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April 2002

## SCWDS BRIEFS: Volume 18, Number 1 (April 2002) -- Special CWD Issue

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# SCWDS BRIEFS

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A Quarterly Newsletter from the  
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## SPECIAL CWD ISSUE

The recent discovery of chronic wasting disease (CWD) in wild deer in new locations in Colorado, Nebraska, South Dakota, and Wisconsin has led to unprecedented attention and concern for a wildlife health issue. A short list of CWD-related events in the last few months includes complete bans on live cervid importation in several states, management plans to kill as many wild cervids as possible in areas in Wisconsin and Colorado where CWD recently was found, depopulation of captive elk herds in the CWD endemic area of northeastern Colorado, and a congressional hearing on CWD. A joint CWD Task Force co-chaired by the Administrator of USDA's Animal Plant and Health Inspection Service (APHIS) and the Director of the U.S. Fish and Wildlife Service was formed in mid-May. Within this task force, six working groups have been directed to develop a 5-year national management plan for CWD.

News about CWD now comes to wildlife management and animal health authorities on a daily basis and it has become challenging to stay informed about this rapidly evolving situation. The purpose of this special issue of the SCWDS BRIEFS is to provide historical and up-to-date facts about CWD and other transmissible spongiform encephalopathies. Several persons assisted with this special issue, and we are grateful to them: Lynn Creekmore, Linda Detwiler, Lisa Ferguson, and Tom Gomez with USDA-APHIS; Bruce Morrison of the Nebraska Game and Parks Commission; Ron Fowler of

the South Dakota Department of Game, Fish, and Parks; and Beth Williams of the University of Wyoming. Additional sources of information, including scientific articles, book chapters, and agency websites, are listed for further reading. Updates on CWD and other wildlife health topics will be provided in future issues of the SCWDS BRIEFS.

## Transmissible Spongiform Encephalopathies

Transmissible spongiform encephalopathies (TSEs) are invariably fatal diseases of the central nervous system that occur in domestic and wild animals and humans. TSEs in domestic animals include scrapie of sheep and goats, transmissible mink encephalopathy (TME), and bovine spongiform encephalopathy (BSE, also known as mad cow disease). Chronic wasting disease (CWD) is the only TSE currently found in free-ranging wildlife (white-tailed deer, mule deer, and elk), and it also has been found in captive animals of like species. Human TSEs include Creutzfeldt-Jakob disease (CJD), new variant CJD, kuru, Gerstmann-Straussler-Scheinker syndrome, and fatal familial insomnia.

Although each of these TSEs is considered different, they all share important characteristics and thus are grouped together. Scientific debate continues about the cause of TSEs, but all are thought to be caused by the same type of infectious agent. The most widely accepted theory describes TSE agents as abnormal, proteinaceous, infectious particles (prions) that are closely related to a cell protein normally

produced in the central nervous system, lymphoid tissue, and many other body tissues. However, abnormal TSE prions cannot be broken down by the body's enzyme protease. Researchers call the normal form of the cell protein PrP<sup>c</sup>, whereas the enzyme-resistant form is shaped differently and is generically known as PrP<sup>res</sup>. Following introduction into the body, PrP<sup>res</sup> promotes conversion of normal PrP<sup>c</sup> to more of the undegradable PrP<sup>res</sup> in lymphoid tissues and brain cells. The end result is a pathologic accumulation of PrP<sup>res</sup> in brain cells that results in nerve cell death and loss of neurologic function. Two unique aspects of PrP<sup>res</sup> include lack of genetic material (DNA or RNA) and extreme resistance to breakdown by environmental conditions, disinfectants, and treatments that normally inactivate infectious disease agents such as viruses, bacteria, fungi, etc. All TSEs are characterized by long incubation periods, ranging up to several years, and currently there are no treatments or vaccines.

There are several ways that TSEs can develop. In human beings, sporadic TSEs may develop when some of the person's normal prions (PrP<sup>c</sup>) spontaneously change into PrP<sup>res</sup> thereby altering the person's normal PrP<sup>c</sup> in a chain reaction. Inherited cases occur in people who receive a defective gene that codes for the PrP<sup>c</sup> protein, causing it to be shaped abnormally. Finally, acquired cases may arise when PrP<sup>res</sup> from an affected individual is transferred to another through contact with infected central nervous system tissue or contaminated medical instruments. In animals, TSEs are acquired; the spontaneous or inherited forms have not been found, although they may occur. Studies in animals reveal that after PrP<sup>res</sup> is ingested, the disease agent spreads from the gastrointestinal tract to the spinal cord and brain. There is no apparent immune response to the disease agent and inflammation is not seen in affected tissues. All TSEs of domestic and wild animals and humans have characteristic damage to brain cells (neurons) that results in a spongiform degenerative change in the gray matter when the brain is examined microscopically, thus the term

“spongiform encephalopathy.” PrP<sup>res</sup> can be detected in affected brain tissue and also in normal-appearing lymphoid cells of infected hosts with certain TSEs such as scrapie and CWD. The latter feature may prove useful for diagnosis via detection of the TSE agent. The distribution of PrP<sup>res</sup> is very limited with other TSEs, such as BSE. (Prepared by Nicole Gottdenker and Vic Nettles).

Additional sources of information:

Williams, Elizabeth S., Michael W. Miller, and E. Tom Thorne. Chronic Wasting Disease: Implications and Challenges for Wildlife Managers. *Transactions of the 67th North American Wildlife and Natural Resources Conference*. In Press.

Centers for Disease Control and Prevention ([www.cdc.gov](http://www.cdc.gov))

National Institutes of Neurological Disorders and Stroke ([www.ninds.nih.gov](http://www.ninds.nih.gov))

USDA's Animal and Plant Health Inspection Service ([www.aphis.usda.gov](http://www.aphis.usda.gov))

### **TSEs of Domestic Animals and Humans**

The earliest historical records of transmissible spongiform encephalopathies (TSEs) began with sheep scrapie more than 250 years ago in England. Microscopic brain lesions of scrapie were described in the 19th century, and its transmissible nature and long incubation period were demonstrated experimentally in the 1930s. The scrapie agent is thought to spread from animal to animal, primarily from an infected ewe to her offspring, and to other lambs through contact with the placenta or placental fluids. Clinical signs usually appear between 2 and 5 years after infection but may not occur until much later. Affected sheep and goats show progressive neurologic disease, including behavioral changes, head and neck tremors, ataxia, and self-trauma due to incessant itching. Death typically occurs within 1 to 6 months or longer after the onset of clinical signs. In the

United States, scrapie was first diagnosed in 1947 in a flock that had received sheep of British origin. Scrapie has been identified in more than 1,000 flocks in the United States, and currently it is the target of an eradication program administered by the USDA's Animal and Plant Health Inspection Service. There is no evidence to indicate that scrapie poses a risk to human health.

