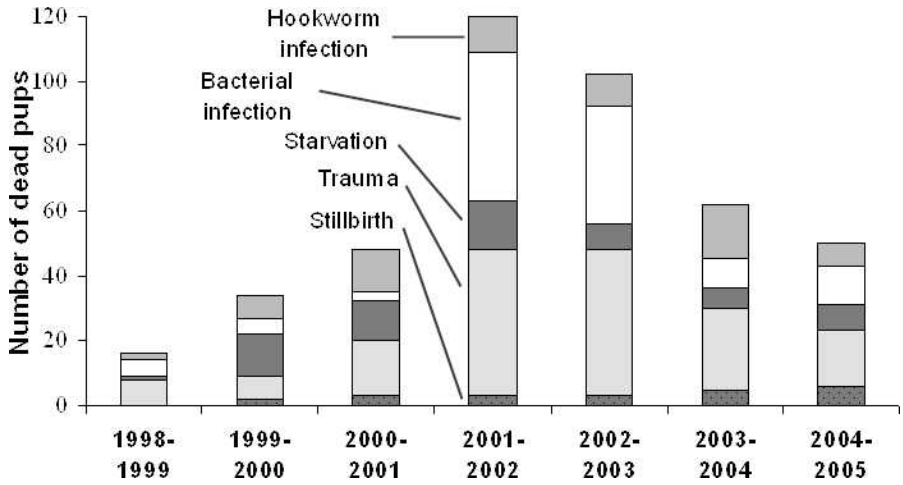

FIGURE 1. Distribution of the primary causes of neonatal mortality in New Zealand (NZ) sea lion pups on Enderby Island, Auckland Islands, for the 1998–99 to 2004–05 breeding seasons.

tions were more prevalent following the epidemics (Fig. 1); however, the proportion of bacterial infections as a primary cause of death returned to the same level as before the mass mortality events. The proportion of pups dead by trauma was not affected by the epidemics, but the absolute number that died from trauma increased in both of the epidemic years (Fig. 1). There were significantly fewer pups dying from starvation during and after the epidemics than before the epidemics ($P<0.005$). Likewise, fewer pups died from hookworm infection during the 2001–02 and 2002–03 seasons; however, this trend was not significant (Table 2). Finally, the percentage of stillbirth was consistently low each year regardless of epidemics (4.2%, Table 2).

Temporal variations of causes of mortality for the years 2000–01, 2001–02, 2002–03, and 2003–04 are shown in Figure 2, which includes one year before the *K. pneumoniae* epidemics, the two seasons of epidemics, and the year immediately after the epidemics. Traumatic injuries as a primary cause of mortality showed a consistent temporal trend each year from 1999–2000 to 2004–05 regardless of epidemics. The first peak was observed during the month following the peak of parturition and coincided with dominant males fighting within the harem. A lesser, but still remarkable, increase of fatal traumatic in-

TABLE 2. Distribution of the primary causes of neonatal mortality in New Zealand sea lion pups on Enderby Island, Auckland Islands, for the 1998–99 to 2004–05 breeding seasons.

Season	No. necropsies	No. pups with diagnosis	Stillbirth	Trauma	Starvation	Bacterial infection	Hookworm infection
1998–99	26	16	0	8	1[a]	5	2
1999–2000	34	34	2	7	13[a]	5	7
2000–001	48	48	3	17	12[a]	3	13
2001–02	126	120	3	45	15	46[b]	11
2002–03	108	102	3	45	8	36[b]	10
2003–04	62	62	5	25	6	9	17
2004–05	51	50	6	17	8	12	7

[a] Significantly different from the following years ($P<0.005$).

[b] Significantly different from the other years ($P<0.001$).

