SEA OTTER MORTALITY IN ORCA INLET, PRINCE WILLIAM SOUND, ALASKA, WINTER 1995-1996

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February 2002

Technical
Report
MMM 02-1

MARINE MAMMALS MANAGEMENT
Fish and Wildlife Service
Region 7, Alaska
U.S. Department of the Interior

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USFWS Technical Report MMM 02-1

February 2002

TABLE OF CONTENTS

LIST OF TABLES ii

LIST OF FIGURES iii

ABSTRACT 1

INTRODUCTION 2

METHODS 4
- Carcass Surveys 4
- Age Determination 4
- Necropsies 5
- Population Surveys 5
- Weather Data 6
- Fisheries Data 6

RESULTS 6
- Carcass Surveys and Age Determinations 6
- Necropsies 7
- Population Surveys 7
- Weather Data 8
- Fisheries Data 8

DISCUSSION 8

ACKNOWLEDGMENTS 14

REFERENCES 14

APPENDIX A 23

APPENDIX B 25

APPENDIX C 26

List of Tables

Table 1. Information on whole sea otter carcasses (all male) collected from Orca Inlet beaches, PWS, Alaska 17

Table 2. Estimated sea otter numbers in Orca Inlet, Prince William Sound Alaska 1993-96 based on aerial surveys 18

List of Figures

Figure 1. Map of Orca Inlet, showing beaches surveyed by D. Monson and M. King for carcass recoveries. 19

Figure 2. Map of Orca Inlet, showing aerial survey areas 20

Figure 3. Distribution of ages at death for sea otters found dead on beaches in Orca Inlet, Prince William Sound, Alaska, during spring 1996. 21

Figure 4. Mean minum air temperatures recorded at Cordova airport for winter months,1975-1996. Error bars represent 95% confidence intervals 22

ABSTRACT

During the winter of 1995-96, residents of Cordova, Alaska, observed a relatively high number of sea otters dead or dying in the harbor and on local beaches, compared with previous winters. Unusual behaviors of sea otters (predation on seabirds, gnawing on pilings, eating fish offals from fish processing plant, and lethargic otters) were observed. Our objective was to evaluate sea otter mortality in Orca Inlet in the late winter of 1996 by examining (1) the demographic pattern of the die-off, using carcass surveys, (2) the physical condition, evidence of disease, and causes of death, by necropsy, (3) changes in abundance in sea otters using aerial surveys, and (4) contributing causes, including weather and adverse interactions with area fisheries. Sixty-eight sea otter carcasses were found on beaches in March and April of 1996. Nine carcasses were intact, and were collected for necropsy. Fifty-nine carcasses were partial, and only skulls were collected. Sex was determined for 54 of the carcasses: 41 (60%) were males, 13 (19%) were females, and 14 (21%) were of unknown sex. Prime age sea otters (2-8 years) comprised 56%, and older sea otters (>9 years) 38% of the sample. There was evidence that 5 of the examined otters had bullet holes in the skulls; there was no evidence of gun shot wounds on any of the other animals examined. Necropsies on the 9 male sea otters revealed that in all cases, the sea otters were emaciated, with starvation reported as a major cause of death. Very heavy parasite loads were observed in all otters and contributed to the deaths in some cases. Results of aerial surveys done in 1993 and 1996 do not provide evidence of a decline in the population of sea otters in Orca Inlet; however, standard errors were relatively large and changes could occur without being detected. The winter of 1995-96 was unusual, with extremely low precipitation, small snow pack, and the ground freezing to an unusual depth for the coast. No coincidental mortality of sea otters or other marine mammals was noted through commercial fishing activities. Carcass surveys were repeated during spring of 1997 in Orca Inlet with the same level of effort as 1996; 17 carcasses were observed. The lower carcass recovery in 1997 suggests elevated mortality rates in 1996 did not persist into the following year. Continued monitoring of beaches for carcass deposition in late winter and early spring may be useful as an indicator of mortality rates and general health of the population near Cordova.

Key words: *Enhydra lutris*, fish processing waste, mortality, parasites, sea otter

INTRODUCTION

Sea otters (*Enhydra lutris*) came close to extinction during the 18th and 19th centuries, when they were heavily hunted by European and American traders for their fur. A few remnant populations survived, and in 1911, they were placed under international protection from further hunting. Today, through growth of remnant populations and translocations to unoccupied habitat, sea otters are found throughout most of their original range including the waters around Cordova, Alaska (Rotterman and Simon-Jackson, 1988, Riedman and Estes 1990, Bodkin *et al.* 1994).

One remnant population was located in southwestern Prince William Sound (PWS), Alaska (Lensink 1962). In the 1970's, this population expanded to the north and east, including Orca Inlet in southeastern PWS. By 1980, sea otters had repopulated most areas of PWS (Garshelis and Garshelis 1984, Rotterman and Simon-Jackson 1988). In the last 20 years, Orca Inlet has supported a density of sea otters which is higher than observed in any other part of the sea otters' range (Monnett and Rotterman 1989). Orca Inlet is located adjacent to the community of Cordova, Alaska, in southeast PWS (Figure 1). Water depths range from intertidal bars to approximately 60 meters (m) in Nelson Bay. Most of the Inlet is shallow, less than 10 m in depth, with a soft sediment bottom. Orca Inlet, from Nelson Bay to Mummy Island, is approximately 30 kilometers (km) long and 3 km wide.

Seasonal movements of sea otters in Orca Inlet are understood as follows: males concentrate in the central and northern parts of the Inlet near Mud Bay at Cordova in the winter months while female sea otters concentrate in the southern parts of Orca Inlet, in the vicinity of Mummy Island and on the south side of Hawkins Island (Monnett and Rotterman 1989). We believe that the central Orca Inlet area, near Cordova, continues to be dominated by males throughout the year, although females are also abundant in southwestern Orca Inlet. The uncommonly high density of sea otters in Orca Inlet raises questions about the long-term sustainability of food resources in the area. Mortality events for sea otters in this region were documented in 1989 and recently during the winter of 1995-96.

Sea otter mortality events have been noted over the last few decades in Alaska. Kenyon (1969) discusses winter mortality at Amchitka Island in the Aleutians, an area of high sea otter density. Generally, mortality was greatest in late winter and early spring, and most of the dying animals were juveniles or aged sea otters in emaciated condition. Kenyon and colleagues concluded that a limited food supply and severe climate were the factors

contributing to the mortality. The estimated number of deaths (measured as carcasses on beaches) over a 25 year period (1937-1963) fluctuated considerably, with the carcass count in "high" mortality years estimated at more than 5 times the number in "low" mortality years. Another high mortality event for sea otters, in which both high animal densities and severe weather were contributing factors, was observed at the Commander Islands, Russia, in the winter of 1990-91 (Bodkin *et al.* 2000). Disease, in conjunction with factors such as severe weather, decreased food resources, and high animal densities, has also been identified as a potential cause of increased mortality. DeGange and Vacca (1989) documented a die-off of sea otters at Kodiak Island, Alaska, in 1987. The cause of the die-off is unknown; however, they postulated that a concurrent outbreak of paralytic shellfish poisoning may have contributed to the deaths.

During the winter of 1995-96, residents of Cordova, Alaska, observed what seemed to be a relatively high number of sea otters dead or dying on beaches in the area, compared with previous winters. There were also reports from residents of unusual behaviors of sea otters (predation on seabirds, gnawing on pilings, eating fish offals from fish processing plant, and lethargic otters hauled out), particularly in the harbor area. These observations generated concerns that sea otters were unhealthy and mortality rates elevated, relative to other years, and that as a consequence, the sea otter population in Orca Inlet might be substantially reduced. This report documents the efforts of the U.S. Fish and Wildlife Service (USFWS), in cooperation with Cordova residents, the Alaska Biological Science Center of the U.S. Geological Survey-Biological Resources Division (USGS-BRD), and the National Marine Fisheries Service-Enforcement (NMFS) in Cordova, Alaska, to evaluate the extent and causes of the sea otter deaths.

The objective of this report is to evaluate the apparent increased mortality of sea otters in Orca Inlet in the late winter of 1996 by examining (1) demographic patterns associated with the die-off, using carcass surveys, (2) necropsy of some dead animals for body condition, evidence of disease, and causes of death, (3) changes in population abundance estimates using aerial surveys, and (4) other potential contributing causes, including the severity of weather and adverse interactions with area fisheries.

METHODS

The study area included Orca Inlet, from Nelson Bay in the northeast to Mummy Island and the mouth of Boswell Bay in the south (Figure 1).

Carcass Surveys

In March and April 1996, a directed effort was made to recover intact, fresh carcasses for necropsies (discussed below), and skulls from scavenged or decomposing carcasses. Only carcasses of otters judged to have died during the winter of 1995-96 were included in the study. Teeth were obtained from skulls for estimation of ages at death. If possible, depending on condition of the carcass and skeletal remains, sex was determined.

On March 22-23, 1996, Mr. Daniel Monson of the Alaska Biological Science Center (ABSC) surveyed beaches from northeast of Cordova, in the vicinity of Orca Cannery, to the Hartney Bay area, southwest of Cordova (Figure 1). Four intact carcasses were recovered and were shipped to Anchorage for necropsy. Seventeen sea otter skulls were recovered and shipped to Anchorage for processing. Five of the skulls were cleaned by boiling until soft tissues could be removed from the bone.

During April 6-11, 1996, Mr. Mark King of Cordova surveyed beaches in the Cordova area, including the mouth of Boswell Bay, Mud Bay, Hartney Bay, Big Point, Boatyard Bay, and Orca Cannery (Figure 1). Several beaches searched by Mr. Monson in March were rechecked as part of this survey. Forty-two decomposed carcasses (unsuitable for necropsy) were found, from which skulls were collected and shipped to Anchorage for processing. Skulls were radiographed (concern was expressed by residents of Cordova that sea otters were being shot) to determine if bullet fragments were present.

Five additional carcasses were picked up on Cordova beaches in April by NMFS personnel, and shipped to Anchorage or Madison, Wisconsin for necropsy.

Age Determination

A first premolar tooth was pulled from each skull and carcass for age determination. Matson's Laboratory (Milltown, Montana) prepared the teeth and read the sections. Age of the animal was determined by counting cementum layers or annuli (rings) in the root of the

tooth; each annulus corresponds to a year (Garshelis 1984; Bodkin *et al.* 1997).

Necropsies

The nine carcasses recovered intact were in relatively fresh condition, and were necropsied to evaluate body condition, examine organ systems and, if possible, note abnormalities that may have contributed to death of the animal (Table 1).

Five carcasses were examined at the USFWS in Anchorage by Dr. Daniel Mulcahy, staff veterinarian with the ABSC. Three carcasses were shipped to the USGS-BRD National Wildlife Health Center (NWHC) in Madison, Wisconsin, where they were necropsied by Dr. Nancy Thomas, a veterinary pathologist. Facilities and expertise at the NWHC allowed for the additional examination of tissues, including bacteriological testing. One carcass was necropsied by USFWS biologists Angela Doroff and Carol Gorbics, following the procedures outlined in Doroff and Mulcahy (1997). All necropsies followed standard veterinary procedures, and similar baseline data were collected in all cases.

