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PATTERNS OF MORTALITY IN SOUTHERN SEA OTTERS (*ENHYDRA LUTRIS NEREIS*) FROM 1998–2001

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ABSTRACT: Detailed postmortem examination of southern sea otters (*Enhydra lutris nereis*) found along the California (USA) coast has provided an exceptional opportunity to understand factors influencing survival in this threatened marine mammal species. In order to evaluate recent trends in causes of mortality, the demographic and geographic distribution of causes of death in freshly deceased beachcast sea otters necropsied from 1998–2001 were evaluated. Protozoal encephalitis, acanthocephalan-related disease, shark attack, and cardiac disease were identified as common causes of death in sea otters examined. While infection with acanthocephalan parasites was more likely to cause death in juvenile otters, *Toxoplasma gondii* encephalitis, shark attack, and cardiac disease were more common in prime-aged adult otters. Cardiac disease is a newly recognized cause of mortality in sea otters and *T. gondii* encephalitis was significantly associated with this condition. Otters with fatal shark bites were over three times more likely to have preexisting *T. gondii* encephalitis suggesting that shark attack, which is a long-recognized source of mortality in otters, may be coupled with a recently recognized disease in otters. Spatial clusters of cause-specific mortality were detected for *T. gondii* encephalitis (in Estero Bay), acanthocephalan peritonitis (in southern Monterey Bay), and shark attack (from Santa Cruz to Point Año Nuevo). Diseases caused by parasites, bacteria, or fungi and diseases without a specified etiology were the primary cause of death in 63.8% of otters examined. Parasitic disease alone caused death in 38.1% of otters examined. This pattern of mortality, observed predominantly in juvenile and prime-aged adult southern sea otters, has negative implications for the overall health and recovery of this population.

Key words: Acanthocephalan, cardiac disease, *Enhydra lutris nereis, Sarcocystis neurona,* shark predation, southern sea otter, *Toxoplasma gondii.*

INTRODUCTION

Evidence linking anthropogenic stressors to unusual patterns of disease and mortality in marine mammals has accumulated over the past decade (Ross et al., 1996; Harvell et al., 1999; Fair and Becker, 2000; Daszak et al., 2001). Habitat degradation, pollutants, municipal runoff, global climate change, and overharvest of marine resources are likely to have complex effects that both directly and indirectly affect marine mammal health. Southern sea otters (*Enhyrda lutris nereis*) are a useful indicator of near-shore marine ecosystem health because they are heavily exposed to human activity in coastal California (USA) and they commonly remain in one geographically localized area for most of their lives. Despite legal protection since 1911 and unlike sympatric California sea lions (*Zalophus californianus*), northern elephant seals (*Mirounga angustirostris*), and harbor seals (*Phoca vitulina*) in California (Carretta et al., 2001), southern sea otters have made a slower than expected recovery after hunting drastically reduced their numbers prior to the 20th century (Estes, 1990). Annual counts of sea otters over their entire range in California have declined since 1995 with the current count at slightly over 2,000 animals (U.S. Geological Survey, unpubl. data). Birth rates in California sea otters appear similar to those observed in other, rapidly growing otter populations (Riedman et al., 1994; Monson et al., 2000), suggesting that increased mortality may be causing the slow recovery rate in the California population.

A high percent of mortality due to infectious disease was detected in detailed necropsies of southern sea otters from

1992–95, which raised concern over the general susceptibility of this species to infection and subsequent mortality (Thomas and Cole, 1996). Furthermore, some diseases were newly recognized as important causes of mortality in sea otters, and several pathogens were implicated that were not expected to cause considerable mortality in a free-ranging marine wildlife species, such as protozoal encephalitis, acanthocephalan peritonitis, and coccidioidomycosis (Thomas and Cole, 1996). Continued monitoring of these newly recognized diseases in sea otters is essential, not only in light of the recent population decline, but also because techniques used to diagnose causes of death in sea otters have improved with time. Furthermore, detailed evaluation of specific patterns of mortality in sea otters may have broad implications for overall ecosystem health and may improve our understanding of the processes that promote disease in a marine mammal population. In order to evaluate the recent pattern of mortality in sea otters, the demographic and geographic distribution of causes of death for freshly deceased sea otters found along the California coast between 1998 and 2001 were evaluated.

MATERIALS AND METHODS

Carcass collection and evaluation

Sea otter carcasses were recovered through a large-scale stranding network conducted by the California Department of Fish and Game (CDFG), the United States Geological Survey (USGS), the Monterey Bay Aquarium (MBA), and The Marine Mammal Center (TMMC). Collaborating veterinary pathologists at CDFG's Marine Wildlife Veterinary Care and Research Center (MWVCRC, Santa Cruz, California) and the University of California (UC) School of Veterinary Medicine (Davis, California) examined three of four freshly dead otters; every fourth carcass was sent to the National Wildlife Health Center (Madison, Wisconsin, USA). Necropsy findings from otters examined at MWVCRC/UC from February 1998 through June 2001 were included in these analyses.

Stranding location for all otters was determined at the time of carcass recovery. Otters were assigned the corresponding latitude and longitude value of their stranding location to the nearest 0.5 km increment along a smoothed California coastline. Stranding locations were also categorized as north or south based on their relation to Cape San Martin (35°89′N, 121°46′W), which is at the center of the southern sea otter range. Age class was determined at necropsy and was estimated as follows: juveniles—milk teeth present (younger than 1 yr), subadults—unworn adult dentition (approximately 1–3 yr old), prime-aged adults—adult dentition with evidence of some tooth wear (approximately 4–9 yr old), and aged adults—marked tooth wear (approximately 10 yr and older). When evaluating age as a risk factor for specific causes of death, age classes were collapsed into a juvenile category (juveniles and subadults) and an adult category (prime-aged adults and aged adults). Pregnancy and lactation status were recorded for females at necropsy.

