A Plague Upon Them

Helping Wildlife Adapt to Climate Changes and Disease

Aimee Delach

Defenders of Wildlife

Acknowledgements

This paper was supported by a grant from the Wallace Global Fund. Reviewed by J. Christopher Haney. Additional reviews by Brian Lapointe, Carl J. Berg and Peter Jenkins. Research assistance provided by Jean Brennan and Zachary Mang.

Introduction

The impacts of climate change on human disease have been well documented (see, for instance, McMichael et al. 2003). Malaria, cholera, dengue fever, hantavirus, and Rift Valley fever have all been documented to respond to climatic factors, and are expected to have increases in outbreak frequency or severity, or wider geographic ranges in response to anthropogenic climate change (Patz et al. 2005, IPCC 2007). Furthermore, the linkages between wildlife disease and human health have been described in the context of climate change (WCS 2008). However, a detailed discussion of conservation implications of climate change on wildlife disease, and the potentially necessary management responses, has been lacking. This review fills that gap by first providing case studies of how climate change is affecting an array of different wildlife diseases, across multiple habitats and taxa, then suggesting how managers and conservation practitioners can help wildlife navigate the effects of climate change and wildlife disease.

Case studies in this report detail a number of mechanisms by which changing climate conditions are interacting with infectious disease to threaten wildlife. These effects fall roughly into several categories:

Benefit to pathogen. In some cases, the effects of climate change confer an advantage to the pathogen. For instance, warmer temperatures decrease the time it takes some organisms to complete their life cycle. In other cases, climate change is lengthening the growing season, allowing some pathogens to avoid overwintering. For those pathogens that do overwinter, warmer winter temperatures may decrease overwintering mortality.

Benefit to vector. While some diseases are transmitted directly, others have complicated lifecycles that require them to complete one or more stages in a completely different organism, such as a mosquito or a tick. These vectors can also benefit from climate change, via enhanced survivorship, higher metabolic activity, or improved opportunities for breeding or feeding on their hosts.

Stress to host. Impacts associated with climate change include air and water temperature increases, increased frequency of severe storms extended droughts, and phenologic mismatches that affect

food availability. These various problems can stress wildlife and can leave them more susceptible to disease than they would be under more normal circumstances.

Synergistic impacts. In some cases, climate change will allow diseases to manifest in the environment via pathways they otherwise would not. For example, synergistic impacts occur when climate conditions exacerbate other stressors, such as pollution, in ways that enhance disease susceptibility.

By no means are all wildlife diseases predicted to be worsened by climate change. In some cases, disease expression may decrease in severity due to conditions associated with climate change. Brucellosis, which causes stillbirths in affected ungulates, impacts elk and bison in the Greater Yellowstone Region. Wildlife managers provide supplemental feeding to elk in the winter months in order to reduce transmission to cattle; unfortunately this practice causes the disease to spread more rapidly within the elk population. Milder winters, which would reduce the need for supplemental feeding, might help lower the incidence of brucellosis among elk (Cross et al. 2007). Similarly, climate change- induced droughts in the American West may reduce cases of plague by reducing rodent activity (Ben Ari et al. 2008). Ranavirus, an amphibian disease, is less lethal at higher temperatures than lower (Rojas et al. 2005).

Nonetheless, the impacts of several diseases on wildlife will constitute a significant conservation threat to their prospects, as will be described in the profiles below. As such, disease must be addressed in management and planning efforts to help wildlife adapt to climate change.

Chapter 1. Avian Malaria

Malaria is a debilitating illness caused by protozoan parasites – single-celled organisms that are much larger and more complex than bacteria or viruses. Because it is a protozoan, malaria cannot be treated with antibiotics, nor has a vaccine been developed. In humans, malaria poses an enormous public health challenge, with 900 million cases per year and 2.7 million deaths (USAID, undated). It is recognized as a major barrier to economic development in Africa and other tropical regions (USAID, undated). However, malaria strikes animals as well: there are dozens of different strains of malaria, each a different species of the genus *Plasmodium*. Four of these strains affect humans (WHO, undated), while other strains strike other mammals, reptiles or birds. Avian malaria in particular has been a major conservation tragedy in the Hawaiian Islands, and climate change is poised to worsen the threat.

The Malaria Life Cycle

All strains of *Plasmodium* share two important characteristics: 1) they cause illness by attacking the host's red blood cells, reducing the blood's ability to deliver oxygen to the cells and tissues of the body; and 2) all depend on a mosquito to help them complete their life cycle. Unlike a cold, which can be transmitted directly between people, malaria requires a "vector" to transmit the disease between potential hosts. The main vector for human malaria is a group of mosquitoes in the genus *Anopheles*. Avian malaria is spread by a different group called *Culex* mosquitoes. Only female mosquitoes bite: they feed on the blood of humans and animals because they need the high protein and iron food in order to lay eggs. If a mosquito bites a person or animal with malaria, some of the infected blood cells within the blood meal will contain specialized male and female forms of the pathogen. These "gametocytes" take up residence in the mosquito's gut, where they reproduce. The offspring do not harm the mosquito, but migrate to its salivary glands in preparation for the mosquito's next blood meal.

When a mosquito bites a vertebrate, it pierces the skin with a syringe-like proboscis and injects some saliva, which is thought to assist in locating a blood vessel on which to feed (Ribiero et al 1984). If the mosquito is infected with malaria, the plasmodium protozoans that were waiting within the salivary glands enter the bloodstream along with the saliva that the mosquito injects. The parasites quickly migrate to the liver, where they quickly divide (via asexual reproduction) into thousands of

identical "daughter cells." These proceed from the liver and infect red blood cells feeding on the hemoglobin and dividing further. The parasites eventually destroy enough cells to cause anemia and other symptoms. The body also has a difficult time fighting the pathogen, because it spends most of its life hidden within the cells of the blood or liver. However, as each new wave of daughter cells moves out to infect new blood cells, an immune response is triggered: the classic recurrent high fever of the malaria patient. Some of the daughter cells also differentiate into male and female "gametocyte" forms, in preparation for the continuation of the disease's life cycle.

Malaria and Climate

Malaria is endemic to tropical regions around the world, found through much of Africa, South and Southeast Asia, and Central and South America. This distribution is tied strongly to climate factors, particularly rainfall and temperature. All mosquitoes begin their life cycle in water. The adult female lays eggs in a small pool of standing water, preferably one containing dead leaves or other organic material. The eggs hatch into larvae, which feed upon the organic matter and grow for a while before developing into pupae. The pupa is a resting phase during which the mosquito transforms into an adult phase, at which point it from the water. Mosquitoes therefore depend on the presence of water, and prefer still or stagnant water where the larvae won't be washed away. They also prefer small pools, which are likely to have fewer predators, but require regular intervals of rainfall to persist.

External temperature strongly influences the development and activity of the mosquitoes that carry malaria. According to one study, *Culex* mosquitoes at 20°C took 25 days after hatching to develop to adults, but only 7.1 days at 35°C (Mottram et al. 1986). These researchers also found that below 15.6°C the mosquitoes didn't develop at all. *Culex* mosquitoes also feed less at lower temperatures (Eldridge 1968).

In addition to affecting mosquito development, low temperatures also inhibit the lifecycle phase that occurs inside the gut of the mosquito. Only female mosquitoes bite: they feed on the blood of humans and animals because they need the high protein and iron food in order to lay eggs. The adult female mosquitoes that transmit malaria don't live very long--an average of 9 to 14 days under normal circumstances (Scholte et al. 2003). Therefore, the developing parasite is in something of a race against the clock. The plasmodium taken up when a mosquito bites an infected person must

mature and reach the mosquito's salivary glands in time to be injected into new victim before the mosquito dies.

Researchers have known since the early 1900s that low temperatures are lethal to the human malaria strains, particularly early in their development. The parasite was killed outright by exposure to temperatures below 17°C within the first 1.5 days after a mosquito bites an infected person (Grassi 1900, Stratman-Thomas 1940). The parasite is better able to tolerate temperature drops after the crucial early phase, but does take longer to develop. Avian malaria shows a similar pattern: long periods of cold exposure shortly after infection curtails development of the parasite (Chao and Ball 1962). Optimal development of the parasite inside a mosquito occurs at 27°C. A temperature of 18°C doubles the length of time it takes for the parasites to develop and migrate to the salivary glands, and the parasites also require an additional five days to mature to the point where they are capable of transmitting an infection (Ball & Chao 1965).

Because temperature determines the rate at which the plasmodium develops in the mosquito, it plays a critical role in determining whether malaria will be passed on. At 17°C, there is only a 0.001% chance that infective parasites will "beat the clock" and mature before the mosquito dies. At 20°C, the chance is 5.9%, and the probability rises to a maximum of 37% at 30°C (Snow et al. undated). Above this 30°C, the probability drops even though the parasites mature very quickly, because the mosquitoes die of heat stress.

Malaria is endemic throughout most of tropical regions where temperatures remain for extended periods within the favorable zone for parasite development and transmission. The main tropical areas outside the favorable zone are at higher elevations where cooler weather predominates and the parasite has less of a chance of getting a foothold. It is no accident that many of the largest cities within the tropics are located at fairly high elevations. For instance Nairobi, Kenya, is located at 5450 feet and Harare, Zimbabwe, is at 4865 feet.

Malaria Conservation Threat

Many tropical birds have evolved with the threat of avian malaria and have thus built up some resistance to the disease over time. This is not the case where avian malaria has been introduced

recently and the native birds are entirely without defense. In these areas, effects of the disease have been devastating.

One case in point is the birds of Hawaii. The isolation of the archipelago in the Pacific Ocean made Hawaii is a hot spot for bird evolution. Since so few colonizing animals were able to traverse the expanse of ocean to reach the islands, those that made it found a wide array of open habitats and niches, along with very few predators. These few colonists diversified via adaptive radiation to fill various niches: it is thought that as many as 53 Hawaiian endemic species evolved from just 15 colonists to the island chain (Berger 1981).

With this extraordinary array of diversity came an unusual fragility: because Hawaii's birds evolved to fill distinct niches and in the absence of predators and diseases, they are highly vulnerable to changes in habitat and to introduction of exotic predators and parasites. Nearly 40 species were pushed to extinction following Polynesian colonization of the islands, probably due to hunting and destruction of lowland habitats (Olson and James 1982). European colonization brought additional habitat destruction, direct exploitation, and exotic species introduction that caused the extinction or endangerment of dozens more. Of the 71 endemic taxa present on the islands at the time of European contact in the late 1700s, twenty-three were extinct by 1995 (Jacobi and Atkinson 1995), and by 2006 a further eleven had most likely vanished (USFWS 2006).

Among the introductions that eventually proved most devastating was the vector for avian malaria, a *Culex* mosquito, which is thought to have first arrived to Maui's port of Lahaina in 1826 as an unwanted guest on the ship the "Wellington" (van Dine 1904). The mosquito's range remained relatively confined for about 50 years, but spread rapidly throughout Maui and the rest of the islands with the advent of road-building and expanding inter-island travel in the late 1800s (van Dine 1904). The date and route of entry for malaria itself is unknown (LaPointe et al 2005). One possibility is that migratory ducks and shorebirds might have brought the disease, which was later transmitted to resident birds following the introduction and spread of the necessary vector (Warner 1968). Others have pointed to the near-absence of the parasite in migratory individuals and implicate the wave of deliberate introductions of Asian birds in the early 1900s as the source of the infection (van Riper et al. 1986)

By the early 1900s it was apparent that birds were disappearing from lowlands, even in suitable forest habitats: "So far as the human eye can see, their old home offers to the birds practically all that it used to, but the birds themselves are no longer there," wrote H.W. Henshaw in 1902. Six endemics had disappeared from Oahu by 1900 (Warner 1968) and birds on Lanai were becoming increasingly confined to the uplands, despite plenty of remaining habitat in the lowlands (Munro 1944). Contemporary chroniclers of Hawaiian avifauna described sightings birds suffering visible symptoms of viral avian pox, another detrimental introduced disease. However, they also recorded sightings of dead birds, particularly in lowland forests, that showed no outward symptoms, and many of these probably suffered from avian malaria (Warner 1968).

Richard Warner (1968) demonstrated the lethality of avian malaria to native birds by exposing highland-caught honeycreepers to lowland mosquito conditions in large-mesh cages. These birds invariably sickened and died, and post-mortem examination revealed high levels of *Plasmodium* infection in each bird's bloodstream. Warner found that infections were more severe and more likely fatal in the honeycreepers, as compared to introduced finches and white-eyes, suggesting immunological naïveté. He also observed that the native birds exhibited a behavioral naïveté as well: the introduced birds tended to sleep with their feet and faces drawn tightly into their feathers, denying the mosquitoes a good place to feed. The native birds, on the other hand, slept with their faces and feet exposed, and the researchers observed many more *Culex* mosquitoes feeding upon the native birds.

The Climate Change Connection

As described above, the development and transmission of the malaria parasite is strongly dependent on temperature. Below a certain threshold, the parasites do not reach infectivity within the lifespan of the vector mosquito. For the strain of malaria in Hawaii, no transmission of malaria occurs below 13ºC, and very little occurs between 13ºC and 17ºC (LaPointe et al 2005). At issue in the conservation of Hawaii's avifauna is to what elevation that temperature threshold corresponds, and is that threshold moving upslope with the onset of climate change? The answers to these questions will ultimately determine the extent and location of the malaria-free refugia for Hawaii's endemic species.

In his 1968 examination of malaria in honeycreepers, Richard Warner cited 600 meters in elevation as the critical threshold for the temperature limitation of avian malaria. The major islands in the archipelago range from 1000 meters to 4000 meters in height, and on the higher islands the upper limit of the forested area is about 1900 meters (Juvik & Juvik 1998). Thus, the 600-meter threshold suggested that substantial areas of most of the islands were available as disease-free refugia for honeycreepers and other malaria-sensitive island endemics.

Unfortunately, later work by other researchers contradicted this assertion: van Riper and colleagues (1986) found breeding mosquitoes and malaria-infected birds up to 1500 meters in wet forest habitats and up to 1350 meters in dry forests, where there are fewer pools of water for mosquitoes to breed in. This higher threshold substantially contracts the available malaria-free habitat zone. All of Oahu and Lanai, and most of Molokai, lie below this elevation, and high-elevation forest habitat on Kauai, Maui and Hawaii is considerably restricted.

Under plausible scenarios of the impacts of impending climate change on the Hawaiian Islands, the malaria-free area constricts sharply. A warming climate shifts the thresholds for malaria up the slopes of the mountains and further shrinks the area where the disease is absent. One recent analysis of the potential effects of a 2^{o}C increase on protected forested areas was particularly discouraging, because it found that the upslope shifts cut sharply into the available forest habitat (Benning et al. 2002). On Maui, a 2^{o}C increase shrinks the area of very low malaria risk (below 13^{o}C) from 665 hectares to 285 hectares of forested habitat, and the area of medium risk (between 13 and 17^{o}C) from 1,236 hectares to 886 hectares. On the other large islands the situation was even worse. Because the maximum elevation of Kauai is less than 1600 meters, the island already lacks the lowest risk temperature zone, and a 2^{o}C increase cuts the medium risk area from over 15,000 hectares to less than 2,500 hectares. Hawaii, despite having the highest elevation of all the islands and therefore the largest low-temperature zone, suffers from the presence of unforested pasture land upslope from its largest reserve, so a 2^{o}C temperature increase shifts the critical isotherms into unsuitable habitat: the lowest risk area shrinks from 3,120 hectares to 130 hectares. However, the medium risk zone on Hawaii remained the largest of the three islands–7,669 hectares, down from the current 9,229 hectares (Benning et al. 2002).

The dire predictions of the model are already being borne out by observations. By 2001, avian malaria was present in 5.4% of forest birds at 1900 meters in Hawaii's Hakalau Forest National Wildlife Refuge (Freed et al. 2005), the same refuge modeled above. This represented a doubling of the presence of malaria compared to a decade earlier. Mean air temperature has risen slightly over the past decade; perhaps more importantly, the mean air temperature during warm spells has increased significantly. A two-week sequence of air temperatures averaging 15.4^{o}C seems to have provided an opening for the parasite to gain a foothold in the upper reaches of the Refuge's forest habitat. Given that forest gives way to pasture shortly above this elevation, it is clear that rising temperatures are in fact depriving Hawaii's birds of their last refuge.

Helping Wildlife Adapt to the Climate Change & Malaria Threat

Protect and restore appropriately located forested habitat. Models of the impact of climate change on malaria and its mosquito vector indicate that on the island of Hawaii, the malaria-free zone is predicted to move upslope and into a region dominated by pasture rather than forest habitat (Benning et al. 2002). These authors conclude that, "restoration of high-elevation forests above [the Hakalau Wildlife Refuge] is crucial to improving the chances for survival of the honeycreeper species, particularly the Hawaii àkepa, a cavity nester that requires large trees." Forest fragmentation and agricultural land use also increase the likelihood of mosquito presence (Reiter & LaPointe 2007), so reforestation of buffer areas adjacent to refuges may also reduce the rate of the spread of malaria within forest refuges.

Locate and protect surviving birds at low elevations. Despite the apparent doom of the climate change and malaria scenario, there is evidence that resistance to the diseases is beginning to emerge in some species. In particular, the amakihi is now found at lower elevations (Atkinson et al 2000, Woodworth et al. 2005) and these low-elevation populations exhibit genetic differences from upslope birds that indicate evolved resistance to malaria (Foster et al. 2007). Therefore, low-elevation habitats, far from being "sacrifice" zones, may contain crucial reservoirs of birds that have evolved resistance to malaria. Birds found at low elevations could be crucial components of captive breeding programs. Tolerance of malaria may be emerging in the 'I'iwi and the Hawaii 'Akepa as well (Freed et al. 2005).

Reduce other stressors to birds in order to increase the likelihood of evolution of resistance. As mentioned previously, malaria is only one in a long list of threats to Hawaii's native birds. Another devastating factor was the introduction of rats, cats and mongoose to islands that were previously free of mammalian predators on nests and nestlings (Atkinson 1977). If, as mentioned above, tolerance of or resistance to malaria is beginning to emerge naturally in Hawaii's birds, a critical conservation issue will be to make sure that birds exhibiting tolerance live to pass it on to their offspring. Reducing nest predation by controlling rodents and other predators may pay great dividends in facilitating the spread of the genes that confer malaria resistance (Kilpatrick 2005).

Continue research on disease and vector control methods. Continued attempts to develop a safe and effective vaccine for avian malaria should continue, as should attempts to control mosquitoes with low-toxicity methods like sterile insect release. Furthermore, control of feral pigs might reduce malaria transmission because their foraging behavior creates small pools that enhance mosquito breeding success.

Prevent the introduction of diseases and vectors in other locations. The role of careless introductions in Hawaii's extinction tragedy serves as a cautionary tale for islands everywhere. As climate change alters landscapes around the world, even more vigilance is needed to prevent the introduction of malaria and other diseases to locations that might previously have proved inhospitable for a tropical disease like malaria. Imports of live poultry and exotic birds pose severe risks, as do any cargo consignments likely to contain stagnant water, such as used tires.

Chapter 2. West Nile Virus

West Nile virus made its first appearance in the United States in the summer of 1999. Authorities in Queens, New York, began to notice that large numbers of American crows were dying mysteriously. That August, several elderly people in the same area developed symptoms of a neurological illness after spending time outdoors during the evening. St. Louis encephalitis was initially suspected in both the human and bird cases, but tests later revealed that the culprit was a closely related new arrival: West Nile virus, which over the next few years spread across the country, causing illness and death in humans, horses and an alarming array of bird species (Nash et al. 2001, Marra et al. 2004).

The West Nile Virus Life Cycle

All viruses consist of a strand of genetic material wrapped in a coating of protein. Because of their extremely simple structure, viruses cannot feed, reproduce or move on their own. Instead they bind to the surface of cells of another organism, by means of host-specific molecular binding sites on the protein coat. The genetic material inside is then absorbed into the host cell and incorporated into its own genetic makeup: in essence, the virus "hijacks" the cell and directs it to make more copies of the virus. Eventually the host cell dies and breaks open, releasing new copies of the virus. Depending on the severity of the infection and the type of cells affected, viruses' impact on their hosts can range from mild (the common cold) to life-threatening (HIV/AIDS, smallpox, ebola). Antibiotics are ineffective against viruses, though vaccines provide protection from many.

West Nile virus was first detected Uganda in 1937 (Smithburn et al 1940), though it has probably been present for hundreds of years or more. It is one of the most widely distributed of the flaviviruses, present across much of Africa, southern Europe and Asia (Hubalek & Halouzka 1999). The exact origin and pathway of the 1999 incursion remain unclear. Genetic analysis revealed that both the first infected people and a flamingo that died at the Bronx Zoo were infected with a strain of West Nile Virus matching one that was isolated from a dead goose in Israel – the only other country to record both human and avian fatalities from WNV-- the previous year (Lancioletti et al. 1999, Petersen & Marfin 2002). Manifestations of West Nile virus range from mild illness to severe and potentially fatal encephalitis (inflammation of the brain). Survivors of encephalitis are often left with permanent neurological injury.

Viruses are categorized according to the type and arrangement of genetic material, virus shape and characteristics of their protein coat. West Nile belongs to a class of viruses called Flaviviridae, a group of single-stranded RNA viruses that also includes Hepatitis C and several forms of encephalitis. More importantly, WNV is also an arbovirus, a contraction for *Ar*thropod-*bo*rne virus, This means it is transmitted by insects, particularly mosquitoes, and other arthropods like ticks, which feed on humans and other vertebrates. Over 60 species of mosquitoes can carry WNV, though the most important vectors appear to be members of the genus *Culex*, a large and widespread group of mosquitoes that bite both humans and birds (CDC 2008). Other arthropods, like ticks, fleas and biting midges, may also transmit the virus, but their role seems to be small compared to mosquitoes (Anderson et al. 2003, Sabio 2005).

If a mosquito bites a person or animal with a viral infection, the mosquito may take up the virus, which may or may not multiply inside the mosquito (Whitman 1937). Whether the mosquito becomes infected with WNV depends on the concentration of the virus in the initial host and the susceptibility of the mosquito. People and other mammals, for instance, appear to carry such low concentrations of the virus that mosquitoes feeding on infected individuals cannot further transmit it. They are thus considered to be "dead end" hosts for the virus. Many bird species, on the other hand, achieve high levels of viral concentration and can therefore be sources of infection.

Once a mosquito ingests blood with a sufficiently high viral load, it takes 1 to 14 days for the virus to move out of the gut of the mosquito and into the rest of its body, including the salivary glands (Kilpatrick et al. 2007). When a mosquito bites takes its next blood meal, it pierces the skin with a syringe-like proboscis and injects some saliva, which is thought to assist in locating a blood vessel on which to feed (Ribiero et al 1984). If the mosquito is infected with WNV, the viruses within the salivary glands enter the bloodstream along with the saliva that the mosquito injects. Birds can also contract the disease through close contact with infected birds or by consuming infected prey. Avian species differ in their reaction to West Nile infection; some display little to no illness, and others demonstrate high susceptibility to fatal disease. Lethargy, inability to stand, and disorientation are common external symptoms; internal symptoms range from enlargement of the heart and spleen to swelling and hemorrhage of the brain and nervous system tissues (Komar 2003).

West Nile Virus and Climate

Mosquito distribution and virus activity are closely tied to climate factors, particularly rainfall and temperature. Outbreaks of both West Nile virus, and the closely related St. Louis encephalitis, have been shown to correlate with periods of heat and drought that favor the development of the vector mosquitoes. For instance, the summer of 1999, when WNV was first detected in New York City, was characterized by July temperatures 9°F above the 30-year average (Epstein & Defilippo 2001).

All mosquitoes begin their life cycle in water. The adult female lays her eggs in a small pool of standing water, preferably one containing dead leaves or other organic material. The eggs hatch into larvae, which feed upon the organic matter and grow for a while before developing into pupae. The pupa is a resting phase during which the mosquito transforms into the adult phase, then emerging from the water. Mosquitoes therefore depend on the presence of water, and prefer still or stagnant water where the larvae won't be washed away. They also prefer small pools, which are likely to have fewer predators. In urban and suburban settings, *Culex* mosquitoes thrive in storm drains and catch basins. Drought conditions actually favor the mosquitoes, because the water remains stagnant and organic matter concentrates, providing a food source. Drought also concentrates birds at the same water sources, where they are more likely to be bitten (Epstein & Mills 2005).

External temperature strongly influences the development and activity of the mosquitoes that carry WNV. According to one study, *Culex* mosquitoes at 20°C took 25 days after hatching to develop to adults, but only 7.1 days at 35°C (Mottram et al. 1986). These researchers also found that below 15.6°C the mosquitoes didn't develop at all. *Culex* mosquitoes also feed less at lower temperatures (Eldridge 1968). High summer temperatures also favor the species *Culex pipiens*, the virus's primary vector, over a closely related mosquito, *C. restuans*, which is a less important vector (Kunkel et al 2006).

Temperature also plays an important role in overwintering survival. *Culex* mosquitoes overwinter as adults, mostly females that have taken a blood meal but not yet developed eggs (Bailey et al. 1982). West Nile virus has been detected in overwintering adult mosquitoes (Bugbee & Forte 2004).

It has long been known that raising the temperature also hastens flavivirus inside the mosquito, shortening the amount of time required for the mosquito transmit the virus after becoming infected (Davis 1932). The turnover time within the mosquito is called "vector competence." More recent

research specifically on WNF has shown that transmission rates clearly increase with temperature: at 18°C, it took 25 days for *Culex pipiens* mosquitoes to become infective after consuming an infected blood meal, and fewer than 30% of those were infective even after 28 days. On the other hand, mosquitoes reared at 30°C began showing infectivity at only four days, and >90% were infective after 12 days. Mosquitoes held at intermediate temperatures showed intermediate results (Dohm et al. 2002). Faster development of infectivity increases the likelihood that the virus will reach infectivity by the time the mosquito takes a subsequent blood meal. Another study found no detectable virus when infected mosquitoes were held at simulated overwintering temperatures of 10°C, but infectivity increased when temperatures were elevated to 26°C after simulated overwintering (Dohm & Turrell 2001), so rapid onset warm spring weather might also favor hastened virus development.

