

EFFECTS OF CLIMATE CHANGE ON ARCTIC MARINE MAMMAL HEALTH

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Abstract. The lack of integrated long-term data on health, diseases, and toxicant effects in Arctic marine mammals severely limits our ability to predict the effects of climate change on marine mammal health. The overall health of an individual animal is the result of complex interactions among immune status, body condition, pathogens and their pathogenicity, toxicant exposure, and the various environmental conditions that interact with these factors. Climate change could affect these interactions in several ways. There may be direct effects of loss of the sea ice habitat, elevations of water and air temperature, and increased occurrence of severe weather. Some of the indirect effects of climate change on animal health will likely include alterations in pathogen transmission due to a variety of factors, effects on body condition due to shifts in the prey base/food web, changes in toxicant exposures, and factors associated with increased human habitation in the Arctic (e.g., chemical and pathogen pollution in the runoff due to human and domestic-animal wastes and chemicals and increased ship traffic with the attendant increased risks of ship strike, oil spills, ballast pollution, and possibly acoustic injury). The extent to which climate change will impact marine mammal health will also vary among species, with some species more sensitive to these factors than others. Baseline data on marine mammal health parameters along with matched data on the population and climate change trends are needed to document these changes.

Key words: Arctic; biotoxins; climate change; disease; health; human interaction; marine mammals; sentinel species; toxicants.

INTRODUCTION

Health is a difficult concept to define, but most simply, it is an absence of disease, with disease defined as “any departure from normality” (Rothman and Greenland 1998). The overall health of an individual animal is a complex interaction between its immune status, body condition, pathogen characteristics, toxicant load, and environment. The consequent effects of a disease at the population level then depend on the fecundity, survival, and dispersal of the population (Gulland and Hall 2006). The effects of climate change on the health of marine mammals in the Arctic may be direct or indirect. Direct effects include changes in health associated with environmental changes such as loss of habitat, temperature stress, and exposure to severe weather. These direct effects are discussed in Laidre et al. (2008). Indirect effects could result from changes in host–pathogen associations due to altered pathogen transmission or host resistance, changes in body condition due to alterations in predator–prey relationships, changes in exposure to toxicants, or increased human

interactions (boat strike, fisheries activities, acoustic injury, and sound pollution) through increased ship traffic, offshore production, or development and exploration (Table 1).

To detect changes in marine mammal health related to climate change, we need baseline data from which change can be evaluated. These data are quite limited; especially lacking are studies on infectious disease epidemiology and biologic effects of toxicants in Arctic marine mammals. Available information includes a detailed study on lungworm in ringed seals (*Phoca hispida*) (Onderka 1989) and data on the prevalence of exposure to some microparasites that have caused disease outbreaks in marine mammals in other parts of the world, although not reported to date in the Arctic. These include serological surveys for *Toxoplasma gondii*, morbilliviruses, *Brucella* sp., calicivirus, herpes viruses, rabies, *Salmonella* sp., and influenza virus A (O'Hara et al. 1998, Duignan 1999, Tryland 2000, Nielsen et al. 2001, Tryland et al. 2001, 2005, Aschfalk et al. 2002, Calle et al. 2002, Dubey et al. 2003, Measures et al. 2004, Philippa et al. 2004). Some studies of pathogenic organisms have been conducted, including surveys and characterizations of poxviruses, *Salmonella* sp., *Giardia*, *Cryptosporidia*, *Brucella* sp., and *Clostridium perfringens* toxin (Aschfalk and Muller 2001, Dailey 2001, Dunn et al. 2001, Hughes-Hanks et al. 2005). Baseline histology, hematology, and serum biochemical data are limited

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TABLE 1. A summary of possible health affects for Arctic marine mammals related to climate change.

