

Chapter 10. Animal Health and Welfare Considerations

Michael J Murray, DVM

The purpose of this section of the feasibility study is to provide information on the potential health and welfare hazards which may negatively impact the success of the reintroduction of sea otters to the Oregon coast. The information will be subdivided into two major sections, animal health (or its converse, disease) and animal welfare. For the purpose of this discussion disease will include both infectious diseases, such as parasitic infections, and non-infectious disease, such as domoic acid intoxication. There may also be circumstances in which a differentiation between northern sea otters (*Enhydra lutris kenyoni*) and southern sea otters (*E. l. nereis*) is made.

The animal welfare section of this chapter will be more subjective and speculative in nature. While animal welfare is becoming more science-based, it is an evaluation of an animal's state at any one point in time, is described on a continuum from good to poor, and will vary, often dramatically, within a group of animals and over time. The subject will be addressed through the lens of a modified list of the five freedoms described by Britain's Farm Animal Welfare Council in 1965 and subsequently released in 1979 (<https://webarchive.nationalarchives.gov.uk/20121010012427/http://www.fawc.org.uk/freedoms.htm>) and the Association of Zoos & Aquariums' Five Opportunities outlined in their accreditation standards (<https://assets.speakcdn.com/assets/2332/aza-accreditation-standards.pdf>): 1) nutritionally complete diets (quantity, familiarity, safety, accessibility); 2) comfortable habitat (appropriate for species, ability to rest, haul out opportunity, anthropogenic risk); 3) health (known disease risk, live stranding response, carcass recovery & processing, potential rehabilitation opportunities); 4) chronic stressors (boat traffic, ecotourism disturbance, adequate refugia, inter-species interactions); 5) social structure (group size, sex ratio, age range, site fidelity).

Lastly, a discussion of health and welfare would be incomplete without the inclusion of animal transportation and post-arrival conditioning. There are a number of federal agencies with regulatory oversight for interstate transportation of animals, and the list becomes longer, albeit less specific when dealing with wildlife, especially marine mammals. Regardless of the source population, the transport will be several hours long, and the potential for transport-related stress and loss of pelage conditioning is high. Some degree of post-arrival recovery and conditioning will likely be a critical component in the maintenance of the otters' health and well-being.

Animal Health

As previously described, animal health will include both infectious and non-infectious diseases. For the purposes of this chapter, a rather stringent definition of infectious disease will be applied. Infectious diseases are those that are caused by a living organism (i.e., viruses, bacteria, fungi, protozoa, or metazoan parasites) under normal (natural) circumstances. It is important to note that the definition does not include or describe modes of transmission. Diseases that are transmitted directly between animals will be described as transmissible, communicable, contagious, or transmitted horizontally. An exception to that definition are the diseases which are known to be transmitted *in utero*, such as Toxoplasmosis, for which transplacental or vertical transmission will be used. While many infectious diseases are transmitted directly between animals, not all are. Examples include both Toxoplasmosis (excepting the vertical transmitted between dam and fetus) and Sarcocystosis. Both are caused by living organisms, protozoa, but they cannot be transmitted directly to other otters (or humans) through

normal mechanisms. Theoretically, they may be transmitted directly if an uninfected otter ate an infected one, but that is not a normal activity.

This document will be selective in its inclusion of non-infectious diseases. An attempt will be made to address those that are considered to potentially impact the success of a sea otter re-introduction program at a population level and are typically considered more as an individual animal malady. Of the eleven major groups of non-infectious diseases (degenerative, allergic, autoimmune, metabolic, neoplastic, nutritional infectious, immunological, toxic, traumatic, and genetic) only four (infectious, toxic, traumatic, and genetic) are salient to this discussion. The reader is referred to Chapter 4 for further information on genetics and disease.

Aspects of this discussion will be necessarily speculative. Information provided will be based upon a combination of published data, work in progress, personal communication with colleagues, and the author's experience in clinical sea otter medicine. In addition, Inference will be drawn from other members of the family, *Muistelidae*, for which a fair bit of information is known about infectious and non-infectious diseases.

Infectious Disease

Morbillivirus

Of the list of viral diseases affecting sea otters, morbillivirus is undoubtedly the most concerning. A member of the *Paramyxoviridae* family, the genus *Morbillivirus* contains two species of significant concern to sea otters, canine distemper and phocine morbillivirus. Prior to 2001, all sea otters tested for morbillivirus were sero-negative (Hanni et al. 2003, Thomas et al. 2020). Live otters from Washington State (henceforth, Washington) were tested in 2001-2002 following the 2000 mortality event and 80% were sero-positive (Brancato et al. 2009). A retrospective evaluation of tissue from 18 deceased otters sampled between 2000 and 2010 using immune-histochemistry and RT-PCR identified canine distemper virus as the cause of either infection (12/18) or disease (6/12) (Thomas et al. 2020). Evidence collected suggests that canine distemper virus was the cause of the 2000 mass mortality event.

Phocine morbillivirus was first associated with a mass mortality event affecting seals in the North Atlantic in 1988. Since then, a second event has occurred, and sporadic deaths are reported. Serologic evaluation of live-captured sea otters in the eastern Aleutians and Kodiak archipelago in 2004-2005 identified 40% sero-positivity to phocine morbillivirus (Goldstein et al. 2009).

The incidence of morbillivirus in southern sea otters appears to be low. A recent compilation of southern sea otter necropsies from 1998-2012 identified three cases of putative morbillivirus infection (3/560) as primary cause of death (COD) and five (5/560) as contributing COD (Miller et al. 2020). In nearly 1000 live strandings seen at the Monterey Bay Aquarium, no cases of morbillivirus have been identified.

Despite the fact that morbillivirus has been associated with marine mammal die offs in the North Atlantic, Gulf of Mexico, and Mediterranean Sea, the only morbillivirus-associated mass die-off affecting sea otters was the 2000 event off the Washington coast. That being said, however, the potential exposure to canine distemper virus to naïve sea otters from terrestrial carnivores, such as canids and raccoons, as well as marine-foraging river otters, cannot be ignored. Additionally, the ongoing loss of sea ice and opening of the Northwest Passage may facilitate movement of phocine morbillivirus by carrier seals. Once established in the Pacific, the potential exposure of sea otters becomes significantly greater.

Influenza virus.

Mustelids are well known as being susceptible to influenza virus infection, so much so that the domestic ferret is often used as an animal model for studying the disease. Marine mammals, particularly pinnipeds, are considered wildlife reservoirs for the virus. Northern sea otters captured in 2011 were evaluated for antibodies to influenza virus H1N1 (LI 2014). Of the 30 otters tested, 70% (21/30) were sero-positive. The source of the infection was unclear, however serologic evidence supported the notion that the source of infection to the sea otter was the northern elephant seal (*Mirounga angustirostris*).

While the mortality associated with influenza virus in sea otters is uncertain, that fact that virus transmission can occur through shared haul out areas is notable. Additionally, the addition of the sea otter as a wildlife reservoir for influenza A virus may have some public health significance.

Bacterial diseases.

Morbidity and mortality associated with bacterial infections is not uncommon in the sea otter. Bacterial infections were the primary COD in 33/560 and 35/560 contributing cause of death in southern sea otters, 1998-2012 (Miller et al. 2020). The examined death assemblage from the 2002-2015 evaluation of Washington State otters identified 14/93 cases of bacterial infection (including 6 cases of Leptospirosis) (White et al. 2018).

Recent sea otter mortality studies have lumped bacteria-caused mortality into a single group, bacterial infection. It is unclear whether the bacterial species is considered a primary or secondary (opportunistic) pathogen. Review of the list of 15+ species recovered at necropsy (Brownstein et al. 2011) suggests that the vast majority of bacterial species are, in fact, opportunistic relying on a breach of the host's intrinsic immune system (skin, mucus membranes), immunosuppression, or co-infection with a primary pathogen to gain access to the body. It is notable that several of the pathogens identified have significant zoonotic potential and may pose a public health risk; *Brucella spp*, *Coxiella brunettii*, *Bartonella spp*, *Erysipelothrix spp*, *Leptospira spp*, and *Salmonella spp*. Most are likely opportunistic in nature.

Streptococcus phocae, one of the more commonly identified opportunistic pathogens, is frequently recovered from deceased sea otters. A true secondary pathogen, the organism requires damaged skin as a portal of entry. It has been recovered from shark bite wounds, breeding-related wounds to the muzzle and nasal pad, and a myriad of bite wound likely associated with intra-specific aggression. Once the organism is established, it often causes abscesses or septicemia (Bartlett et al. 2016).

Recent studies have demonstrated that several sea otter prey species, bay mussels (*Mytilus trossulus*), butter clams (*Saxidomus giganteus*), Dungeness crab (*Metacarcinus magister*), and black turban snails (*Tegula funebris*), are capable of bio-accumulating *S. phocae* (Rouse et al. 2021). It is not clear whether this bacterium is capable of breaching the gastro-intestinal mucosa or if food-borne exposure requires a pre-existing break in the GI tract, such as an ulceration or wound associated with prey handling.

Other beta-Streptococcus species, *Streptococcus bovis/equinus* and *Streptococcus infantarius* subsp *coli* have been strongly associated with vegetative valvular endocarditis, a proliferative disease of the heart valves. While the exact pathogenesis remains unclear, some attribute the unusual mortality event declared in 2006 in Kachemak Bay to be partially or entirely caused by one or both of these Strep species (Carrasco et al. 2014).

Bordetella bronchiseptica is a common primary and secondary pathogen affecting domestic dogs; one of several organisms associated with “kennel cough.” The organism was first identified as a sea otter pathogen affecting the respiratory tract (Staveley et al. 2003). In the sea otter it is considered to be a secondary pathogen and may be associated with morbillivirus infections. This organism may become significant during post-transport holding and acclimation. The stress-mediated immunosuppression of capture, transport, abnormal social structures, and behaviorally-induced inappetence may result in opportunistic infections with this contagious pathogen.

Leptospirosis has historically been an uncommon disease of sea otters. A study of otters in Washington had a sero-positivity rate of 1/30 in 2001 (Brancato et al. 2009); 5/103 in California in 2003 (Hanni et al. 2003); and 3/161 in Alaska and Russia in 2004-2006 (Goldstein et al. 2011). In 2002, six beach cast sea otter carcasses were evaluated and COD attributed to leptospirosis (Knowles et al. 2020). While the incidence seems to remain low, there may be some degree of concern for transfer of infection from terrestrial wildlife. A study of peri-urban wildlife in Northern California identified six species associated with significant risk factors for infection: western gray squirrel, coyote, striped skunk, raccoon, gray fox, and mountain lion (Straub and Foley 2020). Their presence in and around potential sea otter haul outs may pose some degree of inter-species transmission on the Oregon coast.

Overall, bacterial infections are unlikely to pose a significant, population-level threat to a re-introduced sea otter population along the Oregon coast. Recent mortality studies of southern and Washington sea otters identified 68/560 (12%) and 14/93 (15%) cases in which bacterial infections were the primary or secondary COD, respectively (White et al. 2018, Miller et al. 2020).

Fungal diseases.

There is only one fungal disease warranting discussion within this venue, coccidioidomycosis or Valley Fever, a disseminated fungal infection caused by *Coccidioides immitis*. While it is an infectious disease, it is not easily transmitted from one otter to another, and therefore should not be considered communicable. The infectious fungal spores have a limited range, and the primary risk to sea otters is associated with adjacency to the San Joaquin Valley (Figure 10.1). No cases were reported in northern sea otters, and 9/560 were identified by Miller, et al; all of which were found at the southern end of the sea otter range (Miller et al. 2020).

Interestingly, the incidence of Valley fever has increased dramatically in humans at the northern end of the southern sea otter range from 7.3 cases per 100,000 population in 2008 to 54.7 cases per 100,000 in 2018 (<https://www.co.monterey.ca.us/government/departments-a-h/health/diseases/coccidioidomycosis-valley-fever/coccidioidomycosis-local-data>). Some have theorized that the sea otter cases are associated with construction and other disturbances to the topsoil in the valley associated with eastern winds.

