AI Diagnostics Research

The difficulties associated with virus isolation- and PCR-based surveillance for avian influenza viruses (AIV) in wild birds have become readily apparent in recent exploratory surveillance efforts. Conducting AIV surveillance on novel species or in locations where there is little information to guide sample collection can be like “looking for a needle in a haystack,” because of the limited duration of viral shedding and the marked variability in spatial and temporal patterns of AIV infection in different wild avian populations. In addition, using diagnostic techniques with low sensitivity can greatly reduce the chance of detecting low rates of infection.

In the last issue of the SCWDS BRIEFS (Vol. 25, No. 4), we reported the results of our evaluation of a new serological surveillance technique (the IDEXX bELISA) that had good sensitivity and specificity. Here we report results of our evaluation of a new technique for detecting genetic material of AIV. This technique is less dependent on maintaining cold storage during sample handling, shipping, and processing than traditional methods. Difficulties maintaining the “cold chain” most often are associated with adverse field conditions such as remote field sites; however, they also can occur through improper sample handling in the laboratory (repeated freeze-thaw). These interruptions can drastically reduce the likelihood of AIV detection, particularly when attempting virus isolation.

Finders Technology Associates (FTA®) cards are composed of filter paper impregnated with chemicals that lyse cells, bind and stabilize genetic material, and inactivate infectious agents, including viruses. A swab or tissue impression smear is applied to the card and allowed to air dry. Once dried, the FTA® cards can be stored at room temperature and tested with PCR, presumably without the loss of diagnostic sensitivity; there is no need to maintain the cold chain. With the proper permits, the cards can be sent to a laboratory from anywhere in the world.

With funding from the Wildlife Conservation Society GAINS Program, SCWDS has evaluated the reliability of FTA® cards for wild bird AIV surveillance. Our research to date has been conducted under laboratory conditions and has addressed the detection limit for the technique, the effect of room temperature storage, and field sample validation. The same general protocol was used for all of these studies. A sample of AIV from an inoculated chicken egg was pipetted onto the FTA® cards, hole punches were collected from each card, and genetic material was eluted and extracted from the punches using commercial kits. Real-time reverse transcriptase PCR (RRT-PCR) was used to screen the samples for the genes encoding matrix protein of type A influenza virus, as is done with swabs collected from wild birds in some surveillance programs.

The limit of detection for the FTA® cards was evaluated for six wild bird-origin low pathogenic AI (LPAI) virus isolates. The average minimal concentration of detection for all six LPAI isolates was relatively high, and this could greatly limit the sensitivity of FTA® cards for AIV detection in wild birds. To evaluate the effects of room temperature storage, we inoculated cards with the same six LPAI viruses with 10 times the viral concentration that was detectable in the previous experiment. The cards were stored at room temperature and sampled at intervals from one to 120 days post-
inoculation. Viral RNA was consistently detected on the cards for up to 30 days for all subtypes. After 30 days, the reliability of detection decreased, but some subtypes were detectable for up to 90 or 120 days. These results suggest that maintaining the inoculated FTA® cards at room temperature will not affect the sensitivity if viral RNA is extracted within 20 days of sampling. Additional studies should be conducted to evaluate FTA® cards stored at a variety of temperatures likely to be encountered in the field.

To validate the utility of FTA® Cards with field samples, we applied material to the card from 20 known positive and five negative cloacal swab samples from both ruddy turnstones and mallards. No significant difference was observed in the performance of the diagnostic technique between the two species. The diagnostic sensitivity was approximately 68% and the specificity was 100% on all 50 samples, and there was moderate agreement between the results of virus isolation and RRT-PCR of FTA® cards.

Based on our results, the minimal detectable concentration for the six LPAI viruses used in the experiment was higher than is generally considered effective for AIV detection in wild bird field samples. However, results of the room temperature storage and field sample validation experiments supported the potential utility of the FTA® cards as a viable technique for AIV detection. Mallards and ruddy turnstones were used for the field sample validation experiments because both are considered reservoirs for LPAI virus. Consequently, viral concentrations in the feces of these species may have been very high and facilitated the detection of AIV on the cards. Detection frequencies could be significantly lower in other non-reservoir species with greatly reduced viral shedding, and additional experiments are needed to validate FTA® cards in other avian groups.

