

Commentary

Sea otters in a dirty ocean

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The connections between the health of humans, animals, and the environments in which they live have been well recognized and have recently been referred to as one health, one medicine. The one health, one medicine concept is encompassed in the mission statement of the Wildlife Disease Association, the scope of publications in the journal *EcoHealth*, and the concepts of *Conservation Medicine*.¹ It is an underlying principal of the Wildlife Conservation Society and the School of Veterinary Medicine Wildlife Health Program at the University of California, Davis. For four years, one health, one medicine has been the theme of an annual conference hosted by the School of Veterinary Medicine at North Carolina State University. In 2007, the AVMA Executive Board voted to develop the One Health Initiative Task Force. The veterinary profession is inexorably moving toward and embracing broader environmental perspectives and responsibilities. This is all the more important because the broad causes of ill health cross traditional medical disciplines, and the steps taken to address them should be comprehensive to be successful in the long term. By recognizing problems that affect the health of humans, animals, and ecosystems, we can more easily marshal the necessary resources to deal with them.

An excellent example of the interconnectedness of human, animal, and ecosystem health is provided by the situation facing the southern sea otter (*Enhydra lutris nereis*). Near-shore marine habitats are arguably among the global ecosystems most vulnerable to human development. Humans live by the sea in disproportionately large numbers (20 million Californians reside in coastal counties), and the coastal oceans are the ultimate receptacles of urban, industrial, and agricultural effluents. A suite of pollutants apparently emanating from terrestrial sources, including protozoal and bacterial organisms, various persistent organic pollutants, and urea, which influences toxic algal blooms, has been a substantial cause of or contributor to ill-

ness and death in southern sea otters.^{2-6,a} The magnitude of these pollutants' impact on sea otters may be jeopardizing the recovery of this species, identified as threatened in the Endangered Species Act. Sea otters live in California's coastal oceans and are important members of the ecosystem for two main reasons. First, they function as a keystone species.^{7,8} When otters are lost or removed from the ecosystem, kelps and other species of macroalgae, as well as their associated fish and invertebrate communities, are reduced by the abundant herbivores that thrive in the otters' absence.⁹ This trophic cascade has a diverse array of ecologic consequences for kelp-dependent species, and loss of kelp beds leave shorelines more susceptible to severe weather and erosion. Second, sea otters are very effective environmental sentinels: as upper-trophic-level predators, their health reflects that of California's coastal oceans.^{1,5,10} This suite of pollutants also causes morbidity and death in other marine animals and reduces the complexity and stability of the near-shore ecosystem itself.

Sea otters were hunted to near extinction during the Pacific maritime fur trade. The southern sea otter is descended from a remnant colony of about 50 animals that survived along the Big Sur coast, and the population presently numbers about 2,800 animals. This is far fewer than the 16,000 otters that California's coastal oceans are believed to be capable of supporting, and yet population growth effectively ceased around 1994. It is now known that the California sea otter population's sluggish performance is the consequence of increased mortality rate.^{4,11,12} In each of the past four years, the number of sea otter carcasses recovered has equaled or nearly equaled 10% (280/2,800) of the counted population. A number of novel diseases, parasites, and intoxicants have been identified in southern sea otters over the past decade.^{6,13-17,a} Between 1992 and 2004, over 600 southern sea otters, 28% of all otters whose carcasses were recovered during that period, underwent complete postmortem examinations. There are few death assemblages for wild species that are comparable in detail, longevity, and percentage of the population examined. If we assume that sea otters found freshly dead were representative of the population as a whole, during the early to mid 1990s approximately 40% of the otters died of infectious or parasitic diseases.¹⁴ This proportion increased to half (120/241) of the otters examined from 1998 through 2001.³ When intoxication and all forms of disease (infectious

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and noninfectious) were considered, they accounted for 64% (154/241) of the otters examined from 1998 through 2001.³ Interpretation of this striking pattern is complicated by a number of factors, including improvements in our ability to diagnose certain parasitic, infectious, and other diseases and intoxications, and uncertainty as to the potential interactive effects of nutritional stress and impoverished genetic diversity, effects that may increase disease susceptibility. Nonetheless, the central importance of disease to the ecology of California sea otters and their coastal marine ecosystems is quite clear.

Spatial and temporal clusters of deaths have been reported in southern sea otters with a notable proximity to human population centers (Figure 1), but there is limited access to the coast and few beaches on which carcasses might strand in the more unpopulated central portion of their range from San Simeon to Big Sur. These clusters suggest important spatial and temporal relationships, potential sources of pathogens, and exposure risk factors. Spatial and temporal clustering of deaths and the growing body of information regarding the pathogens and traditional pollutants affecting otters suggest important land-sea connections for a substantial proportion of deaths in sea otters (Appendix).