At this point there is no evidence of a population-level threat being posed by coccidioidomycosis to the sea otter re-introduction. That being said, however, a map of prevalence (Figure 10.1) demonstrates the proximity of the fungus to coastal and central Oregon. Given the weather and other impacts associated with climate change, assuming that infection is impossible is probably not wise.

Coccidioidomycosis (Valley fever)

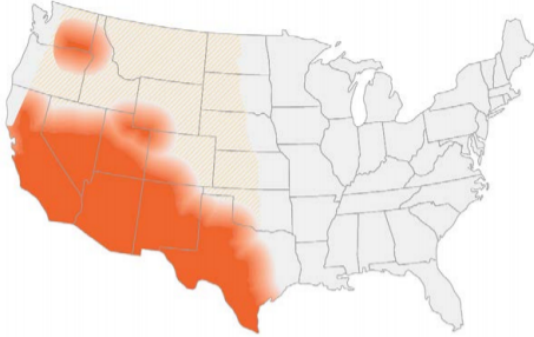


Figure 10.1. Map of distribution of Valley Fever in the U.S. <https://www.cdc.gov/fungal/pdf/more-information-about-fungal-maps-508.pdf>

Parasitic diseases

Unlike parasitic disease in many other wildlife species, the majority of the parasites reported in sea otters tend not to be easily transmitted horizontally. Four of the five parasitic diseases reported to be primary or contributing causes of death in recent studies (White et al. 2018, Miller et al. 2020) are not communicable, and in fact, the sea otter is an aberrant host for three of four infections, protozoal infection (*Sarcocystis*, *Toxoplasma*), acanthocephalid peritonitis, and larval migrans (*Baylisascaris*, *Paragonimus*).

Sarcocystosis

Sarcocystosis is caused by a sporozoan protozoa, *Sarcocystis neurona*. It has a rather complicated life cycle, employing a number of endotherms, including dogs, cats, raccoons, and sea otters, as intermediate hosts, in which it forms tissue cysts. The definitive host, the species in which sexual reproduction occurs and oocysts are produced and shed, is the Virginia opossum, *Didelphis virginiana*.

In the sea otter, positive antibody titres are more common than clinical disease. It is suspected that encysted parasites may not cause significant symptoms. The 2002-2015 Washington State study found Sarcocystosis to account for 28/93 primary causes of death (White et al. 2018), while the California study identified protozoal infection (*Sarcocystis* and *Toxoplasma*) accounting for 50/560 and 58/560 as primary and contributing COD, respectively. While numbers were not provided, Sarcocystosis outnumbered Toxoplasmosis as a primary COD by a factor of five (Miller et al. 2020). The 2004 mass mortality event in Morro Bay, CA was attributed to Sarcocystosis as the primary COD in 15/16 animals (Miller et al. 2010a).

Sarcocystis infections have been identified in California, Washington, British Columbia, and Alaska with spatial clustering most common in California and Washington. There has been a strong association of infection, as defined by positive antibody titres, with terrestrial features (wetlands, croplands, and high human-unit density), soft sediment substrate, and predominance of clams in the diet (Burgess et al. 2020).

A transmission pathway has been proposed, in which oocysts accumulate overtime, remaining viable in the environment for months to years. Freshwater runoff into the nearshore system allows concentration by the local marine habitat features, ocean physical processes, and subsequent invertebrate bio-

accumulation. Benthic invertebrates, such as bivalve mollusks (e.g. razor clams), are then consumed by the sea otter, resulting in infection.

In California, there is good alignment between the dominant freshwater outflows occurring in late winter and early fall followed by a disease peak in sea otters in spring and early summer. This tends to confirm the land-sea transmission epidemiology of Sarcocystosis (Miller et al. 2010a). In addition, disease hot spots have been identified in association with localized oceanic conditions and terrestrial features that affect run-off (Burgess et al. 2020).

Sarcocystosis is of significantly more concern than the other diseases mentioned previously in this chapter. Evidence points to *Sarcocystis* being a more virulent parasite than other apicomplexan parasites. The Virginia opossum is a very well-adapted, non-native mammal introduced into Oregon in 1910-1921, therefore oocyst shedding is likely along the extent of the Oregon coast. Infective stages are shed into the environment and remain infective for extended time periods. The method of transmission from land to sea is now well understood, as is the bio-concentration of the parasite within a normal food item without causing disease in the vector.

Toxoplasmosis.

A second sporozoan (spore-producing) protozoan, *Toxoplasma gondii*, is a significant pathogen in sea otters (Thomas and Cole 1996, Miller et al. 2007). This parasite is found throughout the sea otter's range. There are several serotypes that have been identified with Type II and Type X dominating in sea otters. Type X is the genotype most often associated with fatal disease in sea otters, and Type II, while causing sero-conversion, rarely causes significant, if any, clinical disease (Miller et al. 2008b, Shapiro et al. 2019). Type X has been identified not only in sea otters, but also in domestic cats, bobcats, and mountain lions. Toxoplasmosis is not an uncommon disease in humans, generally associated with undercooked meat, particularly pork. In pregnant women, serious disease in the unborn fetus is possible.

As with for *Sarcocystis*, the sea otter is not the definitive host for the parasite. In the case of *Toxoplasma*, the only known definitive host is a felid, either domestic or wild. Vertical transmission of the parasite is possible, with abortion or peri-natal death as likely outcomes (Miller et al. 2008a, Shapiro et al. 2016).

When evaluated at a large spatial scale, the risk of infection is greatest in areas with higher human population density or high proportion of human-dominated land use, such as impervious surfaces and cropping land. It is thought that this effect is the result of an increased presence of a felid definitive host (Burgess et al. 2018).

At smaller spatial scales, the risk of infection positively correlates to increasing age, sex (male), and prey choice (Burgess et al. 2018). Diets dominated by marine snails are more commonly associated with toxoplasmosis than other feeding strategies (Johnson et al. 2009). It has been theorized that the feeding strategy of snails, like *Tegula*, have a different feeding strategy than other gastropods, such as abalone. The net result is thus an increased exposure to *Toxoplasma* oocysts in *Tegula* diets, than in abalone (Krusor et al. 2015).

The epidemiology of toxoplasmosis is similar to that described for sarcocystosis. The presence of the putative definitive host (felids) which shed large numbers of oocysts into the terrestrial watershed

adjacent to sea otter habitat, a durable infectious stage capable of persistence for extended time periods outside of the host, land-based surface freshwater runoff acting as the source for *Toxoplasma* in the near shore marine environment, and the ability of benthic filter feeders, such as bi-valves, to accumulate infectious stages for eventual consumption by the sea otter (Miller et al. 2002). This pathway has been confirmed for the more virulent genotype, Type X (Shapiro et al. 2019).

While toxoplasmosis is not transmitted horizontally between sea otters, there may be some degree of concern for its potential impact on a recently re-introduced sea otter population. Significant infection, even with the less virulent types, may have impact on reproductive success. Type X infections may be associated with mortality. There may also be some bio-political and public perception issues. While sea otters cannot transmit toxoplasmosis to humans under normal circumstances, it may be difficult for the public to avoid association of sea otters' well described toxoplasma relationship with any publicized human cases.

Acanthocephalid peritonitis.

Acanthocephalid peritonitis (AP) is not an uncommon primary or contributing cause of death in southern sea otters (127/560) but is rarely reported in the northern subspecies (White et al. 2018, Miller et al. 2020). The sea otter is considered an aberrant or dead-end host for the causative agent, *Profilicolis spp.*, of AP. The normal life cycle is complex with a free-living stage, an arthropod intermediate host, and a vertebrate definitive host. In the case of *Profilicolis*, the intermediate hosts are the sand crab, *Emerita analoga*, and the spiny mole crab, *Blepharopoda occidentalis*, and the definitive host is a scoter, gull, or sea duck (Mayer et al. 2003).

While the definitive hosts are found throughout the eastern Pacific coast, the intermediate host are somewhat more inconsistently found in that area. *Emerita* is commonly found in sandy and mixed substrate habitats on the California coast. Sand crab populations are much more sporadically found along the Oregon coast. It has been postulated that the species is re-stocked by larvae drifting northward on the currents with highest number identified during El Nino years (Sorte et al. 2001).

The disease is most often diagnosed in recently weaned pups, sub adults, and aged adult animals living near appropriate habitat for the intermediate host. There may also be a relationship between disease incidence and resource (food) availability (Shanebeck and Lagrue 2020, Tinker et al. 2021b). When the population is at or near carrying capacity, energy recovery rates are lower implying that otters need to work harder to find adequate food. During these periods, the more shallowly located, easily extracted sand crabs may be an attractive source of food. When food is plentiful, hunting is less demanding and even the less physically fit otters are able to forage on normal prey species. This theory is obviously speculative and needs to be interpreted as such, although the positive relationship between sea otter density and the incidence of AP mortality in southern sea otters is statistically significant (Tinker et al. 2021b).

It is unclear how significant AP may be to a recently introduced sea otter population. There may be opportunities to mitigate the risk to some degree through thoughtful release site selection and physical conditioning of animals pre-release. Ample food availability (at least in early years after reintroduction) may result in otters avoiding predation upon some of the high-risk food sources, such as *Emerita* and *Blepharopoda*.

Larval migrans.

In this venue, larval migrans will be used as a generic term to describe the aberrant migration of helminth larvae through various tissues in a non-definitive host, the sea otter. Excluded from this definition is the previously described acanthocephalid peritonitis.

Larval migrans is an uncommon primary or contributing COD in the sea otter. The most commonly described parasite species are the raccoon roundworm (*Baylisascaris sp*) and the lung fluke (*Paragonimus sp*) (White et al. 2018, Miller et al. 2020). Peripheral migration through viscera, muscle, etc. tends not to be clinically significant. On occasion, however, the larva may enter the eye, causing blindness or the brain resulting in an encephalitis. Both diseases tend to be fatal in free-ranging animals due to the untoward impacts on foraging and other life-supporting activities.

Despite their uncommon occurrence, they are included within this discussion as examples of the potential health hazards associated with land – sea pathogen transmission. The presence of freshwater runoff and human-dominated land use, such as impervious surfaces, cropland, and human dwellings, seem to provide increased risk of pathogen pollution of the nearshore habitat.

Non-Infectious Disease

Toxic diseases.

Domoic acid intoxication

While domoic acid intoxication was not identified as a cause of death in the recent Washington death assemblage, it was a significant primary or contribution cause of death (probable/possible) in the California study (White et al. 2018, Miller et al. 2020). Domoic acid is a water-soluble neuronal glutamate receptor analog that is produced by certain strains and species of the diatom *Pseudo-nitzschia* (PN). It is the cause of amnesic shellfish poisoning which was first recognized in Canada in 1987.

Harmful algal blooms (HAB) are known to occur along vast stretches of the eastern Pacific coastline, including Oregon. There are a number of factors that are known or suspected of enhancing PN blooms including changes in the oceanographic conditions, overfishing, eutrophication of marine waters, and global climate change (Landsberg 2002, Chavez et al. 2003, Lefebvre et al. 2016, McKibben et al. 2017). A great deal of work has been done in an attempt to better understand the relationship between oceanographic conditions and HAB along the coast of Oregon.

PN blooms tend to be seen during spring and summer months which are early to mid-point of oceanic upwelling of nutrient rich water. This upwelling tends to be associated with northerly winds. As winds relax, phytoplankton blooms are moved closer to shore where they may interact with benthic invertebrates, prey for sea otters (McKibben et al. 2015). It should be noted that not all PN blooms are associated with the production of domoic acid.

