

UC Office of the President

Coastal Environmental Quality Initiative

Title

Southern sea otters (*Enhydra lutris nereis*) and emergent disease in the near-shore marine ecosystem: Assessment of spatial trends in cause-specific mortality from 1998-2001 and evaluation of an aerial survey method for surveillance of trends in morta...

Permalink

<https://escholarship.org/uc/item/8245j1h0>

Author

Kreuder, Christine

Publication Date

2004-11-29

PROJECT TITLE: Southern sea otters (*Enhydra lutris nereis*) and emergent disease in the near-shore marine ecosystem: Assessment of spatial trends in cause-specific mortality from 1998-2001 and evaluation of an aerial survey method for surveillance of trends in mortality.

INVESTIGATORS: Christine Kreuder, Jonna A.K. Mazet, Tim E. Carpenter Pat A. Conrad, and Linda J. Lowenstine (Wildlife Health Center, School of Veterinary Medicine, University of California, Davis, California 95616); Melissa A. Miller, David A. Jessup, Michael D. Harris, and Jack A. Ames (Marine Wildlife Veterinary Care and Research Center, California Department of Fish and Game, Santa Cruz, California 95060).

Collaborators and Research Assistants: Michelle Staedler, Tim Tinker, Julie Stewart, Mike Kenner, Krista Hanni, George Troup, Terri Nicholson, Ellen Faurot-Daniels, Brian Hatfield Erin Dodd, Debbie Brownstein, and Eva Berberich.

SUMMARY OF PROJECT ACTIVITIES AND FINDINGS:

Project background:

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 (*Enhydra lutris nereis*) are a useful indicator of near-shore marine ecosystem health because they are heavily exposed to human activity in coastal California 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.G.S., unpublished 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. 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. Furthermore, the aerial and shore survey methods that are currently used to count part of sea otter population were compared and evaluated under a variety of viewing conditions in order to permit more accurate assessment of trends in distribution and abundance.

Project # 1: Evaluation of Causes of Mortality

Project Activities

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).

Veterinary pathologists at CDFG's Marine Wildlife Veterinary Care and Research Center (MWVCRC, Santa Cruz, California, USA) and the University of California (UC) School of Veterinary Medicine (Davis, California, USA) examined all freshly dead otters (deceased for 4 days or less) from February 1998 through June 2001. All otters received a complete gross necropsy, as well as microscopic examination of major tissues. Laboratory evaluation of bacterial, fungal and parasite samples and toxicologic screening for domoic acid were performed when indicated. 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. In general, causes of death were diagnosed based on the pathologists' interpretation of gross and microscopic findings in conjunction with any pertinent laboratory results. 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.

Cause-specific 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. The distribution of the four most common causes of death among gender and age classes was evaluated by the standard two-sided chi square test of independence. 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) and the spatial scan statistic (Kulldorf and Nagarwalla 1995).

Project Findings

Sea otter carcass retrieval was spatially clustered along the accessible sandy shores of Monterey Bay and Estero Bay. 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). Carcasses recovered consisted predominantly of prime-aged adults (46.7%), with fewer juveniles (29.5%), subadults (11.4%), and aged adults (12.4%). While 25 different primary causes of death were identified, 53.3% of otters (56/105) died from one of four major causes: *Toxoplasma gondii* brain infection, 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.

Brain inflammation (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 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 (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 due to *Sarcocystis neurona* was the primary cause of death identified in 6.7% of otters, all of which were detected during spring months (March through May). 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. Sea otter carcasses found in Estero Bay had higher *T. gondii* seroprevalence than live and dead otters sampled elsewhere (Miller et al. 2002), suggesting that mortality may be due to high levels of parasite exposure. 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 also been reported in neotropical marsupials and non-human primates, which evolved in ecological 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. 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. Other causes of encephalitis must also be considered, particularly for cases with an as yet unconfirmed cause.