Only otters that were in good postmortem condition (dead for <4 days) were included in this study. Otters heavily scavenged prior to necropsy and those with incomplete histopathology were excluded. For otters that stranded alive but were subsequently euthanized or died while undergoing rehabilitation, the pathologic conditions that were the likely cause of stranding were reported. Otters that remained in rehabilitation for longer than 4 wk were excluded. All otters received a complete gross necropsy, as well as microscopic examination of major tissues including heart, lung, liver, kidney, spleen, stomach, small intestine, colon, omentum, multiple lymph nodes, skeletal muscle, spinal cord, and brain. Tissue sections were placed in 10% neutral buffered formalin, paraffin-embedded, sectioned at 5 μm, and stained with hematoxylin and eosin for examination by light microscopy. Acanthocephalan parasites known to infect sea otters (Hennessy and Morejohn, 1977) were identified to genus by overall size and proboscis morphology at gross necropsy (Amin, 1992). Laboratory evaluation of bacterial, fungal, and parasite samples and toxicologic screening for domoic acid were performed when indicated. Swabs for bacterial culture were collected from heart, blood, intestinal tract, and lung, plated on tryptic soy agar with 5% sheep blood, MacConkey agar, and XLT-4 agar (Hardy Diagnostics, Santa Maria, California). Plates were incubated at 37 C. Bacterial isolates were identified by the Microbiology Laboratory at the UC Veterinary Medical Teaching Hospital (Davis, California) using standard biochemical and molecular techniques. Tissues swabs collected from necropsied otters with suspected fungal infections were also submitted to the Microbiology Laboratory. Samples were placed on wet mounts

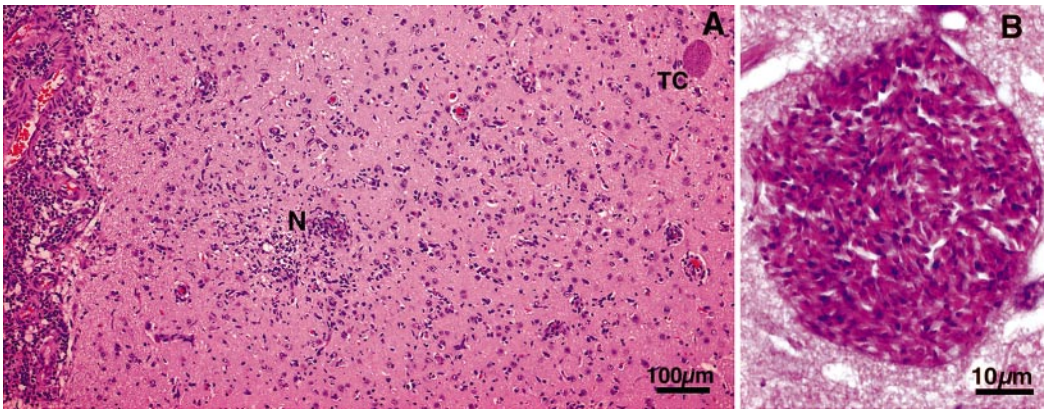

FIGURE 1. A. Low magnification of a hematoxylin and eosin (HE) stained section of cerebrum from a southern sea otter with encephalitis due to *Toxoplasma gondii.* Note moderate perivascular to diffuse mononuclear inflammatory infiltrate with marked expansion of Virchow-Robbins spaces by exocytosing inflammatory cells. Near the center of the affected neuropil is a small focus of necrosis (N.). A *T. gondii* tissue cyst (T.C.) is present in the upper right hand corner of the photo. B. High magnification view of HE-stained sea otter cerebrum showing a *T. gondii* tissue cyst filled with hundreds of elongated bradyzoites.

for the direct observation of spherules. Samples were also cultured on inhibitory mold agar with chloramphenicol (Hardy Diagnostics) and incubated at 30 C in air for 3 wk. If fungal growth occurred, gross colonial morphology was evaluated and a lactophenol cotton blue preparation (Hardy Diagnostics) was made for microscopic examination of arthroconidia for the identification of *Coccidioides immitis* (St Germain and Summerbell, 1996). Suspect *C. immitis* positive cultures were submitted to a specialty laboratory for confirmation (D. Pappagianis, Microbiology and Immunology, UC Davis School of Medicine, Davis, California). On histopathology, *C. immitis* was identified by the presence of round (5–50 μm in diameter), thick-walled spherules containing endospores.

Death was attributed to domoic acid intoxication when substantially elevated levels of domoic acid were detected in urine (Scholin et al., 2000) and gross or histologic lesions suggestive of another cause of mortality were absent. Domoic acid was initially detected by receptor-binding assay (Dr. Vera Trainer, National Oceanic and Atmospheric Administration, Northwest Fisheries Science Center, Environmental Conservation Division, Marine Biotoxin Program, Seattle, Washington, USA) and, when possible, results were confirmed by liquid chromatography-tandem mass spectroscopy (Dr. Francis Van Dolah, National Oceanic Atmospheric Administration, Center for Coastal Environmental Health and Biomolecular Research, Marine Biotoxin Program, Charleston, South Carolina, USA).

Encephalitis was determined to be a primary cause of death when moderate to severe inflammation of the cerebral and/or cerebellar neuropil was present on histopathology (Fig. 1a). This criterion was chosen because moderate encephalitis was identified histologically in otters undergoing rehabilitation with recurrent seizures and severe neurologic dysfunction. Protozoal infection with either *Toxoplasma gondii* or *Sarcocystis neurona* was determined to be the cause of encephalitis when protozoal stages were directly visualized within inflammatory foci in the neuropil on histopathology (Fig. 1) and/or when laboratory tests confirmed infection through isolation of protozoa from brain tissue (Miller et al., 2001b). Antibody titers to *T. gondii* and *S. neurona* were not used to diagnose these parasites as the cause of encephalitis because positive titers do not distinguish between past exposure and active infection. Otters with encephalitis but without histopathologic or laboratory confirmation of protozoal infection were diagnosed with encephalitis of unconfirmed cause.