Tissue samples (including liver, kidney, stomach, intestine, heart, lung, and lymph nodes) from the carcasses were preserved in formalin for histopathology (microscopic examination of cells for abnormalities and evidence of disease). Samples from carcasses necropsied in Anchorage were submitted to Dr. Kathy Burek, a veterinary pathologist in Alaska, and to the Armed Forces Institute of Pathology in Washington, D.C. Histopathology on samples from carcasses necropsied at the NWHC was done by Dr. Thomas.

Population Surveys

Aerial surveys of Orca Inlet were conducted in the summers of 1993-1996 by the ABSC. Methods for the surveys were as described by Bodkin and Udevitz (1999) and are briefly summarized here. The survey area was divided into a series of strip transects, and a subset of these transects, representing about 20% of the total area, was randomly selected. Surveys were flown along this subset of transects. Sea otter density (otters/km^2) was estimated from the transect data, and applied to the full study area to obtain an estimate of the total population size for the area. A survey-specific correction factor was calculated and used to adjust the survey results to account for otters not detected. Surveys were conducted at high tides, from a fixed-wing tandem style aircraft (Piper Super Cub 1993 and a Bellanca Scout 1994-96), flown at an altitude of 91 m (300 feet) and at an air speed of 29 m/s (65 mph). The

area covered in the survey is shown in Figure 2.

Weather Data

Weather data for Cordova were obtained from an archive maintained by the Utah Climate Center at Utah State University (http:\\climate.usu.edu). Temperature data from the Cordova Airport reporting station (ID# 00 040450217702), located 13 miles east of Cordova, were used, as this station had the most complete records for the period of interest. For months November and December 1975-94, and January, February and March, 1976-95, the daily minimum temperatures reported at the airport were averaged to give monthly mean minimum temperatures. The monthly mean minimum values were averaged to obtain a "20 year" mean minimum temperature for each month (November-March), and 95% confidence intervals around the means were computed. The monthly mean minimum temperatures for November 1995 through March 1996 were compared to the 20 year mean and confidence interval for that month. Wind data for a comparable period were not available at the Utah State University or other internet archives.

Fisheries Data

Data on area fisheries in 1995 and 1996 were obtained from the Alaska Department of Fish and Game, Commercial Fisheries Division.

RESULTS

Carcass Surveys and Age Determinations

Sixty-eight sea otter carcasses were found on beaches in March and April of 1996. Nine carcasses were intact, and were collected for necropsy to determine body condition, cause of death, age and sex. Fifty-nine carcasses were partial, and only skulls were collected. Sex was determined for 54 of the carcasses: 41 (60%) were males, 13 (19%) were females, and 14 (21%) were of unknown sex.

Age was estimated from a tooth for 64 carcasses, and the distribution of ages is shown in Figure 3. Juveniles, defined as sea otters of 0 to 1 year of age, comprised only 6% of the animals (all were 1; no 0 age animals were found). Prime age sea otters, defined as those from 2 to 8 years of age, comprised 56%, and older sea otters (those aged 9 and above) 38%

of the sample. The juvenile, prime and older age-classes were based on Monson and Ballachey (1995).

Radiographs of the skulls recovered by M. King showed no evidence of lead fragments, which would have indicated that mortality was due to gunshot. However, upon cleaning, three of five skulls collected by D. Monson were found to have bullet holes in the skull. The remaining skulls collected by D. Monson were not cleaned, nor were they radiographed.

Necropsies

Necropsies were done on 9 sea otters, all males. Necropsy reports and associated data sheets for several of these otters are provided in Appendix C; weights, lengths and estimated ages of the necropsied sea otters are presented in Table 1. In all cases, the sea otters were emaciated, with starvation reported as the major cause of death. Very heavy parasite loads were observed in all otters. In some cases, parasites contributed to the deaths by perforating the gastrointestinal tract, leading to peritoneal infections, and in other cases by causing intestinal intussusceptions (i.e., prolapse of one part of the intestine into an adjoining segment).

The helminthe parasite infestations observed in the gastrointestinal tracts of the otters included acanthocephalans (thorny-headed worms) and cestodes (tapeworms), primarily in the small intestine, and trematodes (flukes) in the gall bladder, all of which have commonly been observed in previous necropsies of sea otters from PWS and other areas of Alaska (USFWS unpublished data). An additional nematode (roundworm) was found in all carcasses examined, primarily in the stomach but also in the intestine. This nematode was identified by Dr. C. Gardiner, a veterinary parasitologist at the Armed Forces Institute of Pathology in Washington, D.C., as Anisakis sp., and further by Dr. Michael Kinsella, a veterinary parasitologist at the NWHC, as *Pseudoterranova decipiens*, an anisakid nematode. This particular parasite was reported twice previously in sea otters from PWS (Kenyon 1969; Margolis *et al.* 1997).

Population Surveys

The results of the population surveys for 1993 and 1996 are shown in Table 2. No evidence of a decline in sea otter abundance during this period can be detected from these results.

Weather Data

Mean monthly minimum temperatures (°F, 20 year means, November-March), from the Cordova Airport reporting station, and the confidence intervals around those means are presented in Figure 4. Monthly average minimum temperatures are presented in Appendix B. Bar graphs on Figure 4 show the mean minimum temperature for each month in the winter of 1995-96. The mean monthly minimum for January 1996 was 1.74°F, which is below the lower bound of the confidence interval for the January 20 year mean. For the other months (November and December 1995, February and March 1996), the mean monthly minimum temperature fell within the confidence interval for each 20 year mean. However, December 1995 was the only month in the winter 1995-96 for which the mean minimum temperature was not relatively low, compared to the average value for the previous 20 years. Wind speed data encompassing a several year period, including the winter of 1995-96, were not available.

Fisheries Data

The following fisheries typically operate in the study area but were closed during the winter of 1995-96: Alaska Roe Herring, Alaska Food/Bait Herring, Eastern District PWS Purse Seine, Copper River Drift Gillnet Salmon, and Dungeness Pot Fishery. The Groundfish Longline or Pot Fishery was open during this period, but less than five boats were believed to have participated during the winter 1995-96 (J. Brady, Alaska Department of Fish and Game, personal communication).

DISCUSSION

Variations or declines in the abundance of sea otters may be caused by a variety of factors, including altered or declining food resources, loss of habitat, adverse weather conditions, and disease. Any one or a combination of these factors could increase mortality rates in a wild population; effects could be either short-term or persistent. For wild animal populations in general, it is thought that as densities increase and food resources decrease, mortality rates may increase, particularly for very young, older, and sick or otherwise injured animals. This pattern has been observed in sea otters and other large mammal populations (Kenyon 1969; Caughley 1977; Emlen 1970).

Residents of Cordova reported increased numbers of dead sea otters on area beaches, relative to previous years, in the winter of 1995-96. Several dead and dying animals were reported to NMFS and USFWS offices. Local residents also reported aberrant behaviors of sea otters. More otters seemed to be present in the harbor area than in previous years. Otters hauled out in the harbor area and on beaches close to town were described as lethargic and disoriented, with uncommonly long haul-out times. Sea otters were seen preying on seabirds on at least one occasion (Stephen Bodnar, personal communication), which has been reported previously (Kenyon 1969, Riedman and Estes 1990, Harding 1994) but is unusual. Kenyon (1969) suggested that "...the flesh of birds is eaten only under the stress of hunger, and particularly in winter." Sea otters also were observed eating chunks of fish, which were by-products from the fish processing plant just outside the harbor area (M. King, personal communication).

No coincidental mortality of other marine or coastal vertebrates were reported. No indication of other perturbations in the sea otters' habitat, such as increased fishing or unusual pollution events, were observed. Because of the lack of the fishing effort during this time period, there is no evidence to suspect increased mortality of sea otters was due to entanglement in fishing gear and subsequent drowning. There was no evidence of paralytic shellfish poisoning, based on human consumption of shellfish.

At necropsy, sea otters were judged to be in very poor body condition. Starvation and intestinal disorders associated with the helminth parasite infestations were judged to be the ultimate causes of death. Parasite loads were generally much heavier than seen in previous necropsies, including those of otters from eastern and western PWS, with the most severe pathologies associated with nematodes, and also heavy cestode infections, causing intussusceptions of the GI tract.

Gastric nematodes identified as *Pseudoterranova decipiens*, an Anisakid nematode, were found in the stomachs of all otters necropsied. *P. decipiens* were previously identified as a parasite of sea otters by Rausch (1953)[1], in a survey of helminth infestations of sea otters dying at Amchitka Island in late winter 1951. Rausch described the nematode as frequently highly pathogenic, especially in the larval stage, and associated with intestinal perforations. Kenyon (1969), however, notes the occurrence of *P. decipiens* in apparently healthy adult sea

[1]Rausch (1953) actually identifies the nematode as *Porrocaecum decipiens*,and Kenyon (1969) as *Terranova decipiens*; both are equal to *Pseudoterranova decipiens* using current nomenclature (N. Thomas, NWHC, pers. comm.).

otters killed at Amchitka during 1955-63, suggesting that infestation with this parasite is not necessarily life-threatening. Anisakid nematodes including *P. decipiens* are commonly found in cetaceans and pinnipeds (Rausch 1953, Margolis and Dailey 1972) and although the life cycle is not well understood, fish, including salmon and herring, are known to serve as an intermediate host. Sea otters at Amchitka frequently prey on fish (Rausch 1953, Kenyon 1969), providing a likely route of nematode infection.

Pseudoterranova decipiens have rarely been observed in necropsies of sea otters from PWS (USFWS unpublished data). However, they were reported in 1962, in a necropsy of a subadult sea otter from Patton Bay on Montague Island (Kenyon 1969, p. 273) and again in 1989, in a necropsy of a sea otter collected dead after the *Exxon Valdez* oil spill (Margolis et al. 1997). Presumably, the apparent scarcity of anisakid nematodes in PWS sea otters may may be due to the fact that sea otters there generally prey primarily on invertebrate species (Calkins 1978, Garshelis et al. 1986, Doroff and Bodkin 1994; USFWS & USGS-BRD unpublished data).

Sea otters in the vicinity of Cordova which foraged on chunks of fish from the processing plant (and perhaps live fish as well) in the winter of 1995-96 could certainly have been exposed to *P. decipiens*. If these otters were already in poor body condition, having endured a relatively cold winter, their ability to resist parasitic infections may have been impaired, particularly if exposure was continual by repeated foraging on fish. Thus they may have been at high risk for heavy helminth parasite infections (*P. decipiens* and several other species), which subsequently became a major factor in their mortality.

Comparable body condition data (weights, lengths) for nine male sea otters taken by a hunter in Valdez Arm in early April 1994 (USFWS unpublished data) suggest that the poor condition of the Orca Inlet otters recovered as carcasses in 1996 may not be markedly lower than that of live male otters in late winter. The average weight of sea otters from Valdez Arm was only slightly higher (mean, SD = 20.8±4.1 kg for Valdez vs 19.8±4.6 kg for Orca Inlet), and average length was slightly less (mean, SD=126±12 cm for Valdez vs 132±12 cm for Orca Inlet). No parasites (n=5) or few to moderate parasites (n=4) were found in the Valdez Arm group. Additional otters harvested in Valdez Arm in 1996 were reported to have either no (n=4), few (n=6) or moderate (n=2) levels of parasitic infection (USFWS unpublished data). Apparently, otters can be in relatively poor body condition and survive, whereas animals in poor body condition with heavy parasite loads are at higher risk of death. However, comparisons across different areas must be viewed with caution, and

unfortunately, we have no information on body condition and parasite loads of live male otters in the Orca Inlet area during the same time period.