Collectively, these results suggest that the FTA® cards can be used to detect AIV in wild birds, but the sensitivity is poor compared to traditional surveillance approaches. At this point, the primary utility of FTA® cards is for situations where maintaining the cold chain is not possible. While this diagnostic technique may never be as effective as traditional surveillance approaches, there are some situations where it could prove useful. (Prepared by Shamus Keeler and Justin Brown)

White Nose Syndrome Update

To the great disappointment of conservationists, white nose syndrome (WNS) has not slowed in its spread across the United States since it was first identified in bats in Howe’s Cave, New York, in 2006. On February 16, 2010, Geomyces destructans, the fungus associated with WNS, was reported for the first time in bats in Worley’s cave in eastern Tennessee, the most southerly state to be affected thus far. Only weeks after WNS was first identified in the state, the disease was found approximately 280 miles westward in Dunbar Cave, located about 40 miles northwest of Nashville. Even more recently, another infected premises, White Oak Blowhole Cave, has been identified in the Tennessee portion of Great Smoky Mountains National Park. On April 19, a National Park Service press release stated that a little brown bat (Myotis lucifugus) from that site was confirmed to be infected by G. destructans. This finding is particularly important because White Oak Blowhole Cave contains the largest known hibernaculum of Indiana bats (Myotis sodalis) in Tennessee.

WNS made another big jump when it was discovered in Pike County, Missouri, northwest of St. Louis. A little brown bat from a privately owned property in the center of Pike County was positive by the polymerase chain reaction (PCR) test. Photographs of the bat suggest a mild infection, with grossly visible fungus evident only on the skin at the wrists. Missouri Department of Conservation personnel report that more than 6,300 caves are present in the state, and 74% are privately owned. The number containing bat colonies is uncertain; 500 hibernacula are known, but as many as 5,000 caves across the state could harbor bats.

Canada also can be included within the advancing line of WNS. On March 19, 2010, the Ontario Ministry of Natural Resources issued a press release stating that WNS had been confirmed in the Bancroft-Minden area, the first report of the disease in Canada. By March 31, it had been identified in six additional sites in the
province. On April 12, 2010, Quebec officials reported they had confirmed WNS in the Outaouais region along the border with Vermont. However, they also report that bats suspected of having WNS have been observed in the Outib-Temiscamingue region of western Quebec, near Ottawa, Ontario.

As the affected region expands, WNS is being found in more caves within the known distribution of the disease. Maryland recently reported its first positive cave, though surrounding states were previously known to be affected. Delaware, which has no known caves with hibernacula, reported its first cases as well. The bats that were confirmed to be infected with the fungus recently had emerged from caves and migrated to summer roost sites in Delaware. Every other affected state except Connecticut and Massachusetts continues to find additional affected premises that fill in the map of counties with WNS.
Those areas that have been affected the longest by WNS have seen dramatic declines in bat numbers. Cumulative mortality rates in some caves affected for multiple years approach 100%. The alarming mortality rates and the rapid advance of WNS greatly increase the urgency of a response. Researchers across the country are working to better understand the disease and to identify response options, but time is limited.

In an effort to better coordinate the response to WNS, 72 state and federal fish and wildlife biologists from across the country met March 16-18, in Louisville, Kentucky, to draft a National Management Plan for WNS. This effort was partially funded by the U.S. Fish and Wildlife Service through a Multistate Conservation Grant to assist state agencies in their ability “to detect, manage, research, and educate the public about disease issues.” This grant was awarded to SCWDS and has been instrumental in expanding the capabilities of state wildlife agencies in their ability to address wildlife disease issues. (Prepared by Kevin Keel)

**More CWD News**

Although we provided an update on chronic wasting disease (CWD) in the last issue of the SCWDS BRIEFS, a few recent events are worthy of mention. The Missouri Department of Agriculture (MDA) reported on February 25, 2010, that CWD had been confirmed for the first time in the state. The positive animal was a captive white-tailed buck at a private shooting enclosure in Linn County in the north-central part of the state. On March 19, the MDA and the Missouri Department of Conservation (MDC) reported that CWD was not detected in 50 additional captive deer that had been destroyed and tested on the 800-acre tract.

