

**REPORT OF THE
DEPARTMENT OF INLAND FISHERIES AND WILDLIFE**

**BEFORE THE JOINT STANDING COMMITTEE ON INLAND FISHERIES &
WILDLIFE**

L.D. 445

**RESOLVE, DIRECTING THE DEPARTMENT OF INLAND FISHERIES AND
WILDLIFE TO STUDY SCENTS USED IN HUNTING DEER AND RENDERINGS
USED IN DEER FEED**

SPONSORED BY: REPRESENTATIVE SANDERSON OF CHELSEA

COSPONSORED BY: REPRESENTATIVE CEBRA OF NAPLES

REPRESENTATIVE COTTA OF CHINA

REPRESENTATIVE DAVIS OF SANGERVILLE

REPRESENTATIVE HARVELL OF FARMINGTON

REPRESENTATIVE MCKANE OF NEWCASTLE

REPRESENTATIVE MORISSETTE OF WINSLOW

REPRESENTATIVE PARRY OF ARUNDEL

REPRESENTATIVE SARTY OF DENMARK

SENATOR TRAHAN OF LINCOLN

JANUARY 9, 2012

Executive Summary

It is known that: 1) the infectious agent causing Chronic Wasting Disease (CWD) in North American deer, a mutant protein or prion, can be carried in bodily fluids such as urine and transmitted to other deer that come into contact with infected excretions; 2) the captive deer industry continues to spread CWD among captive deer farms within and across state boundaries; 3) some deer farms associated with urine products have not been compliant with mandatory disease testing and prevention rules; and 4) to date, testing of free-ranging deer in Maine at a level that guarantees a 99% chance of detecting a 0.1% infection rate has *not* found CWD to be present in Maine; furthermore, no State in New England has detected CWD yet despite similar levels of testing.

At this time, the Maine Department of Inland Fisheries and Wildlife (MDIFW) concurs with the findings of other wildlife biologists and wildlife veterinarians that the risk of introducing infectious prions into the environment through the dissemination of natural urine-based lures taken from captive deer of unknown origin, that could lead to CWD in Maine's free-ranging CWD-free population of white-tailed deer, may not be quantifiable, but the existence of such risk

is undeniable. Given the history of prevalence and spread of CWD among captive deer herds, it seems prudent to prohibit or minimize spread of urine from captive deer of unknown origin across the landscape. Artificial, or synthetic, scents should pose no such risk, but their origins could also be suspect. Such products, if used, should be placed where deer cannot touch them. MDIFW plans to continue its outreach efforts to hunters regarding this issue.

Based on our investigation, the MDIFW finds that the food and feed industry is checked and held in compliance with substantial effort by both federal and state agencies. By federal law, feed for ruminants such as deer containing prohibited materials for feeding of ruminants must be clearly marked “Do not feed to cattle or other ruminants”. Feed labeled as containing “animal protein” may contain prohibited materials, as described by FDA 21CFR589.2000, so should not be fed to deer. Also, *poultry, swine, and pet feed should never be fed to deer* due to relatively high likelihood of containing prohibited materials for ruminants and no labeling requirements for such products (regarding ruminants). Commercial food products for ruminants such as deer should pose little threat of introducing CWD into a CWD-free area as long as people feeding deer use FDA-approved feed for ruminants. People feeding deer should use locally-grown products or should be diligent about checking labels on bags of commercial feed, as federal laws dictate strict labeling requirements for feeds intended for ruminants.

MDIFW recommends that deer feed used in Maine contain proteins that only come from plants. Feeding of deer becomes a risk factor for spreading CWD after the disease has been introduced into an area because it congregates deer in small areas where they can more readily exchange bodily fluids. Feeding bans are common in areas where CWD is found to be newly established.

Background: CWD, Status in Maine, & Captive Deer of North America

Chronic wasting disease (CWD) is an infectious neurological disease of North American deer (Family Cervidae) and belongs to the group of infectious diseases known as transmissible spongiform encephalopathies (TSEs). Cervids of North America include white-tailed deer, black-tailed deer, mule deer, elk, moose, and caribou. CWD was first diagnosed as a TSE in research facilities of captive deer in Wyoming and Colorado in the early 1980s. Other TSEs include bovine spongiform encephalopathy, or “mad cow disease”, in cattle (*Bovis spp.*), scrapie in domestic sheep (*Ovis spp.*), and Creutzfeldt-Jakob disease in humans. There is no evidence of immunity to or recovery from CWD; it is always fatal to infected deer.