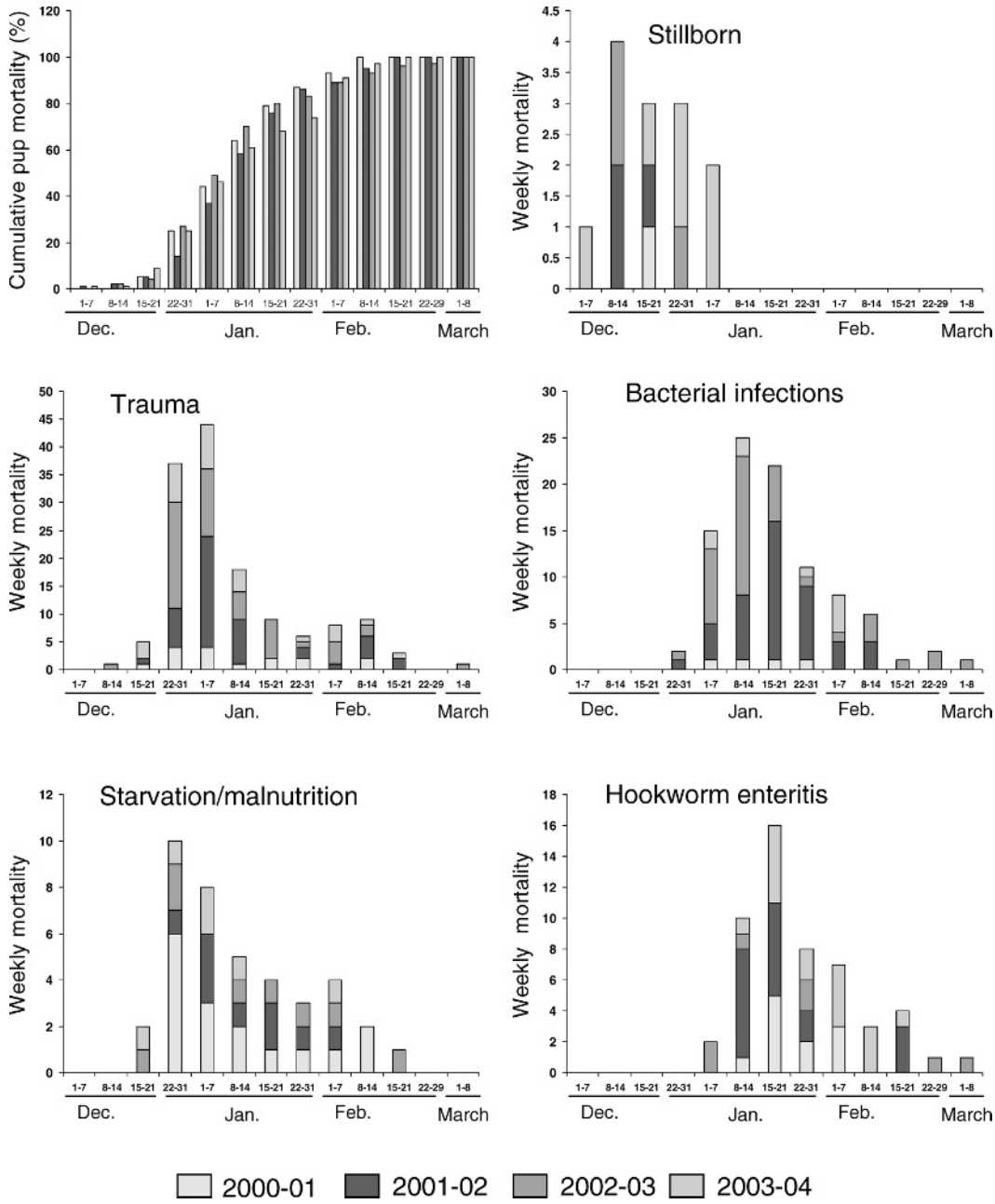

FIGURE 2. Incidence of primary causes of mortality in New Zealand sea lion pups on Enderby Island, Auckland Islands, for the 2000–01, 2001–02, 2002–03, and 2003–04 breeding seasons.

juries usually occurred later in February, this time associated with subadult and nondominant males abducting pups.

The first diagnoses of hookworm infection at necropsy were made during the first week (in 2001–02 and 2002–03) or the second week of January (in 2000–01 and 2003–04), which is approximately 3 wk after the annual peak of birth.

In nonepidemic years, the most prevalent primary cause of death until mid-January was starvation; in mid-January,

the most prevalent primary cause of death was hookworm infection; there was a rise in prevalence of bacterial infections and trauma in early February (Fig. 2). The trends for the two epidemic years, 2001–02 and 2002–03 were similar to the nonepidemic years with respect to the prevalence of causes of death, except that there was a marked increase in the number of pups that died of bacterial infections and trauma in epidemic years (Figs.1, 2). Isolates from bacterial cultures included more normal flora than pathogenic species (Table 3). *Klebsiella pneumoniae* was the most significant pathogen isolated from a wide range of tissues and lesions in both 2001–02 and 2002–03, and it was also isolated from dead pups in the years subsequent to the epidemics. However, attempts to isolate this pathogen from archived samples collected prior to the 2001–02 epidemic failed (Castinel et al., 2007b). Several different *Salmonella enteriditis* subspecies (serotypes Cerro, Derby, and Newport) were also identified from pup feces collected at necropsy over the whole period of study. In 2001–02, six of six cultured samples were *Salmonella* positive, in 2002–003, 17 of 19 samples (89.5%) were positive, and in 2003–04, *Salmonella* was cultured from nine of 14 samples (64.3%).

Of the thirty virology cultures, two were contaminated with *Aspergillus* spp., making any interpretation impossible, and all the others were negative after two passages on Vero cells for viruses identified by cytopathic effect.

A low prevalence of congenital anomalies was reported (12/476 or 2.6%). In some cases, those defects, such as atresia coli, would be inevitably fatal, while others were incidental findings in pups where another primary cause of death was identified (Table 4).

DISCUSSION

The seven continuous years of monitoring allowed the main causes of neonatal mortality to be identified. These included, in order of prevalence: trauma, bacterial and hookworm infections, starvation, and stillbirth. Furthermore, the data enabled an analysis of the impact of variables such as year, sex, age, and stochastic events (epidemics) that could influence the mortality rate and causes of death. The studies on other pinniped species, and in particular on otariid species, with which to compare this survey are limited. Most of the previously published reports present data for only a short period, usually for one breeding season (Mattlin, 1978; Baker et al., 1980; Georges and Guinet, 2000). Furthermore, few other studies have accumulated detailed demographic data over a long period on births and deaths and had access to almost all of the dead pups for detailed necropsy and sampling.

Overall, the mortality recorded for nonepidemic years during the present study for New Zealand sea lion was not unusually high compared to other pinniped populations not subjected to unusual events. Thus, the mean mortality in New Zealand sea lion neonates was 10.2%, which is comparable to 12.5% in gray seals (*Halichoerus grypus*) from the Isle of May sampled in 1986 (Baker and Baker, 1988), 11.25% in California sea lions (*Zalophus californianus*) from Baja California between 1982 and 1985 (Aurioles and Sinsel, 1988), and 13% for South American sea lions (*Otaria byronia*) in years not affected by El Niño (Soto et al., 2004). Slightly higher (20%) mortality has been reported in the New Zealand fur seal, *Arctocephalus forsteri* (Mattlin, 1978). Stochastic events such as El Niño–La Niña cycles can have devastating effects on neonatal otariids and cause up to 100% mortality in species such as the South American sea lion, the Galapagos fur seal (*Arctocephalus galapagoensis*), and the California sea lion (Francis and Heath, 1991; Trillmich and Dellinger, 1991; Soto et al., 2004).

