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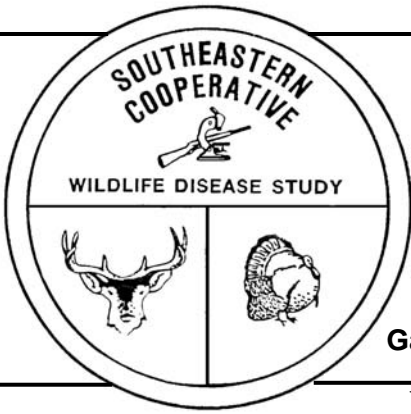
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SCWDS BRIEFS

A Quarterly Newsletter from the
Southeastern Cooperative Wildlife Disease Study
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USDA Seeks Comments on New CWD Rule

On March 31, 2009, the United States Department of Agriculture-Animal and Plant Health Inspection Service (APHIS) published a proposed rule titled *Chronic Wasting Disease Herd Certification Program and Interstate Movement of Farmed or Captive Deer, Elk, and Moose* (Federal Register Vol. 74, No. 60, pp 14495-14506). In this publication, APHIS proposes changes to the 2006 CWD rule (see SCWDS BRIEFS Vol. 22, No. 6) that would establish a herd certification program to eliminate CWD from farmed or captive cervids in the United States. APHIS plans to withdraw the 2006 CWD rule (which never went into effect) and issue a revised final rule based on the 2006 final rule and on the March 2009 proposed rule after evaluating all comments on the proposed rule.

There are several significant changes in the new proposed rule. APHIS will change the 2006 final rule to recognize state bans on cervid importation for reasons that are unrelated to CWD. However, as in the 2006 rule, the new proposed rule indicates that federal CWD requirements will preempt state CWD requirements for interstate movement of cervids.

The new proposed rule requires that captive or farmed cervids moved interstate must be from herds that have been monitored for CWD for at least five years. Previously, interstate movement could begin after one year, with the required monitoring period increasing by one year each year the program was in place, until five years of CWD herd monitoring had been achieved.

Regarding importation of farmed or captive cervids, APHIS is proposing to allow states to

elect to not receive animals from areas in proximity to CWD occurrences in wild cervids and to establish a single federal standard for such proximity in order to make the standard consistent among all states with such restrictions: The premises of origin must not be within 25 miles of an identified case of CWD in wild deer, elk, or moose, or within 25 miles of an area where CWD has become established in wild cervids, as defined by APHIS and the state. In addition, APHIS intends to amend standards for participation and enrollment in the proposed federal program by adding a provision that an application for participation may be denied if APHIS or the state determines that the applicant's herd was established after the effective date of the final rule following this proposal on a premises within 25 miles of CWD in wild cervids as defined above. Furthermore, APHIS proposes to require states to continue to conduct CWD surveillance among wild cervids in order to provide reliable data to determine the proximity of a captive or farmed cervid premises to CWD in free-ranging deer, elk, or moose. Such surveillance will be required for a state CWD program to become an Approved State CWD Herd Certification program, according to the APHIS CWD final rule.

Additional proposed changes include further defining herd inventory standards for participation in the CWD Herd Certification program by requiring an actual physical inventory of assembled animals at the time the herd is enrolled in the federal-state cooperative CWD program, in order to provide a reliable baseline record for the herd's participation. Further physical inventories would be scheduled when an APHIS employee or state representative finds it necessary to verify herd compliance with program standards. The physical inventories usually would be several years apart and never would be ordered more

continued...

than once per year unless lack of program compliance is suspected. The proposed change should allow timing of physical assembly of a herd for inventory so that it is coordinated with cervid testing for other diseases, such as brucellosis or tuberculosis. However, some type of herd inventory would be performed annually, although it may not include physical assembly of the animals.

The new proposed rule would require animal owners to pay for any confirmatory DNA testing of CWD-positive animals in order to assure their identity and herd of origin. APHIS intends to provide additional guidance on how to request confirmatory DNA testing and how to arrange payment for it if this proposed rule is adopted as final.

