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The Risk of Disease and Threats to the Wild Population

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The growth of the southern sea otter population has been steady, but slow in comparison to Alaskan subspecies, and range expansion in California has faltered. Slower growth is occurring in California despite birth rates comparable to those in Alaska, so biologists have reasoned that mortality is hindering the growth of the California population (Riedman and Estes 1990; see Estes et al., this issue). In order to investigate this issue, research efforts have been directed toward identifying the causes of death in southern sea otters.

Several major causes of mortality in the southern population were recognized in the past and are currently being addressed. These include protections from overharvesting and accidental entanglements in fishing gear, as well as oil spill prevention and contingencies. Still to date, the problem of slow population growth and expansion persists.

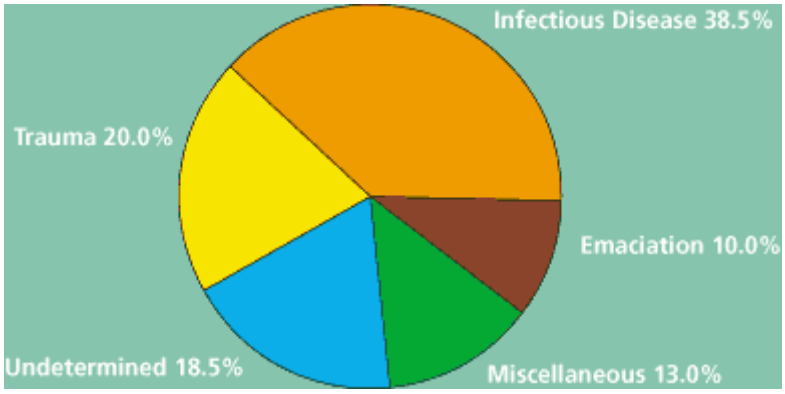
Table 1. Sea otter mortalities from 1968-1989 (n=1,680), taken from Ames 1983 and Riedman and Estes 1990.

Trauma	18.9%
shark attack	12.0%
gunshot	4.6%
mating	2.3%
Fishing net/line	4.6%
Dependent animals	16.5%
Natural causes	4.0%
Undetermined	56.0%

The California Department of Fish and Game has studied sea otter mortality since 1968. A salvage network, maintained through the cooperation of State and Federal agencies and scientific institutions, verified beach-cast carcasses and examined them to estimate the causes of death. By 1989 nearly 1700 dead sea otters had been documented (see Table 1; Ames et al. 1983, unpublished data; Riedman and Estes 1990). Substantial mortality from net drownings was identified and the prevalence of trauma from predators, shooting, fighting and mating activities were documented. Pup abandonment and a variety of miscellaneous conditions and diseases were recorded. Since 1992, laboratory diagnostic efforts at the National Wildlife Health Center (NWHC), Madison, Wisconsin, supplemented the stranding network's activities in California, so that knowledge about the causes of mortality has been further refined. It is these recent perspectives on the causes of mortality in the southern sea otter that are outlined here, along with consideration of their implications for sea otters and the Pacific marine ecosystem.

Causes of mortality

Since 1992 approximately 50 southern sea otter carcasses per year were examined at the NWHC as part of a 5 year intensive necropsy study. Carcasses were selected for the NWHC study on the basis of good post mortem condition from the approximately 110 to 160 carcasses found per year in California. At the NWHC, microscopic examination supplemented gross examination of the carcasses and a variety of laboratory techniques in bacteriology, virology, parasitology, and toxicology were conducted to arrive at a diagnosis. By this means a greater proportion of the animals with inapparent causes of death were diagnosed. Only a preliminary analysis of the 1992-95 data has been done, but some interesting patterns are emerging from the 195 cases studied (and additional samples have been collected through 1996). The proportionate causes of mortality identified at the NWHC bore similarities to past data but also had interesting differences (see Figure 1). Various parasitic, fungal, or bacterial diseases caused the deaths of 38.5% of the sea otters examined at the NWHC. Twenty percent died from traumatic injuries. Another 10% were emaciated at death and no specific cause for their debility could be identified. Miscellaneous conditions, such as neoplasia, or gastrointestinal or urinary obstructions, accounted for 13% of the mortality. In 18.5% of the animals no cause of death could be ascertained.