For all years, the number of New Zealand sea lion dead pups per week was

TABLE 3. List of bacteria isolated from various tissues of New Zealand sea lion pups, from 1998–99 to 2004–05. Frequencies of the different bacterial isolates are given by groups of tissues for the seasons before the *Klebsiella pneumoniae* epidemics (1998–99, 1999–2000 and 2000–01), for the two epidemic years (2001–02 and 2002–03), and for the seasons after the epidemics (2003–04 and 2004–05).

Tissue	Before epidemics	Epidemics	After epidemics
Lymphoid tissues (tonsil, lymph nodes, spleen, thymus)	*Escherichia coli* (2/3); *Streptococcus* sp. (1/3); *Salmonella enteriditis* subspp. (1/3)	*Klebsiella pneumoniae* (41/45); *Staphylococcus intermedius* (1/10); *Staphylococcus aureus* (1/17); *E. coli* (3/25); *Streptococcus canis* (1/2); *Streptococcus dysgalactiae* subsp. *equisimilis* (2/12)	*K. pneumoniae* (33/70); *Klebsiella oxytoca* (5/66); *E. coli* (7/42); *Salmonella enteriditis* subspp. (1/25); *Streptococcus* spp. (3/57); *Staphylococcus* spp (6/38); *Proteus* spp. (4/25)
Subcutaneous tissues (including muscles, abscesses)	*Streptococcus* spp. (1/1)	*K. pneumoniae* (11/12); *Proteus* spp. (1/6); *Streptococcus* spp. (1/6); *E. coli* (1/6)	*Staphylococcus* spp. (1/1); *K. pneumoniae* (2/9); *E. coli* (1/9); *Staphylococcus* spp. (1/9)
Joints fore-flipper (shoulder, elbow, metacarpus, carpus)	—	*K. pneumoniae* (32/34); *Staphylococcus intermedius* (1/13); *E. coli* (2/18); *Streptococcus canis* (1/14)	*K. pneumoniae* (18/32); *K. oxytoca* (4/28); *Streptococcus* spp. (4/24); *Staphylococcus* spp. (1/8); *E. coli* (1/20); *Shigella* spp. (1/4)
Joints back-flipper (hip, metatarsus, tarsus)	—	*K. pneumoniae* (8/11); *Proteus* spp. (2/4); *Staphylococcus intermedius* (2/4); *E. coli* (1/7)	*K. pneumoniae* (1/3); *Staphylococcus* spp. (1/3)
Liver, pancreas	*K. oxytoca* (1/3); *Staphylococcus* sp. (1/3); *E. coli* (1/3)	*K. pneumoniae* (3/3)	*K. pneumoniae* (2/2)
Lung	*Salmonella enteriditis* subsp. (1/4); *Staphylococcus aureus* (1/4); *Proteus* sp. (1/4); *Streptococcus* sp. (1/4)	*K. pneumoniae* (3/4); *E. coli* (1/4); *Streptococcus canis* (1/4); *Proteus* sp. (1/4)	*K. pneumoniae* (7/20); *E. coli* (5/20); *Staphylococcus* sp. (1/20); *Streptococcus* spp. (3/20)
Abdominal cavity (peritoneum)	*E. coli* (1/2); *Streptococcus* sp. (1/2)	*K. pneumoniae* (6/6)	*K. pneumoniae* (6/26); *K. oxytoca* (3/9); *Salmonella enteriditis* subsp. (1/9)
Spino-cerebral tissue (meninges/ brain)	*Staphylococcus* sp. (1/2); *Streptococcus* sp. (1/2)	*K. pneumoniae* (6/8); *Staphylococcus intermedius* (2/8); *Proteus* spp. (2/8)	*K. pneumoniae* (5/8); *E. coli* (1/8); *Streptococcus* sp. (1/18)
Thoracic cavity		*K. pneumoniae* (2/3)	*K. pneumoniae* (1/4); *K. oxytoca* (1/4); *E. coli* (1/4)
Kidney	*E. coli* (1/1)	—	—
Pericardial fluid/myocardium	—	—	*K. pneumoniae* (2/4); *Staphylococcus* spp. (2/4)

TABLE 4. Congenital defects in New Zealand sea lion pups found dead at Sandy Bay Beach from 1998–99 to 2004–05, with prevalence and cause of death.

Congenital defect	Number affected	Cause of death
Intestinal atresia	4/12 (33%)	Atresia coli
Ventricular septal defect	1/12 (8%)	Cardiac anomaly and malnutrition
Hypoplasia of one adrenal and one thyroid gland	1/12 (8%)	Hookworm enteritis and malnutrition
Right renal aplasia and polycystic left kidney	1/12 (8%)	Acute suppurative pleuritis and vaginitis
Right thyroid hypoplasia and left thyroid hyperplasia	1/12 (8%)	Malnutrition
Hiatus hernia	1/12 (8%)	Hookworm enteritis
Scoliosis of cervical spine	1/12 (8%)	Malnutrition

higher in January when they were from 1 to 5 wk old; 50% of the seasonal pup mortality occurred in the first week of January when pups were about two weeks of age. The only comparable data on pup mortality in relation to age were found in studies on fur seals. In sub-Antarctic fur seals (*Arctocephalus tropicalis*), the mortality rate was higher from birth to 2 wk old (9%) and decreased to 3.2% between 2 wk and 1 mo of age (Georges and Guinet, 2000). In Antarctic fur seals (*Arctocephalus gazella*) at South Georgia, half of neonatal mortality had occurred by the age of 2 days and 90% by 1 mo (Doidge et al., 1984). These comparisons suggest that there is a similar trend between studies and that otariid neonates in general are more at risk in their first few weeks of life.