The Conservation Threat

West Nile Virus has been reported in 321 bird species (CDC 2007) and 29 mammal species (Marra et al. 2004), as well as captive alligators (Miller et al. 2003) and possibly other wild reptiles and amphibians (Marra et al. 2004). The strain that entered the U.S. in 1999 is more deadly to American crows than are closely related strains of the virus from North Africa and Australia (Brault et al. 2004). The virus causes high levels of mortality in many of these species, particularly crows and blue jays. By one estimate, up to 72% of Oklahoma crows were lost to West Nile virus in a single year (Caffrey et al. 2005). Other particularly susceptible species include the house finch, black-crowned night-heron, western scrub-jay, and yellow-billed magpie (Wheeler et al. 2009).

One recent study implicated WNV as an important factor in the declines of seven common bird species: American crow, blue jay, American robin, eastern bluebird, Carolina chickadee, black-capped chickadee and house wren (LaDeau et al. 2007). Since this set of species generally associate positively with urbanization and suburbanization, the authors postulate observed declines in these species over the time since the introduction of West Nile could be rightly attributed to the disease rather than to loss of natural habitat. For other declining species, it is much more difficult to separate WNV from other threats, such as habitat loss.

West Nile may upset ecosystem dynamics where it affects important predator species, like raptors. WNV antibodies have been detected in several species of hawks (Hull et al. 2006) and owls

(Fitzgerald et al. 2003), though its mortality rate and population impacts are unknown. Raptor reductions could lead to explosions in populations of their prey, particularly rodents, which are themselves important vectors for Lyme disease, leptospirosis, plague, hantaviruses and hemorrhagic fever (Epstein & Mills 2005).

Another risk is the potential impact on birds that aggregate in large groups or nest in colonies. Because the virus is shed in fecal material and orally as well as spread via mosquitoes, the likelihood of transmission increases when birds gather in large aggregations. West Nile virus reached American white pelican colonies in the northern Great Plains in 2002. With its arrival, mortality levels in older chicks jumped from less than four percent to 44%. The high mortality levels in older chicks, which would likely have otherwise survived to adulthood, has serious implications for the future health of the population (Sovada et al. 2008).

Even more alarming is the potential for West Nile to cause population declines in species that are already imperiled. The virus has already been demonstrated to be a serious threat to at least one such species. Greater Sage grouse have declined by a rate of about 2 percent per year from 1965 to 2003 (Connelly et al. 2004). Sage grouse decline can be attributed to many factors, as summarized by Braun (1998): habitat loss due to agriculture, mining and energy development, suburban and rural development, roads, and reservoirs; habitat fragmentation by fences, power lines, and sagebrush control treatments; habitat degradation from grazing, sagebrush control via mechanical and chemical means, and altered fire regimes; and natural factors including drought, predation, and hunting. The sagebrush habitat the grouse rely upon has also been degraded by invasive cheat grass, which promotes ''larger, earlier and more frequent fires'' due to its dense, continuous ground cover and summertime drought dormancy, as well as increased erosion potential following fires (West 1999). Cheatgrass invasion shortened the fire return interval in the intermountain west from 20–100 years to as little as 5 years, reducing or eliminating fire-sensitive sagebrush from large parts of its range (Pyke 1999).

In 2003, West Nile virus emerged as an additive threat to this already in-trouble species. Sage grouse mortality from WNV was 1st observed on 24 July 2003, prompting monitoring of radio-tagged sage grouse in 4 populations in Alberta, Montana, and Wyoming (Naugle and others 2004). Naugle

and colleagues found that 18 of the 22 carcasses th were positive for WNV. They also found that survival in WNV areas was 26% lower than in their WNV-free control site and was 25% lower than the average survival for those areas from 1998 to 2002, prior to WNV emergence. Equally troubling, they found ‘‘no live sage-grouse seropositive for neutralizing antibodies against WNV;’’ in other words, ‘‘no evidence that sage-grouse are able to survive WNV infection and develop immunity’’. The authors conclude: ‘‘The spread of WNV represents a significant new stressor on sage-grouse and probably other at-risk species’’ (Naugle and others 2004). The extreme susceptibility of the sage grouse to the virus has also been demonstrated in the laboratory: in one experiment all six birds challenged with the virus died, and so did four out of five birds that had been vaccinated prior to exposure (Clark et al. 2006).

The dramatic declines wrought by WNV are among the issues that have forced the U.S. Fish and Wildlife Service (FWS) to take a new look at whether the sage grouse requires the protection of the Endangered Species Act (ESA). FWS had made a determination in January 2005 that protection of the sage grouse under the ESA was not warranted (70 Federal Register 2244-2282). That decision was challenged and a court order remanded the finding back to FWS, which is currently reviewing the bird’s status in light of this recently emerged threat.

West Nile also has the potential to be very detrimental to several species that are already protected under the Endangered Species Act. Florida Scrub-jays have declined precipitously in central Florida following WNV introduction (Fujisaki et al. 2008). California condors, which are critically endangered and have been the subject of an intensive captive breeding and reintroduction campaign, were potentially saved from extinction by a prospective immunization program of the entire population prior to the arrival of WNV in the western U.S. (Gwong-Jen et al 2007). The potential impact of the disease on other imperiled species, such as whooping crane and Kirtland’s warbler, is still uncertain.

West Nile Virus in a Warming World

As described above, increased temperature facilitates the development and spread of both the West Nile flavivirus and its most important mosquito vector. Drought conditions are also favorable for the spread of the vector. Indirect evidence of the importance of heat and drought on West Nile pathology came in 2004: an unseasonably cool summer that year reduced mosquito activity and

lowered mortalities of humans, horses and sage grouse (Naugle et al. 2005). For the latter, the effect was particularly important at the northern extent of WNV range in Alberta.

Unfortunately, under most climate change scenarios, future patterns are not expected to resemble the cool summer of 2004. IPCC projects that warmer days and nights, warmer and more frequent hot spells, and increased areas of drought are all "very likely" or "likely" to occur over the coming century as a result of anthropogenic climate forcing (IPCC 2007). Thus, WNV is likely to continue to threaten wildlife and human health in coming years, and will likely continue to spread to higher latitudes and altitudes, with possibly dire consequences for the naïve fauna of those regions.

Warming temperatures also appear to have helped facilitate the evolution of a new strain of West Nile Virus. This strain, referred to as WN02, was first detected in 2001. WN02 contains three genetic differences from NY99, the strain originally detected in New York in 1999. Natural variation and emergence of new strains is common in viruses and WN02 appears to have evolved naturally from NY99. What is interesting, however, is that the WN02 strain appears to have almost completely displaced NY99, indicating that it is somehow "better" at being passed between mosquitoes and vertebrate hosts (Snapinn et al. 2007; Davis et al. 2005). WN02 has a shorter incubation time within the mosquito WN99; with more effective transmission at 5 to7 days after the mosquito first takes an infected blood meal (Ebel et al. 2004, Moudy et al. 2007). The shorter incubation time gives the WN02 strain it a big advantage over NY99 in the "vector competence" race. The main factor tilting the balance in favor of the WN02 strain is temperature: WN02 is more efficient at replicating within mosquitoes at higher temperatures and responds more strongly to warm temperature than NY99 (Kilpatrick et al. 2008). The differential response to temperature gave the new strain an advantage in transmission and allowed it to outcompete its predecessor.

Helping Wildlife Adapt to West Nile Virus and Climate Change

Continue monitoring and adaptive management for potentially sensitive wildlife species. Continued monitoring of avian mortality has value for detecting potential population-level disease impacts, particularly colonial birds and those species that are already rare or suffering population declines for other reasons. Continued monitoring of mortality in crows and jays also provides vital sentinel data that can help predict human outbreaks and possibly reduce their impacts (McLean 2006) (for instance by warning sensitive groups to take measures to avoid mosquito bites). Monitoring has already proven

to be an important tool in the conservation of sage grouse in the face of WNV (Walker et al 2004). Monitoring programs can also inform mosquito control practices that have fewer negative ecological side effects, such as targeted larviciding rather than broad-scale adult control (Epstein & Miller 2005).

Curtail practices that increase mosquito activity. One way to limit exposure to West Nile is to avoid creating new habitat for the mosquito vectors of the disease. One example of such a practice is coal bed methane extraction in the Powder River Basin of northern Wyoming, in the heart of sage grouse habitat. Coal bed methane infrastructure degrades sage brush habitat, and drilling activity disrupts sage grouse lekking within a radius of up to 3.2 kilometers, resulting in reduced activity by males and abandonment of leks (Walker et al. 2007). In addition, extraction of coal bed methane requires significant discharges of water, which is impounded in small ponds at the surface, creating ideal habitat for larvae of the mosquito that spreads WNV. Coal bed methane discharge ponds resulted in a 75% increase of potential habitat for mosquito larvae in the Powder River Basin from 1999-2004 (Zou et al. 2006).

Enact preventive measures to halt spread of the virus to new areas. West Nile virus has the potential to devastate the unique endemic faunas in places like Hawaii and the Galapagos. Risk assessment models suggest that transport of mosquitoes on airplanes is the most likely pathway by which WNV might reach both archipelagos (Kilpatrick et al. 2004, Kilpatrick et al. 2006). This risk would be reduced by disinfection of airplane passenger cabins and cargo holds, as well as managing air and seaport environs to eliminate larval habitat (Kilpatrick et al. 2004, Kilpatrick et al. 2006). Additional quarantine precautions with respect to live bird imports to the islands would also reduce the risk from that pathway.

The following table summarizes the spread of WNV in the U.S.

Year	Human Cases Recorded	Bird Deaths Recorded	Source
1999	59 E/M, 3 F, 7 deaths (NY)	NY, NJ, CT, MD	
2000	19 E/M, 2 F, 2 deaths (NY, NJ, CT)	+VT, NH, MA, PA, DE, DC, VA, NC	
2001	64 E/M, 2 F, 9 deaths (+AL, FL, GA, LA, MD, MA, PA	Alabama, Arkansas, Connecticut, Delaware, District of Columbia, Florida, Georgia, Iowa, Indiana,	

		Illinois, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Mississippi, Missouri, New Hampshire, New Jersey, New York, North Carolina, Ohio, Pennsylvania, Rhode Island, Tennessee, Virginia, and Wisconsin.	
2002	2946 E/M, 1160 F, 50 unsp, 284 deaths (39 states & DC)	All states except OR, NV, UT, AZ, AK, HI, PR	
2003	2866 E/M, 6830 F, 166 unsp, 264 deaths (45 states + DC; not in WA, OR, ME, AK, HI	All states except WA, OR, AK, HI, PR	
2004	1142 E/M, 1269 F, 128 unsp, 100 Fatalities Alabama, Arizona, Arkansas, California, Colorado, Connecticut, District of Columbia, Florida, Georgia, Idaho, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maryland, Michigan, Minnesota, Mississippi, Missouri, Montana, Nebraska, Nevada, New Jersey, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, South Carolina, South Dakota, Tennessee, Texas, Utah, Virginia, Wisconsin, and Wyoming	Alabama, Arizona, Arkansas, California, Colorado, Connecticut, Delaware, District of Columbia, Florida, Georgia, Idaho, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, Montana, Nebraska, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, South Dakota, Tennessee, Texas, Utah, Vermont, Virginia, West Virginia, Wisconsin, Wyoming, and Puerto Rico.	
2005	1294 E/M, 1607 F, 99 unsp 119 Fatal Alabama, Arizona, Arkansas, California, Colorado, Connecticut, Delaware, District of Columbia, Florida, Georgia, Idaho, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, Montana, Nebraska, Nevada, New Jersey, New Mexico, New York, North Carolina, North	All but Alaska, PR, HI	

	Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, South Dakota, Tennessee, Texas, Utah, Virginia, Wisconsin, and Wyoming		
2006	1459 E/M, 2616 F, 194 unsp (177 fatal) All contig. States except ME, VT, NH, RI, DE	All contiguous states	CDC 2007
2007	1217E/M, 2350 F, 63 unsp (124 fatal) Alabama, Arizona, Arkansas, California, Colorado, Connecticut, Delaware, Florida, Georgia, Idaho, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, Montana, Nebraska, Nevada, New Jersey, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, South Dakota, Tennessee, Texas, Utah, Virginia, Wisconsin, and Wyoming	2182 All but ME, AK, HI	
2008			

http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount99_detailed.htm
http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount00_detailed.htm
http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount01_detailed.htm
http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount02_detailed.htm
http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount03_detailed.htm
http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount04_detailed.htm
http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount05_detailed.htm
http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount06_detailed.htm
http://www.cdc.gov/ncidod/dvbid/westnile/surv&controlCaseCount07_detailed.htm

Chapter 3. Chytridiomycosis

Amphibians—frogs, toads, salamanders—are in alarming decline throughout the world. The 2004 Global Amphibian Assessment classified 1,896 species of amphibians as vulnerable, endangered or critically endangered. This figure represents nearly one-third of all amphibians, a higher rate of endangerment than in birds (12%) or mammals (23%) (GAA 2004). Thirty-five species have been driven to extinction in recent years and a further 130 may be extinct. Many factors drive amphibian decline, with habitat loss and pollution long established as important threats to many species. Over the past decade, however, diseases—particularly the fungus chytridiomycosis—have emerged as another important driver. Even worse, Chytridiomycosis may be interacting with climate change and other stresses to drive species toward extinction.

Disease Life Cycle

Chytridiomycosis is caused by a fungus, the taxonomic group that includes molds, yeasts and mushrooms. Unlike mushrooms and molds, however, these fungi do not form large visible fruiting structures. Chytrids are a fairly distinct group among the fungi, as they are mainly found in aquatic habitats or in soils and each spore has a whip-like flagella for propulsion, to aid dispersal through the water. Once this zoospore lands on a suitable host, it forms a small cyst and then begins to divide and send out tiny root-like structures into the epidermal cells of its host. These rhizoids infiltrate the animal's skin cells and feed on keratin, an important structural protein within the skin. Eventually, the fungus creates new spores, which themselves go on to infect additional cells or new individuals (Longcore et al. 1999; Berger et al. 2005).

Chytrid-infected amphibians show skin lesions with cellular damage evident at the microscopic level. The damage is somewhat analogous to human cases of athlete's foot, caused by another keratin-ingesting fungus; however, the effects are much more dangerous for frogs, because their skin helps regulate the body's oxygen and water content (Berger et al. 1998). It remains uncertain whether amphibians die directly from the fungal damage, from toxins emitted by the fungus, or from secondary bacterial infection (Berger et al. 2005). Tadpoles usually survive infection, because their skin lacks keratin; therefore the fungus only attacks their keratin-containing mouthparts; widespread and often fatal infection follows metamporhosis, when a larger part of the skin becomes keratinized (Berger et al. 1998). The chytrid group contains many species; however the deadly amphibian

parasite *Batrachochytrium dendrobatidis* (often abbreviated as *Bd.*) is the only species within the group to attack vertebrates (Berger et al. 1998).

The Conservation Threat

Origin and Means of Spread

A study of museum specimens found that the chytrid fungus was present on clawed frogs (*Xenopus laevis)* from southern Africa dating to 1938, 23 years earlier than the disease was found anywhere else (Weldon et al. 2004). The disease also had little impact on that species, supporting the theory that the fungus originated somewhere in Africa. A number of other amphibians from different parts of the continent have shown chytrid fungus infection without high levels of mortality, so more research is needed to pinpoint the exact origin and original host within Africa (Weldon et al. 2004).

In 1934, scientists discovered that the African clawed frog can be induced to ovulate when injected with the urine of a pregnant woman. This knowledge was used to develop a protocol for a rapid pregnancy test (Shapiro & Zwarenstein 1934). Subsequently large numbers of African clawed frogs were exported around the world (Weldon et al. 2004). Specimens were also used in embryological research and molecular biology, and feral populations that could potentially serve as infection reserviors became established in the United States, Britain and Chile (Weldon et al. 2004). The American bullfrog *(Rana catesbiana*) may have served as an additional vector for the spread of the chytrid fungus; like the African clawed frog, it is traded widely, has established feral populations in many areas, and can carry the fungus without suffering adverse effects (Weldon et al. 2004). For instance, researchers in the Venezuelan Andes have found populations of introduced bullfrogs carrying the disease but suffering only small skin lesions and low mortality (Hanselmann et al. 2004).

North America

The earliest incidence of chytrid fungus in North America dates to green frogs in Quebec in 1961 (Ouellet et al. 2005). Sampling at National Wildlife Refuges in New York, Vermont, New Hampshire, Massachusetts and Maine detected chytrid fungus in American toads (*Bufo americanus*), bullfrogs (*Rana catesbiana*), green frogs (*Rana clamitans*), pickerel frogs (*Rana palustris*), northern leopard frogs (*Rana pipiens*), mink frogs (*Rana septentrionalis*) and wood frogs (*Rana sylvatica*). The fungus was not detected in the gray tree frog (*Hyla versicolor*) and spring peeper (*Pseudacris crucifer*),

possibly because both of these spend more time in trees and less in ponds and streams than the other species tested (Longcore et al. 2007).

The fungus also infects populations of the mountain yellow-legged frog (*Rana mucosa*) in California. Interestingly, the patterns of population decline in this species varies, with population extinction occuring within a few years in southern Sierra Nevada sites, but populations persisting despite infection at sites in the northern part of the range (Briggs et al. 2005, Fellers et al. 2007). The potential role of habitat and differences in temperature profile between the two areas is currently under investigation. Fungal infection has been associated with die-offs of in boreal toads, Wyoming toads and Yosemite toads (Green & Sherman 2001, Green et al. 2002).

Chytrid fungus has also been detected in the Jemez Mountains of New Mexico in an exclusively terrestrial salamander species, suggesting that the disease could potentially threaten a wider array of species than previously thought (Cummer et al. 2005). The fungus was detected in three species of Arizona frogs, one a threatened species and the other a candidate for listing. Given that all of Arizona's native frogs are experiencing population declines, the emergence of chytrid fungus there poses a serious additional concern for the southwest's amphibian fauna (Bradley et al. 2002). On the other hand chytridiomycosis detected in tiger salamanders in Arizona did not lead to mortalities, indicating that susceptibility might vary among species (Davidson et al. 2003).

Australia & New Zealand

Chytridiomycosis was identified in Australia after scientists discovered large numbers of dead and dying frogs in the Big Tableland area of Queensland in 1993 and 1994; these researchers subsequently found dead and dying frogs in other locations and from captive collections as well, and were able to determing that the cause of death was the chytrid fungus (Berger et al. 1998). The pathogen was probably introduced to Australia in the mid to late 1970s and had caused dramatic declines in mountain streamdwelling frog species (DEH 2006). The sharp-snouted day frog (*Taudactylus acutirostris*) was most likely driven to extinction by chytridiomycosis (Schloegel et al. 2006). The frog was previously abundant in the upland tropical areas of Queensland but populations crashed starting in 1990; by 1994 the species has essentially vanished from the wild, and the last captive specimen died in 1995 (DEH 2006).

The disease also most likely claimed the three other Australian frog species, the northern and southern gastric brooding frogs (*Rheobatrachus vitellinus* and *R. silus*) and the southern day frog (*T. diurnus*). All three went extinct before the disease was postively identified, but their patterns of decline and disapperance fit the epidemiology of chytridiomycosis (DEH 2006). Several species suffered local extinctions of their upland populations, while lowland populations have managed to persist. Overall, the disease has been detected in 49 species of amphibians in Australia, including 14 of the 27 listed as threatened. Further species are expected to be listed due to the disease (DEH 2006).

In New Zealand, where the native amphibian fauna is already greatly reduced due to introduced predators and habitat modification, the chytrid fungus was found in 1999 (Waldman et al 2001).

Central & South America

Amphibian populations in the Monteverde region of Costa Rica underwent a severe crash in 1987; this event led to the high-profile presumed extinction of two species, the golden toad and the harlequin frog (Pounds & Crump 1994), as well as declines in multiple other species (Pounds et al. 1997). Researchers postulated that temperature or moisture stress, or a pathogen, might be interacting with climate disturbance from a particularly strong El Nino event, to cause the alarming declines (Pounds & Crump 1994, Pounds et al. 1997, Ron et al. 2003). Later examination of specimens collected in 1986 from Braulio Carrillo National Park in Costa Rica confirmed the presence of chytrid fungus (Puschendorf et al. 2006a). A large-scale mortality of several species of stream-dwelling frogs observed in Las Tablas, Costa Rica and Fortuna, Panama in 1993 and 1994; these frogs exhibited the diagnostic features of infection by the keratin-attacking chytrid fungus (Lips 1998, 1999). Similar declines and possible extinctions have occurred in Honduras (Puschendorf et al. 2006b), and the disease has also been detected in Guatemala, where several species have experienced “catastrophic” declines (Mendelson et al. 2004). More recently the disease has been detected in El Salvador in two frog species, one of which is critically endangered (Felger et al. 2007).

Chytrid infection has struck frogs along the spine of the Andes, and may be a factor in the extinction of several species in genus *Telmatobius*, a group whose various species live in isolated high mountain streams in the Andes. Many *Telmatobius* were rare or endangered prior to the arrival of

chytrid fungus (Barrionuevo & Mangione 2006). In 1989, the disease was detected in *T. niger*, a species that was once widespread in Ecuador. That species has not been seen since 1994 and is now presumed extinct (Merino-Viteri et al. 2005). *T. marmoratus*, which lives in the high mountains of Peru, is also in decline, with chytrid fungus as one potential cause (Seimon et al. 2005). In 2006, Bd was reported from two species of endangered Argentinean frogs (*Telmatobius atacamensis* and *T. pisanoi*) (Barrionuevo & Mangione 2006).

Chytrid fungus has probably played a role in the disappearance of several species of the brightly colored *Atelopus* toads (also called harlequin frogs) in the Venezuelan lowlands (Bonaccorso et al. 2003) and Andes (Lampo et al. 2006) and Ecuador, where the apparent extinction of the Jambayo toad (*Atelopus ignescens*) followed a warm, dry year in 1987 (Ron et al. 2003). A recent comprehensive survey of this genus found that this group has declined more severely than any other group of amphibians: 42 out of 113 putative species have declined by half and 30 species have not been seen since 1996 and are feared to be extinct (La Marca et al. 2005). These researchers also did a breakdown by country: 100% of Costa Rica's harlequin frogs were declining, 50% in Panama, 27% in Colombia, 58% in Ecuador, 11% in Peru 90% in Venezuela and 25% in French Guiana. Chytrid fungus has been documented in nine declining species and is suspected in others, particularly since many species are declining in protected areas where habitat loss is not thought to be a factor (La Marca et al. 2005).

Chytridiomycosis has been detected across a broad range of locations and altitudes in the Brazilian Atlantic rain forest (Carnaval et al 2006). While the disease has not yet been definitely linked to declines and extinctions, at least 20 species of Brazilian frogs and toads have undergone alarming population declines in recent years, and several of the species of concern are montane stream-dwelling species that have elsewhere been particularly vulnerable to chytrid fungus (Eterovick et al. 2005).

Caribbean

Chytrid fungus was first reported from the Caribbean in 2004 (Burrowes et al. 2004). At least three species of high-elevation dwelling frogs are presumed to have gone extinct in Puerto Rico over the past thirty years. Higher-elevation populations of six other endemics are in decline, and the fungus may be a factor in these declines and extinctions (Burrowes et al. 2004). More recently, the disease

was found in a dead toad in Cuba, and may prove to be an emerging conservation challenge there (Diaz et al. 2007).

Europe

Chytridiomycosis was first detected in Europe in 1998, following the importation of infected frogs from Costa Rica and French Guayana (Mutschmann et al. 2000). By 1999 the disease caused an 86% crash in populations of Spain's common midwife toad; similar to patterns elsewhere, the declines took place in protected areas that had undergone very little change in habitat (Bosch et al. 2001). In 2004, the fungus was detected in a wild population of introduced bullfrogs in a pond in southeastern Britain (Cunningham et al. 2005). The extent of the disease's spread and the impacts on native British amphibian fauna are not yet known. Amphibians also tested positive for the disease in Portugal, Switzerland (Garner et al. 2005) and Italy (Simoncelli et al. 2005).

The Climate Connection

Climate disturbance, particularly extremely dry El Niño events, was proposed early on as a factor in the disappearance of the golden toad and the harlequin frog from their cloud forest habitats in Costa Rica (Pounds & Crump 1994). At first glance, however, it would appear that global warming would hinder the chytrid fungus rather than act synergistically with it. Optimal temperatures for the growth of the fungus occur between 6°C and 28°C (Bradley et al. 2002). The fungus is more pathogenic at moderate temperatures: for instance 100% of experimentally infected frogs died when held at 17°C and 23°C, but only 50% of frogs died at 27°C (Berger et al. 2004). In Australia, chytridiomycosis is more likely to be present at sites where the summer temperature is below 30°C (Drew et al. 2006) and the disease is more prevalent in early spring than in late summer and fall (Kriger & Hero 2006). Infected frogs held at 37°C for fifteen hours are "cured" of the disease (Woodhams et al. 2003)

High temperatures actually inhibit the development of the fungus and increase the rates at which amphibians slough off their skin cells; thus high temperatures prevent the fungus from completing its life cycle before the infected cells are shed (Berger et al. 2005). One might expect, therefore, that warming temperatures might slow or halt the disease. However, as with many issues involving climate change, the story of how the fungus and host interact in the real world is more complicated than simply an effect of temperature.

One set of researchers believes that they have found the answer to this apparent paradox. J.Alan Pounds and colleagues have noted that in the middle elevations of the mountain regions of Central and South America, where disease-related dieoffs have been particularly severe, one of the main manifestations of climate change is an increase in cloud cover. This increase is moderating the temperatures at the middle elevations: raising the night time temperatures and lowering the daytime temperatures. At low elevations, daytime highs are still high enough to kill off the fungus, especially in sunny spots on the forest floor. At the highest elevations, night time temperatures are still low enough to inhibit the fungus. But at the middle elevations, cloud cover has moderated both the daytime and nighttime temperatures, with both periods thereby "shifting towards the growth optimum for these pathogens" (Pounds et al. 2006).