As part of a cooperative response plan, the MDC collected samples from 153 free-ranging white-tailed deer within a 5-mile radius of the affected captive facility to determine whether CWD was present in the wild animals sampled. Samples also were tested from 72 wild deer harvested by hunters in Linn and surrounding counties during the 2009-2010 deer hunting seasons. Testing has been completed, and no evidence of CWD has been detected among any wild deer sampled in Missouri.

The MDC and MDA have been testing for CWD for years in wild and captive cervids, respectively. In 2002, Missouri established a state Cervid Health Committee composed of individuals from MDA, MDC, the Missouri Department of Health and Senior Services, and the U.S. Department of Agriculture to cooperatively mitigate CWD-associated challenges. Since then, more than 24,000 free-ranging deer from all parts of the state have been tested for CWD. The MDC has indicated it will be testing hunter-harvested deer from the northern half of the state during the 2010-2011 hunting seasons and will continue sampling efforts in the area where Missouri’s first case was discovered.

On March 17, 2010, the North Dakota Fish and Game Department (NDFGD) announced that CWD had been confirmed in a free-ranging white-tailed deer killed by a hunter last autumn. The adult buck from western Sioux County appeared sick and is the first case of CWD found in North Dakota. NDFGD, Standing Rock Reservation, and South Dakota Game Fish and Parks Department personnel are working closely to develop surveillance strategies for this fall’s harvest. At present, the plan is to reduce the wild deer population by increased hunting in the management unit (deer unit 3F2), where the positive animal was. In addition, the hunter-harvested surveillance will be augmented with increased targeted surveillance of sick animals and road kills.

A growing challenge with CWD management is how to maximize the efficiency of surveillance programs that must be sustained over long periods, especially as resources to conduct this work become more limited. A paper by Walsh and Miller in the January 2010 issue of the *Journal of Wildlife Diseases* reported their evaluation of the potential effects of using a biased system that gives more weight to animals with high prevalence and low inclusion probability (clinical CWD suspects) and less weight to animals with low prevalence and high probability of inclusion (apparently healthy hunter-killed deer). The authors found that by implementing this type of system, “wildlife agencies should be able to maintain or improve
current surveillance standards while, perhaps, collecting and examining fewer samples, thereby increasing the efficiency and cost-effectiveness of ongoing CWD surveillance programs."

Dealing with CWD surveillance, management, and funding for the last several years has been made much easier by Dr. Dean Goeldner, the lead CWD veterinarian with APHIS-Veterinary Services (VS). An extremely ‘user-friendly’ guy, Dean facilitated the allocation of federal funds to address CWD issues among both captive and free-ranging deer and elk and was a reliable liaison between APHIS-VS and wildlife management agencies across the United States. However, as some of you already know, Dean recently left APHIS to become staff director of the Livestock, Dairy, and Poultry Subcommittee of the U.S. House of Representatives Committee on Agriculture. We are grateful to Dean for his fine service and wish him the best of luck in his new endeavors. (Prepared by John Fischer)

**Pneumonia Kills Bighorn Sheep**

Large numbers of bighorn sheep (BHS) in at least nine herds in five western states died or were culled due to pneumonia in unprecedented die-offs during the winter of 2009-2010. Pneumonia is a severe disease in BHS and is believed to have contributed significantly to the great decline of many North American populations to less than 10% of their historic numbers. Since November 2009, state wildlife management agencies in Montana, Nevada, Utah, Washington, and Wyoming have reported one or more affected BHS herds, with new cases identified through early March 2010. Investigations of the etiologic agents and epidemiological factors contributing to the die-offs are ongoing.