In 1997–98, a mass mortality event killed 60% of New Zealand sea lion neonates and many adults at all three Auckland Islands rookeries (North Auckland Islands, Dundas, and Figure of Eight Islands). It was not possible to attribute the mortality to a single cause because too few necropsies were carried out at that time (Baker, 1999). When further epidemics occurred in 2001–02 and 2002–03, mortality associated with bacterial infections increased threefold, and the pathogen *K. pneumoniae* was isolated in pure culture from almost all pups examined at necropsy (Wilkinson et al., 2006). Species of *Klebsiella* are widespread pathogens capable of causing severe nosocomial outbreaks in humans hospitalized in intensive care units and in neonatal wards resulting in septicemia (Peña et al., 1998; Lebessi et al., 2002). Species of *Klebsiella* have been commonly cultured from various internal tissues in marine mammals (Stroud and Roffe, 1979; Vedros et al., 1982; Baker and McCann, 1989; Hernández-Castro et al., 2005) and in a vast range of wild birds, reptiles, and terrestrial mammals (Bartoszcze et al., 1990; Aguirre et al., 1994; Montgomery et al., 2002; Steele et al., 2005). Yet, species of *Klebsiella* have never previously been reported to cause an epidemic in an animal population (Wilkinson et al., 2006). The origin of infection here is unknown, but the lack of isolates from samples collected prior to 2001–02 suggests a recent introduction of the pathogen into this population.

The present study ranked bacterial infections as the third cause of direct mortality in New Zealand sea lion neonates in nonepidemic years (after trauma and hookworm disease), but they were recorded as the major cause of death in gray seal pups in two studies (Anderson et al., 1979; Baker et al., 1980). In New Zealand sea lions, bacterial infections as a primary cause of death accounted for less than 15% of pup mortality outside the *K. pneumoniae* epidemic years (Fig. 2).

However, the overall prevalence remained intermediate in the years following the epidemics. The abrasive action of sand and a dirty environment has been associated with infection of the umbilicus, which evolves into peritonitis and septicemia after passage of the pathogens through liver. Omphalitis, with subsequent peritonitis, has been observed in other pinnipeds. It contributed to the death of up to 50% of pups in gray seal colonies (Anderson et al., 1979; Baker and Baker, 1988). Somewhat surprisingly, there were only a few cases of omphalitis diagnosed during the survey at Sandy Bay Beach rookery, and cases of peritonitis were not associated with the occurrence of omphalitis. Most of the bacteria involved in these cases of septicemia were *Klebsiella pneumoniae* (during and after the *K. pneumoniae* epidemics), *Streptococcus* spp., and *Escherichia coli.*

The different categories of primary causes of death in New Zealand sea lion pups, in epidemic and in nonepidemic years, were similar to those described in other pinniped species, although their relative ranking in order of importance did vary. For instance, Keyes (1965) reported that the primary diagnoses of neonatal mortality in the northern fur seal were, by order of importance, malnutrition, trauma, hookworm infection, and miscellaneous bacterial infections. In New Zealand sea lions, trauma was the predominant cause of pups' death in all years. Signs of suffocation as well as cranial and thoracic hemorrhage in pups less than 2 wk old were the common presentations of trauma caused by adult animals. Later in the breeding season, internal abdominal hemorrhage and bites and skin lesions were correlated with subadult and peripheral males physically abducting pups while lactating females were feeding at sea (Chilvers et al., 2005). Moreover, Wilkinson et al. (2000) suggested that pup abduction associated with infanticide and cannibalism in New Zealand sea lions could be a significant cause of neonatal mortality. However, in the present study and in other otariid species (Antarctic fur seals: Doidge et al., 1984; Australian sea lions, *Neophoca cinerea*: Higgins and Tedman, 1990; northern fur seals: Kiyota and Okamura, 2005), male harassment and abduction resulting in death of pups seemed to be the result of misdirected aggression or an accidental occurrence. Neonates would simply be too small and too slow to avoid attacks and to survive the injuries that are likely to be insignificant in older animals.

Topography and density of animals on the rookery may also affect neonatal survival. For instance, the open Sandy Bay beach does not provide any shelter for New Zealand sea lion pups to hide from adult males and scavenging birds; the same hazardous conditions have been reported for sub-Antarctic fur seals (Georges and Guinet, 2000) and gray seals (Twiss et al., 2003). In contrast, rookeries on rocky shores with boulders and crevices provide shelter for fur seal pups from fighting males (Bradshaw et al., 2000; Kiyota and Okamura, 2005). In addition, in Antarctic fur seals, there is evidence that frequency of aggressive interactions increases when colony density becomes high (Doidge et al., 1984; Reid and Forcada, 2005). Thus, it is possible that infanticide and cannibalism reported in New Zealand sea lion on Dundas Island by Wilkinson et al. (2000) could be linked to the fourfold higher number of animals in that rookery compared to Sandy Bay (Enderby Island), where no such behavior was observed during the present survey.