Trauma noted at necropsy was attributed to shark attack when shark tooth fragments were recovered from wounds or when multiple stab-like lacerations in soft tissue or scratches, characteristic of contact with serrated shark teeth, were detected on underlying bone or cartilage (Ames and Morejohn, 1980) (Fig. 2). Similarly, trauma was attributed to boat strike in cases with soft tissue lacerations and fractures or cuts in underlying bone in a pattern consistent with a propeller injury (Ames and Morejohn, 1980). Severe blunt trauma suggestive of a high speed collision with a boat hull was also attributed to

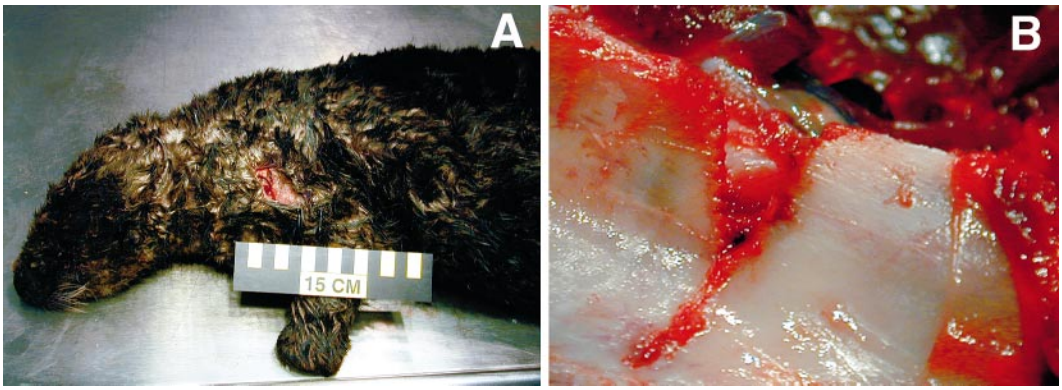

FIGURE 2. A. Southern sea otter with a shoulder laceration caused by shark attack. Note the typical stab wound-like pattern and minimal soft tissue removal associated with penetration by shark teeth. B. Higher magnification view of the scapula from the same sea otter shown in Figure A showing a transverse cut of the scapular spine. This cut has distinct comb-like edges, typical of wounds inflicted by great white sharks, which have serrated teeth.

boat strike. Cases were diagnosed with trauma or lacerations of unknown cause when clear evidence for shark attack or boat strike was not detected.

Causes of death were rigorously standardized so that the primary cause identified for each otter was the most substantial injury or illness initiating the sequence of events leading directly to death. Otters were assigned two equally weighted primary causes of death if two unrelated and independent conditions were each severe enough to have caused death. Contributing causes of death were noted only if pathologic conditions were identified that added to the probability of death, but were not part of the primary disease complex. To ensure independence of primary and contributing causes of death, diagnoses of conditions were based solely on the severity of the individual lesions regardless of other conditions present.

Statistical analyses

Proportionate mortality, noted as a percent, was used to identify the major causes of death in sea otters from 1998 to 2001. Cause-specific proportionate mortality was calculated as the proportion of otters with a specific condition as the primary cause of death among all otters that met the inclusion criteria during the study period. Distribution of the four most common causes of death among sex and age classes was evaluated by the standard two-sided chi square test of independence using statistical software (Epi Info Version 6, Centers for Disease Control and Prevention, Atlanta, Georgia, USA). One-sided chi-square test of independence was used to test associations between each of the four most common primary causes of death and the common contributing causes of death with at least nine cases each. If age or sex were significantly associated with a primary cause of death, associations among primary and contributing causes of death were stratified by the significant variable (age or sex), and the association was evaluated for each stratum. For dichotomous distributions with an expected frequency less than five, the Fisher exact test (Fisher, 1935) was calculated using statistical software (SPSS Base 10.0, SPSS Inc., Chicago, Illinois, USA). When appropriate, strength of the association was estimated by the odds ratio.

Geographic and temporal clustering of overall carcass retrieval and temporal clustering of each primary cause of death were evaluated by the scan test (Carpenter, 2001). This test evaluates whether carcass retrieval was uniform using the binomial distribution to estimate the probability of the maximum observed number of cases within sequential distances of 5 km, 10 km, 15 km, and 20 km for geographic clustering and within 1, 2, 3 and 4 mo for temporal clustering. These spatial and temporal periods were specified because increased carcass retrieval within these time periods was considered biologically significant based on sea otter behavior and habitat use. Geographic clustering of specific primary causes of death was evaluated only for the four most common primary causes of death with 10 cases or more. Specifically, the spatial scan statistic (Kulldorf and Nagarwalla, 1995) was used to test whether a primary cause of death was randomly distributed along the coastline where sea otters carcasses were recovered. The Bernoulli model was selected for this procedure, which used the case and control approach to adjust for differ-

ential retrieval of carcasses along the coastline. In this manner, stranding locations for each specific cause of death were compared to locations for otters with all other causes of death. A Monte Carlo iterative technique was used to determine distribution of the likelihood ratio test statistic (Kulldorf and Nagarwalla, 1995).

RESULTS

Between February 1998 and June 2001, 105 sea otter carcasses met our inclusion criteria and received a complete necropsy and diagnostic work up in order to determine the cause of death. Of these otters, 27 were alive at the time of stranding but died or were euthanized at rehabilitation centers. Sea otter carcass retrieval was spatially clustered along the coastline during our study period. Both Monterey Bay and Estero Bay had significant ($P<0.01$) clusters of beachcast carcasses. No carcasses were retrieved from the remote and rocky 140 km long coastal segment in the center of the sea otter range south of Yankee Point (36°48′N, 121°94′W) to north of San Simeon Point (35°63′N, 121°19′W). Overall carcass retrieval was not temporally clustered during the study period. Carcasses recovered consisted predominantly of prime-aged adults (46.7%), with fewer juveniles (29.5%), subadults (11.4%), and aged adults (12.4%). Distribution of primary causes of death among sex and age classes is shown in Table 1. While 25 different primary causes of death were identified, 53.3% of otters (56/105) died from one of four major causes: *T. gondii* encephalitis, acanthocephalan parasite infection, shark attack, or cardiac disease. Acanthocephalan infection, cardiac disease, and *T. gondii* encephalitis were also recognized as common contributing causes of death (Table 1).