A few outbreaks of transmissible mink encephalopathy (TME) have been reported in ranched mink in Europe and North America. This rare TSE has never been found in wild mink. Outbreaks are believed to be caused by exposure to the infectious agent, such as by eating contaminated feed, and are characterized by 60% to 100% morbidity within a ranched mink population and 100% mortality of affected mink. Affected animals initially show behavioral changes, aggression, and ataxia; later they become moribund and die. Although TME is not a significant disease of domestic animals or humans, it has great utility as an experimental TSE model.

Several TSEs occur in humans. Kuru is a TSE of the Fore natives of the highlands of New Guinea who performed ritual cannibalism of relatives after natural death. Kuru essentially has disappeared since mortuary cannibalism practices were discontinued. Gerstmann-Straussler-Scheinker Syndrome and fatal familial insomnia are extremely rare hereditary TSEs that have been demonstrated to be transmissible under laboratory conditions. They are found in just a few families around the world. Although quite rare, the most common TSE in humans is classic Creutzfeldt-Jakob Disease (CJD), with a worldwide incidence of approximately one case per one million people per year. Disease usually develops later in life; the typical age of onset is around 60 years. As with all TSEs, CJD is characterized by progressive neurological disease that is invariably fatal.

In 1996, researchers recognized a "new variant" of Creutzfeldt-Jakob Disease (vCJD) in British

people. The most likely explanation of this new form of the disease was exposure to the BSE agent. Additional research since that time has found epidemiological and laboratory evidence to further support the causal association between BSE and vCJD, thus linking vCJD with consumption of products contaminated with central nervous system tissue of cattle with BSE. BSE is the only TSE of domestic or wild animals ever linked with human disease. Human patients with vCJD generally were much younger at the age of death (median age of 28) than victims of sporadic CJD (median age of 68), and the microscopic features of vCJD differed from classic CJD. Since it was first recognized, vCJD has been documented as the definite or probable cause of death or disease of 130 people in the United Kingdom (121), France (6), Ireland (1), Italy (1), and the United States (1). The single probable case in the United States was reported in April 2002 in a young woman who had come to the United States after spending much of her life in the United Kingdom. There never has been a case of vCJD that did not have a history of exposure within a country where BSE was occurring. The epidemiological and laboratory evidence linking BSE and vCJD is very strong and a cause of concern among public health officials, citizens, and meat industries of Great Britain and other countries around the world.

Bovine spongiform encephalopathy (BSE) was recognized in the mid-1980s in cattle in Great Britain. BSE is covered at length in the following article. (Prepared by Nicole Gottdenker and John Fischer)

Additional sources of information:

Williams, Elizabeth S., Michael W. Miller, and E. Tom Thorne. Chronic Wasting Disease: Implications and Challenges for Wildlife Managers. *Transactions of the 67th North American Wildlife and Natural Resources Conference*. In Press.

Centers for Disease Control and Prevention ([www.cdc.gov](http://www.cdc.gov))

National Institutes of Neurological Disorders and Stroke ([www.ninds.nih.gov](http://www.ninds.nih.gov))

UK CJD Surveillance Unit ([www.cjd.ed.ac.uk](http://www.cjd.ed.ac.uk))

USDA's Animal and Plant Health Inspection Service ([www.aphis.usda.gov](http://www.aphis.usda.gov))

### **Bovine Spongiform Encephalopathy**

Since 1986, more than 180,000 cases of bovine spongiform encephalopathy (BSE), also known as mad cow disease, have been reported in numerous European countries and Japan, with more than 95% of the cases occurring in Great Britain. Neither BSE nor any other transmissible spongiform encephalopathies (TSEs) have been detected in cattle in the United States, despite more than 12 years of active surveillance. The British BSE epidemic apparently arose through the feeding of ruminant-derived protein contaminated with a TSE agent suspected to be a strain of sheep scrapie or a previously unrecognized TSE in cattle. The epidemic was sustained and boosted by recycling of BSE infected cattle to other cattle via feeds containing ruminant meat and bone meal. Epidemiological studies suggest widespread exposure of British cattle via contaminated commercial feed as early as 1980, and it is believed that changes in rendering procedures in the 1970s and 1980s resulted in failure to inactivate the TSE agent. Transmission of BSE is primarily, if not solely, through this contaminated feed route as additional studies indicate that there has been little or no horizontal spread from animal to animal, as occurs with scrapie and chronic wasting disease. In Great Britain, the BSE epidemic peaked in early 1993 with an incidence of more than 1,000 new cases per week. As a result of feed bans initiated in 1988, the incidence has dropped markedly and currently runs about 25 new cases per week.

Transmissible spongiform encephalopathies have affected other species, including domestic and exotic cats, in which it is known as Feline

Spongiform Encephalopathy (FSE), as well as exotic ruminants in zoological collections. BSE is believed to have affected 10 bovid and felid species from zoos in the British Isles, viz., eland, greater kudu, nyala, oryx, gemsbock, bison, cougar, ocelot, tiger, and cheetah. The agent isolated from several of these cases of FSE and the TSE of exotic ruminants is indistinguishable from BSE in cattle using strain typing in mice, suggesting the occurrence of TSEs in these species resulted from BSE-contaminated feed.

Without doubt, the great publicity and concern surrounding BSE is due to its epidemiological and laboratory-supported link with new variant CJD (vCJD) in humans. This new human TSE was first recognized in Great Britain in 1996 and appears to be associated with the consumption of BSE-infected cattle products, specifically beef products contaminated with central nervous system tissue containing the BSE agent. To date, there have been 130 definite or probable cases of vCJD in humans. Although there is strong evidence that the BSE agent is responsible for vCJD cases, the specific foods possibly associated with transmission to humans are unknown. In cattle experimentally infected by the oral route, the BSE agent has been identified in brain, spinal cord, retina, nerve roots near the vertebrae, and bone marrow. In naturally infected cattle, the agent has been found only in brain, spinal cord, and retina.