Climate conditions in Spain may also be changing in ways that favor the fungus. For instance, the number of days that fall within the most favorable conditions (21-27^{o}C) have increased, while the number of days hot enough to to inhibit it has not (Bosch et al 2007). Changes in humidity, night time temperatures and shorter and milder winters might also be favoring the fungus (Bosch et al. 2007).

It has also been observed that sharp amphibian declines in Ecuador (Ron et al. 2003) and Puerto Rico (Burrowes et al. 2004) were preceded by droughts. The apparent interaction between drought and amphibian die-offs led scientists to speculate about another way that the disease is interacting with a changing climate: chytrid fungus may interfere with the animals' ability to withstand drought. As mentioned above, one of the areas that often shows damage to the skin from the fungus is the pelvic patch, a small but critically important area of skin that helps the animal control absorption of water (Berger et al. 1998). Some researchers have suggested that frogs can survive this damage during wet periods. During drought when the animals need a functioning pelvic patch in order to absorb water from dew (Pough 1983), and under these conditions the fungal damage may prove fatal because it interferes with this vital function (Burrowes et al. 2004). Increased incidence of drought is one "likely" forecast outcome of anthropogenic climate change (IPCC 2007). If this mechanism of action proves true, chytrid fungus may cause future declines in species and areas that have not yet experienced high levels of mortality from the fungus.

Researchers in Australia found that multiple-year warm trends were more likely than single-year warm periods to presage amphibian declines, and suggest that warming might stress amphibians in ways that make them more susceptible to infection (Alford et al. 2007).

Helping Wildlife Adapt to Chytridiomycosis and Climate Change

Focus monitoring and conservation efforts on vulnerable species and habitats. The chytrid fungus has caused amphibian populations in far-flung regions of the world to plummet towards extinction. To prevent further species from suffering the same fate, it will be critical to monitor amphibian population health, with particularly focus on montaine streamside habitats that are most at risk (Kriger & Hero 2007). Intensive interventions such as capture, fungicide treatments and captive breeding may be necessary to prevent further extinctions (Young et al. 2007).

Clean fishing, recreation and research practices. While the chytrid fungus is adapted to aquatic dispersal, the spore is also capable of surviving for several hours in a dry environment and up to three months in a moist environment like mud (Johnson & Speare 2005). The fungus can thus potentially be spread via recreational equipment (boats, boots, fishing gear) and research equipent (Corn 2007). "Clean" campaigns directed at other aquatic nuisance species like zebra mussels and hydrilla have raised awareness of the need to clean and dry recreational equipment before transport to new locations (see, for instance www.protectyourwaters.net). These same protocols might also help slow the spread of aquatic-borne diseaseses like chytrid fungus (Corn 2007).

Reduce the threat from release or transport of infected amphibians. Chytrid fungus was able to spread around the world from its area of origin in southern Africa because of wide-scale trade, transport and release of one of its hosts, the African clawed frog. Release of infected captive animals continues. Fungal spores can also find their way into new habitats via release of water that housed infected captive frogs. International trade in live amphibian imports has reached huge proportions: 28 million individuals passed through the three largest U.S. ports of entry from 2000-2005; and 62% of imported bullfrogs were carrying *Batrachochytrium dendrobatidis* infection (Schoegel et al. 2009). Halting the spread of the disease will require risk abatement planning to reduce the risk from amphibians in trade and captivity (Fisher & Garner 2007).

Reduce other threats, particularly those may act synergistically with chytrid fungus. Disease and climate climate are far from the only threats that amphibians face world wide. Habitat loss, exploitation, introduced species, other pathogens, ultraviolet-B radiation and a range of pollutants and toxins also take their toll on amphibians (Collins & Storfer 2003, Bancroft et al. 2008). All these threats must be abated if amphibians are to survive the climate change and disease bottleneck.

Chapter 4. Harmful Algal Blooms

Harmful algal blooms and red tides are emerging as widening conservation threats. These phenomena diverge somewhat from the pattern of other diseases discussed here-- they are not communicable diseases, like malaria or west Nile virus. Nevertheless, their pervasive and dangerous consequences threaten oceans, estuaries, and vulnerable species in ways that are roughly comparable to other diseases discussed here. Harmful algal blooms share other features with diseases: they are caused by tiny organisms proliferating out of balance, they are deadly to a wide array of species of fish and other creatures, and their effects are exacerbated by human activities on the landscape, most notably pollution, but increasingly climate change as well.

Organisms and their Life Cycles

Harmful algal blooms inflict damage on aquatic and marine ecosystems in two major ways: 1) they directly harm other species through the production of toxins, or simply through their physical structure, particularly spines; and 2) their proliferation and decomposition results in dangerous levels of oxygen depletion in the surrounding waters (Gilbert et al. 2005). Two very distinct types of organisms, cyanobacteria and dinoflagellates, are responsible for most harmful algal blooms. A third group, the diatoms, also has some members with toxic properties.

Cyanobacteria

Sometimes referred to as blue-green algae, cyanobacteria are actually a type of bacteria, one of the most simplest types of living thing, with a very simple cell structure. Unlike true algae, which are more closely related to plants, cyanobacteria cells lack a nucleus, chloroplasts, mitochondria and other organelles. Cyanobacteria are widespread and important, and are found in more types of environments than any other living thing: various species live on bare rock, soil and volcanic ash; from freshwater to marine to extremely salty environments; and from arctic streams to hot springs where no other organisms can live (Chorus & Bartram 1999).

In addition to their ubiquity, Cyanobacteria play important ecological roles. They perform photosynthesis by way of chlorophyll and a bluish pigment called phycocyanin, from which they get their name. In fact, photosynthesis is thought to have evolved in an ancestor of this group (Knoll 2008). Cyanobacteria are also responsible for nitrogen fixation, the conversion of nitrogen from its unusable form in the atmosphere into a compound that plants can use to build proteins and other

structures (Knoll 2008).

A number of blue-green algae produce compounds called cyanotoxins. These pose health threats to humans and wildlife that are exposed via ingestion or skin contact. Three overarching categories of cyanotoxins are recognized: hepatotoxins (microcystins and nodularin), which cause damage to the liver; neurotoxins (anatoxins, saxitoxins and others), which damage the nervous system; and irritants called lipopolysaccharides (Chorus & Bartram 1999). Many species can produce more than one toxin, and blooms often consist of multiple species. Common harmful cyanobacters listed by the Centers for Disease Control include: M*icrocystis aeruginosa*, *Anabaena circinalis*, *Anabaena flos-aquae*, *Aphanizomenon flos-aquae*, *Cylindrospermopsis raciborskii.* There are numerous incidences of people becoming ill with gastrointestinal symptoms, rashes, ear infections and respiratory infections after swimming or canoeing in lakes with algal blooms, and on at least one occasion sheep died after drinking water tainted with neurotoxic cyanobacters (Chorus & Bartram 1999).

Dinoflagellates

Usually referred to as red algae or brown algae, dinoflagellates are also single-celled organisms. However, they are protists rather than bacteria: they have much more complicated cell structures, and so are more similar to plants and animals. These single-celled organisms have features associated with both plants and animals. Like plants, they possess cell walls and convert the sun's energy into chemical energy via photosynthesis. But unlike plants, they can also move using a pair of whip-like flagellae. Some, like *Karlodinium,* produce toxic substances to deter predators (Adolf et al. 2006). Other species are themselves predatory, and may use toxins to "stun" their prey. Through direct exposure, or via bioaccumulation by unaffected predators, these compounds can damage the nervous systems and livers of shellfish, fish and humans. Some of the more commonly occurring toxins that affect people include brevetoxins, saxitoxins, ciguatoxins, okadaic acid and domoic acid.

Brevetoxins are produced by *Karenia brevis*, one of the most common causes of red tides in Florida. Shellfish feed on the dinoflagellates and accumulate the toxin, which does not harm them but can be concentrated to levels that cause neurotoxic shellfish poisoning in anyone who eats affected oysters, clams or other shellfish (Baden 1983). Brevetoxins disrupt the function of electrical impulses between nerve cells by "activating" sodium channels and altering the flow of sodium and calcium in and out of cells. Symptoms in humans include gastrointestinal problems, "pins and needles" tingling

sensations, and sensations of vertigo, weakness, and lack of coordination (Morris et al. 1991). *K. brevis* cells also have fairly weak cell walls, so the cells can break apart at the surface of the water; the toxins can become airborne, causing eye irritation and coughing.

Saxitoxins operate in the opposite manner of brevetoxins; instead of "activating" sodium channels, they are sodium channel blockers. Blocking of sodium movements in and out of cells halts nerve transmissions, causing a condition called paralytic shellfish poisoning. While some saxitoxin poisoning symptoms are similar to brevetoxin symptoms, paralytic shellfish poisoning is considerably more dangerous, since the respiratory functions can be paralyzed leading to death by asphyxiation (Ahmed 1991). Saxitoxins are produced by *Alexandrium fundyense*, which forms algal blooms in the Gulf of Maine. More recently the toxin was discovered in *Pyrodinium bahamense* dinoflagellates from the Indian River Lagoon in Florida, after pufferfish from this water body were implicated in saxitoxin poisoning in humans (Landsberg et al. 2006).

Diarrhetic Shellfish poisoning is caused by a compound called okadaic acid produced by the dinoflagellates *Dinophysis acuta* and *D. acuminata*. These dinoflagellates are more common in European waters but have been detected in the Atlantic near Canada. Okadaic acid exposure causes an acute but rarely serious gastrointestinal illness; of more concern is its apparent role as a promoter of tumor growth (NOAA undated).

Yet another of the harmful dinoflagellates is *Pfisteria piscicida*, which emerged in the 1990s as a potential cause of fish lesions, kills and human illness in estuaries along the eastern seaboard (Burkholder et al. 1992, Steidinger et al. 1996). Watermen working near *Pfiesteria* fish-kills demonstrated decreases in cognitive and visual function; these symptoms were triggered by airborne exposure rather than ingestion of fish, and improved after detoxification therapy (Hudnell 2005). The exact nature of the toxin involved remained elusive for several years, and turned out to be different from any of the others described above. It was recently identified as a complex, rapidly dissipating organic compound containing copper, which induces free-radical damage in cells (Moeller et al. 2007).

Diatoms

Diatoms are a separate group of single-celled algae, characterized by "pillbox" shells of silicon

dioxide. Like cyanobacteria and dinoflagellates, they are a widespread group containing multiple species adapted to many environments. Unfortunately the similarities do not end there: this group also contains toxin-producing members. In particular, diatoms of genus *Pseudo-nitzschia* produce a neurotoxin called domoic acid, which accumulates shellfish and fish. Ingestion of tainted seafood causes amnesic shellfish poisoning, so called because it can cause permanent memory loss. A 1987 outbreak associated with mussels caused three deaths in Prince Edward Island, Canada, and outbreaks on the West Coast closed Washington State's razor clam fishery for a year at a time in 1991, 1998 and 2002 (WDFW 2003).

The Conservation Threat

Harmful algal blooms threaten aquatic and marine ecosystems and wildlife through the creation of hypoxic conditions, and also through the toxins they produce. Hypoxia refers to the severe reduction in the amount of oxygen dissolved in the water associated with blooms of any type of algae. Algal blooms produce oxygen when they are alive and photosynthesizing, but most of this oxygen stays in the warmer, well-lit surface waters. When the algae die, they sink to the bottom, where the water is colder, darker, and in coastal areas, saltier. In summertime, these two layers of water do not mix except in unusual circumstances, like a strong storm system. As the algae decompose, the oxygen in this bottom layer gets used up by the bacteria breaking down the algae. Since there is very little photosynthesis going on in the darker bottom layer, and very little mixing between the upper and lower layers, oxygen levels quickly plunge to levels that cannot sustain fish, crabs and other organisms. Mobile animals like fish tend to leave the area, while more sessile creatures are killed outright, hence the term "dead zone." Dead zones form in Lake Erie the Chesapeake Bay, Gulf of Mexico, and elsewhere.

While the most direct impacts of hypoxia may be confined to the benthic zone of the affected waters, the full effects of harmful algal blooms are felt much more broadly, even among mobile animals that might escape the direct effects of oxygen reduction in the dead zone. For instance, a 2007 bloom of the dinoflagellate *Karlodinium* in a Chesapeake Bay tributary killed over 300,000 fish, probably through a combination of toxins and oxygen depletion (Blankenship 2007). *Karlodinium* is particularly insidious because its toxins attack the gills, rendering fish less able to survive oxygen depletion. *Karlodinium* may even be the ultimate cause of fish kills attributed to *Pfiesteria*, with the latter acting in concert or feeding opportunistically on dead and dying fish (Greer 2007).

For birds and marine mammals that are less vulnerable to aquatic hypoxia, toxin exposure is the bigger threat. Cyanobacterial toxins, particularly microcystins and anatoxin-a, have been implicated in the deaths of over 50,000 lesser flamingos in Kenya (Kotut et al. 2006). Mortalities of great blue herons in Maryland (Driscoll et al. 2002) and alligators and white pelicans in Florida (Burns et al. 2002) have also been linked to cyanotoxins. However, the majority of toxin outbreaks impacting wildlife can be attributed to dinoflagellates or diatoms. These are of particular concern for marine mammals, a group that includes a number of rare and threatened species. The compounds involved are for the most part the same as those which cause human illness, as described above.

Brevetoxin accumulates on sea grasses and in fish, reaching concentrations sufficient to harm and kill the marine mammals at the top of the food chain (Flewelling et al. 2005). The toxin binds to the brains of manatees, gray whales, humpback whales, and sea lions in much the same way as in human brains, so the toxic impacts follow much the same pathway (Trainer & Baden 1999). Bottlenose dolphins have experienced mass mortalities due to brevetoxin: the Florida panhandle saw 152 deaths in 1999-2000 (Mase et al. 2000) and 107 deaths in a two-week period in 2004 (Flewelling et al. 2005).

The deaths of 39 endangered manatees in the lower Caloosahatchee River in 1982 coincided with a *Karenia brevis* (at the time called *Gymnodium breve*) that also killed large numbers of fish and double-crested cormorants (O'Shea et al. 1991). In 1996, 149 manatees died during a *K. brevis* red tide. The affected animals showed inflammation and lesions of the nasal passages, lungs, liver, kidneys and brains, consistent with chronic brevetoxin poisoning (Bossart et al. 1998). Brevetoxin may be responsible for 17 percent of the annual mortality in manatees (Baskin 2006), and is considered to be one of the major threats to this federally listed endangered species numbering just 3300 individuals (USFWS 2007). Strandings of sea turtles also increase during red tide events; investigations have revealed that dead turtles show elevated levels of brevetoxin and live turtles display symptoms of brevetoxicosis (Redlow et al. 2002). The affected turtles include two federally threatened species, the loggerhead turtle and the green turtle, and one federally endangered species, the Kemp's ridley turtle. The 1999 red tide event may also have virtually eradicated a rare pygmy octopus from one of only four locations where it is found in Florida (Tiffany et al. 2006).

Saxitoxin, the cause of paralytic shellfish poisoning, binds to marine mammal brains along similar

pathways as in humans (Trainer & Baden 1999). Fewer poisonings have been attributed to this compound; however, the incidents that have been recorded are disturbing. An unprecedented stranding of 14 federally endangered humpback whales occurred on Cape Cod over a six-week period in late 1987. Examinations revealed that the whales had consumed mackerel whose livers were contaminated with saxitoxin, probably acquired on feeding grounds in the Gulf of St. Lawrence (Geraci et al. 1989). The single animal observed while still alive deteriorated to a terminal condition within a 90-minute period, its rapid death consistent with the respiratory syndromes of saxitoxin poisoning. Saxitoxin has also affected several bird species, including shag, northern fulmar, great cormorant, herring gull, common tern, common murre, Pacific loon and sooty shearwater (USGS 1999). Sublethal saxitoxin exposure may also contribute to reproductive failure in endangered North Atlantic right whales, which number fewer than 400 individuals (Reeves et al. 2001, Doucette et al. 2006).

Paralytic shellfish poisoning is also suspected in massive die-offs of penguins following an HAB event in the Falkland Islands in 2002-2003 (Uhart et al. 2004). Populations of gentoo and rockhopper penguins, which had been increasing, suffered a serious blow from this outbreak. While exact mortality was not directly assessed, long-term monitoring of the population revealed that the outbreak probably killed about 95,000 gentoo penguins and 176,000 rockhopper penguins, negating nearly a decade of recovery for both species (Huin 2007).

Okadaic acid, the cause of diarrhetic shellfish poisoning in humans, has not been implicated in any mass strandings or mortalities. However, the compound is known as a tumor promoter and a suppressant of testosterone production (Asada et al. 2001). Therefore, the effects of long term exposure may be of concern, particularly since it has been detected in Australia in the feeding grounds of the dugong and green turtle, two species of conservation concern (Takahashi et al. 2007).

A 2007 mass stranding of migrating fulmars, grebes, loons and surf scoters in Monterey Bay highlighted a newly heretofore unknown danger from dinoflagellate blooms. Nearly 300 birds died and hundreds more were stranded following a large outbreak of *Akashiwo sanguinea.* The bloom produced a slimy yellow protein film that coated the birds and acted as a surfactant, reducing the birds' buoyancy and insulation. This was the first recorded instance of such a phenomenon from a dinoflagellate bloom (Jessup et al. 2009).

Domoic acid, the toxin produced by diatoms that causes a form of memory loss called amnesiac shellfish poisoning, also impacts a number of wildlife species. In 1961 in Capitola, California, hundreds of sooty shearwaters, which are rarely seen on land, crashed into windows and streetlights. This incident, which probably inspired Alfred Hitchcock's movie "The Birds," is now thought to have been resulted from domoic acid poisoning caused by urea leaking from septic tanks (Coombs 2008). In 1998, over 400 sea lions stranded and died on the California coast during a *Pseudo-nitzschia australis* bloom. Many other sea lions showed neurological problems. Another stranding of 184 animals occurred in 2000. Symptoms in both cases included seizures, listlessness, head waving, and scratching behavior (Gulland et al. 2002). Researchers concluded that the toxic diatom, eaten by the sea lions via anchovies, caused the illness and deaths (Gulland 1998, LeFebvre et al. 1999, Scholin et al. 2000). Domoic acid exposure has recently emerged as a potential threat to federally threatened sea otters. It was reported for the first time in 2003 as the cause of death in 5% of the deaths of southern sea otters recorded from 1998-2001 (Kreuder et al. 2003). The toxin causes inflammation of the heart in otters (Kreuder et al. 2005).

Domoic acid outbreaks appear to be increasing in frequency and virulence. The worst outbreak to date occurred in 2007, killing a wider than ever array of species than ever seen before: seals, sea otters, dolphins, a sperm whale, a minke whale, and large numbers of birds, including grebes, gulls, cormorants, American avocets, loons and endangered California brown pelicans. Pelicans literally fell out of the sky after suffering seizures during flight. International Bird Rescue Research Center director Jay Holcomb says, "In my opinion, domoic acid is the new DDT. If the effects of [domoic acid] poisoning are cumulative in the brain, and we don't know that yet, it could have serious consequences on the population of California Brown Pelicans. As of this point, we just don't know" (IBRRC 2007).

Domoic acid also persists in the food chain following an algal bloom, so strandings can occur after the bloom subsides (Gulland et al. 2002). In pregnant female sea lions, sublethal exposure to the domoic acid has been linked to reproductive failure, premature birth, and increased risk of epilepsy in surviving offspring (Brodie et al. 2006, Ramsell & Zabka 2008, Goldstein et al. 2009). The toxin also can be transferred to offspring via milk, albeit in small quantities, posing a potential additional

chronic risk risk to young (Maucher & Ramsdell 2005). It is not yet known whether similar effects occur in sea otters or other imperiled marine mammals.

The Climate Connection

Multiple factors are involved in the proliferation of harmful algal blooms. One key cause is excess fertilization. Nitrogen and phosphorous are key limiting nutrients in aquatic systems, inputs of these elements stimulate excess growth of many kinds of algae. Nitrogen and phosphorous are key components of human and animal wastes, and are important fertilizers on land as well. Thus, they enter rivers and estuaries from many sources: improperly treated sewage, fertilized lawns and golf courses, and agricultural runoff, both from fertilized fields and animal waste (Carpenter et al. 1998). Discharge of untreated ballast water provides a means for organisms to move into new areas. Overharvesting of fish and shellfish that would keep plankton populations in check also plays a role.

While all these factors are important in promoting algal blooms, climate change is also exacerbate the problem in a number of ways.

Long-term changes in water temperatures may allow tropical species of toxic algae to broaden the seasonality of their activity or to expand their ranges into temperate zones and beyond. *Karenia brevis*, for instance, is limited by water temperatures, growing much more slowly at 15^{o}C than at higher temperatures (Magana & Villareal 2006). Saxitoxin-producing *Alexandrium catenella* also grows faster at temperatures greater than 13^{o}C (Nishitani & Chew 1984) and is predicted to move up the Pacific coast in coming years (Moore et al. 2008). Other dinoflagellates with toxic properties also show affinity for warm waters, and the fossil record demonstrates that they had wider ranges in warmer periods (McMinn 1989).

In addition to promoting harmful algal blooms, waters warmed by climate change may also magnify their detrimental effects on organisms, especially those sensitive to oxygen depletion. Warmer waters are also more likely to experience anoxic conditions because 1) warm water holds less dissolved oxygen; and 2) oxygen demand increases under warm conditions as organisms' metabolism increase (Stanley & Nixon 1992, Buzzelli et al. 2002).

Climate change might affect cyanobacteria blooms in freshwater in some unique ways. First, while

warmer water temperatures promote increased growth of many types of algae, Cyanobacteria appear to get more of a boost from water temperatures over 25°C than other types of algae; therefore at very warm water temperatures they are more likely to outcompete less harmful species (Paerl and Huisman 2008). Second, climate change can tip the balance in favor of cyanobacteria in freshwater lakes by extending the season in which a stable layer of warm water forms on the top of the lake. If the waters are turbulent, the various algae species get mixed around, but when the water is stagnant for a long period, the tiny cyanobacteria float to the top and therefore also get a sunlight advantage over larger-celled species. During the summer 2003 heat wave in Europe, incidence of *Microcystis* blooms increased, probably due to the interaction of these two phenomena (Jöhnk et al. 2007). Ocean waters can also stratify in warm conditions. This may confer an advantage upon dinoflagellates because their ability to swim allows them to travel to lower waters to obtain nutrients, whereas immobile forms of phytoplankton are stuck at the surface where nutrients may be limited (Moore et al. 2008).

Climate change may alter the dynamics of harmful algal blooms in other complicated ways. For instance, the IPCC predicts an increase in heavy precipitation events ("very likely") as well as an increase in incidence the area under drought ("likely") (IPCC 2007). Intense precipitation events are associated with large flows of runoff and associated pulse of nutrient input. In many municipalities combined sanitary and storm sewer systems are periodically overwhelmed by large precipitation events, resulting in release of raw sewage along with the rest of the runoff. Large pulses of nitrogen and phosphorus promote the growth of harmful algal blooms; however, the effect of large storms might be offset somewhat by the mixing effect and increased turbidity that result. Shifts in snowmelt timing might also send nutrients downstream earlier in the year, and the effects these changes are not known (Moore et al. 2008).

Climate change will also alter wind patterns. Since wind-driven nutrient upwellings are another important source of nutrients for algal blooms, changes in the strength and location of upwellings may alter the type, location and size of algal blooms (Bakun 1990, Moore et al. 2008). Increased area in drought condition might also contribute to algal blooms by promoting the formation and transport of dust containing iron, which is an important micronutrient in algal bloom formation (Martin & Fitzwater 1988, Hayes et al. 2001).

Finally, the world's oceans function as an important carbon sink, absorbing carbon dioxide from the atmosphere. Carbon dioxide in solution forms an acid, and oceans have become 0.1 pH units more acidic since the beginning of the industrial revolution (IPCC 2007). Continued acidification of the ocean could inhibit the growth of phytoplankton species that have shells of calcium carbonate, which dissolve in acidic conditions. More acidic oceans would favor those organisms which do not have calcium carbonate shells, including the harmful dinoflagellates (Moore et. al. 2008).

Helping Wildlife Adapt to Climate Change and Harmful Algal Blooms

Reduce the nutrient load on coastal waters. The links between pollution and algal blooms are well established, and one of the best ways to halt harmful algal blooms is to reduce the amount of nitrogen and phosphorus that wash downstream into estuaries and coastal waters. Improvements are needed to municipal storm and sanitary sewage treatment. Better timing and targeting of fertilization of farm fields, golf courses and lawns will also help reduce nutrient loads, as will widespread adoption of best management practices to contain livestock waste.

Reduce overharvest of fish and shellfish species. Overfishing is one of the oldest and most pervasive human impacts on aquatic and marine ecosystems, and it may be an important driver of ecological change in these systems; specifically, removal of species at the top of the food chain leads to a population explosion lower down the food chain (Jackson et al. 2001). Since even the algae considered most toxic are tolerated and eaten by certain species of fish and shellfish, bringing overfishing and overharvest to a halt will moderate the impacts of algal blooms.

Enact Ballast Water Controls to halt the spread of harmful species. Ballast water from cargo ships is an important vector for a wide array of aquatic invasive species; for instance, zebra mussels entered the Great Lakes ecosystem via ballast water. There is also evidence that ballast discharges have played a role in moving harmful bloom-forming dinoflagellates into new ranges (Bolsch & de Salas 2007). As climate changes create favorable conditions for harmful tropical species in temperate latitude, keeping those species out becomes even more important. Enactment of stringent, uniform performance standards for ballast water treatment will help protect aquatic and marine ecosystems from multiple invasive species.

