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## SCWDS BRIEFS: Volume 15, Number 1 (April 1999)

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# SCWDS BRIEFS

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A Quarterly Newsletter from the  
**Southeastern Cooperative Wildlife Disease Study**  
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## Aflatoxicosis in Louisiana Geese

A significant mortality event involving geese was observed by personnel from the Louisiana Department of Wildlife and Fisheries and the U.S. Fish and Wildlife Service starting in mid-November 1998 and continuing through early March 1999. Sick and dead geese were observed in corn fields and adjacent flooded rice fields in several northeast Louisiana parishes, and estimated losses exceeded 10,000 birds. Most of the geese affected were snow (and blue) geese, with lesser numbers of Ross' and white-fronted geese involved. The geese were observed feeding in corn fields that had not been harvested due to high levels of aflatoxin; most of the affected fields had been disked or mowed. Live geese and goose carcasses were collected by biologists and sent to the National Wildlife Health Center (NWHC) in Madison, WI, and SCWDS for examination. At necropsy, degenerative and reactive liver lesions were observed in most geese, and some geese had degenerative lesions in other organs including the spleen, pancreas, and kidneys. These lesions were characteristic of aflatoxicosis, and aflatoxin or its metabolites were detected in stomach contents from 2 snow geese at the NWHC. Analysis of corn from fields where the geese had been feeding and where geese were found dead yielded levels of aflatoxin as high as 8,200 ppb, which greatly exceeds USDA standards.

Aflatoxins are produced by several fungal organisms in the genus *Aspergillus*. The toxins are produced when the fungi grow on cereal grains, including corn, under warm, humid conditions. The toxins are more commonly produced

in grains during storage but can be produced when the fungi grow on grains in the field. Aflatoxins principally affect the liver and can lead to degenerative lesions and reactive changes serious enough to cause death. These toxins also can affect a variety of other organ systems, including the immune system, and there are both acute and chronic forms of toxicosis with these compounds. Most species of mammals and birds are susceptible to aflatoxicosis, with great variability among species and especially between age classes of animals. Birds and monogastric mammals are more susceptible than ruminants, and younger animals are generally more susceptible than adults. There is no medical treatment for aflatoxicosis, and prevention is the key to minimizing wildlife losses.

Losses in wildlife species due to aflatoxicosis have been described; however, large mortality events like the one observed with the geese in Louisiana are rare. Extrapolation from experimental trials in domestic animals and infrequent reports of wildlife mortality events in the field have been the basis of a SCWDS position that grains known to be contaminated with aflatoxin in excess of levels allowable in animal feeds (up to 300 ppb) should not be used in wildlife feeding programs. However, a more problematic issue is what to do with condemned standing crops because so little data exist. Two published cases of waterfowl mortality in Texas revealed that the aflatoxin levels in waterfowl crop (stomach) contents ranged from 10 to 500 ppb, while the peanuts that were tested from one field contained only 110 ppb. In Florida, bobwhite quail from corn fields with mean aflatoxin levels of over 1,000 ppb had aflatoxin

levels in crop contents that averaged only 63 ppb. Nevertheless, some of the quail had liver lesions consistent with aflatoxicosis.

The devastating losses in Louisiana, although unfortunate, provide wildlife managers with an important new reference point documenting the risks of aflatoxicosis in waterfowl. It is apparent that very high levels of aflatoxin (several thousand ppb) in standing crops can present a significant hazard to waterfowl. The Louisiana Department of Agriculture and Forestry estimated that 360,000 acres of corn failed in 1998 due to drought, and much of this grain probably contained high aflatoxin levels. Furthermore, the aflatoxin level present in the corn when farmers made the decision not to harvest was probably lower than that which developed as the corn continued to mold in the field.

