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## SCWDS Briefs: Volume 24, Number 2 (July 2008)

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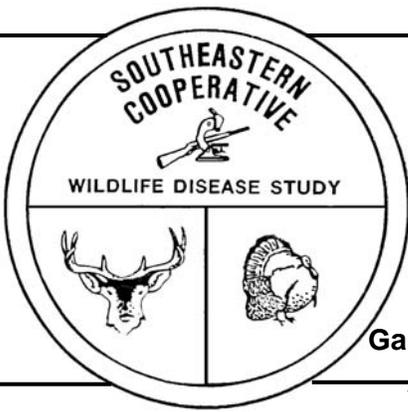


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# SCWDS BRIEFS

A Quarterly Newsletter from the  
**Southeastern Cooperative Wildlife Disease Study**  
**College of Veterinary Medicine**  
**The University of Georgia**  
**Athens, Georgia 30602**

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## Brucellosis in MT and WY

Diseases at the wildlife/livestock interface continue to pose some serious challenges to natural resource and animal health agencies, as well as to livestock producers and sportsmen. In the Greater Yellowstone Area (GYA), which includes parts of Idaho, Montana, and Wyoming, *Brucella abortus* apparently spilled over from domestic cattle to wildlife decades ago and has become firmly established in the wild elk and bison populating the area. Due to the successful national program to eliminate bovine brucellosis in domestic cattle and bison, infected elk and bison in the GYA are the last reservoirs for this disease in the United States. Unfortunately, apparent transmission from infected wildlife to cattle currently is costing Montana its Brucellosis Class-Free Status, and the recent detection of an infected cattle herd in Wyoming is threatening the disease-free status that it regained in 2006.

On June 9, 2008, the Montana Department of Livestock announced confirmation of brucellosis in a cow from Paradise Valley north of Yellowstone National Park. According to federal regulations, the finding of this second infected herd within 24 months will drop Montana from Class Free status to Class A. The first infected herd was detected in May of 2007 and also was located near Yellowstone National Park. The source of infection has not been confirmed in either herd. However, there was no history of commingling of either infected herd with bison, and epidemiological investigations, including genetic analysis of the *B. abortus* organism isolated from the cow in late May 2008, suggest transmission from infected elk, rather than from infected bison or other infected cattle. Montana can reapply for Class Free status in May 2009, which will be 12 months after the last reactor was destroyed.

In June 2008, the Wyoming Livestock Board reported confirmation of brucellosis in a cattle herd near Daniel in Sublette County. Investigation of this situation continues in order to identify the extent of infection, as well as the possible source. The remainder of the Daniel herd is being tested, as are 13 other cattle herds in the area. To date, testing has been completed on two of the area's 13 other herds, with no infected animals found. Wyoming would lose its Class Free status if a second infected herd is detected within 24 months, if all cattle herds in the area are not tested by the end of October 2008, or if the infected Daniel herd is not depopulated by late August 2008.

Federal disease testing requirements increase when Class Free status is lost, and this testing comes at considerable expense to cattle producers. Typically, all sexually intact cattle 18 months of age or older in Class A states must test negative for brucellosis within 30 days prior to export. Among the three states in the GYA, Montana had maintained its Class Free Status since 1985. Wyoming regained Brucellosis Class Free status in 2006 after dropping to Class A in 2004, when two infected cattle herds were detected. A similar situation occurred in Idaho, which lost its Class Free status in late 2005 but regained it in 2007. Transmission of *B. abortus* from infected elk was suspected although it was not confirmed in Wyoming and Idaho in 2004 and 2005, respectively.

Brucellosis is a bacterial disease that results in persistent infections in ungulates and humans. In cattle it causes weak calves and late-term abortions, as well as reduced milk production and possible weight loss. There is no effective treatment for infected animals, and they are euthanized to prevent infection of other animals. Among livestock, *Brucella* bacteria are transmitted by direct contact with infected animals or by use of an environment that has been contaminated with discharges from infected animals, particularly during abortion or calving.