Traumatic causes, emaciation and a variety of miscellaneous conditions were similar to those described in sea otters in the past. Ames summarized the causes and distribution of traumatic injuries in sea otters examined from 1968 to 1989 (as presented in Riedman and Estes 1990). The most frequently identified cause of trauma in the past was shark attack (Ames and Morejohn 1980); the shark-bitten sea otters were collected throughout the range but the largest proportion were found north of Point Sur. In the recent study shark attack was also the most frequently diagnosed source of trauma (7% of all mortality, n=14), and more than 70% of the shark-bitten sea otters were found north of Point Sur. In the past, gunshot injuries were documented predominately south of Cambria. Ames speculated that the frequency of shark attacks in the northern extent of the range and gunshot mortalities in the south may have an impact on range expansion (as presented in Riedman and Estes 1990). Gunshot was diagnosed in 4% (n=8) of the sea otters in the recent study but these cases were equally distributed between the range south of Cambria and north of Point Sur.

Infectious disease

The high frequency of infectious diseases (38.5%, n=75) was unexpected. Many of these diseases were reported but uncommon in the past. Peritonitis induced by acanthocephalan parasites (see below) was the diagnosis for 14% (n=27) of the sea otters examined at the NWHC, comprising the single most frequent cause of death, as well as the most frequent infectious disease. Most (67%, n=18) cases of acanthocephalan peritonitis occurred in pups or juvenile sea otters. Peritonitis occurs when larval acanthocephalan parasites that reside in the intestine aberrantly migrate through the intestinal wall allowing bacteria to infect the abdominal cavity. Additionally, in many of these cases, thousands of acanthocephalans were embedded in the intestinal wall, and there was profuse bloody enteritis. The parasite causing this condition, Polymorphus spp., belongs to one of two genera of acanthocephalans found in southern sea otters (there is a current taxonomic dispute on the Polymorphus species identification). The other, Corynosoma enhydri, was found frequently but caused no detectable

deleterious effects. Acanthocephalans are transmitted to sea otters through invertebrate intermediate hosts. The spectrum of invertebrate hosts of Polymorphus spp. is unknown. Polymorphus spp. were reported in small numbers (2-206 per otter) in 10% of the southern sea otters examined by Hennessy and Morejohn (1977). Both the prevalence and intensity of Polymorphus spp. infections appear substantially greater in recent years.

Protozoal encephalitis was the cause of death in 8.5% (n=17) of the sea otters examined at the NWHC. This is the first report of this organism or disease in sea otters. Most (88%, n=15) of the affected otters were adults or subadults. Otters with encephalitis had no gross evidence of the condition but, microscopically, inflammation in the brain was associated with intermediate stages of protozoal parasites. The characterization of this disease syndrome was confounded by the fact that the protozoan (Toxoplasma gondii) was isolated from the brains of sea otters with and without encephalitis. Immunohistochemical tests indicated that a second protozoan was present in some of the cases, suggesting that two agents may be involved. In each year of the recent study, encephalitis cases were restricted to the spring and summer. The means of transmission of these agents to sea otters is unknown. The typical infectious stage, the oocyst, of T. gondii is shed in the feces of a species-specific definitive host, the cat. Intermediate stages of several protozoan agents are found in animal tissues and may also be directly infectious if ingested. The intermediate stage of T. gondii is found in a wide variety of vertebrates including humans world wide, but serious illness from the infection is sporadic. Illness is more common in the very young, aged, or as a complication of impaired immune function.

Eight cases of coccidioidomycosis (4% of all mortality), a systemic infection caused by the fungus, Coccidioides immitis, were diagnosed from 1992-95 (Thomas et al. 1996). Only one case had been reported previously, in 1976 (Cornell et al. 1979); this may be an emerging problem. The affected sea otters were adult or subadult. All 8 sea otters, and the prior case in 1976, were found at the southern end of the range at San Luis Obispo County. The disease, also known as San Joaquin Valley fever, is endemic in certain arid regions of the Southwest U.S., notably the Central Valley of California. This fungus grows in soil to form arthrospores that produce disease when inhaled by susceptible individuals. Coccidioidomycosis most often manifests itself in the respiratory tract, but many organs, including the brain, may be affected. A wide variety of species, including humans, are susceptible to coccidioidomycosis. The results of exposure vary, depending on both species and individual factors, from no ill effects to death. In all sea otter cases the infection was widely disseminated to affect multiple organs.