Encephalitis due to *T. gondii* was one of the two leading causes of mortality identified in sea otters during the time period studied. This condition, characterized by moderate to severe, multifocal, non-suppurative and necrotizing meningoencephalitis (Fig. 1), was a primary cause of death in 16.2% of the otters examined and was a contributing factor in another 11.4% of otters examined. A marginally significant spatial cluster of *T. gondii* encephalitis cases was detected in a 25 km area at the southern end of Estero Bay in central California ($P=0.06$, centered at 35°31′N, 120°88′W, Fig. 3). Half of the otters (8/16) recovered in this section of Estero Bay were determined to have *T. gondii* encephalitis as the primary cause of death. Encephalitis caused by *S. neurona* was characterized by severe, necrotizing, mixed non-suppurative and suppurative encephalitis, which was often most severe in the cerebellum and brainstem. Encephalitis due to *S. neurona* was the primary cause of death identified in 6.7% of otters, all of which were detected during spring months (March through May). Cases were significantly clustered temporally in the spring of 2001 ($P=0.01$). Purely suppurative encephalitis was detected in only one sea otter, but the primary cause of death in this case was attributed to facial bite wounds, which caused a ganglioneuritis and ascending infection with *Archanobacterium phocae.* Encephalitis of unknown or unconfirmed cause was a primary cause of death for another 4.8% of otters examined. Overall, encephalitis of all types caused death in 28% of otters examined and contributed to death in another 18% of otters.

Similar in proportionate mortality to *T. gondii* encephalitis, infection with acanthocephalan parasites was a primary cause of death in 16.2% of otters examined, and this parasite contributed to death in another 9.5% of otters. Septic peritonitis, caused by migrating *Profilicollis* spp., was the most common consequence of heavy acanthocephalan infection (Fig. 4). This type of peritonitis was the primary cause of death in 14.3% (15/105) of otters examined and was 3.5 times more likely to have caused death in juveniles and subadults than adults or aged adults (two-sided chi square test, $P=0.03$). Acanthocephalan infection either directly caused, or was a contributing factor for, death in 40% (17/43) of the

TABLE 1. Primary and contributing causes of death in 105 southern sea otters from February 1998 to July 2001. Sex and age class distributions are provided for primary causes of death.

Cause of death[a]	Primary	Contributing	Male	Female	Juvenile	Subadult	Adult	Aged adult
Acanthocephalan infection	17	10	9	8	9	2	6	0
Toxoplasma gondii encephalitis	17	12	11	6	4	4	7	2
Cardiac disease	14	12	4	10	0	1	9	4
Shark attack	14	0	6	8	1	4	8	1
Lacerations or trauma (unknown cause)	8	0	6	2	2	0	5	1
Sarcocystis neurona encephalitis	7	0	4	3	3	2	1	1
Encephalitis - unconfirmed cause	5	7	1	4	2	0	3	0
Other intestinal disease[b]	5	0	3	2	2	2	1	0
Boat strike	5	0	5	0	1	0	2	2
Perinatal mortality	4	0	3	1	4	n/a	n/a	n/a
Starvation (early maternal separation)	4	3	1	3	4	n/a	n/a	n/a
Domoic acid intoxication	4	1	3	1	0	0	3	1
Bite wounds[c]	3	0	2	1	0	0	2	1
Gastric ulceration	2	7	1	1	0	0	2	0
Nose wounds due to mating trauma	2	9	0	2	0	0	2	0
Gunshot	2	1	2	0	0	0	2	0
Bacterial pneumonia	2	0	0	2	0	0	2	0
Sepsis (unknown cause)	2	0	2	0	0	0	1	1
Fungal pneumonia (*Coccidioides immitis*)	1	1	1	0	0	0	1	0
Gill net drowning	1	0	1	0	0	0	1	0
Delayed closure of foramen ovale	1	0	1	0	1	0	0	0
Oiling	1	0	1	0	0	0	0	1
Emaciation	0	16	—	—	—	—	—	—
Peritonitis (unknown cause)	0	3	—	—	—	—	—	—
Dental disease	0	3	—	—	—	—	—	—

[a] A total of 14 otters had two equally weighted primary causes of death. Multiple primary diagnoses were shark attack and *T. gondii* encephalitis ($n = 5$), and one otter each with shark attack and encephalitis of unconfirmed cause, shark attack and small bowel volvulus, boat strike and *T. gondii* encephalitis, domoic acid and *T. gondii* encephalitis, gill net drowning and *T. gondii* encephalitis, cardiac disease and encephalitis of unconfirmed cause, cardiac disease and *T. gondii* encephalitis, domoic acid and trauma of unknown cause, and acanthocephalan infection and encephalitis of unconfirmed cause. One otter had the three equally weighted primary causes of acanthocephalan infection, *S. neurona* encephalitis, and small bowel volvulus. A single contributing cause of death was identified in 63 otters and two contributing causes of death were identified in 23 otters.

[b] Other intestinal diseases included small bowel volvulus ($n = 2$), intussusception ($n = 1$), intestinal perforation of unknown cause ($n = 1$), and bacterial enteritis ($n = 1$).

[c] Bite wounds were not characteristic of shark attack and were most likely caused by aggressive interactions with other otters or sympatric species.

juvenile and subadult otters examined. A geographic cluster of acanthocephalan peritonitis was detected at the southern end of Monterey Bay ($P=0.02$, centered at 36°60′N, 121°88′W, Fig. 3). In this 1.8 km area, five of six carcasses recovered had acanthocephalan peritonitis as a primary cause of death, while only one case was expected if this condition had been distributed evenly across locations where carcasses were recovered along the coast. The sixth carcass recovered in this area had acanthocephalan peritonitis as a contributing cause of death.