Southern sea otters consume a wide array of benthic marine invertebrates, and as a mustelid with a high metabolic rate and surface-to-volume ratio, they must consume 25% to 35% of their body weight per day for maintenance.¹⁸ Suspension-feeding shellfish as well as detritus-feeding invertebrates and their predators can accumulate pollutants and pathogens from the water column. A clear association between proximity to freshwater inputs into the ocean and proportion of otters infected with *Toxoplasma gondii* has been reported.² In tank experiments, filter-feeding sea otter prey species, such as blue mussels (*Mytilus* spp), have been determined to accumulate *T gondii* oocysts that remain infective in tissues for a number of weeks.¹⁹ Infections with protozoal pathogens *T gondii* and *Sarcocystis neurona* were the cause of death in 23% of 105 freshly dead sea otters from 1998 through 2001, and *T gondii* infections contributed to the death of another 11%.³ Introduced and invasive terrestrial mammals, most notably domestic cats and opossums, are the primary definitive (oocyst-shedding) hosts for these protozoa. When their shed oocysts are transported from land to sea, these mammals become causes of pathogen pollution.^{5,20,21} In three small coastal communities near Morro Bay, Calif, feral and free-roaming cats deposit an estimated 106.4

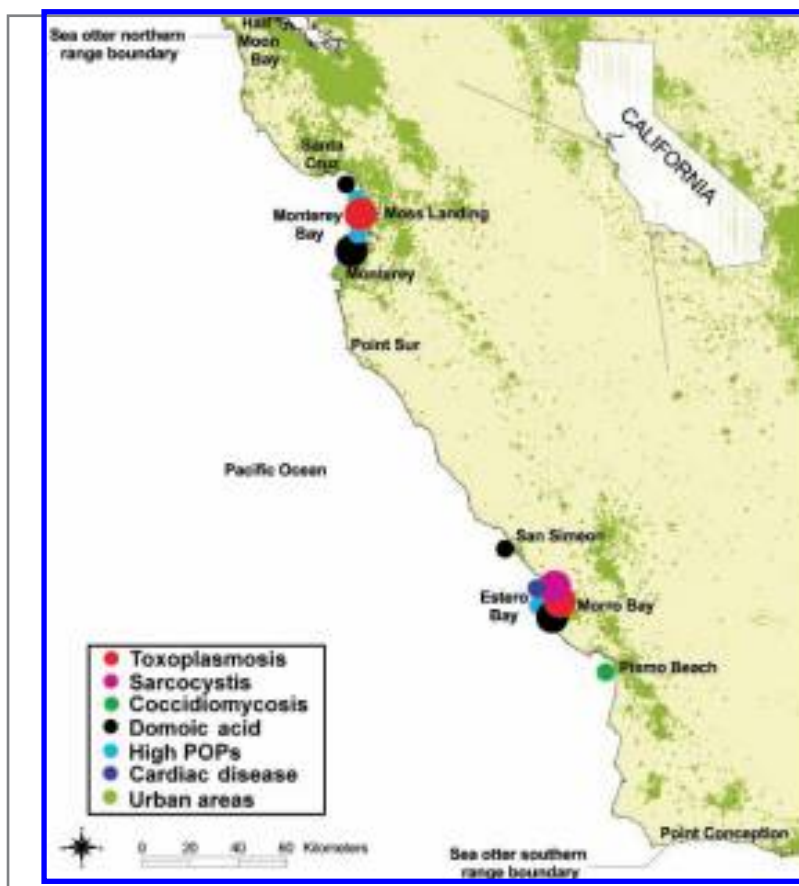


Figure 1—Map of the California coast showing the range of the southern sea otter. Symbols represent approximate locations of clusters of deaths from 1992 to 2004. Larger dots represent 25 or more otters determined as dying from the designated cause. Smaller dots represent fewer than 25 otters dying from the designated cause or in which the designated cause had an indirect role in illness or death. Half of the sea otter population is south of Monterey Bay and north of Estero Bay, but there are few beaches and limited access, which may bias otter carcass recovery. POP = Persistent organic pollutants.

tons of feces per year onto lands that drain immediately into the Pacific Ocean, and *Toxoplasma* oocyst concentrations were estimated to be 97 per square meter of soil.²²