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Humans are susceptible to infection with several species of *Brucella* bacteria. Currently, most human infections in the United States are acquired via consumption of unpasteurized milk from goats infected with *B. melitensis*. Human infection, or undulant fever, is characterized by fever, headache, weakness, profuse sweating, arthritis, and other symptoms. Long-term (6 weeks) antibiotic therapy is the treatment of choice, and relapses may occur due to the intracellular sequestration of the bacteria. The human health considerations, as well as the veterinary implications, precipitated the national brucellosis eradication program in the 1930s. Additional information on brucellosis can be found at the websites of USDA's Animal and Plant Health Inspection Service ([www.aphis.usda.gov](http://www.aphis.usda.gov)), the Montana Department of Livestock ([liv.mt.gov](http://liv.mt.gov)), and the Wyoming Livestock Board ([wlsb.state.wy.us](http://wlsb.state.wy.us)). (Prepared by John Fischer)

### **Lead Fragments in Donated Venison**

Three states (North Dakota, Minnesota, and Wisconsin) stopped distributing hunter-donated ground venison in March and April 2008 because of concerns regarding lead fragments in the meat. In late March, results of a study in North Dakota indicated high levels of lead in a significant portion of tested packages of ground venison. According to the North Dakota Department of Health (ND DOH), 53 out of 95 packages (56%) of donated ground venison showed metal fragments on X-ray examination. Five packages with fragments tested strongly positive for lead. Subsequently, the ND DOH "recommended that food pantries not distribute the ground venison remaining in their possession."

Beginning on May 16, 2008, the U.S. Centers for Disease Control and Prevention (CDC) and the ND DOH began a study to measure the risk, if any, of consuming wild game harvested with lead bullets. Investigators planned to collect samples from 680 persons aged two years and above in order to compare blood lead levels in people who eat wild game with the lead levels of those who do not. By the end of May, samples had been obtained from 738 people in North Dakota's six largest cities. Most samples were collected from adults who had eaten venison from deer harvested with high-velocity lead bullets, although some samples came from persons who had eaten pheasant and waterfowl taken with lead or non-lead pellets. The samples were taken to the CDC laboratory in Atlanta, Georgia, and results

are expected prior to the opening of the autumn hunting seasons.

In Minnesota, officials reported on April 10, 2008, that laboratory tests had confirmed the presence of varying amounts of lead fragments in venison samples collected from food pantries in the state. The Minnesota Department of Agriculture (MDA) found lead fragments in 76 of 299 samples (25%) of donated venison, with laboratory analysis indicating a range of 0.2 - 46.3 parts per million. These highly variable results did not allow for generalizations to be made, but indicated that additional testing should be conducted. However, Minnesota officials elected to have food pantries destroy any remaining donated venison to prevent the meat from being served to at-risk persons such as pregnant women and children under seven years of age.

In Wisconsin, the Division of Public Health asked food pantries to hold venison pending completion of lead analyses of samples. Subsequent testing showed that approximately 16% of 183 samples of venison collected from food pantries and processors contained detectable levels of lead, while 8% of venison samples donated by Department of Natural Resources staff tested positive. Additional work, including testing of food pantry venison this fall is planned. In the meantime, health officials have recommended that venison from food pantries not be consumed or distributed unless the meat has been X-rayed and found to be free of metal fragments. It should be noted that no human cases of lead poisoning have ever been associated with consumption of hunter-killed game in Minnesota, North Dakota, Wisconsin, or elsewhere.

On June 4, 2008, individuals from Iowa, Michigan, Minnesota, Missouri, North Dakota, South Dakota, and Wisconsin attended a regional meeting in Minneapolis, Minnesota, to address the issue of lead fragments in venison. Representatives from wildlife, public health, and food safety agencies, as well as individuals representing hunting, ammunition, and food processing interests, met to develop a consistent and coordinated approach to assist regulators, hunters, and processors in answering questions and provide useful information about managing risks associated with lead fragments in hunter-harvested deer.