An important cautionary note is that reliance of off-shore PN and domoic acid monitoring may not reflect the degree to which benthic sea otter prey is exposed to the biotoxin. Exposure is dependent upon movement of the algal bloom into the more shallow surf zone. This is in turn affected by surf zone hydrodynamics and morphology (Shanks et al. 2018). Dissipative surf zones are often associated with rip currents which are efficient in exchanging water and associated algal blooms with off-shore water masses. More reflective surf zones limit the exchange of water, thereby reducing the entry of algal blooms into nearshore areas (Shanks et al. 2016). The net result is that the degree to which sea otter filter-feeding prey is exposed to domoic acid may vary dramatically on small spatial scales. The use of

data generated over larger scales is likely to be relatively insensitive in predicting sea otter risk to intoxication.

Because domoic acid intoxication occurs in humans, as well as marine mammals and birds, active monitoring programs are carried out by state and local agencies. Several sentinel species, as well as evaluation of the water column for PN are used. Mussels are a common bio-accumulator that are easily managed; therefore, they are commonly used as sentinel species for the presence of domoic acid. There is some suggestion that they are less sensitive than other benthic invertebrates, such as sand crabs (Ferdin et al. 2002). Razor clams, a significant commercial and recreational fishery in Oregon, are highly effective bio-accumulators of DA. They also have a slow depuration rate relative to mussels (Blanco et al. 2002). As a result, high levels of DA in razor clams may represent an acute, high level exposure, or alternatively a chronic, low level exposure over time (McKibben et al. 2015). Because monitoring efforts vary from region to region, and due to differing mechanisms of bioaccumulation between species, the use of human-centric toxicity thresholds, and the emphasis on human-consumed species, the use of established monitoring systems have limited applicability to predicting sea otter exposure (Figure 10.2).

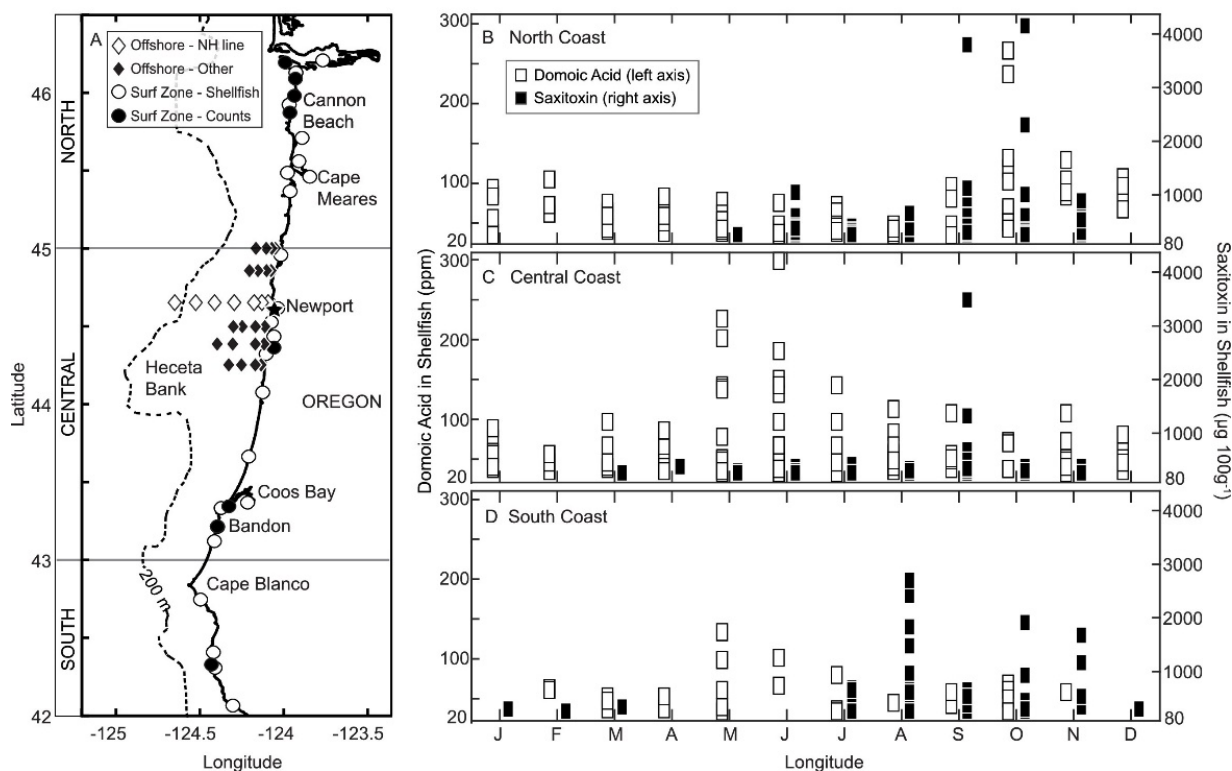


Figure 10.2. A) Map of coastal Oregon (area to the right of solid black line is land), with dashed line showing the continental shelf break at the 200-m isobath. Diamond symbols show offshore locations sampled aboard research vessels. White diamonds highlight the Newport Hydrographic (NH) line at 44.658N. Wind data were collected at Newport, Oregon (star symbol). Circles on coast represent surf zone sampling locations for shellfish DA and STX (white) or *Alexandrium* and *Pseudo-nitzschia* cell counts (black). Surf zone data are binned into north (45–46.58N), central (43–45.8N), and south (42–43.8N) regions. B–D) Monthly STX and DA are shown as black squares (right axis) and white squares respectively for (b) north, (c) central, and (d) south coast locations defined in (a). Only values above the 80 mg * 100 g⁻¹ and 20 ppm harvesting closure thresholds for STX and DA, respectively, are shown (i.e., y-axes start at closure thresholds). From (McKibben et al. 2015).

Other potential sea otter prey items have been evaluated as potential depositories for DA. One study looked at eight benthic invertebrate species representing four feeding groups, filter feeders (*Emerita*

analoga, *Urechis caupo*), a predator (*Citharichthys sordidus*), scavengers (*Nassarius fossatus*, *Pagurus samuelis*), and deposit feeders (*Neotrypaea californiensis*, *Dendraster excentricus*, *Olivella biplicata*). While DA was identified in all eight species, it was above the human safety threshold of 20 ppm in six (*N. fossatus*, *E. analoga*, *U. caupo*, *C. sordidus*, *N. californiensis*, and *P. samuelis* (Kvitek et al. 2008).

The potential impact and pathogenesis of domoic acid exposure is likely to be directly related to the manner in which various prey species respond to the toxin, local and regional environmental factors, and the age/size of the prey (Egmond 2004). Mussels, one of the primary sentinel species for domoic acid, accumulate domoic acid in the digestive gland. As a result, it depurates quickly, but it does accumulate to high levels. Domoic acid accumulates in different body tissues, the mantle and foot, of the razor clam. This accumulatory pathway results in a significantly slower depuration rate (Novaczek et al. 1992). As a result of the rapid accumulation and elimination in mussels, sea otters may be exposed to high levels of domoic acid in a short time; an acute intoxication is the result. Prey species with slower depuration rates, such as razor clams (Blanco et al. 2002), may result in the accumulation of high levels of domoic acid from either profound *Pseudo-nitzschia* blooms or exposure to low, persistent levels of the toxin (McKibben et al. 2015). Domoic acid intoxication is difficult to diagnose ante-mortem. The toxin is readily absorbed via the gut and eliminated via the urine. Its serum half-life is short, making serological evaluation insensitive. Urine is a more sensitive test; however, it too is eliminated within a short time period. There are three major post-mortem presentations of domoic acid intoxication based on dose consumed over time. Acute intoxication is primarily a neurological disease with seizures dominating the clinical presentation. A subacute disease with doses being spread out over time has both neurological changes and some degree of effect on the heart. The chronic form is a cardiac disease often associated with cardiomyopathy and other degenerative diseases of the heart (Miller et al., *in press*).

Given the significance of known or suspected domoic acid-related mortality, and recently published information demonstrating the relationship between DA and cardiac disease in sea otters (Moriarty et al. 2021), the potential for DA-related morbidity and mortality is highly probable in an Oregon coast re-introduction effort. Methods for mitigation are uncertain, although likely sea otter prey items (especially razor clams) should be included in the process of identifying release sites. Additionally, local oceanographic conditions and the potential for anthropogenic eutrophication of nearshore waters warrant consideration.

Saxotoxin intoxication

A second marine biotoxin warranting discussion is saxitoxin (STX), the causative agent of paralytic shellfish poisoning (PSP), which is produced by some species of the dinoflagellate, *Alexandrium*. STX is not a single compound, instead a group of neurotoxins produced by species of dinoflagellates, including *Alexandrium* (Horner et al. 1997). Based on regional native American customs and the apparent ability of some marine mammals to proactively reject toxin-bearing prey, it appears that PSP has been present on the west coast for centuries (Fryxell et al. 1997). For this reason, the Oregon Department of Agriculture (ODA) has been monitoring shellfish for the presence of STX since 1979.

The typical pattern does not involve DA and STX events co-occurring (McKibben et al. 2015). Both are more common in warmer water and are initiated by upwelling-causing northerly winds. As winds decline, the blooms are moved towards shore exposing nearshore invertebrates to biotoxins. Dinoflagellate blooms, including *Alexandrium*, are classically seen later than DA-associated blooms, traditionally peaking in the months of June through November (McKibben et al. 2015).

The marine biotoxin sampling program for DA/STX and *Pseudo-nitzschia/Alexandrium* is inconsistent along the Oregon coast with the north coast being most heavily monitored, followed by the central coast, and the south coast the lowest level. (Figure 2). Mussels are sampled more commonly than razor clams, and the frequency of sampling decreases from north to south. Significant STX and *Alexandrium* have been reported. In 2010, the ODA closed the entire Oregon coast to all harvesting of mussels, scallops, razor clams, oysters, and bay clams; all are potential sea otter prey (McKibben et al. 2015).

Despite the frequency that shellfish commercial and recreational harvesting is closed along the eastern Pacific coast, the incidence of STX intoxication in sea otters is low. Recent comprehensive analyses of causes of death for sea otters in Washington and California did not report any cases of STX intoxication (White et al. 2018, Miller et al. 2020). Sea otters are susceptible to the effects of the neurotoxin; however, experiments involving wild caught sea otters from Kodiak Island suggested that they seemed to detect and avoid heavily toxic loads (Kvitek et al. 1991).

In the butter clam, *Saxidomus gigantus*, approximately 60-80% of the toxin bioaccumulates in the siphon, gills, kidneys, and pericardial glands. STX depurates slowly, and potentially toxic levels can remain in the butter clam one year following a seasonal bloom (Shumway 1990).

Following consumption of toxic levels of STX, sea otters demonstrate a spectrum of neurological and behavioral anomalies, including vocalization, muscle tremors, and agitation. When toxic prey is removed, recovery appears to be complete (Kvitek et al. 1991). This may explain the absence of STX-related mortality in recent mortality reviews for sea otters (White et al. 2018, Miller et al. 2020).

It is likely that despite the prevalence of STX in Oregon shellfish, there is minimal potential for significant population-level impacts on re-introduced sea otters. Sea otters appear to be able to detect and develop an aversion to STX in levels above a certain threshold (Kvitek and Bretz 2004). It is unclear how this occurs and whether it occurs below the surface. The Kodiak Island study (Kvitek et al. 1991) involved wild caught, independent otters. Therefore, it is not clear from previous work whether the STX avoidance behavior is an innate or learned one. If the latter is true, it is possible that naïve, rehabilitated juvenile and sub-adult otters may be a greater risk of Saxitoxinosis.

Microcystin intoxication.