The causative agent for each of these diseases is a prion, or infectious mutant protein. Abnormal prions transform normal proteins into a form that does not perform its normal function. This ultimately kills the cell. The loss of nerve cells creates small holes in the brain, thus the spongiform description. Infectious prions have been found in nearly all types of tissue and bodily fluids of infected deer. Direct and indirect contact among deer is how the disease is most often spread. No link between CWD and disease in humans has been reported, but health agencies recommend that humans do not consume infected deer. It was recently shown that prions are capable of adaptation and evolution, despite the lack of DNA, so caution seems warranted.

Prions are difficult to detect and are persistent. Prions are extremely resistant to heat, cold, disinfectants and the host's immune system. Prions can bind to soil and persist in the environment for several years. Unlike other organisms such as viruses and bacteria, prions have no nucleus or natural antibodies, so common methods used to identify and detect the infectious agent are not possible for CWD. There is no reliable and practical live-test, and infected, contagious animals can look healthy for 1–3 years before developing clinical symptoms. To avoid false-negative test results, the brain-stem or retropharyngeal lymph nodes (in the throat) must be removed and examined in a certified laboratory.

CWD has been documented in: 1) wild elk in Colorado, New Mexico, and Wyoming; 2) wild moose in Colorado and Wyoming; 3) wild deer in Alberta, Colorado, Illinois, Maryland, Minnesota, Nebraska, New Mexico, New York, North Dakota, Saskatchewan, South Dakota, Utah, Virginia, West Virginia, Wisconsin, and Wyoming; 4) captive deer or elk in Alberta, Colorado, Kansas, Michigan, Minnesota, Missouri, Montana, Nebraska, New York, Oklahoma, Saskatchewan, South Dakota, and Wisconsin (see <http://www.cwd-info.org/index.php/fuseaction/about.map>).

The geographic distribution of the disease suggests there are multiple factors involved with the spread of the disease. CWD has spread slowly across Wyoming from the original endemic area on the Wyoming-Colorado border due to overland travel and deer dispersal behavior. The Wisconsin and Illinois cases, discovered in 2002, are 700 miles from the historic endemic area. When Wisconsin first tested free-ranging and captive deer for CWD in 2002, they found it in both groups. In 2005, CWD was diagnosed in captive and adjacent wild deer in New York, a distance of 850 miles from Wisconsin. CWD was also shipped to Canada and Korea in captive deer. CWD was recently introduced to Michigan and Missouri in captive deer. In 2011, CWD was newly discovered in another captive facility in Missouri.

CWD was found in two adjacent captive deer facilities in central New York in spring 2005; it was soon discovered in two free-ranging deer near those captive facilities. There is hope that the disease did not become established (>1% prevalence) in that population because liberal lethal sampling of free-ranging deer for 5 years has produced no more CWD-positive test results. Over 6,000 deer have been killed and sampled in New York's CWD Containment Zone since 2005.

CWD jumped from the historic endemic zone to New Mexico and West Virginia, both discovered in 2005, but captive deer were not implicated in those two cases. These cases suggest that transportation of infected animals, *or the parts or products* of infected cervids, have been the source of some new infected areas away from the initial endemic area. CWD has apparently spread overland from West Virginia through the northern corner of Virginia and into Maryland. From there it is a short distance to southern Pennsylvania.

CWD may be impossible to remove from a free-ranging population of deer after it becomes established at a prevalence rate of >1% of deer. Many southern and Midwestern states have deer populations that may be able to absorb another source of mortality, but populations at the northern extent of the range of the white-tailed deer, such as that in Maine, are already managed to limit mortality by hunting and other causes. CWD would likely negatively affect Maine's

moose population that already exhibits periodic signs of stress, possibly related to parasites such as winter ticks and lungworm.

Following Acts of Congress passed in 2003, the USDA has provided financial assistance to states to test and monitor both captive and free-ranging cervids for CWD. Annual samples taken from hunter-harvested deer and moose from 2002–2010 led to the determination that Maine is currently CWD-free and should be considered “at-risk”, with more than 99% assurance that an infection rate of 0.1% would have been detected. Establishment of CWD in Maine would place the State’s deer and moose resources at considerable risk.