The deaths of 12% (n=23) of the otters were attributed to various bacterial infections. The affected sea otters were all adult or subadult. Bacterial infections were manifested primarily as pneumonia, heart valve infection, abscess or septicemia. The origins of infection were likely to be inhalation or traumatic injuries, but were inapparent in most cases. Bacterial infections occurred as individual cases with no apparent links among them, so direct transmission between sea otters was unlikely. The most frequently isolated bacteria were different strains and species of Streptococcus, but the agents varied widely and mixed infections in the same individual were common.

Significance of disease

The implications of the recent necropsy results appear complex. Past mortality surveys suggested that the influences of "natural" (that is, not directly human-inflicted) mortality might be significant (Ames et al. 1983). The preliminary data presented here corroborate that mortality from natural causes, specifically infectious diseases, is occurring at a high rate. Disease mortality is not only frequent but some diseases appear to be on the rise while others are newly reported. Several of the diseases are predominately affecting prime age, breeding adults. Taken in total, the findings regarding infectious disease mortality appear to bear some disturbing implications for sea otters. The immediate questions are whether this frequency is truly unusual and whether it is remediable.

In comparison to endangered terrestrial top-level predators, these results are unusual, and, at the moment, there is no comparable data available for other, more vigorous sea otter populations. The

NWHC is carrying out similar mortality studies on gray wolves (Canis lupus) in the Great Lakes Region and for the red wolf (Canis rufus) reintroduction program. In these terrestrial species the greatest proportion of mortality (55% for gray wolves and 42% for red wolves) is accidentally or intentionally human-inflicted by shooting, trapping, poisoning, or vehicle trauma. Infectious diseases cause a relatively minor proportion of the mortality in these species, at a rate of 9% in gray wolves and 8% in red wolves.

The question of whether the sea otter's disease mortality has a remedy is not readily answered in our current state of knowledge. Missing details about the transmission cycles of the more frequently encountered diseases may provide the keys to effective control measures. Before focusing on the transmission cycles of individual diseases, it may be useful to consider the underlying significance of a high rate of infectious disease from a broad perspective. In order to exhibit a high frequency and variety of infectious disease mortality, are southern sea otters highly vulnerable to many diseases or are they undergoing high rates of exposure?

A state of general susceptibility is suggested by the variety of infections encountered in sea otters. Unusual vulnerability is also suggested by the fact that otter deaths are repeatedly attributed to diseases that are expected to cause only sporadic mortality in other species. Generalized vulnerability to infections implies that immune function is ineffective. Although assessing and understanding the exact mechanisms of immune function impairment is complicated, in general, immunologic defects may be congenital or have a genetic basis, or arise from certain viral infections, environmental toxins, or malnutrition.

The factors that affect the rates of infectious disease exposure in sea otters differ among the different diseases encountered. The potential sources of the disease agents and means of exposure differ, as do the reasons why an apparent increase or emergence might be taking place. Exposure rates may be linked to population density for certain diseases but density-independent for others. Identification of the key factors that affect exposure and the significance of each disease in relationship to population dynamics will be important distinctions to make if we are to arrive at effective mortality-reduction strategies.

Polymorphus spp.

Over the last 5 years the prevalence and intensity of Polymorphus spp. infections in otters and the frequency of extraintestinal migration of acanthocephala has increased. In order to understand this increase in deleterious Polymorphus spp. infections in sea otters, we need to examine the predator-prey dynamics of the definitive host species (sea birds) and probable intermediate hosts (crabs). It is this predator-prey relationship that the Polymorphus spp. depend upon for completion of the life cycle. Preliminary data suggest that Emerita analoga and Blepharipoda sp. of crab are two intermediate hosts for this acanthocephalan, however there may be more invertebrate species involved. Birds such as gulls, scoters and sea ducks prey upon infected crabs and harbor the egg-producing adult Polymorphus spp. The eggs are deposited with feces into the environment. Sea otters can become infected with the Polymorphus spp., by eating the infected crabs, however, the sea otter is a dead-end host and no egg-producing acanthocephalans have been found. Abiotic or biotic factors that influence the population densities and predator-prey interactions of the bird, invertebrate hosts, or sea otters could affect the risk of infection in sea otters.

