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Current opinion

Sea otter health: Challenging a pet hypothesis

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ABSTRACT

A recent series of studies on tagged sea otters (*Enhydra lutris nereis*) challenges the hypothesis that sea otters are sentinels of a dirty ocean, in particular, that pet cats are the main source of exposure to *Toxoplasma gondii* in central California. Counter to expectations, sea otters from unpopulated stretches of coastline are less healthy and more exposed to parasites than city-associated otters. Ironically, now it seems that spillover from wildlife, not pets, dominates spatial patterns of disease transmission.

The fuzzy pelt that makes sea otters (*Enhydra lutris*) look cuddly also makes for a warm coat, which is why Russian and Aleut fur traders hunted sea otters to near extinction. A remnant population of southern sea otters (*E. lutris nereis*) escaped in the jagged, surf-swept coves at the mouth of Bixby Creek, Big Sur, California and slowly expanded north and south. Due to their near extirpation, sea otters were protected under the North Pacific Fur Seal Treaty of 1911, becoming one of the first species listed under the 1974 U.S. Endangered Species Act. The sea otter population expanded and grew, but in the last few decades has stalled just shy of the legal delisting density of 3090 otters. The failed recovery has motivated considerable research. Sea otters found washed up on the shore often showed pathology from bacterial and parasitic infections (e.g., the acanthocephalan *Profilicolis altmani* and apicomplexans *Toxoplasma gondii* and *Sarcocystis neurona*) (Kreuder et al., 2003). My own review of the literature suggested that infectious disease was preventing the recovery of sea otters (Lafferty and Gerber, 2002), and the most likely source of infection of *T. gondii* was terrestrial runoff containing oocysts defecated by cats (Conrad et al., 2005).

Veterinary pathologists, concerned with sea otter health, speculated that these infectious diseases might be related to human impacts to the environment, because sea otters float in a “dirty ocean” of waste flushed down toilets, tossed into streets, or discharged to waterways (Jessup et al., 2004). Of particular note was that stranded otter carcasses were more likely to be infected with the *T. gondii* parasite if they were found near urban freshwater runoff (Miller et al., 2002). Three hypotheses were raised from these necropsied sea otters: (1) diseases prevent sea otters from recovering, (2) toxoplasmosis is an important disease of sea otters, and (3) feces from pet cats is the main source of exposure to *T. gondii*. Being sensitive to human impacts and easy to observe, Jessup et al. (2004) named sea otters “sentinels of ocean health”. Not only might sea otters tell us something about our effects on the ocean, but we might be able to mitigate our impacts and aid sea otter recovery.

The pet cat hypothesis prompted then Governor Arnold Schwarzenegger to assign into law AB 2485, which included requiring kitty litter to have a label admonishing pet owners not to flush cat feces down the toilet. This law had intuitive appeal to the public. Except for perhaps sea urchin and abalone fishermen, most of the public likes sea otters, and not everyone likes cats, or cat owners, and certainly not cat feces. An Internet search of “sea otter kitty litter” returns more than 14,000 results, almost all of which

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tell a story that blames irresponsible cat owners for the failure of sea otters to recover from the brink of extinction.

A growing fascination with sea otter health inspired wildlife veterinarians and their colleagues to further study human impacts. They surmised that if sea otters are sentinels, then their parasites, sources of mortality, and body condition should map on to human impacts (Tinker et al., 2013c). However, the data from stranded otters used to support the pet cat hypothesis had a weakness: carcasses don't indicate where otters are infected, just the location of the washed-up bodies, which can drift for days. This drift might add error to the data and perhaps obscure the hypothesized links between human impacts and otter health. For otters to be a sentinel species, tighter data were needed. To resolve this deficiency, several scientists set out to compare 135 live sea otters from two ecologically similar locations that differed in human density (Tinker, 2013 and chapters therein). Big Sur, that remote, uncrowded, and wild coastline that attracts adventurers, writers, and artists, was chosen as the pristine site. The impacted site was at nearby Monterey, with a busy harbor, and surrounded by intensive agriculture and nearly 50,000 pet cats, which make up for in abundance what they lack in *T. gondii* prevalence (VanWormer et al., 2013). Starting in 2009, the researchers captured, probed, and assayed otters from each population. Then, they tagged, implanted, and tracked them. Based on their previously published findings, the researchers predicted that the otters from urban Monterey would show more parasitism, worse health, and higher mortality than the otters from wild Big Sur. The city otters should be sicker than their country cousins.