Starvation is recognized as an important cause of death in pinniped pups. The estimate of 13% in this study is similar to that reported in gray seals by Anderson et al. (1979) but contrasts with other studies describing starvation as the major factor of neonatal mortality in New Zealand fur seals (Mattlin, 1978) and in Hawaiian monk seals, *Monachus schauinslandi*

(Banish and Gilmartin, 1992). It is most likely that starvation in New Zealand sea lion neonates was primarily due to failure of the mother-pup bond to persist. This may happen for various reasons, such as the death of the female from shark predation, disease, or male aggression (Wilkinson et al., 2000; Chilvers et al., 2005). Given that lactating New Zealand sea lion females forage at great distances from rookery sites and at greater depth than most other otariids, they constantly live at their physiological limits (Costa and Gales, 2000; Chilvers et al., 2006). As a consequence, New Zealand sea lions are very likely to be susceptible to fluctuations of food resources as caused by El Niño/La Niña events or other climatic fluctuations. The effect of these events has not been investigated in New Zealand sea lion, but foraging patterns of other marine species that share the same latitudes have been studied in parallel with El Niño (low prey abundance) and La Niña (high prey abundance) events (Boyd et al., 1994; Peacock et al., 2000; Bowen et al., 2001; Soto et al., 2004). Availability of prey in the equatorial Pacific region varies considerably with the El Niño–Southern Oscillation (ENSO) (Barber and Chavez, 1983; Glynn, 1988). Periods of low prey abundance have led the lactating females of some otariid species to extend the duration of their foraging trips with increased activity and energy expenditure (Cape fur seals, *Arctocephalus pusillus pusillus*: Bowen et al., 2001; Antarctic fur seals: Boyd et al., 1994). When female Cape fur seals cannot cope with extreme physiological conditions, they abandon their pup (Bowen et al., 2001). Similarly, pup growth in New Zealand fur seals partly reflects maternal attendance of lactating females and food availability (Bradshaw et al., 2000). Although the first recorded New Zealand sea lion mass mortality was concurrent with 1997–98 ENSO event, there was no epidemiologic evidence from the 1997–98 necropsy data to show any relationship between the occurrence of El Niño or La Niña years (1997–98 for El Niño and 1998–99 for La Niña) and starvation and malnutrition in New Zealand sea lion pups.

Uncinaria spp. represent a constant primary cause of pup mortality in this colony of New Zealand sea lion. Every year, adult hookworms and associated intestinal lesions could be observed for the first time about three weeks after the majority of pups were born at Sandy Bay rookery. Thus, it would appear to take about three weeks for the infective larvae to develop into mature adults feeding on the intestinal mucosa in New Zealand sea lion pups (Castinel, unpubl. data). This observation is based on the hypothesis that transmammary transmission is the only mode of transmission to pups that completes the life cycle in the northern fur seal (Olsen and Lyons, 1965). Although the prevalence of infection is high, the prevalence of parasitic disease as a primary cause of death is low. The lower proportion of pups diagnosed with hookworms in 2001–02 (9.2%) and 2002–03 (9.8%) is more likely to be due to pups dying from acute bacterial infection and septicemia (38.3% and 33.3%, respectively) before 3 wk of age rather than a lower intensity of parasites during the epidemic years. The epidemiology of hookworm infection in New Zealand sea lion pups was investigated at Sandy Bay Beach rookery and is reported as a separate study (Castinel et al., 2007a).

Hookworms, and to some extent any parasites, including bacteria and viruses (May and Anderson, 1979), may play a role in pup mortality by causing debilitation leading to trauma, malnutrition, and hypothermia. Such a scenario has also been reported for Hawaiian monk seal neonates (Banish and Gilmartin, 1992) and for northern fur seal pups (Spraker et al., 2004). To further investigate this hypothesis in New Zealand sea lions, studies are under way to correlate infection by enteric bacteria with the extent of lesions caused by *Uncinaria* spp. embedded in the mucosa.

Viral infections have been associated with neonatal mortality in other pinniped species. Phocid herpes viruses (PhHV-1 and PhHV-2) have been isolated from harbor seal (*Phoca vitulina*) neonates that died in a seal rehabilitation center from pneumonia or adrenal necrosis (Osterhaus et al., 1985; Gulland et al., 1997). Morbillivirus infection (phocine distemper virus) may also cause neonatal mortality in phocid seals but has not been reported in otariids (Daoust et al., 1993; Duignan et al., 1993, 1995). Caliciviruses may be involved in abortion and neonatal death in California sea lions (Smith et al., 1979). In the present study, no viral infections were identified in tissues cultured from New Zealand sea lion pups, but the sample size was limited.

This paper is the first to report the primary causes of mortality in pups from birth to three months of age in New Zealand sea lions. Stillbirth, starvation, and hookworm disease remained relatively prevalent over the seven years of the survey, while bacterial infections and trauma increased dramatically for two years of epidemics caused by the bacillus *Klebsiella pneumoniae*. Even though the proportions of primary causes of mortality were reestablished soon after the epidemics, long-term consequences of increased pup mortalities are unknown in the demographics of this threatened species (Wilkinson et al., 2006). Aside from the demographic effect of stochastic events such as the *Klebsiella* epidemics, it is now possible to include relatively prevalent causes of mortality such as hookworm and starvation into demographic models for this species. Furthermore, the data presented here can assist in the management of this threatened pinniped species.

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LITERATURE CITED

AGUIRRE, A. A., G. H. BALAZS, B. ZIMMERMAN, AND T. R SPRAKER. 1994. Evaluation of Hawaiian green turtles (*Chelonia mydas*) for potential pathogens associated with fibropapillomas. Journal of Wildlife Diseases 30: 8–15.