Effect	Result
Direct	
Loss of sea ice platform	1) Reduction of suitable habitat for feeding, resting, and breeding. 2) Movement and distribution (e.g., polar bears) will be affected. 3) Loss of substrate for prey. 4) Increases in drownings and strandings of animals such as polar bears and walrus.
Increased incidence of severe weather	Hypothermia, drowning, strandings from exhaustion.
Indirect	
Changes in infectious disease transmission (changes in host–pathogen associations due to altered pathogen transmission or host resistance)	1) Increased host density due to reduced habitat, increasing density-dependent diseases. 2) Epidemic disease due to host or vector range expansion. 3) Increased survival of pathogens in the environment. 4) Interactions between diseases, loss of body condition, and increased immunosuppressive contaminants, resulting in increased susceptibility to endemic or epidemic disease.
Alterations in the predator–prey relationship	Affected body condition and, potentially, immune function.
Changes in toxicant pathways (harmful algal blooms [HABS], variation in long-range transport, biotransport, runoff, use of the Arctic)	1) Mortality events (e.g., HABS). 2) Toxic effects of contaminants on immune function, reproduction, skin, endocrine systems, etc.
Other negative anthropogenic impacts	Increased likelihood of ship strikes, fisheries interactions, or acoustic injury, and chemical and pathogen pollution due to shipping or agricultural practices.

(Bossart et al. 2001), and there are no studies that relate these health parameters to infectious agents or population-level effects. Numerous reports are available that document concentrations of contaminants for Arctic marine mammals; however, very few data are available evaluating the effects of these contaminants on marine mammals or subsistence consumers (AMAP 1998, 2004, Hoekstra et al. 2005, O'Hara et al. 2005). While reports on body condition and reproductive rates are available, studies on possible causes of decreased condition and reproduction often are lacking.

The lack of integrated long-term data on health, diseases, and toxicant effects in Arctic marine mammals, as well as the complicated and geographically variable changes in climate, limit our ability to predict the effects of Arctic climate change on marine mammal health. Nevertheless, we can extrapolate from information in the literature on other species to predict what may occur with climate change and various factors that impact health. In this review we summarize available information on trends in health status of marine mammals in the Arctic, describe potential impacts that changes in climate may have on health, and summarize information needs to assess the impact climate change will have on health of Arctic marine mammals.

HEALTH EFFECTS

Infectious diseases

Infectious diseases are the result of a pathogen invading the host and negatively affecting the host's ability to function. Host–pathogen–environment interactions that increase either the host's susceptibility or the pathogen's virulence or transmission rate may result in a change in the incidence of infectious disease.

Climatic warming may alter the host–pathogen relationship by several mechanisms, including: (1) changes in pathogen transmission rates due to changes in host density or movement patterns; (2) increased pathogen survival, including relaxed overwintering restrictions and increased survival of pathogens in the environment; (3) carrier host species or vector range extension (Harvell et al. 2002, Bradley et al. 2005); (4) changes in host susceptibility to disease due to nutritional or toxic stresses (Seth and Boetra 1986, O'Hara and O'Shea 2001).

Pathogen transmission.—Transmission of infectious diseases among marine mammals may be density dependent or frequency dependent. Density-dependent diseases are those in which infection occurs through random contacts between infected and susceptible individuals, and the likelihood of a susceptible host acquiring infection in a given time interval is proportional to the density of infected individuals. Density-dependent diseases reported in marine mammals include morbilliviruses, which have caused die-offs of thousands of phocids when the susceptible host population was greater than a threshold level (Kennedy 1998). Frequency-dependent diseases are those for which the probability of an individual acquiring the disease is a function of the frequency of diseased animals, rather than the absolute density, and frequency-dependent diseases are less well documented in marine mammals, but in terrestrial mammals are usually transmitted by sexual contact, a vector, or contact within a social group. Many marine mammal diseases are likely both density and frequency dependent due to the social behavior of many of these animals; at low population numbers, most pinnipeds will continue to congregate at similar densities

on rookeries and haul-outs (Harwood and Hall 1990, Heide-Jorgensen and Harkonen 1992).