There was no evidence by radiograph that any of the 42 sea otter skulls collected in April had fragments of metal in the bone and none of the nine animals necropsied had evidence of having been gunshot[2]. However, five of the skulls collected by D. Monson in March were cleaned and three of the five had what appeared to be bullet holes in the skull. This suggests that some proportion of the deaths were attributed to gunshot.

Carcass recoveries in western PWS and in other parts of the sea otters' range have shown that ages at death can provide useful information on the "normalcy" of mortality patterns (Monson 1995, Monson and Ballachey 1996), and that, for an area with breeding animals (females and territorial males), the proportion of "prime-age" animals (defined as ages 2-8 years) in the sample of recovered carcasses generally was less than 20%. Elevation of the proportion of prime-age animals, relative to juveniles and older ages, suggests abnormal mortality. However, the carcasses recovered in 1996 were predominately of males, presumably from the male group in Orca Inlet. The age structure of a male group very likely differs from that of a breeding population. For example, it might be expected that there would be no pups of the year and few juveniles (1 year of age) in a male group, and that the number of older animals in a male group would be proportionately fewer, as females generally are recognized to live longer than males. Additionally, older males (perhaps ages 5+) may be less common in a male group because they are spending more time in breeding areas. Thus, the age distribution obtained for the carcasses from Orca Inlet in 1996 (6% 0-1, 57% 2-8, and 39% 9+ year olds; n=64), which at first consideration may appear to have a relatively large percentage of prime-age otters, in fact may be "normal" for that population. Further, many of the prime-age category were 8 year olds (Figure 3), by which age mortality rates may start to be higher than for younger individuals. Until we have more years of carcass collection data, and a better understanding of distribution of ages-at-death for otters living in a male area, we cannot judge whether or not the 1996 age data suggest abnormal mortality for Orca Inlet.

Overwinter mortality of sea otters may be exacerbated by harsh storm events and cold temperatures. Kenyon (1969), in the Aleutian Islands, observed greater numbers of carcasses

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[2] One otter (BS96001) was euthanised by gunshot on the beach due to its morbid condition.

on beaches following major storms, and concluded that strong winds limited the ability of otters to forage. We were unable to locate wind data for the Cordova area over a long-term (several years or more) period, to allow comparisons with the winter of 1995-96. Lack of information on wind is limiting because if there is a relationship between weather conditions and mortality, wind speeds may be a more important factor than temperature. However, a summary of temperature data for Cordova from 1976-95 indicates that January 1996 was particularly cold, with the mean minimum temperature for that month falling below the confidence interval for the 20 year (1976-95) mean minimum January temperatures. Furthermore, the mean minimum temperatures for November 1995 and February and March 1996 were relatively low, compared to means for the previous two decades, although they do not fall outside the confidence intervals computed for those months. According to Stephen Bodnar, a scientist with the Prince William Sound Science Center in Cordova, the winter of 1995-96 was unusual, with extremely low precipitation, small snowpack, and the ground freezing to an unusual depth for the coast (S. Bodnar, personal communication). Perhaps a period of severe cold in January, plus generally cold conditions in the other months that winter, significantly worsened conditions for sea otters and contributed to the subsequent mortality in March and April.

Results of aerial surveys done in 1993 and 1996 (Table 2) do not provide evidence of a decline in the population of sea otters in Orca Inlet. However, standard errors from the surveys are relatively large, and changes could have occurred without being detected. We conclude that further survey effort focusing on Orca Inlet specifically (using the same methods with replication) would give a tighter estimate of the Orca Inlet area.

In the spring of 1997, Mr. Mark King of Cordova repeated the carcass survey in the Orca Inlet area, with approximately the same level of effort as expended in 1996. He recovered 14 carcasses, and observed 3 more carcasses floating in the water. This number (17 total) is lower than the 68 carcasses recovered a year previously, and supports the observation that mortality in the previous winter (1995-96) was relatively high. The lower carcass recovery in 1997 strongly suggests that apparent elevation of mortality rates in 1996 did not persist into the following year. Continued monitoring of carcass prevalence on Cordova area beaches in late winter may be useful as an indicator of mortality rates and general health of the population.

Sea otters have reoccupied the Orca Inlet area for less than two decades. We can speculate that the high densities of sea otters presently residing there exceed carrying capacity and that

as a consequence, invertebrate prey resources are being depleted. Consumption of fish and seabirds and apparent relatively high mortality of otters in the winter of 1995-96 may support this concept, but certainly does not confirm it, particularly in consideration of the relatively cold temperatures that winter, and involvement of parasitic infections in their deaths. The fact that the surveys did not show a decline in otter numbers in the summer of 1996, and that there was no report of a large mortality in the following winter (1996-97), alleviate concerns over a major and continuing die-off and consequent reduction in numbers of sea otters in Orca Inlet.

It is not possible to definitively state the cause of the observed high number of dead and moribund animals on the beaches near Cordova during the winter of 1995-96. However, assuming that the carcasses examined were representative of the dying population, heavy helminth parasite infections appear to be a major factor in the mortality. Parasites, particularly *P. decipiens*, may have been present at greater than normal levels because of consumption of fish parts by otters in the Cordova harbor area. Whether fish parts were consumed because other prey were becoming depleted or simply because they offered an easily retrieved alternative is not known. Additionally, temperatures during that winter, especially during January, were relatively cold; this may have imposed a further stress on the otters and contributed to their poor condition and demise. No declining trend or large scale die-off could be detected from the aerial survey results, however, given the precision of the surveys, a die-off in the winter of 1995-96 could have gone undetected. Similar patterns of mortality were not evident in 1997, based on a lower number of carcasses recovered in late winter 1997, and lack of reports or observations from local residents of abnormal sea otter behavior or mortality. Concern over a possible large-scale population decline in the Cordova area has abated; however, continued monitoring of the population is advisable.

We recommend the following for continued monitoring of the sea otter population in Orca Inlet, 1) annual carcass surveys should continue to monitor patterns of mortality in the Cordova area,
2) results of necropsies of hunter killed animals should be monitored to evaluate body condition and parasite infestations in healthy animals and, 3) periodic surveys to monitor trends in population abundance and demographics should be continued.

ACKNOWLEDGMENTS

We thank Mark King, Daniel Monson, Mark Kirkland and the NMFS staff in Cordova for their detailed reporting and willing recovery of sea otter carcasses for examination; Drs. Daniel Mulcahy and Nancy Thomas for sea otter necropsies; Drs. Kathy Burek and Tom Lipscomb for histopathology; and Dr. Pam Tuomi for radiographs of skulls. Jim Bodkin and George Esslinger conducted aerial surveys of the sea otters. Stephen Bodnar provided assistance in locating Cordova weather records. Mary Cody, Doug Burn, and Mary Whalen provided assistance in preparation of data and figures for the report. We thank George Esslinger and Linda Comerci for review of the draft report. This work could not have been completed without the support of the Alaska Sea Otter Commission; U.S. Fish and Wildlife Service; USGS-BRD; and the Armed Forces Institute of Pathology.

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Table 1. Information on whole sea otter carcasses (all male) collected from Orca Inlet beaches, PWS, Alaska.

Otter Number	Collection Date	Necropsy Date	Estimated Age	Weight (kg)	Length (cm)	Wt/Length Ratio
BS96001	6 April 96	11 April 96	Adult	-	130	-
BS96002	3 April 96	5 April 96	8	22.3	140	0.159
BS96003	22 March 96	28 March 96	10	23.6	140	0.169
BS96004	22 March 96	28 March 96	10	24.5	142	0.173
BS96005	22 March 96	28 March 96	3	20.5	138	0.149
BS96006	23 March 96	26 March 96	4	20.5	127	0.161
BS96007	11 April 96	15 April 1996	1	10.3	110	0.094
BS96008	11 April 96	15 April 1996	1	16.1	115	0.140
BS96010	16 April 96	24 May 96	5	20.4	139	0.150

Table 2. Estimated sea otter numbers in Orca Inlet, Prince William Sound Alaska 1993-96 based on aerial surveys.

Month/Year	Population Estimate	Standard Error	Coefficient of Variation
August 1993	1,936	592	0.30
August 1994	5,260	1,956	0.37
March 1996	1,514	393	0.26
August 1996	2,239	852	0.38

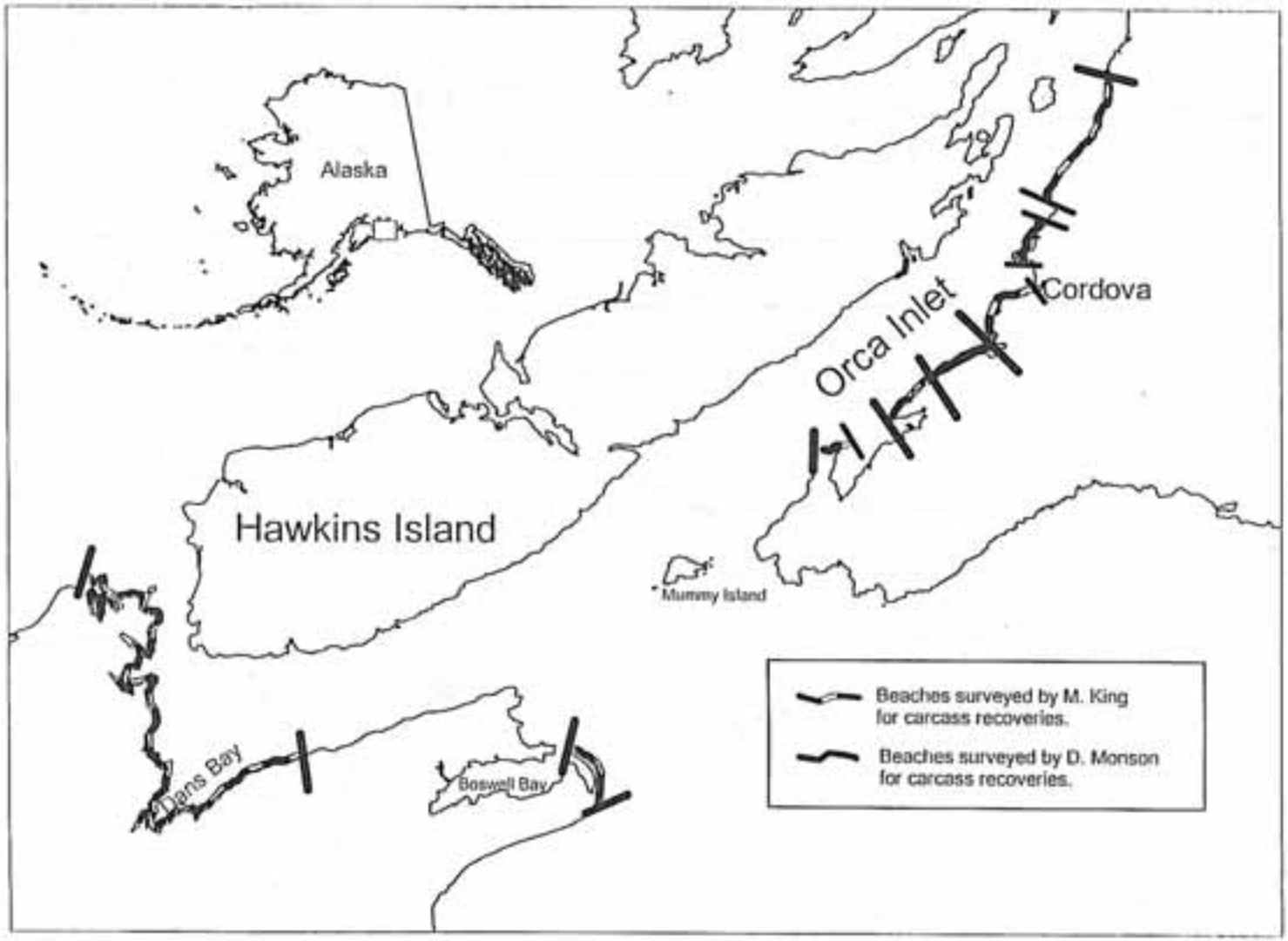

Figure 1. Study area depicting shoreline areas searched for sea otter carcasses, March and April 1986.