APHIS encourages the submission of comments on the new proposed rule by all interested parties. All comments received by June 1, 2009 will be considered. Comments can be mailed to Docket No. 00-108-7, Regulatory Analysis and Development, PPD, APHIS, Station 3A-03.8, 4700 River Road Unit 118, Riverdale, MD 20737-1238. Comments should be identified as pertaining to Docket No. 00-108-7. Additional information can be obtained from APHIS Senior Staff Veterinarian Dr. Dean Goeldner at 301-734-4916. (Prepared by John Fischer)

Some of the Intricacies of CWD

One of the more difficult problems of chronic wasting disease (CWD) research is that the efficiency of transmission apparently exceeds our ability to easily detect the infectious protein in infective material. The use of genetically altered (transgenic) mice that are sensitive to infection by the CWD prion has greatly facilitated detection of the prion. Another technique, which only recently is being explored, is termed the protein misfolding amplification assay (PMA). Similar to the familiar polymerase chain reaction assay (PCR), which amplifies DNA, the PMA basically amplifies small amounts of prion to levels that can be detected in the laboratory. These transgenic mice and PMA techniques potentially can identify small quantities of CWD prion that are undetectable by the conventional assays, and in recent months important new information concerning potential CWD

transmission routes has been revealed by these methods.

For many years, deer biologists and deer disease experts have been concerned about the potential infectivity of urine passed by CWD-positive deer. In a March 2009 article published in *PLoS ONE* (www.plosone.org), Haley et al. described detection of the infectious protein in the urine and saliva from CWD-infected deer using the mouse bioassay. The quantity of prion in the samples apparently was low because it could not be detected by western blot or enzyme-linked immunosorbent assay (ELISA). In addition, of the nine mice inoculated intracerebrally with urine, only two tested positive, whereas eight of nine mice inoculated with saliva were positive. Nevertheless, the demonstrated infectivity of urine justifies the concerns raised about urine-based deer lures that some hunters distribute in the environment to attract deer.

The mouse bioassay and PMA also have been used recently to detect the CWD prion in antler velvet of infected elk. Angers et al., reported in the March 2009 issue of *Emerging Infectious Diseases* that western blot, ELISA, and immunohistochemistry techniques failed to detect the infectious protein in the antler velvet of 14 CWD-affected elk. However, velvet from a subset of these elk was used to infect susceptible transgenic mice. As with the urine and saliva trials described above, an attack rate of less than 100% implied that the prion levels in the velvet were low.

Beyond the laboratory, an obvious concern regarding CWD is the potential negative impact upon free-ranging cervid populations. Models previously suggested that CWD can depress deer numbers over time, but there have been few opportunities to evaluate the impacts of the disease in natural populations. One such study recently compared annual survival of CWD-infected mule deer in Boulder, Colorado, with apparently uninfected mule deer, using a cohort design applied to deer tested by tonsil biopsy. The results indicated that positive deer have a much shorter life expectancy than negative deer. The researchers also discovered that positive deer were nearly four times more likely to be killed by mountain lions, but such selective predation apparently was insufficient to limit

infection rates among susceptible animals, as approximately 25% of the sampled deer were infected. Based on these findings, the authors concluded that CWD was a plausible explanation for the local deer population decline observed there since the 1980s.

There have been no new CWD foci detected in wild cervids in North America since our last update (BRIEFS, Vol. 22, No. 4). However, infected free-ranging deer, elk, and an occasional moose continue to be found in or near previously recognized CWD foci, many of which appear to be expanding. The single exception is New York, where CWD was found in two wild deer in 2005 following detection in captive deer there. No additional infected animals have been found in New York despite testing.