Improve research, monitoring, coordination and veterinary techniques. Harmful algal blooms affect wildlife health, public health, fisheries, tourism, recreation and local economies. Because of their wide range of impacts, a variety of state and federal agencies as well as private entities engage in research, monitoring and response to bloom events and wildlife mortalities. However, as climate change alters bloom dynamics and potentially exposes more wildlife species to toxin risk, more research, monitoring, and rapid response will be needed (Gaydos 2006). In particular, veterinary techniques and treatments to reduce wildlife mortality have been developed for certain species; their applicability to other species should be investigated, and rapid response protocols should be developed and disseminated.

Adopt a precautionary approach toward climate change mitigation proposals that involve promotion of algal blooms. One proposal to help mitigate climate change involves promoting algal blooms by "fertilizing" stretches of open ocean with iron, mentioned above as an important limiting nutrient. Under this scenario, the resultant algal blooms would take up large amounts of carbon dioxide, before dying and sinking to the bottom, effectively locking up the carbon on the ocean floor (WHOI 2003). Other research has called into question the efficacy of this "geo-engineering" approach (Watson et al. 1994). Even more problematic, one of the species that responds best to this approach is the domoic acid-producing *Pseudo-nitzschia*. Any assessment of this geo-engineering technology would have to include potential impacts to wildlife and fisheries resources from elevated toxicity (Marchetti et al. 2008).

Chapter 5. Coral Reef Diseases

One of the most important features of coral reefs is the symbiotic nature of their inhabitants. Corals are primitive animals related to jellyfish. Each individual coral is a tiny polyp, a very simple organism consisting mostly of a stomach topped by a tentacle-bearing mouth and surrounded by a calcium carbonate exoskeleton that it secretes. Thousands of identical polyps live colonially together, each embedded in the calcium carbonate exoskeleton matrix, which over long periods of time forms the structure of the reef. The coral animals use their tentacles to sting and ingest plankton and other small creatures.

But the corals themselves are only part of the story. The corals ingest, but do not digest, tiny algae called zooxanthellae. Zooxanthellae are single-celled dinoflagellates – members of the same large and diverse group that includes some red-tide forming organisms – that are capable of photosynthesis. The zooxanthellae live inside the stomach cavity of the coral, together in a symbiotic relationship. The coral provides the algae with carbon dioxide, the essential building block of photosynthesis, important nutrients like nitrogen and phosphorus, as well as a protected place to live and reproduce. In turn, the photosynthetic action of the zooxanthellae provides the coral with up to 90% of its energetic requirements, as well as oxygen, a by-product of photosynthesis.

Because of this unique relationship, coral reefs are tremendously productive even in nutrient-poor waters. In fact, in waters that are enriched with nitrogen and phosphorus—for instance, near river outlets—other types of algae quickly outcompete the coral. Coral reefs are found exclusively in clear, shallow waters where enough light reaches the bottom to enable photosynthesis. Furthermore, corals have a fairly narrow water temperature tolerance range, about 18^{o}C to 30^{o}C (Harvell et al. 2007).

Vast numbers of organisms feed, shelter or live in association with coral reefs. Australia's Great Barrier Reef, for instance, contains 350 species of hard corals and 1500 species of fish; huge numbers of sponges, anemones and other invertebrates also live in association with reefs. While there has been no comprehensive inventory of the number of species that live on coral reefs, the number almost certainly exceeds 93,000, or five percent of the world's known species, and may up to ten times that (Reaka-Kudla 1996). Given that coral reefs only account for about one-tenth of

one percent of the world's area, this represents a remarkable concentration of species.

The structure, biodiversity and sheer beauty of coral reefs provide enormous ecological and economic benefits. By one estimate, the world's coral reefs provide annual economic benefits totaling $29.8 billion, through tourism and recreation ($9.6 billion), coastal protection ($9.0 billion), fisheries ($5.7 billion), and biodiversity ($5.5 billion) (Cesar et al. 2003). Unfortunately, 27% of the world's coral reefs have been destroyed by overexploitation, pollution, sedimentation and disease. Worse, another 30% may be lost in coming years (Cesar et al. 2003). And the loss of coral reefs has negative consequences for biodiversity: for instance, in the Caribbean, where coral cover has decreased by 80% since the 1970s, populations of reef fish have plummeted by 32 to 72% (Paddack et al. 2009). Evidence is mounting that climate change is exacerbating the threats to coral reefs, particularly the various diseases ravaging these unique environments.

Coral diseases

The disease pathology of coral reefs is incredibly complicated, due to the huge array of coral species and the multiplicity of diseases affecting them. In the Caribbean alone, 20 different diseases have been described, affecting 45 species of hard and soft corals, sponges, and other reef organisms (Harvell et al. 2007). Making matters even more difficult, causative agents have been definitively identified for only a small subset of the diseases whose symptoms have been observed. Those that have been identified span a wide array of organisms: viruses, bacteria, cyanobacteria, protozoans and fungi (Harvell et al. 2007).

Given these uncertainties, most coral diseases are referred to according to the description of their visible symptoms: white band, yellow blotch, dark band and others. Some of the more important and widespread coral diseases include:

Coral bleaching is the most serious global disease threat to corals (Harvell et al. 2007). Bleaching occurs when the zooxanthellae algae vacate their places within the coral structure. As the coral animals themselves are transparent, the loss of the brightly colored algae leaves the skeletal calcium carbonate structure visible, leading to the bleached appearance. The coral can survive for short periods by way of their predatory behavior, but because the photosynthetic action of the zooxanthellae provides such a large portion of their energy, they will eventually die if they do not re-

establish their dinoflagellate flora. The mechanisms of coral bleaching are not well understood, but there are several leading hypotheses. Rising sea temperatures might directly cause coral bleaching (Brown 1997, Jokiel 2004). It has also been proposed that bleaching can present an opportunity, allowing recombination of corals and algae that may be better adapted to changing conditions (Buddemeier & Fautin 1993). In several cases, however, bleaching has been attributed to the presence of *Vibrio* bacteria, a relative of the causative agent of human cholera. The bacteria penetrate the coral and multiply, producing a toxin that interferes with algal photosynthesis (Banin et al. 2000, Banin et al. 2001). This phenomenon of bacterial bleaching has been seen in the Mediterranean Sea and in the Indian Ocean.

Black Band Disease is one of the most common and widespread coral diseases. In the Caribbean it affects 19 species of hard coral and 6 species of soft coral (Harvell et al. 2007). In the Great Barrier Reef National Park it was found in nearly three-quarters of the reefs surveyed (Page & Willis 2006). It is also found in the Philippines (Sussman et al. 2006) and East Africa (McClanahan 2004). Infection takes the form of a narrow black band encroaching on living tissue, with the dead white coral skeletons above. The band's color comes from concentrations of photosynthetic pigments called phycocyanins that build up in infected tissue. The disease was first described on Caribbean corals in 1983 and attributed to the cyanobacteria *Phormidium corallyticum* (Rützler & Santavy 1983), though more recent research indicates that several taxa are involved (Frias-Lopez et al. 2003), including sulfate-reducing bacteria called *Desulfovibrio* (Viehman et al. 2006). These bacteria are usually found in oxygen poor environments like lake sediments, where their use of sulfate instead of oxygen for their metabolism creates the "rotten-egg" smell characteristic of some marshes. *Desulfovibrio* does tolerate oxygen, so it can live in the coral environment. But its presence in the coral creates low-oxygen conditions and a buildup of sulfide byproducts, which kill the coral (Viehman et al. 2006). Cyanobacteria are likely responsible for red-band diseases of Caribbean corals (Richardson 1992).

White plague and white band are the names given to a set of diseases with similar symptoms; namely, a spreading lesion of white skeleton. In the white plagues, this is sharply distinguished from apparently healthy tissue, with no zone of discolored tissue in between, and the white band diseases sometimes show a bleached band between the healthy tissue and the lesion (Bythell et al. 2004). The various types are distinguished by which species they infect, how quickly they spread, and the pattern of

spread exhibited (Bythell et al. 2004). Diseases of branching staghorn and elkhorn (*Acropora)* corals are generally termed white band and those on massive species are called white plague. In the Caribbean, white band has devastated corals that had been building for thousands of years (Aronson and Precht 2001). White plague I and II both affect multiple species and start at the base of the coral and work upwards, but II progresses much faster than I, at a rate of up to 2 cm per day. White plague III is very fast-acting like II, but starts in the middle of the coral (Bythell et al. 2004). The responsible pathogen has been definitively identified for two of these diseases: white plague II is caused by the bacteria *Aurantimonas coralicida,* and white band caused by another member of the *Vibrio* family, *V. carchariae* (Harvell et al. 2007). One or more species of *Vibrio* bacteria also appear to be responsible for *Porites* ulcerative white spot disease, which affects a dozen or so species in the Philippines (Harvell et al. 2007), as well as yellow blotch/band disease in the Caribbean (Cervino et al. 2004). Yet another disease in the "white" category is white pox, which causes multiple blotchy circular lesions on elkhorn corals in the Caribbean and is responsible for 85% loss in coral cover in affected areas. White pox is caused by *Serratia marcescens*, a bacteria species associated with untreated sewage (Patterson et al. 2002).

While most of the diseases described above are bacterial or cyanobacterial, protozoans cause several diseases as well: skeletal eroding band, found in Australia, resembles black band disease, but is attributable to *Halofolluculina corallasia* ciliate protozoans that destroy the limestone skeleton of the coral (Antonius & Lipscomb 2001). An as-yet-unidentified ciliate appears to be responsible for brown band syndrome, which is found in the Great Barrier Reef and is similar in appearance to black band syndrome (Ulstrup et al. 2007). Fungi also cause coral reef disease: *Aspergillosis*, which causes purple blotches on several species of sea fans, is caused by *Aspergillus sydowii*, a member of a large group of terrestrial fungi that also trigger mold allergies and other infections in humans (Geisner et al. 1998). Finally, emerging research indicates that viruses may also play a role in coral reef disease, but their extent and pathology remain uncertain (Davy et al. 2006).

The Climate Connection

The loss of coral reefs is a complicated phenomenon and involves a host of factors. Pollution from onshore has long been recognized as a significant threat to coral reefs. Sewage effluent, for instance, damages coral reefs through three major mechanisms: nutrient input, sedimentation and toxic elements (Pastorak & Bilyard 1985). Agricultural runoff poses similar problems. Nutrient

enrichment promotes algal blooms in the water as well as the growth of algae on top of the coral itself. The algae block the light and suffocate the corals. Sedimentation reduces light availability, and also directly abrades the surface of the corals. And pollutants like PCBs, pesticide residues, heavy metals and other chemicals are also damaging to corals (Pastorak & Bilyard 1985).

In addition to being detrimental on their own, pollutants can exacerbate the impact of diseases. One study found that the nutrient enrichment doubled the amount of damage caused by the coral diseases aspergillosis and yellow band (Bruno et al. 2003). A similar effect has been found for black band disease (Voss & Richardson 2006).

Overfishing is also a problem, since the main mechanism to keep algae in check is grazing, and reduced fish stocks allow algae to proliferate even further. Aquaculture and discharge of ballast water are also hastening the pace at which exotic and potentially damaging species enter new marine environments.

While all these factors are important, mounting evidence suggests that climate change is exacerbating the disease threat to corals. The Fourth Assessment IPCC states that "recent warming is strongly evident at all latitudes in sea surface temperatures over the oceans" (IPCC 2007). Sea temperatures have already risen by 0.7^{o}C, and are currently the warmest they have been at any time in the past 420,000 years (Hoegh-Guldburg et al. 2007). Other important effects of climate change include increased incidence of high temperature anomalies, changes in wind patterns, and an increase in extreme events, and ocean acidification.

The physical effects of climate change worsen coral disease via several mechanisms: 1) thermal stress to the coral animals and their associated algae; 2) creation of advantageous conditions for the disease organisms; 3) other impacts from weather events, acidification and altered weather patterns.

Thermal stress to corals. Geographic and seasonal patterns of disease prevalence in corals strongly suggest that increases in temperature also play important role in disease dynamics.
Disease prevalence increases when approaching the equator: north to south in the Caribbean (Harvell et al. 2007) and in the southern hemisphere, black band disease increases from south to north in Great Barrier Reef (Page and Willis 2006).

The various coral reef diseases also show seasonal trends, with increased disease progression in the warmer months. Black band disease in the Great Barrier Reef progresses nearly twice as quickly in the summer months and showed accelerated rates of transmission as well; both warmer temperatures and increased light availability contributed to the acceleration of the disease (Boyett et al. 2007). White pox disease also shows a strong seasonal component, with large losses of coral in the summer months and slight regrowth of the coral during the winter, implying that warmer temperatures accelerate the disease (Patterson et al. 2002).

There is evidence that unusually warm sea temperatures stress corals and increase their vulnerability to disease. Coral bleaching, characterized by the loss of photosynthetic algae, intensifies under warm sea surface temperatures, even in the absence of a pathogen (Brown 1997). For instance, warm sea surface water temperature anomalies associated with the El Niño Southern Oscillation (El Niño) produce coral bleaching events. A warm water upwelling event in the eastern Pacific in 1982 produced high coral mortality in the Gulf of Panama (Glynn & D'Croz 1990). Similarly, the 1997-98 El Niño event produced a temperature anomaly increasing the average temperature of tropical oceans by about 3 degrees C. Major worldwide coral mortality occurred during this high sea surface temperature anomaly (Sheppard and Rioja-Nieto 2005). During this event, two of the major coral reef systems in the Indian Ocean suffered roughly 25% mortality from bleaching (Arthur 2000), and severe and widespread bleaching occurred in the Palau archipelago of the western Pacific (Bruno et al. 2001). Extensive bleaching occurred in the waters of Belize as well (Aronson et al. 2002).

Another temperature anomaly in 2005 was followed by bleaching of over 30% of a reef colony in the Virgin Islands; additionally, some of the coral suffered subsequent mortality from white plague II, raising the possibility that the thermal bleaching left the coral more susceptible to the disease (Whelan et al. 2007).

In the temperate coral communities of the Mediterranean Sea, outbreaks of *Vibrio coralliilyticus* caused extensive mortality of *Paramuricea clavata* during climate anomalies that elevated sea level temperatures; the authors of this study went so far as to title their findings "a new case of emerging disease linked to climate change" (Bally & Garrabou 2007).

Thermal stress has also been linked to outbreaks of white syndrome across a large scale (1,500 km region) of Australia's Great Barrier Reef, though researchers also found that coral cover was an important factor in the spread of the pathogen (Bruno et al. 2007).

Recent research has demonstrated one of the mechanisms of thermal stress for *Gorgonia* fan corals and other invertebrates in the Mediterranean: warm water temperatures are stressful to corals and other marine invertebrates in that warmer water holds less dissolved oxygen, so animals must expend more energy to breathe. Unfortunately, the warm temperatures also increase the stratification of the water, so plankton, which tend to move to cooler waters, become less available as a food source. The period of unfavorable summer conditions has been lengthening for a period of about one day per year since the 1970s, becoming an increasing source of stress for many members of the marine community (Coma et al. 2009).

Advantages to pathogen. High temperatures also directly benefit the disease organisms. *Auratimonas coralicida*, the bacterium that causes white plague II, shows optimal growth between 30 and 35ºC. Furthermore, higher temperatures also allow the bacteria to thrive in more acidic conditions. This is important because the outer protective coating of the coral is a mucopolysaccharide layer that has a low pH. Thus warmer temperatures confer a double benefit to the disease: pathogens are better able to survive the coral's defense mechanisms and mount an infection, and they grow faster as well (Remily & Richardson 2006). Similarly, *Vibrio shiloi*, the bacteria that causes bacterial bleaching, adheres to the surface of the coral more readily at higher sea temperatures. Warmer temperatures allow the bacteria to produce an adhesive compound that helps it burrow through the mucopolysaccharide layer (Rosenberg and Ben-Haim 2002).

The specifics of high temperatures' benefit to the disease and the stress to the corals may be difficult to unravel, and in many cases both factors may be at work. For instance, when corals are inoculated with the *Vibrio* bacteria that cause yellow blotch/band, the disease progresses much more rapidly and causes much higher mortalities at 32ºC than at 20ºC (Cervino et al. 2004). The disease attacks the zooxanthellae, and the authors postulate that the bacteria's toxicity to the algae may be enhanced at high temperatures. However, researchers also found that healthy control corals held at the same temperatures began to show signs of thermal bleaching (expulsion of the zooxanthellae) at the highest temperatures, so both benefits to the bacteria and stress to the coral may be at work.

The combination effect is also evident in the dynamics of Aspergillosis infection of *Gorgonia* sea fans. The *Aspergillus* fungus shows and optimal growth rate at 30°C; and in addition, the defense mechanisms of the sea fan were less effective at the higher temperatures (Alker et al. 2001).

Other impacts. Climate change impacts extend beyond changes in water temperature. As discussed above, aspergillosis disease of fan corals is caused by a terrestrial fungus. It was hypothesized several years ago that dust storms from Asia and Africa might carry viable micro-organisms all the way to the Caribbean (Garrison et al. 2003). Soon after, researchers isolated the spores of Aspergillus from African dust storm deposits in the Virgin Islands and were able to determine that they were the same as those sampled from diseased corals in the Caribbean (Weir-Brush et al. 2004). Transatlantic dust deposition has been on the increase over the past thirty years as drought conditions have intensified in the Sahara and Sahel regions (Prospero and Lamb 2003); therefore it seems the purple blotches on Caribbean fan corals may be yet one more fingerprint of climate change.

Hurricanes are important tropical phenomenon, and since climate change may result in increased intensity of some hurricanes, they deserve mention as well. However, the balance of hurricane impacts is far from clear. In the Caribbean, loss of coral cover from the passage of a hurricane averages 17% (Gardner et al. 2005). In addition, growth and recruitment of massive corals in the Caribbean and Belize was lower in years when they were impacted by hurricanes or tropical storms than in years without storm passage (Crabbe et al. 2007).

On the other hand, one coral reef that was being monitored for long term impacts of white pox actually showed a slight increase in live coral cover after the passage of Hurricane Georges in 1998 (Patterson et al. 2002). In the summer of 2005, intensive bleaching was underway in the coral reefs off of Florida and the U.S. Virgin Islands; however, the Florida reefs experienced some recovery following the passage of Hurricanes Rita and Wilma (Manzello et al. 2007). Hurricane passage is associated with wind-driven mixing of water layers and a reduction of sea temperature by up to 5°C and thus may actually alleviate some of the thermal stress behind bleaching and other disease outbreaks (Manzello et al. 2007). Hurricanes have also scoured invasive algae off of reefs off the coast of Florida (La Pointe et al. 2006).

One more relevant outcome of anthropogenic carbon dioxide output is the acidification of the oceans. As carbon dioxide emissions have risen, a significant proportion of the carbon dioxide—roughly 25%-- has been taken up by the oceans (Canadell et al. 2007). The fraction taken up by the ocean dissolves to form carbonic acid, which, in sufficient quantity, decreases the pH of the ocean water. The pH of the oceans has already dropped by 0.1 pH units, to the lowest level of the past 420,000 years (Hough-Guldberg et al. 2007). Acidification reduces availability of carbonate to living systems, inhibiting the ability of reef-forming organisms to lay down their aragonite crystal skeletons. Corals exposed to lower pH conditions exhibit decreased growth rates and lowered density of their skeletons (Hough-Guldberg et al. 2007). It is not yet known whether acidification leads to greater disease susceptibility or interacts with thermal stress in other ways (Eakin et al 2007), but this important consequence of increasing carbon dioxide emissions should not be ignored.

Helping Coral Reefs Adapt to Climate Change and Disease

Improve water quality in tropical oceans. As discussed throughout this document, the dynamics of coral reef disease are very complicated and affected by a wide variety of factors including, but not limited to, climate change. Pollution from onshore sources has been long recognized as a destructive force to coral reefs. Steps should be taken to limit this stress on these ecosystems as an important means of implementing climate change adaptation for corals.

Reduce overfishing. As with many marine systems, overfishing has harmed coral reef ecosystems by removing fish that would graze upon algae that threaten to overgrow corals. But coral reefs are critically important nursery areas for the young of many commercially fished species. Thus, the loss of coral reefs will potentially exacerbate the effects of over-fishing, since recruitment depends on these nursery habitats. Fishing-free, fishing-reduction zones or other management measures may help corals adapt to climate change.

Manage ballast water to prevent spread of exotic organisms, invasive species or pathogens. Given the uncertainty about the origins of many coral diseases, and the potential for these organisms to be spread to new regions in the ballast water of ships, enactment of stringent, uniform performance standards for ballast water treatment is as important for the protection of coral reefs as it is for reducing the spread of harmful algal blooms. Measures should also be put in place to halt the release of exotic

aquarium species into waterways where they could transmit pathogens to corals or to other reef species. This is particularly prudent given the uncertainties regarding the nature and origins of many coral pathogens.

Expand research into the origins of coral disease and potential treatments. Coral diseases are largely uncharted scientific territory: for all but a handful of the disease syndromes, the causative organism or set of organisms have not even been definitively identified. We are only beginning to understand the dynamics of infection and resistance. The possibility remains that some coral diseases might be treatable, for instance by inoculation with viruses that can help the corals combat invading microbes (Efrony et al. 2007). Given the tremendous biodiversity and economic benefits provided by coral reefs, a greater societal investment in basic research and protection of these systems is warranted.

Chapter 6. Diseases of Freshwater Fish

Aquatic ecosystems are hardly immune from the effects of climate change and diseases. In some ways, aquatic wildlife may be even more vulnerable to the effects. For instance, trout and salmon, which require clear, cold water, are increasingly being stressed by climate change (Trout Unlimited 2007). Additionally, the discreteness of stream ecosystems prevents natural migration of fish and other aquatic animals between watersheds when conditions become climatically unfavorable. Pest and disease species, on the other hand, have an unfair advantage: many are tiny and can be transported unintentionally between systems via recreational equipment, bait buckets, the surfaces and ballast water of ships, and other means. The exotic zebra mussel serves as a particularly vivid and well-documented example: in 1988 it arrived in Great Lakes through ballast water and has since spread throughout much of the east, and is pushing westward as well (Benson 2008). Two protozoan parasites, whirling disease and icthythophonus, are of particular conservation concern because of their potential to interact with climate change.

Whirling Disease

Life cycle

Whirling disease, so named because it causes infected fish to swim in circles, is caused by a tiny parasite called a "myxozoan," a tiny animal related to jellyfish and corals. The organism, termed *Myxobolus cerebralis*, has a complicated life cycle that was not entirely deduced until the 1980s (Markiw and Wolf 1983, Wolf et al. 1986). The life cycle is presented in detail by Gilbert & Granath (2003). When a fish infected with the parasite dies, spores of the parasite are released. If some of these spores are ingested by a tiny, aquatic worm called *Tubifex tubifex*, the spores sprout tiny filaments and attach themselves inside the worm's gut, where they then begin to reproduce. The offspring are called triactinoyxons (TAMs), which are released into the water column in the feces of the worm. The free-floating TAMs (once thought to be an entirely separate organism) attach to the skin of a salmonid fish and bore in, reproducing first in the skin layers and then migrating to feed on nerve and cartilage tissue. Inflammation from the infection constricts the spinal column and puts pressure on the brain stem, leading to the characteristic whirling behavior (Rose et al. 2000). The whirling behavior, as well as skeletal malformations from the infection, can sufficiently interfere with regular feeding and swimming as to be fatal (MacConnell & Vincent 2001). The disease causes high levels

of mortality in young fish whose skeletons are mostly cartilage; the disease has less impact on older salmonids whose skeletons have hardened into bone.

Conservation Threat

Whirling disease is native to Europe, where it appears to have co-evolved with the brown trout (*Salmo trutta*). It was introduced to the U.S. in the 1950s. Originally a problem mainly at hatcheries, has since escaped and spread to 23 states. Rainbow trout/ steelhead (*Oncorhynchus mykiss*) and sockeye salmon (*Oncorhynchus nerka*) are the most susceptible species (Whirling Disease Initiative undated). Brown trout, introduced from Europe in the 1880s, show markedly less susceptibility to whirling disease than native species like the rainbow trout (Hedrick et al. 1999). In some parts of Montana the disease has caused 90% reductions in the rainbow trout population (Potera 1997) and has decimated populations of Yellowstone cutthroat trout (*Oncorhynchus clarki bouvieri*), a keystone species in the Greater Yellowstone Ecosystem (Koel et al. 2007). The disease may also prevent a barrier to the recovery of rare species like the greenback cutthroat trout (*O. c. stomias*) (Cooney, undated), and Gila (*Oncorhynchus gilae gilae*) and Apache trout (*O. g. apache*) (AZGFD 2007).

Climate Connection

Trout and salmon species favor cool water -- maximum temperature tolerance for most salmonids is in the range of 20° to 24°C (Eaton & Scheller 1996). Climate models predict that stream temperatures will rise in many locations over the 20th century (ISAB 2007, Eaton & Scheller 1996). As stream temperatures increase, up to 50% of existing salmonid nationwide habitat will become too warm (Eaton & Scheller 1996). More specific models of the North Platte River in Wyoming predict that each degree Centigrade of temperature increase leads to approximately a 10 to 15% loss of stream habitat (Keleher and Rahel 1996, Rahel et al. 1996). These researchers also predict that as the fish become increasingly restricted to the colder headwater streams, their populations will be more fragmented and more isolated, since the populations will no longer be able to intermingle in the warmer mainstems downstream.

Unfortunately, as climate change contracts the range of many cold-water fishes, it will push them more firmly into the stronghold of whirling disease, which itself will also most likely expand due to climate change. Like many pathogenic organisms, both the whirling disease microorganism and the tubifex worm that is critical in the disease's life cycle respond to changes in water temperature. An

early study of the effect of water temperature on disease development concluded that 15°C is the optimum temperature for disease development (El-Maboutli et al. 1999). A later study refined this conclusion, determining that development of the myxozoan within the worm occurs most rapidly at 17°C, but that the largest number of TAMs is produced at 13°C (Blazer et al. 2003). Both studies found that TAM production dropped off at incubation temperatures above 20°C, at temperatures approaching the thermal maxima for many salmonids.