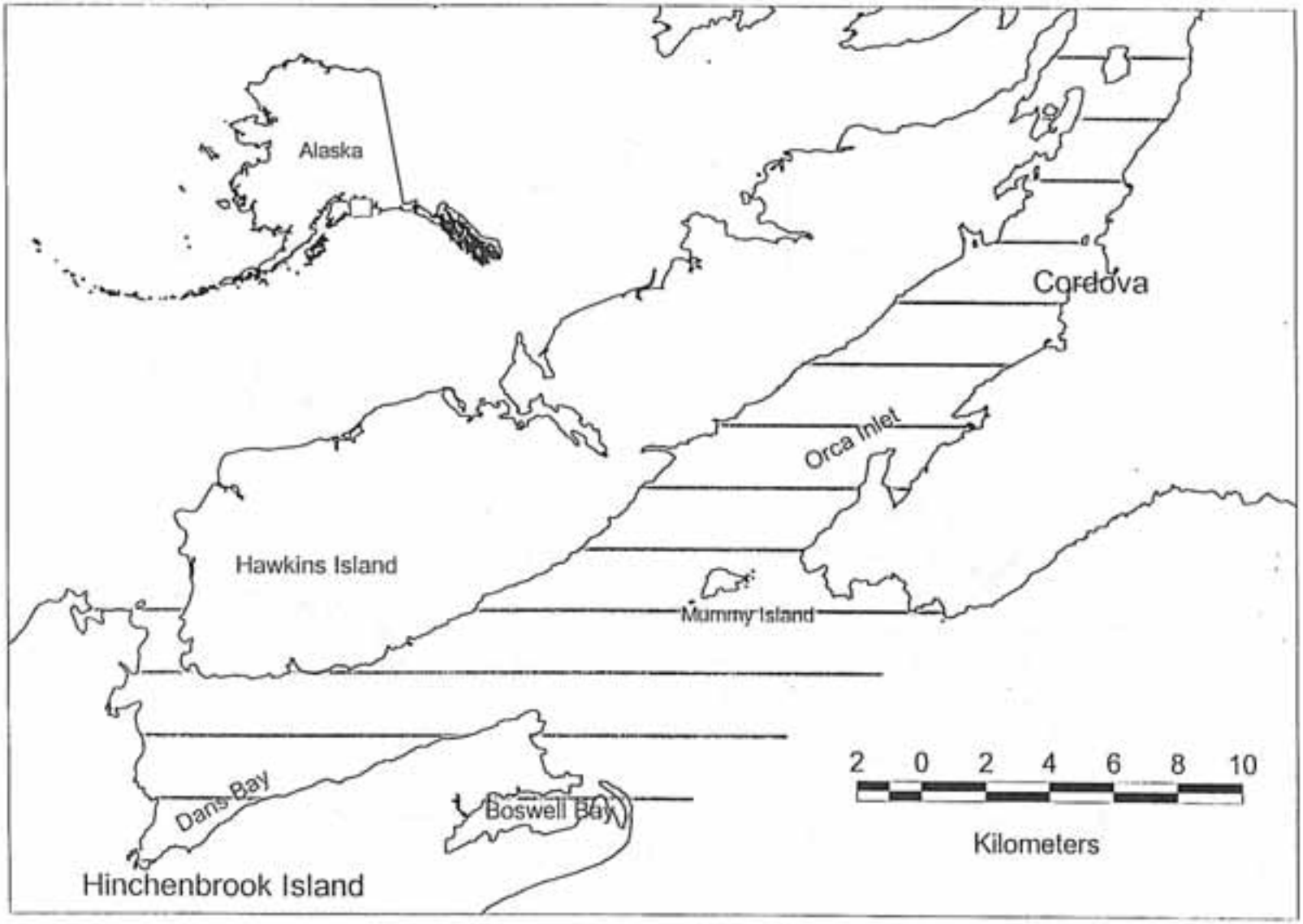

Figure 2. Map of Orca Inlet showing transects sampled during 1996 aerial surveys.

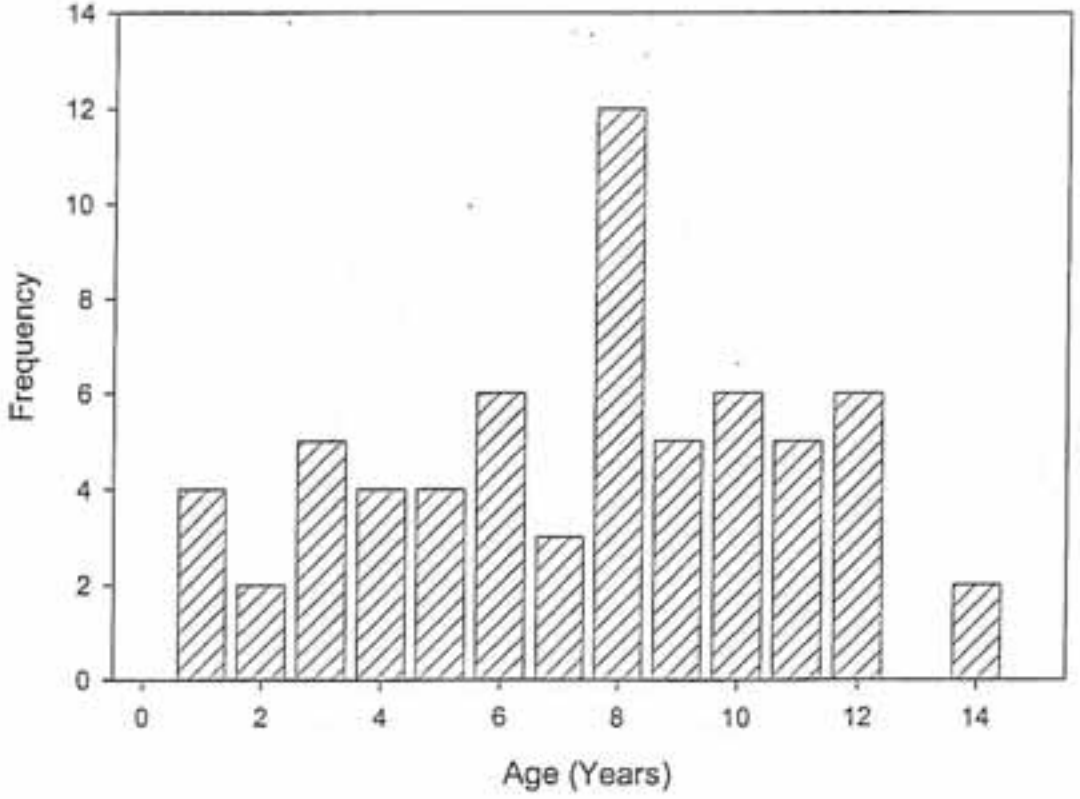

Figure 3. Distribution of ages at death for sea otters found dead on beaches in Orca Inlet, Prince William Sound, Alaska, during spring 1996.

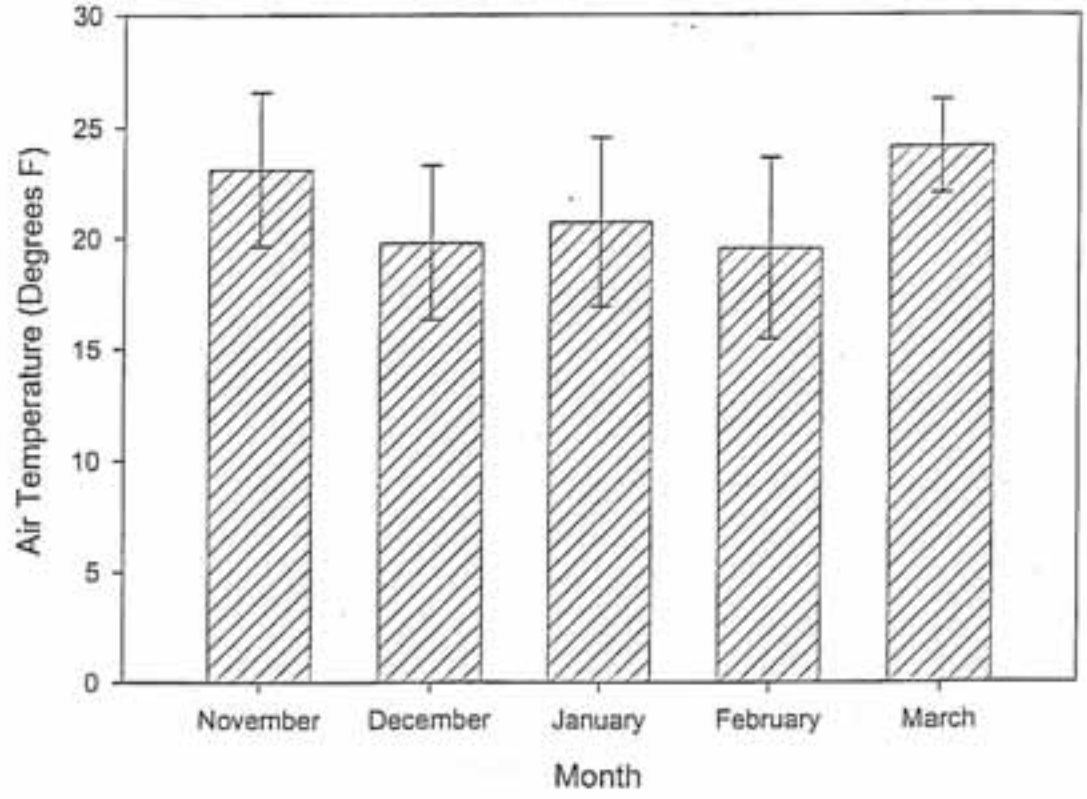

Figure 4. Mean minumum air temperatures recorded at Cordova airport for winter months,1975-1996. Error bars represent 95% confidence interval.

APPENDIX A. Ages determined from sea otter teeth of beachfound or necropsied animals.