The researchers were surprised when, four years later, it became clear that otters from Big Sur are not healthier (Tinker, 2013). When there are differences in health measures, it is mostly the sea otters from waters adjacent to the city that seem to be doing a bit better (Murray and Tinker, 2013): the Monterey sea otters are bigger, live longer, and have a higher population growth rate (Tinker et al., 2013b). Furthermore, contrary to past results, infectious disease was not a particularly common source of death in the small sample of 17 tagged sea otters that were found dead during the study (Miller et al., 2013), despite the tendency for false positives with the indirect fluorescent antibody test used (Miller et al., 2002). Although infectious diseases might take their toll on sea otters that are under other types of stresses, such as malnourishment, most primary causes of adult sea otter death in this study were shark bite, bacterial septicemia from wounds, mating trauma, and lactation stress; the only cause of death associated with humans was boat strike (Miller et al., 2013). As for the pet cat hypothesis, "sea otters at Monterey that were adjacent to human population centers and areas heavily impacted by runoff or sewage were *not* more likely to be exposed to pathogens than otters at the more pristine Big Sur site, at least in the case of *T. gondii*. In fact [our] results showed exactly the opposite pattern …" (Burgess et al., 2013). More specifically, sea otters from the Monterey area, that haven of domestic cats, are 40-fold less likely to have detectable antibodies to *T. gondii* than are sea otters from the wilds of Big Sur, especially for the stay-at-home female otters (Burgess et al., 2013). Kitty litter seems not to be the main source of infection after all.

Why were sea otters more infected away from humans? One reason could be that the shorter, steeper watersheds and narrower kelp beds of Big Sur might bring sea otters into more contact with untreated terrestrial runoff. Another compelling explanation is that the pet cat hypothesis had blamed the wrong cats. Though pet cats are rare in unpopulated Big Sur, there is pristine habitat for thousands of bobcats and mountain lions (VanWormer et al., 2013). These wild cats have a high prevalence of infection (VanWormer et al., 2013) and maintain a sylvatic cycle of *T. gondii* dominated by the North American type X haplotype that commonly infects sea otters today (Dubey et al., 2011), and probably has for thousands of years. In contrast, domestic cats are more likely to shed the common type II haplotype than the "otter" haplotype (VanWormer et al., 2013). This is not to say that domestic cats are not a source of infection for city otters; a cluster of the less pathogenic "domestic" haplotype occurs in central Monterey Bay otters, near where this haplotype is also common in domestic cats (Miller et al., 2004, 2008). But it seems that domestic cats, by themselves, are not a substantial source of infection. What about other sea otter parasites? Shorebirds and diving ducks are the final hosts for the acanthocephalans that can cause peritonitis in sea otters (Kreuder et al., 2003). Such parasites are most abundant in areas where birds are common (Smith, 2007), suggesting city beaches with high human disturbance should be safer for sea otters. In other words, the dirty ocean doesn't make sea otters sick; instead, the comparison between Big Sur and Monterrey suggests that parasites from wildlife spillover into sea otters (Fig. 1). Nonetheless, further study is needed to determine the generality of this pattern beyond these two sites.

Science works best when it challenges pet hypotheses. It took dedicated efforts by many open-minded scientists to overturn their initial expectations and unravel the complex and sometimes counterintuitive ways that sea otter health interacts with the environment. I expect that future directions in sea otter health research will continue this recognition that marine diseases are part of nature, and that sea otter parasites might, ironically, indicate wilderness, not a dirty ocean.

Box 1

How are marine mammals exposed?