The first fatal case of pneumonia was discovered November 15, 2009, in a BHS herd along the East Fork of the Bitterroot River in Montana. The Montana Department of Fish, Wildlife and Parks (MDFWP) removed 75 sick BHS from this population of 200-220 animals in order to prevent the spread of disease to other BHS populations nearby. On January 12, 2010, sick sheep were reported on the Lower Blackfoot River, and by February 5 MDFWP removed 93 BHS from this herd of 160-180 animals. Additional affected BHS populations were reported in lower Rock Creek and upper Rock Creek, but MDFWP elected not to intervene due to difficult terrain.

In Nevada, coughing BHS were first reported during the second week of December 2009 in the Ruby Mountains, and in late December in the East Humboldt Range. There is documented movement of rams between these two ranges during the rut. Eighty-eight dead BHS were found, and herd mortality was estimated at 50% in the Ruby Mountains and 60-80% in the East Humboldt Range. The Nevada Department of Wildlife captured and marked 30 BHS and treated one group with a combination of an antibiotic, a non-steroidal anti-inflammatory drug, and vitamin E. The other group was treated with the same combination, minus the antibiotic. Most of the animals were sick and in poor body condition when captured, and preliminary results indicated little, if any, difference in outcome between BHS that received antibiotic and those that did not. An additional 46 BHS were darted with antibiotic without being captured and marked.

In Washington, sick BHS were discovered on both sides of the Yakima River Canyon in early December 2009. Approximately 90% of the northern subpopulation and 25% of the southern subpopulation on the west side of the river were coughing. By mid-February 2010, 20 BHS carcasses had been found, and the Washington Department of Fish and Wildlife elected to cull the sickest remaining animals to prevent spread of disease to a large population to the northeast. By mid-March 2010, 40 BHS had been culled, and 10 more pneumonia mortalities had been found.

The Utah Division of Wildlife Resources attempted to eliminate all 40-60 BHS in a herd on the northern slope of the Uinta Mountains after finding sick animals in mid-February 2010. By February 20, 26 wild sheep had been culled. These efforts were made to prevent spread of pneumonia to the herds at nearby Bare Top, Carter Creek, and Sheep Creek.

Bighorn sheep showing signs of pneumonia were discovered in Wyoming in early March 2010. Only 5-7% of approximately 55 BHS
present were coughing. Collection and necropsies of two young BHS revealed early stages of pneumonia in both animals.

In addition to conducting management actions, all involved state wildlife management agencies collected biological samples from BHS for diagnostic testing. Although final results are pending, a variety of bacterial organisms have been cultured or identified by PCR testing, and *Pasteurella* and *Mycoplasma* were the dominate genera found.

Wild sheep are very susceptible to pneumonia, especially to infection with *Pasteurella* and related organisms. Two epidemic patterns in BHS have been described: Some epidemics are believed to be due to endemic respiratory pathogens, including *Pasteurellaceae*, *Mycoplasma ovipneumoniae*, viruses including parainfluenza-3 and respiratory syncytial virus, and lungworms, with or without environmental stressors. Other outbreaks appear to be initiated by introductions of novel respiratory pathogens into BHS populations. In the latter form, contact with domestic sheep and goats is suspected to provide the opportunity for pathogen transmission. The potential role of domestic sheep contact continues to be assessed in the 2009-2010 die-offs. In some of the locations, such contact is suspected; in others, there is no documented contact. Domestic sheep in Montana were tested for pathogens at the time of bighorn sheep testing, but results are not yet available.

A workshop to evaluate response protocols and discuss future strategies for managing disease issues among BHS will take place during the 17th Northern Wild Sheep and Goat Council meeting at Hood River, Oregon, June 7-11, 2010.

Information for this article was excerpted from a summary of the 2009-2010 die-offs compiled by the Wild Sheep Working Group of the Western Association of Fish & Wildlife Agencies. The summary can be found at (http://wolves.files.wordpress.com/2010/03/wafwa-wswg-summary-on-winter-2009-10-bhs-dieoffs.pdf).