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Key messages developed at the meeting include, but are not limited to:

- Lead particles found in hunter-harvested venison have not been linked to any illnesses.
- The neurotoxicity of lead depends on the level and frequency of exposure. It is particularly harmful to children under seven years old and pregnant women, who should not consume venison if there is any concern.
- Physiological effects may occur when levels are below those that would cause noticeable signs of sickness.
- Initial tests suggest that ground venison has a higher tendency to contain lead particles, while they are rare in whole-muscle cuts.
- Hunters should be provided with guidelines and suggestions to eliminate or minimize the potential risk of consuming lead fragments. Recommendations include:
  - use ammunition less prone to fragment
  - discard deer with excessive shot damage
  - trim a generous distance from the wound channel
  - discard meat that is bruised, discolored or contains hair, dirt, or bone fragments

Many states have programs in which venison from hunter-killed animals is donated to food pantries that distribute it to low income families. The goal of these programs is to provide a safe and nutritious source of protein to those in need. In addition, venison donation programs are tools that help wildlife officials manage wild deer populations. Because of the importance of venison donation programs to human nutrition and wildlife management, midwestern and other states are developing and conducting additional studies to further assess the issue, determining research needs, and establishing guidelines for hunters, meat processors, and consumers to eliminate or minimize lead exposure from processed game. More information on lead in donated venison can be found at the websites of the ND DOH ([www.ndhealth.gov](http://www.ndhealth.gov)) and MDA ([www.mda.state.mn.us](http://www.mda.state.mn.us)). (Prepared by John Fischer)

## The Threat of Plague

Plague is an ancient disease that has been cycling through rodent populations, with an occasional spill over into carnivore and human populations, since at least 1320 BC. Recent events, including the death of a wildlife biologist at Grand Canyon National

Park, and an outbreak threatening reintroduced black-footed ferrets in South Dakota, have brought plague back into focus as a deadly zoonotic disease. Understanding its natural history and practicing appropriate preventive measures are essential to decreasing plague's impact on wildlife, pets, and humans.

Plague is caused by the bacterium, *Yersinia pestis*, and affects animals and people in several countries in Africa, Asia, North America, and South America. Plague is endemic in parts of the western United States. At the root of most plague outbreaks are diseased rodents and their fleas. Wildlife or domestic animals that eat the rodents or are exposed to their fleas are at risk of becoming infected. In addition, people that come in frequent contact with infected wildlife or domestic animals and their fleas are more likely to be exposed to plague and should take proper precautions. Clinically, plague occurs in three forms in susceptible mammals, including humans: bubonic (inflamed lymph nodes known as buboes); pneumonic (infection of the lungs, with coughing, chest pain, and difficulty breathing); and/or septicemic plague (bacteria replicating in the blood, leading to fever, shock, and hemorrhages in skin and other organs).

Bubonic plague typically occurs following a flea bite and is the most common form of the disease. It does not spread to other animals through casual contact. Septicemic plague can be secondary to bubonic plague and can spread to infect the lungs. Pneumonic plague is the rarest form, but it is the deadliest. It is especially dangerous because the bacteria can be aerosolized and spread from one individual to another.

Between periodic outbreaks, plague circulates in rodent populations. The fleas that feed on the infected rodents carry the disease to other rodent populations or to unsuspecting wildlife, pets, or humans. The domestic rat flea (*Xenopsylla cheopis*) is the primary vector of plague in Asia, Africa, and South America. In the southwestern United States, rock squirrels and their fleas (*Diamanus montanus*) are the most frequent source of infection for humans. On the Pacific coast, the California ground squirrel and its fleas are the most common source. Other hosts include wood rats, commensal rats, antelope ground squirrels, chipmunks, mice, and prairie dogs (both white-tailed and black-tailed). Certain rodent species, such as prairie dogs, suffer high mortality in the face of the

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disease, while species such as deer mice and voles can be asymptomatic reservoir hosts and can maintain the disease agent in wildlife populations. In addition, rodent fleas can survive for several months in abandoned rodent burrows, allowing the plague organism to remain latent in an area.

Mammals other than rodents, including lagomorphs, felids, canids, mustelids, and some ungulates, have been naturally infected with *Y. pestis*. Birds and other non-mammalian vertebrates appear to be resistant. Mammals that feed upon infected rodents or are present in their territory are at the greatest risk for acquiring the disease.

Domestic animals also can become exposed to plague by eating rodents or by infestation with rodent fleas. Pets with plague, especially cats and occasionally dogs, can serve as a source of infection for their owners, veterinary staff, and shelter workers. Cats with plague commonly have abscessed lymph nodes in the submandibular region from oral exposure via consumption of infected prey. Cats also can acquire the pneumonic form of plague, which they can spread via aerosol. In domestic animals, risk factors for infection include living in or visiting an area where plague is endemic, exposure to a rodent or rabbit carcass, access to wild rodent or rabbit habitat, and infestation by fleas. Public health veterinarians recommend regular trips to the veterinarian, flea control, and preventing pets from roaming and hunting.