Microcystin intoxication is not a common cause of sea otter morbidity or mortality; however, its prevalence in freshwater systems is becoming a worldwide problem (De Figueiredo et al. 2004). As with several other causes of sea otter mortality, there is a freshwater link to the disease. Microcystin is an environmentally stable toxin produced by several species of cyanobacteria, formerly known as blue-green algae. It is found in both freshwater and estuarine waters throughout North America and worldwide. In a case study published in 2010 (Miller et al. 2010b), microcystin was transported from freshwater systems into Monterey Bay via nutrient-impaired rivers. Based on experimental evidence, it is believed that the toxin bio-magnified up to 107 times in the tissues of bi-valves (Miller et al. 2010b). Sea otters that consumed toxic levels of microcystin-containing prey died of acute liver failure. The ability of benthic filter feeders to bio-accumulate the toxin above ambient levels and depurate the compound slowly poses a potential health threat to otters foraging adjacent to freshwater streams and rivers.

It is unlikely that microcystin is a significant, population-level health threat to a re-introduced sea otter population. It does, however, warrant some degree of consideration during the evaluation of release

sites. The Oregon Health Authority, Public Health Division publishes guideline for cyanobacterial blooms in freshwater bodies, a potential resource for this evaluation.

(<https://www.oregon.gov/oha/PH/HEALTHYENVIRONMENTS/RECREATION/HARMFULALGAEBLOOMS/Documents/2019%20Advisory%20Guidelines%20for%20Harmful%20Cyanobacterial%20Blooms%20in%20Recreational%20Waters.pdf>)

Tributyltin or organotins

Tributyltin (TBT) was employed as an anti-fouling agent in marine paint for boat hulls starting in the 1960s, until its use was regulated in 1988 (Huggett et al. 1992). As TBT ablated from its original site of application, levels increased in the water column, sediments, and local organisms. It became apparent that the effects of TBT extended beyond target organisms, such as barnacles and marine worms, to include oysters, snails, other mollusks, and crustaceans (Kannan et al. 1998). In fish and mammals, TBT tends to bio-accumulate primarily in the liver, however significant levels are also found in the brain and kidney. Likely as a result of the sea otter's diet and high energetic demands, levels found in sea otters are more than twice that seen in cetaceans (Kannan et al. 1998).

It appears that TBT is associated with immunosuppression in birds and mammals (Snoeijs et al. 1987, De Vries et al. 1991). A study of butyltin residues and cause of death for southern sea otters recovered from 1992-1996 did not demonstrate a strong association between TBT levels and immunosuppression as evidenced by disease as cause of death (Kannan et al. 1998). This finding was supported in a study of organotins in sea otter carcasses from California, Washington, Alaska, and Kamchatka, Russia from 1992-2002 (Murata et al. 2008). Again, the correlation between tissue levels and infectious disease was not strong, although infectious disease cases tended to have higher TBT levels in general. Immunosuppressive effect may be relatively long term, as the half-life of the compound is estimated to be three years (Murata et al. 2008).

Since the use of organotin compounds as marine anti-biofouling agents was federally regulated in 1988, the levels seen are likely declining. Residues have historically been higher in enclosed marinas, such as Monterey Harbor and Morro Bay, and lower in open areas. There is some evidence that the compound may persist longer in larger harbors, which attract larger vessels and those from foreign fleets.

Other Contaminants.

There has been a significant amount of work done looking at contaminants and (to a lesser degree) their potential impact on sea otters (Kannan et al. 1998, Nakata et al. 1998, Bacon et al. 1999, Kannan et al. 2006b, Jessup et al. 2010, Reese et al. 2012). Organic compounds may be found concentrated in the water, such as methylmercury, or in sediments, such as PCBs. The mechanism for introduction into sea otter tissues is not completely understood, but is most likely associated with bio-accumulation and slow depuration in benthic invertebrate prey (Rudebusch et al. 2020). Unfortunately, with the exception of localized concentrations of PCBs associated with military base activity in the Aleutian Islands (Reese et al. 2012, Tinker et al. 2021a), there is little information available for linking environmental concentrations to those found in sea otters. There is also little or no information showing population-level consequences of contaminant exposure for sea otters. Therefore, it is unclear if contaminant levels previously identified in sea otters are biologically significant. Again, site selection for a translocated population will be important in the potential for exposure to anthropogenic contaminants.

While consideration of the degree to which a release site is polluted, compromised, or nutrient enriched should be a part of the decision-making process, its importance should not be over-emphasized relative

to other factors. As with many, if not most, estuarine habitats on coastal North America, Oregon's estuaries are likely to suffer negatively from anthropogenic impacts, including high levels of pollution (see Chapter 6). However, published evidence from a large California estuary, Elkhorn Slough, does not support the notion that polluted ecosystems and thriving sea otter populations are necessarily mutually exclusive. Despite having the most elevated levels of the organic contaminants DDT and DDE recorded within the southern sea otter range (Jessup et al. 2010), pollutants that are known to have deleterious effects on sea otters (Kannan et al. 2006a), Elkhorn Slough also supports some of the highest sea otter densities in California (Tinker et al. 2021c). The Elkhorn Slough sea otter population has been found to have high survival and growth rates even in the presence of these high pollutant levels (Mayer et al. 2019). Perhaps more importantly, the net result of this thriving sea otter population has been the contribution of important ecosystem services, such as positive effects on eelgrass and salt marsh habitats (Hughes et al. 2013, Hughes et al. 2019). It thus seems apparent that one should not consider the sea otter to be a benign occupant of an ecosystem and a passive recipient of negative effects from pollution, but rather as a functioning component of a resilient ecosystem that can help mitigate problems like pollution through positive effects on habitats such as eelgrass (Tinker, pers comm).

Oil Spills

A discussion of anthropogenic contaminants would not be complete without including oil spills. While the incidence of direct oil-associated impacts on sea otters is uncommon, the experiences of the 1989 Exxon Valdez Oil Spill (EVOS) graphically illustrate the potential devastation that oil can have on sea otter populations.

The short term, acute effects of oil exposure are dramatic and well known. Affected otters suffer from life threatening loss of thermoregulatory capacity due to the fouling of the fur with oil. Loss of thermoregulation results in a cascade of metabolic events associated with not only the toxicity of the petroleum compounds, but also the animals inability to meet caloric and fluid needs, either through active loss of heat or inability to hunt. Acute toxic effect observed during EVOS included pulmonary and mediastinal emphysema, gastric erosion and hemorrhage, hepatic necrosis, and hepatic and renal tubular lipidoses (Lipscomb et al. 1993).

Long-term effects of oil contamination can also be significant. This includes animals which may have been exposed to sub-lethal amounts of oil, persistent exposure to low levels of lingering oil, effects of oil on prey populations, and exposure to petroleum compounds bio-accumulated in prey species (Bodkin et al. 2011). In EVOS-affected areas of Prince William Sound, Alaska, lingering oil in intertidal sediments provided both direct and indirect exposure to foraging sea otters (Monson et al. 2000). At the population level, sea otter survival rates decreased in EVOS impacted area and population growth slowed significantly as a result of both continued mortality and movements of new animals into the affected areas (Monson et al. 2011).

The potential for oil related morbidity and mortality in a re-introduced sea otter population in Oregon cannot be ignored. It seems most likely that exposure would most likely affect low numbers of otters at a time, because of small spills from recreational or commercial vessels and runoff from adjacent lands. Catastrophic oil spills may also occur along the Oregon coast. While they historically have not reached the level of EVOS, spills such as the *New Carissa* spill of as much as 70,000 gallons in Coos Bay in

Feb/Mar 1999 (http://www.mhhe.com/biosci/pae/es_map/articles/article_29.mhtml) may be devastating to a newly introduced population, were a spill to happen at the wrong time and place.

Fortunately, most of Oregon's power comes from hydro-electric plants, renewable sources, and natural gas. The last oil refinery stopped in 2008. A small portion of the state's energy is fueled by oil refined primarily by Puget Sound refineries. It is then transported to Oregon via the Olympic Pipeline or by barge. The oil shipped from Puget Sound is refined and not the problematic "Bunker C" oil that causes the worst contamination of wildlife and habitats; nonetheless, opportunities for oil spills in Oregon do exist.

The Oregon Department of Environmental Quality's Emergency Response Program has the responsibility of working together with industry and other agencies to prevent and respond to oil spills. While facilities and training for oil spill response in Oregon likely exist, there is probably not much consideration of sea otters and oil spill response. As a re-introduction program becomes more likely, a pro-active sea otter-based response plan and training program should be considered. Fortunately, California, Alaska, and Washington are good resources for such a program.

Trauma Caused Disease

Shark bite.

Shark bite trauma is the most common primary COD described for the southern sea otter from 1998-2012 (Miller et al. 2020) with dramatic increases being recorded since 2003 (Tinker et al. 2016). A recent analysis indicates that shark-bite mortality has a greater impact on overall population recovery in California than any other cause of death (Tinker et al. 2021b). The reported incidence in Washington State otters is not nearly as common, with only 2/93 reported between 2002-2015 (White et al. 2018). Predation, although not specifically attributable to sharks, is also thought to be an important limiting factor on sea otter populations in southwest Alaska (Estes et al. 1998).

Shark-related mortality of southern sea otters have been attributed to white shark (*Carcharodon carcharias*) bites, as a result of recovered tooth fragments and parallel scratches on sea otter bones. Unlike other marine mammal bites, sea otter attacks are non-consumptive, probably exploratory bites. The nature of the resulting wound and tissue trauma occurs later because of blood loss, tissue trauma, or the loss of thermal integrity and subsequent metabolic collapse.

The nature of the shark bite-related mortalities involving northern sea otters was not provided, however, the pathogenesis of the ultimate death was likely similar to that observed in southern sea otters (White et al. 2018). There is an increasing body of anecdotal evidence to suggest that shark-related sea otter mortality may be important in coastal Oregon. Reports involving beach cast sea otter carcasses for the first 11 months of 2021 (USFWS, unpublished data; T Waterstrat, pers. comm.) suggest the seven of eight had evidence of shark bites, although the timing of shark bite, ante- or post-mortem, could not be reliably determined.

The potential threat posed by shark predation to a re-introduced sea otter population in Oregon is unclear. It is likely to depend on several factors, including prey availability, kelp canopy cover, numbers and species of predatory sharks, and water temperatures (Tinker et al. 2016, Nicholson et al. 2018, Moxley et al. 2019). Tagging data (T. Chapple, unpublished data) and anecdotal evidence indicate a presence of white sharks in Oregon; however, recent personal communication with shark biologists

from California State University Long Beach (C. Lowe) and Oregon State University (T. Chapple) suggest that there is not currently a good sense of the abundance or distribution of white sharks off the Oregon coast. Recent evidence does suggest that white shark distribution in California may be moving northward (Tanaka et al. 2021) with warming conditions. While these size classes do not feed on marine mammals, it is possible that the larger size class of white sharks, which does feed on marine mammals may be experiencing a similar northward distribution shift. This would mirror a hypothesized northward shift in white shark distribution along the US east coast (Bastien et al. 2020).

A second shark species with the potential for sea otter predation is the broadnose sevengill shark (*Notorynchus cepedianus*). Broadnose Sevengill sharks are circumglobally distributed ectothermic predators; on the west coast of North America they range from Baja Mexico to southeast Alaska, typically occupying shelf waters (<200m) including bays and estuaries. With the exception of the White sharks, Broadnose Sevengills are thought to be the dominant shark predator in coastal marine ecosystems where they reside, foraging individually or cooperatively and transitioning from a fish-based feeding structure to a diet focused on other elasmobranchs and marine mammals as they grow (Ebert 2002). While not considered to be a significant threat to sea otters in California, their potential impact in Oregon is less certain given their high trophic level and abundance in estuarine and coastal systems. There is a well described and documented migration pattern between the continental shelf and the shallow nearshore and estuarine habitats in this shark species (Williams et al. 2012).

Sevengill sharks feed on a broad spectrum of animals, including other sharks, batoids, teleost fishes, and marine mammals (Ebert 1991, Lucifora et al. 2005). The sevengill shark employs multiple hunting strategies, including stealth, similar to that of the white shark, but also social facilitation, in which a pack of sharks surround its victim to prevent escape prior to subduing it; a strategy employed at depth (Ebert 1991).