Shark-inflicted mortality was detected in 13.3% of otters examined. All cases had bite wounds that were consistent with attack by white sharks (*Carcharodon carcharias,* Fig. 2). A significant spatial cluster of otters attacked by sharks was noted in the 80 km stretch of coastline from Point Año Nuevo to Santa Cruz ($P=0.02$, centered at 37°11′N, 122°33′W, Fig. 3). In this area, six of 10 otters recovered were killed by sharks, while only 1.3 cases of shark attack were expected had this condition been distributed evenly in areas where carcasses were recovered. Histopathologic

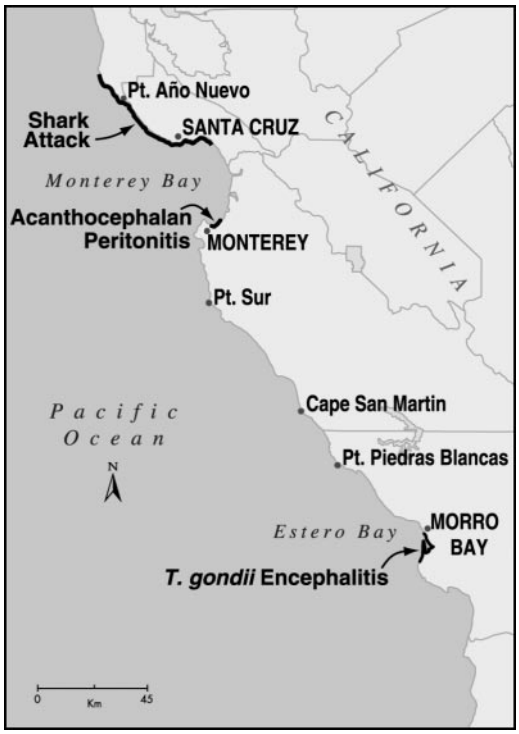

FIGURE 3. Central California showing significant clusters of major causes of mortality in sea otters from 1998 through July 2001. Each arrow points to the highlighted section of coastline where beachcast otters had a significantly higher likelihood of having the specified cause of death.

examination of brain tissue from shark-attacked otters revealed that eight of 14 otters (57%) attacked by sharks had pre-existing encephalitis. Otters with moderate to severe *T. gondii* encephalitis were 3.7 times more likely to be attacked by sharks than otters without this condition (one-sided Fisher exact test, $P=0.05$). Shark-inflicted mortality was not significantly associated with acanthocephalan infection, cardiac disease, or emaciation.

Cardiac lesions in otters were newly recognized during this study period and cardiac disease was diagnosed as a primary cause of death in 13.3% of otters examined. Cardiac lesions consisting of mild to severe non-suppurative (lymphocytic) myocarditis (Fig. 5) were a common finding in otters examined ($n=41$). These lesions were considered a cause of death only when myocarditis was accompanied by a grossly enlarged, rounded, dilated, and thin walled heart (Fig. 5) and/or strong evidence for congestive heart failure (pulmonary edema, pleural and peritoneal effusion, and hepatic congestion). Nearly all cardiac disease cases were observed in prime-aged adults or aged adults, and this condition was 3.5 times more likely to be a primary cause of death in females than males (two-sided chi square test, $P=0.04$). Severe nose wounds were

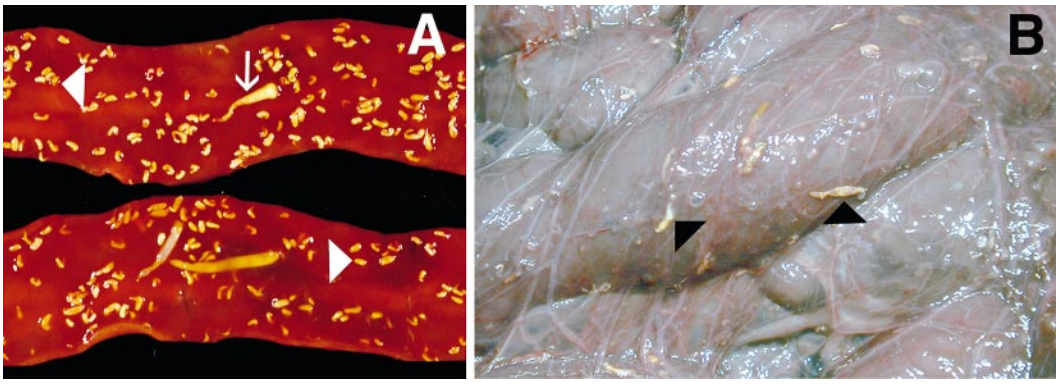

FIGURE 4. A. Mucosal surface of the small intestine in a sea otter with intestinal acanthocephalidiasis. Both *Profilicollis* spp. (arrowhead) and *Corynosoma enhydri* (arrow) are adhered to the mucosa. The smaller *Profilicollis* spp. are in various stages of mural penetration. B. Omentum from a southern sea otter with acanthocephalan peritonitis. *Profilicollis* spp. have migrated through the intestinal wall and are attached to the omentum (arrowheads), which is grossly thickened and opaque.