Among captive deer and elk, CWD continues to be found in Canada and the United States. In Canada, most infected captive cervid herds and all of the recently detected cases have been in Saskatchewan. It was in Saskatchewan that CWD was first identified in commercial captive cervids in 1996, and an extensive epidemiological investigation revealed that elk in 38 captive herds apparently became infected through direct or secondary contact with a single source herd. This led to the destruction of nearly 8,000 elk at a cost of \$19 million Canadian dollars in indemnification and cleanup. Current provincial policy is depopulation of all captive cervid facilities in which CWD is confirmed. In spite of this policy, and the millions of dollars spent to eliminate CWD from captive cervids in Saskatchewan, the problem persists. The Canadian Food Inspection Agency confirmed CWD in four captive cervid facilities there in 2008 and two more so far in 2009. Infected captive elk and captive white-tailed deer herds have been found, and CWD prevalence reached as high as 32% in one captive elk breeding operation.

In the United States, additional infected captive deer and elk facilities have been found in Colorado, Minnesota, and Wisconsin, where CWD previously had been confirmed in captive deer or elk. Michigan was added to the list in August 2008, when an infected white-tailed deer was found in a captive herd in Kent County. In April 2009, the owners of this herd pleaded

guilty to violating Michigan's Animal Industry Act by removing and transporting a deer from their herd while it was under quarantine.

In response to the CWD finding, the Michigan Department of Natural Resources increased sampling efforts in Kent County, and in 2008 1,942 wild deer there were tested for CWD. Another 7,399 wild deer were tested from other counties in the state. All wild deer were negative. (Prepared by Kevin Keel)

3rd International CWD Symposium

The Utah Division of Wildlife Resources is hosting the 3rd International Symposium on Chronic Wasting Disease (CWD) in Park City, Utah, July 22-24, 2009. The theme of this symposium is "CWD – Advancing the Science and Developing the Tools." Early registration at a reduced fee continues until May 22, 2009.

Researchers, wildlife managers, and others will be presenting more than 60 state-of-the-art papers in sessions on regulating CWD; ecology and epidemiology of CWD; human dimensions of CWD; research in prion biology of CWD; environmental contamination issues and research; surveillance, management, and control of CWD in free-ranging cervids; CWD diagnostics and detection; and cervid industry CWD issues. In addition, more than 30 posters will be presented on a wide range of CWD-related issues.

Complete information, including on-line registration for the symposium and hotel reservations at a group rate, can be found at the conference website: http://www.regonline.com/CWD_symposium, or by contacting Ms. Leslie McFarlane of the Utah Division of Wildlife Resources (telephone 801-538-4891 or email lesliemcfarlane@utah.gov). (Prepared by John Fischer)

White Nose Syndrome Update

White nose syndrome (WNS) was first recognized in bats in Howes Cave in Upstate New York in 2006 (see *SCWDS BRIEFS* Vol. 24, No. 1). One year after the index case, five caves within 10 miles of Howes Cave were confirmed to contain infected bats. As of March 2009, the syndrome has been identified in

multiple caves in eight states: Connecticut, Massachusetts, New Hampshire, New Jersey, Pennsylvania, Vermont, Virginia and West Virginia, and biologists in adjacent states are conducting surveillance to detect its potential presence.

White nose syndrome is named for the characteristic fluffy white mold that grows around the muzzles of affected bats. This mold also can be found growing on the wings, ears, and tail. Since its discovery, WNS has killed an estimated 500,000 to one million bats of five species, including the little brown bat (*Myotis lucifugus*), the eastern pipistrelle (*Pipistrellus subflavus*), the northern long-eared bat (*Myotis septentrionalis*), the small-footed bat (*Myotis leibii*) and the endangered Indiana bat (*Myotis sodalis*). The disease has caused up to 97% cumulative mortality among hibernating bats at affected sites.

Recently, microbiologists with the U.S. Geological Survey's National Wildlife Health Center (NWHC) have identified the fungus as a previously undescribed species of *Geomyces*. No viral or bacterial pathogens have been isolated consistently from bats with WNS, and other fungal isolates seem to consist of sporadic contaminants. Fungi in the genus *Geomyces* are best known as saprophytes (feeding on dead organic material) in colder climates.