Means by which CWD can be moved across state boundaries include the captive deer trade, transport of infected deer carcasses, importation of contaminated deer-feed products, introduction by contaminated deer products (e.g., urine), and overland movement of contaminated free-ranging deer. The nearest known CWD-endemic area with relation to Maine is currently Maryland. This assumes that depopulation of two captive deer facilities and liberal lethal sampling of free-ranging deer has successfully eradicated the disease from central New York before it had a chance of becoming 1% prevalent in the population after initial detection in spring of 2005 (no CWD-positive sampling results since). It should take many decades for CWD to reach Maine by overland travel from Maryland, but more immediate threats exist such as long-distance transportation of live or dead infected deer, or products made from infected deer.

It is widely accepted that key to management of this disease is prevention; in CWD-free states, agencies should take all reasonable measures to keep from getting it. Agencies must develop strategies to minimize risk factors. Risk factors include captive deer operations, high free-ranging deer densities, inter-state transportation of deer carcasses, deer feeding and baiting, and importation and use of captive-deer products such as urine. If these risk factors can be managed and minimized, diseases such as CWD should be unlikely to be introduced to Maine from distant locations.

Strict safeguards are the most effective means of preventing the introduction and establishment of this disease into Maine. Maine has already taken steps to minimize risk factors such as importation restrictions for captive deer, monitoring of CWD in captive deer herds, and a ban on whole-deer carcass imports from other states and provinces except those that directly border Maine and have had testing of wild deer for CWD. USDA and States’ rules in place since 2003 should have prevented spread of the disease among captive facilities since then, but it continues to be spread among captive facilities within and across state boundaries.

Spread of the infectious mutant protein, or prion, occurs via lymph tissues, antler velvet, blood, saliva, feces, and urine and can persist in soils for years. Prions have now been found in the body tissues that produce the various forms of solid and liquid excretions. A risk-based audit of captive cervid facilities in Michigan found that 9 of 11 facilities associated with deer-urine products were not compliant with mandatory rules for disease testing and prevention (D. O’Brien, Michigan DNR, unpublished data and personal communication).

Urine-Based Scent Lures: Strategies

Many states and provinces are providing information and educational materials explaining the risk of natural urine-based attractants from captive deer of unknown origin, while a few others have taken a more hard-line approach. Nova Scotia was the first to ban urine-based products in 2007. Ontario was the next to institute an outright ban in August 2010. The Ontario Ministry of Natural Resources explains the changes in regulations on their website in this way (accessed 16 November 2011):

“Ontario has passed an amendment to O. Reg 665/98 (Hunting) under the *Fish and Wildlife Conservation Act, 1997*. This amendment prohibits possession and use of products that contain, or purport to contain, body parts of any member of the deer family including blood, urine, gland oils and other fluids, for the purposes of hunting. This prohibition applies to body parts and fluids from hunter-harvested deer or moose and applies to all hunting in Ontario.

One potential pathway for the spread of CWD is from possession and use of hunting attractants that contain body parts of members of the deer family. These products contain urine, blood, gland oil or other bodily fluids obtained from captive/farmed deer, elk or other cervids. This regulation also prohibits hunters from possessing or using these types of materials (i.e., body fluids and parts) obtained from wild and farmed cervids for the purposes of hunting in Ontario. These products may contain infectious material and may be capable of introducing CWD to Ontario.”

In 2011, Manitoba introduced a ban on the possession of any product that contains urine, feces, saliva or scent glands of a cervid (Michigan Dept. of Natural Resources, 2011, A CWD Regulations Table for States and Provinces). No state has yet advertised a ban of such products, but state and provincial deer biologists from the Northeast Deer Technical Committee have been discussing the merits and drawbacks of doing so (L. Kantar, MDIFW, personal communication). Some Midwestern states are warning hunters about risks associated with deer-urine products and are considering bans or alternatives to outright bans (D. Lopez, CWD Coordinator, Wisconsin DNR, pers. comm.).

[*Excerpted from a November 2, 2010 article by Jeffrey L. Frischkorn of The News Herald, northern Ohio:* "There is an undeniable yet unquantifiable risk associated with the use of all-natural urine-based attractants," said Mike Tonkovich, deer project leader for the Ohio Division of Wildlife. "For that reason, the Division of Wildlife encourages hunters to use synthetic alternatives wherever possible until more is known about the risk of disease transmission through the use of these products."]