(Prepared by Carolyn Hodo, senior veterinary student, University of Georgia College of Veterinary Medicine)

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**MCF in a Key Deer**

On January 5, 2010, SCWDS was asked by U.S. Fish and Wildlife Service personnel to assist in the investigation of the death of an endangered Key deer held at a South Florida petting zoo. The deer died suddenly, and a postmortem examination was conducted by the attending veterinarian. Lesions included multifocal necrosis of the liver and severe pulmonary edema. Tissues were sent to a private pathology laboratory, and clinicians there described a vasculitis thought to be consistent with hemorrhagic disease (HD).

Almost immediately after we contacted the clinician regarding this deer, the other Key deer in the facility became feverish and lethargic. It died within two days, in spite of supportive care and the administration of antibiotics. The clinician again performed a necropsy and described subcutaneous hemorrhages and edema in the wall of the small intestine and associated mesentery. Samples of frozen and formalin-fixed tissues were submitted to SCWDS for evaluation.

In this case, the origin of the deer suggested that HD was an unlikely cause of death. Although we regularly find circulating antibodies to HD viruses in Key deer, indicating exposure, we have never identified a clinical case of HD in these animals. This probably is due to a combination of factors. Deer in the extreme South are exposed to the viruses of HD on a regular basis from birth and, therefore, have a high degree of herd immunity. In that situation, deer are not likely to develop clinical HD. In addition, experimental results suggest that white-tailed deer that populate these areas of high exposure may be genetically resistant to these viruses. The maintenance of the deer in a mixed-animal collection and a second look at the description of the vasculitis in the first deer increased our suspicion that another disease had killed these deer.

Formalin-fixed tissues from the second deer were examined microscopically using standard techniques. This examination revealed all major organs had severe vasculitis. These lesions were characterized by massive accumulations of lymphocytes and a particular degenerative
change to arteries called fibrinoid necrosis. This combination of lesions is suggestive of malignant catarrhal fever, a disease caused by some types of herpesvirus that are host-adapted to, and carried by, certain species of ungulates. Results of polymerase chain reaction tests (PCR) matched ovine herpesvirus-2 (OVHV-2), the causative agent of sheep-associated malignant catarrhal fever (MCF).

Cases of MCF in mixed-animal collections are not uncommon. There have been sporadic cases in a number of species, all of which occurred as the result of a herpesvirus being transmitted by an asymptomatic carrier to another susceptible ungulate species. The two herpesviruses most commonly associated with MCF are OVHV-2 and alcelaphine herpesvirus-1, a herpesvirus commonly carried by wildebeest. However, another herpesvirus, caprine herpesvirus-1, recently has been associated with MCF in sika deer and white-tailed deer that had close contact with goats. These viruses are not highly infectious, and they usually require close contact between a susceptible host and an unapparent carrier.

This case emphasizes our inability to recognize the disease risks that apparently healthy individuals can pose. Every animal harbors many bacteria, viruses, and parasites that may not be causing disease in that individual; however, they may have pathogenic potential for other hosts or under different conditions. (Prepared by Kevin Keel)

National Feral Swine Mapping System

In 1982, SCWDS produced a series of maps depicting the nationwide distribution and density of several species of wild or feral cloven-hoofed animals that are susceptible to foot and mouth disease. To meet the continuous demand for information on the dramatically expanding range of feral swine, SCWDS updated this map in 1988, 2004, 2008, 2009 and 2010. In 1982 17 states reported feral swine, in 2004 28 states reported feral swine, and currently 36 states report populations of feral swine. It is important to note that our maps are of established populations, not incidental or occasional sightings. Established populations are those that have been present for at least two years and where there is evidence of, or a demonstrated potential for, reproduction.

In response to the need for current data on the feral swine distribution, SCWDS developed the National Feral Swine Mapping System (NFSMS) in 2008 in cooperation with USDA-APHIS-Veterinary Services (see SCWDS BRIEFS Vol. 24, No. 1). This is an interactive data collection system that can be accessed at http://www.feralswinemap.org/. The site was developed to collect and display current distribution data on feral swine in the United States.