It still has not been definitively proven that the *Geomyces* sp. is the cause of WNS, but scientists at the NWHC currently are trying to verify this by attempting to infect normal bats with isolates from diseased bats. If the experimentally inoculated bats develop the disease, it will prove the fungus alone is sufficient to cause WNS. There has been great concern that other factors, such as changing environmental conditions, toxins, or viral infections, are involved that might predispose the bats to infection with this fungus.

The *Geomyces* sp. of interest grows very well at 5° C to 10° C, which is within the temperature range of bat hibernacula (2° C to 14° C). All species of *Geomyces* are keratinophilic, meaning they grow on hair and skin. The *Geomyces* sp. associated with WNS is no exception, and it appears to be well adapted for growth in the outer layers of the skin and even

gets into hair follicles, sweat glands, and sebaceous glands. The bats don't have any consistent internal lesions. The mechanisms by which the fungus causes disease have not yet been completely described. However, once infected, bats often are aroused from hibernation and quickly deplete their fat stores needed to survive the winter. Affected bats often move to colder parts of the cave and many have been reported flying outside during daylight hours in the wintertime, which is extremely unusual behavior for bats during hibernation.

Regardless of the ultimate cause of the disease, there is no doubt that WNS is causing the most catastrophic mortality ever seen in bats in the region. It has caused precipitous population declines among several bat species in the Northeast and threatens endangered species, particularly the Indiana bat. As it moves south, it also threatens gray bats (*Myotis grisescens*), another endangered species. Even though many of the bat species that are affected are not endangered, the tremendous regional declines in their populations could have unforeseen consequences on the ecosystem. Some bats consume a large number of insects (one bat can eat as many as 3,000 mosquitoes per night), and no one can predict the effects of reduced insect mortality. Agriculture could be threatened due to increases in plant pests, and disease dynamics could be shifted due to increases in insect vectors. (Prepared by Kevin Keel and Victoria Watson)

Wildlife Poisoning in Kansas

During the first week of January 2009, the Kansas Department of Wildlife and Parks (KDWP) received numerous reports of large groups of dead wild turkeys at multiple locations in Logan County. From January 4-8, KDWP recovered 29 dead turkeys from a city park in the small town of Russell Springs, along with two smaller groups of turkeys within five miles of town. In addition, a dead raccoon was recovered with one group of turkeys. All together, 45 wild turkeys and one raccoon were collected over a four-day period. Three weeks later, an American badger was found dead seven miles southeast of Russell Springs on private property that serves as a recovery site for black-footed ferrets that were reintroduced as an experimental population in 2007. Based on

the widespread use of rodenticides to control black-tailed prairie dog populations in Logan County, poisoning was suspected in this case.

Six wild turkeys, the raccoon, and the badger were delivered to SCWDS for postmortem examination. All of the animals were in good nutritional condition. The turkeys had pooled and clotted blood accumulated within body cavities. In addition, the lungs of most birds were dark red and wet. The crop of each bird was distended with grass and wheat kernels, some of which had green pigment on the surface. The intestines of the raccoon and badger were distended with bloody contents. Several areas of hemorrhage were present under the skin and on the surface of many organs. In addition, the badger's lungs and chest were filled with blood. The presence of widespread hemorrhages in multiple organ systems suggested loss of blood vessel integrity or a disruption of blood clotting mechanisms. Based on these findings and the history of the use of chlorophacinone (Rozol®), an anticoagulant rodenticide, and zinc phosphide in the area, toxicologic testing was performed.

Chlorophacinone was detected in the liver of the American badger and raccoon, and two other anticoagulant rodenticides, brodifacoum and bromadiolone, also were present in the raccoon. Phosphene gas was detected in the crop contents of all wild turkeys, which is consistent with zinc phosphide poisoning. Additionally, the crop contents of one turkey contained chlorophacinone, indicating a non-lethal exposure. It is of interest to note that in 2002 SCWDS had diagnosed Rozol® poisoning in two wild turkeys found dead in a baited prairie dog town in nearby Cheyenne County.