There are three broad facets to examine to determine why increases may be occurring in otters.

(1) Factors impacting bird populations. A larger infected bird population (as higher prevalence and/or higher acanthocephalan burdens) could be due to factors that caused an increase in certain sea bird populations, or factors inducing bird aggregations (for example, beach utilization by humans, distribution and management of landfills, aggregation of crab species or other food items).

(2) Factors influencing intermediate host populations. A larger infected intermediate host community (as increased prevalence and/or larval acanthocephalan burdens) could be related to an increase in infected

birds that contaminate the beaches with feces and acanthocephalan eggs. Add to this the complicating fact that some acanthocephala species alter their intermediate host's behavior, thereby increasing the risk of predation so that birds prey disproportionately on infected crabs. Certain conditions such as a rise in water temperature and phytoplankton proliferation can favor the survival of crab offspring, thereby providing a larger intermediate host community for potential infection.

(3) Factors affecting the sea otter population. A food habit change by some sea otters could increase their predation on infected crustaceans. This prey selection might occur if more optimal prey items were unavailable or they may be taken by inexperienced or incapacitated sea otters that seek easier prey such as Emerita and Blepharipoda.

A combination of two or more of the above factors may be interacting to cause an increase in the parasitism of the sea otter. Given the complexity of the ecology influencing the predator-prey dynamics, several species of invertebrates and vertebrates need to be considered in order to identify the factors influencing the sea otter's Polymorphus spp. infections.

Eradicating Polymorphus spp. from the normal bird and invertebrate hosts would be impossible. However, a reversal of the sea otter's current mortality trend from Polymorphus spp. is feasible if we could reduce the prevalence of this parasite. Effective management strategies may be to disperse the responsible bird species or increase the availability of alternate uninfected prey species to sea otters. However, before such management strategies can be developed basic questions must be answered, including which crab species act as intermediate hosts, which beaches have highly infected intermediate or definitive host communities, and what factors correlate with the distribution of infected sea otters.

Toxoplasma gondii

The source of Toxoplasma gondii exposure to sea otters is most likely oocysts from cat feces. Transmission by preying on infected intermediate hosts is unlikely because only warm-blooded animals have been reported as intermediate hosts, and the sea otter is reported only rarely to eat birds. Sea otters could ingest oocysts in water contaminated with cat feces through run-off from beach soils, or possibly by eating filter feeding invertebrates that are transporting oocysts in their alimentary tracts. If oocysts are entering the water via sewage effluent, the contamination of the marine ecosystem could be much greater than just that from beach or adjacent shoreline run-off. This would be an important source to identify and is potentially manageable. To investigate the connection between sewage disposal and otter exposure, the survivability of oocysts through local sewage treatment systems and the viability of oocysts in sea water should be evaluated. Temporal studies could determine whether peaks in contamination correlate with seasonal patterns of disease. Laboratory studies could be designed to investigate whether crabs and other crustaceans could act as phoretic agents in the transmission of T. gondii to sea otters. Vertical transmission of T. gondii from mother to offspring can cause abortion in sheep, goats, and humans. Toxoplasmosis in the very young sea otter may be under- represented in post mortem sampling because carcasses of fetuses or neonates are less likely to be found (Riedman and Estes 1990).

Toxoplasmosis, may be more than the other diseases, raises concern about the sea otter's general ability to resist disease. Infection with T. gondii does not always cause disease in the host. Disease and death more often occurs in animals that are vulnerable due to age, immuno-compromise, or in species (e.g., Australian marsupials or arboreal New World monkeys) that evolved isolated from felids or their feces. Subadult and adult sea otters, not just the young or aged, had protozoal encephalitis, so age does not appear to be an important factor. Sea otter immune competence has not been well studied, so its status to date is unknown. Regarding the species experience with T. gondii, there is little information. Most reports of T. gondii infection of marine mammals are from captive animals. However, reports of this protozoan causing disease in the wild northern fur seal (Callorhinus ursinus) (Holshuh et al. 1985), the West Indian manatee (Trichechus manatus) (Buergelt and Bonde 1983) and five species of dolphin (Di Guardo et al. 1995) indicate that other wild marine mammals are also exposed to T. gondii. Regardless of whether resistance to toxoplasmosis is an individual or species phenomenon, we can evaluate the sea

otter's degree of susceptibility by evaluating the frequency of exposure. Such studies would help discern if most otters are exposed to T. gondii and resist disease or if the few that are exposed break with disease.