The home page is available to the public, but a password available only to designated state/federal agency personnel provides access to the interactive data collection areas of the site. Operation of the NFSMS is a collaborative effort between SCWDS, the Information Technology Department in the University of Georgia’s (UGA) College of Veterinary Medicine, and the Center for Remote Sensing and Mapping Science in the UGA Department of Geography. Since the system went online in 2008 we have had over 330 individual additions made to the map through the NFSMS, and several states have provided completely new maps with numerous changes.

As with previous maps, personnel with state and territorial natural resources agencies, as well as with USDA-APHIS-Wildlife Services, provide distribution data for the NFSMS. Agency personnel can submit this information online at any time by simply locating the selected areas on the website map and drawing additions to the feral swine distribution directly onto the map. Populations and sightings are distinguished as shaded areas or single points. Areas where feral swine have been eliminated can be deleted from the map in the same manner. The data are carefully evaluated by SCWDS, and we update the national map monthly.

In addition to providing the national map through the website, SCWDS can furnish state or regional maps on request. Although it was developed primarily for evaluating the local risk posed by feral swine for spreading pseudorabies virus and swine brucellosis, the NFSMS provides access to up-to-date data on the

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distribution of feral swine in the United States. These data are important to state/federal wildlife resources managers, agricultural agencies, and public health agencies throughout the United States. (Prepared by Joseph Corn)

**Brucella suis** in North America

Each of the six characterized species within the *Brucella* genus has its own principal host species; however, all are capable of causing disease in humans and a variety of wild and domestic mammals. *Brucella suis* is an important pathogen of wild and domestic pigs, with multiple biovars that affect a number of other species. Biovars 1, 2, and 3 are capable of infecting swine, while biovar 4 is maintained in reindeer and caribou. Biovar 2 also occurs in European hares, and biovar 5 is found in small rodents. All subtypes of *B. suis* have zoonotic potential, but biovars 1, 3 and 4 in particular pose a significant threat to human health.

Brucellosis in humans can be a serious, debilitating, and often chronic disease with nonspecific signs. Clinical signs can include fever, headache, gastrointestinal upset, anorexia, and arthritis. Humans usually become infected by direct contact with tissues, blood, urine, fetuses and placentas of infected animals and by ingestion of raw milk or dairy products. Like most *Brucella* species, *B. suis* is found in high numbers in tissues associated with an abortion or stillbirth from an infected animal. Transmission in swine primarily is through coitus and ingestion of contaminated or aborted material. Among reindeer and caribou, transmission occurs mainly through contact with infective uterine discharges following abortion.

Historically, human cases of *B. suis* most commonly occurred among swine slaughterhouse workers. However, human infections from domestic swine have disappeared in the United States since the National Cattle Brucellosis Eradication Program was expanded in 1972 to include commercial swine herds and *B. suis* was eradicated from domestic pigs. Currently, human *B. suis* infections in the continental United States predominantly are associated with exposure to feral swine. Feral swine are a significant brucellosis reservoir and pose a threat to domestic swine and human health. In 2009, the U.S. Centers for Disease Control and Prevention (CDC) published a report detailing *B. suis* infections in three men who had hunted and handled feral swine in Florida (*SCWDS BRIEFS* Vol. 25, No. 2).

Serologic investigations of infected feral swine herds in the Southeast have revealed *B. suis* antibody prevalence of 5-44%. In 2007, 80 pigs were euthanized and necropsied from a known enzootically infected herd in South Carolina that had been used for brucellosis vaccine research. *Brucella suis* biovar 1 was isolated from 69% of the animals, and *B. abortus* (two vaccine strains and one field strain) was isolated from 35%. However, only 49% of the animals were positive for *Brucella* antibodies, suggesting that the sensitivity of serologic tests may not be as high as previously thought. In a recent SCWDS study, *B. suis* was found in feral swine herds in South Carolina but not in North Carolina (*SCWDS BRIEFS* Vol. 25, No. 4). This was attributed to the possibility that feral swine in the study area in North Carolina had been established after brucellosis had been eradicated from domestic swine and have remained free of infection thus far. The distribution of feral swine in the United States has been steadily expanding, primarily due to human-assisted movements, and it can be expected that *B. suis* will spread to uninfected feral herds.