ANDERSON, S. S., J. R. BAKER, J. H. PRIME, AND A. BAIRD. 1979. Mortality in grey seal pups: Incidence and causes. The Journal of Zoology, London 189: 407–417.

AURIOLES, D., AND F. SINSEL. 1988. Mortality of California sea lion pups at Los Islotes, Baja California Sur, Mexico. The Journal of Mammalogy 69: 180–183.

BAIRD, S. J. (compiler). 1996. Non fish species and fisheries interactions. New Zealand Fisheries Assessment Working Group Report 96/1, May 1996 Ministry of Fisheries, Wellington, New Zealand.

BAKER, A. J. 1999. Unusual mortality of the New Zealand sea lion, *Phocarctos hookeri*, Auckland Islands, January–February 1998: A report of a workshop held 8–9 June 1998, Wellington, and a contingency plan for future events. pp. 29–33, Department of Conservation, Te Papa Atawhai, Wellington, New Zealand, http://www.doc.govt.nz/publications/004~Science-and-Research/Miscellaneous/PDF/sealion.pdf. Accessed 12 August 2006.

———, AND R. BAKER. 1988. Effects of environment on grey seal (*Halichoerus grypus*) pup mortality.

Studies on the Isle of May. The Journal of Zoology, London 216: 529–537.

———, AND T. S. MCCANN. 1989. Pathology and bacteriology of adult male Antarctic fur seals, *Arctocephalus gazelle*, dying at Bird Island, South Georgia. British Veterinary Journal 145: 263–275.

———, S. S. ANDERSON, J. H. PRIME, AND A. BAIRD. 1980. The pathology of the grey seal (*Halichoerus grypus*). I. Pups. British Veterinary Journal 136: 401–412.

BANISH, L. D., AND W. G. GILMARTIN. 1992. Pathological findings in the Hawaiian monk seal. Journal of Wildlife Diseases 28: 428–434.

BARBER, R. T., AND F. P. CHAVEZ. 1983. Biological consequences of El Niño. Science 222 (4629): 1203–1210.

BARTOSZCZE, M., J. MATRAS, S. PALEC, J. ROSZKOWSKI, AND E. WYSTUP. 1990. *Klebsiella pneumoniae* infection in chinchillas. The Veterinary Record 127: 119.

BOWEN, W. D., J. HARWOOD, D. GOODMAN, AND G. L. SWARTZMAN. 2001. Review of the November 2000 Biological Opinion and incidental take statement with respect to the western stock of the Steller sea lion. Final report for the North Pacific Fisheries Management Council, Anchorage, Alaska, 52 pp.

BOYD, I. L., J. P. Y. ARNOULD, T. BARTOUN, AND J. P. CROXALL. 1994. Foraging behaviour of Antarctic fur seals during periods of contrasting prey abundance. The Journal of Animal Ecology 63: 703–713.

BRADSHAW, C. J. A., L. S. DAVIS, C. LALAS, AND R. G. HARCOURT. 2000. Geographic and temporal variation in the condition of pups of the New Zealand fur seal (*Arctocephalus forsteri*): Evidence for density dependence and differences in the marine environment. The Journal of Zoology, London 252: 41–51.

CASTINEL, A., P. J. DUIGNAN, W. E. POMROY, E. T. LYONS, S. A. NADLER, M. D. DAILEY, I. S. WILKINSON, AND B. L. CHILVERS. 2006. First report and characterization of adult *Uncinaria* spp. in New Zealand sea lion (*Phocarctos hookeri*) pups. Parasitology Research 98: 304–309.

———, ———, E. T. LYONS, W. E. POMROY, N. GIBBS, N. LOPEZ-VILLALOBOS, B. L. CHILVERS, AND I. S. WILKINSON. 2007a. Epidemiology of hookworm (*Uncinaria* spp.) infection in New Zealand (Hooker's) sea lion (*Phocarctos hookeri*) pups on Enderby Island, Auckland Islands (New Zealand) during the breeding seasons from 1999/2000 to 2004/2005. Parasitology Research 101: 53–62.

———, A. GRINBERG, R. PATTISON, R. DUIGNAN, P. J. DUIGNAN, W. E. POMROY, L. ROGERS, AND I. S. WILKINSON. 2007b. Characterization of *Klebsiella pneumoniae* isolates from New Zealand sea lion (*Phocarctos hookeri*) pups during and after the epidemics on Enderby Island, Auckland Islands. Veterinary Microbiology 122: 178–184.

CAWTHORN, M. W., M. C. CRAWLEY, R. H. MATTLIN, AND G. J. WILSON. 1985. Research on pinnipeds in New Zealand. WRLG Research Review Number 7, Pinniped Research Review Subcommittee Department of Conservation, Wellington, New Zealand, 34 pp.

CHILVERS, B. L., B. C. ROBERTSON, I. S. WILKINSON, P. J. DUIGNAN, AND N. J. GEMMEL. 2005. Male harassment of female New Zealand sea lions, *Phocarctos hookeri*: Mortality, injury, and harassment avoidance. Canadian Journal of Zoology 83: 642–648.

———, I. S. WILKINSON, P. J. DUIGNAN, AND N. J. GEMMEL. 2006. Diving to extremes: Are New Zealand sea lions (*Phocarctos hookeri*) pushing their limits in a marginal habitat? Journal of Zoology 269: 233–240.

COSTA, D. P., AND N. G. GALES. 2000. Foraging energetics and diving of lactating New Zealand sea lions, *Phocarctos hookeri.* The Journal of Experimental Biology 203: 3655–3665.