These results have important implications for the role of climate change in altering the infectivity of whirling disease. The disease is most dangerous to young hatchling salmonids, for instance, the effects on rainbow trout are most severe if the young fish come into contact with TAMs in the first nine weeks after hatching (Ryce et al. 2004). Since the development and release of TAMs occurs more slowly at low temperatures, cold water, especially in the spring, may have a protective effect on young trout (Kerans et al. 2005). However, climate change is predicted to decrease snowpack in the Rocky and Cascade Mountains (NAST 2000). Reduced amounts of coldwater snowmelt in spring could lead to a faster spring warming of streams when young hatchlings are most vulnerable to whirling disease. A temperature-mediated increase in the rate of disease development would mean release of infective TAMs when more hatchlings are still quite young, thereby increasing the severity of the disease's impact.

Ichthyophonus

Life Cycle

Ichtyhophonus hoferi is a fish parasite, classified in a group of protozoans that appear to be intermediates between animals and fungi. Fish contract the disease when they eat an infected fish whose tissue contains the fungus-like spores. In the acidic environment of the fish's stomach, the spores germinate and send finger-like projections called hyphae through the wall of the stomach. Upon contact with a blood vessel, apparently mediated by a change to neutral pH, the hyphae burst and send small amoeba-like cells into the bloodstream. Settling in the heart, liver or muscle tissue, the amoeba-like parasites set about producing more spores (Spanggaard et al. 1995). The spore "factories" are visible as white nodules on the muscle and organs of infected fish (Kocan et al. 2004). Infected fish die of multiple organ failure (Rahimian 1998) or of cardiac damage that reduces swimming stamina during spawning (Kocan et al. 2006)

Conservation Threat

Ichthyophonus appears to have a global distribution and a wide range of potential freshwater and marine hosts. For instance, outbreaks have been reported among rainbow trout from Korea (Chun & Kim 1981) and Japan (Miyazaki & Kubota 1977), largemouth bass (*Micropterus salmoides*) in Taiwan (Tung et al. 1986), sea bream (*Sparus aurata*) and rainbow trout in Greece (Athanassopoulou 1992), sea bass (*Dicentrachus labrax*) from Spain (Sitja-Bobadilla and Alvarez-Pellitero 1990), yellow-tail flounder (*Limanda ferruginea* Storer) in the Atlantic (Ruggieri et al. 1971), and herring (*Clupea* spp.) in both the Atlantic (Scattergood 1948, Rahimian, 1998) and Pacific (Jones & Dawe 2002).

In the mid-1980s, the disease appeared in Alaska's Yukon River Chinook salmon (*Oncorhynchus tshawytscha*), where it was noticed by subsistence fishermen (Kocan et al. 2004). By 2003 infection prevalence reached 45% (Kocan et al. 2004). Infection prevalence decreases far upstream near spawning grounds, presumably because a high proportion infected fish die along the very long spawning distance due to cardiac damage from the disease (Kocan & Hershberger 2006, Kocan et al. 2006). Loss of a substantial portion of the spawning population in their largest river system could have serious consequences for the Chinook salmon, as well as grizzly bears, orcas and other species that feed on them, not to mention the subsistence, sport and commercial fishing economies they support. Total catches of Yukon Chinook have declined dramatically, with the 2000-2004 catch in Alaska and Canada averaging about 60% of historical averages (JTC 2006).

Climate Connection

Several lines of evidence suggest that warm temperatures exacerbate ichthyophonus infection. Laboratory experiments on rainbow trout have demonstrated that the disease severity is correlated with temperature: experimentally inoculated fish held at 4°C did not develop the disease, fish held at 10°C all developed the disease but had 90% survival, and fish held at 15°C and 20°C all suffered fatal infections (Okamoto et al. 1987). In a multi-year study of infection patterns in Yukon Chinook, fish that spawned later in the summer, when river temperatures tended to be in the 15-20°C, had much more severe infections than fish spawning earlier in spring when water temperatures were cooler (Kocan et al. 2003). These researchers also found that the lowest level of disseminated disease occurred in 2002, when the average water temperature was lowest, and was higher in the years with higher average water temperature. From 1975 to 2001, mean water June water temperature rose from less than 11ºC to about 15ºC (Kocan et al. 2003). Climate change is predicted to continue to

raise river temperatures in Alaska (ACCAAG 2008), potentially further exacerbating the impacts of ichthyophonus on Chinook salmon.

Helping Fish Navigate Climate Change and Aquatic Diseases

Help fish navigate the rivers. Dams block access to quality upstream habitat. Dam removal, fish passages will broaden the range of habitat available for fish; more importantly, it will allow access to the upstream habitats that are in general cleaner and colder than the downstream areas.

Prioritize streamside habitat restoration. Restoring vegetation along stream habitats shades and cools the water, and also filters nutrients and sediment that reduce water quality. Forestry practices, including sufficiently wide buffers and watershed cover, to maintain cool and other essential water quality characteristics.

Clean fishing and hatchery practices. Boats, fishing and other recreation gear, bait dumping and the transport of hatchery fish are important vectors for the introduction of pathogens and other invasive species. Efforts are underway to educate the fishing and water recreation public as to the importance of clean boating practices. Continuation and expansion of these programs is recommended. Hatcheries should also implement best management practices to test for and control diseases that could spread in the wild fish population.

Chapter 7. Emerging Case Studies in Climate Change and Wildlife Disease

In addition to the detailed profiles described throughout this document, new evidence linking climate change to wildlife disease is emerging in many parts of the world. Several of these diseases have only recently been discovered to impact certain wildlife species, and in many cases much more needs to be learned about these diseases. However, even given these knowledge gaps, a pattern is emerging that suggests climate change exacerbating multiple wildlife and disease interactions.

Nematode Infections in Muskoxen

Muskoxen are large mammals, more closely related to sheep than to oxen, found in the harshest expanses of the Arctic tundra. Nearly 6 feet tall and weighing 600 pounds, they are distinguished by their large curving horns and thick fur that reaches almost to the ground. They live in the northernmost reaches of Nunavut and the Northwest Territories, the islands of the Canadian High Arctic, and in parts of coastal Greenland. Once extirpated from Alaska, they were reintroduced in the 1930s and now number about 2200, divided between the Yukon- Kuskokwim Delta area and northern and northwestern coasts of Alaska.

A previously unknown parasite of muskoxen was discovered in 1988 and determined to be a new species in 1995 (Gunn et al. 1991, Hoberg et al. 1995). The parasite is carried by snails that live in the grass. Muskoxen acquire it while grazing if they accidentally ingest snails infected with larvae that are near adulthood (Kutz et al. 1999). Once ingested the larvae move into the muskox's lungs, where they mature into adults, form cysts in the lung tissue, and lay their eggs. The new larvae move out of the lungs and into the throat of the animal, and pass through the digestive system. Once excreted, the larvae burrow into the foot of a snail, where they move through their larval stages until picked up by another grazing muskox (Kutz et al. 2001).

For a disease that was unknown to science less than 25 years ago, the muskox lungworm has achieved a remarkably high prevalence in just a few short decades. Researchers postulate that the relative lateness of the nematode's discovery can be attributed to a combination of low prevalence of the disease and relatively low levels of researcher effort on the topic (Kutz et al. 2004). Independent of researcher effort, however, a real and dramatic expansion of the disease also seems to be taking

place, with nearly 100% incidence of infection in some parts of Nunavut Territory (Kutz et al. 2004). Evidence is mounting that this expansion may be driven by climate change.

In general, temperature is an important factor regulating the life cycle nematodes. For instance, the larvae move through their development cycle more quickly when summer temperatures are warmer (Greifenhagen and Noland 2003). Another important temperature effect occurs in winter: very cold winters kill off eggs and larvae, so survivorship of eggs and larvae is higher in milder winters (Greifenhagen and Noland 2003). Susan Kutz and her colleagues (2002) have found that under normal temperatures, the muskox lungworm nematode requires two years to move through its larval phases inside a gastropod. However, warm summer temperature speed up the cycle and allow them to complete development in a single year. The extension of the growing season via warmer springs and autumn temperatures also helps the larvae complete the cycle. Developing in one year rather than two confers an enormous advantage on the nematode: it means that the larvae don't have to overwinter at all, and are thus freed from a significant cause of mortality. Instead, at the end of just one season, they are ready to move into the relative safety (from a temperature perspective) of the inside of a muskox, where they can then lay their eggs and begin the cycle anew.

In their 2004 article on muskox lung nematodes, Susan Kutz and colleagues point out that temperatures in the area of highest infection have risen 2.0^{o}C over the past sixty years, and that eighteen of the twenty years from 1983-2003 were above historical averages. Data from the Canadian Meteorological Service shows that this trend has continued: following near-normal years in 2004 and 2005, summer temperatures in the western Canadian High Arctic have been 1.0 to 1.5 Centigrade degrees above normal in 2006-2008 (CMS website). They postulate that this change is tilting the ecological balance in favor of the parasite.

Nematode infection damages the lungs of the animals and may impair with their ability to flee from grizzly bears and other predators (Kutz et al. 2004). The disease may have other effects as well, such as interfering with growth, reproduction or survival; the potential impacts have not been fully investigated. However, muskoxen populations appear to be declining in places where lungworm infectivity rates are highest, and both of these are correlated with climate warming over the past ten to fifteen years (Kutz et al. 2004).

More importantly, muskoxen may not be alone among arctic ungulates in suffering increased levels of parasitism due to climate change. The climate-mediated alteration in the dynamics between host and parasite may be playing out in other species as well. As with the muskoxen lungworm, Arctic warming has lengthened the "growing season" for a two nematode parasites of Dall's sheep, one infecting the muscle and the other the lungs (Jenkins et al. 2006). The researchers studying this species predict that the two parasites will spread northward and cause more intense infections in coming years. Similarly, northern fur seals often suffer infections from hookworms that cause anemia, which is particularly deadly to pups. Like other nematodes, hookworms' life cycle is tied to temperature and humidity, so they might also become more prevalent as the Arctic warms (Burek et al. 2008).

Disease Convergence in Lions

Climate change may cause synergistic effects between diseases. Researchers in Tanzania have found this effect in lions. Two diseases that generally cause little mortality when acting alone-- canine distemper and *Babesia* – produce devastating die-offs when they hit at the same time. Extreme climate events, particularly droughts, increase the likelihood of concurrence.

Canine distemper is a virus in the morbillivirus family, related to measles in humans. Despite the implication from the name that it is a disease of dogs, it affects a variety of species: in addition to dogs, wolves and coyotes, it can be found in weasels, raccoons (Appel & Gillespie 1972), seals (Kennedy et al. 2000) and large felids (Appel et al. 1994). Symptoms of CDV include fever, gastrointestinal problems, loss of appetite, difficulty breathing, and seizures characterized by chewing motions and waving of the legs (Appel et al. 1994).

The first fatal occurrence of CDV in wild lions occurred in the Serengeti National Park in Tanzania in 1994, killing nearly 30% of the lions in the Serengeti-Masa region (Roelke-Parker et al. 1996). Fatal cases showed pneumonia and swelling of the brain. It was originally thought that this was the first exposure of the Serengeti lions to the CDV virus and that the high fatalities were attributable to "immunological naïveté" among the lions (Roelke-Parker et al. 1996). However, later research demonstrated that CDV epidemics had occurred sporadically in the area from1976 to 2006, but only two of these caused significant lion fatalities: the 1994 Serengeti epidemic and a 2001 epidemic in nearby Ngorongoro Center (Munson et al. 2008). It was at first unclear what circumstances caused a

subset of CDV epidemics to prove so much more fatal than others. The difference, it turned out, was co-infection with *Babesia*.

Babesia is a protozoan blood parasite similar to malaria in that it attacks red blood cells and causes infected individuals to suffer from a lack of oxygen to cells and tissues. Unlike malaria, which is transmitted by mosquitoes, *Babesia* is transmitted by ticks. Low levels of infection are common in lions and do not appear to be particularly problematic (Penzhorn 2006). Linda Munson and her colleagues (2008) found, however, that many lions had unusually high *Babesia* infection levels during the 1994 and 2001 fatal canine distemper epidemics. *Babesia* infection levels were not unusually high during the nonfatal CDV outbreaks, which led the scientists to hypothesize that some environmental factor was contributing to high *Babesia* levels, which in turn reduced the lions' ability to survive the concurrent CDV outbreaks.

The researchers found that the key environmental factor in the 1994 and 2001 epidemics was the occurrence of a severe drought. On important prey species, the Cape buffalo, was particularly affected by the drought. In their malnourished and weakened state, they had much higher than usual loads of parasites, particularly ticks. As the Cape buffalo were killed off by the drought, the lions took advantage of the large food source. But as the lions fed on the buffalo, they acquired a much higher than usual load of *Babesia* infected ticks. Suffering from unusually heavy *Babesia* infections, the lions were less able to cope with canine distemper virus, and the combination of the two diseases killed many more lions than either disease commonly would acting on its own (Munson et al. 2008).

Climate change looms large as a threat in this story. Two pathogens, each relatively benign on their own, combined with deadly force in the face of an extreme weather event, in this case severe drought, that is predicted to become more frequent under current climate change scenarios (IPCC 2007). Munson and her colleagues warn that, "If extreme weather events become increasingly frequent owing to global climate change, the consequent synchronization of proliferating pathogens or their vectors may cause disease to become a major threat to historically stable populations that had previously coexisted with multiple viral and parasitic pathogens."

Distemper in Marine Mammals

As described above, canine distemper virus affects a wide array of animals, including seals (Kennedy et al. 2000). In 1988, several thousand Baikal seals in Lake Baikal, Siberia were killed by canine distemper (Grachev et al. 1988). In the Caspian Sea in the spring and summer of 2000, over 10,000 Caspian seals died from pneumonia and lymph gland damage due to canine distemper. The exact pathology and causes of this epidemic have not been pinpointed and a number of factors may be at work, including contamination with immune-suppressing organochlorine pollutants (Kuiken et al. 2006). Researchers did note, however, that the winter preceding the epidemic was significantly warmer than the 10-year average, and ice cover disappeared earlier that year than in any of the previous ten years. They hypothesized that the rapid ice loss that year might have reduced available locations for the seals to haul out, and in turn led to an unusually high rate of concentration, allowing for rapid spread of fatal canine distemper (Kuiken et al. 2006).

Another member of the morbillivirus family is phocine distemper, which is very similar but is restricted to seals and sea lions. Phocine distemper epidemics killed 18,000-23,000 harbor seals in northern Europe in 1988 and 21,700-30,000 in 2002 (Jensen et al. 2002, Müller et al. 2004, Harkonen et al. 2006). The harbor seal epidemics were probably caused by contact with a migratory species, such as grey seals, which may have acted vector spreading the disease between harbor seal populations (Harkonen et al. 2006). As climate change causes changes in ranges and feeding patterns, there may be increasing instances where species with previously separate ranges encounter each other, providing new opportunities for diseases to spread to species not previously affected (Burek et al. 2008). In fact, Burek and colleagues' 2008 prediction may be playing out already: phocine distemper appears to have jumped from the Atlantic to the Pacific Ocean due to retreating sea ice. The virus had not previously been found in the Pacific basin, but researchers studying a 2006 mortalities of sea otters in south-central Alaska found them infected with a strain of phocine distemper that matched the 2002 strain that killed harbor seals in northern Europe (Goldstein et al. 2009). The species vector responsible for transmitting the virus to the otter has not yet been identified; however, the authors suspect that sea ice reductions allowed infected Atlantic seals to transmit the virus to Pacific marine mammals. They conclude that "All Pacific marine mammal species are now at risk for phocine distemper–induced population decreases" (Goldtein et al. 2009).

Avian Influenza

Very few studies have considered the potential impacts of climate change on the range, rate of spread, pathogenicity, or species affected by avian influenza (Gilbert et al. 2008). One study has attributed the spread of H5N1 in Europe to migratory birds, particularly swans, ducks and geese (Kilpatrick et al. 2006). On the other hand, the pathogenicity of H5N1 may hamper infected birds' ability to migrate, reducing their ability to spread the virus widely (Weber and Stilianakis 2007).

One of the only treatments of the topic to date suggests that if climate change alters the breeding or migratory patterns of ducks, swans, geese or shorebirds, these species may spread one or more strains of avian influenza to new places. The greatest potential for wide-scale spread probably occurs during fall molt migration staging, when large numbers of multiple species, many temporarily flightless, congregate in narrow geographic areas. Any changes that further restrict these staging areas might increase transmission (Gilbert et al. 2008).

Conclusions

As demonstrated through the case studies in this report, climate change is interacting with wildlife disease to create an ever-widening threat to terrestrial, marine and freshwater wildlife species. A wide variety of measures should be undertaken to meet this threat, including: 1) improved surveillance for diseases; 2) additional research into the dynamics of disease, climate change, and other wildlife threats; and 3) measures to ameliorate the threats of climate change and wildlife disease.

Addressing these challenges will require a greater combined commitment from federal and state wildlife agencies, the public health community, conservation organizations, researchers and funders. Unfortunately, the threat of disease has not been at the forefront of climate change adaptation research and planning for most agencies. Defenders of Wildlife recently conducted interviews with 68 wildlife professionals from federal and state agencies, non-governmental conservation organizations and scientists who are working on climate change adaptation. The experts interviewed were asked to discuss their planning efforts, techniques and practices related to helping wildlife adapt to climate change (Theoharides et al. 2009). Not a single one of the professionals interviewed volunteered disease as one of the climate change threats, or as an issue that needed to be addressed in the context of climate change adaptation (K. Theoharides; pers. comm.).

A recent federal funding appropriation enabled the USGS to create a National Climate Change and Wildlife Science Center. “In December 3–4, 2008, the USGS convened a workshop that brought together nearly 200 representatives from invited state and federal agencies, tribal organizations, academia and nongovernmental organizations to identify research needs and priorities, devise strategies for partnerships and collaboration, and to begin to design a structure for the Center” (USGS 2008). The summary report from this workshop mentioned the word “disease” only once.

The disconnect works in reverse as well: the most important web-based wildlife disease resource, the USGS/University of Wisconsin’s Wildlife Disease Information Node, contains very little mention of climate change (WDII, undated).

One notable exception to the nearly universal disconnect between attention to thinking on climate change and wildlife disease is the U.S. Fish and Wildlife Service's draft "Strategic Plan for Responding to Accelerating Climate Change in the 21st Century." This report identifies disease as one of the conservation consequences of our changing climate (USFWS 2008). As one of the Plan's goals, the Service intends to "…deliver landscape conservation that supports climate change adaptations by fish wildlife and plant populations of ecological and societal importance." One of the nine objectives under this goal is to "reduce susceptibility to diseases, pathogens, pests and contaminants." The Service's plan to do so entails improving surveillance and response, improving predictability about the effects of climate change on wildlife disease, and reducing the vulnerability of wildlife to disease threats (USFWS 2008). All of these are very important goals for the Service and will be critical components of helping wildlife adapt to climate change. It is crucial that the final Strategic Plan retain this focus, and that future plans and actions stemming from the Strategic Plan more fully flesh out how the Service will achieve these goals. More attention to climate change and wildlife disease is also needed at other federal agencies and research centers, such as the USGS National Climate Change and Wildlife Science Center, National Wildlife Health Center, and the other federal wildlife research centers. Equally important, states must recognize disease as they update their wildlife action plans and other strategic plans to address the threat of climate change to wildlife.

References

Introduction

Ben Ari, T., A. Gershunov, K.L. Gage, T. Snäll, P. Ettestad, K.L. Kausrud and N.C. Stenseth. 2008. Human plague in the in the USA: The importance of regional and local climate. *Biology Letters* 4(6):737-40.

Cross, P.C., W.H. Edwards, B.M. Scurlock, E.J. Maichak, and J.D. Rogerson. 2007. Effects of management and climate on elk brucellosis in the Greater Yellowstone Ecosystem. *Ecological Applications* 17(4):957-964.

IPCC 2007. Summary for Policymakers. In: *Climate Change 2007: Impacts, Adaptation and Vulnerability*. Contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. M.L. Parry, O.F. Canziani, J.P. Palutikof, P.J. van der Linden and C.E. Hanson, Eds., Cambridge University Press, Cambridge, UK, 7-22.

McMichael, A.J., D.H. Campbell-Lendrum, C.F. Corvalán, K.L. Ebi, A.K. Githeko, J.D. Scheraga, and A. Woodward. *Climate Change and Human Health: Risks and Responses*. World Health Organization, Geneva. 322 pages.

Patz, J.A., D. Campbell-Lendrum, T. Holloway and J.A. Foley. 2005. Impact of regional climate change on human health. *Nature* 438:310-317.

Rojas, S., K. Richards, J.K. Jankovich and E.W. Davidson. 2005. Influence of temperature on Ranavirus infection in larval salamanders *Ambystoma tigrinum*. *Diseases of Aquatic Organisms* 63:95-100.

Wildlife Conservation Society. 2008. The Deadly Dozen. Available at: http://www.wcs.org/media/file/DEADLYdozen_screen.pdf

Chapter 1:

Atkinson, C.T., R.J. Dusek, K.L. Woods and W.M. Iko. 2000. Pathogenicity of avian malaria in experimentally-infected Hawaii amakihi. Journal of Wildlife Diseases 36(2):197-204.

Ball, G.H. and J. Chao. 1965. Temperature stresses on the mosquito phase of *Plasmodium relictum*. Journal of Parasitology 50(6):748-752.

Berger, A. J. 1981. *Hawaiian Birdlife*. Honolulu: University Press of Hawaii.

Chao, J. and G.H. Ball. 1962. The effect of low temperature on *Plasmodium relictum* in *Culex tarsalis*. *Journal of Parasitology* 48:252-254.

Eldridge, B.F. 1968. The effect of temperature and photoperiod on blood-feeding and ovarian development in mosquitoes of the *Culex Pipiens* complex. *American Journal of Tropical Medicine and Hygiene* 17(1):133-140.

Foster, J.T., B.L. Woodworth, L.E. Eggert, P.J. Hart, D. Palmer, D.C. Duffy and R.C. Fleischer. 2007. Genetic structure and evolved malaria resistance in Hawaiian honeycreepers. *Molecular Ecology* 16(22):4738-4746.

Freed, L.A., R.L. Cann, M.L. Goff, W.A. Kuntz and G.R. Bodner. 2005. Increase in avian malaria at upper elevation in Hawaii. *The Condor* 107:753-764.

Henshaw, H.W. 1902. *Birds of the Hawaiian Islands; being a complete list of the birds of the Hawiaan possessions, with notes on their habits*. Thos. G. Thrum, Honolulu.

Jacobi, J.D. and C.T. Atkinson. 1995. Hawaii's Endemic Birds. Pp. 376-381. In LaRoe, E.T., Farris, G.S., Puckett, C.E., Coran, P.D., and Mac M.J. (eds.) *Our Living Resources: A Report to the Nation on the Distribution, Abundance and Health of U.S. Plants, Animals and Ecosystems*. U.S. Department of Interior. National Biological Service. Washington, DC 530 pp.

Kilpatrick, A.M. 2005 Facilitating the evolution of resistance to avian malaria in Hawaiian birds. *Biological Conservation* 128(4):475-485.

LaPointe, D., T.L. Benning and C. Atkinson. 2005. Avian malaria, climate change and the native birds of Hawaii. Pp. 317- 321 In Lovejoy, T.E. and L. Hannah (eds). *Climate Change and Biodiversity*. New Haven, CT: Yale University Press. 418 pp.

Mottram, P., B.H. Kay and D.S. Kettle. 1986. The effect of temperature on eggs and immature stages of *Culex annulirostris* Skuse (Diptera: Culicidae). *Australian Journal of Entomology* 25 (2):131–136.

Munro, G.C. 1944. Birds of Hawaii. Tongg Publishing Company, Oahu.

Olson, S.L. and H.F. James. 1982. Fossil birds from the Hawaiian Islands: Evidence for wholesale extinctions by man before Western contact. *Science* 217:633-635.

Reiter, M.E. and D.A. LaPointe. 2007. Landscape factors influencing the spatial distribution and abundance of mosquito vector *Culex quinquefasciatus* (Diptera: Culicidae) in a mixed residential-agricultural community in Hawaii. *Journal of Medical Entomology* 44(5):861-868.

Ribiero, J.M.C., P.A. Rossignol and A. Spielman. 1984. Role of mosquito saliva in blood vessel location. *Journal of Experimental Biology* 108:1-7.

Scholte, E-J., B.N. Njiru, R.C. Smallegange, W. Takken and B.G.J. Knols. 2003. Infection of malaria (*Anopheles gambiae s.s.*) and filariasis (*Culex quinquefasciatus*) vectors with the entomopathogenic fungus *Metarhizium anisopliae*. *Malaria Journal* 2:29. Open Access Journal available at: http://www.malariajournal.com/content/2/1/29 (Accessed 19 June 2008).

Snow, R.W. A. Ikoku, J. Omumbo & J. Ouma. Undated. The epidemiology, politics and control of malaria epidemics in Kenya: 1900-1998. Available at: http://www.who.int/malaria/docs/ek_report2.htm#p22 (Accessed 19 June 2008).

Stratman-Thomas, W.K. 1940. The influence of temperature on *Plasmodium vivax*. *American Journal of Tropical Medicine* 20:703-715.

U.S. Agency for International Development. Malaria Programs Page. http://www.usaid.gov/our_work/global_health/mch/ch/techareas/malaria_brief.html (accessed 13 June 2008).

U.S. Fish and Wildlife Service. 2006. Revised Recovery Plan for Hawaiian Forest Birds. Region 1, Portland, OR. 622 pp. Available at http://ecos.fws.gov/docs/recovery_plan/060922a.pdf (Accessed 26 June 2008).

Van Dine, D.L. 1904. Mosquitoes in Hawaii. Hawaii Agricultural Experiment Station, Bulletin No. 6. Available at http://www.mosquitocatalog.org/pdfs/135320-0.PDF (accessed 23 June 2008).

Van Riper III, C., S.G. van Riper, M..L. Goff and M. Laird. 1986. The epizootiology and ecological significance of malaria in Hawaiian land birds. *Ecological Mongraphs* 56(4):327-344.

Warner, R.E. 1968. The role of introduced diseases in the extinction of the endemic Hawaiian avifauna. *The Condor* 70:1101-1120.