Zinc phosphide is a rapidly acting toxin commonly sold as a grain-based formulation for the control of many rodent species. Once the ingested bait contacts stomach acid, a toxic gas is produced that is inhaled and absorbed, resulting in blood vessel damage and tissue necrosis. Death usually occurs within six hours of ingestion, often due to fluid filling the lungs.

Anticoagulant rodenticides interfere with production of clotting mechanisms, and death generally occurs due to severe blood loss. Unlike zinc phosphide mortality, death due to

anticoagulant rodenticides may require repeated ingestion of the toxin, although the various compounds vary widely in their toxicity.

These mortality events document nontarget intoxication of multiple species with widely available rodenticide compounds. Based on the predatory nature of badgers, the chlorophacinone exposure may have been from the consumption of Rozol®-laden carcasses (secondary poisoning). This also is possible with the raccoon; however, primary consumption of multiple baits cannot be ruled out. The exact source of anticoagulant exposure could not be determined in any of these cases; however, large amounts of Rozol® are used for prairie dog control in Logan County. Zinc phosphide is not as commonly utilized in the area, and the exact source of exposure of the turkeys could not be determined.

The perception of prairie dogs both as a nuisance species and a keystone species presents a significant challenge to wildlife, land, and livestock managers. In Logan County, elimination of prairie dogs on public and private lands is mandatory by law. Multiple agencies and private groups are actively involved in controlling prairie dog populations outside of black-footed ferret recovery zones, which currently are confined to private lands. The potential risk to black-footed ferrets and other carnivores should be considered when selecting prairie dog control methods. Risks to carnivores may include secondary poisoning or a decrease in prey base. The extent of nontarget and secondary poisoning of wildlife in Logan County currently is unknown and warrants further investigation. (Prepared by Mark Ruder and Kevin Keel)

Salmonellosis in Your Backyard

Beginning in late January 2009, the SCWDS Diagnostic Laboratory began receiving numerous submissions of songbirds that were found sick or dead at birdfeeders in residential areas around northeast Georgia. Postmortem examinations of these birds revealed pale yellow nodules in the esophagus, and the bacterium *Salmonella typhimurium* was isolated from several organs. Over the remainder of the winter and spring, small salmonellosis die-offs continued to be reported in songbirds throughout

the Southeast. To date, SCWDS has confirmed salmonellosis in 92 birds from seven states (Florida, Georgia, North Carolina, South Carolina, Tennessee, Virginia, and West Virginia), and countless other die-offs are suspected of being caused by *S. typhimurium*. The primary species affected were pine siskins, American goldfinches, and northern cardinals. Disease also was found infrequently in red-winged black birds, house finches, and purple finches.

Salmonellosis, caused by bacteria in the genus *Salmonella*, is a common disease in both wild and domestic animals. Several species of *Salmonella* exist, each with the unique ability to infect and cause disease in different types of animals. Certain species commonly cause mortality in songbirds and are responsible for scattered outbreaks at bird feeders each year. Affected birds typically have fluffed and ruffled feathers, closed eyes, and are extremely lethargic, often allowing humans and predators to approach closely. The illness usually progresses rapidly to death, and birds may appear to die suddenly while feeding or perching in nearby trees.

Salmonella inhabits the intestinal tracts of many birds and mammals, where it may be tolerated without causing significant illness. The bacteria are shed into the environment in feces. Songbirds become infected when they ingest feed or water that has been contaminated by bacteria-laden feces, or through direct contact with infected birds. In the winter months, when natural forage is less abundant and bird feeders provide an easy meal, songbirds may congregate at feeding stations. Higher densities of birds at these feeders increase the likelihood of fecal contamination and bird-to-bird contact, thus increasing the risk of *Salmonella* transmission.

