Good Medicine for Conservation Biology: Comments, Corrections, and Connections

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Lafferty and Gerber (2002) provide an excellent review of the conservation significance of a number of diseases to wildlife populations and some examples of the significance of several diseases to southern sea otters (*Enhydra lutris nereis*). Many of the points they make seem relatively new to conservation theory and practice, and are pertinent in light of new and emerging disease threats to wildlife populations. Their article is both provocative and comprehensive. I offer a few comments, corrections, and some connections that might help bridge the gap between those whose focus is conservation biology or traditional wildlife management and those who work in the biomedical and veterinary realms.

Comments

Although one message from epidemiology to conservation might be that "with the exception of factors that impair a host's ability to battle a normally benign infection, infectious diseases are irrelevant. . . . [E]xceptions . . . include disease agents with dynamics controlled by . . . reservoir populations" (Lafferty & Gerber 2002), political and financial forces can conspire to create additional exceptions to this rule. Currently newsworthy examples in North America include the prion protein causing chronic wasting disease (CWD) of cervids, tuberculosis (TB) in deer (*Odocoileus virginianus*), and Brucellosis in elk (*Cervus elaphus*) and bison (*Bison bison*). None of these diseases currently have any known or significant reservoir population of domestic animals, and an impaired host immune response does not seem to be important. They do not cause significant mortality or threaten the viability of large wildlife populations. All three diseases are important (relevant) for their potential impact on the export of meat and animal products, human health, and political issues. All three are receiving more public and scientific attention and research funding than diseases that have greater conservation significance. The point is that biological or conservation relevance isn't the whole picture. When conflict occurs between conservation and agriculture, human health, or political interests on the disease front, conservation usually loses. Conservation might be better served if the intrinsic and monetary values of wildlife, as well as the full ecological impacts of projects promoting human and domestic animal health, were more carefully considered.

Another situation where impaired host immunity or reservoirs of disease are not necessary for conservation relevance to exist are newly introduced, emerging, or exotic diseases that are virulent and cause high death rates in a wide variety of new hosts or in keystone species. These diseases may affect the ecosystems they enter. Examples include (1) the effects of the rinderpest pandemic on African ungulate populations and grazing ecology, (2) the more recent morbillivirus and tuberculosis outbreaks in lions and other carnivores in southern and eastern Africa and their subsequent ecological cascade effects, and (3) the many extirpations and/or near extirpations of bighorn sheep (*Ovis Canadensis*) herds in North America due to virulent *Pasteurella* pneumonia. These severe epidemics, although often relatively short-lived, can be the stochastic event that causes the extirpation of subspecies and local populations. This concept is mentioned by Lafferty and Gerber in their synthesis, but these examples make the point.

Lafferty and Gerber make a number of cogent observations about the limitations of population viability analysis in dealing with health issues (subsequently, the World Conservation Union Conservation Species Breeding Group has developed epidemiologic models) and disease hazards of reintroductions, reserves, and conservation strategies that may increase density. Specific ex-

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amples of the latter they did not include, but two that are now seen as increasingly controversial are provision of artificial water sources (guzzlers in the desert) and feed sources (for elk with Brucellosis on wintering grounds in Wyoming [U.S.A.], for deer at feeding stations in the midwestern United States where TB and CWD occur, and at backyard feeders across the United States where mycoplasmosis may be transmitted between songbirds).

The role of disease in the faltering recovery of the southern sea otter holds many lessons and offers suggestions for further subsequent study. Lafferty and Gerber point out that *Toxoplasma gondii* and *Sarcocystis neurona* infections of the brain may play an important role in preventing the recovery of southern sea otters. Kreuder et al. (in press) show that they are now the most important infectious cause of sea otter mortality—causing at least 23% of all deaths, nearly triple the 8.4% estimate by Thomas and Cole (1996). Deaths due to protozoal disease often occur in prime-age and young adults, and, as noted by Lafferty and Gerber, the infective stages of these protozoa are shed in the feces of cats and opossums, both introduced invasive terrestrial species in California, where southern sea otters occur.

Using a sensitive and validated serologic test, Miller et al. (2002) show that 42% of apparently healthy live otters have been exposed to *T. gondii* and that 62% of dead otters have evidence of exposure. Although the absolute difference between seroprevalence in morbid and healthy segments of the populations is not large (an indirect indicator of the importance of the disease to the population cited by Lafferty and Gerber), we hypothesize that protozoal disease is having a significant effect on population trends. Also, sea otters living in the area of Morro Bay, California, are nine times more likely to have toxoplasmosis than sea otters elsewhere in their range (Miller et al. 2002). Factors that may account for this include (1) the presence of large numbers of feral cats, (2) the discharge of primary treated sewage, (3) historic halogenated hydrocarbon contamination, and (4) a power plant that discharges warmed sea water. These factors do not imply causality, but they serve as further examples of the potential power of on-the-ground epidemiology to influence conservation and resource management decision-making (all four factors above are subject to regulation and enforcement).

Bacterial infections of sea otters are also more complex and interesting than summarized by Lafferty and Gerber. Besides secondary *Streptococcal* wound infections, a number of odd cases of overwhelming bacterial infections caused by organisms usually considered opportunistic pathogens of humans and domestic animals have been documented. Sea otters have also died of gram-negative (fecal) bacterial infections that may be related to sewage contamination. A recent pilot project looking for organisms that cause bacterial and protozoal intestinal infections in humans (conducted in an area with a history of sewage spills) revealed that seven of eight intestinal pathogens are present in the feces of sea otters, that 35% of otters have more than one potentially harmful intestinal pathogen, and that several otters have died as a result of these infections (Melissa A. Miller & D.A.J., unpublished data).