Control measures for BSE in the United Kingdom and other affected countries include bans on animal-derived proteins in livestock feeds; prohibitions on the use of specified risk materials in any food, feed, or biological product; culling of cattle at highest risk of infection; and stringent regulations or bans on the movement of animals and animal products. Countries without BSE have employed import restrictions and similar measures to prevent its introduction and spread.

Since 1989, the United States has banned importation of ruminants and most ruminant

products from countries identified as having BSE or at risk for having BSE. Cattle imported from high-risk countries prior to the ban have been euthanized and tested for BSE or have been quarantined and monitored. There is a U.S. ban on the importation of processed animal protein products, regardless of species, from BSE-restricted countries. Since 1997, the U.S. Food and Drug Administration (FDA) has banned the use of most mammalian protein in ruminant feeds and has issued guidelines concerning the use of bovine materials from BSE-restricted countries in human medical products. Since 1990, the USDA's Animal and Plant Health Inspection Service (APHIS) has conducted an active surveillance program for BSE. Samples are obtained from the target high-risk population -- adult cattle that exhibit neurological signs, non-ambulatory ("downer") cows, and animals that die of unknown causes. More than 26,000 animals in the United States have been tested for BSE to date with uniformly negative results.

In 1998, the USDA entered into a cooperative agreement with Harvard University's School of Public Health to conduct an analysis and evaluation of potential pathways for BSE to enter the U.S. national cattle herd or food supply. Harvard assessed the pathways by which BSE could potentially occur in the United States, pathways by which humans could potentially be exposed to the BSE agent from beef products, and identified additional measures that could be taken to protect human and animal health in the United States. The results of this analysis, made public in November 2001, found that the United States is highly resistant to the introduction and spread of the BSE agent in the U.S. cattle herd due to existing federal regulatory programs. The measure identified as most effective in reducing the spread of BSE is the ban instituted by FDA to prevent recycling of potentially infectious cattle tissues. (Prepared by John Fischer)

Additional sources of information:

Centers for Disease Control and Prevention  
([www.cdc.gov](http://www.cdc.gov))

United Kingdom's Department for Environment,  
Food, and Rural Affairs ([www.defra.gov.uk](http://www.defra.gov.uk))

USDA's Animal and Plant Health Inspection  
Service ([www.aphis.usda.gov](http://www.aphis.usda.gov))

### **Chronic Wasting Disease 101**

Concern about chronic wasting disease (CWD) is increasing for wildlife managers, animal health authorities, captive cervid owners, and the public across North America. Many biological features of CWD pose significant challenges for those attempting to control or eradicate the disease. Perhaps even greater challenges are those associated with balancing complex and often competing and conflicting interests of the general public, sportsmen, the game farming industry, traditional livestock industries, and many state and federal animal health and public health agencies.

Chronic wasting disease was first recognized in the 1960s as a syndrome of captive mule deer held in several wildlife research facilities in Ft. Collins, Colorado. Originally believed to be a nutritional malady, CWD was not recognized as a transmissible spongiform encephalopathy (TSE) until 1978. CWD soon was identified in captive deer and elk from other wildlife research facilities near Kremmling and Meeker Colorado, and at Wheatland, Wyoming, as well as in at least two zoological collections. By the 1980s, CWD had been documented in free-ranging deer and elk in northeastern Colorado/southeastern Wyoming, and active surveillance in 2000 revealed that this endemic area extended into adjacent western Nebraska. From 1996 through early 2002, CWD has been diagnosed in privately owned captive elk herds in Colorado, Kansas, Montana, Nebraska, Oklahoma, South Dakota, and the Canadian provinces of Alberta and Saskatchewan. From late 2000 to April 2002, CWD also has been detected in wild deer at locations in northwestern Nebraska,

Saskatchewan, South Dakota, Wisconsin, and on the Western Slope of Colorado.

The origin of CWD is unknown, and it may never be possible to definitively determine how or when CWD arose. It is possible that CWD was derived from scrapie, a TSE of domestic sheep recognized in the United States since 1947. Arguments can be made both for and against this hypothesis. It also is possible that CWD is a spontaneous TSE that arose in deer in the wild or in captivity and has biological features promoting transmission to other deer and elk. The majority of cases of the human TSE Creutzfeldt-Jakob Disease (CJD) are thought to be spontaneous. Occurrence of spontaneous CJD is approximately one case per one million humans per year. It is possible, but difficult, to prove that spontaneous CWD may have occurred in deer.

Only three species of the family Cervidae are known to be naturally susceptible to CWD: mule deer (*Odocoileus hemionus*), white-tailed deer (*Odocoileus virginianus*), and Rocky Mountain elk (*Cervus elaphus nelsoni*), though it is very likely that other subspecies of *C. elaphus* are susceptible. Susceptibility of other cervid species to CWD is not known. Cattle and other domestic livestock appear to be resistant to natural infection; to date, only 3 of 13 cattle have become infected with the CWD agent following experimental intracerebral inoculation, although this and other experimental studies begun in 1997 are not yet completed.

Chronic wasting disease-affected deer and elk show loss of body condition and changes in behavior. The clinical disease often is more subtle and prolonged in elk than in deer. Affected animals may walk repetitive courses; show subtle ataxia and a wide-based stance; have subtle head tremors; be found near water sources or in riparian areas; have periods of somnolence; and may carry their head and ears lowered. Chronic wasting disease-affected animals continue to eat, but amounts of feed consumed are reduced, leading to gradual loss

of body condition. Excessive drinking, urination, salivation, and drooling are common in the terminal stages. Death is inevitable once clinical disease occurs.

The clinical course of CWD varies from a few days to approximately a year, with most animals surviving from a few weeks to several months after onset of signs. While a protracted clinical course is typical, occasionally acute death may occur. This may be more common in the wild than in the relative security of captivity. Aspiration pneumonia is a common finding at postmortem examination of terminal CWD cases and presumably is due to difficulty swallowing, hypersalivation, and inhalation of foreign material into the lungs. Thus the brain should be examined for evidence of CWD on every prime age cervid that dies with pneumonia. (Excerpted and modified from: Williams, Elizabeth S., Michael W. Miller, and E. Tom Thorne. Chronic Wasting Disease: Implications and Challenges for Wildlife Managers. *Transactions of the 67th North American Wildlife and Natural Resources Conference*. In Press.)