The current trend towards a warming climate has been associated with a worldwide increase in reports of disease affecting marine organisms (Kuiken et al. 2006). In five mass mortality events of pinnipeds with available data, mean monthly temperatures preceding the mortalities were higher than the 10-year average (Lavigne and Schmitz 1990). For species that haul out on ice, fewer suitable haul-outs could increase haul-out densities, increasing transmission rates (Lavigne and Schmitz 1990). Changes in haul-out patterns (concentrations and distributions) may also increase risk of contact between marine mammals and terrestrial mammals harboring pathogens to which the former are susceptible but rarely exposed. In the Beaufort Sea region of northern Alaska and in the Chukchi Sea region of eastern Siberia, increasing numbers of polar bears are remaining on land in summer and early autumn (S. Amstrup and S. Schliebe, *personal communication*). Because these coastal regions are already occupied by grizzly bears, this more regular co-occurrence could increase the likelihood of disease transmission between these two closely related but normally spatially separated species. In Kaktovik, Alaska, where remains of subsistence-hunted bowhead whales are stockpiled, encounters between polar and grizzly bears have become common. These encounters suggest similar situations could occur at walrus haul-outs and other places where marine mammal carcasses regularly occur (S. Miller, *personal communication*).

Pathogen survival and range extension.—Climate influences pathogen distribution, and weather influences the timing of disease outbreaks (Dobson and Carper 1993). Therefore, changes in climate and weather can change the distribution and timing of outbreaks. The range of a pathogen can increase if environmental conditions increase its survival and/or its vectors. In turn, the expansion of the range of a pathogen can have dramatic consequences if it is introduced into a previously naive population (e.g., one that lacks pathogen-specific immunity). For example, in the Gulf of Mexico, intensity and prevalence of “dermo,” an endemic disease of oysters caused by the protozoan *Perkinsus marinus*, drop during cool and wet El Niño years and rise during warm and dry La Niña years (Powell et al. 1996). Increasing winter water temperatures along the northeast coast of the United States have reduced overwinter mortality of the protozoa and allowed subsequent northward spread of epidemics in oysters as far as Maine (Cook et al. 1998).

Data on the influence of climate on the bacterial diseases of marine mammals are lacking. However, the changing epidemiology of *Vibrio* species, a marine bacterium infecting a range of animals including humans and marine mammals, may serve as a model according to which changes in distribution of other bacteria may be predicted. Outbreaks of human cholera due to *Vibrio cholerae* have been associated with El Niño events in

Peru, and the prevalence of diarrhea cases due to *Vibrio cholerae* in Bangladesh is associated with increased sea surface temperatures (Lipp et al. 2002). *Vibrio parahaemolyticus* is found in the environment only when water temperatures are $>17^{\circ}\text{C}$ and salinities are <13 parts per thousand (Kelly and Stroh 1988). In 2004, an outbreak of food poisoning due to farmed oysters occurred in Prince William Sound, Alaska, extending the known range of an oyster source of this disease by 1000 km north. This was correlated with increased temperatures of the ocean water (McLaughlin et al. 2005). *Vibrio parahaemolyticus* is a pathogen of marine mammals. In 2005 in Prince William Sound, the first cases of infection by this bacteria of marine mammals in Alaska were documented; northern sea otters (*Enhydra lutris kenyoni*) died due to septicemia with *V. parahaemolyticus*, and clinical cases of enteritis due to *V. parahaemolyticus* occurred in live-stranded northern sea otters (P. Tuomi, *personal communication*). However, because of lack of historical information on specific causes of death in this otter subspecies in Alaska, it is not known whether this is a new disease in this region. An increase in water temperature will likely enhance survival of some marine bacterial pathogens and increase exposure resulting in an increased prevalence of bacterial diseases such as *V. parahaemolyticus*.

Helminth parasites of terrestrial wildlife that release eggs or free-living stages into the environment or use invertebrate intermediate hosts to complete life cycle stages are very susceptible to changes in temperature and humidity (Harvell et al. 2002). Hookworms (*Uncinaria lucasi*) have been an important nematode parasite of northern fur seals (*Callorhinus ursinus*). Larvae of these parasites are transmitted to newborn pups through the milk, with adult parasites present only in neonatal animals. Eggs are shed in the feces of infected pups, and the larvae embryonate in the environment and are transmitted transdermally to animals of all ages. Relatively high numbers of adult hookworms in neonatal pups can cause fatal anemia due to hemorrhagic enteritis (Dailey 2001). Hookworms were an important cause of northern fur seal pup mortality with a strong correlation between proportions of northern fur seal pup deaths due to hookworm anemia and the estimated numbers of pups born on St. Paul Island, Alaska (Raga et al. 1997). Since transmission depends on transdermal migration of free-living larvae, environmental factors such as temperature and humidity are important in the maintenance of this parasite.