In Wisconsin, where the CWD issue first took on a head of steam, that state's Department of Natural Resources restricts how much deer-attracting scent can be used. While up to two ounces of scent material is allowed for deposition on the ground or on some type of vegetation, anything more cannot be accessible to deer and also must be removed by day's end, Wisconsin law requires.

Jerry Feaser, press secretary for the Pennsylvania Game Commission, said his agency's wildlife veterinarian has also studied the question. He then presented his findings to the Game Commission's board of game commissioners.

"As part of that, we outlined some of the things that could be done to further protect Pennsylvania of having CWD accidentally introduced," Feaser said. "One of those was the possibility of CWD being introduced through urine, though our board has not taken any action, nor is it considering it at this time."]

The Vermont Fish and Wildlife Department initiated an outreach campaign to inform hunters of the risks of natural urine-based products from captive deer of unknown origins. Outreach

involved information and links posted to their website, press releases, notice in the annual regulations booklets, discussion in annual deer harvest reports, and information provided at regular public deer meetings and presentations. By monitoring internet chat-room activities and public comments during public meetings, it was evident that among hunters there was as much, or more, support for added protections against CWD than there was against it (S. Haskell, MDIFW, personal observation). There was some concern among those that sell such products. Most states, like Maine, have opted to pursue outreach campaigns about the use of natural urine-based deer attractants rather than attempting to impose strict bans on the use of these products.

At this time, the Maine Department of Inland Fisheries and Wildlife (MDIFW) concurs with the findings of other wildlife biologists and wildlife veterinarians that the risk of introducing infectious prions into the environment through the dissemination of natural urine-based lures taken from captive deer of unknown origin, that could lead to CWD in Maine's free-ranging CWD-free population of white-tailed deer, may not be quantifiable, but the existence of such risk is undeniable. Given the history of prevalence and spread of CWD among captive deer herds, it seems prudent to prohibit or minimize spread of urine from captive deer of unknown origin across the landscape. Artificial, or synthetic, scents should pose no such risk, but their origins could also be suspect. Such products, if used, should be placed where deer cannot touch them.

Deer Feed

In 1997 the federal Food and Drug Administration (FDA) passed a new rule that prohibits feeding cattle some commonly used protein feed ingredients made from certain mammalian tissues. These rules were intended to prevent the establishment and spread of bovine spongiform encephalopathy (BSE) in the U.S., commonly referred to as "mad cow disease", and to keep the BSE disease-causing agent out of both the human food supply. The rule also ensured protections for the wider order of ruminants that are animals with 4-chambered stomachs including cows, sheep, goats, bison, deer, and elk (including all cervids as described above). As part of the FDA Title 21 Part 589 "Substances Prohibited from use in Animal Food or Feed", 21CFR589.2000 "Animal Proteins Prohibited in Ruminant Feed" extended protections to prevention of the introduction of all transmissible spongiform encephalopathies (TSEs) into feed for ruminants. Thus, the TSE rule covers the infectious prion protein that causes CWD in cervids.

The TSE rule prohibits feeding protein derived from mammalian body parts, such as muscle, organs, and bones to ruminants. These proteins are commonly referred to as "prohibited materials". Some mammalian proteins are exempted and are referred to as "nonprohibited materials". These "nonprohibited materials" that may be fed to ruminants include blood and blood products, tallow, fats and oils, milk and milk proteins, and human plate waste. Proteins from poultry, fish, and vegetables are also considered "nonprohibited materials", as they should contain no prions harmful to ruminants.

Examples of prohibited materials are meat and bone meal, animal by-product meal that come from ruminant animals. A problem may occur when a labeler uses the collective term "animal protein products" on their feed tags. Both prohibited and non-prohibited proteins are included in this group of animal protein product ingredients. A feed manufacturer must be capable of

substantiating the actual protein source used if the collective term is listed as an ingredient in a particular feed.

