Report to the AFWA Fish and Wildlife Health Committee
Wildlife Disease Issues of Interest to State Managers and Directors
USGS National Wildlife Health Center, Madison, WI

September 16, 2007

The following information is of a topical nature for wildlife management agencies and entities; many partners and collaborators are involved in gathering and researching the information herein.

AVIAN INFLUENZA

Live Bird Surveillance (AK, CA, HI, OR, TX, WA, UT)

During 2006, as part of the program for avian influenza surveillance in live birds, 18,633 birds were tested and 316 birds were positive for avian influenza, according to the matrix RT-PCR test. Nine of those were of the H5 subtype based on RT-PCR. No H5N1 was detected during the 2006 surveillance in live birds. As of Sept. 1, 2007, the NWHC has received 4,375 samples and most of these are from Alaska; live bird collection in the lower 48 states is just beginning. Overall, 32 birds tested positive for avian influenza by RT-PCR, but no H5 positives have been identified so far.

Hunter-Harvest Surveillance (AK only)

During the 2006 hunter-harvest surveillance season (April 1, 2006 – March 31, 2007), 7,513 birds were tested and 384 birds were positive for avian influenza, according to the matrix RT-PCR test. Of these, 16 were of the H5 subtype. None were of the H5N1 variety. During the 2007 season (as of Sept. 1), 2,200 samples were received from Alaska. Six samples tested positive for avian influenza, but none were positive for H5 so far.

Mortality Investigation Surveillance (Nationwide)

During the 2006 surveillance season, 1,150 birds were submitted as part of mortality event investigations and were tested for avian influenza. The presence of influenza virus was detected in 41 birds. None of the mortality investigation samples were positive for H5. During the 2007 season (as of Sept. 1), a total of 301 birds have been submitted for avian influenza testing as part of the mortality investigation program. Ten birds have tested positive for avian influenza so far this year. None of them are of the H5 subtype.

There is no evidence of the introduction of the highly pathogenic avian influenza H5N1 by migratory wild birds in the United States at the present time.

Avian Influenza Virus Isolation (AK, CA, UT, WA)

Eight H5 avian influenza viruses were detected in Alaska, another six from California, and two each from Utah and Washington. No H5N1 viruses of either high or low pathogenicity were identified in the surveillance effort, but H5N2, H5N3, and H5N9 subtypes were isolated. Some of the H5 viruses isolated are discussed in an upcoming publication in the Journal of Virology. No H7 avian influenza viruses were identified during the initial screening efforts using the National Animal Health Laboratory Network

(NAHLN) procedure. This was an unexpected result, as the H7 subtype is relatively common in wild birds. We re-tested all of the matrix RT-PCR positive samples after the season with a different RT-PCR test and isolated 23 H7 viruses. NWHC will be working with both the Southeast Poultry Research Laboratory and the National Veterinary Services Laboratories to modify the current H7 RT-PCR test of the National Animal Health Laboratory Network to improve detection of H7 avian influenza viruses in North American wild birds. Other avian influenza viruses identified in the surveillance program include H2N1, H2N4, H2N5, H3N6, H3N8, H4N6, H4N7, H4N9, H6N1, H8N4, H10N2, H10N7, H10N8, H11N2, H11N9, and H16N3 viruses. A number of new bird species not previously recorded with avian influenza infection were discovered during the 2006 surveillance program. These include the surprising role of marine birds, such as glaucous gulls and several species of eiders. Moreover, sequence characterization of the influenza viruses isolated from these birds have revealed evidence of intercontinental mixing of genes between Eurasian and North American viruses and supports our hypothesis that these birds might play a role in the potential introduction of H5N1 into North America.

SYLVATIC PLAGUE

Prairie Dogs and Black-footed Ferrets

Plague (*Yersinia pestis*), a bacterial disease of wild rodents transmitted by fleas, can rapidly decimate colonies of prairie dogs, the primary food of the endangered black-footed ferret. Moreover, recent information indicates that the ferrets are also highly susceptible to this disease. The occurrence of plague in prairie dog populations, and its potentially devastating effect on black-footed ferrets, has made it a major impediment to the ferret recovery programs of the Bureau of Land Management, the Fish and Wildlife Service, the National Park Service, and numerous State agencies. A recombinant vaccine against sylvatic plague that can be delivered by ingestion of a gelatin-based bait was used experimentally to successfully protect black-tailed prairie dogs from challenge with the plague bacteria. This vaccine offers potential for being delivered in baits to free-ranging prairie dog colonies. Vaccine studies using an F1-V plague vaccine were done in black-footed ferrets. Vaccinated ferrets were shown to be completely protected from plague after ingesting plague-infected mice. Field tests of the vaccine in ferrets also showed high titers to F-1 for over one year. Preliminary data from field trials showed the vaccine improved survival of vaccinates about twice that over unvaccinated animals.