Infectious disease outbreaks can occur when carrier animals in which the pathogen is generally “non-pathogenic” come in contact with a susceptible host species in which clinical disease occurs. During the morbillivirus outbreaks in northern Europe, migrating Arctic and subarctic seal species, such as harp seals (*Phoca groenlandicus*) from Greenland (Dietz et al. 1989) or gray seals (*Halichoerus grypus*) (Hammond et al.

2005, Harkonen et al. 2006), may have been carriers that introduced a morbillivirus into immunologically naive harbor seals. If northern expansion of subarctic species occurred into the Arctic regions due to changes in air and water temperatures, there would be the potential for contact between carrier and naive host species. To this date marine mammal morbillivirus outbreaks have not been reported in the Pacific Basin. With the projected decrease in sea ice and projected expansion in ranges of various subarctic and Arctic phocids (Walsh 2008), another scenario would be contact between Atlantic phocids with endemic morbillivirus such as grey and harp seals with Pacific Basin phocids that are immunologically naive. Although currently an unlikely scenario, small changes in host ranges have the potential to result in large disease epidemics, so that a new introduction could have significant population impacts.

Warming temperatures also may allow extension of domestic animals into the Arctic (Charron 2002). Domestic animal production can amplify pathogens due to high densities of animals and associated stresses and can introduce new pathogens into wildlife species that come into contact with domestic animals. Terrestrial examples include transmission of pathogenic *Pasteurella multocida*, *Mannheimia haemolytica*, and *P. trehalosi* endemic in domestic sheep to susceptible bighorn (*Ovis canadensis*) and Dall sheep (*Ovis dalli*), resulting in epidemic disease with high mortality (Miller 2001). Other examples include local population extinction of African wild dogs (*Lycaon pictus*) due to rabies from domestic dogs; mortality in several African antelope species due to rinderpest from domestic cattle; mortality of African lions (*Panthera leo*) due to canine distemper virus most likely acquired from domestic dogs; and epidemics of canine distemper in Baikal seals (*Phoca sibirica*) and Caspian seals probably transmitted to seals via domestic dogs (Kennedy et al. 2000, Gog et al. 2002). The true reservoir for canine distemper in Caspian seals has not been determined, and the Caspian seal population itself or another terrestrial carnivore as yet undetermined could act as a reservoir (Kuiken et al. 2006).

Diseases transmitted by arthropod vectors may increase their range if vector survival is enhanced by warming in the Arctic. The greatest effect of climate change on transmission of vector-borne disease is likely to be observed at the extremes of the ranges of temperatures at which transmission occurs (Githeko et al. 2000). Incidences of tick-borne encephalitis and other tick-borne diseases have increased in humans in Sweden since the mid-1980s in association with the northward expansion and increased density of the disease-transmitting tick *Ixodes ricinus* (Lindgren et al. 2000). Outbreaks of the most common North American pathogenic arbovirus for humans, St. Louis encephalitis virus, have occurred farther north during unseasonably warm years (Patz et al. 1996). Cetaceans are susceptible to St. Louis encephalitis virus (fatal encephalitis was

reported in a captive killer whale [Buck et al. 1993]), so extension of the vector range for this virus could impact cetaceans. West Nile virus is transmitted to mammals from avian hosts by mosquitoes, especially *Culex* spp., and caused mortality of phocids in display facilities as it spread across the United States in the current epidemic (Del Piero et al. 2006). This suggests phocids may be very susceptible to this virus, so that establishment of West Nile virus and its vectors in the Arctic could cause mortality of subarctic and, potentially, Arctic phocids.

It seems likely that climate change will cause some changes in distribution of pathogens and patterns of diseases due to the factors discussed above. However, it is difficult to predict how significant this will be at the population level. Even with the introduction of a "new" epidemic disease such as can cause initial extensive mortality and morbidity, once a proportion of the population develops immunity or the numbers are reduced below a threshold level, the epidemic will resolve. In the case of the morbillivirus epidemics of the 1980s and 1990s, there was an initial drop in population, and age and sex distributions were skewed for several years after the epidemic, but these changes were followed by a recovery to pre-epidemic numbers, followed by another epidemic when enough immunologically naive animals were again present in the population (Heide-Jorgensen et al. 1992, Gulland and Hall 2006, Harkonen et al. 2006). Severe epidemics might reduce host population density to such an extent that stochastic events or previously unimportant ecological factors may further reduce the host population size (Harwood and Hall 1990).