The TSE rule applies to renderers, blenders, feed manufacturers, feeders, mixers, distributors and haulers. The following are requirements of FDA 21CFR589.2000 (excerpted from a website by the Colorado Department of Agriculture and verified in FDA 21CFR589.2000):

Renderers

- Renderers who do not separate prohibited and nonprohibited materials must label all products "Do not feed to cattle or other ruminants."
- They must maintain receipt and distribution records for one year and, upon request, provide them to FDA or state regulatory personnel.
- If a renderer does separate prohibited and nonprohibited materials they must, in addition to the requirements listed above, obtain their nonprohibited material from single species slaughter houses, prevent commingling of materials and document their procedures to prevent commingling. To prevent commingling or cross-contamination, the renderer must use separate processing equipment and storage facilities and use clean-out procedures such as sweeping, flushing, or sequencing. Written procedures used by the firm must be maintained on site. Any imported products processed by the renderer must meet the same requirements as domestic products. Export products must be clearly marked "FOR EXPORT ONLY."

Feed Manufacturers and Blenders

The same requirements as listed for renderers also apply to feed manufacturers and blenders.

A feed manufacturer or blender that produces feed or feed products that contain prohibited materials must label the end products with the caution statement "Do not feed to cattle or other ruminants." In addition they must have procedures in place to prevent any cross-contamination of a ruminant feed with a prohibited material. These procedures must be in writing and available for review by the FDA or state regulatory inspectors.

Ruminant feeds must not contain any prohibited materials, either in the feed formula or from cross-contamination.

In summary, feed manufacturers and blenders must do the following if they manufacture ruminant feeds and/or handle prohibited materials:

All non-ruminant feeds that contain prohibited materials must contain the label caution statement "Do not feed to cattle or other ruminants." In the case of bulk shipments, the statement should be prominently imprinted on the invoice, bill of lading, or the paperwork that accompanies the shipment.

- When switching from making a non-ruminant feed that contains a prohibited material to a ruminant feed a system clean out or adequate sequencing plan, or a combination of separation and clean out must be followed.

This plan must be documented and maintained on site.

- In the feed mill, any prohibited materials should be kept separate from the mixing/processing area where they may contaminate another feed. If a dog or cat is kept on the premises, their food that

may contain prohibited materials should also be kept in an area separate from the mixing/processing area.

- All transport vehicles used to deliver both ruminant and non-ruminant feeds must have a written clean out procedure that is followed. Attention should also be paid to vehicles that deliver feed ingredients to the manufacturer/blender. Have these vehicles hauled any prohibited materials in the past? If so, did the hauler clean out the vehicle before carrying any ingredients intended for use in ruminant feeds?
- Receiving records and/or records for incoming feed materials, and distribution records for products that contain prohibited materials must be maintained for one year. These records must be sufficient to track the materials throughout their receipt, processing and distribution. These records must be made available for inspection and copying and maintained for one year from the shipment date. These records should contain the following information:
 - Date of receipt or purchase and sale or delivery
 - Name and address of the seller
 - Name and address of the consignee (purchaser)
 - Identification of the product
 - Quantity

**Please note that pet food and food that is clearly labeled as intended for laboratory animals is exempt from the BSE rule requirements for listing a caution statement and record keeping unless it is sold as "damaged" or "distressed" and diverted to another use other than feeding pets or laboratory animals. In this case, it must follow all of the requirements listed above.*

If a non-ruminant feed that contains prohibited material is sold to a livestock feeder that also feeds ruminants, the seller of the product should inform the purchaser that the product must not be fed to ruminants.

Distributors

A distributor is "any person who sells, offers to sell, exchange, or barter commercial feed or to supply, furnish, or otherwise sell commercial feed to a contract feeder." Distributors include transporters of feed and feed ingredients, retail feed stores, feed warehouses, renderers, blenders, feed manufacturers.

Distributors must do the following if they handle prohibited materials or feed that contains a prohibited material:

- All products that contain or may contain a prohibited material must bear the caution statement "Do not feed to cattle or other ruminants."
- Records must be kept to track any prohibited materials or feed containing prohibited material throughout their receipt, processing and distribution. The records should contain:
 - Date of receipt or purchase and sale or delivery
 - Name and address of the seller
 - Name and address of the consignee (purchaser)
 - Identification of the product

- Quantity
- These records must be maintained for one year and made available for copying by FDA or state feed control personnel.