Otter Number	Estimated Age (years)	Age Certainty (years)
BS96002	8	± 1
BS96003	10	± 1
BS96004	3	± 1
BS96005	3	± 1
BS96006	4	± 1
BS96007	1	± 0
BS96008	1	± 0
BS96010	5	± 0
CD96025	12	± 1
CD96026	11	± 2
CD96027	8	± 2
CD96028	12	± 1
CD96030	10	± 1
CD96031	8	± 2
CD96032	12	± 1
CD96033	3	± 1
CD96034	8	± 2
CD96035	2	± 0
CD96036	9	± 1
CD96037	7	± 1
CD96038	7	± 1
CD96039	4	± 0
CD96040	5	± 0
CD96041	7	± 1
CD96042	11	± 1
CD96043	10	± 1
CD96044	1	± 1
CD96045	5	± 1
CD96046	8	± 2
CD96047	8	± 1
CD96048	5	± 1
CD96049	6	± 1

CD96050	2	± 0
CD96051	3	± 0
CD96052	8	± 1
CD96053	8	± 2
CD96054	6	± 1
CD96055	9	± 1
CD96056	14	± 1
CD96057	4	± 1
CD96058	11	± 1
CD96059	14	± 1
CD96060	3	± 1
CD96061	8	± 1
CD96063	3	± 1
CD96064	8	± 1
CD96065	6	± 1
CD96066	6	± 1
COD9603	12	± 1
COD9604	4	± 1
COD9605	5	± 1
COD9606	9	± 1
COD9607	8	± 1
COD9608	6	± 1
COD9609	12	± 1
COD9610	11	± 1
COD9611	10	± 1
COD9612	9	± 1
COD9613	1	± 0
COD9614	12	± 1
COD9615	8	± 1
COD9616	11	± 1
COD9617	9	± 1
COD9618	10	± 1
COD9619	6	± 1

APPENDIX B. Monthly average minimum temperatures (°F) recorded at the Cordova airport weather station, November-March 1975-1996.

Year	November	December	January	February	March
1975/76	16.7	11.7	15.2	9.0	22.2
1976/77	31.1	31.1	24.1	33.9	23.7
1977/78	14.6	8.6	21.6	29.6	27.8
1978/79	23.5	17.1	14.7	-1.6	28.9
1979/80	30.7	13.6	12.0	28.7	26.9
1980/81	32.5	3.5	33.6	24.7	31.5
1981/82	23.8	17.2	7.4	16.4	20.4
1982/83	23.2	25.4	19.8	24.4	24.6
1983/84	29.1	17.0	24.7	26.4	29.9
1984/85	19.6	19.4	31.4	12.9	22.9
1985/86	4.9	30.0	28.6	19.5	17.4
1986/87	23.8	33.6	27.1	28.8	20.8
1987/88	32.1	19.1	19.8	27.0	30.1
1988/89	22.8	22.6	5.9	6.3	15.9
1989/90	19.2	29.0	15.8	6.7	24.4
1990/91	10.0	15.7	-	24.5	18.8
1991/92	28.6	22.7	26.0	18.0	25.1
1992/93	27.6	11.6	12.1	22.2	26.1
1993/94	27.8	26.5	22.7	13.0	28.3
1994/95	16.1	20.8	20.0	19.5	15.9
1995/96	20.4	20.2	1.7	17.5	22.8

UNITED STATES DEPARTMENT OF THE INTERIOR

NATIONAL BIOLOGICAL SERVICE

Alaska Science Center
1011 E. Tudor Road
Anchorage, AK 99503-6199
01 April 1996

A. Doroff, Wildlife Biologist
U.S. Fish and Wildlife Service
1011 East Tudor Road
Anchorage, AK 99503

Dear Angie:

Attached are copies of preliminary necropsy reports from the last three sea otters we examined. Histopathology samples from the first animal we did (BS 96002?) were submitted to Dr. Burek for histopathology. Let me know whether you wish the remaining samples from the other animals to be submitted.

Upon consideration of this series of necropsy, and with the caution that Dr. Burek's report may change this interpretation, I am concluding that the ultimate cause of death is starvation. There are varying proximal causes of death (peritonitis, colitis, heavy parasitism), but, at this point, I feel they are the result of severe debilitation.

The value of having more than one carcass from the same event, and those in very good condition, cannot be overemphasized in extending our conclusions from the death of a single animal to the dynamics of a subpopulation. Because several of these animals had discrete proximal causes of death, had we examined only one, we might have dismissed this event as having no meaning to the subpopulation.

I am concerned about the heavy infestations of the nematode parasites (probably *Porrocaecum decipiens*) in the gastrointestinal tracts of all of these animals. I have not seen this parasite before, in the hundreds of carcasses examined from the oil spill. I wonder if this infestation is limited to eastern Prince William Sound (not likely), or whether it itself represents an epizootic, possibly the major cause of debilitation and deaths of these animals? Alternatively, the lessened immune response of starving animals may let a normal infection become worse, or starving animals may be shifting their foraging to previously untapped resources, thereby becoming exposed to the intermediate stages of this parasite.

Thanks again for the opportunity and the loan of your considerable necropsy skills.

Sincerely,

Daniel M. Mulcahy, Ph.D., D.V.M.
Wildlife Veterinarian

cc: Ballachey

USGS-BIOLOGICAL RESOURCES DIVISION
NATIONAL WILDLIFE HEALTH CENTER
6006 Schroeder Road
Madison, Wisconsin 53711-6223
608-271-4640 (FAX 608-264-5431)

DIAGNOSTIC SERVICES CASE REPORT

Case: #14189, 001 Epizoo: RHT: LCG

Submitter:
Angie Doroff
USFWS - Region 7
Marine Mammals Management
1011 East Tudor Road
Anchorage, AK 99503

Specimen description/identification:
Sea Otter, Reference # BS96001.

Date Submitted: 04/10/96

Location: Prince William Sound, Valdez-Cordova Burrough, AK.

General Diagnosis: Subacute gunshot wound, left shoulder
Emaciation
Verminous gastritis and peritonitis; heavy gastrointestinal parasitism

Comments: The necropsy findings in this otter included multiple debilitating conditions. A gunshot wound had fractured the left humeral head. Nematodes were causing ulcers and abscesses in the stomach and were associated with localized peritonitis at sites where they lay in the abdominal cavity. Other intestinal parasites (cestodes, acanthocephalans and trematodes) were also found in great numbers. These conditions would have at least contributed to the animal's emaciated condition. Frozen skeletal muscle will be returned to Carol Gorbics in the near future.

__X__ Final Report (08/26/97)
date

__X__ See attached necropsy records for individual specimen observations.

N. J. Thomas

Pathologist: Nancy J. Thomas, DVM, MS
Diplomate, American College of Veterinary Pathologists

Linda C. Glaser DVM

If you have questions regarding this case, contact: Linda C. Glaser, DVM at 608-271-4640. Include above Case Number. Diagnostic findings may not be used for publication without the pathologist's knowledge and consent.

NATIONAL WILDLIFE HEALTH CENTER
NECROPSY REPORT

Submitter's Name, Affiliation Address
Angie Doroff
USFWS - Region 7
Marine Mammals Management
1011 East Tudor Road
Anchorage, AK 99503

LEGAL No
Case: 14189
Accession: 001
Collected: 04/06/96
Exam Date: 04/11/96
Pathologist: N. Thomas
Prosector: N. Thomas

Species: Sea Otter Specimen: Carcass nit
Bandtype: (E) Ref/Band No: (BS96001) Euth: (N) Weight (Gm): (12.5 kg)
History Summary: This animal was found dead near Cordova, AK in Prince William Sound.

EXTERNAL/INTERNAL OBSERVATIONS - LABORATORY RESULTS

External: The corneas are mildly cloudy and mildly sunken. The teeth are all present and in good condition. There is minimal wear on the canine teeth, premolars and molars. The gingiva has receded and food accumulated between right mandibular premolar 3 and molar 1. There are thin, white, coalescing streaks and plaques on the ventral aspect of the right base of the tongue. The body and abdomen are narrow. The dorsal vertebral processes and bony prominences of the hips and shoulders are all protruding. Black moist feces soil the anus. Right front digit 4 is lacerated at the margin of the hair and there is no evidence of hemorrhage or healing at this site.

Internal: There is serous atrophy of subcutaneous fat. Moderate diffuse to ecchymotic hemorrhage is present in the subcutaneous tissue and between muscle planes over the manubrium, the caudal ventral cervical region, left shoulder and over the lateral aspect of the left scapula. There is moderate diffuse intramuscular and subcutaneous edema surrounding the left shoulder joint. The base of the left humeral head has multiple small fractures and fracture lines evident near the greater tubercle. Small fragments of brown and gray discolored necrotic bone lie free or are attached to the joint capsule within the joint. The exposed humeral bone is partially fragmented and discolored brown with minute gray foci. The submandibular lymph nodes are flat and discoid. The prescapular lymph nodes are small. The axillary and inguinal nodes are rounded but small, approximately 2 cm in diameter. There is no abdominal or coronary fat. A moderate number of thin nematodes are lying on the peritoneum. Several are clustered on the omentum while single nematodes are found on the intestinal serosa or the abdominal wall. Catarrhal green exudate in clumps up to 1 cm in diameter is adhering to the omentum near the parasites. The peritoneal cavity contains scant cloudy brown fluid and has clumps of soft fibrin lightly adhering to the abdominal wall. Red-brown mucoid fluid coats the organs in the right upper quadrant. The mesenteric lymph nodes are diffusely gray and soft (autolysis), but they are large and plump. One mesenteric node is 2.5 cm in diameter and 7 cm long. The gall bladder and all bile ducts are severely dilated, but thin walled and mildly flaccid. The gall bladder is approximately 10 cm in length. The bile is clear, watery, and orange. There is severe mediastinal emphysema. The costochondral junctions of the ribs are mildly thickened and abruptly angular in a progression of severity from rib 8 caudally. The spleen is red-brown and contracted. The urinary bladder is distended with clear straw-colored urine. The testis are narrow and have mildly firm parenchyma; the left measures 40 x 12 mm and the right 37 x 13 mm. There is scant white foam in the trachea. The tracheobronchial lymph nodes are small. The right caudal lung lobe is heavy, red, and wet. There are occasional small pale foci beneath the capsule (probable air bubbles). All lung lobes have a uniform smooth texture. Several large ascarid-like nematodes are found in the lower esophagus near the cardia of the stomach. The stomach contains cloudy brown-red fluid and has numerous large nematodes lying free in the lumen and many more small thin nematodes adhering to the mucosa. The small worms are in tight clusters and the mucosa has minute pale foci at their adhesion sites. A few large and small nematodes are also found free in the upper duodenum. In the middle and lower duodenum the lumen is distended by numerous broad cestodes and a moderate number of small, thin-walled acanthocephalan parasites are lightly attached to the mucosa. In the lower duodenum and upper jejunum small acanthocephalans are numerous so that 2 to 5 are attached to the mucosa per square centimeter. The upper to middle jejunum has a doughy texture and is heavy in association with severe parasitism. The cestodes decrease in frequency and are no longer present by the middle jejunum. The small acanthocephalans also

progressively decrease in number through the tract and only rarely occur in the ileum or colon. Rarely a small thin nematode is found in the lower intestine. Intestinal contents other than parasites are scant and no food items are found. The lower jejunum and colon contain dark red-brown tarry fluid. A rare streak of hemorrhage is found in the lower small intestinal wall. No lesions are seen in the brain, pituitary gland, hyoid bones, thyroid glands, esophagus, trachea, pleura, diaphragm, heart, pericardial sac, heart valves, tracheobronchial lymph nodes, pancreas, kidneys, ureters, urinary bladder, adrenal glands, intestinal mucosa, right shoulder, bilateral elbow, hip and stifle joints, or testes. The total length is 139 cm. Tooth condition is 2. The grizzle is only present within 2 cm of the nose.