### **CWD Epidemiology**

Chronic wasting disease (CWD) is both transmissible and infectious, but many details of its transmission remain to be determined. In contrast to bovine spongiform encephalopathy (BSE), CWD is not a foodborne disease associated with rendered ruminant meat and bonemeal. Instead, observations of CWD among captive deer and elk provide strong evidence of lateral transmission, which is more similar to scrapie of sheep and goats. Experimental and epidemic modeling data support these anecdotal observations. Maternal transmission may occur but appears to be relatively rare. Some interspecies transmission probably occurs among the three natural host species. Apparent transmission from mule deer to elk, mule deer to white-tailed deer, and elk to mule deer and white-tailed deer has been observed.

The presumed CWD agent has been demonstrated by special procedures in various lymphoid tissues, including those of the digestive tract (e.g., tonsil, Peyer's patches, and mesenteric lymph nodes). These distribution patterns suggest that the agent may be shed through the alimentary tract. Because TSE agents are extremely resistant in the environment, transmission may be both direct and indirect. Concentrating deer and elk in captivity or by artificial feeding probably increases the likelihood of direct and indirect transmission between individuals. Contaminated pastures appear to have served as sources of infection in some CWD epidemics; similar phenomena have been suspected in some outbreaks of sheep scrapie. The apparent persistence of the CWD agent in contaminated environments represents a significant obstacle to eradication of the disease from either farmed or free-ranging cervid populations.

The overall duration of CWD infection (time from exposure to end-stage clinical disease) has been difficult to determine in natural cases. Without clear knowledge of when animals become infected, it is impossible to accurately determine the overall course of disease. Experimental CWD challenge studies based on single-dose oral exposure to infectious brain tissue have yielded some insights into the disease course; however, experimental data probably underestimate time frames for natural infections. Experimentally, minimum incubation time was about 15 months, and mean time from oral infection to death was about 23 months (range 20 to 25 months) in mule deer. The range of incubation observed in orally infected elk was approximately 12 to 34 months. The maximum disease course is not known, but can exceed 25 months in experimentally infected deer and 34 months in elk. Duration is less certain in naturally occurring cases. The youngest animal diagnosed with clinical CWD was 17 months old, suggesting 16 to 17 months may be the minimum natural incubation period. Among deer and elk residing in facilities with a long history of CWD, most natural cases occur in 2- to 7-year-old animals; however, deer have

lived more than 7 years in heavily infected facilities without succumbing to CWD, and elk more than 15 years of age have succumbed to CWD. It is not known when during the course of infection an animal may become infectious, but it appears likely that shedding of the CWD agent is progressive through the disease course; epidemic models suggest shedding probably precedes onset of clinical disease in both deer and elk.

Chronic wasting disease can reach remarkably high prevalence in captive cervid populations. In one infected research facility, more than 90% of mule deer resident for more than 2 years died or were euthanized while suffering from CWD. Recently, high CWD prevalence (about 50%) has been demonstrated in white-tailed deer confined in association with an infected Nebraska elk farm. Among captive elk, CWD was the primary cause of adult mortality (5 of 7, 71%; 4 of 23, 23%) in 2 research herds, and high prevalence (59%) was detected in a group of 17 elk slaughtered from an infected farm herd.

To estimate prevalence in infected free-ranging populations, tissues from deer and elk harvested by hunters in CWD-endemic areas have been collected and examined at random. Within endemic areas, prevalence of preclinical CWD has been estimated at less than 1% in elk and less than 1% to 15% in mule deer. Modeled CWD epidemics failed to achieve a steady-state equilibrium in infected deer populations, suggesting that CWD may lead to local extirpations of infected deer populations if left unmanaged. (Excerpted and modified from: Williams, Elizabeth S., Michael W. Miller, and E. Tom Thorne. *Chronic Wasting Disease: Implications and Challenges for Wildlife Managers. Transactions of the 67th North American Wildlife and Natural Resources Conference*. In Press.)

### **CWD Diagnosis**

Chronic wasting disease (CWD) cannot be diagnosed on the basis of clinical signs, because



several neurological diseases and other maladies may produce similar physical or behavioral abnormalities in deer and elk. Currently there is no approved live animal test for CWD, and the diagnosis is made by postmortem microscopic examination of brain for lesions (spongiform change with degeneration and loss of neurons) and/or for accumulation of abnormal prions (PrP<sup>CWD</sup>), the presumed causative agent of CWD.

Microscopic lesions are apparent only after the onset of clinical disease in deer and elk, but PrP<sup>CWD</sup> accumulation can be detected in animals before clinical signs develop. Although there is widespread PrP<sup>CWD</sup> accumulation in the brain later in the course of infection when clinical signs are apparent, early accumulation of PrP<sup>CWD</sup> is known to occur only in a very specific site in clinically normal animals. This site is located in the medulla oblongata of the brainstem directly beneath the cerebellum. With practice, this portion of the brainstem can easily be removed and preserved in formalin for testing. Immunohistochemical (IHC) staining of this area is used to demonstrate the presence or absence of PrP<sup>CWD</sup>, and currently it is regarded as the only meaningful test for clinically normal animals. A positive IHC result, with or without spongiform lesions, is sufficient to diagnose CWD infection. In some cases, supplementary tests may be employed to confirm a positive IHC result.

Recent investigations have demonstrated a potential live animal test for CWD in mule deer. The procedure involves IHC staining for PrP<sup>CWD</sup> within lymphoid tissue obtained via tonsil biopsy. Studies have shown that PrP<sup>CWD</sup> accumulates in lymph nodes and tonsil of mule deer early during CWD incubation. Although useful in special situations for mule deer and potentially of use in whitetails, the tonsil test does not appear to be of use for antemortem CWD testing of elk, due to differences in CWD pathogenesis in this species. Furthermore, there is little chance that this live animal test would have much application on a large scale in wild populations, because individual animals would

have to be captured and anesthetized for the biopsy procedure. If test results are positive, the animal would then have to be located and euthanized in most CWD control programs. Several other diagnostic tests for transmissible spongiform encephalopathies in other species, including BSE in cattle and scrapie in sheep, are being evaluated for potential use in CWD diagnostic testing of deer and elk. Some of these tests, if validated, may provide more rapid testing than current IHC procedures. (Prepared by Rick Gerhold)

Additional information:

Williams, Elizabeth S., Michael W. Miller, and E. Tom Thorne. Chronic Wasting Disease: Implications and Challenges for Wildlife Managers. *Transactions of the 67th North American Wildlife and Natural Resources Conference*. In Press.