A retail feed store should make sure that they know which of the products they sell contain prohibited materials/bear the caution statement. Records regarding the sale of these products must be kept for a period of one year. Customers purchasing a product bearing the caution statement should be reminded that the product must not be fed to ruminants. Feeds containing prohibited material should be stored in area where the chance of cross contamination (broken bags) is minimal. *Prohibited materials are most commonly found in poultry, swine and pet foods.* Pet food labels are not required to bear the caution statement; so as a rule of thumb, try not to store ruminant feeds next to pet food. Check the poultry and swine feed labels for the caution statement. If they contain prohibited materials, store them in an area that is separate from the ruminant feeds.

It may be helpful if the store makes a list of the products containing prohibited materials and keeps this list by the register so that it can be referred to easily. If a computer sales system is used, products containing prohibited material can be "flagged" in the system for easy identification.

Ruminant Feeders

Establishments and individuals that are responsible for feeding ruminant animals must do the following to be in compliance with the FDA BSE rule:

- Maintain copies of all purchase invoices for all feeds received that contain animal protein.
- Maintain copies of labeling for all feeds containing animal protein products. If the labeling is part of an invoice, as with bulk deliveries, the invoice is the labeling.
- Make copies of invoices and labeling available for inspection and copying by the FDA or state feed control personnel.
- Maintain the records for a minimum of one year from the date of receipt of the products.

 [Much of the information provided in the section below comes from personal communication with Shannon Jordre who works as a Consumer Safety Officer in FDA’s Center for Veterinary Medicine’s Division of Compliance and manages for FDA the BSE/Ruminant Feed Inspection Compliance Program which includes coordinating work planning, inspections, training, and enforcement. Any errors of interpretation are not the responsibility of Mr. Jordre.]

The State of Maine relies on compliance checks of ruminant feed standards conducted by the federal Department of Health and Human Services’ FDA and their cooperators. The FDA contracts compliance checks with 35 states that provide about 75% of field efforts. The FDA Office of Compliance has been responsible for 7,000–9,000 compliance checks at all stages of the ruminant feed industry annually since 1997. Compliance with standards governing the production and shipment of ruminant feed tends to be excellent, with the potential stigma of being a producer that inadvertently introduces “mad cow disease” into ruminant or human feed. Results of these checks are available in tabular form on an FDA website.

Maine does not typically participate in the federal compliance program due to lack of funding. Most feed products for ruminants in Maine are grown and used locally, or the feed products are

being checked in other states from which they are being exported. This is typical for states of New England compared to states west of New England that have much larger operations. State-funded efforts in Maine focus on registration of commercial feeds and occasional investigation of complaints regarding such feeds (H. Prince, MDAFRR, Director of Division of Quality Assurance and Regulation; personal communication). Several compliance officers in other states are also deer hunters that check deer feed products at the stores that sell them, ensuring proper ingredients, storage, and labeling practices.

Materials from cervids known to be CWD-positive may not be used in any animal feed or feed ingredients, as the FDA recognizes such material as adulterated. Feed that is later found to contain a part of a cervid infected with CWD must be recalled at great expense to the industry, and some of the larger producers of feed no longer accept cervids due to financial risk. However, cervids considered to be at high risk of being CWD-positive (defined in FDA guidelines) can be rendered into feed for animals other than ruminants. A potential problem associated with such a situation and the definition of “nonprohibited materials” is that this definition includes blood and fat of a rendered deer, that if infected with CWD, could carry the infectious prions.

The question is: how often does deer blood and fat end up in deer feed as nonprohibited materials? The answer is: 1) probably very rarely; 2) if so, at very low concentrations; and 3) the likelihood that it would have infectious prions in it is also minimal. Large rendering and feed-producing plants often collect blood from slaughtered pigs and ruminants, but such facilities often do not accept the relatively small shipments of captive cervids for processing. The smaller rendering and feed-producing plants might accept relatively small shipments of cervids for processing but would often find it impractical to handle blood separately that would often need to be kept fresh during shipment to another facility. Thus, most cervid blood would likely get rendered with the carcass into feed for animals other than ruminants.

Cervids are notoriously lean and not typically desired for their fat content. The fat that is allowable as unprohibited material, that could potentially be incorporated into ruminant feed, must meet strict purity standards. These standards allow for <0.15% of insoluble impurities (such as protein from bone meal) to be present in fat that could be considered unprohibited material for use in ruminant feed. If cervid fat were ever rendered into materials later used to produce feed for deer, it would likely be diluted to a great extent by fat from other sources such as plants, cows, or pigs that would not have any potential to be harmful to deer.