Preliminary Diagnosis: Acute to subacute fracture of left humeral head; Exam Type: (GO)
emaciation; mild fibrinous peritonitis associated with nematode parasitism; severe gastrointestinal parasitism.
Sex (M) Age (A)/(__) Body Cond. (P) Postmortem State (F) Giz. Lead (0)/(0)

Necropsy comments:

Laboratory Tests/Samples Saved:

1. VIROLOGY: Tongue with mucosa.

2. PARASITOLOGY: Omentum, parasites from the peritoneal cavity, parasites from the stomach, GI tract, gall bladder, nasal flush, brain.

3. BACTERIOLOGY: Peritoneal swab, left humeral swab, lung, omentum.
   Results: Lung = *Streptococcus equisimilus* and group G
   Enterococcus faecium
   Vibrio parahaemolyticus E26509
   Pasteurella multocida E22301
   Omentum = *Escherichia coli* E15901
   Peritoneal swab = *Escherichia coli* E15901
   Enterococcus faeciem
   Streptococcus fecalis E25016
   Shoulder joint = *Streptococcus equisimilis* and group G

4. HISTOPATHOLOGY: Several tissues.

5. Frozen for Tox: Brain, liver, kidney, skeletal muscle.

6. Saved frozen return to submitter: Upper premolar.

7. Photographs taken.

8. Carcass is incinerated.

RADIOGRAPH: A radiograph of the proximal left humerus shows an irregular fracture line through the greater tubercle, and numerous small chip fractures and bone loss in the humeral head near its junction with the greater tubercle. Numerous small irregular, intensely radiopaque fragments are scattered through the fracture site.

Radiographic diagnosis: Gunshot wound resulting in multiple acute fractures of the proximal left humerus.

HISTOPATHOLOGY:

Skeletal muscle (abdominal wall): Several thick walled sarcocysts are present in myofibers. A cluster of degenerating macrophages and granulocytes lie along 1 surface.

Tongue: Several sarcocysts similar to those in the previous section.

Lung: Severe diffuse congestion with mild intra-alveolar hemorrhage.

Thyroid: Several follicles are severely distended with glovular to laminated inspissated colloid.

Spleen: Lymphoid sheaths are small and few. Reticuloendothelial cells commonly contain golden pigment (hemosiderin). The mesothelial cells are plump.

Liver: Moderate centrilobular congestion. Centrilobular hepatocytes are small.

Testis: Inactive spermiogenesis.

Mesentery: The mesentery is thickened by a mixed cell infiltrate including lymphocytes, neutrophils and macrophages. Pools of fibrinous exudate along the surface contain epithelioid cells and layers of granulocytes with macrophages, as well as clusters of bacterial rods. One nematode is seen nearby in cross and longitudinal section.

Intestine: Small trematodes occasionally lie between villi and are numerous in 1 section. One nematode is seen in the superficial mucosa.

Stomach: The mucosa is split at a site with numerous intact and partial nematodes in section, and the underlying submucosa is severely thickened by granulomatous inflammation, fibrosis, and microabscesses associated with intact or degenerative nematodes.

No microscopic lesions are seen in brain, heart, adrenal gland, kidney, or pancreas.

Histopathologic diagnoses:

Severe focal pyogranulomatous gastritis (verminous gastritis)
Pyogranulomatous peritonitis (verminous)
Moderate intestinal trematodiasis
Moderate sarcosporidiosis, skeletal muscle

Comments on histopathology: The microscopic changes correspond well with the gross description of complications from peritoneal and gastrointestinal parasitism.

Final Diagnosis (in order of importance)

	topog.	morph.	etiol.	funct.	disease	link
1. Subacute gunshot, left shoulder	(TY1220)	(M12000)	(E94440)	(_____)	(_____)	(AW)
2. Emaciation	(T00010)	(M70700)	(E00040)	(_____)	(_____)	(PL)
3. Verminous peritonitis and gastritis	(TY4500)	(M42100)	(E45100)	(_____)	(_____)	

Diagnostic findings may not be used for publication without the pathologist's knowledge and consent.

COD (16)

NECROPSY REPORT

NOTE: THIS REPORT MAY BE REVISED FALLOWING RESULTS OF THE HISTOPATHOLOGICAL EXAMINATION OF TISSUES.

DATE OF NECROPSY: 28 March, 1996 SPECIES: Sea otter (Enhydra lutris)

NECROPSY DONE BY: D. Mulcahy, A. Doroff, J. DeGroot

SAMPLE NUMBER: BS 96003 SEE DATA SHEET FOR ADDITIONAL DETAILS

SOURCE OF CARCASS: Collected on beach near Cordova by Dan Monson

GENDER: Male

BACULUM LENGTH: 14.8 cm RIGHT FOREPAW WIDTH: 5.5 cm.

BODY WEIGHT: 52.0 lb/23.6 kg. SKULL LENGTH (SKINNED): 13.3 cm

BODY LENGTH: 139.7 cm. SKULL WIDTH (SKINNED): 9.4 cm

GIRTH AT MANUBRIUM: 64.4 cm.

GENERAL OBSERVATIONS: The carcass was never frozen, and was relatively fresh. This was an mature (est. 6 years) adult male sea otter in very poor body condition. The dorsal spinous processes of the spinal column could be clearly distinguished beneath the pelage. The pelvis, scapulae, and ribs were easily palpable. There were no external abnormalities. Paws and flippers were normal. There was no subcutaneous fat present. The inguinal fat depot was absent.

ORAL CAVITY: There were no oral lesions. The tonsils were not examined. There was medium wear of the canines and molars. The width of the upper left canine was 9.4 mm.

THORAX. There was no pleural inflammation and no pleural effusion.

RESPIRATORY SYSTEM: Moderate mediastinal emphysema was present in the thorax as caudal as the heart. Severe pulmonary emphysema was also present. There was profound hemorrhage in the tracheal mucosa.

HEART: The pericardium and epicardium were normal. There was vegetative endocarditis of the mitral valve which appeared fibrotic. There was no fat present around the base of the heart. The myocardium was normal.

ARTERIES, VEINS, LYMPHATICS: The great vessels were normal. The axillary and inguinal lymph nodes were normal in appearance and size. The mesenteric lymph nodes were greatly enlarged in size where they drained the jejunal perforation noted below.

LIVER: The liver was diminished in size, but was largely normal in appearance and color. The gall bladder was normal size and color and contained about 30 cc blood-tinged bile. There were no parasites present in the gall bladder wall. No bile was collected. The bile duct was patent.

KIDNEYS: Both kidneys appeared normal in size, consistency, shape, and structure.

ENDOCRINE GLANDS: The adrenal glands were enlarged (≅3.0 x 2.0 cm) and firm, with multiple cauliflower-like growths on the surface. The pituitary gland was not examined. The pancreas was normal. The thyroid gland was normal.

REPRODUCTIVE TRACT: Normal. Both testicles were descended.

GASTROINTESTINAL SYSTEM: The abdomen contained about 200 cc of clear bloody fluid. The omentum was blackened and thickened. The esophagus appeared normal. About one-third of the exterior of the stomach was darkly discolored. The stomach contained approximately 40cc of bloody fluid but no solid matter. The stomach contained extensive areas of nematode infestations (about 100 cc of worms in mass). There were no erosions of the gastric mucosa. The duodenum contained a yellowish viscous material but no parasites. The jejunum had many cestodes and 0 to 6 acanthocephalans per 20cm strip. The jejunum contained a small amount of what appeared to be blood. No digesta was present throughout the lengths of the small and large bowels. There was a single area of perforation of the jejunum associated with a large nematode colony. The adjacent bowel was black and necrotic. The draining mesenteric lymph nodes were greatly enlarged. Proximal to the perforation was a large intussusception of the jejunum, with 75 cm of bowel enveloped into a 20 cm length. The colon contained a large amount of digested blood and mucus. The colon wall was thin. The rectum contained a single, very large (ca. 3 cm) colony of nematodes. There were no mesenteric fat stores.

URINARY BLADDER: The bladder was large and contained about 50 cc bloody urine. The ureters appeared to be normal.

EYES AND EARS: Both eyes were present and grossly normal. The ears were normal.

BRAIN: The brain was not examined.

SPINAL CORD: The spinal cord was not examined.

BONE MARROW: The bone marrow was not examined.

BONES AND JOINTS: There were no obvious fractures or malformations noted.

MUSCULATURE: The musculature was symmetrical but reduced in bulk.

HISTOPATHOLOGY SAMPLES: Liver, kidney, bladder, colon, lung, mitral valve, myocardium, pancreas, thyroid gland, gall bladder, lymph node (mesenteric), stomach (with roundworms), rectum (with parasites), heart, spleen, jejunum (2 pieces of perforation), colon, bladder, testicle, and adrenal gland. Urine crystals were also collected.

REMARKS AND WORKING DIAGNOSES: Abnormalities found were: cachexia;peritonitis, pulmonary, mediastinal and subcutaneous emphysema; subcutaneous and gastrointestinal hemorrhage; adrenal hypertrophy; and gastrointestinal parasitism. It is probable that the emphysema was acute and agonal. The animal was chronically debilitated prior to death, and probably succumbed to peritonitis subsequent to the paraite-induced perforation of the jejunum.

Daniel M. Mulcahy Ph.D., D.V.M.
Wildlife Veterinarian

NECROPSY REPORT

NOTE: THIS REPORT MAY BE REVISED FALLOWING RESULTS OF THE HISTOPATHOLOGICAL EXAMINATION OF TISSUES.

DATE OF NECROPSY: 28 March, 1996 SPECIES: Sea otter (Enhydra lutris)

NECROPSY DONE BY: D. Mulcahy, A. Doroff, C. Gorbics

SAMPLE NUMBER: BS 96004 SEE DATA SHEET FOR ADDITIONAL DETAILS

SOURCE OF CARCASS: Collected on beach near Cordova by Dan Monson

GENDER: Male

BACULUM LENGTH: 17.0 cm RIGHT FOREPAW WIDTH: 5.4 cm.

BODY WEIGHT: 54 lb/24.5 kg. SKULL LENGTH (SKINNED): 13.0 cm

BODY LENGTH: 142 cm. SKULL WIDTH (SKINNED): 10.5 cm

GIRTH AT MANUBRIUM: 66 cm.

GENERAL OBSERVATIONS: The carcass was never frozen, but was autolytic and somewhat smelly. This was an older (est. 7-9 years) adult male sea otter in very poor body condition. The dorsal spinous processes of the spinal column could be clearly distinguished beneath the pelage. The pelvis, scapulae, and ribs were easily palpable. The only obvious external abnormality was a large (ca. 15 cm area on the nape of the neck where scavengers had opened the skin and had eaten the most superficial layer of musculature. Blood was present on the muzzle. The right flipper had what appeared to be a well-healed bite wound extending from the second to the fourth digits, with a nonhealing cutaneous wound at the level of the second and third phalanx of the third digit. There was no subcutaneous fat present. The inguinal fat depot was absent. There was a small area of subcutaneous hemorrhage on the side of the back.