Body condition

There will very likely be changes in nutritional status of Arctic marine mammals due to prey shifts and changes in the food web due to climate change (Stirling 2002, Stirling and Smith 2004, Bluhm and Gradinger 2008). For some species this will be a negative change; for others, it may be positive, depending on how generalized or specific they are in their feeding habits and how dependent they are on the sea ice platforms. Food depletion and stress are well-known causes of immune suppression in humans, laboratory rodents, and terrestrial mammals (Seth and Boetra 1986, Beck et al. 2004, Cunningham-Rundles et al. 2005, Landgraf et al. 2005). Although the effects of food depletion and stress on marine mammal immune responses have not been well investigated, the few data available suggest marine mammal immunity is affected by similar factors as that of terrestrial mammals. These changes in immune function may increase host susceptibility to endemic pathogens, thus increasing prevalence of diseased individuals in these populations. For example, stranded harbor seals with higher circulating cortisol levels (as an indication of physiologic stress) are more likely to die of endemic herpesvirus than animals with lower cortisol levels (Gulland et al. 1999). Poor nutrition is also a well-

known factor in reduced reproductive success, affecting age of onset of puberty, fertility, and success in maintaining pregnancies in domestic animals (Gerloff and Morrow 1986), all of which could have major effects at the population level (Harwood et al. 2000).

Toxicant exposures

Toxicants can be produced by organisms (biotoxins), released from geologic stores (e.g., heavy metals, some hydrocarbons), or are anthropogenic (some of the persistent organic pollutants, petroleum hydrocarbons, heavy metals, and radioactive substances). Marine mammals are exposed to a wide range of types and amounts of toxicants and are expected to show different dose-dependent levels of exposure and effects with climate change. Scientists have been monitoring contaminant residues in marine mammal tissues, especially organochlorines and heavy metals, for a longer period of time than any other group of mammals. Ironically, marine mammals are among the least studied in terms of controlled experiments related to effects of toxicants, making it difficult to interpret results of surveys of residues in tissues in terms of significance to the health of the animal. There have been several studies demonstrating that polychlorinated biphenyls (PCBs) and dichlorodiphenyltrichloroethanes (DDTs) are associated with immunosuppression, reproductive failure, and endocrine disruption in marine mammals and domestic and laboratory animals. Bone and reproductive pathology in seals has been linked with elevated levels of organochlorines (AMAP 1998, 2004, O'Hara and Shea 2001, Beckmen et al. 2003). In some cases, epidemics caused by morbilliviruses have had higher mortality in populations from polluted regions where elevated PCBs and dichlorodiphenyldichloroethylene (DDE) in tissues may have compromised immune function. In other cases, organochlorine and zinc levels do not appear to be a factor in the mortality event (Kuiken et al. 2006). During times of nutritional stress, lipophilic contaminants stored in the blubber are released into the blood at an increased rate, and it is possible that contaminants could work in concert with nutritional and disease stresses to cause significant morbidity and mortality.

Climate change can be expected to alter the exposure levels of marine mammals to a variety of toxicants through a variety of means including changes in distribution of harmful algal blooms (HABs), changes in long-range atmospheric and oceanographic transport (including interactions with sea ice), biotransport, changes in feeding ecology, increased and altered runoff, and increased human involvement in the Arctic. The possible changes in exposure of the different toxicants are difficult to predict and will largely depend upon the chemical features of the exact toxicant (Macdonald et al. 2005).