WEST NILE VIRUS (WNV) UPDATE

WNV Sampling in Greater Sage Grouse (NV, OR)

In August 2007, WNV was again isolated from moribund greater sage-grouse in Oregon. The USGS National Wildlife Health Center (NWHC) sampled 114 greater sage-grouse and 371 alternate host species (passerines) at sites in Nevada and Oregon in August 2007. Sample testing is in progress.

Vesper sparrows:

An experimental study conducted at the NWHC has shown that vesper sparrows are moderately susceptible to West Nile virus (WNV), developing high titers following

exposure to the virus. However, WNV infection in vesper sparrows was not fatal. Vesper sparrows are a very common passerine species in sage habitat and thus may serve as an amplification host to WNV.

Merial WNV Vaccine:
An experimental trial of the Merial Recombitek Equine WNV vaccine shows the vaccine to be highly efficacious in preventing WNV when used in a prime-boost strategy. In addition, it was moderately efficacious at preventing WNV and reducing the virus' pathogenicity when used as a single dose of vaccine. In captured free-ranging wildlife, the vaccine might be employed to prevent WNV in a single dose fashion. Studies are currently underway to determine the duration of protection provided by a single dose of the Merial vaccine.

White Pelicans:
In collaboration with Northern Prairie Wildlife Research Center and the U.S. Fish and Wildlife Service, we have been investigating whether there is evidence of developing herd immunity to WNV in white pelicans at two pelican breeding sites in North Dakota and South Dakota. We are in the second summer of obtaining serum samples from white pelican chicks prior to the WNV transmission season and from healthy fledgling birds late in the summer. We tested the chicks for the passive transfer of WNV antibody from female to chick and tested the fledglings for evidence of chick survival during the summer transmission season.

CHRONIC WASTING DISEASE (CWD) RESEARCH

The susceptibility of various small rodent species to CWD is being examined by intra-cerebral challenge studies at the NWHC. Meadow voles (*Microtus pennsylvanicus*) appear to be very susceptible to intra-cerebral CWD challenge, with 100% penetrance and a median post-challenge survival time of 270 days. On the other hand, deer mice (*Peromyscus maniculatus*) and white-footed mice (*P. leucopus*) have not developed clinical signs 1 year post-challenge. Red-backed voles (*Myodes gapperi*) have been under challenge for about 3 months and have not yet presented with clinical signs of illness. Experiments are being initiated with the University of Wisconsin to further explore the implications of voles' susceptibility to CWD, especially the likelihood of voles acquiring infections via natural routes. Among other things, we will be examining whether soil minerals potentiate the oral infectivity of CWD in voles, as has been demonstrated by Aiken and Pederson for a prion/hamster model system.

In conjunction with the Wisconsin Department of Natural Resources and other partners, the NWHC has been developing statistical spatial-temporal epidemiological models of CWD epidemics in free-ranging cervids. Substantial progress has been made in developing new statistical "backcasting" models based on dynamic process theory that allow the estimation of the rates at which the disease is growing and spreading. The analyses have discovered that substantial fine-scale spatial heterogeneity exists in infectivity, and spatial patterns in infectivity seem quite stable over time.

Status Update (CO, IL, KS, MN, MT, NE, NM, NY, OK, SD, UT, WV, WI, WY, Alberta and Saskatchewan)
CWD has not been detected in any new states during 2006 or thus far in 2007. Recent disease detections include New Mexico (within the existing known range) and Illinois (LaSalle County, about 25 miles south of previous cases; this is the first documented case in the Illinois River basin). Currently CWD has been detected in free-ranging populations in 11 states and 2 Canadian provinces (Colorado, Illinois, Kansas, Nebraska, New Mexico, New York, South Dakota, Utah, West Virginia, Wisconsin, Wyoming, Alberta and Saskatchewan) and in captive facilities in 9 states and 2 Canadian provinces (Colorado, Kansas, Minnesota, Montana, Nebraska, New York, Oklahoma, South Dakota, Wisconsin, Alberta and Saskatchewan).