The aflatoxin crisis is over for now, but history tells us it will recur, if not in Louisiana, then somewhere else in the South. Mortality events like this raise many questions about what preventive actions can be taken when vast acres of crops are involved. Can programs be developed to help farmers harvest crops that have no commercial feed value? Can contaminated corn be salvaged for uses other than animal feeds? Is it possible to completely cover grains by plowing or will plowing enhance availability to wildlife species? And finally, will hazing or other deterrent measures work? These are questions that need to be examined, and wildlife management agencies should develop partnerships with the agricultural community to address these issues. (Prepared by Todd Cornish and Victor Nettles)

### **Prevention of Meningeal Worm Infection**

White-tailed deer are the normal host for the meningeal worm (*Parelaphostrongylus tenuis*). Adult worms reside in the meninges of the brain of white-tailed deer and produce eggs that are carried to the lungs via blood circulation. In the lungs the eggs hatch and produce larvae that enter airways and migrate to the larynx where they are swallowed and passed in the feces of deer. The

larvae then penetrate and grow within required intermediate hosts, i.e., terrestrial slugs and snails. The life cycle is completed when deer accidentally ingest infected mollusks while browsing.

Infection typically causes no clinical disease in white-tailed deer, but when infected snails and slugs are ingested by other cervids or ungulates, severe neurologic disease can develop due to aberrant parasite migration in the spinal cord and brain. Clinical signs of meningeal worm infection (parelaphostrongylosis) in these abnormal hosts are variable and include weakness, lameness, circling, blindness, head tilt, paralysis, abnormal behavior, and sometimes death. Species known to be susceptible include other native and exotic cervids (black-tailed deer, mule deer, elk, caribou, moose, fallow deer, axis deer), domestic livestock (goats and sheep especially), llamas, and exotic antelope species (blackbuck, sable antelope, and others). All exotic ungulates should be regarded as potentially susceptible.

There currently is no single effective method of preventing meningeal worm infection in ungulates that share range with infected white-tailed deer. Eliminating white-tailed deer via lethal methods is not a practical or effective method of control in most situations. Limiting deer access to pastures can reduce the potential for environmental fecal contamination, but it is important to remember that the mollusk intermediate hosts actually harbor the infective larval stages for up to a year. Deer-proof fencing is one option for small-scale operations, and it is sometimes possible to make pastures less appealing by limiting trees and cover favorable to deer. Furthermore, feeding livestock and captive exotic species in an enclosed building will limit attraction of deer to available feed.

Reducing exposure to infected slugs and snails is important in preventing meningeal worm infection. Susceptible animals should not be allowed access to pastures that contain thick vegetation or moist shaded areas favorable to the snail and slug intermediate hosts. Feeding

animals in elevated bunks rather than directly on the ground is also advisable. A variety of techniques has been suggested to control slugs and snails in pastures, including: destruction of slug and snail habitat; erection of physical barriers around the perimeter of the pasture; trapping mollusks with home-made or commercial traps; application of commercial molluscides; and even manually collecting slugs and snails. More information on methodology is available at the following website: [www.ipm.ucdavis.edu/PMG/PESTNOTES/pn028.html].

A final potential method for prevention of meningeal worm infection in susceptible animals is prophylactic treatment with anthelmintics or deworming drugs. At the present time, no studies have been performed that demonstrate complete prevention of infection with these agents. Despite this fact, some veterinarians currently prescribe daily oral administration of pyrantel tartrate or subcutaneous injections of ivermectin every 3 weeks during the spring, summer, and fall as a preventive. More research needs to be performed to identify effective deworming programs, and producers or owners interested in these methods are encouraged to contact their veterinarian for specific advice and recommendations. (Prepared by Todd Cornish and Cariann Turberville)

### **Meningeal Worms in Cattle**

It was once commonly believed that cattle were refractory to infection with the deer meningeal worm (*Parelaphostrongylus tenuis*) and subsequent development of clinical disease (parelaphostrongylosis). However, in the last 2 years there have been 2 reports of clinical disease in bovine calves due to the meningeal worm. A 7-month-old Angus heifer in Virginia and a 3-month-old mixed-breed bull calf in Michigan developed central nervous system lesions with associated neurologic signs, and *P. tenuis* was recovered from each animal at necropsy. Given the rarity of such reports, clinical infection appears to be very uncommon in cattle, and cattle owners should not be alarmed. Parelaphostrongylosis should be considered

among the possible diagnoses in young cattle with neurologic signs; however, based upon the very low number of documented cases of clinical disease, cattle still are considered highly resistant to infection with this parasite. (Prepared by Todd Cornish)

### **Composted Chicken Litter Safer for Wildlife**

The Southeast is this Nation's largest poultry producer. Along with this distinction comes the dilemma of what to do with tons of poultry litter, which is composed of manure, bedding material, and wasted feed. It is high in nitrogen and is used extensively as a low-cost fertilizer or ruminant feed additive. For wildlife managers, the advantages of recycling poultry manure come with concerns that spreading poultry litter on fields may expose wildlife to infectious diseases and parasites. SCWDS frequently receives questions about the risks posed by poultry litter and ways to reduce the risks.