DAOUST, P. Y., D. M. HAINES, J. THORSEN, P. J. DUIGNAN, AND J. R. GERACI. 1993. Phocine distemper virus in a harp seal (*Phoca groenlandica*) from the Gulf of St. Lawrence, Canada. Journal of Wildlife Diseases 29: 114–117.

DOIDGE, D. W., J. P. CROXALL, AND J. R. BAKER. 1984. Density-dependent pup mortality in the Antarctic fur seal *Arctocephalus gazella* at South Georgia. Journal of Zoology, London 202: 449–460.

DOONAN, I. J., AND M. W. CAWTHORN. 1984. Impacts of incidental mortality on the Hookers sea lions. Internal report number 6 (unpublished report), Fisheries Research Division, New Zealand Ministry of Fisheries, Wellington, New Zealand, 52 pp.

DUIGNAN, P. J., S. SADOVE, J. T. SALIKI, AND J. R. GERACI. 1993. Phocine distemper in harbor seals (*Phoca vitulina*) from Long Island, New York. Journal of Wildlife Diseases 29: 465–469.

———, J. T. SALIKI, D. J. ST. AUBIN, G. EARLY, S. SADOVE, J. A. HOUSE, K. KOVACS, AND J. R. GERACI. 1995. Epizootiology of morbillivirus in North American harbour seals (*Phoca vitulina*) and gray seals (*Halichoerus grypus*). Journal of Wildlife Diseases 31: 491–501.

———, N. GIBBS, AND G. JONES. 2003. Autopsy of pinnipeds incidentally caught in commercial fisheries, 2001/2002. DOC Science Internal Series 131, 41 pp, http://www.doc.govt.nz/Publications/004~Science-and-Research/DOC-Science-Internal-Series/PDF/dsis131.pdf. Accessed 25 July 2006.

FRANCIS, J., AND C. HEATH. 1991. Population abundance, pup mortality, and copulation frequency in the California sea lion in relation to

the 1983 El Niño on San Nicolas Island. *In* Pinnipeds and El Niño: Responses to environmental stress, F. Trillmich and K. A. Ono (eds.), Springer-Verlag, New York, pp. 119–128.

GALES, N. J., AND D. J. FLETCHER. 1999. Abundance, distribution and status of the New Zealand sea lion, *Phocarctos hookeri*. Wildlife Research 26: 35–52.

———, AND R. H. MATTLIN. 1997. Summer diving behaviour of lactating New Zealand sea lions, *Phocarctos hookeri*. Canadian Journal of Zoology 75: 1695–1706.

GEORGES, J.-Y., AND C. GUINET. 2000. Early mortality and perinatal growth in the sub-Antarctic fur seal (*Arctocephalus tropicalis*) on Amsterdam Island. Journal of Zoology, London 251: 277–287.

GLYNN, P. W. 1988. El Niño–Southern Oscillation 1982–1083: Nearshore population, community, and ecosystem response. Annual Review of Ecology and Systematics 19: 309–345.

GULLAND, F. M., L. J. LOWENSTINE, J. M. LAPOINTE, T. SPRAKER, AND D. P. KING. 1997. Herpesviruses infection in stranded Pacific harbor seals of coastal California. Journal of Wildlife Diseases 33: 450–458.

HERNÁNDEZ-CASTRO, R., L. MARTÍNEZ-CHAVARRÍA, A. DÍAZ-AVELAR, A. ROMERO-OSORIO, C. GODÍNEZ-REYES, A. ZAVALA-GONZÁLEZ, AND A. VERDUGO-RODRÍGUEZ. 2005. Aerobic bacterial flora of the nasal cavity in Gulf of California sea lion (*Zalophus californianus*) pups. The Veterinary Journal 170: 359–363.

HIGGINS, L. V., AND R. A. TEDMAN. 1990. Effects of attacks by male Australian sea lions, *Neophoca cinerea*, on mortality of pups. Journal of Mammalogy 71: 617–619.

KEYES, M. C. 1965. Pathology of the northern fur seal. The American Journal of Veterinary Medicine Association 147: 1090–1095.

KIYOTA, M., AND H. OKAMURA. 2005. Harassment, abduction and mortality of pups by nonterritorial male northern fur seals. Journal of Mammalogy 86: 1227–1236.

LEBESSI, E., H. DELLAGRAMMATICAS, P. T. TASSIOS, L. S. TZOUVELEKIS, S. IOANNIDOU, M. FOUSTOUKOU, AND N. J. LEGAKIS. 2002. Extended-spectrum beta-lactamase-producing *Klebsiella pneumoniae* in a neonatal intensive care unit in the high-prevalence area of Athens, Greece. Journal of Clinical Microbiology 40: 799–804.

LUNA, L. G. 1993. Histopathologic methods and color atlas of special stains and tissue artifacts. American Histolabs Publication Department, Gaithersburg, Maryland, 767 pp.

MATTLIN, R. H. 1978. Pup mortality of the New Zealand fur seal (*Arctocephalus forsteri* Lesson). The New Zealand Journal of Ecology 1: 138–144.

MAY, R. M., AND R. M. ANDERSON. 1979. Population biology of infectious diseases: Part II. Nature 280: 455–461.

MOLLOY, J., AND A. DAVIS. 1994. Setting priorities for the conservation of New Zealand's threatened plants and Animals. 2nd Edition. Department of Conservation, Wellington, New Zealand, 32 pp.