Historically, salmonellosis outbreaks at bird feeders occur sporadically each winter in the southeast and typically subside after a few weeks. However, both the extent and duration of disease this year appear to be worse, with greater numbers of sick birds reported and submitted to SCWDS, and with outbreaks continuing into the warmer spring months. The reasons behind the increase in severity of disease this year are not clear, but may be

related to an incursion of dense flocks of pine siskins, one of the species most severely affected. A host of other factors, including the age, level of stress, and species of bird infected, as well as the species of *Salmonella* involved, are not well understood but may influence disease outbreak patterns. Several of these factors currently are being investigated.

In order to prevent or control salmonellosis in backyard birds, feeders and water baths should be cleaned weekly to reduce the risk of *Salmonella* contamination. Feeders should be washed with a 10:1 water to bleach solution, rinsed well, and dried. During an outbreak, it may be necessary to remove the feeders for several weeks until the event has passed. Piles of seeds or hulls that collect on feeders or on the ground below should be removed or raked out. Birds can be discouraged from congregating in high numbers by providing multiple feeders or feeding only intermittently. Humans are susceptible to *Salmonella*, but basic sanitary procedures minimize the chances of infection. Individuals should wash their hands after touching carcasses or birdfeeders that may be associated with a *Salmonella* outbreak, and care should be taken to avoid fecal contamination of surfaces that come in contact with food. (Prepared by Andrew Cartoceti)

Trichomonosis in Songbirds

During winter and spring 2009, the SCWDS Diagnostic Laboratory received numerous dead passerines, including American goldfinches, house finches, northern cardinals, pine siskins, and purple finches. The accessions generally had similar case histories of numerous sick and dead birds found at or near bird feeding and watering/bathing stations. Although many of the birds had salmonellosis, at least 12 were diagnosed with either trichomonosis or a combination of trichomonosis and salmonellosis. The two diagnoses were obtained by one or more of the following methods: wet mounts, histology, culture, and/or polymerase chain reaction assay (PCR). Results of PCR testing on several samples are pending, and additional trichomonosis cases may be confirmed.

Trichomonosis, caused by the microscopic protozoal parasite *Trichomonas gallinae*, often is referred to as crop canker by bird watchers,

because it appears as large, ulcerative, yellowish-white lesions in the mouth and esophagus of affected birds. The disease predominantly is observed in members of the dove family (*Columbidae*) and raptors. Columbids feed nestlings with "crop milk" that includes sloughed lining of the parent's crop, and adults perform a mating behavior termed "billing." Given that *T. gallinae* infects the oral cavity, esophagus, and crop, these columbid behaviors greatly facilitate the transmission of the parasite from parent to offspring, or from one mate to another. Although *T. gallinae* does not form an environmentally resistant stage, it can form pseudocysts that survive for several hours in feed and water. Consequently, trichomonosis outbreaks often are associated with contaminated bird baths and feeders. Raptors become infected by ingesting infected prey, and the disease has been reported in North America from numerous raptor species, including Cooper's hawks, red-tailed hawks, peregrine falcons, and great-horned owls. Generally, trichomonosis occurs as focal outbreaks affecting low numbers of birds; however, large outbreaks were reported in 1952 in mourning doves from multiple southeastern states, in 2003 in Cooper's hawks in Arizona, and in 2006 in band-tailed pigeons in California.

The diagnosis of trichomonosis in passerine birds at SCWDS this year is not unprecedented and appears to be consistent with a developing pattern. Recently, variably sized outbreaks of trichomonosis have been reported in several passerine species from California, Kentucky, the United Kingdom, and Canada. The Kentucky cases occurred in the summer of 2006 and comprised house finches, American goldfinches, and a gray catbird. Affected birds were weak and emaciated and often were found near artificial water and feeding stations. In all, 18 birds were sent to SCWDS but the total number of affected birds was unknown. Trichomonosis was diagnosed by post mortem examination and culture in 10 birds, West Nile virus was isolated from one, and none of the birds were infected with *Salmonella*.