### **CWD Surveillance, Doing the Numbers**

The known distribution of chronic wasting disease (CWD) has expanded at an alarming rate in the past 2 years, and there will be increasing pressure on wildlife managers to evaluate the status of deer and elk populations under their authority. Unfortunately, detection of CWD or confirmation that the disease is not present will require both hard work and money. The primary test currently available for CWD requires brain tissue from deer or elk, and laboratory costs alone are \$20 or greater per animal. The funds necessary to collect and test the number of samples required to reach a defensible position on the absence of CWD in a deer or elk herd will strain wildlife agency budgets, and the flood of samples will overwhelm existing laboratory capabilities nationwide.

How to look for CWD is no simple matter. Presently, the two surveillance strategies generally used are: (1) testing of sick or "target animals" discovered or reported to wildlife agencies (a passive surveillance system) or (2) conducting random surveys of normal deer or elk killed by hunters or agency personnel (an

active surveillance system). CWD “target” animals are defined as deer or elk of 18 months of age or older that are emaciated and showing some combination of signs including abnormal behavior, increased salivation, tremors, stumbling, incoordination, difficulty swallowing, excessive thirst, and excessive urination. Perhaps it would be better to simplify the definition to include *sick-appearing deer or elk that are emaciated or have neurologic disorders*.

The best epidemiological information available on CWD in wild populations comes from the long-term study of deer and elk in the endemic area in northeastern Colorado and southeastern Wyoming. A crucial fact from this study is low prevalence of CWD infection found in the wild. Overall, prevalence rates in the endemic zone were 4.7% for mule deer, 2% for white-tailed deer, and 0.5% for elk, although there were “hot spots” within the study area that ran as high as 15% in mule deer. Unfortunately, what this means for wildlife biologists is that sample sizes in active surveillance programs must be massive before any credence can be given to negative results. Thus, costs of active surveillance are going to be high.

In contrast, when the aforementioned researchers evaluated their success in diagnosing CWD from clinically ill (target) deer and elk in the endemic area, their overall case positive rates were 57%, 35%, and 44% for mule deer, white-tailed deer, and elk, respectively. They concluded that “targeted surveillance appears to be an effective strategy for detecting new CWD foci.” However, it also was concluded that the first clinically ill animals were not apparent in various management units until the prevalence rate was about 1%. Furthermore, the numbers of sick deer submitted from a given management unit did not correspond with the prevalence rate determined by random sampling of that area. They found that the success of targeted surveillance was tied to human activity in the area and the level of concern about the disease.

For the past 4 years, SCWDS has sent a surveillance questionnaire to wildlife management agencies and veterinary diagnostic laboratories seeking reports of target deer and elk that were examined for CWD. We believe that the questionnaires are stimulating increased attention in regard to CWD; however, they revealed marked inconsistencies among states in the detection of “target animals.” This was best exemplified by numerous states indicating that no target animals were identified over the course of a year or more. “Targeted surveillance” is a passive system that relies heavily on the public reporting sick animals to appropriate agency personnel and then on follow-up action to acquire and test samples. The aim of targeted surveillance is to locate and test the animals most likely to be infected, thereby reducing surveillance costs, while at the same time enabling coverage of a large geographic area.

Given the expanding concern about CWD, and the very real threat CWD poses to native cervids, wildlife managers can no longer afford to be casual about the discovery of any sick deer or elk. It is imperative that these animals are obtained and tested for CWD. Furthermore, given that active surveillance has been the method by which CWD has been detected among wild deer in several new locations including Saskatchewan, Nebraska, South Dakota, and Wisconsin, it is becoming clear that both surveillance methods should be strongly considered by wildlife management agencies. (Prepared by Vic Nettles)

#### Additional Information:

Miller, Michael W., et al. 2000. Epizootiology of chronic wasting disease in free-ranging cervids in Colorado and Wyoming. *Journal of Wildlife Diseases* 36(4): 676-690.

#### CWD Management

There is no treatment or vaccine for chronic wasting disease (CWD). Once clinical signs develop, CWD is invariably fatal. Furthermore,

long incubation periods, subtle early clinical signs, absence of a reliable live animal test, extremely resistant infectious agent, possible environmental contamination, and incomplete understanding of transmission all limit options for controlling or eradicating CWD.

In captive facilities, management options currently are limited to quarantine or depopulation of CWD-infected herds. Two attempts to eradicate CWD from cervid research facilities failed. The causes of these failures were not determined, but residual environmental contamination following depopulation and/or facility clean-up was likely in both cases. Attempts to eliminate CWD from farmed elk populations are more recent, and consequently the efficacy of these attempts remains uncertain. Whether contaminated environments can ever be completely disinfected remains questionable. Until effective cleaning and disinfection procedures are identified, a very conservative approach must be taken when considering reintroduction of captive cervids into commercial facilities where CWD has occurred; moreover, free-ranging cervids also should be excluded from previously infected premises. Inherent difficulties in managing infected captive herds and premises underscore the need for aggressive surveillance to prevent movements of infected animals in commerce.

Managing CWD in free-ranging animals presents even greater challenges. Long-term, active surveillance programs to monitor CWD distribution and prevalence have been instituted in the endemic area in Colorado, Nebraska, and Wyoming to determine its extent and to assist in evaluating both temporal changes and effects of management intervention. Management programs established to date focus on containing CWD and reducing its prevalence in localized areas. Ultimate management goals vary among affected states and provinces. In Nebraska, Wisconsin, Saskatchewan, and on Colorado's Western Slope where CWD may not yet be endemic, eradication is the stated goal for CWD management. In contrast, wildlife managers in the CWD endemic area in Colorado

and Wyoming have refrained from committing to eradication because it appears unattainable in their situations.

A variety of specific strategies for managing CWD in free-ranging wildlife have been adopted in affected jurisdictions. Translocating and artificially feeding cervids in endemic areas have been banned in attempts to limit range expansion and decrease transmission. Selective culling of clinical suspects has been practiced throughout the endemic area of Colorado and Wyoming for a number of years, but this approach alone is insufficient to reduce prevalence in affected populations. Localized population reduction in an area of high CWD prevalence has been undertaken in Colorado as a management experiment, but efficacy remains to be determined. Although it seems intuitive that lowered deer and elk densities should reduce both transmission and likelihood of emigration of infected animals to adjacent areas, historic migration patterns and social behaviors characteristic of some deer and elk populations may diminish the effectiveness of wholesale density reduction in controlling CWD.