Plague is zoonotic, meaning it can spread from animals to humans. Pet owners, biologists, hunters, naturalists, and animal health providers in plague-endemic areas are at an increased risk of acquiring this disease. Approximately 10 human cases are reported every year in the United States, primarily from Arizona, California, Colorado, and New Mexico. It is of utmost importance for individuals living and working in these areas to be aware of zoonotic diseases such as plague and to be protected against infection. The plague bacterium spreads quickly through the body and has an incubation period of 2 and 6 days. The treatment for plague in humans consists primarily of antibiotic therapy. However, to be most effective it is critical that treatment be initiated within 24 hours of the onset of symptoms. The U.S. Public Health Service requires that all suspected plague cases be reported to local and state health departments and that the diagnosis be confirmed by the U.S. Centers for Disease Control and Prevention (CDC).

Based on CDC recommendations, field conditions should be evaluated for risk (exposure to rodents and their fleas, or the possibility for aerosolization of material from infected animals). When handling animals, traps, and samples or performing necropsies, contact with animal fluids and excreta should be avoided, and appropriate clothing, boots, goggles, and impermeable gloves should be worn. Insect repellent should be applied before field work and before handling animals or nesting material. Personal protective equipment such as goggles and a mask or a face shield may be necessary especially during necropsy. Wildlife biologists should contact the health and safety personnel at their institution to discuss specific precautions for their work. For more information, see the winter 2007 issue of *The Wildlife Professional* (Volume 1, Issue 4).

Plague is an important zoonotic wildlife disease that will continue to circulate in rodent populations. Because of the natural history of the disease agent, plague should remain on the list of differential diagnoses for mammals and humans in endemic areas. Precautions should be taken to avoid exposure to the hosts and vectors of plague in order to prevent further spread of the disease. For more information on plague and how to minimize the risk of human exposure, visit the CDC plague homepage <http://www.cdc.gov/ncidod/dvbid/plague/>. Prepared by Joy Gary, Colorado State University College of Veterinary Medicine and Biological Sciences, Class of 2009)

## **Recent Cases of Plague in the U.S.**

On May 15, 2008, the U.S. Centers for Disease Control and Prevention (CDC) confirmed the presence of sylvatic plague in black-tailed prairie dogs in the Conata Basin area of Buffalo Gap National Grasslands in southwestern South Dakota. The outbreak is threatening one of the most robust populations of free-ranging black-footed ferrets in North America. The threat is two-fold: ferrets are highly susceptible to the disease, and surviving ferrets may lose their primary food source. Initial investigation and U.S. Forest Service mapping suggested that approximately 4,000 acres of prairie dog habitat was involved in the epizootic; however, within a few weeks the affected area increased to more than 9,000 acres.

After the reintroduction of ferrets to this area in 1997, the South Dakota population thrived and is

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considered to be one of the most successful ferret recovery efforts in the country. It was speculated that much of this success was due to an absence of sylvatic plague in the South Dakota recovery area, because mortality in prairie dog colonies often approaches 100% during plague epizootics. Sylvatic plague is known to have severely complicated black-footed ferret recovery in other areas for the past 27 years. Unfortunately, plague was detected in the Conata Basin recovery area in 2004.

The ferret population in Conata Basin was estimated at 290 individuals in the fall of 2007. The extent of the mortality sustained since then is not known at this time; however, it is known that ferrets live among and prey upon many of the prairie dog colonies already impacted by the disease. Shortly after the detection of plague in mid-May 2008, multiple government agencies, private organizations, and conservation groups collaborated to implement management strategies in order to minimize mortality of both prairie dogs and ferrets. By the end of May, biologists began dusting prairie dog burrows with powdered insecticides in an attempt to reduce flea populations and slow transmission of the disease. Furthermore, ferrets are being captured and vaccinated in order to minimize mortality. Forty individuals had been vaccinated as of July 15, 2008. The plague vaccine being used is a modification of a human vaccine developed by the U.S. Army Medical Research Institute for Infectious Disease. The U.S. Geological Survey's National Wildlife Health Center (NWHC) has been modifying and testing the vaccine for use in wildlife. A recent study evaluated the use of this vaccine in black-footed ferrets and yielded promising results. In addition, the NWHC currently is trying to develop an oral vaccine for wildlife that could be put into a bait, thereby making vaccination much less labor intensive. The USGS also is evaluating the efficacy of the disease management strategies used during this epizootic, as this is the first time concurrent insecticide application and vaccination have been used in an outbreak of this magnitude. The data obtained will provide valuable information for managing future plague outbreaks.