Biotoxins.—There is evidence that the frequency and distribution of toxigenic phytoplankton blooms are increasing worldwide (Tester 1994, Van Dolah 2000,

Mos 2001). The factors leading to these increases are unclear, but they likely involve changes in water temperature, micronutrient availability, and salinity that could result from large-scale oceanographic changes or smaller scale changes in terrestrial effluent (Patz et al. 1996) or an increase in the global dust load due to desertification (Mos 2001). The effects of these algal blooms on marine mammal health are still poorly understood, although marine mammal mortality events associated with these blooms are becoming increasingly recognized (Scholin et al. 2000), including die-offs resulting from exposure to saxitoxin, domoic acid, and brevetoxin (Van Dolah 2000). To date, HABs have not been reported in Arctic marine mammals, although this may be a result of lack of monitoring rather than true absence. However, with sufficient changes in water temperature, toxigenic species from the *Pseudonitzschia*, *Alexandrium*, and *Karenia* genera could potentially bloom in Arctic waters and cause marine mammal mortalities. Recent evidence indicates that domoic acid, typically produced by *Pseudonitzschia* sp., are present at low levels in razor clams in Alaska (Horner et al. 1997), and this toxin has been found at low levels in sea otters from this region (V. Gill and P. Tuomi, *personal communication*). The actual effects of this toxin on the health of sea otters are still to be determined. The exact hydrographic and atmospheric conditions required to produce a HAB have not been determined well, making it difficult to predict the likelihood of HAB occurrence in the warming Arctic.

Contaminants.—

1. *Long-range atmospheric and oceanographic transport.*—It is likely that climatic warming, causing changes in temperature, precipitation, and weather patterns, will alter the pathways, dynamics (e.g., persistence), and concentrations of pollutants entering the Arctic via long-range transport on air and ocean currents (Wania et al. 1998, AMAP 2004, Macdonald et al. 2005). Numerous contaminants travel to the Arctic via the atmosphere and the hydrologic cycle through air movement, precipitation, river outflow, and ocean currents. Sea ice dynamics are important for transport of contaminants due to the presence of contaminants embedded in ice and for retention of contaminants by capping of the ocean, trapping some volatile contaminants within the water. Changes in these abiotic factors may alter deposition and re-volatilization of numerous toxicants. Whether these will be increases or decreases depends on the chemistries of these individual components, and there are extensive reviews on these projected changes (Macdonald et al. 2005).

2. *Biotransport.*—Biotransport, transport by migratory animals rather than passive transport via air or water, is receiving increasing attention (Ewald et al. 1998, Wania 1998). Deposition of contaminants by biotransport occurs through excretion (urine, feces, shedding/molting, etc.), carcasses, eggs/roe, and predator-prey relationships. Clearly climate-driven changes in

migratory patterns and ranges of these species will alter the dynamics of biotransport. Marine mammals are vehicles for the biotransport of chemicals to the Arctic as well as local recipients. As an example, as sea ice recedes, gray whales may remain longer in Arctic waters. The Eastern Pacific stock of gray whales spends the summer in the Bering and Chukchi seas, and they winter off the Pacific coast of Mexico and California (Wania 1998). Relatively reliable population estimates and recent contaminant concentrations in stranded individuals exist for this stock, allowing for an estimate of the amount of some persistent organic pollutants (POPs) contained in these whales. Wania (1998) estimated that the total amount of PCBs and DDT contained in the eastern North Pacific (ENP) gray whale population and therefore transported annually with gray whales is in the range of 20–150 kg PCBs and 1–40 kg DDT. The total amount of PCBs and DDTs in the world's baleen whales is likely on the order of a few tons (Wania 1998). For DDTs, the gross fluxes provided by whales may be comparable to those of air and ocean currents. Where these contaminants enter the food chain depends on where the animal is consuming food vs. where excretion and death occur.

3. *Changes in feeding ecology with resultant changing toxicant exposure.*—Shifts in prey will likely occur with climate due to changes in prey availability (Laidre et al. 2008). Highly specialized feeders are less likely to shift prey as easily as the generalist consumers of prey. Even though some species can shift prey more easily it should be recognized that “new” prey could be greater or lesser sources of contaminants. Animals that shift prey types could alter the associated level of toxicant exposure, especially if prey are from different trophic levels. For example, ringed seals feeding on invertebrates as opposed to fish will reduce exposure to mercury and some organohalogens, while increasing exposure to cadmium (Hoekstra et al. 2003, Dehn et al. 2005), whereas beluga whales shifting between benthic and epibenthic prey will alter mercury and cadmium exposure in the opposite direction (Dehn et al. 2005). Minke whales are documented as having varying tissue composition based on history of invertebrate vs. fish consumption (Haug et al. 1996, Olsen and Holst 2001). Dietary preference has been shown to affect PCB concentrations in killer whales (Ross et al. 2000).