Similarly, mussels and other shellfish have been determined to concentrate protozoa species such as *Giardia*, *Cryptosporidia*, and *Microsporidia*.^{23,24} Strains of *Cryptosporidium* spp associated with cats have been detected in marine bivalves from high-risk coastal estuaries in the southern sea otter range where fecal contamination by cats is observable.²⁴ A number of bacterial enteric pathogens have been isolated from sea otter feces and their invertebrate prey, with risk factor analysis revealing an increased risk for detecting enteric bacteria in mussels collected near human sewage sources.²⁵

In 2003, an unusual mortality event was declared for southern sea otters by the United States Fish and Wildlife Service and the National Oceanic and Atmospheric Administration/National Marine Fisheries Service when deaths greatly exceeded the 10-year averages for three months. Blooms of toxic algae *Pseudonitzschia australis*, which produce domoic acid (the cause of amnesic shellfish poisoning), appear to have been an important contributor to this event.⁴ Although the causes of such harmful algal blooms are complex, nutrients, specifically nitrogen in the form of urea, that are provided to marine environments by runoff, sewage, and riverine inputs may trigger these events.²⁶

Persistent organic pollutants detected in tissues of dead sea otters from California embayments show that southern sea otters have considerably higher exposure to polychlorinated biphenyls (PCBs), dichloro-diphenyl-trichloroethanes, and tributyltin than sea otters examined elsewhere.^{27,28} Summed PCB concentrations in blood samples of living California sea otters are similar to concentrations from sites in Alaska where there are known and well-studied point sources of PCBs.^{29,30,b} Concentrations of summed dichloro-diphenyl-trichloroethanes and PCBs in blood of live, apparently healthy sea otters in California are 50 to 100 times as high as those detected in Alaskan otters, and other organic pollutants have similar patterns.^b

People live by and recreate in the ocean and may consume the same types of shellfish (ie, clams, mussels, and crabs) that sea otters eat. Many of the diseases that plague southern sea otters have a human counterpart (Appendix). Eating uncooked or undercooked seafood containing oocysts may result in toxoplasmosis, an infection increasingly recognized in immunocompromised people and well known for causing fetal deformation as a result of perinatal infection.²¹ Some investigators have speculated that maternal or latent *Toxoplasma* spp infections may alter human behaviors and may be associated with schizophrenia in some people.^{31,32} *Sarcosystis neuroana* infection is a risk factor for myocarditis in sea otters,¹⁷ but effects on human beings are as yet undocumented. Human gastrointestinal and systemic bacterial and protozoal infections resulting from fecal contamination of marine waters or ingestion of raw shellfish are well documented. The first recorded outbreak of amnesic shellfish poisoning in people was in Canada in 1989 and resulted

from ingestion of contaminated mussels.³³ Three people died and dozens were affected. Negative health effects of some persistent organic pollutants in humans are well known, but cumulative effects, lower-level exposures, and additive effects are not well understood.³⁴

It has taken excellent cooperation, collaboration, and transdisciplinary efforts to amass and meld sufficient biological, ecologic, pathologic, microbiologic, and epidemiologic data to understand the nature, source, and scope of southern sea otter health problems. In our opinion, it is an example of the one health, one medicine approach. Pollutants in the form of disease-causing organisms that come from terrestrial and anthropogenic sources; bacteria and protozoal parasites from sewage, agricultural, and street runoff; contaminants; and nutrients that cause algal blooms and intoxications are all part of the problem. That is, sea otters inhabit a near-shore coastal ecosystem that is far richer in potential disease-causing organisms and pollutants that could compromise disease resistance than that in which they evolved. This poses a threat not only to the health of sea otters but the health of humans and the near-shore marine ecosystem.

A number of steps can be taken to mitigate these problems, including tracing apparent nonpoint pollution sources back to their origins (converting them to point sources subject to regulatory action) and enforcing existing state and federal water pollution laws. Improvement of aging sewage infrastructures as well as the level and effectiveness of sewage treatment and elimination or improvement of septic systems could be other vital steps. In harbors, aggressive enforcement of live aboard sewage disposal and regulations on boat paints and effluents are still needed. Processes to improve storm and waste water management so that flushes of runoff and the sediments, nutrients, pathogens, and contaminants they carry are either retained on site or filtered, perhaps through artificial wetlands, are now being considered in land-use planning in many large urban areas. But implementation will take many years, during which time human population and urbanization will continue to increase. Greater use of agricultural best management practices and perhaps movement from voluntary to mandatory farm runoff standards may be needed. Eliminating the outdoor feeding of cats (and inadvertently opossums at feeders) in coastal areas and an increase in public education that focuses on the sources of all these problems and what can be done about them are other steps we have recognized. Overall, these actions may cost many millions of dollars and may inconvenience hundreds of thousands of people, but they need to be done to protect human health, animal health, and the health and sustainability of near-shore ocean ecosystems. In this case, the one medicine may be expensive and somewhat bitter, but the one health it can bring is priceless.