It is important for wildlife professionals to use the utmost care when working with wild rodents, carnivores, and feral cats in plague-endemic regions. Sadly, on November 2, 2007, a 37-year-old wildlife biologist working for the U.S. National Park Service (NPS) in the Arizona section of Grand Canyon National Park died from pneumonic plague after performing a necropsy on an infected mountain

lion. He began exhibiting flu-like symptoms three days after exposure and was found dead in his home several days later. This serves as a tragic reminder of the risks that this zoonotic pathogen poses, not only to wildlife health but also to human health. People living, working, or visiting places where plague is known to exist should take proper precautions to minimize their risk of acquiring the disease. The NPS has a fact sheet on plague that can be found on their website at [www.nps.gov/public\\_health/inter/info/factsheets/fs\\_p\\_lague.htm](http://www.nps.gov/public_health/inter/info/factsheets/fs_p_lague.htm). (Prepared by Mark Ruder)

## Raccoon Roundworm – Public Health Update 2008

The raccoon roundworm, *Baylisascaris procyonis*, is an intestinal parasite that can cause clinical disease in numerous species of animals other than raccoons. Raccoons carry the worms but are not clinically affected. Raccoons hosting mature roundworms pass *B. procyonis* eggs into the environment in their feces. Depending on environmental conditions, eggs larvate and become infective in 11-14 days. If another raccoon ingests the eggs, the roundworm completes its normal life cycle and matures in the digestive tract of the infected animal. However, following ingestion of larvated eggs by an aberrant host, the normal life cycle of the parasite is disrupted. Numerous avian and mammalian species infected with *B. procyonis* may display different manifestations of the disease, depending on the number of larvae present and the tissues that are damaged by larval migration.

The three common forms of the disease are based on the location of the roundworm larvae: visceral larval migrans (VLM), ocular larval migrans (OLM), and neural larval migrans (NLM). Larvae emerge in the stomach of the aberrant host within 24 hours and aggressively migrate throughout the host's body. Some of these larvae become encapsulated in various tissues, but others may continue to migrate through the central nervous system tissue, causing neurological problems. These forms of the disease also are seen in humans and other animals infected with other roundworm species, such as those of dogs and cats.

Since 2005 there have been 15 confirmed human cases of VLM and NLM in the United States due to *B. procyonis*, resulting in five deaths and several cases of mental impairment in young children. Numerous cases of OLM also have been

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diagnosed, frequently impairing vision or causing total blindness. The majority of cases occur in young children because they are more likely to ingest *Baylisascaris* eggs by placing contaminated hands, sand, dirt, or other objects or material in their mouths.

Historically, *B. procyonis* has been found more commonly in the Northeast, Midwest, and a few western states, and prevalence can be as high as 90%. The parasite has been less frequently reported in the Southeast, with rare reports coming from the mountainous regions of Kentucky, Tennessee, Virginia and West Virginia. However, in 2002 researchers at the U.S. Centers for Disease Control and Prevention (CDC) found *B. procyonis* in 11 of 50 (22%) raccoons captured in DeKalb County, Georgia, one of the metro Atlanta counties. During the same year, *B. procyonis* was found in a young raccoon being cared for by a wildlife rehabilitator in Athens, Georgia. Additional positive raccoons in Athens, Georgia, were detected in 2006 during routine necropsies of clinical cases submitted to SCWDS.

SCWDS recently initiated a study in conjunction with the University of Georgia's Warnell School of Forestry and Natural Resources to determine the distribution and prevalence of this important zoonotic parasite in the Athens, Georgia, area. Since August 2007, we have found *B. procyonis* in 7 of 66 raccoons (10.6%), from the Athens area.