Cases of San Joaquin Valley fever (coccidioidomyocosis) in southern sea otters are clustered in the area around the mouth of the Santa Maria River. Although this disease is commonly an air-borne infection in humans, an alternative hypothesis for the entry of these fungal organisms into the marine environment, which is not discussed by Lafferty and Gerber but which more closely reflects the observed epidemiology, is that they may be carried in the particulate matter of run-off. Bivalves, which filter and concentrate protozoal oocysts (Graczyk et al. 1999), could also concentrate infective fungi and serve as sources of infection. Both routes of infection may be viable, and construction and agricultural development that disturb soils in the watershed and coastal areas where these cases occur and may be the ultimate source.

Sea otters are a valuable sentinel species. It is increasingly clear that pathogen pollution, possibly complicated by chemical pollution that seems to have terrestrial origins, is a significant cause of sea otter mortality (overall, infectious diseases and parasites currently account for 45% of deaths and contribute to the death of another 26%). The peculiar epidemiology of the diseases of southern sea otters has lead to speculation about the immune competence of this remnant subspecies, but no obvious or consistent immune dysfunction has yet been shown. The decades of necropsy work conducted on southern sea otters have been the efforts of the California Department of Fish and Game (CDFG), the U.S. Geological Survey's National Wildlife Health Center (NWHC), and the University of California–Davis (UCD).

Corrections

The genus of acanthocephalans that cause peritonitis in southern sea otters has been renamed *Profilicolis* from *Polymorphus* (Nickol et al, 1999), and *kenti* is the most pathogenic species. This disease syndrome is somewhat more complicated than presented by Lafferty and Gerber because concurrent superinfection by flukes, which also use the sand crab as a host, may add to *P. kenti*'s pathogenicity by debilitating the otter. Acanthocephalan infestations may reduce otter survival rates in some areas, and cluster analysis (Kreuder et al. in press) shows the highest mortality in the north-central portion of their

range, where larger numbers of sea otters feed over the sandy-bottom habitats favored by the intermediate hosts *Emerita* and *Blepharipoda*. It is unclear whether *P. kenti* is an emerging pathogen, what its relationship is with sea bird populations, and whether any sort of management of sea birds would be appropriate or effective.

In Lafferty and Gerber's Table 1, "cholera" is listed as a disease of bighorn sheep, with the source being domestic sheep. The agent(s) Lafferty and Gerber are referring to are pneumonic forms of *Pasteurella* and *Mannheimia* spp. bacteria (Miller 2001). None of the pertinent disease literature on bighorn sheep pneumonias uses the term cholera, although fowl cholera is a term used to describe septicemic (as opposed to pneumonic) *Pasteurella multocida* infections in waterfowl. The epidemiology and pathogenesis of these infections are very different and shouldn't be confused by the use of an archaic term. The original reference specifically linking domestic sheep contact with *Pasteurella* pneumonias of bighorn sheep is that of Foreyt and Jessup (1982), not that of Goodson (1982; as cited by Lafferty & Gerber 2002), who reviewed bighorn dieoffs and supposition about the causes.

This brings up another point, the difference between the peer-reviewed literature on diseases and various less formal technical reports, proceedings, and reviews. Lafferty and Gerber's literature cited section contains 97 citations, many of them from the grey literature. Although this shows a willingness to search for obscure information, these types of reports often contain inaccuracies, suppositions, and conclusions that peer review could eliminate. Worldwide, the primary periodical source for peer-reviewed information on wildlife disease is the *Journal of Wildlife Disease*. Two new and well-referenced texts are *Infectious Diseases of Wild Mammals* (Williams & Barker 2001) and *Parasitic Diseases of Wild Mammals* (Samuelet al. 2001), with *Infectious and Parasitic Diseases of Wild Birds* due out in 2003.

Connections

Fruitful connections between ecologists and epidemiologists are currently being made, notably in work on the southern sea otter. The full suite of ecological and behavioral studies now being conducted by biologists of the U.S. Geological Survey are well integrated with biomedical studies that look at baseline health, immunology, genetics, and exposure to diseases and contaminants conducted by the California Department of Fish and Game and the University of California–Davis. Full postmortem examinations are done on study animals by the same people examining dead animals from the general population. This approach recognizes that at some point the questions ecologists ask merge with those asked by veterinary epidemiologists and that the answers to these questions may be critical for the recovery of the southern sea otter. These efforts may serve as a model for cooperation between ecologists and veterinary epidemiologists.

Lafferty and Gerber have provided a good overview and an ecological perspective frequently lacking in more-focused biomedical and veterinary research journals. They also note that "a detailed understanding of pathogen life history will illuminate the intersection of epidemiology and conservation theory." This type of detailed understanding is the primary focus of a few organizations, notably the Wildlife Disease Association (WDA), which has been actively publishing in this arena for 50 years, long before the recent coining of the phrase "conservation medicine." The WDA's mission is "to acquire, disseminate and apply knowledge of the health and diseases of wild animals in relation to their biology, conservation, and ecology, including interactions with humans and domestic animals." It is a multidisciplinary organization: approximately half its members are veterinarians who treat wildlife populations and their ecosystems, as well as individual animals, and the other half comes from a variety of disciplines (epidemiology, microbiology, public health, wildlife management). The WDA has four international sections and members in 52 countries. Additional information is available at www.wildlifedisease.org. The WDA and the Society for Conservation Biology have a great number of common interests and concerns and some common membership. Formal exchange and cross-participation at future annual meetings might be a good idea.

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