ORAL CAVITY: The state of decomposition of the carcass made it impossible to detect oral lesions. There was heavy wear of the incisors and canines. The tonsils were not examined. The upper left canine was broken distally, with exposure of the root. The lower left first molar was partially fractured, with a portion of the lateral wall missing. The upper left first premolar was the only first premolar present and was fractured while being extracted (no tooth age will be available for this animal). The width of the upper left canine was 11.0 mm.

THORAX. There was no pleural inflammation and no pleural effusion.

RESPIRATORY SYSTEM: Slight mediastinal emphysema was present in the cervical region and in the thorax as caudal as the heart. Moderate pulmonary emphysema was also present. There was no hemorrhage in the tracheal mucosa.

HEART: The pericardium and epicardium were normal. The heart and major vessels appeared normal. There was no fat present around the base of the heart. The myocardium was normal.

ARTERIES, VEINS, LYMPHATICS: The great vessels were normal. The axillary and inguinal lymph nodes were normal in appearance and size. The mesenteric lymph nodes were normal in size.

LIVER: The liver was diminished in size, but was largely normal in appearance and color. The gall bladder was normal size and color and contained about 20 cc blood-tinged bile. There was no hemorrhage and no parasites present in the gall bladder wall. No bile was collected. The bile duct was patent.

KIDNEYS: Both kidneys had a small amount of white, gritty material adherent to the capsules. Otherwise, the kidneys appeared normal in size, consistency, shape, and structure.

ENDOCRINE GLANDS: The adrenal glands were enlarged (≅3 x 2 cm) and firm. The pituitary gland was not examined. The pancreas was normal. The thyroid gland was normal.

REPRODUCTIVE TRACT: Normal. Both testicles were descended.

GASTROINTESTINAL SYSTEM: The esophagus appeared normal. The stomach contained approximately 100cc of bloody fluid but no solid matter. The stomach contained multiple areas of nematode infestations. A few roundworms could be found loose, outside of the stomach. There were no erosions of the gastric mucosa. The duodenum contained a yellowish viscous material but no parasites. The jejunum held no cestodes and about six to 18 acanthocephalans per 20cm strip. The jejunum contained a small amount of what appeared to be blood. No digesta was present throughout the lengths of the small and large bowels. The rectum contained a small amount of mussel shell. There were no mesenteric fat stores. The colon contained some black, tarry fluid, presumably digested blood. There were no erosions present.

URINARY BLADDER: The bladder was large but empty. The ureters appeared to be normal.

EYES AND EARS: Both eyes were present and grossly normal. The ears were normal.

BRAIN: The brain was not examined.

SPINAL CORD: The spinal cord was not examined.

BONE MARROW: The bone marrow was not examined.

BONES AND JOINTS: There were no obvious fractures or malformations noted.

MUSCULATURE: The musculature was symmetrical but reduced in bulk.

HISTOPATHOLOGY SAMPLES: Esophagus, liver, kidney, bladder, colon, lung (2), myocardium, pancreas, thyroid gland, gall bladder, lymph node (right axillary), stomach

(with roundworms), spleen, duodenum, colon, bladder, and adrenal gland. (Note: the scribe did not record these on the sample sheet.)

REMARKS AND WORKING DIAGNOSES: Abnormalities found were: cachexia; pulmonary, mediastinal and subcutaneous emphysema; subcutaneous and gastrointestinal hemorrhage; adrenal hypertrophy; and gastrointestinal parasitism. It is probable that the emphysema was acute and agonal.

Daniel M. Mulcahy Ph.D., D.V.M.
Wildlife Veterinarian

NECROPSY REPORT

NOTE: THIS REPORT MAY BE REVISED FALLOWING RESULTS OF THE HISTOPATHOLOGICAL EXAMINATION OF TISSUES.

DATE OF NECROPSY: 28 March, 1996 SPECIES: Sea otter (Enhydra lutris)

NECROPSY DONE BY: D. Mulcahy, A. Doroff, S. Kalxdorff

SAMPLE NUMBER: BS 96005 SEE DATA SHEET FOR ADDITIONAL DETAILS

SOURCE OF CARCASS: Collected on beach near Cordova by Dan Monson

GENDER: Male

BACULUM LENGTH: 15.3 cm

RIGHT FOREPAW WIDTH: 4.6 cm.

BODY WEIGHT: 45.5 lb/20.5 kg.

SKULL LENGTH (SKINNED): 13.2 cm

BODY LENGTH: 138 cm.

SKULL WIDTH (SKINNED): 10.2 cm

GIRTH AT MANUBRIUM: 62.3 cm.

GENERAL OBSERVATIONS: The carcass was never frozen, and was relatively fresh. This was an young (est. 3-4 years) adult male sea otter in very poor body condition. The dorsal spinous processes of the spinal column could be clearly distinguished beneath the pelage. The pelvis, scapulae, and ribs were easily palpable. There were no external abnormalities. Paws and flippers were normal. There was no subcutaneous fat present. The inguinal fat depot was absent. There was an area of hemorrhage dorsal and caudal to the left scapula. An large area of subcutaneous emphysema was present dorsal to the pelvis on the right side.

ORAL CAVITY: There were no oral lesions. The tonsils were not examined. There was light wear of the canines and molars. The width of the upper left canine was 8.1 mm.

THORAX. There was no pleural inflammation and no pleural effusion.

RESPIRATORY SYSTEM: Severe mediastinal emphysema was present in the cervical region and in the thorax as caudal as the heart. Severe pulmonary emphysema was also present. There was profound hemorrhage in the tracheal mucosa.

HEART: The pericardium and epicardium were normal. The heart and major vessels appeared normal except for some mitral valve fibrosis. There was no fat present around the base of the heart. The myocardium was normal.

ARTERIES, VEINS, LYMPHATICS: The great vessels were normal. The axillary and inguinal lymph nodes were normal in appearance and size. The mesenteric lymph nodes were normal in size.

LIVER: The liver was diminished in size, but was largely normal in appearance and color. The gall bladder was normal size and color and contained about 30 cc blood-tinged bile. There were no parasites present in the gall bladder wall. No bile was collected. The bile duct was patent.

KIDNEYS: Both kidneys appeared normal in size, consistency, shape, and structure.

ENDOCRINE GLANDS: The adrenal glands were enlarged (≅3.5 x 1.7 cm) and firm. The pituitary gland was not examined. The pancreas was normal. The thyroid gland was normal.

REPRODUCTIVE TRACT: Normal. Both testicles were descended.

GASTROINTESTINAL SYSTEM: The esophagus appeared normal. The stomach contained approximately 40cc of bloody fluid but no solid matter. The stomach contained multiple areas of nematode infestations. There were no erosions of the gastric mucosa. The duodenum contained a yellowish viscous material but no parasites. The jejunum held no cestodes and no acanthocephalans per 20cm strip. The jejunum contained a small amount of what appeared to be blood. No digesta was present throughout the lengths of the small and large bowels. The colon contained a small amount of mussel shell and a large amount of digested blood. The colon wall was thin. There were no mesenteric fat stores.

URINARY BLADDER: The bladder was large and contained about 5 cc urine, with about 4 cc of fine crystals. The ureters appeared to be normal.

EYES AND EARS: Both eyes were present and grossly normal. The ears were normal.

BRAIN: The brain was not examined.

SPINAL CORD: The spinal cord was not examined.

BONE MARROW: The bone marrow was not examined.

BONES AND JOINTS: There were no obvious fractures or malformations noted.

MUSCULATURE: The musculature was symmetrical but reduced in bulk.

HISTOPATHOLOGY SAMPLES: Liver, kidney, bladder, colon, lung, myocardium, pancreas, thyroid gland, gall bladder, lymph node (mesenteric), stomach (with roundworms), heart, spleen, duodenum, colon, bladder, testicle, and adrenal gland. Urine crystals were also collected.

REMARKS AND WORKING DIAGNOSES: Abnormalities found were: cachexia; pulmonary, mediastinal and subcutaneous emphysema; subcutaneous and gastrointestinal hemorrhage; adrenal hypertrophy; and gastrointestinal parasitism. It is probable that the emphysema was acute and agonal.

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NECROPSY REPORT

NOTE: THIS REPORT MAY BE REVISED FALLOWING RESULTS OF THE HISTOPATHOLOGICAL EXAMINATION OF TISSUES.

DATE OF NECROPSY: 26 March, 1996 SPECIES: Sea otter (Enhydra lutris)

NECROPSY DONE BY: D. Mulcahy, A. Doroff, L. Thomas

SAMPLE NUMBER: BS 96006 SEE DATA SHEET FOR ADDITIONAL DETAILS

SOURCE OF CARCASS:Found dead 22 March, 1996 on Cordova boat dock (Dan Monson)

GENDER: Male

BACULUM LENGTH: 14.4cm RIGHT FOREPAW WIDTH: 5.6 cm.

BODY WEIGHT: 45 kg. SKULL LENGTH (UNSKINNED): 14.1cm

BODY LENGTH: cm. SKULL WIDTH (UNSKINNED): 11.1cm

GIRTH AT MANUBRIUM: cm.

GENERAL OBSERVATIONS: The carcass was never frozen. This is an adult male sea otter in very poor body condition. The dorsal spinous processes of the spinal column can be clearly distinguished beneath the pelage. The plvis, scapulae, and ribs are easily palpable. There are no obvious external abnormalities. Paws and flippers are normal. There is no subcutaneous fat present. The inguinal fat depot was absent. On being skinned, there were two areas of subcutaneous hemorhage over pressure points on the left and right hind legs, and an additional deep subcutaneous hemorrhage just cranial to the wing of the right ileum. There was crepitus in the right axilla, which proved to be subcutaneous emphysema on dissection.

ORAL CAVITY: There were no oral lesions present. There was light wear of the incisors and canines. The tonsils were not examined. The upper right first premolar was missing (taken by D. Monson in Cordova) and the upper left first premolar was taken for aging. The width of the upper right canine was 8.3mm.

THORAX. There was no pleural inflammation and no pleural effusion.

RESPIRATORY SYSTEM: Marked mediastinal emphysema was present from the cranial cervical region to the diaphragm. There was also severe pulmonary emphysema. There were extensive hemorrhage in the tracheal mucosa. The right lung was livid, suggesting a right lateral position of the animal after death.

HEART: The pericardium and epicardium were normal. The heart and major vessels appeared normal. There was no fat present around the base of the heart. The myocardium was normal. The bicuspid valve was firm, white in color and thickened. The tricuspid valve seemed normal.

ARTERIES, VEINS, LYMPHATICS: The great vessels were normal. The axillary and inguinal lymph nodes were normal in appearance and size. The mesenteric lymph nodes were normal in size

LIVER: The liver was diminished in size, but was largely normal in appearance and color. There were several irregular areas of beige, tightly adherent material on the surface of the liver. A similar area on the accesory lobe of the liver extended into the liver parenchyma and a piece was taken for histopathology. The gall bladder was thick-walled, white in color, and was elongated to about 10cm. It contained about 12cc of opaque, canary yellow bile. There was no hemorrhage and no parasites present in the gall bladder. No bile was collected. The bile duct was patent.

KIDNEYS: Extensive subcapsular hemorhage nearly surrounded the right kidney and was present to a lesser amount around the left kidney. The kidneys appeared normal in size, consistency, shape, and structure.