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. Infection of the abdominal cavity (septic peritonitis), caused by migrating *Profilicollis spp.*, was the most common consequence of heavy acanthocephalan infection. This type of infection 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. Sea otters become infected with this parasite when eating sand crabs (*Emerita analoga*) and possibly spiny mole crabs (*Blepharipoda occidentalis*) (Hennessy and Morejohn 1977), which may not be preferred sea otter prey, but they are abundant and easy to capture. Therefore, 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. A geographic cluster of acanthocephalan peritonitis was detected at the southern end of Monterey Bay (centered at 36°60' N, -121°88' W, Fig. 3). In this 1.8 km area, five out of six carcasses recovered had acanthocephalan peritonitis as a primary cause of death, while only 1 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. The sand and mole crab intermediate hosts for these acanthocephalan parasites are found in predominantly sandy habitat. It is therefore expected that otters foraging in sandy bays, such as Monterey Bay, will have a high level of exposure to *Profilicollis spp.*

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*). 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 (centered at 37°11' N, 122°33' W, Fig. 3), which is an area known for white shark predation on pinnipeds (Long et al. 1996). In this area, six out of 10 otters recovered were killed by sharks, while only 1.3 cases of shark attack were expected if this condition been distributed evenly in areas where carcasses were recovered. 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. Shark-inflicted mortality was not significantly associated with acanthocephalan infection, cardiac disease or emaciation. 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, personal communication). 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.

Cardiac disease in otters was newly recognized during this study period and was diagnosed as a primary cause of death in 13.3% of otters examined. Sea otters had inflammation of heart tissue (myocarditis) and, in some cases, heart inflammation was accompanied by an enlarged, rounded, heart (cardiomyopathy) with 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. Separate spatial-temporal clusters of myocarditis and DCM were identified in the southern aspect of the sea otter range from May to November 2000. Risk factors for myocarditis and cardiomyopathy were investigated in detail since this condition was newly detected in sea otters with no identified underlying cause. Significant risk factors associated with myocarditis included adult age, good nutritional body condition, and exposure to domoic acid and *S. neurona*. Domoic acid, a marine toxin produced by *Pseudonitzschia australis*, is commonly produced along coastal California. Myocarditis associated with domoic acid occurred predominantly in the southern part of the range, whereas myocarditis associated with *S. neurona* occurred in the northern part of the range. Adult age and suspected previous exposure to domoic acid were identified as major risk factors for cardiomyopathy. Otters with cardiomyopathy also had significantly lower concentrations of myocardial L-carnitine than otters with myocarditis and otters with normal hearts at necropsy. Domoic acid toxicosis and infection with *S. neurona* are likely to be two important causes of myocarditis in sea otters. Domoic acid-induced myocarditis appears to progress to DCM and depletion of myocardial L-carnitine may play a key role in this pathogenesis.

Overall, disease was 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 ($\underline{n} = 30$) compared to juveniles ($\underline{n} = 19$), subadults ($\underline{n} = 10$) and aged adults ($\underline{n} = 8$). 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. 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.

Nonetheless, detailed examination of sea otter carcasses found along the coast of California has provided an exceptional opportunity to determine important causes of mortality and geographic patterns of disease in a threatened marine mammal species. 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. The high percentage 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. The underlying processes promoting increased levels of parasitic disease and domoic acid exposure in sea otters should be a focus of future investigation. 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 be substantially altered in terms of prey abundance, water quality and pathogens in the time since the near-extirpation and early recovery of sea otters.