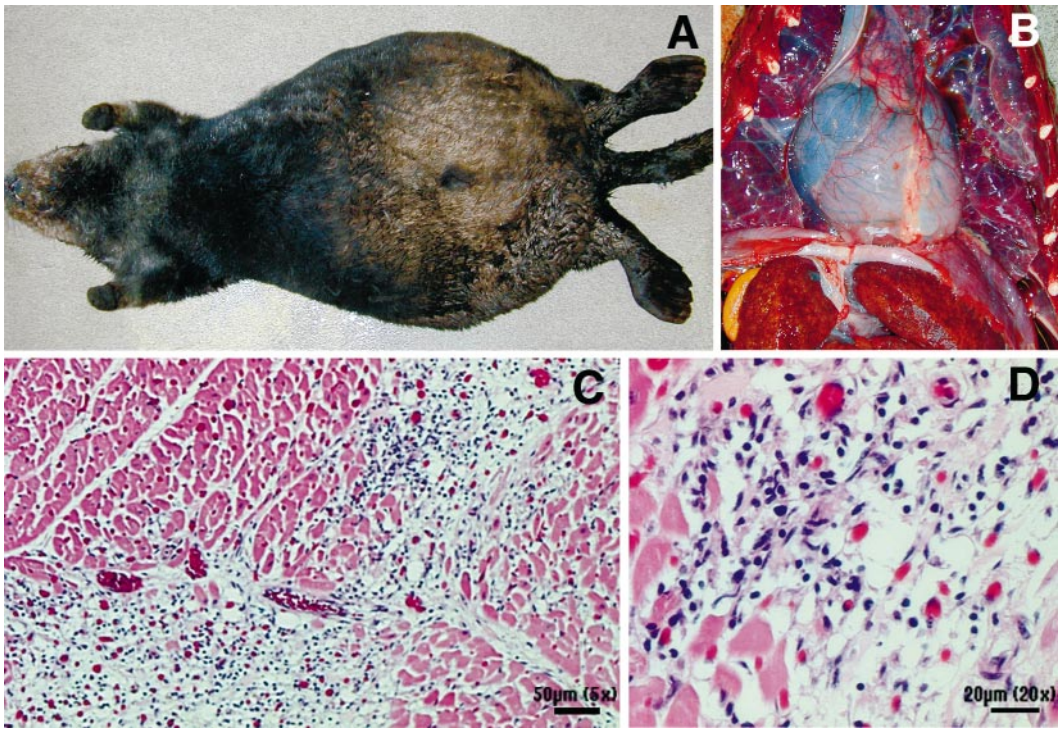

FIGURE 5. A. Southern sea otter with congestive heart failure secondary to cardiac disease. Note gross abdominal distension caused by hepatomegaly and peritoneal effusion. B. Gross photographs of the chest and abdomen from the otter in Fig. A, showing the enlarged and rounded heart. Also visible are the markedly enlarged liver, characterized by prominent rounding of the hepatic lobes, and diffuse pulmonary edema. C. HE-stained ventricular myocardium from a sea otter showing multifocal to coalescing areas of fibrosis and inflammation with accompanying myofiber loss. D. Higher magnification photomicrograph of the same site in Fig. 5C showing the predominantly lymphocytic inflammatory infiltrate.

found in half of the otters (7/14) with cardiac disease as a primary cause of death. Nose wounds are commonly acquired by females during mating (Staedler and Riedman, 1993) but also result from intraspecific aggression in males. Both male otters with severe nose wounds had cardiac disease as a primary cause of death (one-sided Fisher exact test, stratified on sex, $P<0.01$) and five of nine female otters with severe nose wounds had cardiac disease as a primary cause of death (one-sided Fisher exact test, stratified on sex, $P=0.01$). In all cases, severe nose wounds were acute or subacute and seemed to have occurred after development of cardiac disease. While significant localized geographic clusters of cardiac disease cases were not detected, otters with this condition were 5.6 times more likely to be recovered from the southern half of the California sea otter range (two-sided chi square test, $P<0.01$), which is also where the cluster of *T. gondii* encephalitis was detected. Otters diagnosed with cardiac disease were 2.9 times more likely to have concurrent *T. gondii* encephalitis than otters without cardiac disease (one-sided chi square test, $P=0.01$). Cardiac disease was not associated with any other common contributing cause of death.

While emaciation was not determined to be a primary cause of death in any otters examined, emaciation was identified as a contributing cause of death in 15% of otters. Emaciation was most significantly associated with cardiac disease (one-sided Fisher exact test, $P=0.01$) and was 3.2

times more likely to be a contributing cause of death in females than males (two-sided chi square test, $P=0.03$). Of 27 adult and aged adult females that were necropsied, nine were confirmed to be lactating at the time of death. Most lactating females (7/9) were emaciated at death and four of nine lactating females had cardiac disease as a primary cause of death.

Infectious diseases (caused by parasites, bacteria, and fungi) and diseases without a specified etiology (cardiac disease, intestinal disease, and encephalitis of unconfirmed cause) were implicated as a primary cause of death in 63.8% of otters examined. Disease was most commonly a primary cause of death in prime-aged adults ($n=30$) compared to juveniles ($n=19$), subadults ($n=10$), and aged adults ($n=8$). Parasitic diseases alone, caused by *T. gondii, S. neurona,* and *Profilicollis* spp, were determined to be a primary cause of death in 38.1% of the otters examined.

DISCUSSION

Encephalitis, caused by *T. gondii* and *S. neurona,* was first recognized as a source of mortality in sea otters only after detailed necropsies were begun in 1992 (Cole et al., 2000; Lindsay et al., 2000; Miller et al., 2001a). Because encephalitis can only be diagnosed by microscopic examination of brain tissue, this cause of death was likely missed in the past when carcasses were not examined in such detail. However, the reported percent of mortality attributed to protozoal encephalitis appears to have increased substantially over the short period during which detailed examinations have been undertaken. Of otters examined from 1992–95, 8.5% had protozoal encephalitis while 22.9% of otters we examined had protozoal encephalitis as a primary cause of death. This apparent increase in prevalence may be a consequence of improved diagnostics, a difference in criteria for establishing this diagnosis among laboratories and pathologists, or temporal variation in the occurrence of this disease. Other causes of encephalitis should also be considered, particularly for cases with an as yet unconfirmed cause. However, the magnitude of this most recent temporal trend certainly warrants attention, particularly as the increased prevalence of this condition coincides with a period of population decline.

Protozoal infections in sea otters may be examples of spillover of land-based pathogens into the marine ecosystem because the only identified definitive hosts for *T. gondii* and *S. neurona* are felids and opossums (*Didelphis virginiana*), respectively. The spatial cluster of mortality due to *T. gondii* encephalitis in Estero Bay may be a consequence of local factors, such as increased sea otter exposure to *T. gondii,* enhanced parasite virulence, or increased sea otter susceptibility in this particular area. In a separate study, sea otter carcasses found in Estero Bay had higher prevalence of antibodies to *T. gondii* than otters sampled elsewhere (Miller et al., 2002), suggesting that mortality may be due to high levels of parasite exposure. Seasonal peaks of *T. gondii* encephalitis mortality in otters were not detected even though coastal freshwater runoff has been associated with increased odds of exposure to *T. gondii* in sea otters (Miller et al., 2002). Runoff is highest during the winter rainy season and spring in California. However, many unrecognized environmental and host factors could affect the timing of death from this disease. Otters with *S. neurona* encephalitis, which may be a more acutely severe and rapidly progressive disease, stranded only in spring months (March through May), which follows maximal seasonal runoff and coincides with the season when opossums were more likely to shed sporocysts (Rickard et al., 2001).