CWD-Positive Tissue Bank
Several agencies have encountered positive cervids over the past two years and the agency response has varied considerably. In response to the continued need for known positive control tissues and known positive sources of infected material, the USGS NWHC entered into a working agreement with the Wyoming Division of Wildlife and the University of Wyoming to develop a positive tissue bank. The bank consists of elk, mule deer and white-tailed deer samples. All animals were inoculated orally with infected brain material from conspecific animals. Infected animals were killed at 6 month intervals post-exposure and complete necropsies were conducted.

Harvested tissues from all major organ systems and lymphatics were split or doubled such that all tissues are both frozen and fixed in 10% neutral-buffered formalin. In addition, prior to euthanasia, whole blood samples were collected and serum or plasma (collected in heparin and ETDH) were harvested and frozen. Urine was collected from the urinary bladder at necropsy and fecal pellets were collected from the distal colon. Environmental samples in the form of fresh fecal pellets and urine soaked soil were collected from animal holding areas and frozen. Immunohistochemistry assays specific for CWD are being performed on all tissues. All test animals were infected. By collecting animals incrementally, we are describing how this disease moved through cervid hosts over time up to two years post infection.

Field Investigations Team Summaries, January – September 2007

Lead poisoning in waterfowl leads to eagle deaths in Oregon
During the months of January and February 2007, approximately 1,500 mallards died from lead poisoning after feeding in a flooded agricultural field near Klamath National Wildlife Refuge, Oregon. Mallards collected had liver lead concentrations ranging from 19.99 – 46.68 ppm (wet weight) in January and 17.51 – 54.00 ppm in February. These levels are known to be lethal in mallards. Lead pellets were present in the gizzards of some ducks. Furthermore, after observing approximately 100 bald eagles scavenging mallards, refuge staff live-trapped 8 bald eagles and 1 golden eagle to collect blood for lead analysis. Blood lead levels for all eagles ranged from 0.04 to 0.31 ppm, wet weight, and none were below the detection limit of 0.02 ppm. Biologists suspect that the mallards obtained the lead pellets on this former waterfowl hunting area. Alternatively, contamination of agricultural land with lead pellets resulting from efforts to control

nuisance birds is also possible. Cold weather conditions may have concentrated local mallards for an extended period in one of the few remaining unfrozen water bodies in south-central Oregon.

Immature greater shearwaters die of suspected starvation along the Atlantic and Gulf coasts (FL, GA, SC, NC, VA, MD)

From the coasts of Florida to Maryland, hundreds of immature greater shearwaters were found washed up dead on the beaches in mid-June. Over 200 dead shearwaters were counted, but it is estimated there were at least 500 dead. When approached, the few live birds observed were unwilling to move and appeared emaciated. It is suspected that the young shearwaters starved to death while on their migratory route to Newfoundland, but the reason for the starvation is as yet undetermined. In August, shearwaters began arriving on the Texas coast in poor condition and appeared emaciated; about 100 immature shearwaters were found dead and NWHC is currently investigating cause-of-death.

Marine bird mortality along Oregon coast (OR)

During March 2007, unusually high numbers of rhinoceros auklets, horned and tufted puffins were found dead along with eight other species on the Oregon coast. The birds were found during volunteer surveys for beached birds. An estimated 200 dead birds have been counted. Horned and tufted puffins submitted to the USGS NWHC were emaciated with no evidence of food in the digestive tract. The higher than expected numbers of dead birds observed could reflect a change in the population this year, a change in winter distribution, or a change in ocean conditions. Biologists with the NOAA report that there was abundant phytoplankton, but numbers of small forage fish may now be down. USFWS biologists suggest that the puffins may have moved down from Alaska in poor condition due to reduced food resources there. The generally poor condition of birds in these populations may also be linked to the poor marine food resources in 2006. See the *Newport News-Times* from April 6, 2007.

***Botulism type E in Great Lakes kills endangered piping plover and thousands of fish-eating birds* (MI, NY)**

Annual mortalities of thousands of fish-eating birds such as ring-billed gulls, Caspian terns, and black-backed gulls from Lake Michigan, Lake Ontario, and Lake Erie are attributed to botulism type E exposure as a result of a complex cycle involving toxic algae blooms, zebra mussels, round gobies and the bacterium *Clostridum botulinum*. Based on recorded patterns, botulism type E activity is expected to continue into at least late November and affect more than 10,000 birds. Since early June 2007 and as of September 2007, botulism type E has likely killed several thousand fish-eating birds in these 3 Great Lakes, and has been confirmed in 3 of 4 endangered piping plovers collected at Sleeping Bear Dunes National Lakeshore, Lake Michigan. Botulism type E at Sleeping Bear Dunes occurred from late June to mid-July 2007 and resulted in at least 45 bird deaths. Compared to fish-eating birds, shorebird mortalities from botulism type E are much less common. Although a source of the toxin for the plover is not known, scientists speculate that the plover fed upon maggots that had infested the carcasses of affected fish-eating birds or other invertebrates on the shore. Shorebirds have recently