The California cases involved house finches, northern mockingbirds, and several corvid species (American crow, common raven, scrub jay, and Stellar's jay) that were submitted to a wildlife rehabilitation center in Northern

California from 2001 to 2005 (Anderson et al., 2009. *Veterinary Parasitology* Vol. 161, pp. 178-186). Prevalence of *Trichomonas* spp. infection in the house finches, mockingbirds, and corvids was 1.71%, 0.92%, and 0-6.25%, respectively. Case fatality rates in the house finches, mockingbirds, and corvids were 95.5%, 37.5%, and 0-100%, respectively. The high rate in the house finches suggests that most finches infected with *Trichomonas* spp. died, and incidental findings of *Trichomonas* in house finches were rare. As with the current outbreak diagnosed at SCWDS, the passerine birds from the outbreaks in California, Kentucky, Canada, and the United Kingdom were associated with artificial feeders and waters. The most likely source of the corvid infections was ingestion of other infected birds.

It is not surprising that during this current outbreak several birds were diagnosed with a combination of trichomonosis and salmonellosis, because both diseases often are associated with contaminated feeders. Additionally, *T. gallinae* causes ulcerative lesions in the upper gastrointestinal tract, which may facilitate bacterial invasion, leading to septicemia. Routine cleaning of feeders and birdbaths with a 10% bleach solution and removing moldy and wet feed is recommended to reduce and prevent outbreaks.

Recent SCWDS research and the findings of Anderson et al. in California showed significant genetic variability in *T. gallinae* isolates collected from wild birds. SCWDS studies strongly suggested at least three species comprise the *T. gallinae* complex (see SCWDS BRIEFS Vol. 23 no. 4), with rock pigeons, white-winged doves, and common ground-doves each having their own species of *Trichomonas*. Several isolates from the current outbreak in passerines are being analyzed to determine their genotype and better elucidate the epidemiology of this outbreak. (Prepared by Rick Gerhold)

Dr. Al Franzmann

Dr. Albert Wilhelm Franzmann died February 13, 2009, at age 78. Al was recognized world-wide as a pioneer in bridging the veterinary and wildlife professions. He was a native of Ohio and received his DVM degree from Ohio State University. He then served for two years in the

United States Air Force Veterinary Corps before engaging in private veterinary practice from 1956 until 1968. In 1968 Al entered graduate school at the University of Idaho and received his PhD degree in Forestry Science in 1971. His graduate research was on the physiology of Rocky Mountain bighorn sheep.

In 1972, Al and his family moved to Soldotna, Alaska, where he became a research biologist with the Alaska Department of Fish and Game and was Director of the Moose Research Center. His research produced over 250 publications. He was appointed Affiliate Associate Professor of Wildlife Biology at the University of Alaska, Fairbanks, and the Institute of Arctic Biology. Al retired in 1987 and pursued international wildlife veterinary consulting as a Director of the International Wildlife Service, Inc. He worked on projects in Africa, Asia, Europe, South America, and North America. He compiled and edited the book *Ecology and Management of the North American Moose* published in 1998. Al was appointed by Governor Wally Hickel to the Alaska Board of Game (1992-1995), and he was elected to the board of directors of the Alaska Outdoor Council, the Alaska Fish and Wildlife Conservation Fund, and the Alaska Challenger Center for Space Science Technology.

Al was active in professional wildlife organizations such as The Wildlife Society, Wildlife Disease Association, American Association of Wildlife Veterinarians (Founder President), World Association of Wildlife Veterinarians, and the American Association of Zoo Veterinarians. He also was a life member of the Isaac Walton League, the Nature Conservancy, the National Rifle Association, the National Wildlife Federation, and Alaska Outdoor Council, and he was a regular member of many other conservation, wildlife, veterinary and civic organizations.