While infection with *T. gondii* is common in terrestrial mammals, it is usually subclinical in immune competent hosts (Frenkel, 1988). Disseminated systemic disease with severe brain infection is more typical of immune suppressed humans, such as HIV infected AIDS patients (Arnold et al., 1997). Fatal *T. gondii* infections

have been reported in neotropical marsupials and nonhuman primates, which evolved in ecologic isolation from domestic cats (Frenkel, 1988). Expansion of domestic cat and opossum populations and decreased natural filtration of watershed runoff through coastal estuaries may have increased sea otter exposure to a pathogen for which they are immunologically ill-prepared. These protozoal pathogens are not likely to have been abundant in the marine environment centuries ago. The substantial proportionate mortality caused by protozoal encephalitis in juvenile and prime aged adult otters is of concern, particularly because *T. gondii* encephalitis was shown here to be associated with two other important causes of death, shark attack and cardiac disease. If *T. gondii* infection increases the risk of death from shark attack or cardiac disease, this parasite may have a complex but critical role in a high level of mortality in sea otters.

Toxoplasma gondii infection may have other deleterious effects that are difficult to document by the methods used here. Fetal infection with *T. gondii* is associated with serious birth defects and a high frequency of abortion in terrestrial animals and humans. These effects would be difficult to detect in free-living sea otters because aborted fetuses and pups that die shortly after birth are less likely to be found in fresh condition. Underlying causes for the perinatal mortality noted in recovered pup carcasses could not be determined but all were near full term pups that died shortly after birth and none had pathologic findings consistent with protozoal infection.

The high prevalence of pre-existing *T. gondii* encephalitis in shark attacked otters suggests that otters with encephalitis may exhibit aberrant behavior that renders them more vulnerable to attack by sharks. Otters with protozoal encephalitis frequently exhibit fine muscle tremors, recurrent seizures, dull mentation, and decreased or abnormal motor function (M. Murray, pers. comm.). Neurologic dysfunction might cause otters to be less able to evade attacks, to move offshore out of the protected areas, or to attract shark attention through abnormal movements and seizures. Wounds inflicted upon otters by sharks were typically large gashes with minimal soft tissue removal, consistent with open-mouth slashing or raking by sharks (Fig. 2). This bite pattern is similar to that seen when sharks attack humans (Byard et al., 2000) and is not consistent with a bite pattern used for intended prey. Sharks feeding on pinniped prey often remove large pieces of soft tissue (Lucas and Stobo, 2000). The wound pattern observed in otters suggests that they may not be intended prey and the high percent of shark-attacked otters with pre-existing encephalitis is consistent with the hypothesis that sharks are more likely to attack otters if they are acting abnormally.

The area from Santa Cruz to Point Año Nuevo with high shark-related mortality in sea otters is known for white shark predation on pinnipeds (Long et al., 1996). Shark-inflicted mortality has been long-recognized as an important cause of mortality in sea otters since carcasses were first evaluated in 1968 and the number of shark-bitten beachcast carcasses has varied considerably by year (Ames et al., 1996). However, the annual proportion of shark-attacked sea otter carcasses relative to the annual population count has increased through time particularly during periods of population decline (Estes et al., 2003). Because interactions with sharks may be modified by encephalitis in otters, prevalence of shark attack may be linked to prevalence of encephalitis. Both shark attack and encephalitis may be under-represented as a cause of death if carcasses with penetrating wounds from shark bites are more likely to sink than wash ashore (Lucas and Stobo, 2000) or if otters are consumed by sharks, although the latter has never been observed.

Cardiac disease has not previously been reported in sea otters and the substantial percent of deaths attributed to cardiac dis-

ease was unexpected. Stress-related cardiac disease has been documented in stranded cetaceans (Turnbull and Cowan, 1998), but the contraction band-necrosis of myocardium detected in dolphins and whales differed from the pattern of nonsuppurative myocarditis and gross cardiac dilation observed in sea otters. The inflammatory nature of the cardiac lesions noted here suggests an infectious etiology although infectious agents were not detected during microscopic examination of sea otter myocardium. Our observation that otters with cardiac disease are more likely to have concurrent *T. gondii* encephalitis implies that these two conditions are causally linked or are somehow coupled by an unknown mechanism. Rare accounts of myocarditis in animals infected with *T. gondii* have been reported in dolphins (Inskeep et al., 1990), a northern fur seal (*Callorhinus ursinus*; Holshuh et al., 1985), and a California sea lion (*Zalophus californianus*; Migaki et al., 1977), although disseminated toxoplasmosis with protozoal cysts in cardiac myofibers were observed in all cases. In humans, *T. gondii*-related cardiomyopathy has been documented in AIDS patients (Matturri et al., 1990) and immune-suppressed heart transplant patients (Arnold et al., 1997). Efforts are currently being directed at understanding the immunopathology of toxoplasmosis in sea otters, improving detection methods for protozoal stages, and evaluating other possible etiologies and risk factors for cardiac disease.

Cardiac disease was a common cause of death in female otters and was often associated with severe nose wounds and emaciation. Emaciation and severe nose wounds may be a secondary consequence of the debilitating effects of heart failure, which could prevent afflicted otters from foraging effectively and defending themselves against aggressive males. Adult females appear to be highly susceptible to both mating trauma and emaciation, particularly at the end of lactation, possibly because they are at a nutritional disadvantage after caring for a pup and entering estrus, which attracts male attention. The transition from pup care and lactation to estrus and breeding appears to be a particularly vulnerable period for adult female sea otters in California.