Woodworth, B.L., C.T. Atkinson, D.A. LaPointe, P.J. Hart, C.S. Spiegel, E.J. Tweed, C. Henneman, J. LeBrun, T. Denette, R. DeMots, K.L. Kozar, D. Triglia, D. Lease, A. Gregor and D. Duffy. 2005. Host population persistence in the face of introduced vector-borne disease: Hawaii amakihi and avian malaria. *Proceedings of the National Academy of Sciences* 102(5):1531-1536.

World Health Organization. Malaria. 2007. Fact Sheet Number 94. Available at: http://www.who.int/mediacentre/factsheets/fs094/en/index.html (accessed 16 Jun 2008).

Chapter 2.

Anderson, J., A. Main, T. Andreadis, S. Wikel, and C. Vossbrinck. 2003. Transstadial transfer of West Nile virus by three species of ixodid ticks (Acari: Ixodidae). *American Journal of Tropical Medicine and Hygiene* 48:726-738.

Bailey, C.L., M.E. Faran, T.P. Gargan II, and D.E. Hayes. 1982. Winter survival of blood fed and non-blood-fed *Culex pipiens*. *American Journal of Tropical Medicine and Hygiene* 31:1054-1061.

Braun, C.E. 1998. Sage grouse declines in western North America: What are the problems? *Proceedings of the Western Association of State Fish and Wildlife Agencies* 78:139-156.

Brault AC, Langevin SA, Bowen RA, Panella NA, Biggerstaff BJ, Miller BR, and N. Komar. 2004. Differential virulence of West Nile strains for American Crows. *Emerging Infectious Diseases* 10:2161-2168.

Bugbee, L.M. and L.R. Forte. 2004. The discovery of West Nile virus in overwintering *Culex pipiens* (Diptera:Culicidae) mosquitoes in Lehigh County, Pennsylvania. *Journal of the American Mosquito Control Association* 20(3):326-7.

Caffrey, C., S.C.R. Smith and T.J. Weston. 2005. West Nile virus devastates an American crow population. *The Condor* 107(1):128-132.

Centers for Disease Control & Prevention (CDC). 2008. West Nile virus activity- United States, 2007. *Morbidity and Mortality Weekly Report* 57(26):720-723 (July 4, 2008).

Clark, L., J. Hall, R. McLean, M. Dunbar, K. Klenk, R. Bowen and C.A. Smeraski. 2006. Susceptibility of greater sage-grouse to experimental infection with West Nile virus. *Journal of Wildlife Diseases* 42(1): 14-22.

Connelly, J.W., S.T. Knick, M.A. Schroeder, and S.J. Stiver. 2004. *Conservation Assessment of Greater Sage-grouse and Sagebrush Habitats*. Cheyenne, WY: Western Association of Fish and Wildlife Agencies.

Davis C.T., G.D. Ebel, R.S. Lanciotti, A.C. Brault, H. Guzman, et al. 2005. Phylogenetic analysis of North American West Nile virus isolates, 2001–2004: Evidence for the emergence of a dominant genotype. *Virology* 342: 252–265.

Davis, N.C. 1932. The effect of various temperatures in modifying the extrinsic incubation period of the yellow fever virus in *Äedes aegypti*. *American Journal of Epidemiology* 16:163-176.

Dohm, D.J., M.L. O'Guinn and M.J. Turell, 2002. Effect of environmental temperature on the ability of *Culex pipiens* (Diptera: Culicidae) to transmit West Nile virus. *Journal of Medical Entomology*, 39 (1): 221-225.

Dohm, D.J. and M.J. Turell. 2001. Effect of incubation at overwintering temperatures on the replication of West Nile virus in New York *Culex pipiens* (Diptera: Culicidae). *Journal of Medical Entomology* 38(3):462-464.

Ebel GD, Carricaburu J, Young D, Bernard KA, Kramer LD. 2004. Genetic and phenotypic variation of West Nile virus in New York, 2000–2003. *American Journal of Tropical Medicine and Hygiene* 71: 493–500.

Eldridge, B.F. 1968. The Effect of Temperature and Photoperiod on Blood-Feeding and Ovarian Development in Mosquitoes of the *Culex Pipiens* Complex. *American Journal of Tropical Medicine and Hygiene* 17(1): 133-140.

Epstein, P.R. and C. Defilippo. 2001. West Nile virus and drought. *Global Change and Human Health* 2(2):1-3.

Epstein, P.R. and E. Mills. 2005. *Climate Change Futures: Health, Ecological and Economic Dimensions*. Center for Health and the Global Environment, Harvard Medical School. http://www.climatechangefutures.org/pdf/CCF_Report_Final_10.27.pdf

Fitzgerald, S.D., J.S. Patterson, M. Kuipel, H. A. Simmons, S.D. Grimes, C.F. Sarver, R. M. Fulton, B.A. Steficek, T.M. Cooley, J. P. Massey and J.G. Sikarskie. 2003. Clinical and pathologic

features of West Nile virus infection in native North American owls (Family Strigidae). *Avian Diseases* 47:602-610.

Fujisaki, I., E.V. Pearlstine and M. Miller. Detecting population declines of birds using long-term monitoring data. *Population Ecology* 50(3):275-284.

Gwong-Jen, J.C., B.S. Davis, S. Stringfield, and C. Lutz. 2007. Prospective immunization of the endangered California condor (*Gymnogyps californianus*) protects this species from lethal West Nile virus infection. *Vaccine* 25(12):2325-2330.

Hubálek Z. and J. Halouzka. 1999. West Nile Fever–a Reemerging Mosquito-Borne Viral Disease in Europe. *Emerging Infectious Diseases* 5(5):643-650.

Hull, J., A.. Hull, W. Reisen, Y. Fang, and H. Ernest. 2006. Variation of West Nile virus antibody prevalence in migrating and wintering hawks in central California. *The Condor* 108(2):435-439.

Intergovernmental Panel on Climate Change. 2007. Summary for Policymakers. In: *Climate Change 2007: The Physical Science Basis*. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change [Solomon, S., D. Qin, M. Manning, Z. Chen, M. Marquis, K.B. Averyt, M.Tignor and H.L. Miller (eds.)]. Cambridge University Press, Cambridge, United Kingdom and New York, NY, USA.

Kilpatrick, A.M., Y. Gluzberg, J. Burgett and P. Daszak. 2004. Quantitative risk assessment of the pathways by which West Nile virus could reach Hawaii. *Ecohealth* 1:205-209.

Kilpatrick, A.M.., P. Daszak, S.J. Goodman, H. Rogg, L.D. Kramer, V. Cedeño and A.A. Cunningham. 2006. Predicting pathogen introduction: West Nile virus spread to Galápagos. *Conservation Biology* 20(4): 1224-1231.

Kilpatrick. A. M., S.L. LaDeau, and P.P. Marra. 2007. Ecology of West Nile virus transmission and its impact on birds in the Western Hemisphere. *The Auk* 124(4):1121-1136.

Kilpatrick AM, Meola MA, Moudy RM, Kramer LD (2008) Temperature, viral genetics, and the transmission of West Nile Virus by *Culex pipiens* mosquitoes. *PLoS Pathology* 4(6): e1000092.

Komar, N. 2003. West Nile virus: Epidemiology and ecology in North America. *Advances in Virus Research* 61: 185-234.

Kunkel, K.E., R.J. Novak, R.L. Lampman and W. Gu. 2006. Modeling the impact of variable climatic factors on the crossover of Culex restuans and Culex pipiens (Diptera: Culicidae), vectors of West Nile virus in Illinois. *American Journal of Tropical Medicine and Hygiene* 74(1): 168-173.

Lanciotti R.S., J.T. Roehrig, V. Deubel, J. Smith, M. Parker, K. Steele, B. Crise, K.E. Volpe, M.B. Crabtree, J.H. Scherret, R.A. Hal, J.S. MacKenzie, C.B. Cropp, B. Panigrahy, E. Ostlund, B. Schmitt, M. Malkinson, C. Banet, J. Weissman, N. Komar, H.M. Savage, W. Stone, T. McNamara, and D.J. Gubler. 1999. Origin of the West Nile virus responsible for and outbreak of encephalitis in the northeastern United States. *Science* 286: 2333-2337.

Marra, Peter P., S. Griffing, C. Caffrey, A. M. Kilpatrick, R. McLean, C. Brand, E. Saito, A.P. Dupuis, L. Kramer and R. Novak. 2004. West Nile virus and wildlife. *BioScience* 393 54(5): 393-402.

McLean, R.G. West Nile Virus in North American birds. *Ornithological Monographs* 60(1):44-64.

Miller, D. M. Mauel. C. Baldwin, G. Burtle, D. Ingram, M. Hines and K. Frazier. 2003. West Nile virus in farmed alligators. *Emerging Infectious Diseases* 9:794-794-799.

Mottram, P., B. H. Kay and D. S. Kettle. 1986. The effect of temperature on eggs and immature stages of *Culex annulirostris* Skuse (Diptera: Culicidae). *Australian Journal of Entomology* 25 (2): 131–136.

Moudy RM, Meola MA, Morin LL, Ebel GD, Kramer LD. 2007. A newly emergent genotype of West Nile virus is transmitted earlier and more efficiently by *Culex* mosquitoes. *American Journal of Tropical Medicine and Hygiene* 77: 365–370.

Nash, D., F. Mostashari, A. Fine, J. Miller, D. O'Leary, K. Murray, A. Huang, A. Rosenberg, A. Greenberg, M. Sherman S. Wong, and M. Layton. 2001. The outbreak of West Nile virus infection in the New York City area in 1999. *The New England Journal of Medicine* 344(24): 1807-1814.

Naugle D.E, C.L. Aldridge, B.L. Walker, T.E. Cornish, B.J. Moynahan, M.J. Holloran, K. Brown, G.D. Johnson, E.T. Schmidtmann, R.T. Mayer, C.Y. Kato, M.R. Matchett, T.J. Christiansen, W.E. Cook, T. Creekmore, R.D. Falise, E.T. Rinkes and M.S. Boyce. 2004. West Nile virus: Pending crisis for greater sage-grouse. *Ecology Letters* 7(8): 704-713.

Naugle, D.E., C.L. Aldridge, B.L. Walker, K.E. Doherty, M.R. Matchett, J. McIntosh, T.E. Cornish, and M.S. Boyce. 2005. West Nile virus and sage-grouse: What more have we learned? *Wildlife Society Bulletin* 33(2):616-623.

Petersen, L.R. and A.A. Marfin. 2002. West Nile virus: A primer for the clinician. *Annals of Internal Medicine* 137(3): E173-E179.

Pyke, D.A. 1999. Invasive exotic plants in the sagebrush ecosystems of the Intermountain West. Pp. 43-62 In Entwhistle, P.G., A.M. Debolt, J.H. Kaltenecker and K. Steenhof, compilers. *Proceedings: Sagebrush Steppes Ecosystems Symposium*. USDI BLM Publication NO. BLM/ID/PT-001001+1150.

Ribiero, J.M.C., P.A. Rossignol and A. Spielman. 1984. Role of mosquito saliva in blood vessel location. *Journal of Experimental Biology* 108:1-7.

Sabio, I.J. 2005. *Seasonal Abundance and Detection of West Nile Virus in Ceratopogonids (Diptera:Ceratopogonidae) in East Baton Rouge Parish, Louisiana*. Master's Thesis, Louisiana State University. http://etd.lsu.edu/docs/available/etd-11032005-144604/unrestricted/Sabio_thesis.pdf

Smithburn KC, Hughes TP, Burke AW, Paul JH. 1940. A neurotropic virus isolated from the blood of a native of Uganda. *American Journal of Tropical Medicine* 20:471-92.

Snapinn KW, Holmes EC, Young DS, Bernard KA, Kramer LD, et al. 2007. Declining growth rate of West Nile virus in North America. *Journal of Virology* 81: 2531–2534.

Sovada, M.A., P.J. Pietz, K.A. Converse, D.T. King, E.K. Hofmiester, P. Scherr and H.S. Ip. 2008. Impact of West Nile virus and other mortality factors on American white pelicans at breeding colonies in the northern plains of North America. *Biological Conservation* 141:1021-1031.

Walker, B.L., D.E. Naugle, K.E. Doherty and T.E. Cornish. 2004. From the field: Outbreak of West Nile virus in greater sage-grouse and guidelines for monitoring, handling and submitting dead birds. *Wildlife Society Bulletin* 2(3): 1000-1006.

Walker, B.L., D.E. Naugle, and K.E. Doherty. 2007. Greater sage-grouse population response to energy development and habitat loss. *Journal of Wildlife Management* 71(8):2644-2654.

West, N.E. 1999. Synecology and disturbance regimes of sagebrush steppe ecosystems. Pp. 15-26 In Entwhistle, P.G., A.M. Debolt, J.H. Kaltenecker and K. Steenhof, compilers. *Proceedings: Sagebrush Steppes Ecosystems Symposium*. USDI BLM Publication NO. BLM/ID/PT-001001+1150.

Wheeler, S.S., C.M. Barker, Y. Fang, M.V. Armijos, B.D. Carroll, S. Husted, W.O. Johnson and W.K. Reisen. 2009. Differential impact of West Nile Virus on California birds. *The Condor* 111(1):1-20.

Whitman, L. 1937. The multiplication of the virus of yellow fever in *Äedes aegypti*. *Journal of Experimental Medicine* 66:133-143.

Zou, L., S.N. Miller, and E.T. Schmidtmann. 2006. Mosquito larval habitat mapping using remote sensing and GIS: Implications of coalbed methane development and West Nile virus. *Journal of Medical Entomology* 43(5):1034-1041.

Chapter 3

Alford, R.A., K.S. Bradfield, and S.J. Richards. 2007. Global warming and amphibian losses. *Nature* 447:E3-E4.

Bancroft, B.A., N.J. Baker and A.R. Blaustein. 2008. A meta-analysis of the effects of ultaviolet-B radiation and its synergistic interactions with pH, contaminents and disease on amphibian survival. *Conservation Biology* 22(4):987-996.

Barrionuevo, S. and S. Mangione. 2006. Chytridiomycosis in two species of *Telmatobius* (Anura: Leptodactylidae) from Argentina. *Diseases of Aquatic Organisms* 73:171-174.

Berger, L., A.D. Hyatt, R. Speare and J.E. Longcore. 2005. Life cycle stages of the amphibian chytrid *Bactrachochytrium dendrobatidis*. *Diseases of Aquatic Organisms* 68: 51-63.

Berger, L. R. Speare, P. Daszak, D.E. Green, A.A. Cunningham, C.L. Goggin, R. Slocombe, M.A. Ragan, A.D. Hyatt, K.R. McDonald, H. B. Hines, K.R. Lips, G. Marantelli and H. Parkes. 1998. Chytridiomycosis causes amphibian mortality associated with population declines in the rain forests of Australia and Central America. *Proceedings of the National Academy of Sciences* USA 95:9031-9036.

Berger, L. R. Speare, H.B. Hines, G. Marantelli, A.D. Hyatt, K.R. Mcdonald, L.F. Skerratt, V. Olsen, J.M. Clarke, G. Gillespie, M. Mahony, N. Sheppard, C. Williams, and M.J. Tyler. 2004. Effect of season and temperature on mortality in amphibians due to chytridiomycosis. *Australian Veterinary Journal* 82(7):31-36.

Bonaccorso, E., J.M. Guayasamin, D. Méndez and R. Speare. 2003. Chytridiomycosis as a possible cause of population decline of *Atelopus cruciger* (Anura:Bufonidae). *Herpetological Review* 34(4):331-334.

Bosch, J., I. Martínez-Solano, and M. García-París. 2001. Evidence of a chytrid fungus infection involved in the decline of the common midwife toad (*Alytes obstetricans*) in protected areas of Central Spain. *Biological Conservation* 97:331–337.

Bosch, J., L.M. Carrascal, L. Duran, S.Walker, and M.C. Fisher. 2007. Climate change and outbreaks of amphibian chytridiomycosis in a montane area of Central Spain; is there a link? *Proceedings of the Royal Society* 274:253-260.

Bradley, G.A., P.C. Rosen, M.J. Sredl, T.R. Jones, and J.E. Longcore. 2002. Chytridiomycosis in native Arizona frogs. *Journal of Wildlife Diseases* 38:206–212.

Briggs, C.J., V.T. Vredenburg, R.A. Knapp, and L.A. Rachowicz. 2005. Investigating the population-level effects of chytridiomycosis, an emerging infectious disease of amphibians. *Ecology* 86(12):3149-3159.

Burrowes, P. A., R. L. Joglar, and D.E. Green. 2004. Potential causes for amphibian declines in Puerto Rico. *Herpetologica* 60(2):141–154.

Collins, J.P. and A. Storfer. 2003. Global amphibian declines: sorting the hypotheses. *Diversity and Distributions* 9:89-98.

Corn, P. S. 2007. Amphibians and disease: Implications for conservation in the Greater Yellowstone Ecosystem. *Yellowstone Science* 15(2):11-16.

Cummer, M.R., D.E. Green, and E.M. O'Neill. 2005. Aquatic chytrid pathogen detected in terrestrial plethodontid salamander. *Herpetological Review* 36(3):249-249.

Davidson, E.W., M. Parris, J.P. Collins, J.E. Longcore, A.P. Pessier, and J. Brunner. 2003. Pathogenicity and transmission of chytridiomycosis in tiger salamanders (*Ambystoma tigrinum*). *Copeia* 2003(3):601–607.

Díaz, L.M., A. Cádiz, A. Chong, and A. Silva. 2007. First report of chytridiomycosis in a dying toad (Anura: Bufonidae) in Cuba: A new conservation challenge for the island. Ecohealth: DOI:10.1007/s10393-007-0094-4

Cunningham, A.A., T.W.J. Garner, V. Aguilar-Sanchez, B. Banks, J. Foster, A.W. Sainsbury, M. Perkins, S.F. Walker, A.D Hyatt and M.C. Fisher. 2005. Emergence of amphibian chytridiomycosis in Britain. *Veterinary Record* 157:386-387.

Department of Environment and Heritage (Australia). 2006. Background Document for the Threat Abatement Plan: Infection of Amphibians with Chytrid Fungus Resulting in Chytridiomycosis. Available at: www.deh.gov.au/biodiversity/threatened/publications/tap/chytrid/ (accessed 21 August 2008).

Drew, A., E.J. Allen, and L.J.S. Allen. 2006. Analysis of climatic and geographic factors affecting the presence of chytridiomycosis in Australia. *Diseases of Aquatic Organisms* 68:245-50.

Eterovick, P.C., A.C. Oliviera de Queiroz Carnaval, D.M. Borges-Nojosa, D.L. Silvano, M.V. Segalla, and I. Sazima. 2005. Amphibian declines in Brazil: An Overview. *Biotropica* 37(2):166-179.

Felger, J., J. Enssle, D. Mendez and R. Speare. 2007. Chytridiomycosis in El Salvador. *Salamandra* 43(2):122-127.

Fellers, G.M., D.F. Bradford, D.Pratt, and L.L. Wood. 2007. Demise of repatriated populations of mountain yellow-legged frogs (*Rana mucosa*) in the Sierra Nevada of California. *Herpetological Conservation and Biology* 2(1):5-21.

Fisher, M.C. and T.W.J. Garner. 2007. The relationship between the emergence of *Batrachochytrium dendrobatidis*, the international trade in amphibians, and introduced amphibian species. *Fungal Biology Reviews* 21:2-9.

Garner, T.W.J., S. Walker, J. Bosch, A.D. Hyatt, A.A. Cunningham, and M.C. Fisher. 2005. Chytrid fungus in Europe. *Emerging Infectious Diseases* 11(10):1639-1641.

Global Amphibian Assessment. 2004. www.globalamphibians.org/summary. Accessed 14 Aug 2008.

Green, D.E., K.A. Converse, and A.K. Schrader. 2002. Epizootiology of sixty-four amphibian morbidity and mortality events in the U.S.A., 1996-2001. *Annals of the New York Academy of Sciences* 969:323-339.

Green, D.E. and C.K. Sherman. 2001. Diagnostic histological findings in Yosemite toads (*Bufo canorus*) from a die-off in the 1970s. *Journal of Herpetology* 35(1):92-103.

Hanselmann, R., A. Rodríguez, M. Lampo, L. Fajardo-Ramos, A.A. Aguirre, A.M. Kilpatrick, J.P. Rodríguez, and P. Daszak. Presence of an emerging pathogen of amphibians in introduced bullfrogs *Rana catesbiana* in Venezuela. *Biological Conservation* 120:115-119.

IPCC. 2007. Summary for Policymakers. In: Climate Change 2007: Impacts, Adaptation and Vulnerability. Contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change, M.L. Parry, O.F. Canziani, J.P. Palutikof, P.J. van der Linden and C.E. Hanson, Eds., Cambridge University Press, Cambridge, UK, 7-22.

Johnson, M. L., and R. Speare. 2005. Possible modes of dissemination of the amphibian chytrid *Batrachochytrium dendrobatidis* in the environment. *Diseases of Aquatic Organisms* 65:181–186.

Kriger, K.M. and J.-M. Hero. 2006. Large-scale seasonal variation in the prevalence and severity of chytridiomycosis. *Journal of Zoology* doi:10.1111/j.1469-7998.2006.00220.x.

Kriger, K.M. and J.-M. Hero. 2007. The chytrid fungus *Batrachochytrium dendrobatidis* is non-randomly distributed across amphibian breeding habitats. *Diversity and Distributions* DOI: 10.1111/j.1472-4642.2007.00394.x.

La Marca, E., K.R. Lips, S. Lötters, R. Puschendorf, R. Ibáñez, J.V. Rueda-Almonacid, R. Schulte, C. Marty, F. Castro, J. Manzanilla-Puppo, J.E. García-Pérez, F. Bolaños, G. Chaves , J.A. Pounds, E. Toral, and B.E. Young. 2005. Catastrophic population declines and extinctions in neotropical harlequin frogs (Bufonidae: *Atelopus*). *Biotropica* 37(2):190-201

Lampo, M., A. Rodríguez-contreras, E. La Marca and P. Daszak. 2006. A chytridiomycosis epidemic and a severe dry season precede the disappearance of *Atelopus* species from the Venezuelan Andes. *The Herpetological Journal* 16(4):395-402.

Lips, K.R. 1998. Decline of a tropical montane amphibian fauna. *Conservation Biology* 12(1):1-13.

Lips, K.R. 1999. Mass mortality and population declines of anurans at an upland site in western Panama. *Conservation Biology* 13(1):117-125.

Longcore, J.R., J.E. Longcore, A.P. Pessier, and W.A. Halteman. 2007. Chytridiomycosis widespread in anurans of Northeastern United States. *Journal of Wildlife Management* 71(2):435-444.

Longcore, J.E., A.P. Pessier, and D.K. Nichols. 1999. *Batrachochytrium dendrobatidis* gen. et sp. nov., a chytrid pathogenic to amphibians. *Mycologia* 91:219-227.

Mendelson, J.R. III, E.D. Brodie, Jr., J.H. Malone, M.E. Acevedo, M.A. Baker, N.J.
Smatresk & J.A. Campbell. 2004. Factors associated with the catastrophic decline of a cloudforest frog fauna in Guatemala. *Revista de Biologia Tropical* (*International Journal of Tropical Biology and Conservation*):52(4):991-1000.

Merino-Viteri, A., L.A. Coloma and A. Almendáriz. 2005. Los Telmatobius de los Andes de Ecuador y su disminución poblacional. In: Lavilla EO, De la Riva I (eds) Studies on the Andean frogs of the genera Telmatobius and Batrachophrynus. Monografías de Herpetología 7, Asociación
Herpetológica Española, Valencia, p 9–37.

Mutschmann, F., L. Berger, P. Zwart and C. Gaedicke. 2000. Chytridiomycosis in amphibians—first report from Europe. *Berl. Münch. Tierärztl. Wochenschr.* 113(10):380-383. [In German, summary in English]

Ouellet, M., I. Mikaelian, B.D. Pauli, J. Rodrigue, and D.M. Green. 2005. Historical evidence of widespread chytrid infection in North American amphibian populations. *Conservation Biology* 19(5):1431-1440.

Pough, F. H., T. Taigen, M. Stewart, and P. Brussard. 1983. Behavioral modification of evaporative water loss by a Puerto Rican frog. *Ecology* 64:244–252.

Pounds, J.A., M.R. Bustamante, L.A. Coloma, J.A. Consuegra, M.P.L. Fogden, P.N. Foster, E. La Marca, K.L. Masters, A. Merinio-Viteri, R. Puschendorf, S.R. Fon, G.A. Sánchez-Asofeifa, C.J. Still and B.E. Young. 2006. Widespread amphibian extinctions from epidemic disease driven by global warming. *Nature* 439:161-167.

Pounds, J.A. and M.L. Crump. 1994. Amphibian declines and climate disturbance; the case of the golden toad and the harlequin frog. *Conservation Biology* 8(1):72-85.

Pounds, J.A., M.P.L. Fogden, J.M. Savage, and G.C. Gorman. 1997. Tests of null models for amphibian declines on a tropical mountain. *Conservation Biology* 11(6):1307-1322.

Puschendorf, R. F. Bolaños, and G. Chaves. 2006a. The amphibian chytrid fungus along an altitudinal transect before the first reported declines in Costa Rica. *Biological Conservation* 132(1):136-142.

Puschendorf, R.F., F. Castañeda, and J.R. McCranie. 2006. Chytridiomycosis in wild frogs from Pico Bonito National Park, Honduras. *EcoHealth* DOI: 10.1007/s10393-006-0026-8.

Ron, S.R., W.E. Duellman, L.A. Coloma, and M.R. Bustamante. 2003. Population decline of the Jambato toad *Atelopus ignescens* (Anura:Bufonidae) in the Andes of Ecuador. *Journal of Herpetology* 37(1):116-126.

Schloegel, L.M., J.-M. Hero, L.Berger, R. Speare, K. Macdonald and P. Daszak. 2006. The decline of the sharp-snouted day frog (*Taudactylus acutirostris*): The first documented case of extinction by infection in a free-ranging wildlife species? *EcoHealth* 3:35-40.