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- a. Jessup DA, Miller MA, Harris M, et al. The 2003 southern sea otter (*Enhydra lutris nereis*) unusual mortality event: a preliminary report to NOAA and USFWS. Unpublished report, 2004.
 - b. Jessup DA, California Department of Fish and Game, Santa Cruz, Calif: Unpublished data, 2006.
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Appendix

Specific causes of or potential contributors to diseases that cause illness and death in southern sea otters, their likely sources or connection to land, relative importance as mortality factors, and potential human health connection.

Otter disease or mortality factor	Likely sources	Relative importance to sea otter mortality rate	Potential human health impacts
Toxoplasmosis	Cat feces, runoff	High to moderate	Systemic or neonatal toxoplasmosis, mental disorders
Sarcocystis	Opossum feces, runoff	High to moderate	Unknown
Fecal bacteria	Sewage, farm runoff, bite wounds, other	Moderate to low	Enteritis, diarrhea, sepsis
Domoic acid	Harmful algal blooms caused by urea, nutrients	Moderate	Amnesic shellfish poisoning
Persistent organic pollutants	Agriculture, street runoff, other	Unknown	Various
<i>Cryptosporidium</i> and <i>Giardia</i> spp	Runoff, animal and human feces	Unknown	Enteritis, diarrhea
Cardiac diseases	Domoic acid exposure, sarcocystis, other	Moderate	Unknown