Unfortunately, the risk posed by shark attacks on sea otters in a re-introduction program is unknown, and unlikely to be known prior to embarking on such a program. Similarly, it is purely speculative to predict the potential impact that the broadnose sevengill shark may have on the population. Their known presence in both nearshore and estuaries is of some concern. While the white shark population of Oregon is uncertain, the effects of ocean warming due to climate change on white shark distribution may place Oregon-resident sea otters in harm's way. An example of the northward shift of white shark populations is exemplified by the recent documentation of a nursery area in Monterey Bay (Tanaka et al. 2021).

Anthropogenic trauma.

There are several direct human-caused health risks warranting discussion during an evaluation of a potential re-introduction of sea otters to the Oregon coast. While coastal Oregon has not been closely evaluated to date, a recent evaluation of anthropogenic risks for sea otters in San Francisco Bay was published, and may serve as a roadmap for an Oregon introduction (Rudebusch et al. 2020). In this study, anthropogenic risks were subdivided into four groups: vessel traffic, contaminants, commercial fishing, and major oil spills. These categories cover the majority of direct human-caused primary and contributing COD reported for northern and southern sea otters (White et al. 2018, Miller et al. 2020), the exceptions being blunt trauma to the skull and gunshot.

Vessel traffic.

The incidence of boat strike-related mortality was low in both the California and Washington State studies; 25/560 and 1/93, respectively (White et al. 2018, Miller et al. 2020). The negative effects of vessel traffic are not limited to boat strikes. Any disturbance of resting or grooming otters, normal social structure, and foraging efforts may also have significant impacts both directly and indirectly through stress (the immunosuppression caused by chronic adrenocortical hormone release), as well as the energetic expense of responding to the disturbance (Barrett 2019). Consideration of anthropogenic disturbance should include not only commercial boating and fisheries traffic, but also recreational fishing, watersports, such as kayaks, and boat-based nature watching tours. The risk associated with vessel traffic will likely be site specific, and as human numbers continue to grow, can be expected to increase.

Fishing gear related trauma.

Trauma in sea otters associated with commercial and recreational fisheries is most frequently attributable to net entanglement, fishhook injuries/consumption, or entrapment in fish/invertebrate traps (Figure 10.3). Since fishing regulations in California were changed to move gill net fisheries into deeper water, the incident of net entanglement has decreased significantly (Wendell et al. 1986). However, it still does occur on occasion, either as a result of illicit fishing practices or lost/abandoned/damaged net entanglements. By mandating gill nets be set at depths deeper than sea otters dive (40m), the hazard seems avoidable.

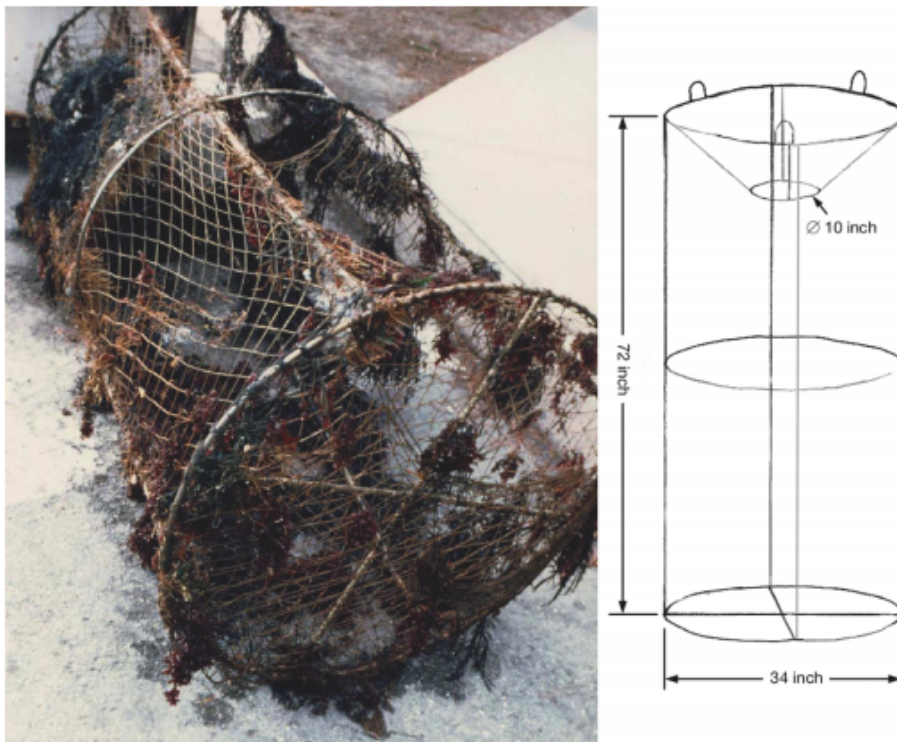


Figure 10.3. A) photograph, and B) line drawing, of a derelict fish trap that drifted into Monterey Harbor 1987, containing 2 drowned sea otters (1 adult female and 1 large male pup). Note the 10" diameter (25.4 cm) fyke opening. Figure from (Hatfield et al. 2011)

Rigid traps, especially those used for Dungeness crabs, have been recognized as a potential entrapment threat, especially for younger sea otters, who may be capable of entering the trap. Following extensive testing using rehabilitated otters at the Monterey Bay Aquarium, a solution to the mortality in fish and shellfish traps was identified. By reducing the fyke size from a 10" circle to a 3" x 9" rectangle, most independent sea otters were excluded from the traps, and yet crab capture rates were not significantly impacted (Hatfield et al. 2011).

Anthropogenic trauma.

Two forms of direct anthropogenic trauma – gunshot and blunt trauma to the skull – have been reported that are most assuredly malicious in nature (trauma from boat strikes is discussed above under the sub-heading “vessel traffic”) (White et al. 2018, Miller et al. 2020). These incidents seem to be uncommon. Published reports do not identify locations, either specifically or generically, nor do they postulate the “justification” for the use of deadly force. Rather than speculating without adequate basis, suffice to say public reaction to a sea otter re-introduction program is unlikely to be universally embraced. It is incumbent upon project managers to recognize the potential for this type of trauma and take steps necessary to mitigate its occurrence, if possible. Public outreach and education may be the most effective mitigation strategies.

Animal Welfare.

Animal welfare and its application to free ranging wildlife is a challenging subject: welfare assessments tend to be focused on individual animals, while conservation goals tend to be focused on populations, and these two underlying goals are not always consistent (Estes and Tinker 2017). While aspects of animal welfare have gained increasing degrees of scientific grounding, they still remain predominantly subjective, and by the nature of welfare, are not static and change frequently.

Original concepts of animal welfare were based on The Five Freedoms written for Britain’s Farm Animal Welfare Council in 1965 and released in 1979:

(<https://webarchive.nationalarchives.gov.uk/20121010012427/http://www.fawc.org.uk/freedoms.htm>).

These have been subsequently modified and renamed the Five Opportunities for the Association of Zoos & Aquariums’ animal welfare and accreditation standards:

(<https://assets.speakcdn.com/assets/2332/aza-accreditation-standards.pdf>). The modification was made to better align the concept of animal welfare with wildlife, particularly wildlife under human care.

Within the context of this chapter, the Five Opportunities will provide the structure upon which welfare considerations will be outlined. By their nature, they will be subjective, and attempts will be made to apply them to a re-introduced population whenever possible. At times, however, it may be necessary to consider the individual animal within the context of the opportunities:

- Nutritionally complete diets
- Comfortable living experiences
- Physical health
- Social groupings
- Avoidance of chronic stress

Animal welfare is a hot-button topic in the public's eyes, especially as it applies to marine mammals. The inclusion of animal welfare as a component of the feasibility study may be of benefit if, and when, the project is formally proposed. Consideration not only of the scientifically and model-based aspects, but also the humane and welfare considerations of the project will undoubtedly be helpful as the Alliance strives to gain public support.

Nutritionally complete diets.

Several aspects of nutrition and diet will need to be included in release site selection. Not only will availability of prey be important, but also the spectrum of species and the otters' recognition of the species as food warrants consideration. The availability of a variety of prey may provide some degree of insulation from naturally occurring recruitment cycles and other forms of variability of species availability. Prey will also need to be present in sufficient quantities at depths attainable by re-introduced otters.

Wholesomeness (or health risks) of food items also warrants consideration. Areas with large aggregations of *Emerita* and *Blepharopoda*, the intermediate hosts of the cause of acanthocephalan peritonitis, may be problematic. Similarly, food-based risk factors associated with toxoplasmosis and those known to bioaccumulate domoic acid effectively are noteworthy.

Comfortable living experiences.

A great deal of effort has been made in identifying appropriate habitat suitable for release of sea otters, particularly animals that will be unfamiliar with the release site(s). It is important to factor into the decision-making process the ability of animals to rest comfortably without undue disturbance from boat traffic and other noxious stimuli. In addition, while the potential for shark attack is unknown, risk factors that have been identified in California warrant consideration in the release site evaluation process (Moxley et al. 2019).

As plans for pre-release holding and conditioning are developed, animal welfare will be an important consideration. The federal Animal Welfare Act and Regulations:

https://www.aphis.usda.gov/animal_welfare/downloads/AC_BlueBook_AWA_508_comp_version.pdf has established minimum standards for marine mammal enclosures for exhibition and research animals based on animal size (§ 3.104(f)), but their applicability to animals in a re-introduction program is doubtful. Regardless, however, there must be some consideration for the size of animal enclosures. Tanks used for surrogate reared, pre-release juveniles at the Monterey Bay Aquarium are approximately 20 ft. in diameter and 3 ft. deep. Animal comfort appears to decrease significantly with group sizes exceeding six animals. Population density within holding facilities will be an important consideration.

Physical health.

Much of the discussion about the welfare considerations of animal health are found in the first section of this chapter. There are several additional considerations which are not disease specific. There should be a protocol developed which describes the frequency (i.e., pre-transport, pre-release, and post-release) with which individual animal health assessments are made. It is readily apparent that starting with healthy animals before re-introducing them to a new site is important.

After otters have been released, what will be the response to animals in distress? There will undoubtedly be a public expectation that attempts will be made to capture and rehabilitate sick or injured sea otters associated with the re-introduction program. Development and implementation of a

stranding response program may be able to rely on some pre-existing coastal marine mammal rehabilitation centers, but facilities, protocols, and even regulatory agencies will be different for sea otters.

One of the confounding knowledge gaps in review of the previous Oregon re-introduction program has been the lack of information about “why it failed.” To better understand the outcome of the project, plans for post release monitoring and carcass recovery and analysis should be made. The development and implementation of these post release efforts warrant further discussion and investigation.

Social groupings.

Successful re-introduction of sea otters into sea otter-free habitats may be difficult; there are no conspecifics to attract and keep the neophytes there. It is not known what the critical mass for re-introduction is, but data from the previous sea otter translocations may be informative. The numbers of otters available for the project will be dependent upon the source populations (see Chapters 3 and 9).

This will be further complicated by the potential need to hold otters at the release site for a time to allow acclimation and recovery of the pelage post transportation. Holding times will be directly proportional to the distance travelled.

The Monterey Bay Aquarium has occasionally released pairs of juvenile animals which spent enough time together to develop a bond while under human care. Despite this pre-release relationship, the otters commonly split up immediately upon release. On occasion, they might re-encounter one another, but there was no evidence of retention of the bond. In some cases, there was a loose re-association at common rafting or feeding areas, or they may remain separated but within the same general location (Staedler, M, Mayer, K, Hazan, S; pers comm). It is important, however, to recognize that these observations were made in release sites already occupied by sea otters, which may have served as anchors to recently released individuals.