Schloegel, L.M., A.M. Picco, A.M. Kilpatrick, A.J. Davies, A.D. Hyatt and P. Daszak. 2009. Magnitude of the U.S. trade in amphibians and presence of *Batrachochytrium dendrobatidis* and ranavirus infection in imported North American bullfrogs (*Rana catesbiana*). Biological Conservation *in press*.

Seimon, T.A., G. Hoernig, P. Sowell, S. Halloy, and A. Seimon. (2005). Identification of chytridiomycosis in Telmatobius marmoratus at 4450 m in the Cordillera Vilcanto of Southern Peru. In: Lavilla EO, De la Riva I (eds) Studies on the Andean frogs of the genera Telmatobius and Batrachophrynus. Monografías de Herpetología 7, Asociación Herpetológica Española, Valencia, p 273–281.

Shapiro, H.A. and H. Zwarenstein. 1934. A rapid test for pregnancy on *Xenopus laevis*. *Nature* 133:762.

Simoncelli, F., A. Fagotti, R. Dall'Olio, D. Vagnetti, R. Pascolini, and I. Di Rosa. 2005. Evidence of *Batrachochytrium dendrobatidis* in water frogs of the *Rana esculenta* complex in central Italy. *EcoHealth* 2:307-312.

Waldman, B., K.E. van de Wolfshaar, J.D. Klena, V. Andjic, P.J. Bishop, and R.J.B. Norman. 2001. Chytridiomycosis in New Zealand frogs. *Surveillance* 28(3):9-11.

Weldon, C., L.H. du Preez, A. D. Hyatt, R. Muller, and R. Speare. 2004. Origin of the amphibian chytrid fungus. *Emerging Infectious Diseases* 10(12):2100-2105.

Woodhams, D.C., R.A. Alford, and G. Marantelli. 2003. Emerging disease of amphibians cured by elevated body temperature. *Diseases of Aquatic Organisms* 55:65-67.

Young, S., L. Berger and R. Speare. 2007. Amphibian chytridiomycosis: strategies for captive management and conservation. *International Zoo Yearbook* 41:1-11.

Chapter 4

Adolf, J.E., T.R. Bachvaroff, D.N. Krupatkina, H. Nonogaki, P.J.P. Brown, A.J. Lewitus, H.R. Harvey and A.R. Place. 2006. Species specificity and potential roles of *Karlodinium micrum* toxin. *African Journal of Marine Sciences* 28(2):415-419.

Ahmed, F.E. (ed.) 1991. *Seafood Safety*. Washington DC: National Academy Press. 432 pages.

Asada, S., M. Koga, K. Nagao, M. Tabata, and K. Takeda. 2001. Okadaic acid remarkably suppresses testosterone production in murine Leydig cells. *Journal of Health Science* 41(1):60-64.

Baden, D.G. 1983. Marine food-borne dinoflagellate toxins. *International Review of Cytology* 82:99-150.

Bakun, A. 1990. Global climate change and intensification of coastal ocean upwelling. *Science* 247:198-201.

Baskin, Y. 2006. Sea sickness: The upsurge in marine diseases. *BioScience* 56(6):464-469.

Blankenship, K. 2007. Algal blooms plague Bay, rivers despite favorable dry conditions. *Bay Journal*, September.

Bolch, C.J.S. and M.F. de Salas. 2007. A review of the molecular evidence for ballast water introduction of the toxic dinoflagellates *Gymnodinium catenatum* and the *Alexandrium* "*tamarensis* complex" to Australia. *Harmful Algae* 6(4):465-485.

Bossart G.D., D.G. Baden, R.Y. Ewing, B. Roberts and S.D. Wright. 1998. Brevetoxicosis in manatees (*Trichechus manatus latirostris*) from the 1996 epizootic: gross, histologic, and immunohistochemical features. *Toxicologic Pathology* 26(2):276-82.

Brodie, E.C., F.M.D. Gulland, D.J. Greig, M. Hunter, J. Jaakola, J. St. Leger, T.A. Leighfield and F.M. Van Dolah. 2006. Domoic acid causes reproductive failure in California sea lions (*Zalophus californianus*). *Marine Mammal Science* 22(3):400-407.

Burkholder, J.M., E.J. Noga, C.W. Hobbs, H.B. Glasgow and S.A. Smith. 1992. New "phantom" dinoflagellate is the causative agent of major estuarine fish kills. *Nature* 358:407-410.

Burns, J., C. Williams and A. Chapman. 2002. Cyanobacteria and their toxins in Florida surface waters. Pp. 16-21 In D. Johnson & R.D. Harbison (eds). *Proceedings of Health Effects of Exposure to Cyanobacteria Toxins: State of the Science*. August 13-14 2002. Available at: http://www.doh.state.fl.us/environment/community/aquatic/pdfs/Cyanobacteria_200208.pdf [Accessed 29 December 2008].

Buzzelli, C.P., R.A. Luettich, Jr., S.P. Powers, C.H. Peterson, J.E. McNinch, J.L. Pinckney and H.W. Paerl. 2002. Estimating the spatial extent of bottom-water hypoxia and habitat degradation in a shallow-water estuary. *Marine Ecology Progress Series* 230:103-112.

Carpenter, S.R., N.F. Caraco, D.L. Correll, R.W. Howarth, A.N. Sharpley and V.H. Smith. 1998. Nonpoint pollution of surface waters with phosphorus and nitrogen. *Ecological Applications* 8(3):559-568.

Chorus, I. and J. Bartram. 1999. *Toxic Cyanobacteria in Water: A guide to their public health consequences, monitoring and management*. FN Spon. London, 400 pages. Available at: http://www.who.int/water_sanitation_health/resourcesquality/toxcyanobacteria.pdf (accessed 5 December 2008)

Coombs, A. 2008. Urea pollution turns tides toxic. *Nature News*, October 27.

Doucette, G.J., A.D. Cembella, J.L. Martin, J. Michaud, T.V.N. Cole and R.M. Rolland. 2006. Paralytic shellfish poisoning (PSP) toxins in North Atlantic right whales Eubalaena glacialis and their zooplankton prey in the Bay of Fundy, Canada. *Marine Ecology Progress Series* 306:303-313.

Driscoll, C.P., P.C. McGowan, E.A. Miller and W.W. Carmichael. 2002. Case Report: Great blue heron (Ardea Herodias) morbidity and mortality investigation in Maryland's Chesapeake Bay. *Proceedings of the Southeast Fish and Wildlife Conference*, Baltimore, MD, October 24, 2002 (poster).

Eastough J. and S. Sheperd. 1989. Infections and toxic syndromes from fish and shellfish consumption. *Archives of Internal Medicine* 149:1735–1739.

Farstad, D.J. and T. Chow. 2001. A brief case report and review of ciguatera poisoning. *Wilderness and Environmental Medicine* 12(4): 263–269.

Flewelling, L.J., J.P. Naar, J.P. Abbott, D.G. Baden, N.B. Barros, G.D. Bossart, M-Y. D. Bottein, D.G. Hammond, E.M. Haubold, C.A.. Heil, M.S. Henry, H.M. Jacocks, T.A. Leighfield, R.H. Pierce, T.D. Pitchford, S.A. Rommel, P.S. Scott, K.A. Steidinger E.W. Truby, F.M. Van

Dolah and J.H. Landsberg. 2005. Red tides and marine mammal mortalities. *Nature* 435:755-756.

Gaydos, J.K. 2006. Bottlenose dolphins and brevetoxins: a coordinated research and response plan. NOAA Technical Memorandum NMFS-OPR-32. Available at: http://www.nmfs.noaa.gov/pr/pdfs/health/bottlenose_brevetoxins.pdf [Accessed 6 January 2009].

Geraci, J.R., D.M. Anderson, R.J. Timperi, D.J. St. Aubin, G.A. Early, J.H Prescott, and C.A. Mayo. 1989. Humpback whales (*Megaptera novaeangliae*) poisoned by dinoflagellate toxin. *Canadian Journal of Fisheries and Aquatic Science* 46:1895-98.

Gilbert, P.M., D.M. Anderson, P. Gentien, E. Granéli, and K.G. Sellner. 2005. The global, complex phenomena of harmful algal blooms. *Oceanography* 18(2):131-141.

Goldstein, T., T.S. Zabka, R.L. DeLong, E.A. Wheeler, G. Ylitalo, S. Bargu, M. Silver, T. Leighfield, F. Van Dolah, G. Langlois, I. Sidor, J.L. Dunn, and F.M.D. Gulland. 2009. The role of domoic acid in abortion and premature parturition of California sea lions (*Zalophus californianus*) on San Miguel Island, California. *Journal of Wildlife Diseases* 45(1):91-108.

Greer, J. 2007. Was There Another Killer? The Case for a Toxic Culprit. *Chesapeake Quarterly* 5(1):16-19.

Gulland, F.M.D. 2000. Domoic acid toxicity in California sea lions (*Zalophus californicus*) stranded along the central California coast May-October 1998. Report to the National Marine Fisheries Service Working Group on Unusual Marine Mortality Events. U.S. Department of Commerce, NOAA Technical Memo NMFS-OPR-17

Gulland F.M.D., M. Haulena, D. Fauquier, M. E. Lander, T. Zabka, R. Duerr, and G. Langlois. 2002. Domoic acid toxicity in California sea lions (*Zalophus californicus*): clinical signs, treatment and survival. *The Veterinary Record* 150(15):475-480.

Hayes, M.L., J. Bonaventura, T.P. Mitchell, J.M. Prospero, E.A. Shinn, F. Van Dolah and R.T. Barber. 2001. How are climate and marine biological outbreaks functionally linked? *Hydrobiologia* 460:213-220.

Hudnell, H.K. 2005. Chronic biotoxin-associated illness: multiple-system symptoms, a vision deficit, and effective treatment. *Neurotoxicology and Teratology* 27(5):733-43.

Huin, N. 2007. Falkland Islands Penguin Census. Falklands Conservation, 31 pp. Available at http://www.falklandsconservation.com/wildlife/birds/PenguinCensusReport05-06.pdf [Accessed 13 July 2009].

Intergovernmental Panel on Climate Change. 2007: Summary for Policymakers. In: *Climate Change 2007: Impacts, Adaptation and Vulnerability*. Contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change, M.L. Parry, O.F. Canziani, J.P. Palutikof, P.J. van der Linden and C.E. Hanson, Eds., Cambridge University Press, Cambridge, UK, 7-22.

International Bird Rescue Research Center (2007). Algae Bloom Kills Sea Birds, Other Sea Life In Southern California In Record Numbers. *ScienceDaily*, April 28. Available at http://www.sciencedaily.com /releases/2007/04/070427084149.htm [Accessed December 31, 2008].

Jackson, J. B. C., M. X. Kirby, W.H. Berger, K.A. Bjorndal, L.W. Botsford, B.J. Bourque, R.H. Bradbury, R. Cooke, J. Erlandson, J.A. Estes, T.P. Hughes, S. Kidwell, C.B. Lange, H.S. Lenihan, J.M. Pandolfi, C.H. Peterson, R.S. Steneck, M.J. Tegner and R.R. Warner. 2001. Historical Overfishing and the Recent Collapse of Coastal Ecosystems. *Science* 293 (5530): 629-637.

Jessup, D.A., M.A. Miller, J.P. Ryan, H.M. Nevins, H. A. Kerkering, A.Mekebri, D.B. Crane, T. A. Johnson and R.M. Kudela. 2009. Mass stranding of marine birds caused by a surfactant-producing red tide. *PLoS One* 4(2): e4550. doi:10.1371/journal.pone.0004550.

Jöhnk, K.D., J. Huisman, J. Sharples, B. Sommeijer, P.M. Visser and J.M. Stroom. 2008. Summer heatwaves promote blooms of harmful cyanobacteria. *Global Change Biology* 14(3):495-512.

Knoll, A.H. 2008. Cyanobacteria and earth history. Pages 1-21 In: A. Herrero and E. Flores (eds). *The Cyanobacteria: Molecular Biology, Genetics and Evolution*. Caister Academic Press; Norfolk, UK. 484 pp.

Kotut, K., A. Ballot and L. Krienitz. 2006. Toxic cyanobacteria and their toxins in standing water in Kenya: Implications for water resource use. *Journal of Water and Health* 4(2):233-245.

Kreuder, C., M. A. Miller, D. A. Jessup, L. J. Lowenstine, M. D. Harris, J. A. Ames,
T. E. Carpenter, P. A. Conrad, and J. A. K. Mazet. 2003. Patterns of mortality in southern sea otters (*Enhydra lutris nereis*) from 1998 to 2001. *Journal of Wildlife Diseases* 39(3):495-509.

Kreuder, C., M. A. Miller, L. J. Lowenstine, P. A. Conrad, T. E. Carpenter, D. A. Jessup, and J. A. K. Mazet. 2005. Evaluation of cardiac lesions and risk factors associated with myocarditis and dilated cardiomyopathy in southern sea otters (*Enhydra lutris nereis*). *American Journal of Veterinary Research* 66(2):289-299.

Landsberg, J.H., S. Hall, J.N. Johannessen, K.D. White, S.M. Conrad, J.P. Abbott, L.J. Flewelling, R.W. Richardson, R.W. Dickey, E.L.E. Jester, S.M. Etheridge, J.R. Deeds, F.M. Van Dolah, T. A. Leighfield, Y.Zou, C.G. Beaudry, R.A. Benner, P.L. Rogers, P.S. Scott, K.Kawabata, J.L. Wolny, and K.A. Steidinger. 2006. Saxitoxin Puffer Fish Poisoning in the United States, with the First Report of *Pyrodinium bahamense* as the Putative Toxin Source. *Environmental Health Perspectives* 114(10): 1502–1507.

Lefebvre , K.A., C.L. Powell, M. Busman, G.J. Doucette, P.D.R. Moeller , J.B. Silver, P.E. Miller, M. P. Hughes, S. Singaram, M.W. Silver and R.S. Tjeerdema. 1999. Detection of domoic acid in northern anchovies and California sea lions associated with an unusual mortality event. *Natural Toxins* 7(3):85-92.

Magana, H.A. and T.A. Villareal. 2006. The effect of environmental factors on the growth rate of Karenia brevis (Davis) G. Hansen and moestrup. *Harmful Algae* 5(2):192-198.

Martin, J.H. and S.E. Fitzwater. 1988. Iron deficiency limits phytoplankton growth in the northeast Pacific subarctic. *Nature* 331:341-343.

Marchetti, A., N. Lundholm, Y. Kotaki, K. Hubbard, P.J. Harrison and E.V. Armbrust. 2008. Identification and assessment of domoic acid production in oceanic *pseudo-nitzschia* (bacillariophyceae) from iron-limited waters in the northeast subarctic Pacific. *Journal of Phycology* 44:650-661.

Mase, B., W. Jones, R. Ewing, G. Bossart, F. VanDolah, T. Leighfield, M. Busman, J. Litz, B. Roberts, T. Rowles. 2000. Epizootic in bottlenose dolphins in the Florida panhandle: 1999-2000. In *Proceedings of the American Association of Zoo Veterinarians and the International Association of Aquatic Animal Medicine* (Baer ed.). New Orleans, LA 2000:522-525.

Maucher, J.M. and J.S. Ramsdell. 2005. Domoic acid transfer to milk: Evaluation of a potential route of neonatal exposure. *Environmental Health Perspectives* 113(4)461-464.

McMinn, A. 1989. Late Pleistocene dinoflagellate cysts from Botany Bay, New South Wales, Australia. *Micropaleontology* 35:1-9.

Moeller, P.D.R., K.R. Beauchesne, K.M. Huncik, W.C. Davis, S.J. Christopher, P. Riggs-Gelasco, and A.K. Gelasco. 2007. Metal complexes and free radical toxins produced by *Pfiesteria piscicida*. *Environmental Science and Technology* 41(4): 1166-1172

Moore, S.K., V.L. Trainer, N.J. Mantua, M.S. Parker, E.A. Laws, L.C. Backer and L.E. Fleming. 2008. Impacts of climate variability and future climate change on harmful algal blooms and human health. *Environmental Health* 7(Suppl 2):S4.

Morris, P.D., D.S. Campbell, T.J. Taylor, and J.I. Freeman. 1991. Clinical and epidemiological features of neurotoxic shellfish poisoning in North Carolina. *American Journal of Public Health* 81(4):471-47.

Nishitani, L. and K.K. Chew. 1984. Recent developments in paralytic shellfish poisoning research. *Aquaculture* 39:317-329.

NOAA. Diarrhetic Shellfish Poisoning. http://www.nwfsc.noaa.gov/hab/habs_toxins/marine_biotoxins/dsp/index.html [Accessed 23 December 2008].

O'Shea, T.J., G.B. Rathbun, R.K. Bonde, C.D. Buregelt and D.K. O'Dell. 1991. An epizootic of Florida manatees associated with a dinoflagellate bloom. *Marine Mammal Science* 7(2):165-179.

Paerl, H.W. and J. Huisman. 2008. Blooms like it hot. *Science* 320:57-58.

Ramsdell, J.S. and T.S. Zabka. 2008. In utero domoic acid toxicity: A fetal basis to adult disease in the California sea lion (*Zalophus californianus*). *Marine Drugs* 6(2):262-290.

Redlow, T., A. Foley, and K. Singel. 2002. Sea turtle mortality associated with red tide events in Florida. In: *Proceedings of the 22nd Annual Symposium on Sea Turtle Biology and Conservation*, J. Seminoff, compiler. U.S. Available at: http://www.seaturtle.org/ists/docs/22turtle.pdf [Accessed 30 December 2008]

Reeves, R.R.; Rolland, R.; Clapham, P.J., editors. 2001. Causes of reproductive failure in North Atlantic right whales: new avenues of research. Report of a workshop held 26-28 April 2000, Falmouth, Massachusetts. Northeast Fish. Sci. Cent. Ref. Doc. 01-16; 46 pp. Available at: http://www.nefsc.noaa.gov/nefsc/publications/crd/crd0116/0116.htm [Accessed 30 December 2008].

Scholin, C.A., F. Gulland, G.J. Doucette, S. Benson, M. Busman, F.P. Chavez, J. Cordaro, R. DeLong, A. De Vogelaere, J. Harvey, M. Haulena, K. Lefebvre, T. Lipscomb, S. Loscutoff, L.J. Lowenstine, R. Marin III, P.E. Miller, W.A. McLellan, P.D.R. Moeller, C.L. Powell, T. Rowles, P. Silvagni, M. Silver, T.Spraker, V.Trainer and F.M. Van Dolah. 2000. Mortality of sea lions along the California coast linked to a toxic diatom bloom. *Nature* 403(6765):80-4.

Stanley, D.W. and S.W. Nixon. 1992. Stratification and bottom-water hypoxia in the Pamlico River estuary. *Estuaries and Coasts* 15(3):270-281.

Steidinger, K.A., J.M. Burkholder, H.B. Glasgow, Jr., C.W. Hobbs, J.K. Garrett, E.W. Truby, E.J. Noga and S.A. Smith. 1996. *Pfiesteria piscicida* gen. et sp. nov. (Pfiesteriaceae fam. nov.), a new toxic dinoflagellate with a complex life cycle and behavior. *Journal of Phycology* 32(1):157-164.

Takahashi, E.M., K.E. Arthur and G.R. Shaw. 2007. Occurrence of okadaic acid in the feeding grounds of dugongs (*Dugong dugon*) and green turtles (*Chelonia mydas*) in Moreton Bay, Australia. *Harmful Algae* 7(4):430-437.

Tiffany, B.N., N.A. Fangue and W.A. Bennett. 2006. Disappearance of a population of pygmy octopus following a harmful algal bloom in a northwestern Florida bay, USA. *American Malacological Bulletin* 21:11-15.

Trainer, V.L. and D.G. Baden. 1999. High affinity binding of red tide neurotoxins to marine mammal brain. *Aquatic Toxicology* 46(2):139-148.

Uhart, M., Karesh, W., Cook, R., Huin, N., Lawrence, K., Guzman, L., Pacheco, H., Pizarro, G., Mattsson, R. & Mörner, T. 2004. Paralytic Shellfish poisoning in Gentoo penguins (*Pygoscelis papua*) from the Falkland (Malvinas) Islands. In 2004 proceedings of the AAZV, AAWV, WDA joint conference. pp 481- 486.

U.S. Fish & Wildlife Service. 2007. West Indian Manatee (*Trichechus manatus*): 5-Year Review, Summary and Evaluation. 6 April 2007. Available at http://ecos.fws.gov/docs/five_year_review/doc1042.pdf [Accessed 30 December 2008].

USGS, Biological Resources Division. 1999. Field Manual of Wildlife Diseases: General Field Procedures and Diseases of Birds. Chapter 36, Algal Toxins. Information and Technology

Report 1999-001. 426 pages. Available at: http://www.nwhc.usgs.gov/publications/field_manual/ [Accessed 30 December 2008].

Washington Department of Fish and Wildlife. 2003. Domoic Acid- A Major Concern to Wahington State's Shellfish Lovers. http://wdfw.wa.gov/fish/shelfish/razorclm/domacid.htm [Accessed 23 December 2008]

Woods Hole Oceanographic Institution. 2003. Will Ocean Fertilization To Remove Carbon Dioxide From The Atmosphere Work?. *ScienceDaily*, April 10. Retrieved January 5, 2009, from http://www.sciencedaily.com /releases/2003/04/030410073330.htm

Yasumoto, T., I.Nakajima, and R. Bagnis. 1977. Finding of a Dinoflagellate as a likely culprit of ciguatera. *Bulletin of the Japanese Society of Scientific Fisheries* 43(8): 1021-1026.

Chapter 5.

Alker, A.P., G.W. Smith and K. Kim. 2001. Characterization of *Aspergillus sydowii* (Thom et Church), a fungal pathogen of Caribbean sea fan corals. *Hydrobiologia* 460:105-111.

Antonius, A. and D. Lipscomb. 2001. First protozoan coral-killer identified in the Indo-Pacific. *Atoll Research Bulletin* 481:1-21.

Aronson, R.B. and W.F. Precht. 2001. White band disease and the changing face of Caribbean coral reefs. *Hydrobiologia* 460(1-3):25-38.

Aronson, R.B., W.F. Precht, M.A. Toscano and K.H. Koltes. 2002. The 1998 bleaching event and its aftermath on a coral reef in Belize. *Marine Biology* 141:435-117.

Arthur, R. 2000. Coral bleaching and mortality in three Indian reef regions during an El Niño Southern Oscillation event. *Current Science* 79(12):1723-1729.

Bally, M. and J. Garrabou. 2007. Thermodependent bacterial pathogens and mass mortalities in temperate benthic communities: a new case of emerging disease linked to climate change. *Global Change Biology* 13(10):2078-2088.

Banin, E., T. Israely, A. Kushmaro, Y. Loya, E. Orr, and E. Rosenberg. 2000. Penetration of the coral-bleaching bacterium *Vibrio shiloi* into *Oculina patagonica*. *Applied and Environmental Microbiology* 66:3131-3136.

Banin, E., K.H. Sanjay, F. Naider, and E. Rosenberg. 2001. A proline-rich peptide from the coral pathogen *Vibrio shiloi* that inhibits photosynthesis of zooxanthellae. Applied and *Environmental Microbiology* 67:1536-1541.

Boyett, H.V., D.G. Bourne and B.L. Willis. 2007. Elevated temperature and light enhance progression and spread of black band disease on staghorn corals of the Great Barrier Reef. *Marine Biology* 151(5):1711-1720.

Brown, B.E. 1997. Coral bleaching: causes and consequences. *Coral Reefs* 16(S1):S129-S138.

Bruno, J.F., L. Petes, C.D. Harvell and A. Hettinger. 2003. Nutrient enrichment can increase the severity of two coral reef diseases. *Ecology Letters* 6:1,036-1,061.

Bruno J.F., E.R. Selig, K.S. Casey, C.A. Page, B.L.Willis, C.D. Harvell, H. Sweatman and A.M. Melendy. 2007. Thermal stress and coral cover as drivers of coral disease outbreaks. *PLoS Biology* 5(6):1220-1227.

Bruno, J.F., C.E. Siddon, J.D. Witman, P.L. Colin and M.A. Toscano. 2001. El Niño related coral bleaching in Palau, western Caroline Islands. *Coral Reefs* 20:127-136.

Buddemeier, R.W. and D.G. Fautin. 1993. Coral bleaching as an adaptive mechanism: a testable hypothesis. *BioScience* 43(5):320-326.

Bythell, J. C., O. Pantos, and L. Richardson. 2004. White plague, white band and other "white" diseases. Pp. 351-363. *In* E. Rosenberg and Y. Loya (eds.), *Coral Health and Disease.* Springer-Verlag, Berlin, Germany.

Canadell, J.G., C. Le Quéré, M.R. Raupach, C.B. Field, E.T. Buitenhuis, P. Ciais, T.J. Conway, N.P. Gillett, R.A. Houghton and G. Marland. 2007. Contributions to accelerating CO_2 growth from economic activity, carbon intensity, and efficiency of natural sinks. Proceedings of the *National Academy of Sciences* 104(47):18,866-18,870.

Cervino, J.M., R.L. Hayes, S.W. Polson, S.C. Polson, T.J. Goreau, R.J. Martinez and G.W. Smith. 2004. Relationship of *Vibrio* species infection and elevated temperatures to yellow blotch/band disease in Caribbean corals. *Applied and Environmental Microbiology* 70(11):6,855-6,864.

Cesar, H.J.S., Burke, L., and Pet-Soede, L. 2003. The Economics of Worldwide Coral Reef Degradation. Cesar Environmental Economics Consulting, Arnhem, and WWF-Netherlands, Zeist, The Netherlands. 23pp. Online at: http://assets.panda.org/downloads/cesardegradationreport100203.pdf

Coma, R., M. Ribes, E. Serrano, E. Jiménez, J. Salat and J. Pascual. 2009. Global warming-enhanced stratification and mass mortality events in the Mediterranean. *Proceedings of the National Academy of Sciences* 106(15):6176-6181.