Project # 2: Evaluation of the Aerial Survey Method for Measuring Sea Otter Abundance *Project Activities*

We estimated detection probability for both the shore and aerial survey under a range of viewing conditions. All individual and environmental factors, such as otter behavior and age, survey site characteristics, and weather/sea state conditions that were suspected to influence sea otter counts by either the shore or aerial survey method, were scored by each observer. Information collected on individual sea otters included age class (adult vs pup), activity (resting, swimming along the surface, diving/feeding, grooming), and location relative to an estimated line 900m distant from shore. Survey site characteristics included habitat type, site elevation for shore-based observer, kelp density, kelp type, glare, wind, and wave height. Twelve 1.2 km areas of coastline within the northern part of the sea otter range were selected to be representative of the variety of common sea otter habitat. All twelve sites were surveyed simultaneously by the aerial and shore methods once during each of ten study days. Independently and concurrently conducted shore-based counts were performed as previously described (Estes and Jameson 1988). All sea otter sightings by both shore and aerial observers were recorded on detailed maps depicting kelp bed configurations (Ecoscan, Resource Data, Freedom, California, 1990). The maps of individual otter sightings were then compared at the end of each survey day to determine which individual otters were counted by both methods, by the shore survey only, or by the aerial survey only. Multinomial logistic regression was used to test the effect of visibility factors on otter detection by the two survey methods, with the condition that otters were detected by at least one method.

Project Findings

The shore and aerial surveys recorded a total of 1,687 sea otter sightings in the survey areas during the ten study days. A total of 774 sea otter sightings were made by both survey methods, 632 sightings were by the shore survey only, and 281 sightings were by the aerial survey only. Based on a crude comparison of overall total counts, the aerial survey counted 75% as many otters as the shore survey. Detection of individual sea otters by each survey method varied considerably according to sea otter age, activity, distance from shore, group size, and weather conditions relating to glare, wind and wave height.

Overall, the shore survey observed 83.3% of all otters that were detectable by the combination of the shore and aerial survey methods (Table 1). The shore survey was generally able to detect more otters than the aerial survey, and was substantially more effective at detecting otters within 900m from shore than the aerial survey. During the 10 survey days, the shore survey observed 92% of the all otters counted within 900 m, but only 58% of all otters past 900m (Table 1). Once all other visibility factors were adjusted for, the shore survey was estimated to detect 87% of adult otters within 900m from shore, but only 31% of adult otters past 900 m from

shore. Therefore, efficacy of the shore survey drops off by 56% for otters past 900m from shore.

Within 900m, the shore survey was far more likely to detect pups and diving otters than the aerial survey. In fact, otters that were within 900m were more likely to be seen by the shore survey than the aerial survey regardless of the individual characteristics or viewing conditions that applied, except in one survey site with mixed habitat. This site contained both numerous offshore rocks and an embayment which lacked any elevation for suitable vantage points from shore. The effect of distance from shore on detection by the shore survey was also modified by glare and waves higher than 1m. Otters within 900m were more observable by the shore survey than the aerial survey when waves were < 1m. However, otters past 900m had an estimated detection probability of only 25% when waves were above 1m, which is slightly lower than the expected negative effect from distance from shore alone. When waves are higher than 1m, otters that are far from shore are only intermittently observable from shore when they are at the crest of the swell, thereby decreasing detectability. The shore survey was impacted negatively by glare even within 900m, but only 8% of the otters past 900m were estimated to be detectable by the shore survey under glare conditions. Most likely glare substantially worsened shore detectability of otters past 900m because otters at a distance are observed through a telescope, which is very difficult under conditions with glare. While the shore survey was found to be much more useful than the aerial survey in conditions with high wind, detection of otters by the shore survey dropped off substantially for otters past 900m under conditions with both high wind and waves over 1m.

The aerial survey observed 62.5% of all otters that were detected by the two survey methods (Table 1). During the 10 study days, the aerial survey observed 56% of otter sightings within 900m and 83% of the otter sightings past 900m from shore (Table 1). When the effects of all other visibility factors were taken into account, an adult otter past 900m from shore had an estimated aerial survey detection probability that was 15% higher than the detection probability for an adult within 900m from shore, suggesting that the aerial survey counted a larger share of all otters observed past 900m. The high speed and altitude at which the aerial survey is conducted may explain why pups and diving otters were less likely to be observed by the aerial survey. The aerial survey was less effective at counting otters at sites designated as rocky habitat compared to sites designated as open water habitat. Airspeed and altitude may also contribute to obscuring otters near large obstructive rocks located close to shore. Rocky, densely rocky, and mixed habitat sites generally had dense kelp canopies with abundant *Nereocystis* and some of the effect ascribed to rocky habitat may also be due to kelp density or kelp type.