The presence of *B. procyonis* in the Athens and Atlanta areas is important because of the health implications for wildlife, humans, and domestic animals. It also is important to note that all positive animals have been captured in urban or suburban areas. Raccoons are well adapted to human activity and disturbance, and they can quickly contaminate areas being used by people and urban-dwelling animals. It has been estimated that a single adult female *B. procyonis* worm can shed 115,000-179,000 eggs per day, so when the worms are present in an urban environment there is substantial risk of human infection.

In general, ascarid eggs are highly resistant to desiccation and chemical disinfection, and *B. procyonis* is no exception. *B. procyonis* eggs have been found to be viable in the environment for more than two years, and when kept at 4 degrees C they can remain infective for 9-12 years. Currently, the only successful ways to decontaminate an area are

to completely burn the area or saturate the area with boiling water, followed by an application of a strong lipophilic solvent such as a 50/50 solution of xylene and absolute alcohol. These decontamination treatments are difficult to perform when raccoon feces contaminates wooden decks, roofs, chimneys, or other such areas. Working with researchers at the CDC, SCWDS is exploring alternative methods to decontaminate areas that have been contaminated with raccoon feces.

The only effective way to prevent contamination of an area with raccoon feces and *B. procyonis* is to restrict raccoon activity in the area. People should secure pet food and garbage in proper storage containers, hollow trees should be removed from the property, and attics and crawl-spaces under the house or deck should be properly barricaded. Wildlife rehabilitators should be aware that raccoons may be infected and take appropriate precautions, including using dedicated pens that will house only raccoons, and when these animals have been released the areas should be properly decontaminated. Numerous outbreaks of *Baylisascaris* larval migrans have been reported in animals in rehabilitation centers that housed animals in cages or pens that had previously housed infected raccoons. (Prepared by Emily Blizzard and Michael Yabsley)

## Avian Pox in a Nestling Bald Eagle

Throughout the spring of 2008, many wildlife enthusiasts around the world were able to view the daily activities of a pair of bald eagles and a nestling in Norfolk, Virginia, via a popular web cam, known as the "WVEC.com Eagle Cam". The nest is located at the Norfolk Botanical Gardens, which co-sponsors the webcam with the Virginia Department of Game and Inland Fisheries (VDGIF).

On May 16, 2008, viewers of the Eagle Cam first reported a grape-size mass on the nestling's beak, and on May 22 the bird was removed from the nest and examined by Dr. Jonathan Sleeman of the VDGIF. A 3-cm diameter, soft, lobulated mass protruded from the left side of the upper beak and occluded air flow through the left nares (Figure 1). Dr. Sleeman collected a biopsy of the lesion and submitted it to SCWDS. Since the lesion was beginning to distort the conformation of the beak and was growing rapidly, officials decided to remove the bird from the nest. The nestling was admitted to The Wildlife Center of Virginia in Waynesboro,

Virginia, where the bird received supportive care and further diagnostics, while officials awaited results of the biopsy. Microscopic examination of the biopsy revealed that the mass was characteristic of avian pox, and a canarypox virus was isolated from fresh tissue obtained during the biopsy (Figure 2).



Figure 1 Mass on upper beak of nestling bald eagle from Norfolk, Virginia, after biopsy was performed. Photo by Mark Ruder

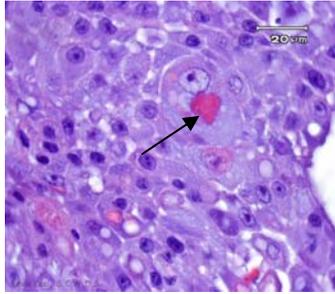


Figure 2. Hyperplastic epithelial cells are variably sized and often contain Bollinger bodies (arrow). Photo by Kevin Keel

Avian pox is caused by viruses in the genus *Avipoxvirus* of the family *Poxviridae* and is a significant infectious disease for many species of wild birds in multiple orders. Some poxvirus strains exhibit high host specificity, while others have a very broad host range. The geographic distribution of avian poxviruses is nearly worldwide, but disease appears to be more prevalent in warm, temperate regions. The primary reservoir is infected individual birds, and transmission is primarily by blood-sucking arthropods or via close contact. Thus, transmission of avian pox is enhanced by increasing the density of the host and/or vector; therefore the prevalence of disease tends to be higher in captive birds (e.g., zoos, rehabilitation facilities, game farms, and captive rearing facilities) and in flocking species in warm climates.