T. gondii oocysts are tough, and survive the trip from land to ocean in creeks, sloughs, and storm drains. Filter feeders, like mussels, can concentrate live oocysts (Miller et al., 2008), so sea otters could become exposed by eating infected bivalves. Although eating bivalves is not correlated with *T. gondii* exposure in otters (Johnson et al., 2009), oocysts stick to kelp and thereby can enter the food web through kelp-grazing snails (Shapiro et al., 2014), and eating snails is a risk factor for infection in otters (Johnson et al., 2009). However, more otters feed on snails at Monterey (14%) than at Big Sur (8%) (Tinker et al., 2013a), so diet doesn't account for the difference in risk between sites observed in the current study. Furthermore, *T. gondii* infects many coastal marine mammals besides sea otters, from to pinnipeds to toothed whales (Tenter et al., 2000), which don't eat snails. How does such a diversity of marine mammals ingest *T. gondii* oocysts? Either the oocysts can be transferred through fish as well as snails, or there is another route of transmission. An alternative to diet is floatation. Oocysts are light (Dubey et al., 1970) and this means that they can concentrate on the ocean surface where marine mammals breath and where the kelp canopy floats. Otters should be especially prone to exposure by floating cysts given the amount of time they spend resting, feeding and grooming on the surface. However, the relative importance of exposure to floating oocysts is anyone's guess.

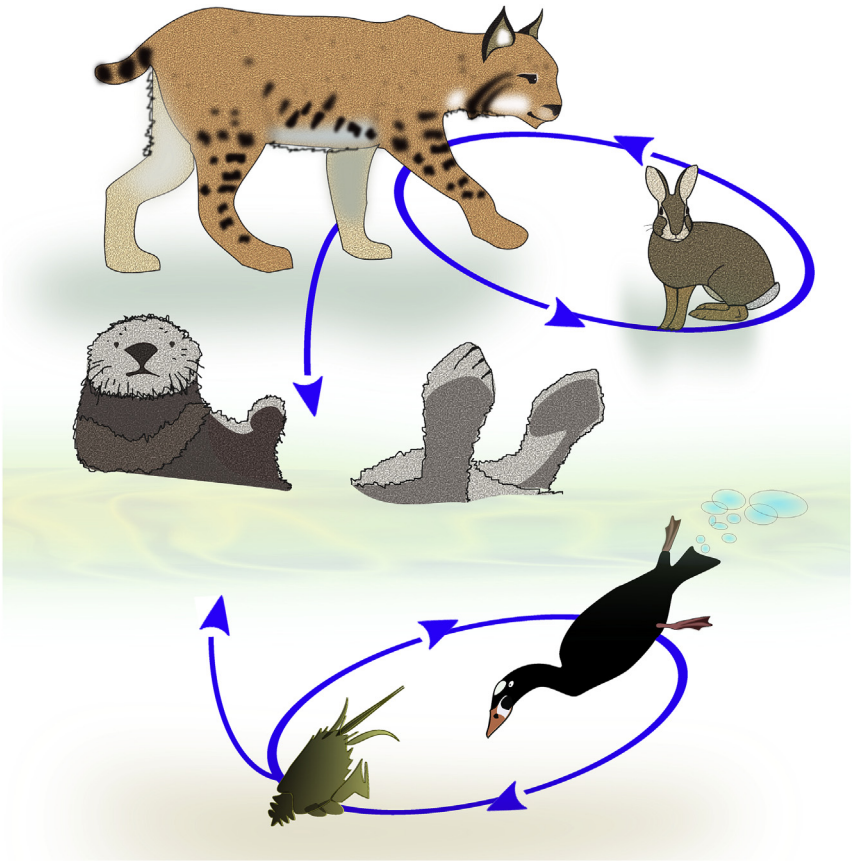

Fig. 1. Parasites that spillover from wildlife to sea otters. Top cycle: cats (like this bobcat) are the final host for the protozoan *Toxoplasma gondii*, for which sea otters are normally a dead-end host. Opossums drive a similar cycle for *Sarcosystis neurona*. Although pet cats were once blamed as the primary source of toxoplasmosis in sea otters, new evidence shows stronger associations with locations where wild cats are common (Burgess et al., 2013). Bottom cycle: diving ducks (like this surf scoter) and shorebirds are the final hosts for acanthocephalans that use sand crabs as intermediate hosts. Otters become accidental hosts if they eat sand crabs. Although many papers and the popular press purport that human actions put sea otter health at risk, these parasites are a natural, long-standing problem for sea otters.

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