References

- Aguirre AA, O'Harra T, Spraker TR, et al. Monitoring the health and conservation of marine mammals, sea turtles, and their ecosystems. In: AA Aguirre, RS Ostfeld, CA House, et al, eds. *Conservation medicine: ecologic health in practice*. New York: Oxford University Press, 2002;79–94.
- Miller MA, Gardner IA, Paradies D, et al. Coastal freshwater runoff is a risk factor for *Toxoplasma gondii* infection of southern sea otters (*Enhydra lutris nereis*). *Int J Parasitol* 2002;32:997–1006.
- Kreuder C, Miller M, Jessup DA, et al. Patterns of mortality in the southern sea otter (*Enhydra lutris nereis*) from 1998–2001. *J Wildl Dis* 2003;39:495–509.
- Gerber LR, Tinker MT, Doak DF, et al. Mortality sensitivity in life-stage simulation analysis: a case study of southern sea otters. *Ecol Appl* 2004;14:1154–1165.
- Jessup DA, Miller M, Ames J, et al. Southern sea otter (*Enhydra lutris nereis*) as a sentinel of marine ecosystem health. *EcoHealth* 2004;1:239–245.
- Miller MA, Griggs ME, Kreuder C, et al. An unusual genotype of *Toxoplasma gondii* is common in California sea otters (*Enhydra lutris nereis*) and is a cause of mortality. *Int J Parasitol* 2004;34:275–284.
- Paine RT. A note on trophic complexity and community stability. *Am Nat* 1969;103:91–93.
- Power ME, Tilman D, Estes JE, et al. Challenges in the quest for keystones. *BioScience* 1996;46:609–620.
- Estes JA, Duggins DO. Sea otters and kelp forests in Alaska: generality and ariation in a community ecological paradigm. *Ecol Monogr* 1995;65:75–100.
- Conrad PA, Miller MA, Kreuder C, et al. Transmission of *Toxoplasma*: clues from the study of sea otters as sentinels of *Toxoplasma gondii* flow into the marine environment. *Int J Parasitol* 2005;35:1125–1168.
- Estes JA, Brian B, Hatfield K, et al. Causes of mortality in California sea otters during periods of population growth and decline. *Mar Mamm Sci* 2003;19:198–216.
- Tinker MT, Doak DF, Estes JA, et al. Incorporating diverse data and realistic complexity into demographic estimation procedures for sea otters. *Ecol Appl* 2006;16:2293–2312.
- Cole RA, Lindsay DS, Howe DK, et al. Biological and molecular characterizations of *Toxoplasma gondii* strains obtained from southern sea otters (*Enhydra lutris nereis*). *J Parasitol* 2000;86:526–530.
- Thomas NJ, Cole RA. Risk of disease and threats to wild populations. *Endangered Species Update* 1996;13:23–27.
- Lindsay DS, Thomas NJ, Dubey JP. Biological characterization of *Sarcocystis neurona* isolated from a southern sea otter (*Enhydra lutris nereis*). *Int J Parasitol* 2000;30:617–624.
- Stavely CM, Register KB, Miller MA, et al. Molecular and antigenic characterization of *Bordetella bronchiseptica* isolated from a wild southern sea otter (*Enhydra lutris nereis*) with severe suppurative bronchopneumonia. *J Vet Diagn Invest* 2003;15:570–574.
- Kreuder C, Miller M, Lowenstine LJ, et al. Evaluation of cardiac lesions and risk factors associated with myocarditis and dilated cardiomyopathy in southern sea otters (*Enhydra lutris nereis*). *Am J Vet Res* 2005;66:289–299.
- Estes JA, Riedman ML, Staedler MM, et al. Individual variation in prey selection by sea otters: patterns, causes, and implications. *J Anim Ecol* 2003;72:144–155.
- Arkush KD, Miller MA, Leutenegger CM, et al. Molecular and bioassay-based detection of *Toxoplasma gondii* oocyst uptake by mussels (*Mytilus galloprovincialis*). *Int J Parasitol* 2003;33:1087–1097.
- Daszak PA, Cunningham AA, Hyatt AD. Anthropogenic environmental change and the emergence of infectious diseases in wildlife. *Acta Trop* 2001;78:103–116.
- Dubey JP, Beattie CP. *Toxoplasmosis of animals and man*. Boca Raton, Fla: CRC Press, 1998.
- Dabritz HA, Atwill ER, Gardner IA, et al. Outdoor fecal deposition by free-roaming cats and attitudes of cat owners and non-owners toward stray pets, wildlife, and water pollution. *J Am Vet Med Assoc* 2006;229:74–81.
- Graczyk TK, Fayer R, Lewis EJ, et al. *Cryptosporidium* oocysts in Bent mussel (*Ischadium recurvum*) in Cheasapeake Bay. *Parasitol Res* 1999;85:518–521.
- Miller WA, Miller MA, Gardner IA, et al. New genotypes and factors associated with *Cryptosporidium* detection in mussels (*Mytilus* spp.) along the California coast. *Int J Parasitol* 2005;35:1103–1113.
- Miller WA, Miller MA, Gardner IA, et al. *Salmonella* spp., *Vibrio* spp., *Clostridium perfringens*, and *Plesiomonas shigelloides* detected in marine and freshwater invertebrates from coastal California ecosystems. *Microb Ecol* 2006;52:198–206.
- Howard MDA, Cochlan WP, Ladizinsky N, et al. Nitrogenous preference of toxigenic *Pseudo-nitzschia australis* (Bacillariophyceae) from field and laboratory experiments. *Harmful Algae* 2007;6:206–217.
- Nakata H, Kannan K, Jing L, et al. Accumulation pattern of organochlorine pesticides and polychlorinated biphenyls in southern sea otters (*Enhydra lutris nereis*) found stranded along coastal California, USA. *Environ Pollut* 1998;103:45–53.

28. Kannan K, Guruge KS, Thomas NJ, et al. Butyltin residues in southern sea otters (*Enhydra lutris nereis*) found dead along California coastal waters. *Environ Sci Technol* 1998;32:1169–1175.
29. Bacon CE, Jarman WM, Estes JA, et al. Comparison of organochlorine contaminants among sea otter (*Enhydra lutris*) populations in California and Alaska. *Environ Toxicol Chem* 1999;8:452–458.
30. Estes JA, Bacon CE, Jarman WM, et al. Organochlorines in sea otters and bald eagles from the Aleutian archipelago. *Mar Pollut Bull* 1997;34:486–490.
31. Lafferty KD. Can the common brain parasite, *Toxoplasma gondii*, influence human culture?, in *Proceedings. R Soc Biol Sci* 2006;2749–2755.
32. Brown AS, Schaefer CA, Quesenberry CP Jr, et al. Maternal exposure to toxoplasmosis and risk of schizophrenia in adult offspring. *Am J Psychiatry* 2005;162:767–773.
33. Perl TM, Bédard L, Kosatsky T, et al. An outbreak of toxic encephalopathy caused by eating mussels contaminated with domoic acid. *N Engl J Med* 1990;322:1775–1780.
34. Jensen AA, Slorach SA, eds. *Chemical contaminants in human milk*. Boca Raton, Fla: CRC Press, 1990.