4. *Change in contaminant pathways related to increased human presence in the Arctic.*—With warming, it is anticipated that agricultural practices will increase in the subarctic and Arctic. Land use practices are well-known causes of contamination, both chemical and microbial, and increased erosion. Many regions of the Arctic are experiencing growth in human habitation. The run-off from these communities and associated facilities, including landfills, power generation facilities, fuel storage areas, sewage, waste combustion, etc., will contaminate the local coastal environment. The health of polar bears may be compromised by their attraction

to landfills and dumpsters for food, possibly resulting in increased mortality as humans defend themselves and their property and bears are exposed to a wide variety of contaminants. Numerous other industries could enter a warmer north and increase coastal activity (i.e., ports, shipping, tourism, offshore oil and gas exploration and drilling, etc.), introducing sources of contamination, including petroleum hydrocarbons, tributyl tins, heavy metals, and ballast water.

OTHER NEGATIVE ANTHROPOGENIC FACTORS

Decreased sea ice will most likely result in the opening of the northern waters (Hovelsrud et al. 2008), with the attendant increase in human activity (cargo transport, tourism, military activity, commercial fisheries) that may pose a physical risk to marine mammals due to boat strike, fisheries interactions, acoustic injury, and noise pollution. Boat collisions with large whales are an increasing cause of injury and mortality (Gulland et al. 2001, Jensen and Silber 2003). Propeller injuries are most common in manatees, but are also observed in pinnipeds, cetaceans, and sea otters (Gulland et al. 2001). Entanglement of marine mammals in fishing gear, packing bands, and rope is commonly observed in stranded animals, and observations of entangled live animals and incidental take are noted in all areas in which there are active ocean fisheries (Gulland et al. 2001). Noise pollution is known to alter the movements of migrating bowhead whales and other cetaceans and is thought to be a form of stress (Richardson et al. 1995).

CONCLUSION AND SUGGESTIONS FOR FUTURE RESEARCH

Baseline data on the health status of Arctic marine mammals are needed to determine the direct and indirect effects of climate change on health. The occurrence of infectious disease is a complex process in the individual, and the determination of its significance at the population level is a daunting task (Gulland and Hall 2006). Data on baseline health parameters; identity, prevalence, and intensity of pathogens; and effects of toxicants and information on modes of disease transmission, host specificity, temporal and spatial patterns in diseases, and the relationship to environmental factors are all needed to understand the potential effects of climate change on marine mammal health. These data must be integrated with demographic data on marine mammals to determine whether effects of diseases and toxicants are significant at the population level. A similar situation exists for marine mammals in the Antarctic in which very little baseline data are available, but these species face similar climate change-related stresses. It would be optimal to develop collaborations between Arctic and Antarctic marine mammal health researchers and to determine representative species for in-depth studies in order to be able to use resources most advantageously. Optimal species would be those predicted to be most sensitive to climate change (Laidre et al. 2008), of reasonable ease to sample (in order to get

reasonable sample sizes, a difficult task in the environment of the Arctic), and of importance to subsistence users. Sampling should be structured by age, sex, and geographic distribution.

In the Arctic, there is a close tie between the native communities and marine mammals, the latter being vital to the nutrition and culture of the human communities. Factors that affect marine mammals can also directly or indirectly affect these native communities. Many of the disease agents to which marine mammals are susceptible are zoonotic and of concern to human health, including *Brucella* sp., influenza A, *Giardia* sp., *Leptospira* spp., and West Nile virus. The toxicants of concern (see *Health effects: Toxicant exposures: Biotoxins and Contaminants*) are also of possible concern for people. It would be optimal for the veterinary, native, and human health communities to work together to monitor these diseases, contaminants, and marine mammals, structuring these studies to use marine mammals as sentinels for human as well as marine mammal and ecosystem health (Bradley et al. 2005, Rabinowitz et al. 2005).

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