Host susceptibility also plays a critical role in the epizootiology of the disease. Two major clinical manifestations of avian pox exist: cutaneous pox is typified by proliferative wart-like growths on the nonfeathered regions of the skin, while the diphtheritic form results in moist, necrotic lesions of the mucous membranes, especially in the mouth and upper GI tract. The cutaneous form predominates in wild birds, and most infections are mild, as lesions generally dry up, scab over, and fall off uneventfully. However, pox lesions that interfere with vision, respiration, prehension of food, or ambulation often lead to death, directly or indirectly. Although generally a self-limiting disease, avian pox can negatively impact certain wild populations.

There is no risk to humans or other mammals; however, some poxvirus strains found in wild birds can infect domestic poultry. Vaccination of poultry is considered protective.

The eagle remains at the Wildlife Center of Virginia, and the pox lesion had regressed with supportive care by early July; however, the bird's beak became severely mal-aligned due to disruption of germinal tissue responsible for beak growth. On July 12, 2008, surgery was performed to correct the mal-alignment. The bird's suitability for release currently is being evaluated. Options for captivity will be explored if the bird is deemed non-releasable. For more information regarding the Eagle Cam or this bird, visit the Eagle Cam website at <http://www.wvec.com/cams/eagle.html> and click on the links for updates from the VDGIF and the Wildlife Center of Virginia. (Prepared by Mark Ruder)

## A Note to Our Readers

We thank you for your sustained interest in our quarterly newsletter, the SCWDS BRIEFS. Our latest issue marked the beginning of the 24<sup>th</sup> year of publication. We continue to receive positive feedback from many readers, which lets us know that we are still providing some items of interest in each issue.

One difficult aspect of putting out a publication such as the BRIEFS is maintaining the mailing list. We want to reach as many of you as we can, but can do so only if you let us know you want to be included on the mailing list, notify us of any address changes, or inform us of someone else you know who would like to be added to the mailing list. Also, if you are in an administrative job and have people working for you that you want to be added, please let us know. Of course, if you want to reduce the volume of mail coming into your home or office you may opt to be removed from the regular mailing list and have your name added to our email list to be informed when each new issue is posted on our website. This way, you usually can read the newsletter at least 10 days before a mailed copy would arrive. As always, if you have suggestions for improvement of the Briefs, please let us hear from you. Our goal is to provide timely information of interest to you. (Prepared by Gary Doster)

## Recent SCWDS Publications Available

Below are some recent publications authored or co-authored by SCWDS staff. Many of these publications can be accessed online from the web pages of the various journals. If you do not have access to this service and would like to have a copy of any of these papers, fill out the request form and return it to us: Southeastern Cooperative Wildlife Study, College of Veterinary Medicine, University of Georgia, Athens, GA 30602.