Group size had a similar, mostly minimal effect on detection probability by the aerial and shore surveys. High speed and altitude may have been less likely to affect detection of individuals within large groups because most large groups, particularly those with over 20 otters, were circled by the aircraft until an adequate count could be completed. Sea otter detection was more affected by increasing wind speed for the aerial survey than the shore survey. Aerial surveys conducted in winds at 1 to 3 knots had an estimated detection probability of 65%, which was 11% lower than for surveys conducted with no wind at all. While wind speeds up to 16 knots had a similar negative effect on detectability, wind speeds over 17 knots had an even more substantial negative effect on detectability. When wind speed exceeded 17 knots, sightability of otters deteriorated in general and only slightly more than half of the detectable otters were estimated to be observed by the aerial survey both within and past 900m.

Our findings suggest that detection of sea otters by the shore and aerial survey are heavily influenced by a number of otter-related, site-related, and weather-related viewing conditions. Sea otter population count methods can be optimized by combining survey methods and using each at appropriate distances under the ideal conditions described to ensure high detection probabilities and more accurate population counts. By quantifying detection probabilities under a range of viewing conditions, we have enabled more confidence in the aerial survey as we are now better able to understand when this survey method is likely to provide useful information.

The range of detection probabilities shown here for shore and aerial surveys suggests that there is a high potential for variability in population abundance counts when these survey methods are used. Surveys conducted under different viewing conditions are likely to produce very different abundance counts if no adjustment is made for viewing conditions. Counts performed over a short period of time, in consistently good or bad viewing conditions, should be expected to produce outlier abundance counts. Fairly large and abrupt changes in abundance counts over time should be interpreted with caution if counts were performed under substantially different viewing conditions. Surveys should record a standardized measure of all significant viewing conditions, so that future population estimates may be adjusted if necessary. Quantification of the factors influencing wildlife population surveys is an important first step towards producing reliable and unbiased population counts that are critical for evaluating long-term trends in wildlife abundance and distribution.

Table 1: Distribution of sea otter sightings by survey method under viewing conditions of interest during 10 survey days in Northern California.

	Both Surveys	Shore Survey Only	Aerial Survey Only	Total
Age				
Adult	723	505	271	1,499
Pup	51	127	10	188
Activity				
Resting	689	468	202	1,359
Diving	40	112	12	164
Surface swimming	45	52	27	124
Unknown	0	0	40	40
Group Size				
Single	264	336	139	739
Group of 2	53	94	40	187
Group 3 to 9	291	152	76	519
Group of 10 to 20	116	38	19	173
Group over 20	50	12	7	69
Distance from Shore				
Within 900m	598	558	99	1,255
Past 900m	176	74	182	432
Total Observed	774	632	281	1,687

PUBLICATIONS RESULTING FROM THIS WORK:

Patterns of mortality in southern sea otters (*Enhydra lutris nereis*) from 1998 – 2001. C Kreuder, MA Miller, DA Jessup, LJ Lowenstine, M D Harris, JA Ames, TE Carpenter, PA Conrad, and JAK Mazet. *Journal of Wildlife Diseases* 39(3):495-509, 2003.

Risk factors for myocarditis and dilated cardiomyopathy in southern sea otters (*Enhydra lutris nereis*). C Kreuder, MA Miller, LJ Lowenstine, PA Conrad, TE Carpenter, DA Jessup and JAK Mazet. *American Journal of Veterinary Research* (in press).