Crabbe, M.J.C., E. Martinez, C. Garcia, J. Chub, L. Castro and J. Guy. 2007. Growth modeling indicates hurricanes and severe storms are linked to low coral recruitment in the Caribbean. *Marine Environmental Research* 65(4):364-368.

Davy, S.K., S.G. Burchett, A.L. Dale, P. Davies, J.E. Davy, C. Muncke, O. Hoegh-Guldberg and W.H. Wilson. 2006. Viruses: agents of coral disease? *Diseases of Aquatic Organisms* 69(1):101-110.

Eakin, C.M., D.K. Gledhill, S.F. Heron, W. Skirving, T. Christensen, J. Morgan, G. Liu and A.E. Strong. 2007. Few like it hot: Coral reef responses to elevated temperatures and CO_2. *Eos. Trans. AGU* 88(52), Fall Meeting Supplement, Abstract B53E-05.

Efrony, R., Y. Loya, E. Bacharach and E. Rosenberg. 2007. Phage therapy of coral disease. *Coral Reefs* 26(1):7-13.

Frias-Lopez, J., G.T. Bonheyo, Q. Jin and B.W. Fouke. 2003. Cyanobacteria associated with coral black band disease in Caribbean and Indo-Pacific reefs. Applied and Environmental *Microbiology* 69(4):2409-2413.

Gardner, T.A., I.M. Côté, J.A. Gill, A. Grant, and A.R. Watkinson. 2005. Hurricanes and Caribbean coral reefs: Impacts, recovery patterns and role in long-term decline. *Ecology* 86(1):174-184.

Garrison, V.H., E.A. Shinn, W.T. Foreman, D.W. Griffin, C.W. Holmes, C.A. Kellogg, M.S. Majewski, L.L. Richardson, K.B. Ritchie and G.W. Smith. 2003. African and Asian Dust: From desert soils to coral reefs. *BioScience* 53(5):469-480.

Geisner, D.M., J.W. Taylor, K.B. Ritchie and G.W. Smith. 1998. Cause of sea fan death in the West Indes. *Nature* 394:137-138.

Glynn, P.W. and L. D'Croz. 1990. Experimental evidence for high temperature stress as of El Niño-coincident coral mortality. *Coral Reefs* 8(4):181-191.

Harvell, D., E. Jordán-Dahlgren, S. Merkel, E. Rosenberg, L. Raymundo, G. Smith, E. Weil and B. Willis (Coral Disease Working Group). 2007. Coral disease, environmental drivers and the balance between coral and microbial associates. *Oceanography* 20(1):172-195.

Hough-Guldberg, O., P.J. Mumby, A.J. Hooten, R.S. Steneck, P. Greenfield, E. Gomez, C.D. Harvell, P.F. Sale, A.J. Edwards, K. Caldiera, N. Knowlton, C.M. Eakin, R. Iglesias-Prieto, N. Muthiga, R.H. Bradbury, A. Dubi and M.E. Hatziolos. 2007. Coral reefs under rapid climate change and ocean acidification. *Science* 318:1737-1742.

Intergovernmental Panel on Climate Change: Trenberth, K.E., P.D. Jones, P. Ambenje, R. Bojariu, D. Easterling, A. Klein Tank, D. Parker, F. Rahimzadeh, J.A. Renwick, M. Rusticucci, B. Soden and P. Zhai, 2007: Observations: Surface and Atmospheric Climate Change. In: *Climate Change 2007: The Physical Science Basis*. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change [Solomon, S.,D. Qin, M. Manning, Z. Chen, M. Marquis, K.B. Averyt, M. Tignor and H.L. Miller (eds.)]. Cambridge University Press, Cambridge, United Kingdom and New York, NY, USA.

Jokiel, P.L. 2004. Temperature stress and coral bleaching. Pp. 401-426 *In* E. Rosenberg and Y. Loya (eds.), *Coral Health and Disease*. Springer-Verlag, Berlin,Germany, 517 pp.

La Pointe, B.E., B.J. Bedford and R. Baumberger. 2006. Hurricanes Frances and Jeanne remove blooms of the invasive green alga *Caulerpa brachypus* forma *parvifolia* (Harvey) Cribb from coral reefs off northern Palm Beach County, Florida. *Estuaries and Coasts* 29(6):966-971.

Manzello, D.P., M. Brandt, T.B. Smith, D. Lirman, J.C. Hendee and R.S. Nemeth. 2007. Hurricanes benefit bleached corals. *Proceedings of the National Academy of Sciences* 104(29):12035-12039.

McClanahan, T.R. 2004. Coral bleaching, diseases and mortality in the western Indian Ocean. Pp.

157-176 *In* E. Rosenberg and Y. Loya (eds.), *Coral Health and Disease*. Springer-Verlag, Berlin, Germany 517pp.

Paddack, M.J., J.D. Reynolds, C. Aguilar, R.S. Appeldoorn, J. Beets, E. W. Burkett, P.M. Chittaro, K. Clarke, R. Esteves, A.C. Fonseca, G.E. Forrester, A.M. Friedlander, J. García-Sais, G. González-Sansón, L.K.B. Jordan, D. B. McClellan, M.W. Miller, P.P. Molloy, P.J. Mumby, I. Nagelkerken, M. Nemeth, R. Navas-Camacho, J. Pitt, N.V.C. Polunin, M.C. Reyes-Nivia, D.R. Robertson, A. Rodríguez-Ramírez, E. Salas, S.R. Smith, R.E. Spieler, M.A. Steele, I.D. Williams, C.L. Wormald, A.R. Watkinson and I.M. Côté. 2009. Recent region-wide declines in Caribbean reef fish abundance. *Current Biology* 19(7):590-595.

Page, C.A. and B.L. Willis. 2006. Distribution, host range and large-scale spatial variability in black band disease prevalence on the Great Barrier Reef, Australia. *Diseases of Aquatic Organisms* 69(1):41-51.

Pastorak, R.A. and G.R. Bilyard. 1985. Effects of sewage pollution on coral-reef communities. *Marine Ecology Progress Series* 21:175-189.

Patterson, K.L., J.W. Porter, K.B. Ritchie, S.W. Polson, E. Mueller, E.C. Peters, D.L. Santavy, and G.W. Smith. 2002. The etiology of white pox, a lethal disease of the Caribbean elkhorn coral, *Acropora palmate*. *Proceedings of the National Academy of Sciences* 13:8,725-8,730.

Prospero, J.M. and P.J. Lamb. 2003. African droughts and dust transport to the Caribbean: climate change implications. *Science* 302:1024-1027.

Reaka-Kudla, M.L. 1996. The global biodiversity of coral reefs: a comparison with tropical rain forests. Pages 83-108 *In*: Reaka-Kudla, M.L., D.E. Wilson and E.O. Wilson (eds.). *Biodiversity II: Understanding and Protecting our Biological Resources*. Joseph Henry Press, 560 pages.

Remily, E.R. and L.L. Richardson. 2006. Ecological physiology of a coral pathogen and the coral reef environment. *Microbial Ecology* 51(3):345-352.

Richardson, L.L. 1992. Red band disease: A new cyanobacterial infestation of corals. *Proceedings of the 10th Annual American Academy of Underwater Science* 153-160.

Rosenberg, E. and Y. Ben-Haim. 2002. Microbial diseases of coral and global warming. *Environmental Microbiology*. 4(6):318-326.

Rützler, K., and D.L. Santavy. 1983. The black band disease of Atlantic reef corals. I. Description of a cyanophyte pathogen. *Marine Ecology* 4:301-319.

Sheppard, C. and R. Rioja-Nieto. 2005. Sea surface temperature 1871-2099 in 38 cells in the Caribbean region. *Marine Environmental Research* 60(3):389-396.

Sussman, M., D.G. Bourne and B.L. Willis. 2006. A single cyanobacterial ribotype is associated with both red and black bands on diseased corals from Palau. *Diseases of Aquatic Organisms* 69(1):111-118.

Ulstrup, K.E., M. Kühl and D.G. Bourne. 2007. Zooxanthellae harvested by ciliates associated with brown band syndrome of corals remain photosynthetically competent. Applied and *Environmental Microbiology* 73(6): 1968-1975.

Viehman, S., D. K. Mills, G. W. Meichel, and L. L. Richardson. 2006. Culture and identification of Desulfovibrio spp. from corals infected with black band disease on Dominican and Florida Keys reefs. *Diseases of Aquatic Organisms* 69(1):119-127.

Voss, J.D. and L.L. Richardson. 2006. Nutrient enrichment enhances black band disease progression in corals. *Coral Reefs* 25(4):569-576.

Weir-Brush, J.R., V.H. Garrison, G.W. Smith and E.A. Shinn. 2004. The relationship between gorgonian coral (Cnidaria: Gorgonacea) diseases and African dust storms. *Aerobiologia* 20:119-126.

Whelan, K.R.T., J. Miller, O. Sanchez and M. Patterson. 2007. Impact of the 2005 coral bleaching event on *Porites porites* and *Colpophyllia natans* at Tektite Reef, U.S. Virgin Islands. *Coral Reefs* 26(3):689-693.

Chapter 6:

Alaska Climate Change Adaptation Advisory Group. 2008. *Natural Systems Impacts Framework*. Available at: http://www.akclimatechange.us/ewebeditpro/items/O97F17334.pdf [Accessed 16 Oct 2008].

Arizona Game and Fish Department. 2007. Whirling disease discovered in trout at Lee's Ferry. Press Release, June 16.

Athanassopoulou, F. 1992. Ichthyophoniasis in sea bream, *Sparus aurata* (L.), and rainbow trout, *Oncorhynchus mykiss* (Walbaum), from Greece. *Journal of Fish Diseases* 15:437-441.

Benson, A. J. 2008. Zebra mussel sightings distribution. http://nas.er.usgs.gov/taxgroup/mollusks/zebramussel/zebramusseldistribution.asp. [Accessed 1 Oct 2008].

Blazer V.S., T.B. Waldrop, W.B. Schill, C.L. Densmore and D. Smith. 2003. Effects of water temperature and substrate type on spore production and release in eastern Tubifex tubifex worms infected with Myxobolus cerebralis. *Journal of Parasitology* 89(1):21-26.

Chun, S.K., and Y.G. Kim. 1981. Infection by an *Ichthyophonus* sp. fungus in rainbow trout *Salmo gairdneri*. *Bulletin of the Korean Fisheries Society* 14:37-42.

Cooney, S. undated. Global warming: Implications for the recovery of the greenback cutthroat trout (*Oncorhynchus clarki stomias*). http://www.nrel.colostate.edu/projects/star/papers/greenback.pdf Accessed 3 October 2008.

Eaton, J.G. and R.M. Scheller. 1996. Effects of climate warming on fish thermal habitat in streams of the United States. *Limnology and Oceanography* 41(5):1109-1115.

El-Matbouli, M., T. S. McDowella, D. B. Antonio, K. B. Andree and R. P. Hedrick. 1999. Effect of water temperature on the development, release and survival of the triactinomyxon stage of *Myxobolus cerebralis* in its oligochaete host. *International Journal for Parasitology* 29(4):627-641.

Gilbert, M.A. and W.O. Granath, Jr. 2003. Whirling disease of salmonid fish: life cycle, biology and disease. *Journal of Parasitology* 89(4):658-667.

Hedrick, R.P., T.S. McDowell, M. Gay, G.D. Marty, M.P. Georgiadis, and E. MacConnell. 1999. Comparative susceptibility of rainbow trout (*Oncorhynchus mykiss*) and brown trout (*Salmo trutta*) to *Myxobolus cerebralis*, the cause of salmonid whirling disease. *Diseases of Aquatic Organisms* 37:173-183.

Independent Scientific Advisory Board. 2007. *Climate Change Impacts on the Columbia River Basin Fish and Wildlife*. ISAB 2007-2. http://www.nwcouncil.org/library/isab/ISAB%202007-2%20Climate%20Change.pdf, Accessed 2 October 2008.

JTC (Joint Technical Committee of the Yukon River US/Canada Panel). 2006. *Yukon River salmon 2005 season summary and 2006 season outlook*. Alaska Department of Fish and Game, Division of Commercial Fisheries, Regional Information Report No. 3A06-03, Anchorage.

Jones, S.R.M. and S.C. Dawe. 2002. *Ichthyophonus hoferi* Plehn & Mulsow in British Columbia stocks of Pacific herring *Clupea pallasi* Valenciennes, and its infectivity to Chinook salmon Oncorhynchus *tshawytscha* (Walbaum). *Journal of Fish Diseases* 25:415-421.

Keleher, C.J. and F.J. Rahel. 1996. Thermal limits to salmonid distributions in the Rocky Mountain region and potential habitat loss due to global warming: a geographic information system (GIS) approach. *Transactions of the American Fisheries Society* 125(1):1-13.

Kocan, R., P. Hershberger and J. Winston. 2003. Effects of I*chthyophonus* on Survival and Reproductive Success of Yukon River Chinook Salmon. Final Report for Study 01-200.

Kocan, R., P. Hershberger and J. Winston. 2004. Ichthyophoniasis: An emerging disease of Chinook salmon in the Yukon River. *Journal of Aquatic Animal Health* 16:58-72.

Kocan, R. and P. Hershberger. 2006. Differences in *Ichthyophonus* prevalence and infection severity between upper Yukon River and Tanana River chinook salmon, *Oncorhynchus tshawytscha* (Walbaum), stocks. *Journal of Fish Diseases* 29(8): 497-503.

Kocan, R., S. LaPatra, J. Gregg, J. Winton and P Hershberger. 2006. *Ichthyophonus*-induced cardiac damage: a mechanism for reduced swimming stamina in salmonids. *Journal of Fish Diseases* 29(9):521-527.

Koel, T.M., D.L. Mahony, K.L. Kinnan, C. Rasmussen, C.J. Hudson, S. Murcia, and B.L. Kerans. 2007. Whirling disease and native cutthroat trout of the Greater Yellowstone Ecosystem. *Yellowstone Science* 15(2):25-33.

MacConnell, E. and E. R. Vincent. 2001. The effects of *Myxobolus cerebralis* on the salmonid host. 2001 Whirling Disease Symposium.

Markiw, M. and K. Wolf. 1983. *Myxosoma cerebralis* (Myxozoa: Myxosporea) etiologic agent of salmonid whirling disease requires tubificid worm (Annelida:Oligochaeta) in its life cycle. *Journal of Protozoology* 30:561-564.

Miyazaki, T. and S. Kubota. 1977. Studies on Ichthyophonus disease of fishes. I. Rainbow trout fry. *Bulletin of the Faculty of Fisheries of Mie University* 4:45-56.

National Assessment Synthesis Team, US Global Change Research Program. 2000. *Climate Change Impacts on the United States: The Potential Consequences of Climate Variability and Change*.

Okamoto, N., K. Nakase, and T. Sano. 1987. Relationships between water temperature, fish size, infective dose, and *Ichthyophonus* infection in rainbow trout. *Nippon Suisan Gakkaishi (Bulletin of the Japanese Society of Scientific Fisheries)* 53(4):581-84.

Potera, C. 1997. Fishing for answers to whirling disease. *Science* 278(5336):225-226.

Rahel, F.J., C.J. Keleher, and J.L. Anderson. 1996. Potential habitat loss and population fragmentation for cold water fish in the North Platte River drainage of the Rocky Mountains: Response to climate warming. *Limnology and Oceanography* 41(5):1116-1123.

Rahimian, H. 1998. Pathology and morphology of *Ichthyophonus hoferi* in naturally infected fishes off the Swedish west coast. *Diseases of Aquatic Organisms* 34:597-598.

Ruggieri, G.D., R.F. Nigrelli, P.M. Powles, and D.G. Garnett. 1971. Epizootics in yellowtail flounder, *Limanda ferruginea* Storer, in the western North Atlantic caused by *Ichthyophonus*, an ubiquitous parasitic fungus. *Zoologica* 55:57-61.

Ryce, E.K.N., A.V. Zale and E. MacConnell. 2004. Effects of fish age and parasite dose on the development of whirling disease in rainbow trout. *Diseases of Aquatic Organisms* 59:225-233.

Scattergood, L.W. 1948. A report on the appearance of the fungus *Ichthyosporidum hoferi* in the herring of the northwestern Atlantic. *U.S. Fish and Wildlife Service Special Scientific Report on Fisheries* 58:1-33.

Sitja-Bobadilla, A. and P. Alvarez-Pellitero. 1990. First report of *Ichthyophonus* disease in wild and cultured sea bass *Dicentrachus labrax* from the Spanish Mediterranean area. *Diseases of Aquatic Organisms* 8:145-150.

Spanggard, B., H.H. Huss, and J. Bresciani. 1995. Morphology of *Ichthyophonus hoferi* assessed by light and scanning electron microscopy. *Journal of Fish Diseases* 18(6):567-577.

Trout Unlimited. 2007. *Healing Troubled Waters: Preparing Trout and Salmon Habitat for a Changing Climate*. http://www.tu.org/atf/cf/{ED0023C4-EA23-4396-9371-8509DC5B4953}/final_climate_change_report.pdf

Tung, M.-C., S.-T. Huang, and S.-S. Tsai. 1986. And epizootic in largemouth bass, *Micropterus salmoides* Lacepede, caused by *Ichthyophonus* in freshwater ponds in southern Taiwan. *Council of Agriculture Fisheries Series* 8:6-13.

Whirling disease initiative. http://whirlingdisease.montana.edu/about/transmission.htm Accessed 3 October 1008.

Whirling disease interactive mapping project. http://bsi1.msu.montana.edu/whirlingdisease/default.aspx

Wolf, K., M. Markiw and J.K. Hiltunen. 1986. Salmonid whirling disease: *Tubifex tubifex* identified as the essential oligochaete in the protozoan life cycle. *Journal of Fish Diseases* 9:83-85.

Chapter 7:

Appel, M.J.G. and J.E. Gillespie JE: 1972. Canine distemper monograph. Pp. 34-63 *In* Gard S., C. Hallauer, andK.F. Meyer (eds), *Handbook of Virus Research*. Springer-Verlag, New York, NY.

Appel, M.J.G., R.A. Yates, G.L. Foley, J.J. Bernstein, S. Santinelli, L.H. Spelman, L.D. Miller, L.H. Arp, M. Anderson, M. Barr, S. Pearce-Kelling and B.A. Summers. 1994. Canine distemper epizooitic in lions, tigers and leopards in North America. *Journal of Veterinary Diagnostic Investigation* 6:277-288.

Burek, K.A., F.M.D. Gulland, and T.M. O'Hara. 2008. Effects of climate change on arctic marine mammal health. *Ecological Applications* 18(2) Supplement: S126-S134.

Goldstein, T., J.A.K. 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. Emerging Infectious Diseases 15(6):925-927).

Grachev M.A., Kumarev V.P., Mamaev L.V., Zorin V.L., Baranova L.V., Denikina N.N., Belikov S.I., Petrov E.A., Kolesnik V.S., Kolesnik R.S., Dorofeev V.M., Beim A.M., Kudelin V.N., Nagieva F.G., Sidorov V.N. 1989. Distemper virus in Baikal seals (Correspondence). *Nature* 338**:**209.

Greifenhagen, S. and T.L. Noland. 2003. A Synopsis of Known and Potential Diseases Associated with Climate Change. Forest Research Information Paper No. 154. Ontario Forest Research Institute, Ontario Ministry of Natural Resources.

Gunn, A., F. Leighton and G. Wobeser. 1991. Wildlife diseases and parasites in the Kitikmeot Region, 1984-1990. Final Report No. 104, Department of Renewable Resources, Government of the Northwest Territories, Coppermine. 52 pages.

Harkonen, T., R. Dietz, P. Rejinders, J. Teilmann, K. Harding, A. Hall, S. Braseur, U. Siebert, S.J. Goodman, P.D. Jepson, T.D. Rasmussen, and P. Thompson. 2006. The 1988 and 2002 phocine distemper virus epidemics in European harbor seals. *Diseases of Aquatic Organisms* 68:115-130.

Hoberg, E.P., L. Polley, A. Gunn and J.S. Nishi. 1995. *Umingmakstrongylus pallikuukensis* gen. nov. et sp. nov. (Nematoda: Protostrongylidae) from muskoxen (*Ovibos moschatus*) in the central Canadian Arctic, with comments on biology and biogeography. *Canadian Journal of Zoology* 73:2266-2282.

Intergovernmental Panel on Climate Change. 2007. Summary for Policymakers. In: *Climate Change 2007: The Physical Science Basis.* Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change [Solomon, S., D. Qin, M. Manning, Z. Chen, M. Marquis, K.B. Averyt, M.Tignor and H.L. Miller (eds.)]. Cambridge University Press, Cambridge, United Kingdom and New York, NY, USA.

Jenkins, E.J., A.M. Veitch, S.J. Kutz, E.P. Hoberg and L. Polley. 2006. Climate change and the epidemiology of protostrongylid nematodes in northern ecosystems: *Parelaphostrongylus odocoilei and Protostrongylus stiles*i in Dall's Sheep (*Ovis d. dalli*). *Parasitology* 132:387-401.

Jensen, T., M. van de Bildt, H. Henrik Dietz, T.H. Andersen, A.S. Hammer, T. Kuiken and A. Osterhaus. 2002. Another phocine distemper outbreak in Europe. *Science* 297(5579):209

Kennedy, S. T. Kuiken, P. D. Jepson, R. Deaville, M. Forsyth, T. Barrett, M.W.G. van de Bildt, A.D.M.E. Osterhaus, T. Eybatov, C. Duck, A. Kydyrmanov, I. Mitrofanov, and S. Wilson. 2000. Mass die-off of Caspian seals caused by canine distemper virus. *Emerging Infectious Diseases* 6(6):637-639.

Kilpatrick, A.M., A.A. Chmura, D.W. Gibbons, R.C. Fleischer, P.P. Marra, and P. Daszak. 2006. Predicting the global spread of H5N1 avian influenza. *Proceedings of the National Academy of Sciences* 103(51):19368-19373.

Kuiken, T. S. Kennedy, T. Barrett, M. W. G. Van de Bildt, F. H. Borgsteede, S. D. Brew, G. A. Codd, C. Duck, R. Deaville, T. Eybatov, M. A. Forsyth, G. Foster, P. D. Jepson, A. Kydyrmanov, I. Mitrofanov, C. J. Ward, S. Wilson and A. D. M. E. Osterhaus. 2006. The 2000 canine distemper epidemic in Caspian seals (*Phoca caspica*): Pathology and analysis of contributory factors. *Veterinary Pathology* 43:321-338.

Kutz, S.J., E.P. Hoberg, J. Nagy, L. Polley and B. Elkin. 2004. "Emerging" parasitic infections in Arctic ungulates. *Integrative and Comparative Biology* 44:109-118.

Kutz, S.J., E.P. Hoberg, J.S. Nishi and L. Polley. 2002. Development of the muskox lungworm *Umingmakstrongylus pallikuukensis* (Protostrongylidae) in gastropods in the Arctic. *Canadian Journal of Zoology* 81:1977-1985.

Kutz, S.J., E.P. Hoberg and L. Polley. 1999. Experimental infection of muskoxen (*Ovibos moschatus*) and domestic sheep with *Umingmakstrongylus pallikuukensis* (Nematoda: Protostrongylidae): Parasite development, population structure and pathology. *Canadian Journal of Zoology* 77:1562-1572.

Kutz, S.J., E.P. Hoberg and L. Polley. 2001. Development of *Umingmakstrongylus pallikuukensis* in gastropods: Morphology, morphometrics and the influence of temperature. *Journal of Parasitology* 87:527-535.

Munson, L., K.A. Terio, R. Kock, T. Mlengeya, M.E. Roelke, E. Dubovi, B. Summers, A.R.E. Sinclair and C. Packer. 2008. Climate extremes promote fatal co-infections during canine distemper epidemics in African lions. *PLoS One* 3(6):e2545.

Muller, G, P. Wohlsein, A. Beineke, L. Haas , I. Greiser-Wilke, U. Siebert, S. Fonfara, T. Harder, M. Stede, A.D. Gruber, and W. Baumgärtner. 2004. Phocine distemper in German seals, 2002. *Emerging Infectious Diseases* 10(4):723-275.

Penzhorn, B.L. 2006. Babesiosis of wild carnivores and ungulates. Veterinary Parasitology 138:11-21.

Roelke-Parker, M.E., L. Munson, C. Packer, R. Kock, S. Cleaveland, M. Carpenter, S.J. O'Brien, A. Pospischil, R Hofmann-Lehmann, H. Lutz, G.L.M. Mwamengele, M.N. Mgasa, G.A. Machange, B.A. Summers and M.J.G. Appel. 1996. A canine distemper virus epidemic in Serengeti lions (*Panthera leo*). *Nature* 379(1):441-445.

Weber, T.P. and N.I. Stilianakis. 2007. Ecologic immunology of avian influenza (H5N1) in migratory birds. *Emerging Infectious Diseases* 13(8):1139-1143.

Conclusion

Theoharides, K., G. Barnhart, P. Glick, N. Matson, A. Choudhury and A. Staudt. 2009. Climate Change Adaptation Across the Landscape: A survey of federal and state agencies, conservation organizations and academic institutions in the United States. Available at: http://www.defenders.org/resources/publications/programs_and_policy/gw/climate_change_adaptation_across_the_landscape.pdf [Accessed 13 May 2009].

U.S. Fish and Wildlife Service. 2008. Rising to the Urgent Challenges of a Changing Climate: Strategic Plan for Responding to Accelerating Climate Change in the 21st Century. Internal Dicussion Draft, Available at: http://www.fws.gov/home/climatechange/pdf/climate_change_draft_strategic_plan.pdf [Accessed 12 May 2009].

U.S. Geological Survey. 2008. National Climate Change and Wildlife Science Center Summary Workshop Report. Available at: http://nccw.usgs.gov/documents/NCCWSC_Summary_Workshop_Report.pdf [Accessed 13 May 2009].

Wildlife Disease Information Node. USGS Wildlife Health Center and the University of Wisconsin. Available at: http://wildlifedisease.nbii.gov/ [Accessed 13 May 2009].