In the Arctic region, the major reservoir of *B. suis* is rangiferine cervids rather than swine. *Brucella suis* biovar 4 has long been enzootic in many caribou herds throughout Canada and Alaska. Antibodies also have been found in moose, wolves, grizzly bears, arctic foxes, sled dogs, and humans. A survey of caribou, grizzly bears, and wolves in Alaska from 1975-1998 showed that antibody prevalence for *Brucella* ranged from 0-9% for caribou, 0-25% for wolves, and 0-24% for bears. For all three species, prevalence was higher in the northern part of the state. Brucellosis has been found on multiple occasions in sled dogs in Alaska that were fed raw caribou meat as part of their diet. Based on published reports, it can be concluded that brucellosis is present in much of the rangiferine cervid population of Alaska and Canada and is commonly transmitted to predators.

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Most human cases of \textit{B. suis} biovar 4 are acquired by ingesting raw caribou meat or bone marrow, a common practice among the indigenous people of the Arctic region. Brucellosis in humans in the Arctic has been documented since the early 19th century but was most commonly caused by \textit{B. abortus} in unpasteurized cow's milk. In 1959, the first cases of \textit{B. suis} were described in residents in the villages of Wainwright and Barrow, Alaska, and other cases of \textit{B. suis}-related illness have been described in Alaska since then. Between 1982-1990, 12 human brucellosis cases were attributed to \textit{B. suis} biotype 4, and all involved clinically ill Alaskans who utilized caribou as part of their diet. Serum tests performed in 1963 revealed that 5-20\% of the residents in communities where caribou is eaten regularly had positive titers to \textit{Brucella}. Due to the often nonspecific signs of brucellosis, it is probable that cases go undiagnosed. Also, cases of human brucellosis may resolve spontaneously, requiring no treatment. For these reasons, it is difficult to assess the true prevalence of brucellosis in the human population.

Persons who hunt, handle, and/or consume swine, caribou, reindeer, or other animals in areas where \textit{B. suis} is present are at risk for infection. Hunters can greatly reduce their risk of exposure by wearing rubber gloves while field dressing animals and by cooking the meat thoroughly. Because there are still \textit{Brucella}-free herds of both feral swine and caribou, care should be taken to avoid further spread of the disease. Wildlife species susceptible to \textit{B. suis} should not be relocated, especially from areas where there is known infection, in order to prevent spread to naïve herds and commercial livestock. Education plays a fundamental role in preventing \textit{B. suis} infections in humans, and efforts should be made to inform hunters of the risks involved and the recommended precautions for hunting and handling potentially infected animals. (Prepared by Carolyn Hodo, senior veterinary student, University of Georgia College of Veterinary Medicine)

**Sam Hamilton**

United States Fish and Wildlife Service

Director Sam Hamilton died suddenly and unexpectedly on February 20, 2010. Sam was 54 years old and had been appointed director just last September; however, he had more than 30 years of experience in natural resources conservation and was deeply respected across the country.

Sam got an early start in conservation. At the age of 15, he began working as a Youth Conservation Corps member on the Noxubee National Wildlife Refuge near his hometown of Starkville, Mississippi. He graduated from Mississippi State University in 1977 and went on to serve in numerous capacities in the Fish and Wildlife Service, rising in the ranks to become director of the agency's Southeast region in 1997.

It was during his 12 years based in Atlanta, Georgia, that we came to know Sam as a dedicated partner in natural resources conservation, a friend of SCWDS, and a fine man. Our paths crossed regularly, and our visits were always fruitful and enjoyable. Like numerous friends and colleagues across the country, we mourn Sam’s loss along with his wife, sons, and grandson, and we deeply regret that he served only a few months as director of the U.S. Fish and Wildlife Service. All of us who knew Sam recognized that the agency and the natural resources of the United States could not have been in better hands. (Prepared by John Fischer)
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