Computerized epidemiologic modeling indicates that early and aggressive intervention through population reduction offers the best chance to prevent establishment of new endemic foci. Unfortunately, surveillance limitations (both cost and sensitivity) may delay detection of newly infected free-ranging populations for years after CWD has been introduced. In Nebraska, Saskatchewan, Wisconsin, and on Colorado's Western Slope, aggressive reductions of deer numbers in newly identified positive locations are underway or planned in attempts to eliminate CWD from these areas. The recent development of tonsil biopsy as a live animal test for CWD in deer might aid control efforts under some special conditions, but large-scale applications to free-ranging populations seem impractical.

Where it occurs, CWD in captive and free-ranging cervids presents serious management problems. Captive populations are quarantined,

thus limiting use and value of infected or exposed animals. Indemnity for depopulated cervids has been made available only recently in the United States. In Canada, the magnitude of infection in farmed elk herds detected thus far has cost the Canadian government over \$19,000,000 in indemnity and cleanup funds. Guidelines for management of captive herds with CWD are being developed by state and provincial animal health officials. A national program is nearing adoption in Canada, and a similar program currently is under review in the United States. Spillover of CWD into local free-ranging cervid populations may have occurred in two locations; further spillover could establish more endemic foci, thereby impairing long-term viability of both cervid farming and wildlife management in those areas.

Implications for management of free-ranging populations of deer and elk may be even more significant. Agencies do not translocate deer and elk from CWD endemic areas. Ongoing surveillance programs are expensive and draw resources from other wildlife management needs. Perhaps most important, impacts of CWD on population dynamics of deer and elk are presently unknown. Modeling suggests that CWD could substantially harm infected cervid populations by lowering adult survival rates and destabilizing long-term population dynamics. Ultimately, public and agency concerns and perceptions about human health risks associated with all transmissible spongiform encephalopathies may erode participation in sport hunting in the endemic area and also may have dramatic influence on management of free-ranging cervid herds where CWD is endemic. It follows that wildlife management and animal health agencies should continue working to understand, prevent introduction, and limit the distribution and occurrence of CWD in free-ranging and farmed cervids. (Excerpted and modified from: Williams, Elizabeth S., Michael W. Miller, and E. Tom Thorne. Chronic Wasting Disease: Implications and Challenges for Wildlife Managers. *Transactions of the*

*67th North American Wildlife and Natural Resources Conference.* In Press.)

### **CWD in Captive Cervids**

Although chronic wasting disease (CWD) was recognized in captive deer held in publicly owned research facilities in Colorado in the 1960s, it was not detected in privately owned commercial cervids until 1996 in Saskatchewan. Since then, 1 or more cases of CWD have been confirmed in elk in 39 captive herds in Saskatchewan. Primary or secondary epidemiological links have been found between every infected herd and a single source herd in the province that apparently imported infected elk from a private South Dakota herd in 1989. In another example of international movement of CWD, the disease was diagnosed in captive elk in Korea that reportedly had been imported from Saskatchewan. It was announced in early 2002 that one infected captive elk herd had been found in Alberta, and an epidemiological investigation is underway. CWD is a reportable disease under Canada's federal Health of Animals Act, and the Canada Food Inspection Agency has responsibility for controlling the disease. Nearly 8,000 captive elk in Saskatchewan have been destroyed in eradication efforts to date at a cost of more than \$19,000,000 for indemnification and cleanup.

In 1997, South Dakota became the first state to detect CWD within the commercial elk industry. As of May 2002, CWD has been diagnosed in 20 privately owned elk herds in Colorado (7), South Dakota (7), Kansas (1), Montana (1), Nebraska (3), and Oklahoma (1). Epidemiological investigations have disclosed animal movement among some of the infected elk ranches. A group of interconnected facilities near Rapid City, South Dakota, that experienced particularly severe infection appeared to be the original source of CWD for other South Dakota game farms. Additional investigations have revealed that two herds of the infected elk in Nebraska originated in Colorado, infected elk in Oklahoma apparently originated in Montana, and CWD subsequently was confirmed in the

Colorado and Montana source herds. In late 2001, CWD was found in captive elk shipped from an infected Colorado elk ranch to two herds in Colorado and one herd in Kansas. Over the course of several years, exposed animals from the source herd in Colorado had been shipped to captive elk operations in 19 states as well as to more than 40 other commercial facilities in Colorado. The other living elk shipped from this facility have been traced, euthanized, and tested for CWD with negative results.

All but two of the infected captive herds found in the United States have been depopulated. One Nebraska herd was quarantined, monitored for additional cases of CWD, and released from quarantine after no more cases were found in 3 years. The Oklahoma herd has not been depopulated despite the recognition of infected elk in 1998, but it recently was appraised in preparation for euthanasia, testing, and disposal of the animals. As is the case with the Oklahoma herd, there are other situations where there was an extended delay between the diagnosis of CWD and herd depopulation. In several instances this delay occurred while arrangements were made with state and federal animal health authorities for compensation of the owner.

Indemnification for captive elk destroyed in CWD control efforts is one component of the proposed program currently being developed by USDA's Animal and Plant Health Inspection Service (APHIS). The APHIS program, which was requested by the commercial elk industry, is being put together with their assistance as well as with input from animal health officials, wildlife management agencies, and other organizations. Drafts of the proposed program have been informally reviewed, and a formal document is being prepared for publication in the Federal Register. The major components of the program are indemnification for elk destroyed in CWD-control efforts, a monitoring and certification program for captive elk herds, and for herd plan guidelines for positive facilities. The programs as currently proposed

would be voluntary unless interstate shipment of animals is involved and will apply only to captive elk with no provisions for other cervid species.

In September 2001, the USDA issued a declaration of emergency regarding CWD, and approximately \$2.6 million were made available in Commodity Credit Corporation (CCC) funds for indemnification and surveillance. In February 2002, an Interim Rule was published in the Federal Register regarding CWD indemnification and another \$12.2 million in CCC funds were released to support CWD control efforts. In addition to compensating owners for the destruction of positive or exposed cervids, the USDA recently purchased approximately 1,350 elk in the northeastern portion of Colorado where CWD is endemic in wild deer and elk. Owners of nearly all of the captive elk herds in the endemic area participated in this buyout and agreed to never restock their property with cervids because of the continued disease risk. There will be no new captive cervid facilities constructed in Colorado's endemic area. Requests for similar federal assistance are expected from other states that recently have found CWD in wild animals.