Poultry scientists have shown that many organisms pathogenic for domestic chickens and turkeys can be present in poultry litter. Fortunately, some of these pathogens are highly sensitive to sunlight and/or drying and will perish quickly in open fields. Others can persist for longer periods, some up to 4 years. Specific information on the disease risks of poultry litter to wildlife is scarce. A SCWDS study (see SCWDS BRIEFS Vol. 8, No. 1) evaluated the risk of histomoniasis (blackhead disease) transmission from domestic chickens to wild turkeys. For histomoniasis, it was determined that manure from young broiler chickens was safe but manure from older chickens such as layers or breeder birds could be infected. Therefore, wildlife managers have an estimate of risk for one important avian disease, but the potential risks associated with other pathogens are poorly understood.

Composting poultry litter has gained popularity in recent years because the process reduces volume and provides a more nutrient-rich fertilizer. Furthermore, it is likely that composting will eliminate many pathogenic organisms. Studies

on human pathogens in sewage sludge have shown that most bacteria, viruses, protozoan cysts, and helminth eggs are eliminated when composting temperatures are maintained at 140°F to 158°F for 3 days. There has not been a similar comprehensive report for poultry pathogens, although individual studies contain similar findings. For example, *Pasteurella multocida*, the bacterium that causes fowl cholera, can persist in soil for up to 3 months but is destroyed by composting in just 22 hours. Likewise, the viruses that cause infectious bursal disease (Gumboro) and Newcastle disease are killed by composting. Even hardy protozoan organisms such as coccidial oocysts are inactivated by composting. Further research will be required to more clearly define the value of composting to destroy other avian pathogens, but available data indicate that composting will reduce risk substantially. Therefore, we favor the use of composted poultry litter over uncomposted litter in areas frequented by wildlife. Practical guidelines for composting poultry litter are available in the 1992 publication entitled *On Farm Composting Handbook (NRAES-54)* which is available for \$20 from Northeast Regional Agricultural Engineering Service, Cooperative Extension Service, Department of Agricultural and Biological Engineering, Cornell University, Ithaca, NY 14853-5701 (Telephone 607-255-7654). Key portions of this publication can be viewed via the internet at [[www.cals.cornell.edu/dept/compost/OnFarmHandbook/](http://www.cals.cornell.edu/dept/compost/OnFarmHandbook/)], and other information on composting is provided at [[www.cals.cornell.edu/dept/Composting\\_Homepage.html](http://www.cals.cornell.edu/dept/Composting_Homepage.html)]. (Prepared by Randy Davidson)

### **Hazards of Deer Relocation**

The following article appeared in the April issue of *The Missouri Conservationist* (Vol. 60, No. 4), the monthly publication of the Missouri Department of Conservation (MDC). The article describes some of the pitfalls encountered when the St. Louis suburb of Town and Country started a capture and relocation program in an effort to reduce the local deer population.

### **"Mixed Success**

"Missouri's first experiment with trapping and relocation to control suburban deer numbers has not gone as smoothly as hoped, but the trial is yielding information that will help other communities decide how to deal with similar problems. Town and Country officials sought and received permission to have deer trapped and removed by a private contractor at city expense. The Missouri Department of Conservation is monitoring the program. Between late December 1998 and mid-February 1999, 51 female deer and 29 male deer were captured with nets and taken from Town and Country to a conservation area south of St. Louis. The city is attempting to remove 122 does each year for the next two or three years to help reduce its deer population – currently estimated at approximately 600 – by half. After that, smaller removals will be needed periodically to maintain the herd at a level where the risk of deer-vehicle accidents, browsing on landscape plantings and other deer-related concerns are acceptable to city residents. The Conservation Department has tracked the movements of relocated deer with radio transmitter collars. By late February 19% of the relocated deer had died of capture myopathy. Town and Country undertook the trapping and relocation program because a majority of citizens surveyed did not want deer killed to solve the overpopulation problem. The cost of deer trapping and removal so far has been more than \$350 per deer. The private contractor who conducted the first round of deer trapping has withdrawn from the project. Town and Country has been unable to find another contractor willing to do the work."