been observed to eat maggots of beached cormorant carcasses. Management strategies to reduce the exposure of the piping plover and other shorebirds to toxin involve the removal of beached carcasses. However, the magnitude of potentially affected beaches will make it difficult to keep fish-eating bird carcasses collected, worrying those concerned about the recovery of the endangered piping plover.

Coccidiosis outbreak in lesser scaup in North Dakota thought to be caused by aeration of lake

In mid-April, about 350 dead and 100 sick lesser scaup were found on the shores of Pheasant Lake in Dickey County. The emaciated birds had difficulty flying and diving. When on the ground, many birds used their wings to move themselves rather than their feet, and when they were not moving, the birds left their wings spread out and down on the ground as if using them to balance. The outbreak lasted for about a week and a half, at the end of which approximately 1,157 birds had died. The disease, identified as coccidiosis in this case, was caused by an intestinal protozoan parasite *Eimeria aythyae.* Outbreaks of coccidiosis have occurred at this same lake in 1989, 1990, 1991, and 2006. It is speculated that aeration of the lake may have stimulated sporulation of oocysts, and in addition prevented winter ice from forming and artificially concentrated returning spring migrants. Future management steps include ceasing aeration of the lake while scaup are present to possibly reduce the amount of coccidia present and discourage the birds from congregating.

Botulism type C outbreaks with concurrent West Nile virus in pelicans in multiple states during summer months (CA, FL, IL, KY, MD, MT, ND, NM, NV, OR, SD, UT, WI)

Warmer temperatures and lower water levels common during summer weather conditions contribute to the occurrence of botulism type C mortality events in waterfowl and shorebirds in multiple states across the country. Botulism has been reported from CA, FL, IL, KY, MD, MT, ND, NM, NV, OR, SD, UT, and WI. NWHC has investigated 20 events with mortality estimates of over 2,000 birds, which is part of a continued decline in the number of events reported to the NWHC. Increased familiarity with the disease by field biologists may have lead to decreased reporting rates for national trends. Concurrent West Nile virus outbreaks have occurred in American white pelicans in MT, ND, and SD at locations with ongoing botulism events. The events have resulted in mortality estimates of over 500 hatch-year pelican chicks. Biologists are encouraged to report morbidity/mortality and submit samples to determine cause-of-death because of the concurrent outbreaks.

Exotic parasite discovered in Mississippi River (WI, MN)

In June 2007, scientists at the USGS National Wildlife Health Center (NWHC) discovered *Leyogonimus polyoon*, an exotic parasitic trematode affecting American coot, in exotic faucet snails collected from the Upper Mississippi National Wildlife and Fish Refuge, near La Crosse, Wisconsin. The snails came from Lake Onalaska, a major backwater lake of the Upper Mississippi River. USGS documented the first record of *L. polyoon* in North America in 1998 when it infected and killed thousands of American coots in north-central Wisconsin. The discovery of snails infected with *L. polyoon* in

Lake Onalaska confirms that the parasite has become established in the Upper Mississippi River System (UMRS) and can potentially spread into its many tributaries. (For additional information see USGS Wildlife Health Bulletin 07-01, http://www.nwhc.usgs.gov/publications/wildlife_health_bulletins/WHB07-01LeyoPoly.pdf)

Based on data from 2004–2006 surveys, *L. polyoon* was not present in the Upper Mississippi River until now. The life cycle of *L. polyoon* involves the exotic faucet snail (*Bithynia tentaculata*) and various species of aquatic insect larvae, such as dragonflies and damselflies. The American coot is the only natural definitive host thus far reported to be susceptible to infection in North America. Coot fall victim to the parasite by feeding on infected insect larvae. (See USGS Fact Sheet: Exotic Parasite Causes Large Scale Mortality in American Coots, http://www.nwhc.usgs.gov/publications/fact_sheets/pdfs/fact_lpolyoon.pdf). In Eastern Europe, where the faucet snail and parasite are native, the spread of the parasite is limited by the geographic distribution of the faucet snail. In the United States, coot and aquatic insects are widely distributed, so control of the parasite will focus on controlling the snail.