Al and his work often were recognized: he received the Distinguished Moose Biologists Award, and he was the first Honorary Diplomat of the American College of Zoological Medicine. He also received an Emeritus Award from the Wildlife Disease Association "in recognition of meritorious contributions to the study and understanding of disease of wildlife." In 1997, Al received the Distinguished Alumnus Award from

The Ohio State University College of Veterinary Medicine. In 2001, Al became an Honor Roll member of the American Veterinary Medical Association and was given the Lifetime Conservation award by the Kenai Chapter of Safari Club International. In 2004, he was inducted into the University of Idaho Hall of Fame "for his leadership and contributions in the field of wildlife veterinary research."

Al's avocations included hunting, fishing, gardening, golf, travel, and photography. He had over 100 photographs published and received several photographic awards. His family, friends, and co-workers knew him as a dedicated professional who loved his work. He would often comment that "he could not believe that he was getting paid for doing such neat things." (Prepared by Gary Doster)

Staff & Student Recognition

Dr. Michael Yabsley is the 2009 recipient of the Junior Achievement in Research Award from The University of Georgia Chapter of Gamma Sigma Delta (GSD), the honor society for agriculture and related sciences. GSD awards are given to faculty each year for teaching, research, and extension/outreach. Michael was not honored for a single event, but was recognized for his continuing superior work ethic since becoming a faculty member at UGA in 2004.

Key to Michael's success is the group of outstanding graduate students he mentors, and several of them also recently were acknowledged for their first rate work at SCWDS. At the annual Graduate Student Symposium held at the University of Georgia's Warnell School of Forestry and Natural Resources in February 2009, three of Michael's students won awards for their presentations. During the three-day event, which culminated in an awards banquet, 36 UGA students gave presentations in six categories. Liz Gleim won first place in her category, Emily Blizzard won second place in hers, and Ben Wilcox won third place in his.

Another of Michael's students, Dawn Roellig, also was distinguished recently by being selected to attend the Third European Wildlife Disease Association Student Workshop held in

Veyrier-du-Lac, France, in March 2009. The workshop is held every other year. This year, 40 students from 14 countries were chosen for this four-day event, and it is a signal honor to be included. Dawn was one of only a few individuals selected from the United States. The central theme of the workshop this year was Infectious Diseases at the Wildlife/Domestic Animal/Human Interface.

Soon after returning from the workshop in France, Dawn attended the Annual Meeting of the Southeastern Society of Parasitologists in Murray, Kentucky, and won the Byrd-Dunn Student Presentation Award for her paper entitled "Oral Transmission of *Trypanosoma cruzi* with Opposing Evidence for the Role of Carnivory in Horizontal Transmission." In addition to a plaque and a cash award, Dawn will receive a travel grant to the 84th Annual Meeting of the American Society of Parasitologists to be held in Knoxville, Tennessee, August 14-17, 2009.

Jess Gonynor was honored recently by being elected as an officer of the Gopher Tortoise Council (GTC) and was appointed as the new website manager for the organization. The GTC was formed in 1978 by a group of biologists and others concerned about the range-wide decline of the gopher tortoise (*Gopherus polyphemus*). Jess also is president of the Warnell Graduate Student Association for 2009-2010. For her PhD research, Jess is studying the ecology of *Mycoplasma* in gopher tortoises and box turtles at the 29,000-acre Joseph W. Jones Ecological Research Center located at Ichauway Plantation in Baker County, Georgia.

Congratulations to Michael, Emily, Ben, Liz, Dawn, and Jess for their fine work. We are proud they are part of the "SCWDS family." (Prepared by Gary Doster)

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Information on SCWDS and recent back issues of the *SCWDS BRIEFS* can be accessed on the internet at www.scwds.org. The BRIEFS are posted on the web site at least 10 days before copies are available via snail mail. If you prefer to read the BRIEFS online, just send an email to Gary Doster (gdoster@uga.edu) or Michael Yabsley (myabsley@uga.edu) and you will be informed each quarter when the latest issue is available.

SCWDS BRIEFS

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