

## **DISEASE AND ENVIRONMENTAL STRESSORS: HOW THEY COMBINE TO IMPACT ENDANGERED SPECIES**

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### **INTRODUCTION**

Working together, wildlife biologists, ecologists and veterinarians are slowly elucidating the complex relationships between host, environment, and pathogen that combine to cause disease in wildlife. Understanding the complexity of factors that underlie disease, malnutrition, or even the reduction in fecundity is challenging but enables managers to identify opportunities to address human actions that ultimately can prevent or mitigate disease in wild animals using tools that go beyond traditional veterinary concepts of disease prevention like vaccination or deworming. Being able to mitigate disease is especially important when trying to recover free-ranging endangered species. Two endangered species, southern sea otters (*Enhydra lutris nereis*) and the southern resident killer whales (*Orcinus orca*) provide examples of the complex relationship between disease and the environment and unique opportunities for reducing disease to benefit recovery of these declining populations.

### **SOUTHERN SEA OTTERS**

Southern sea otters are an endangered sea otter sub-species that has a limited distribution off the coast of California (USA). Infectious disease has been identified as an important cause of death in this species. Protozoal encephalitis caused by *Sarcocystis neurona* or *Toxoplasma gondii* infection, apicomplexan protozoa that have complex multihost life cycles and an infective life stage (oocysts) that can persist in the environment. Sea otter exposure to these parasites is closely tied to otter population density, prey selection, and human alteration of coastal habitat.

Interestingly, otters that occur in low density around San Nicholas Island and are not prey-limited ingest food faster, spend less time foraging, and take more food in than resource-strapped otters that live in higher densities along the central California coast. In the food-poor, high otter density environment of California's central coast, otters tend to specialize in their diet and eat a lower diversity of marine invertebrates.<sup>1</sup> This increases their susceptibility to *Sarcocystis neurona* or *Toxoplasma gondii* infection.<sup>2</sup> In fact, otters feeding on abalone, which is the preferred prey in a resource-abundant marine ecosystem, had a very low risk of infection with either pathogen, whereas otters consuming small *Tegula* spp. marine snails were more likely to be infected with *T. gondii*. Further complicating this and possibly increasing risk of pathogen exposure, multiple species of abalone have declined along the California coast due to human overharvest. Even more complicating is the fact that climate change has been clearly linked to epizootics of abalone withering syndrome, a disease is caused by the gastrointestinal rickettsial-like organism *Candidatus Xenohalotis californiensis*, potentially further reducing availability of this important otter prey item.

A novel toxin that has recently emerged as a mortality factor in southern sea otters demonstrates how what happens on land (freshwater specifically in this case) can cause disease in marine mammals. Between 1999 and 2007, the toxin microcystin, produced by freshwater Cyanobacteria, caused hepatitis (hepatocellular vacuolation, apoptosis, necrosis and hemorrhage) and mortality in 21 sea otters.<sup>3</sup> This toxin, which is produced in freshwater lakes, then carried into the ocean, is likely biomagnified in filter feeding clams, mussels and oysters, where it has a slow depuration rate, and can be indirectly consumed by otters foraging on these bivalves. Monitoring has shown increased toxin levels are associated with rains and freshwater runoff into the ocean.

### **SOUTHERN RESIDENT KILLER WHALES**

A distinct population of fish (salmon) eating killer whales resides in and around the waters of Washington State (USA) and British Columbia (Canada). As with most endangered species, the factors leading to the decline of the southern residents are multiple and interactive. They include a history of live capture, high levels of contaminants, reduced prey abundance and disturbance by vessel noise.<sup>4</sup> Disease is not implicated in the population's decline, but could impede their recovery<sup>5</sup> and, as seen in the southern sea otter example, interacts directly with other factors

While they do eat other fish species, southern resident killer whales specialize in eating the largest of the five species so salmon - the king or Chinook salmon (*Oncorhynchus tshawytscha*). In the summer months when diet has been better studied Chinook salmon comprise 70% of their diet. Unfortunately Chinook are the

least common species of salmon in the northeastern Pacific and long- and short term trends in the abundance of wild stocks are predominantly downward, with some populations exhibiting severe recent declines. Overall, compared to historical salmon return numbers for the Pacific Northwest, salmon returns are down to 6-7% of what they were historically. Salmon declines have been caused by four major factors, often called the 4 H's: habitat, harvest, hatcheries and hydropower. Declines in salmon are closely linked to Southern resident health and a one-year lag has been shown between declines in major salmon runs and increases in Southern resident mortality.