- \_\_\_\_ Bartholomew, J.L., S.D. Atkinson, S.L. Hallett, L.J. Lowenstine, M.M. Garner, C.H. Gardiner, B.A. Rideout, M.K. Keel, and J.D. Brown. 2008. Myxozoan parasitism in waterfowl. *International Journal for Parasitology* 38: 1199-1207.
- \_\_\_\_ Brown, J.D., D.E. Stallknecht, and D.E. Swayne. 2008. Experimental infection of swans and geese with highly pathogenic avian influenza virus (H5N1) of Asian lineage. *Emerging Infectious Diseases* 14(1): 136-142.
- \_\_\_\_ Brown, J.D., D.E. Stallknecht, S. Valeika, and D.E. Swayne. 2007. Susceptibility of wood ducks to H5N1 avian influenza virus. *Journal of Wildlife Diseases* 43(4): 660-667.
- \_\_\_\_ Davis, A.K., M.J. Yabsley, M.K. Keel, and J.C. Maerz. 2007. Discovery of a novel alveolate pathogen affecting southern leopard frogs in Georgia: Description of the disease and host effects. *EcoHealth Journal Consortium* 4: 310-317.
- \_\_\_\_ Fischer, J.R. 2008. Why wildlife health matters in North America. Pp. 107-114. *In* E. Fearn and K.H. Redford (eds.), *State of the Wild: A Global Portrait of Wildlife, Wildlands, and Oceans*. Island Press, Washington, DC. 286 pp.
- \_\_\_\_ Gerhold, R.W. and M.J. Yabsley. 2007. Toxoplasmosis in a red-bellied woodpecker (*Melanerpes carolinus*). *Avian Diseases* 51: 992-994.
- \_\_\_\_ Gerhold, R.W., A.B. Allison, D.L. Temple, M.J. Chamberlain, K.R. Strait, and M.K. Keel. 2007. Infectious canine hepatitis in a gray fox (*Urocyon cinereoargenteus*). *Journal of Wildlife Diseases* 43(4): 734-736.
- \_\_\_\_ Hall, C.R., C. Polizzi, M.J. Yabsley, and T.M. Norton. 2007. *Trypanosoma cruzi* prevalence and epidemiologic trends in lemurs on St. Catherines Island, Georgia, USA. *Journal of Parasitology* 93(1): 93-96.
- \_\_\_\_ Hanson, B.A., M.P. Luttrell, V.H. Goekjian, L. Niles, D.E. Swayne, D.A. Senne, and D.E. Stallknecht. 2008. Is the occurrence of avian influenza virus in charadriiformes species and location dependent? *Journal of Wildlife Diseases* 44(2): 351-361.
- \_\_\_\_ Harvey, J.W., M.R. Dunbar, M.J. Yabsley, and T.M. Norton. 2007. Laboratory findings in acute *Cytauxzoon felis* infection in American pumas (*Puma concolor*). *Journal of Zoo and Wildlife Medicine* 38(2): 285-291.
- \_\_\_\_ Kelly, R., D.G. Mead, and B.A. Harrison. 2008. Discovery of *Culex coronator* Dyer and Knab (Diptera: Culicidae) in Georgia. *Proceedings of the Entomological Society of Washington* 110(1): 258-260.
- \_\_\_\_ Manangan, J.S., S.H. Schweitzer, N. Nibbelink, M.J. Yabsley, S.E.J. Gibbs, and M.C. Wimberly. 2007. Habitat factors influencing distributions of *Anaplasma phagocytophilum* and *Ehrlichia chaffeensis* in the Mississippi Alluvial Valley. *Vector-Borne Zoonotic Diseases* 7(4): 563-573.
- \_\_\_\_ Munderloh, U.G., M.J. Yabsley, S.M. Murphy, M.P. Luttrell, and E.W. Howerth. 2007. Isolation and establishment of the raccoon *Ehrlichia*-like agent in tick cell culture. *Vector-Borne and Zoonotic Diseases* 7(3): 418-425.
- \_\_\_\_ Stallknecht, D.E. Impediments to wildlife disease surveillance, research, and diagnostics. 2008. *In* *Current Topics in Microbiology and Immunology*. J.E. Childs, J.S. Mackenzie, and J.A. Richt (Editors). CTMI Volume on Wildlife and Emerging Zoonotic Diseases: The Biology, Circumstances, and Consequences of Cross-Species Transmission. Springer Verlag, Berlin 315: 445-461.
- \_\_\_\_ Wiley, F.E., S.B. Wilde, A.H. Birrenkott, S.K. Williams, T.M. Murphy, C.P. Hope, W.W. Bowermann, and J.R. Fischer. 2007. Investigation of the link between avian vacuolar myelinopathy and a novel species of cyanobacteria through laboratory feeding trials. *Journal of Wildlife Diseases* 43(3): 337-344.

\_\_\_\_\_ Wimberly, M.C., M.J. Yabsley, A.D. Baer, V.G. Dugan, and W.R. Davidson. 2008. Spatial heterogeneity of climate and land cover constraints on the distributions of tick-borne pathogens. *Global Ecology and Biogeography* 17: 189-202.

\_\_\_\_\_ Yabsley, M.J., A.D. Loftis, and S.E. Little. 2008. Detection of an *Ehrlichia* sp. that is closely related to *Ehrlichia ruminantium* in white-tailed deer (*Odocoileus virginianus*). *Journal of Wildlife Diseases* 44(2): 381-387.

\_\_\_\_\_ Yabsley, M.J., J. McKibben, C.N. MacPherson, et al. 2008. Prevalence of *Ehrlichia canis*, *Anaplasma platys*, *Babesia canis vogeli*, *Hepatozoon canis*, *Bartonella vinsonii berkhoffi*, and *Rickettsia* spp. in dogs from Grenada. *Veterinary Parasitology* 151: 279-285.

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