Individual states may implement more stringent regulations than are outlined in the proposed federal CWD control program. In response to the identification of CWD in captive and wild cervids in several locations, numerous states recently have imposed bans or stringent regulations on live cervid movement. In some states, these regulations apply to animals originating from areas where CWD has been detected, while in others there is total prohibition of live cervid importation. Additionally, some states now require mandatory CWD surveillance of captive deer and elk. (Prepared by John Fischer)

Additional sources of information:

Canadian Food Inspection Agency  
([www.inspection.gc.ca](http://www.inspection.gc.ca))

USDA's Animal and Plant Health Inspection Service ([www.aphis.usda.gov](http://www.aphis.usda.gov))

### **CWD in Wild Deer and Elk**

In recent years, wildlife agencies in numerous states and Canadian provinces have worked diligently to identify the distribution and prevalence of CWD in wild deer and elk. This surveillance has resulted in the discovery of several new foci of CWD among wild cervids since 2000. This report summarizes the circumstances of occurrence and detection of CWD in wild deer and elk as of late May 2002.

Endemic area in Colorado, Wyoming, and Nebraska: In 1981, CWD was diagnosed for the first time in clinically ill free-ranging elk in northeastern Colorado. Later, clinically affected wild elk were found in adjacent southeastern Wyoming. By 1990, wild mule deer and white-tailed deer with CWD had been found in the same regions of both states. Initially, the detection of CWD in these two states resulted from submission of clinically affected animals for diagnosis. Additionally, active surveillance programs conducted by the Colorado Division of Wildlife and the Wyoming Game and Fish Department were developed in which hunter-killed, road-killed, or culled deer and elk were tested for CWD. This combination of targeted and active surveillance provided the information that allowed delineation of the CWD-endemic focus within Colorado and Wyoming. During 2000, active surveillance of hunter-killed deer in the adjacent southwestern corner of Nebraska disclosed an infected mule deer in Kimball County. Further active surveillance in 2001 has disclosed two additional infected deer in Kimball County and one in Cheyenne County. In early 2002, the Nebraska Game and Parks Commission (NGPC) announced that a clinically affected deer had tested positive for CWD in Scotts Bluff County, two counties to the north of Kimball County on Nebraska's western border. Prevalence of infection in the Colorado and Wyoming portions of this endemic focus has been estimated at less than 1% for elk, about 5% for mule deer, and about

2% for white-tailed deer. Local prevalence varied widely, however, and some subpopulations of mule deer had prevalences of 15%.

Saskatchewan: In Saskatchewan, CWD first was detected in 2000 in a mule deer killed by a hunter near the Alberta border. This animal was tested as part of active surveillance of approximately 1,400 hunter-harvested deer and elk following confirmation of CWD in more than 30 captive elk herds in Saskatchewan. Subsequent active surveillance conducted by Saskatchewan Environment and Resource Management personnel disclosed a second positive mule deer within the same general area. Although the source of infection in these wild animals is unknown, it is regarded as a possible spillover from infected farmed elk. Herd reduction efforts are underway in an effort to control the spread of CWD in wild cervids. Active province-wide surveillance of more than 4,000 hunter-killed wild deer and elk in the fall of 2001 yielded uniformly negative results.

Northwestern Nebraska: In 2001, active surveillance in the vicinity of a CWD-positive captive elk herd in Sioux County disclosed several infected white-tailed deer. On the ranch with the positive elk, a CWD infection rate of approximately 50% was found among 179 wild deer inside a high fence enclosure built on the property at the time the elk enclosure was constructed in the early 1990s. Among the positive deer were four fawns approximately 8-months old. Within a radius of 5 miles surrounding the property, 7 of 103 deer (6.8%) were CWD positive, and this prevalence dropped to 3.5% among 57 deer tested in the zone between 5 and 10 miles from the property. Of 125 deer tested between 10 and 20 miles from the facility, a single positive animal was found at a distance of 11 miles. The Nebraska Game and Parks Commission (NGPC) continues to monitor this focus of infection and has implemented an aggressive disease management plan to prevent the spread of CWD throughout the state's wild deer.

Southwestern South Dakota: The South Dakota Department of Game, Fish, and Parks began active surveillance for CWD after several positive captive elk herds were identified in 1997-1998. In 1998, a single positive white-tailed deer was found among 30 formerly wild deer that had been enclosed within one of the private elk facilities. From 1997-1999, positive free-ranging animals were not detected among approximately 1,000 deer and elk tested throughout the state. Sampling did not occur in 2000; however, 1 positive deer was found among approximately 500 hunter-killed deer and elk tested in 2001. The positive animal was from Fall River County in southwestern South Dakota near the Nebraska border. The animal was harvested in the area where CWD previously had occurred in captive elk herds as well as within 50 miles of the northwestern Nebraska captive elk facility where CWD had been diagnosed in captive and wild cervids. Active surveillance has been conducted in the vicinity where the positive South Dakota deer was found as well as in the intervening area between this location and the affected area in northwestern Nebraska. To date, additional positive animals have not been found.

Western Slope of Colorado: In March 2002 the Colorado Division of Wildlife (CDOW) announced it had confirmed CWD infection for the first time in mule deer west of the continental divide in Routt County, Colorado. In accordance with CDOW policy, wild deer and elk that had been inadvertently confined with captive-bred elk in a facility constructed in the summer of 2001 were destroyed in order to prevent them from escaping back into the wild. Two deer of 340 entrapped wild deer and elk killed in the enclosure tested positive for CWD. From April 1-3, 2002, personnel from CDOW and USDA-APHIS' Wildlife Services collected 308 wild deer within a 5-mile radius outside the private facility. Two deer collected during this operation tested positive for CWD, and 1 more infected deer was found among 18 deer subsequently killed in the same immediate area. Beginning April 15, 2002, an additional 135 elk and 285 deer were killed within 5 miles of the

private enclosure in an aggressive attempt to kill as many wild deer and elk as possible before the animals began seasonal migration out of the area. This task was carried out by CDOW and Wildlife Services personnel, assisted by volunteers from the Mule Deer Association, Rocky Mountain Elk Foundation, Traditional Bowhunters Association, Colorado Outfitters Association, and Colorado Bowhunters Association. None of the elk tested positive, but three more positive deer were identified. This brings the total number of positive deer outside the facility to 6 of 633 killed and tested, indicating a prevalence of less than 1% in the wild deer in the area. The source of this focus has yet to be determined. Since 1996, CDOW has tested more than 2,000 animals throughout the non-endemic portion of the state for CWD, including animals from each of the large mule deer herds on the Western Slope. None has been infected, with the exception of the recent Routt County outbreak.