Acanthocephalan peritonitis continues to be a substantial cause of mortality in sea otters, and the recent prevalence (16.2%) is similar to the 14% reported from 1992–95 (Thomas and Cole, 1996). Sand crabs (*Emerita analoga*) and possibly spiny mole crabs (*Blepharipoda occidentalis*) serve as intermediate hosts for these acanthocephalan parasites (Hennessy and Morejohn, 1977), and these crabs are found in predominantly sandy habitat. It is therefore likely that otters foraging in sandy bays will have a high level of exposure to *Profilicollis* spp. As expected, mortality due to acanthocephalan peritonitis during this study period was detected exclusively in carcasses retrieved from Monterey Bay and Estero Bay, both of which are largely sandy habitat. While sand crabs may not be preferred sea otter prey, they are abundant and easy to capture. Juvenile otters may ingest large numbers of these prey species when they are first searching for suitable home-range habitat and learning to forage on their own, which may explain the high level of exposure and subsequent mortality in this age class.

Even though acanthocephalan peritonitis is readily apparent at necropsy through gross evidence of peritonitis associated with parasites in the abdominal cavity, this condition was reported only once in necropsies performed on otters from 1968–76 (Hennessy and Morejohn, 1977), suggesting that fatal acanthocephalan infections may have increased in prevalence. The apparent increased prevalence of acanthocephalan peritonitis may reflect increased host susceptibility to infection or increased exposure through a shift in preferred prey availability, intermediate host abundance, foraging behavior, or habitat use. Southerly otter range expansion into the predominantly sandy habitat south of Point

Estero is unlikely to have contributed substantially to the higher prevalence of this condition in recent years because 80% of the otters with fatal acanthocephalan infections from 1998–2001 were retrieved from Monterey Bay, which has been occupied by sea otters since the 1970s (Riedman and Estes, 1990).

Domoic acid intoxication has not been previously reported in sea otters and necropsy findings and preliminary laboratory tests suggest that domoic acid intoxication was a primary cause of death in four otters examined here. Domoic acid is a marine biotoxin produced by *Pseudonitzchia* spp. and is known to cause severe neurologic dysfunction and death in California sea lions (Scholin et al., 2000) and seabirds (Work, 1993). Domoic acid exposure caused characteristic brain and cardiac lesions in sea lions (Scholin et al., 2000), and histologic lesions associated with domoic acid exposure in sea otters are currently being evaluated. Domoic acid intoxication may be established as a more common cause of mortality in sea otters once diagnostic capabilities are refined and screening is performed more routinely.

Coccidioidomycosis, caused by the soil fungus *C. immitis* was diagnosed as a primary cause of death in only one otter (1%) during our study period. In otters examined from 1992–95, eight cases had severe disseminated coccidioidomycosis (Thomas and Cole, 1996), so there may be some variation in mortality due to this pathogen.

Our approach of identifying more than one primary cause of death in sea otters with two equally severe, yet independent, causes of death has resulted in slightly higher proportionate mortality for some conditions than if diagnoses had been limited to one primary cause of death per otter. This strategy allowed for an unbiased account of important causes of death, but the proportionate mortality reported here may not be comparable to mortality studies using more traditional methods. Also, many of these causes of mortality may be subject to substantial inter-annual variation and other causes of mortality are likely to be identified retrospectively as new diagnostic techniques are developed and implemented as screening tools.

In this study, carcasses were recovered primarily along accessible sandy shores. Sea otter carcasses are not likely to wash ashore along rocky precipitous coastlines and those that do are not likely to be recovered while in fresh condition because most of these areas are not readily accessible. Therefore, very little is known of causes of mortality in sea otters living along the rocky and remote coastline in the center of the sea otter range in California. A directed search effort would be necessary to collect fresh carcasses from these unpopulated and inaccessible areas. An estimated one-half of all recently deceased sea otters were recovered as beachcast carcasses, and one-fourth of these were retrieved in adequately fresh condition to allow detailed necropsy for specific cause of death determination (Estes et al., 2003). Based on these estimates and the specific inclusion criteria for this study, approximately one of ten deceased southern sea otters was evaluated. Because the age distribution of carcasses reported here closely matches the age distribution of all carcasses recovered recently (Estes et al., 2003), the carcasses included in this study are likely to be representative of all recovered carcasses. Consequently, the major causes of death reported here are probably similar to what would be observed if detailed cause of death information could be obtained from decomposed or heavily scavenged carcasses. While a relatively high proportion of the population was available for cause of death determination, certain causes of death, such as drowning due to accidental entanglement in fish traps or nets, may be under-represented in beachcast carcasses. These carcasses may be less likely to float and wash ashore prior to decomposing, and drowning is very difficult to document even with detailed necropsy. Therefore, the cause-specific proportionate mortality reported here may

not be directly referable to the southern sea otter population.

Identification of pathogens responsible for substantial morbidity and mortality in sea otters and the geographic distribution of these pathogens is an important first step toward understanding the role of population health in the recovery of this threatened species. However to fully understand the underlying mechanisms creating the observed pattern of mortality, factors that may be affecting nutrition, immune system competency, and sources of increased pathogen exposure must be evaluated. Studies have detected elevated tissue levels of potentially immunotoxic contaminants in southern sea otters with infectious disease (Kannan et al., 1998; Nakata et al., 1998), but immune system function in wild animals is difficult to measure because species-specific tests must be developed and validated for different age classes.

The high percent of prime-aged adults among beachcast sea otter carcasses in California, and the very high prevalence of disease noted in this age class are not consistent with a healthy population destined for recovery. Sea otter populations experiencing a decline from increased levels of mortality typically have a high percent of prime-aged adults among beachcast carcasses (Estes et al., 2003). Certainly, the prevalence of acanthocephalan-related disease in juveniles and the prevalence of *T. gondii* encephalitis, cardiac disease, and shark attack in prime aged adults have the potential to exert a population-level effect and may be limiting sea otter population growth in California. The underlying processes promoting increased levels of parasitic disease in sea otters should be a focus of future investigation. Special attention should be given to non-native terrestrial species that are endemically infected with protozoal pathogens and have the capacity to serve as a reservoir for persistent exposure and infection in sea otters. Recovery of the southern sea otter population may face a significant challenge as this isolated population struggles to expand in a near-shore system that may have been substantially altered in terms of prey abundance, water quality, and pathogens in the time since the near-extirpation and early recovery of sea otters.

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