Producers and buyers of deer feed often desire to have a component of >10% protein to aid in milk production of lactating female deer and antler growth of bucks during spring and summer. Ruminant protein products could be used for such purposes. Ruminant blood meal is commercially available at a cost of about \$700/ton, and ruminant meat and bone meal is available for about \$300/ton. However, most of these products appear to originate from cows, not deer, and would not be capable of introducing the infectious prions associated with CWD.

Plants such as soybeans can be more affordable (\$200/ton), can be high in protein, and are used to introduce high levels of protein into some deer feeds. Ingredients of deer feeds must be listed in plain sight, and several types of deer feed examined in stores in Maine listed that proteins were from plant materials (S. Haskell, MDIFW, personal observation). As described above, according to federal laws any feed product that contains “prohibited materials” for feeding of ruminants

must be clearly marked “Do not feed to cattle or other ruminants”. Deer feed should be stored separately from poultry, swine, and pet foods that are likely to contain prohibited materials as described by FDA 21CFR589.2000.

Based on our investigation, the MDIFW finds that the food and feed industry is checked and held in compliance with substantial effort by both federal and state agencies. By federal law, feed for ruminants such as deer containing prohibited materials for feeding of ruminants must be clearly marked “Do not feed to cattle or other ruminants”. Feed labeled as containing “animal protein” may contain prohibited materials, as described by FDA 21CFR589.2000, so should not be fed to deer. Also, *poultry, swine, and pet feed should never be fed to deer* due to relatively high likelihood of containing prohibited materials for ruminants and no labeling requirements for such products (regarding ruminants). Commercial food products for ruminants such as deer should pose little threat of introducing CWD into a CWD-free area as long as people feeding deer use FDA-approved feed for ruminants. People feeding deer should use locally-grown products or should be diligent about checking labels on bags of commercial feed, as federal laws dictate strict labeling requirements for feeds intended for ruminants.

MDIFW recommends that deer feed used in Maine contain proteins that only come from plants. Feeding of deer becomes a risk factor for spreading CWD after the disease has been introduced into an area because it congregates deer in small areas where they can more readily exchange bodily fluids. Feeding bans are common in areas where CWD is found to be newly established.

Key References Regarding CWD

- Andrievskaia, O., J. Algire, A. Balachandran, and K. Nielson. 2008. Prion protein in sheep urine. *Journal of Veterinarian Diagnostic Investigations* 20:141–146.
- Angers, R.C., H.-E. Kang, D. Napier, S. Browning, T. Seward, C. Mathiason, and others. 2010. Prion strain mutation determined by prion protein conformational compatibility and primary structure. *Science* 328:1154–1158.
- Angers, R.C., T.S. Seward, D. Napier, M. Green, E. Hoover, T. Spraker, K. O’Rourke, A. Balachandran, and G.C. Telling. 2009. Chronic wasting disease prions in elk antler velvet. *Emerging Infectious Diseases* 15:696–703.
- Baeten, L.A., B.E. Powers, J.E. Jewell, T.R. Spraker, and M.W. Miller. 2007. A natural case of chronic wasting disease in free-ranging moose (*Alces alces shirasi*). *Journal of Wildlife Diseases* 43:309-314.
- Bartelt, G., J. Pardee, and K. Thiede. 2003. Environmental impact statement on rules to eradicate chronic wasting disease in Wisconsin’s free-ranging white-tailed deer herd. Wisconsin Department of Natural Resources, Madison.
- Bishop, R.C. 2002. The economic effect in 2002 of chronic wasting disease (CWD) in Wisconsin. University of Wisconsin-Madison, Staff paper no. 450.
- Canadian Cooperative Wildlife Health Centre. 2003. A comprehensive review of the ecological and human social effects of artificial feeding and baiting of wildlife. Saskatoon, Saskatchewan. 69 pp.
- Canadian Cooperative Wildlife Health Centre. 2011. A proposal for Canada’s national chronic wasting disease control strategy. 18 pp.
- Cooke, C.M., J. Rodger, A. Smith, K. Fernie, G. Shaw, and R.A. Somerville. 2007. Fate of prions in soil: detergent extraction of PrP from soils. *Environmental Science and Technology* 41:811-817.
- Demarais, S., R.W. DeYoung, L.J. Lyon, E.S. Williams, S.J. Willimason, and G.J. Wolfe. 2002. Biological and social issues related to confinement of wild ungulates. *Wildlife Society Technical Review* 02-3.
- Dube, C., K.G. Mehren, I.K. Barker, B.L. Peart, and A. Balachandran. 2006. Retrospective investigation of chronic wasting disease of cervids at the Toronto Zoo, 1973-2003. *Canadian Veterinary Journal* 47:1185-1193.