Wisconsin: The Wisconsin Department of Natural Resources (DNR) began active surveillance of hunter-killed white-tailed deer in 1999 and through the 2001 hunting season had sampled more than 1,000 deer. On February 28, 2002, it was announced that three deer, all harvested within 2 miles of each other in Deer Management Unit (DMU) 70A in Dane County in 2001, were CWD positive by immunohistochemistry assay of brain stem. One deer was emaciated and lethargic when shot by the hunter. Subsequent collection and testing of 516 deer within a 5-mile radius centered around the positive animals disclosed an additional 15 (2.9%) infected animals during March-April 2002. The origin of this focus of CWD is not yet clear, but the absence of any positive deer in other DMUs argues against the concept of a long-term, naturally occurring enzootic presence in the state. In response to this crisis, the Wisconsin DNR currently is developing and implementing an aggressive disease management program to kill as many deer as possible within a 328-square-mile area. In one component of the program, landowners will be authorized to shoot deer on their

property in the "Chronic Wasting Disease Eradication Zone" for 1 week per month during June, July, August, and September. Thousands of wild whitetails are targeted for depopulation in this effort to prevent CWD's spread throughout Wisconsin's deer herd. (Prepared by Randy Davidson)

Additional sources of information:

Colorado Department of Wildlife  
([www.wildlife.state.co.us](http://www.wildlife.state.co.us))

Nebraska Game and Parks Department  
([www.ngpc.state.ne.us/wildlife/cwd/cwdinfo.html](http://www.ngpc.state.ne.us/wildlife/cwd/cwdinfo.html))

Saskatchewan Environment and Resource Management ([www.serm.gov.sk.ca](http://www.serm.gov.sk.ca))

South Dakota Game, Fish and Parks  
([www.state.sd.us/gfp/](http://www.state.sd.us/gfp/))

Wisconsin Department of Natural Resources  
([www.dnr.state.wi.us](http://www.dnr.state.wi.us))

Wyoming Game and Fish ([gf.state.wy.us](http://gf.state.wy.us))

### **Public Health Concerns About CWD**

There currently is no convincing evidence that the agent of chronic wasting disease (CWD) affects humans. However, public health officials recommend that human exposure to the CWD agent be avoided as they continue to evaluate any potential risk. Contrary to a story that circulated in the popular press, an investigation by the Centers for Disease Control and Prevention (CDC) did not find a link between CWD and Creutzfeldt-Jakob Disease (CJD) in three persons, aged 28-30 years, who consumed venison. The investigators concluded: "Although the occurrence of 3 unusually young patients with CJD who consumed venison suggested a possible relationship with CWD, our follow-up investigation found no strong evidence for a causal link. Ongoing CJD surveillance remains

important for continuing to assess the risk, if any, of CWD transmission to humans."

The tendency toward a natural "species barrier" reducing human susceptibility to CWD and other prion diseases has been demonstrated by *in vitro* studies. However, lingering uncertainty about any potential risk that CWD may pose to humans is fostered by differing experiences with two more common animal TSEs. Although there is a long history of human exposure to scrapie through handling and consuming sheep tissues, including brain, there is no evidence that scrapie presents a risk to human health. In contrast, massive exposure of British and perhaps other European citizens to the BSE agent has resulted in approximately 130 human cases of variant CJD as of April 2002. In 2000, the World Health Organization (WHO) published the proceedings of the *WHO Consultation on Public Health and Animal Transmissible Spongiform Encephalopathies: Epidemiology, Risk and Research Requirement*. The consultants consisted of researchers from around the world who are experts on human and animal spongiform encephalopathies. The consultants summarized their findings with the statement: "There currently is no evidence that Chronic Wasting Disease in Cervidae is transmitted to humans."

This situation has not changed. Despite the lack of a causal link between CWD in deer and elk and CJD in humans, the WHO Consultation made the following recommendations regarding CWD: Authorities should encourage awareness and surveillance for CWD around the world; no part or product of any animals with evidence of CWD or other TSEs should be fed to humans or any species of domestic or captive animal; work should continue to improve the understanding of CWD; and precaution should be taken to prevent introduction and spread of CWD when translocating wild or domestic cervids.

Similarly, public health and wildlife management professionals encourage hunters, meat processors, and taxidermists to take some common sense measures to prevent potential



exposure to the CWD agent and to other known zoonotic pathogens. For example, the Colorado Division of Wildlife recommends the following precautions for hunters in the CWD endemic area: Do not shoot, handle or consume any animal that appears sick; contact the Division of Wildlife if you see or harvest an animal that appears sick.

- Wear rubber gloves when field dressing carcasses.
- Bone out the meat from your animal.
- Minimize the handling of brain and spinal tissues.
- Wash hands and instruments thoroughly after field dressing is completed.
- Avoid consuming brain, spinal cord, eyes, spleen, tonsils, and lymph nodes of harvested animals.
- Avoid consuming the meat from any animal that tests positive for the disease.
- Request that your animal be processed individually, without meat from other animals being added to meat from your animal.

(Prepared by John Fischer)

#### Additional Sources of Information:

Belay, Ermias D. et al. 2001. Creutzfeldt-Jakob Disease in unusually young patients who consumed venison. *Archives of Neurology* 58:1673-1678.

Williams, Elizabeth S., Michael W. Miller, and E. Tom Thorne. Chronic Wasting Disease: Implications and Challenges for Wildlife Managers. *Transactions of the 67th North American Wildlife and Natural Resources Conference*. In Press.

Centers for Disease Control and Prevention ([www.cdc.gov](http://www.cdc.gov))

Colorado Division of Wildlife ([www.wildlife.state.co.us](http://www.wildlife.state.co.us))

World Health Organization ([www.who.int](http://www.who.int))

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