Personnel from MDC performed necropsies on dead relocated deer and forwarded tissues to SCWDS for examination. SCWDS diagnosticians detected capture myopathy lesions in 16 of 17 deer examined. Several of the deer submitted for necropsy had case histories indicative of bobcat predation, and it was likely that capture myopathy in these deer caused them to be more susceptible to predation.

Capture myopathy is a complex degenerative disease of skeletal muscle associated with the increased muscular exertion and over stimulation of the nervous system as a result of the capture, restraint, and transportation of animals (see SCWDS BRIEFS Vol. 9, No. 4). Illness and death may result due to disruption of normal circulation, muscle tissue damage, and electrolyte imbalance. Affected animals may show muscle tremors or muscle rigidity, weakness, hyperthermia, respiratory difficulty, collapse, and acute death. Animals that do not die acutely may succumb later due to inadequate oxygen supply to the kidneys and from toxic muscle breakdown products.

The following suggestions may help reduce the occurrence of capture myopathy:

- Capture crews should be comprised of well-trained personnel.
- Enough people should be on hand to work the animals quickly and effectively, however extraneous personnel should be avoided.
- Noise and movement should be kept to a minimum.
- Blindfolding the animal may help reduce stress.
- Use of traps rather than chases will greatly reduce the animal's level of exertion. When chases are necessary, distance and speed must be minimized, and a maximum chase time should be predetermined.
- Capture should be avoided on extremely warm days. Wetting the skin of animals captured on warm days may help prevent overheating.
- Chemical immobilization can reduce stress to the animal; however, some drugs compound the problems of muscle necrosis and decreased blood pressure. The drug, dosage, and delivery system must be chosen carefully.

- The stress of transport may be minimized by proper selection of crates and crate mates. Food and water should be provided to reduce the additional stress of hunger and dehydration.
  - When captured animals are to be held in captivity, handling should be minimal during the first 2-3 weeks to allow recovery from the initial stress of capture.
- (Prepared by Joe Gaydos and John Fischer)

### **Rodenticide Hazards to Wildlife**

In the summer of 1998, SCWDS diagnosed rodenticide poisoning in gray squirrels from Richmond, VA. Two dead gray squirrels were collected by a biologist with the Virginia Department of Game and Inland Fisheries in response to reports of several dead squirrels and birds in a residential area. The carcasses of the collected squirrels were frozen and submitted to SCWDS for necropsy. Significant findings at necropsy included unclotted blood in their intestinal tracts and chest cavities, as well as scattered hemorrhages in subcutaneous tissues and various muscles. The pattern of hemorrhages observed was typical of lesions caused by anticoagulant rodenticide poisoning. Toxicologic testing revealed residues of 2 rodenticides, bromadiolone and diphacinone, in the livers of the squirrels. These rodenticides interfere with the action of Vitamin K in the production of normal blood clotting factors and predispose animals to fatal hemorrhages that can be triggered by trauma or even normal daily activities. Some of the newer anticoagulant rodenticides, including bromadiolone and diphacinone, can be fatal with only a single exposure.

Anticoagulant compounds such as bromadiolone and diphacinone are used in rat and mouse control products and are available under several trade names. There are numerous other anticoagulant rodenticides that are also commercially available (see SCWDS BRIEFS Vol. 11, No. 4). All of these compounds are non-specific and are considered toxic to other mammals and birds. These compounds must be

consumed to be effective and are typically incorporated into grain-based pellets or wax baits. Unfortunately, incorrect application of such baits makes them available to non-target species including wildlife and domestic animals. There have been numerous documented cases of such poisonings involving wildlife species ranging from raccoons and opossums to deer and even a mountain lion, and anticoagulant rodenticide toxicosis is an all-too-common event in domestic animals. Furthermore, secondary toxicosis associated with eating poisoned rodent carcasses also can lead to fatalities in wildlife species, especially raptors such as hawks and owls, and domestic animals including dogs and cats. To minimize the risk of secondary poisonings, areas that have been treated with anticoagulant rodenticide baits should be searched for dead rodents, and all carcasses found should be disposed of in a secure manner. An excellent review of anticoagulant rodenticide poisonings in New York wildlife was published recently in the *Journal of Wildlife Diseases* by Dr. Ward B. Stone and associates (Vol. 35., pp 187-193). (Prepared by Victor Nettles)