While not causally related to social groupings, some consideration should be made to animal age. On one hand, younger animals are less likely to have strong site fidelity and desire to swim back to their original territory. Another perspective, though, is that young animals, particularly rehabilitated sea otters may not be as athletic or physically conditioned as wild otters of similar age. In addition, rehabilitated otters have not experienced the realities of the open sea or estuary. Their lives have been confined to tanks of varying sizes and depths.

The animal welfare aspects of social groupings may be the most problematic of the five opportunities. The questions are relatively straightforward, the answers less so. The options available are not numerous and involve a series of trade-offs.

Avoid chronic stress.

This animal welfare opportunity is a bit oxymoronic. There is no way to avoid stress during a re-introduction, and some of it may be prolonged. Every aspect of the project will be associated with some degree of stress to the otters. A more realistic goal will be to minimize stress whenever possible during the process. Minimizing human contact, both directly and indirectly, management of isolation, and segregation of sexes are examples of actions which will reduce stress. Other stressors are likely to be mitigated through the attention paid to the other four opportunities. The design of a re-introduction

program will be such that opportunities for success are maximized. Minimizing sea otter stress and discomfort will be a natural outcome of the plans to succeed.

Summary

A chapter on the animal welfare concerns associated with a re-introduction of sea otters onto the Oregon coast would not be complete without some discussion of the potential for failure. While the success or failure of the project is determined by population level metrics, both outcomes are based on the sum of individual otters, which is where animal welfare is relevant. The concept of failure will need to be evaluated and defined on different levels, which may impact the decision to continue re-introductions, re-evaluate site of release, and modification of methods for animal capture, transportation and release.

The preceding paragraphs represent an attempt to identify, summarize, and extrapolate information regarding sea otter health and welfare from known circumstances to an anticipated re-introduction site. It is impossible to predict all of the potential health threats that may exist in the future, or that occur cryptically along a coastline free from sea otters for several centuries. That being said, a good faith effort has been made to identify those of greatest concern, either known or suspected. A summary table (Table 10.1) ranks the population-level risks and likelihoods of the diseases described within this chapter.

Based on a review of all the risk factors in Table 10.1, it appears that the most substantial threat to sea otters living along the Oregon coast is likely to be domoic acid intoxication. Its presence in shellfish has been recognized as a potential human health threat for well over a decade; a concern most directed towards acute intoxication of shellfish consumers. Monitoring activities and associated toxicity thresholds have been designed to protect the public, therefore it is likely that chronic, low levels which have been shown to be a driver of cardiac disease in sea otters may go undetected (Moriarty et al. 2021).

A second disease of high concern, but one which is one with uncertain potential is shark bite trauma. Shark bites are a significant cause of mortality for southern sea otters, and the white shark has been accepted as the primary source of the injury. White sharks have been found off the Oregon coast, however, their population numbers and locations are unknown. A second potential sea otter predator, the broadnose sevengill shark is present in high numbers in coastal, offshore, and estuarine systems. A known marine mammal predator, its proclivity to interact with sea otters is unclear.

While it is unlikely that infectious disease will have population-level impacts on the re-introduction program, it may have significant impacts in specific areas, and may also increase over time as sea otter numbers increase, in the case of density-dependent diseases (Tinker et al. 2021b). Contagious diseases, such as one of the morbillivirus infections, have been associated with epizootics in a spectrum of marine and terrestrial mammals. They tend to be density-dependent due to the mode of transmission; a population spread out over a relatively lengthy stretch of coastline may be advantageous, especially for a disease like canine distemper. The same consideration may not apply to other morbilliviruses, such as phocine or cetacean morbillivirus, which may be carried by animals with large home ranges or a few animals making longer-distance movements (Jameson 1989, Ralls et al. 1996).

Table 10.1. Summary of health threats for sea otters in the case of a reintroduction to Oregon, including a subjective ranking of the potential population impact and the relative likelihood of the threat occurring, as well as other attribute of the threats.

Health Concern	Category	Contagious	Population Impact	Likelihood	Source	Site Specificity
Domoic acid	Non-infectious, toxic	No	High	High	Prey, HAB	Possible
Shark bite	Trauma	No	Med-High	Med-high	White shark, 7-gill shark	No
Morbillivirus, phocine	Infectious, viral	Yes	Med-High	Med	Phocid seals	No
Morbillivirus, canine distemper	Infectious, viral	Yes	Med-High	Med	Terrestrial carnivores	No
Sarcocystis	Infectious, parasitic	No	Med-High	High	Land-sea, runoff, prey	Freshwater runoff
Toxoplasma	Infectious, parasitic	No	Med-High	High	Land-sea, runoff, prey	Freshwater runoff
Oil spill	Non-infectious, toxic	No	Med-High	Med-low	Vessels, land-based run-off	Site specific increase
Streptococcus phocae	Infectious, bacterial	Possible	Med	Med-high	Bite wounds, prey	No
Acantocephalid peritonitis	Infectious, parasitic	No	Med	Med	Prey, sandy substrate	Sandy seafloor
Microcystin	Non-infectious, toxic	No	Med	Med	Freshwater runoff	Freshwater runoff
Saxitoxin	Non-infectious, toxic	No	Low	Med-high	Prey, HAB	Widespread
Tributyl tin	Non-infectious, toxic	No	Low	Low	Prey, sediment association	Marinas, large harbors
Influenza	Infectious, viral	Yes	Low	Low	Pinnipeds	No

Health Concern	Category	Contagious	Population Impact	Likelihood	Source	Site Specificity
Leptospirosis	Infectious, bacterial	Yes	Low	Low-med	Pinnipeds	Possible pinniped haul outs, rookery
Bordetella bronchiseptica	Infectious, bacterial	Yes	Low	Low	Open	No
Coccidioidomycosis	Infectious, fungal	No	Low	Low	Environment	Possible
Fishing gear	Anthropogenic	No	low	Low	Nets, crab pots	Possible
Larval migrans	Infectious, parasitic	No	Low	Low	Land-sea, runoff, prey	Freshwater runoff
Vessel traffic	Anthropogenic, trauma	No	Low	Low	Commercial, recreational,	Heavily travelled, populated areas
Contaminants	Anthropogenic	No	Low	Low	Sediments, water column	Yes
Strep bovis/equinus	Infectious, bacterial	Possible	Uncertain	Med	Probable prey	No
Bacterial infections, not specified	Infectious, bacterial	Possible	Uncertain	High	Multiple	No

Non-contagious infectious diseases, such as Sarcocytosis and Toxoplasmosis, are not density-dependent in terms of their transmission processes, but in some cases their impacts on population health can be greater at higher population densities because individual animals are in poorer health and/or selecting sub-optimal prey species (Johnson et al. 2009, Burgess et al. 2018, Tinker et al. 2021b). Such diseases may also have significant impacts on small populations in localized areas, especially those associated with freshwater runoff. A significant first-flush runoff may flush a large pathogen load into the nearshore system, and bio-accumulation by sea otter prey may be the result. This would be unlikely to have a significant impact on an established population, but may be devastating to a recently introduced one.

The animal welfare issues associated with the re-introduction are important for the effect they may have on the population, albeit one otter at a time, and for their role in maintaining public confidence and support. This will be most notable during their time under human care; the capture (if that is needed as an animal source), transportation, acclimation, and release of sea otters in Oregon. During those activities, animals will be best considered individuals. Each of the five opportunities, nutrition, comfort, health, social structure, and stress relief will need to be addressed. Many of the considerations and recommendations are not well defined, as they are dependent upon animal numbers, sources, and release plans. Once these parameters have been set, it will be important to address these.

An additional health and welfare consideration which does not fit well into the previously described categories is post-release activities. Tracking after release may provide important insight into the acclimation and adjustments being made by the otters. It will also be important in identifying otters in distress, retrieval of carcasses, and perhaps following those who emigrate from the release site. Tracking questions are naturally associated with consideration of tagging technologies and the myriad of associated decisions (refer to Chapter 9).

Although not necessarily a population-level health consideration, plans for management of live otters in distress (i.e., sick or injured) must be made. Will they go to a rehabilitation center? If so, which one? A plan for retrieval of beach-cast otter carcasses is important. A component of the carcass program will be the postmortem examination of dead animals. Development of a standardized necropsy protocol is recommended. Again, the questions of who, where, and what, need to be answered before a reintroduction begins.

There are no glaring concerns which suggest that re-introduction of sea otters to the Oregon coast would be likely to face insurmountable health and welfare issues. There are known diseases and conditions which may be somewhat problematic, but this is the case for every extant sea otter population. There are also several unknowns that should be recognized. The effects of climate change through direct impacts on weather patterns, oceanographic parameters, and sea level rise will have an impact at some point in time. Indirect effects, such as changes in prey species, pathogen distribution, and animal movements also exist. Lastly, if 2020's SARS-Coronavirus19 pandemic has taught us anything, it may be that there are things out there which can have devastating effects on animal (and human animal) populations; things we don't know about, and have difficulty predicting. While there are no fail-proof insurance policies for such unknowns, the most prudent strategy for reducing potential for failure is likely to consist of frequent, close monitoring of individuals in a newly established population, with the flexibility to respond quickly should un-anticipated risks emerge.

Final Conclusions

The discussion above is not intended as an all-inclusive list of the potential diseases, infectious and non-infectious, which may have impact on sea otters, or of the considerations for animal welfare. It is an attempt to present information on those which have been shown to have the potential for population-level effects on a re-introduced sea otter population. Much of the information provided is interpreted as an extrapolation of data from the southern sea otter and the Washington populations. Alaskan otters also warrant consideration; however, the nature of the Alaskan coast and subsequent access to otters, especially distressed or dead otters, and the incidence of scavenging upon dead and moribund beach-cast otters, makes mortality investigations problematic for that region.

Literature Cited

- Bacon, C. E., W. M. Jarman, J. A. Estes, M. Simon, and R. J. Norstrom. 1999. Comparison of organochlorine contaminants among sea otter (*Enhydra lutris*) populations in California and Alaska. *Environmental Toxicology and Chemistry* **18**:452-458.
- Barrett, H. E. 2019. The Energetic Cost of Anthropogenic Disturbance on the Southern Sea Otter (*Enhydra lutris nereis*).
- Bartlett, G., W. Smith, C. Dominik, F. Batac, E. Dodd, B. A. Byrne, S. Jang, D. Jessup, J. Chantrey, and M. Miller. 2016. Prevalence, pathology, and risk factors associated with *Streptococcus phocae* infection in southern sea otters (*Enhydra lutris nereis*), 2004–10. *Journal of Wildlife Diseases* **52**:1-9.
- Bastien, G., A. Barkley, J. Chappus, V. Heath, S. Popov, R. Smith, T. Tran, S. Currier, D. Fernandez, and P. Okpara. 2020. Inconspicuous, recovering, or northward shift: status and management of the white shark (*Carcharodon carcharias*) in Atlantic Canada. *Canadian Journal of Fisheries and Aquatic Sciences* **77**:1666-1677.
- Blanco, J., M. B. de la Puente, F. Arévalo, C. Salgado, and Á. Moroño. 2002. Depuration of mussels (*Mytilus galloprovincialis*) contaminated with domoic acid. *Aquatic Living Resources* **15**:53-60.
- Bodkin, J. L., B. E. Ballachey, and G. G. Esslinger. 2011. Trends in sea otter population abundance in western Prince William Sound, Alaska: Progress toward recovery following the 1989 Exxon Valdez oil spill. *US Geological Survey Scientific Investigations Report* **5213**:14.
- Brancato, M. S., L. Milonas, C. Bowlby, R. Jameson, and J. Davis. 2009. Chemical contaminants, pathogen exposure and general health status of live and beach-cast Washington sea otters (*Enhydra lutris kenyoni*).
- Brownstein, D., M. A. Miller, S. C. Oates, B. A. Byrne, S. Jang, M. J. Murray, V. A. Gill, and D. A. Jessup. 2011. Antimicrobial susceptibility of bacterial isolates from sea otters (*Enhydra lutris*). *Journal of Wildlife Diseases* **47**:278-292.
- Burgess, T. L., M. Tim Tinker, M. A. Miller, J. L. Bodkin, M. J. Murray, J. A. Saarinen, L. M. Nichol, S. Larson, P. A. Conrad, and C. K. Johnson. 2018. Defining the risk landscape in the context of pathogen pollution: *Toxoplasma gondii* in sea otters along the Pacific Rim. *Royal Society open science* **5**:171178.
- Burgess, T. L., M. T. Tinker, M. A. Miller, W. A. Smith, J. L. Bodkin, M. J. Murray, L. M. Nichol, J. A. Saarinen, S. Larson, and J. A. Tomoleoni. 2020. Spatial epidemiological patterns suggest mechanisms of land-sea transmission for *Sarcocystis neurona* in a coastal marine mammal. *Scientific reports* **10**:1-9.
- Carrasco, S. E., B. B. Chomel, V. A. Gill, R. W. Kasten, R. G. Maggi, E. B. Breitschwerdt, B. A. Byrne, K. A. Burek-Huntington, M. A. Miller, and T. Goldstein. 2014. Novel *Bartonella* infection in northern