MONTGOMERY, J. M., D. GILLESPIE, P. SASTRAWAN, T. M. FREDEKING, AND G. L. STEWART. 2002. Aerobic salivary bacteria in wild and captive Komodo dragons. Journal of Wildlife Diseases 38: 545–551.

OLSEN, O. W., AND E. T. LYONS. 1965. Life cycle of *Uncinaria lucasi* Stiles, 1901 (Nematoda: Ancylostomatidae) of fur seals *Callorhinus ursinus* on the Pribilof Islands, Alaska. Journal of Parasitology 51: 689–700.

OSTERHAUS, A. D., H. YANG, H. E. SPIJKERS, J. GROEN, J. S. TEPPEMA, AND G. VAN STEENIS. 1985. The isolation and partial characterization of a highly pathogenic herpesvirus from the harbor seal (*Phoca vitulina*). Archives of Virology 86: 239–251.

PEACOCK, L., M. PAULIN, AND J. DARBY. 2000. Investigations into climate influence on population dynamics of yellow-eyed penguins *Megadyptes antipodes*. New Zealand Journal of Zoology 27: 317–325.

PEÑA, C., M. PUJOL, C. ARDANUY, A. RICART, R. PALLARES, J. LIÑARES, J. ARIZA, AND F. GUDIOL. 1998. Epidemiology and successful control of a large outbreak due to *Klebsiella pneumoniae* producing extended-spectrum beta-lactamases. Antimicrobial Agents and Chemotherapy 42: 53–58.

REID, K., AND J. FORCADA. 2005. Causes of offspring mortality in the Antarctic fur seal, *Arctocephalus gazella*: The interaction of density dependence and ecosystem variability. Canadian Journal of Zoology 83: 604–609.

REIJNDERS, P., S. BRASSEUR, J. VAN DER TOORN, P. VAN DER WOLF, I. BOYD, J. HARWOOD, D. LAVIGNE, AND L. LOWRY. 1993. Seals, fur seals, sea lions and walrus. Status Survey and Conservation plan IUCN/SSC Seal Specialist Group, IUCN, Gland, Switzerland, 88 pp.

SMITH, A. W., T. G. AKERS, A. B. LATHAM, D. E. SKILLING, AND H. L. BRAY. 1979. A new calicivirus isolated from a marine mammal. Archives of Virology 61: 255–259.

SOTO, K. H., A. W. TRITES, AND M. ARIAS-SCREIBER. 2004. The effects of prey availability on pup mortality and the timing of birth of South American sea lions (*Otaria flavescens*) in Peru. The Journal of Zoology, London 264: 419–428.

SPRAKER, T. R., E. T. LYONS, R. L. DELONG, AND R. R. ZINK. 2004. Penetration of the small intestine of a California sea lion (*Zalophus californianus*) pup by adult hookworms (*Uncinaria* spp.). Parasitology Research 92: 436–438.

STEELE, C. M., R. N. BROWN, AND R. G. BOTZLER.

2005. Prevalences of zoonotic bacteria among seabirds in rehabilitation centers along the Pacific coast of California and Washington, USA. Journal of Wildlife Diseases 41: 735–744.

STROUD, R. K., AND T. J. ROFFE. 1979. Causes of death in marine mammals stranded along the Oregon coast. Journal of Wildlife Diseases 15: 91–97.

TAYLOR, R. H. 1971. Influence of man on vegetation and wildlife of Enderby and Rose Island, Auckland Islands. New Zealand Journal of Botanics 9: 225–268.

TRILLMICH, F., AND T. DELLINGER. 1991. The effects of El Niño on Galapagos pinnipeds. *In* Pinnipeds and El Niño: Responses to environmental stress, F. Trillmich and K. A. Ono (eds.), Springer-Verlag, New York, pp. 119–128.

TWISS, S. D., DUCK, C., AND P. P. POMEROY. 2003. Grey seal (*Halichoerus grypus*) pup mortality not explained by local breeding density on North Rona, Scotland. Journal of Zoology 259: 83–91.

VEDROS, N. A., J. QUINLIVAN, AND R. CRANFORD. 1982. Bacterial and fungal flora of wild northern fur seals (*Callorhinus ursinus*). Journal of Wildlife Diseases 18: 447–456.

WILKINSON, I. S., S. J. CHILDERHOUSE, P. J. DUIGNAN, AND F. M. D. GULLAND. 2000. Infanticide and cannibalism in the New Zealand sea lion, *Phocarctos hookeri*. Marine Mammal Science 16: 494–500.

———, J. BURGESS, AND M. W. CAWTHORN. 2003. New Zealand sea lions and squid—Managing fisheries impacts on a threatened marine mammal. *In* Marine mammals: Fisheries, tourism and management issues, N. Gales, M. Hindell and R. Kirkwood (eds.), CSIRO Publishing, Melbourne, pp. 192–207.

———, P. J. DUIGNAN, A. CASTINEL, A. GRINBERG, B. L. CHILVERS, AND B. C. ROBERTSON. 2006. *Klebsiella pneumoniae* epidemics: Possible impacts on New Zealand sea lion recruitment. *In* Sea lions of the world, A. Trites, S. Atkinson, D. De Master, L. Fritz, T. Gelatt, L. Rea and K. Wynne (eds.), Alaska Sea Grant College Program, University of Alaska, Fairbanks, pp. 455–471.

WOODLEY, T. H., AND D. M. LAVIGNE. 1993. Potential effects of incidental mortalities on the Hooker's sea lions (*Phocarctos hookeri*) population. Aquatic Conservation of Marine and Freshwater Ecosystems 3: 139–148.

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