### **NWTF Information on Wild Turkey Diseases**

As many of you probably are aware, the National Wild Turkey Federation (NWTF) has developed and produced a high-quality manual called *NWTF Wildlife Bulletin* that contains useful information on various aspects of wild turkey biology and management. The *NWTF Wildlife Bulletin* format is a ringbound notebook that contains a series of brief, single subject, full-color articles. Two of these bulletins, Nos. 25 and 26, were authored by SCWDS staff members and describe avian pox and blackhead disease. Avian pox and blackhead disease generally are believed to be the 2 most common disease-related mortality factors for wild turkeys in the Southeast. These 2 bulletins are designed for use by the general public and would be an excellent resource for anyone who has to answer questions regarding disease problems of wild turkeys. A more comprehensive review of wild turkey health issues is contained in a recent

book entitled *The Wild Turkey: Biology and Management* edited by Dr. Jim Dickson. Both the Bulletin and the book are available from the NWTF, which can be contacted by calling 1-800-THE-NWTF. (Prepared by Randy Davidson)

### **SCWDS Hosts WDA Conference**

The Annual Conference of the Wildlife Disease Association (WDA) is the World's best single-source meeting for wildlife population health information. This year, the WDA Conference will be held August 8-12, 1999, at The University of Georgia's Center for Continuing Education in Athens, GA, USA. The meeting will be sponsored by SCWDS and the College of Veterinary Medicine. General sessions will begin on Monday, August 9, 1999. The program will include a Symposium on International Issues in Rabies Re-Emergence, hosted in conjunction with the Centers for Disease Control and Prevention, Atlanta, GA.

Pre-registration is \$175 for WDA members and \$225 for non-members. Late registrants will be charged an additional fee of \$50 and may have difficulties getting transportation and accommodations. The registration fee includes the Sunday evening reception, all conference sessions, lunches during the sessions, and coffee breaks and snacks. Picnic and banquet tickets are optional, but it is recommended that tickets for these events be purchased at pre-registration or at least 10 days before the meeting.

Airline service directly into Athens is provided on US Air Express from Charlotte, NC. However, Athens is only about 1.5 hours east-northeast of Atlanta, GA, and most persons traveling by air will likely fly into Atlanta. Direct ground transportation from Atlanta to Athens is available by rented car or by AAA Airport Express shuttle van. This shuttle leaves Atlanta 7 times a day and will deliver you directly to the Center for Continuing Education. For shuttle reservations, telephone 1-800-354-7874. For those who are driving, directions to

Athens and to the Georgia Center can be found at [<http://www.vet.uga.edu/par/wda2>].

For other questions, please contact Charlotte Quist or Susan Little, Local Arrangements Co-Chairs, College of Veterinary Medicine, The University of Georgia, Athens, GA 30602, USA; e-mail: [WDAmail@calc.vet.uga.edu]; Telephone: (706) 542-5349; FAX: (706) 542-5977. Additional information on the Conference including a registration form, meeting outline, area accommodations, and other items of interest can be obtained via the WDA webpage at [<http://www.vpp.vet.uga.edu/wda>] and the linked meeting website that includes all the meeting information. (Prepared by Victor Nettles)

### **Last Call for Wildlife Maps**

In past years, SCWDS produced a number of maps depicting the distribution and density of various species of big game in the United States or the Southeast. Although dated, the maps still provide valuable information. For some species, these maps still represent the most current information of its type, and the historical information may be useful to some researchers. We have a supply of some of these maps on hand which we offer to our readers free upon request. See the list below. We will ship these out until June 30, 1999. Contact us by telephone (706-542-1741), FAX (706-542-5865), or e-mail [gdoster@calc.vet.uga.edu].

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Recent back issues of *SCWDS BRIEFS* can be accessed on the Internet at [SCWDS.org](http://SCWDS.org).