- and southern sea otters (*Enhydra lutris kenyoni* and *Enhydra lutris nereis*). *Veterinary microbiology* **170**:325-334.
- Chavez, F. P., J. Ryan, S. E. Lluch-Cota, and M. Ñiquen. 2003. From anchovies to sardines and back: multidecadal change in the Pacific Ocean. *Science* **299**:217-221.
- De Figueiredo, D. R., U. M. Azeiteiro, S. M. Esteves, F. J. Gonçalves, and M. J. Pereira. 2004. Microcystin-producing blooms—a serious global public health issue. *Ecotoxicology and environmental safety* **59**:151-163.
- De Vries, H., A. Penninks, N. Snoeij, and W. Seinen. 1991. Comparative toxicity of organotin compounds to rainbow trout (*Oncorhynchus mykiss*) yolk sac fry. *Science of the Total Environment* **103**:229-243.
- Ebert, D. 1991. Observations on the predatory behaviour of the sevengill shark *Notorynchus cepedianus*. *South African Journal of Marine Science* **11**:455-465.
- Ebert, D. A. 2002. Ontogenetic changes in the diet of the sevengill shark (*Notorynchus cepedianus*). *Marine and Freshwater Research* **53**:517-523.
- Egmond, H. P. 2004. Marine biotoxins. Food & Agriculture Org.
- Estes, J. A., and M. T. Tinker. 2017. Rehabilitating sea otters: Feeling good versus being effective. Pages 128-134 in P. Kareiva, M. Marvier, and B. Silliman, editors. *Effective Conservation Science*. Oxford University Press, Oxford.
- Estes, J. A., M. T. Tinker, T. M. Williams, and D. F. Doak. 1998. Killer whale predation on sea otters linking oceanic and nearshore ecosystems. *Science (Washington D C)* **282**:473-476.
- Ferdin, M. E., R. G. Kvitek, C. Bretz, C. L. Powell, G. J. Doucette, K. Lefebvre, S. Coale, and M. Silver. 2002. *Emerita analoga* (Stimpson)—possible new indicator species for the phycotoxin domoic acid in California coastal waters. *Toxicon* **40**:1259-1265.
- Fryxell, G. A., M. C. Villac, and L. P. Shapiro. 1997. The occurrence of the toxic diatom genus *Pseudonitzschia* (Bacillariophyceae) on the West Coast of the USA, 1920–1996: a review. *Phycologia* **36**:419-437.
- Goldstein, T., V. A. Gill, P. Tuomi, D. Monson, A. Burdin, P. A. Conrad, J. L. Dunn, C. Field, C. Johnson, and D. A. Jessup. 2011. Assessment of clinical pathology and pathogen exposure in sea otters (*Enhydra lutris*) bordering the threatened population in Alaska. *Journal of Wildlife Diseases* **47**:579-592.
- Goldstein, T., J. A. Mazet, V. A. Gill, A. M. Doroff, K. A. Burek, and J. A. Hammond. 2009. Phocine distemper virus in northern sea otters in the Pacific Ocean, Alaska, USA. *Emerg Infect Dis* **15**:925-927.
- Hanni, K. D., J. A. K. Mazet, F. M. D. Gulland, J. Estes, M. Staedler, M. J. Murray, M. Miller, and D. A. Jessup. 2003. Clinical pathology and assessment of pathogen exposure in southern and Alaskan sea otters. *Journal of Wildlife Diseases* **39**:837-850.
- Hatfield, B. B., J. A. Ames, J. A. Estes, M. T. Tinker, A. B. Johnson, M. M. Staedler, and M. D. Harris. 2011. Sea otter mortality in fish and shellfish traps: estimating potential impacts and exploring possible solutions. *Endangered Species Research* **13**:219.
- Horner, R. A., D. L. Garrison, and F. G. Plumley. 1997. Harmful algal blooms and red tide problems on the US west coast. *Limnology and Oceanography* **42**:1076-1088.
- Huggett, R. J., M. A. Unger, P. F. Seligman, and A. O. Valkirs. 1992. ES&T Series: The marine biocide tributyltin. Assessing and managing the environmental risks. *Environmental Science & Technology* **26**:232-237.
- Hughes, B. B., R. Eby, E. Van Dyke, M. T. Tinker, C. I. Marks, K. S. Johnson, and K. Wasson. 2013. Recovery of a top predator mediates negative eutrophic effects on seagrass. *Proceedings of the National Academy of Sciences of the United States of America* **110**:15313-15318.

- Hughes, B. B., K. Wasson, M. T. Tinker, S. L. Williams, L. P. Carswell, K. E. Boyer, M. W. Beck, R. Eby, R. Scoles, M. Staedler, S. Espinosa, M. Hessing-Lewis, E. U. Foster, K. M. Beheshti, T. M. Grimes, B. H. Becker, L. Needles, J. A. Tomoleoni, J. Rudebusch, E. Hines, and B. R. Silliman. 2019. Species recovery and recolonization of past habitats: lessons for science and conservation from sea otters in estuaries. *PeerJ* **7**:e8100.
- Jameson, R. J. 1989. Movements, home range, and territories of male sea otters off central California. *Marine Mammal Science* **5**:159-172.
- Jessup, D. A., C. K. Johnson, J. Estes, D. Carlson-Bremer, W. M. Jarman, S. Reese, E. Dodd, M. T. Tinker, and M. H. Ziccardi. 2010. Persistent Organic Pollutants In The Blood Of Free-Ranging Sea Otters (*Enhydra lutris* Ssp.) In Alaska And California. *Journal of Wildlife Diseases* **46**:1214-1233.
- Johnson, C. K., M. T. Tinker, J. A. Estes, P. A. Conrad, M. Staedler, M. A. Miller, D. A. Jessup, and J. A. Mazet. 2009. Prey choice and habitat use drive sea otter pathogen exposure in a resource-limited coastal system. *Proc Natl Acad Sci U S A* **106**:2242-2247.
- Kannan, K., T. Agusa, E. Perrotta, N. J. Thomas, and S. Tanabe. 2006a. Comparison of trace element concentrations in livers of diseased, emaciated and non-diseased southern sea otters from the California coast. *Chemosphere* **65**:2160-2167.
- Kannan, K., K. S. Guruge, N. J. Thomas, S. Tanabe, and J. P. Giesy. 1998. Butyltin residues in southern sea otters (*Enhydra lutris nereis*) found dead along California coastal waters. *Environmental Science & Technology* **32**:1169-1175.
- Kannan, K., E. Perrotta, and N. J. Thomas. 2006b. Association between perfluorinated compounds and pathological conditions in southern sea otters. *Environmental Science & Technology* **40**:4943-4948.
- Knowles, S., D. Lynch, and N. Thomas. 2020. Leptospirosis in Northern Sea Otters (*Enhydra lutris kenyoni*) from Washington, USA. *The Journal of Wildlife Diseases* **56**:466-471.
- Krusor, C., W. A. Smith, M. T. Tinker, M. Silver, P. A. Conrad, and K. Shapiro. 2015. Concentration and retention of *Toxoplasma gondii* oocysts by marine snails demonstrate a novel mechanism for transmission of terrestrial zoonotic pathogens in coastal ecosystems. *Environ Microbiol* **17**:4527-4537.
- Kvitek, R., and C. Bretz. 2004. Harmful algal bloom toxins protect bivalve populations from sea otter predation. *Marine Ecology Progress Series* **271**:233-243.
- Kvitek, R. G., A. R. Degange, and M. K. Beitler. 1991. Paralytic shellfish poisoning toxins mediate feeding behavior of sea otters. *Limnology and Oceanography* **36**:393-404.
- Kvitek, R. G., J. D. Goldberg, G. J. Smith, G. J. Doucette, and M. W. Silver. 2008. Domoic acid contamination within eight representative species from the benthic food web of Monterey Bay, California, USA. *Marine Ecology Progress Series* **367**:35-47.
- Landsberg, J. H. 2002. The effects of harmful algal blooms on aquatic organisms. *Reviews in Fisheries Science* **10**:113-390.
- Lefebvre, K. A., L. Quakenbush, E. Frame, K. B. Huntington, G. Sheffield, R. Stimmelmayer, A. Bryan, P. Kendrick, H. Ziel, T. Goldstein, J. A. Snyder, T. Gelatt, F. Gulland, B. Dickerson, and V. Gill. 2016. Prevalence of algal toxins in Alaskan marine mammals foraging in a changing arctic and subarctic environment. *Harmful Algae* **55**:13-24.
- Lipscomb, T., R. Harris, R. Moeller, J. Pletcher, R. Haebler, and B. E. Ballachey. 1993. Histopathologic lesions in sea otters exposed to crude oil. *Veterinary pathology* **30**:1-11.
- Lucifora, L. O., R. C. Menni, and A. H. Escalante. 2005. Reproduction, abundance and feeding habits of the broadnose sevengill shark *Notorynchus cepedianus* in north Patagonia, Argentina. *Marine Ecology Progress Series* **289**:237-244.