Variability in detection probability of sea otters: A model for evaluating the relative efficacy of survey methods. C Kreuder, WO Johnson, JA Estes, MD Harris, JA Ames, TE Carpenter, and JAK Mazet. *Ecology* (in preparation).

LITERATURE CITED

- Arnold, S. J., M. C. Kinney, M. S. McCormick, S. Dummer, and M. A. Scott. 1997. Disseminated toxoplasmosis: Unusual presentations in the immunocompromised host. *Archives of Pathology and Laboratory Medicine* **121**:869-873.
- Carpenter, T. E. 2001. Methods to investigate spatial and temporal clustering in veterinary epidemiology. *Preventive Veterinary Medicine* **48**:303-320.
- Carretta, J. V., J. Barlow, K. A. Forney, M. M. Muto, and J. Baker. 2001. U.S. Pacific Marine Mammal Stock Assessments. NMFS-SWFSC-317, NOAA Technical Memorandum.
- Daszak, P., A. A. Cunningham, and A. D. Hyatt. 2001. Anthropogenic environmental change and the emergence of infectious diseases in wildlife. *Acta Tropica* **78**:103-116.
- Estes, J. A. 1990. Growth and equilibrium in sea otter populations. *Journal of Animal Ecology* **59**:385-402.
- Estes, J. A., and R. J. Jameson. 1988. A double-survey estimate for sighting probability of sea otters in California. *Journal of Wildlife Management* **52**:70-76.
- Fair, P. A., and P. R. Becker. 2000. Review of stress in marine mammals. *Journal of Aquatic Ecosystem Stress and Recovery* **7**:335-354.
- Frenkel, J. K. 1988. Pathophysiology of toxoplasmosis. *Parasitology Today* **4**:273-278.
- Harvell, C. D., K. Kim, J. M. Burkholder, R. R. Colwell, P. R. Epstein, D. J. Grimes, E. E. Hofmann, E. K. Lipp, A. D. M. E. Osterhaus, R. M. Overstreet, J. W. Porter, G. W. Smith, and G. R. Vasta. 1999. Emerging marine diseases-climate links and anthropogenic factors. *Science* **285**:1505-1510.
- Hennessy, S., and G. Morejohn. 1977. Acanthocephalan parasites of the sea otter, *Enhydra lutris*, off coastal California. *California Fish and Game* **63**:268-272.
- Kulldorf, M., and N. Nagarwalla. 1995. Spatial disease clusters: detection and inference. *Statistics in Medicine* **14**:799-810.
- Long, D. J., K. D. Hanni, P. Pyle, J. Roletto, R. E. Jones, and R. Bandar. 1996. White shark predation on four pinniped species in central California waters: geographic and temporal patterns inferred from wounded carcasses. Pages 263-274 in A. P. Klimley and D. Ainley, G. editors. *Great White Sharks: The biology of *Carcharodon carcharias**. Academic Press, London.
- Miller, M. A., I. Gardner, C. Kreuder, D. Paradies, K. Worcester, D. Jessup, E. Dodd, M. Harris, J. Ames, A. Packham, and P. Conrad. 2002. Coastal freshwater runoff is a risk factor for *Toxoplasma gondii* infection of southern sea otters (*Enhydra lutris nereis*). *International Journal for Parasitology*:997-1006.
- Monson, D. H., J. A. Estes, J. L. Bodkin, and D. B. Siniff. 2000. Life history plasticity and population regulation in sea otters. *Oikos* **90**:457-468.
- Riedman, M. L., J. A. Estes, M. M. Staedler, A. A. Giles, and D. R. Carlson. 1994. Breeding patterns and reproductive success of California sea otters. *Journal of Wildlife Management* **58**:391-399.
- Ross, P., R. De Swart, R. Addison, H. Van Loveren, J. Vos, and A. Osterhaus. 1996. Contaminant-induced immunotoxicity in harbour seals: Wildlife at risk? *Toxicology* **112**:157-169.