ENDOCRINE GLANDS: The adrenal glands were enlarged (≅3 x 21/2cm), firm and the cortex was very white in color, while there appeared to be hemorrhage present in the medulla. The pituitary gland was not examined. The pancreas was normal. The thyroid gland was normal.

REPRODUCTIVE TRACT: The distal 5 cm of the baculum was at a different angle (≅20°) than the remainder of the structure suggesting an old fracture, but no callus could be palpated. Both testical were descended.

GASTROINTESTINAL SYSTEM: The esophagus appeared normal. The stomach contained approximately 50cc of bloody fluid but no solid matter. There were some erosions of the gastric mucosa. There were three patches of gastric mucosa with embedded roundworms of about 21/2 to 3 cm in length. Several single worms were also found attached. The duodenum contained a yellowish viscus material but no parasites. The jejunum held numerous cestodes throughout its length and about eight to ten acanthocephalans per 20cm strip. A single small cestode with a scolex, several acanthocephalans and some sections of cestodes were collected for examination at Purdue. The jejunum contained a constant ribbon of what appeared to be blood. No digesta was present throughout the lengths of the small and large bowels. There were no mesenteric fat stores. The colon contained large amounts of black, tarry fluid, presumably digested blood. There were no erosions present, but the colonic mucosa appeared to be uniformly absent, to the point that the colonic wall was nearly transparent.

URINARY BLADDER: The bladder was empty but there were extensive petechial hemorrhages. The bladder wall was thickened. The ureters appeared to be normal.

EYES AND EARS: Both eyes were present and grossly normal. The ears were normal.

BRAIN: The brain was not examined.

SPINAL CORD: The spinal cord was not examined.

BONE MARROW: The bone marrow was not examined.

BONES AND JOINTS: There were no obvious fractures or malformations noted.

MUSCULATURE: The musculature was symmetrical but reduced in bulk.

HISTOPATHOLOGY SAMPLES: Esophagus, liver, kidney, bladder, colon, lung (2), myocardium, bicuspid valve, pancreas, thyroid gland, gall bladder, lymph node (near stomach), stomach (with roundworms), spleen, duodenum, colon, bladder, and adrenal gland.

REMARKS AND WORKING DIAGNOSES: Abnormalities found were: cachexia; pulmonary, mediastinal and subcutaneous emphysema; subcutaneous, visceral, gastrointestinal and urological hemorrhage; adrenal hypertrophy; valvular fibrosis; colitis; and heavy gastrointestinal parasitism. It is possible that the emphysema was acute and agonal. There may have been a coagulopathy and anemia due to chronic disease (gastrointestinal hemorrhage). The colitis seemed profound.

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Wildlife Veterinarian

USGS-BIOLOGICAL RESOURCES DIVISION
NATIONAL WILDLIFE HEALTH CENTER
6006 Schroeder Road
Madison, Wisconsin 53711-6223
608-271-4640 (FAX 608-264-5431)

DIAGNOSTIC SERVICES CASE REPORT

Case: #14200, 001-002 Epizoo: #96-040 RHT: LCG

Submitter:
Angie Doroff
USFWS - Region 7
Marine Mammals Management
1011 E. Tudor Rd.
Anchorage, AK 99503

Specimen description/identification:
(001) Sea Otter BS96007
(002) Sea Otter BS96008

Date Submitted: 04/15/96

Location: Cordova, Valdez-Cordova Burrough, AK.

General Diagnosis: 001: Liver abscess; emaciation
002: Perforating verminous gastric ulcer; peracute volvulus

Comments: Otter 001 had a large liver abscess associated with watery exudate in the abdominal cavity. Microscopically there was evidence that bacteria had entered the circulation, at least terminally. The cause of this large abscess was not evident but the isolation of *E. coli* indicates an intestinal origin of the bacteria and the proximity to GI parasites is suspicious. This animal was badly emaciated, a condition that may have been caused or at least complicated by the abscess.

Otter 002 had a perforating gastric ulcer in which many degenerating nematodes were seen microscopically. This otter also had a terminal twist in its intestine. This animal had evidence of recent fat atrophy.

Skeletal muscle samples from each otter will be sent to Carol Gorbics in the near future.

X Final Report (08/26/97)
date

X See attached necropsy records for individual specimen observations.

N. J. Thomas

Pathologist: Nancy J. Thomas, DVM, MS
Diplomate, American College of Veterinary Pathologists

Linda C. Glaser DVM

If you have questions regarding this case, contact: Linda C. Glaser, DVM
at 608-271-4640. Include above Case Number. Diagnostic findings may not be used for publication without the pathologist's knowledge and consent.

NATIONAL WILDLIFE HEALTH CENTER
NECROPSY REPORT

Submitter's Name, Affiliation Address
Angie Doroff
USFWS - Region 7
Marine Mammals Management
1011 E. Tudor Rd.
Anchorage, AK 99503

LEGAL No
Case: 14200
Accession: 002
Collected: 04/11/96
Exam Date: 04/15/96
Pathologist: N. Thomas
Prosector: N. Thomas

Species: Sea Otter Specimen: Carcass nit
Bandtype: (E) Ref/Band No: (BS96008) Euth: (N) Weight (Gm): (16.1 kg)
History Summary: This is one of two animals found dead near Cordova, Alaska.

EXTERNAL/INTERNAL OBSERVATIONS - LABORATORY RESULTS

External: Blood tinged fluid is draining from the nares and staining the muzzle. A tag of blood-tinged fibrin adheres to the margins of the left nares. There is a 1 mm punctate ulcer on the nose at the margin of the right nares. There are hypopigmented scars on the upper lip including a cleft on the right side. All teeth are present and in good condition with the exception of a missing upper right first premolar (postmortem removal). There is mild tartar accumulation and mild pitting of the premolars and molars. There is a gingival and possibly a bony defect forming a cleft between the third mandibular premolar and the first molar. Food items have accumulated in this deep pocket bilaterally. Oral mucous membranes are bright pink. The thorax and abdomen are moderately narrow but the abdomen has a fluid-filled texture on balloting.

Internal: There is serous atrophy of subcutaneous fat remnants visible along the ventral midline, inguinal regions and along the hind legs. The peripheral lymph nodes including submandibular, prescapular, axillary and inguinal nodes are mildly prominent but flat; they have brown and white mottled cut aspects. The peritoneal cavity contains approximately 2 - 3 liters of cloudy red-grey fluid. Strands of fibrinopurulent material have accumulated deep in the peritoneal caviry. A moderate number of nematodes are lying free on the peritoneal surfaces and congregated along the omentum in the cranial abdomen. A few small peritoneal tags are present on the splenic capsule. There is severe pseudomelanosis of the liver, spleen, kidneys and other abdominal organs. One segment of fluid distended small intestine is moderately red-purple. This segment and a contiguous short segment tightly filled with ingesta are suspended from a twisted mesenteric root. The mesentery is abruptly hemorrhagic at the site of the twist and the mesentery and serosa of the involved intestinal segment are moderately congested. Mesenteric lymph nodes are moderately prominent. The urinary bladder is empty. The gall bladder is moderately contracted and has a thick but pliable wall. The bile is deep orange and mildly cloudy with minute particles. The left lung lobes are heavy, wet and red. The larger bronchi contain pink cloudy fluid with fibrin or catarrhal strands. The right lung lobes are mildly heavy, but not wet. All lung lobes have a uniformly soft texture. The esophagus contains red-yellow fluid. Visible on the serosal aspect of the gastric fundus is a 1 cm circular perforation with smooth margins. The perforation is surrounded on the serosa by a ring of purple and grey discoloration and fibrin tags. On the mucosal surface the ulcer is visible as an approximately 2 X 5 mm slit with mucosa everting to cover the margins. The mucosa is mildly raised to form a circular rim around the ulcer site but exposed submucosa is not visible from the interior. The stomach contains a small amount of brown fluid and a few small nematodes. The nematodes lightly adhere to the gastric mucosa. The duodenum contains bile-stained fluid and a thick bolus of broad cestodes. The cestodes persist into the middle to lower jejunum in progressively fewer numbers. In the middle jejunum there are also a moderate number of medium sized acanthocephalans mixed with a small amount of yellow cloudy fluid and occasional shell fragments. The volvulus in the lower jejunum and ileum is preceded by an accumulation of thick, green-yellow fluid with loose shell fragments. The upper 30 cm of the intestinal segment entrapped in the volvulus is packed with small black to black-purple shell fragments with a pearlized surface. The lower portion, an additional 1 meter, of the entrapped intestine is distended by profuse red-brown watery fluid. The remainder of the intestinal tract contains brown fluid and sparse shell fragments. The brain is congested. No lesions are seen in the pituitary gland, oral mucosa, tongue, hyoid bones, thyroid glands, trachea, esophagus, heart, pericardial sac, heart valves, tracheobronchial

lymph nodes, pleura, diaphragm, kidneys, ureters, urinary bladder, pancreas, adrenal glands, or the skeletal system including bilateral shoulder, elbow, hip, stifle, or right talus. The right testis is 31 X 10 mm. The left testis is 32 X 10 mm. The total length 115 cm; grizzling extends to the eyes; teeth have mild wear.

Preliminary Diagnosis: Subacute perforating gastric ulcer; peracute intestinal volvulus Exam Type: (GO)
Sex (M) Age (A)/(__) Body Cond. (F) Postmortem State (P) Giz. Lead (0)/(__)

Necropsy comments:

Laboratory Tests/Samples Saved:

1. PARASITOLOGY: Nasal flush, GI tract, nematodes from the peritoneal cavity, nematodes from stomach, gall bladder, omentum.

2. BACTERIOLOGY: Peritoneal fluid - RC.
   Results: Peritoneal fluid = *Escherichia coli* E15901
   Streptococcus fecalis E25016

3. HISTOPATHOLOGY: Variety of tissues.

4. Saved Frozen for TOX: Brain, liver, kidney, skeletal muscle.

5. Photos taken.

6. Saved For Return to Submitter: Upper Premolar, whiskers.

7. Carcass incinerated.

HISTOPATHOLOGY:

Heart: A small sarcocyst is seen in a myofiber.

Lung: Moderate diffuse edema. One colony of small bacterial rods is associated with blanching of nearby tissue.

Liver: There is one focus of acute coagulative necrosis.

Intestine: Severe autolysis. Large ruptured parasite eggs are seen in the superficial mucosa in 1 section.

Stomach: The muscularis is thickened and incorporates numerous small pools of pyogranulomatous inflammation encompassing degenerating parasite cuticle or muscle. Large zone of granulation tissue are also present. The adventitia is hyperplastic and includes macrophages; it is coated by a layer small bacterial rods.

No microscopic lesions are seen in brain or kidney.

Histopathologic diagnoses:

Chronic pyogranulomatous gastritis (verminous gastritis)
Mild sarcosporidiosis, heart, and moderate, skeletal muscle
Moderate acute pulmonary edema

Final Diagnosis (in order of importance)

	topog.	morph.	etiol.	funct.	disease	link
1. Perforating verminous gastric ulcer	(T63000)	(M38000)	(E45100)	(____)	(____)	(_)
2. Peracute volvulus	(T64000)	(M34200)	(_____)	(____)	(____)	(_)
3. _____	(_____)	(_____)	(_____)	(____)	(____)	

Diagnostic findings may not be used for publication without the pathologist's knowledge and consent.

COD (12)