Coccidioides immitis

The occurrence of infections by the soil-associated fungus, Coccidioides immitis, in the marine environment is puzzling. The means of transmission to sea otters presumably is similar to that in other species, specifically by inhalation of airborne arthrospores. Recurrent dry winds from the east may disperse the spores from the soil in endemic sites around the city of San Luis Obispo or in the San Joaquin Valley. Coccidioidal spores survive and may even grow in salt water, so the accumulation of spores in the marine environment has also been postulated (Dzawachiszwili et al. 1964). The apparent increase in prevalence of coccidioidomycosis in sea otters since 1992 may be due to the application of more sensitive diagnostic methods, but it coincides with a marked increase in human cases in 1991 to 1993. The human outbreak was tentatively attributed to unusual weather and environmental conditions rather than human-related factors, so occurrence in sea otters may lend support to that hypothesis. Prospects for control of this disease in sea otters are not promising, but knowledge about coccidioidomycosis prevalence has predictive value that may particularly apply to the population in the southern extent of range.

Many of the infectious diseases of concern in southern sea otters are not species specific; they may affect other marine mammals, sea birds and human beings. Some of the epizootiologic factors that potentially play a role in promoting disease emergence or frequency in otters also may have broad effects in the marine environment. The successful recovery of the southern sea otter population is of immediate concern, but obstacles to their recovery could have wider implications for the marine ecosystem. The sea otter could be a sensitive indicator of the health of the Pacific near-shore marine ecosystem.

Conclusions and recommendations

The following series of recommendations are proposed as means to assess the impact and implications that have arisen from investigations into sea otter mortality.

(1) Complete the 5 year intensive necropsy study. The 5 year database under development at the NWHC will be completed at the end of 1996. The recent health and mortality data have undergone only preliminary evaluation. Further analyses for age, gender, and temporal and geographic differences are necessary to distinguish significant patterns and compare with previous data.

(2) Analyze the population data. Further examination of recent necropsy data by integration with other mortality and demographic data may provide valuable insight into the significance of disease and the role of mortality in population recovery. The relationship between the various infectious disease conditions and population dynamics needs to be explored. Identification of mortality factors with the greatest potential for limiting population growth should guide research priorities.

(3) Identify key factors in the disease cycles. The key to reducing mortality in the southern sea otter may be found by identifying critical points at which important disease transmission cycles can be interrupted by knowledge-based management strategies. The greatest gaps involve the transmission of acanthocephalan peritonitis and protozoal encephalitis. Elucidation of disease cycles may also provide a measure of the role of human intervention or human responsibility in the emergence of sea otter disease.

(4) Develop comparative data. Comparison with similar data from a more vigorous sea otter population would provide another means to assess the significance of mortality and various mortality factors to the southern sea otter population. The best comparison would include both the causes and rates of mortality.

(5) Continue mortality monitoring. Continuation of a mortality monitoring system is the basis for identifying trends, investigating, and interpreting the patterns in sea otter mortality. The activities of the sea otter stranding network are critical to that end.

(6) Evaluate the rates of disease exposure. By comparing the rates of disease exposure to the rates of disease mortality we can evaluate the general vulnerability of the southern sea otter to infectious disease.

(7) Assess immune function. Evaluation of the adequacy of the wild southern sea otter's immune function is a means to investigate whether generalized vulnerability to infectious disease exists.

(8) Assess environmental contaminant exposure. Otter tissue analyses for organochlorine compounds and heavy metals were discontinued in 1980. No serious threats to sea otter health were revealed in these early analyses. Evaluation of recent environmental contaminant burdens in sea otters in correlation with mortality factors is a means to assess the current role of toxic contaminants in sea otter disease vulnerability, debility, or neoplasia.

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