Increases in underwater noise complicate the already low abundance of salmon by making it more difficult for killer whales to echolocate and find fish to eat. Anthropogenic noise can interfere with echolocation, reduce their ability to capture prey, increase stress and cause associated stress-related physiological changes such as alternations in hormones and decreased immune function. Unfortunately, in the inland waters of Washington and British Columbia, whale watching boats often outnumber the whales being watched and shipping and other vessel traffic create a scenario where underwater noise is almost ubiquitous. In the presence of underwater noise, killer whales compensate by changing their call's amplitude and frequency. The effective range of their echolocation also is decreased, making it even more difficult to detect an already reduced prey.

Southern resident killer whales have been exposed to Dichlorodiphenyltrichloroethane (DDT) since the 1920's and Polychlorinated biphenyls (PCBs) since 1930's. Although their tissue levels likely peaked in the 1960's and have since declined; they are recognized as some of the most PCB contaminated cetaceans in the world.<sup>6</sup> Blubber biopsies revealed sum PCB concentrations (mean  $\pm$  SEM) of  $146,000 \pm 32,700$  ng/g lipid for males (n = 4; approximate ages 18, 37, 40, 44; mean 35) and  $55,400 \pm 19,300$  ng/g lipid for females (n = 2; ages 14 and 49; mean 32) biopsied between 1993 and 1996. In a more recent study, sum PCB concentrations (mean  $\pm$  SEM) were  $66,000 \pm 26,000$  ng/g lipid in the adult male Southern residents (n = 7; approximate ages 15, 15, 15, 18, 18, 29, 55; mean 24) and  $45,000$  ng/g lipid in the single adult female (age = 27).<sup>7</sup> Interestingly, sum PCB levels in juveniles increase continuously until sexual maturity. Males continue to accumulate throughout their lives, however reproductive females sharply decrease their own burden by passing PCBs to their offspring through contaminated milk, possibly impacting the viability of first born calves and reducing the population's overall potential recovery rate. Despite reductions in PCB levels, Southern residents have shown recent increases in polybrominated diphenyl ethers (PBDEs), flame retardant compounds with very similar structure and likely mechanism of action as PCBs. It is believed that high PCB and PBDE levels in Southern resident killer whales decreases their immune system's capacity to fight disease.

Little is known about diseases of free-ranging killer whales, however it is believed that marine *Brucella* spp., cetacean poxvirus, cetacean morbilliviruses, and herpesviruses all have the ability to impede the recovery of the Southern resident killer whale population.<sup>5</sup> Diseases that can reduce fecundity or recruitment in this population could slow its recovery. Marine *Brucella* spp. are Gram negative bacteria closely related to better known terrestrial pathogens in the genus *Brucella* that can cause abortion in captive bottlenose dolphins (*Tursiops truncatus*). Antibodies to *Brucella* have been detected in killer whales however *Brucella*'s ability to cause abortion or reduced fecundity in killer whales is unknown. Poxvirus can cause neonatal and calf mortality in immunologically naive cetaceans. Cetacean poxvirus has been documented to cause cutaneous lesions in killer whales and has the potential to cause neonatal calf mortality. The small size of the Southern resident killer whale population and the gregarious social nature of these animals mean that the introduction of a highly virulent and transmissible pathogen has the potential to catastrophically affect the long-term viability of the population. Cetacean morbillivirus and herpesvirus are two examples of highly virulent pathogens that have been detected in sympatric odontocetes and have the ability to impact Southern resident killer whales.

Despite being a regional icon that is responsible for driving millions of dollars in tourism annually, the factors for decline in this population are all human caused. Anthropogenic factors have caused declines in all salmon species, including the Southern resident's favored diet of Chinook salmon. Vessel traffic and boat associated noise has the ability to further reduce the ability of these animals to find and catch prey. In the absence of reduced prey consumption, killer whales mobilize adipose stores, increasing metabolism of organochlorine compounds such as PCBs and PBDEs, which have the potential to decrease their immune function, making them more susceptible to disease.

## CONCLUSION

Human-caused factors including toxic contamination, alteration of wildlife population density, depletion of prey availability, or increase of external stressors such as noise, all have the potential to alter or increase pathogen exposure and disease in free-ranging endangered species. For example, maintenance of sea grass beds in nearshore marine coastal areas caused a 50% reduction in relative abundance of potential bacterial pathogens (*Enterococcus* spp.) as compared to locations where seagrass meadows were destroyed. More specifically, this study showed a two-fold reduction in disease in corals located adjacent to seagrass meadows

making the point that maintenance of healthy native habitats can reduce disease in wildlife.<sup>8</sup> Understanding the complexities between disease and the environment is important because it give us options for reducing disease that are out of the normal realm in which veterinarians think (e.g. vaccines or individual animal treatment).

## REFERENCES

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