- Mayer, K. A., M. D. Dailey, and M. A. Miller. 2003. Helminth parasites of the southern sea otter *Enhydra lutris nereis* in central California: Abundance, distribution and pathology. *Diseases of Aquatic Organisms* **53**:77-88.
- Mayer, K. A., M. T. Tinker, T. E. Nicholson, M. J. Murray, A. B. Johnson, M. M. Staedler, J. A. Fujii, and K. S. Van Houtan. 2019. Surrogate rearing a keystone species to enhance population and ecosystem restoration. *Oryx*:1-11.
- McKibben, S. M., W. Peterson, A. M. Wood, V. L. Trainer, M. Hunter, and A. E. White. 2017. Climatic regulation of the neurotoxin domoic acid. *Proceedings of the National Academy of Sciences* **114**:239-244.
- McKibben, S. M., K. S. Watkins-Brandt, A. M. Wood, M. Hunter, Z. Forster, A. Hopkins, X. Du, B.-T. Eberhart, W. T. Peterson, and A. E. White. 2015. Monitoring Oregon Coastal Harmful Algae: Observations and implications of a harmful algal bloom-monitoring project. *Harmful Algae* **50**:32-44.
- Miller, M., P. Conrad, E. James, A. Packham, S. Toy-Choutka, M. J. Murray, D. Jessup, and M. Grigg. 2008a. Transplacental toxoplasmosis in a wild southern sea otter (*Enhydra lutris nereis*). *Veterinary Parasitology* **153**:12-18.
- Miller, M. A., P. A. Conrad, M. Harris, B. Hatfield, G. Langlois, D. A. Jessup, S. L. Magargal, A. E. Packham, S. Toy-Choutka, A. C. Melli, M. A. Murray, F. M. Gulland, and M. E. Grigg. 2010a. A protozoal-associated epizootic impacting marine wildlife: Mass-mortality of southern sea otters (*Enhydra lutris nereis*) due to *Sarcocystis neurona* infection. *Veterinary Parasitology* **172**:183-194.
- Miller, M. A., I. A. Gardner, C. Kreuder, D. M. Paradies, K. R. Worcester, D. A. Jessup, E. Dodd, M. D. Harris, J. A. Ames, A. E. Packham, and P. A. Conrad. 2002. Coastal freshwater runoff is a risk factor for *Toxoplasma gondii* infection of southern sea otters (*Enhydra lutris nereis*). *International Journal for Parasitology* **32**:997-1006.
- Miller, M. A., M. E. Grigg, W. A. Miller, H. A. Dabritz, E. R. James, A. C. Melli, A. E. Packham, D. Jessup, and P. A. Conrad. 2007. *Toxoplasma gondii* and *Sarcocystis neurona* infections of pacific coastal sea otters in California, USA: evidence for land-sea transfer of biological pathogens. *Journal of Eukaryotic Microbiology* **54**:48S-49S.
- Miller, M. A., R. M. Kudela, A. Mekebri, D. Crane, S. C. Oates, M. T. Tinker, M. Staedler, W. A. Miller, S. Toy-Choutka, C. Dominik, D. Hardin, G. Langlois, M. Murray, K. Ward, and D. A. Jessup. 2010b. Evidence for a Novel Marine Harmful Algal Bloom: Cyanotoxin (Microcystin) Transfer from Land to Sea Otters. *PLoS One* **5**:Article No.: e12576.
- Miller, M. A., W. A. Miller, P. A. Conrad, E. R. James, A. C. Melli, C. M. Leutenegger, H. A. Dabritz, A. E. Packham, D. Paradies, M. Harris, J. Ames, D. A. Jessup, K. Worcester, and M. E. Grigg. 2008b. Type X *Toxoplasma gondii* in a wild mussel and terrestrial carnivores from coastal California: new linkages between terrestrial mammals, runoff and toxoplasmosis of sea otters. *Int J Parasitol* **38**:1319-1328.
- Miller, M. A., M. E. Moriarty, L. Henkel, M. T. Tinker, T. L. Burgess, F. I. Batac, E. Dodd, C. Young, M. D. Harris, D. A. Jessup, J. Ames, and C. Johnson. 2020. Predators, Disease, and Environmental Change in the Nearshore Ecosystem: Mortality in southern sea otters (*Enhydra lutris nereis*) from 1998-2012. *Frontiers in Marine Science* **7**:582.
- Monson, D. H., D. F. Doak, B. E. Ballachey, and J. L. Bodkin. 2011. Could residual oil from the Exxon Valdez spill create a long-term population "sink" for sea otters in Alaska? *Ecological Applications* **21**:2917-2932.
- Monson, D. H., D. F. Doak, B. E. Ballachey, A. Johnson, and J. L. Bodkin. 2000. Long-term impacts of the Exxon Valdez oil spill on sea otters, assessed through age-dependent mortality patterns. *Proceedings of the National Academy of Sciences of the United States of America* **97**:6562-6567.

- Moriarty, M. E., M. T. Tinker, M. A. Miller, J. A. Tomoleoni, M. M. Staedler, J. A. Fujii, F. I. Batac, E. M. Dodd, R. M. Kudela, and V. Zubkousky-White. 2021. Exposure to domoic acid is an ecological driver of cardiac disease in southern sea otters☆. *Harmful Algae* **101**:101973.
- Moxley, J. H., T. E. Nicholson, K. S. Van Houtan, and S. J. Jorgensen. 2019. Non-trophic impacts from white sharks complicate population recovery for sea otters. *Ecology and Evolution* **9**:6378–6388.
- Murata, S., S. Takahashi, T. Agusa, N. J. Thomas, K. Kannan, and S. Tanabe. 2008. Contamination status and accumulation profiles of organotins in sea otters (*Enhydra lutris*) found dead along the coasts of California, Washington, Alaska (USA), and Kamchatka (Russia). *Marine Pollution Bulletin* **56**:641-649.
- Nakata, H., K. Kannan, L. Jing, N. Thomas, S. Tanabe, and J. P. Giesy. 1998. Accumulation pattern of organochlorine pesticides and polychlorinated biphenyls in southern sea otters (*Enhydra lutris nereis*) found stranded along coastal California, USA. *Environmental Pollution* **103**:45-53.
- Nicholson, T. E., K. A. Mayer, M. M. Staedler, J. A. Fujii, M. J. Murray, A. B. Johnson, M. T. Tinker, and K. S. Van Houtan. 2018. Gaps in kelp cover may threaten the recovery of California sea otters. *Ecography* **41**:1751-1762.
- Novaczek, I., M. Madhyastha, R. Ablett, A. Donald, G. Johnson, M. Nijjar, and D. E. Sims. 1992. Depuration of domoic acid from live blue mussels (*Mytilus edulis*). *Canadian Journal of Fisheries and Aquatic Sciences* **49**:312-318.
- Ralls, K., T. C. Eagle, and D. B. Siniff. 1996. Movement and spatial use patterns of California sea otters. *Canadian Journal of Zoology* **74**:1841-1849.
- Reese, S. L., J. A. Estes, and W. M. Jarman. 2012. Organochlorine contaminants in coastal marine ecosystems of southern Alaska: inferences from spatial patterns in blue mussels (*Mytilus trossulus*). *Chemosphere* **88**:873-880.
- Rouse, N. M., K. L. Counihan, C. E. Goertz, and K. N. Duddleston. 2021. Competency of common northern sea otter (*Enhydra lutris kenyoni*) prey items to harbor *Streptococcus lutetiensis* and *S. phocae*. *Diseases of Aquatic Organisms* **143**:69-78.
- Rudebusch, J., B. B. Hughes, K. E. Boyer, and E. Hines. 2020. Assessing anthropogenic risk to sea otters (*Enhydra lutris nereis*) for reintroduction into San Francisco Bay. *PeerJ* **8**:e10241.
- Shanebeck, K. M., and C. Lagrue. 2020. Acanthocephalan parasites in sea otters: Why we need to look beyond associated mortality.... *Marine Mammal Science* **36**:676-689.
- Shanks, A. L., S. G. Morgan, J. MacMahan, A. J. Reniers, M. Jarvis, J. Brown, A. Fujimura, L. Ziccarelli, and C. Griesemer. 2018. Persistent differences in horizontal gradients in phytoplankton concentration maintained by surf zone hydrodynamics. *Estuaries and coasts* **41**:158-176.
- Shanks, A. L., S. G. Morgan, J. MacMahan, A. J. Reniers, R. Kudela, M. Jarvis, J. Brown, A. Fujimura, L. Ziccarelli, and C. Griesemer. 2016. Variation in the abundance of *Pseudo-nitzschia* and domoic acid with surf zone type. *Harmful Algae* **55**:172-178.
- Shapiro, K., M. A. Miller, A. E. Packham, B. Aguilar, P. A. Conrad, E. Vanwormer, and M. J. Murray. 2016. Dual congenital transmission of *Toxoplasma gondii* and *Sarcocystis neurona* in a late-term aborted pup from a chronically infected southern sea otter (*Enhydra lutris nereis*). *Parasitology* **143**:276-288.
- Shapiro, K., E. VanWormer, A. Packham, E. Dodd, P. A. Conrad, and M. Miller. 2019. Type X strains of *Toxoplasma gondii* are virulent for southern sea otters (*Enhydra lutris nereis*) and present in felids from nearby watersheds. *Proceedings of the Royal Society B* **286**:20191334.
- Shumway, S. E. 1990. A review of the effects of algal blooms on shellfish and aquaculture. *Journal of the world aquaculture society* **21**:65-104.
- Snøeij, N., A. Penninks, and W. Seinen. 1987. Biological activity of organotin compounds—an overview. *Environmental research* **44**:335-353.

- Sorte, C. J., W. T. Peterson, C. A. Morgan, and R. L. Emmett. 2001. Larval dynamics of the sand crab, *Emerita analoga*, off the central Oregon coast during a strong El Niño period. *Journal of Plankton Research* **23**:939-944.
- Staveley, C. M., K. B. Register, M. A. Miller, S. L. Brockmeier, D. A. Jessup, and S. Jang. 2003. Molecular and antigenic characterization of *Bordetella bronchiseptica* isolated from a wild southern sea otter (*Enhydra lutris nereis*) with severe suppurative bronchopneumonia. *Journal of veterinary diagnostic investigation* **15**:570-574.
- Straub, M. H., and J. E. Foley. 2020. Cross-sectional evaluation of multiple epidemiological cycles of *Leptospira* species in peri-urban wildlife in California. *Journal of the American Veterinary Medical Association* **257**:840-848.
- Tanaka, K. R., K. S. Van Houtan, E. Mailander, B. S. Dias, C. Galginitis, J. O'Sullivan, C. G. Lowe, and S. J. Jorgensen. 2021. North Pacific warming shifts the juvenile range of a marine apex predator. *Scientific reports* **11**:1-9.
- Thomas, N., C. L. White, J. Saliki, K. Schuler, D. Lynch, O. Nielsen, J. Dubey, and S. Knowles. 2020. Canine distemper virus in the sea otter (*Enhydra lutris*) population in Washington State, USA. *Journal of Wildlife Diseases* **56**:873-883.
- Thomas, N. J., and R. A. Cole. 1996. The risk of disease and threats to the wild population. *Endangered Species Update* **13**:23-27.
- Tinker, M. T., J. Bodkin, L. Bowen, B. Ballachey, G. Bentall, A. Burdin, H. A. Coletti, G. Esslinger, B. Hatfield, M. C. Kenner, K. Kloecker, B. Konar, A. K. Miles, D. Monson, M. Murray, B. P. Weitzman, and J. A. Estes. 2021a. Sea otter population collapse in southwest Alaska: assessing ecological covariates, consequences, and causal factors. *Ecological Monographs* **Early Edition**.
- Tinker, M. T., L. P. Carswell, J. A. Tomoleoni, B. B. Hatfield, M. D. Harris, M. A. Miller, M. E. Moriarty, C. K. Johnson, C. Young, L. Henkel, M. M. Staedler, A. K. Miles, and J. L. Yee. 2021b. An Integrated Population Model for Southern Sea Otters. US Geological Survey Open-File Report No. 2021-1076. Reston, VA.
- Tinker, M. T., B. B. Hatfield, M. D. Harris, and J. A. Ames. 2016. Dramatic increase in sea otter mortality from white sharks in California. *Marine Mammal Science* **32**:309-326.
- Tinker, M. T., J. L. Yee, K. L. Laidre, B. B. Hatfield, M. D. Harris, J. A. Tomoleoni, T. W. Bell, E. Saarman, L. P. Carswell, and A. K. Miles. 2021c. Habitat Features Predict Carrying Capacity of a Recovering Marine Carnivore. *The Journal of Wildlife Management* **85**:303-323.
- Wendell, F. E., R. A. Hardy, and J. A. Ames. 1986. An assessment of the accidental take of sea otters, *Enhydra lutris*, in gill and trammel nets. California Fish and Game, Marine Resources Technical Report 54.
- White, C. L., E. W. Lankau, D. Lynch, S. Knowles, K. L. Schuler, J. P. Dubey, V. I. Shearn-Bochsler, M. Isidoro-Ayza, and N. J. Thomas. 2018. Mortality trends in northern sea otters (*Enhydra lutris kenyoni*) collected from the coasts of Washington and Oregon, USA (2002–15). *Journal of Wildlife Diseases* **54**:238-247.
- Williams, G., K. S. Andrews, S. Katz, M. L. Moser, N. Tolimieri, D. Farrer, and P. Levin. 2012. Scale and pattern of broadnose sevengill shark *Notorynchus cepedianus* movement in estuarine embayments. *Journal of Fish Biology* **80**:1380-1400.