- Edmunds, D.R., F.G. Lindzey, R.G. Grogan, W.E. Cook, T.J. Kreeger, and T.E. Cornish. 2009. Causes of mortality and vital rates for a white-tailed deer population in the CWD-enzootic area of Wyoming. (abstract) Page 40 in 3rd International Chronic Wasting Disease Symposium, July 22-24, 2009, Park City, Utah.
- Fox, K. A., J. E. Jewell, E. S. Williams, and M. W. Miller. 2006. Patterns of PrPCWD accumulation during the course of chronic wasting disease infection in orally inoculated mule deer (*Odocoileus hemionus*). *Journal of General Virology* 87:3451–3461.
- Gonzalez-Romero, D., M. A. Barria, P. Leon, R. Morales, and C. Soto. 2008. Detection of infectious prions in urine. *FEBS Letters* 582:3161–3166.
- Gross, J.E., and M.W. Miller. 2001. Chronic wasting disease in mule deer: disease dynamics and control. *Journal of Wildlife Management* 65:205-215.
- Haley, N. J., C. K. Mathiason, S. Carver, M. Zabel, G.C. Telling, and E.A. Hoover. 2011. Detection of chronic wasting disease prions in salivary, urinary, and intestinal tissues of deer: potential mechanisms of prion shedding and transmission. *Journal of Virology* 85(13):6309. DOI: 10.1128/JVI.00425-11
- Haley, N. J., D. M. Seelig, M. D. Zabel, G. C. Telling, and E. A. Hoover. 2009. Detection of CWD prions in urine and saliva of deer by transgenic mouse bioassay. *PLoS ONE* 4(3): e4848. doi:10.1371/journal.pone.0004848.
- Inslerman, R.A., J.E. Miller, D.L. Baker, R. Cumberland, P. Doerr, J.E. Kennamer, E.R. Stinson, and S.J. Williamson. 2006. Baiting and supplemental feeding of game wildlife species. *The Wildlife Society, Technical Review* 06-1.
- Johnson, C., J. Johnson, J.P. Vanderloo, D. Keane, J.M. Aiken, and D. McKenzie. 2006. Prion protein polymorphisms in white-tailed deer influence susceptibility to chronic wasting disease. *Journal of General Virology* 87:2109-2114.
- Johnson, C.J., J.A. Pedersen, R.J. Chappell, D. McKenzie, and J.M. Aiken. 2007. Oral transmissibility of prion disease is enhanced by binding to soil particles. *PLoS Pathogens* 3:874–881.
- Joly, D.O., C.A., Ribic, J.A. Langenberg, K. Beheler, C.A. Batha, B.J. Dhuey, R.E. Rolley, G. Bartelt, T.R. Van Deelen, and M.D. Samuel. 2003. Chronic wasting disease in free-ranging Wisconsin white-tailed deer. *Emerging Infectious Diseases* 9:599-601.
- Kahn, S., C. Dube, L. Bates, and A. Balachandran. 2004. Chronic wasting disease in Canada: Part I. *Canadian Veterinary Journal* 45:397-404.
- Keane, D.P., D.J. Barr, P.N. Boschler, S.M. Hall, T. Gidlewski, K.I. O'Rourke, T.R. Spraker, and M.D. Samuel. 2008. Chronic wasting disease in a Wisconsin white-tailed deer farm. *Journal of Veterinary Diagnostic Investigations* 20:698–703
- Keel, K., J.M. Crum, R.W. Gerhold, J.A. Bryan, and J.R. Fischer. 2009. Assessment of the distribution and prevalence of chronic wasting disease in Hampshire County, West Virginia (abstract). Page 90 in 3rd International Chronic Wasting Disease Symposium, July 22-24, 2009, Park City, Utah.
- Kim, T.Y., H.J. Shon, Y.S. Joo, U.K. Mun, K.S. Kang, and Y.S. Lee. 2005. Additional cases of chronic wasting disease in imported deer in Korea. *Journal of Veterinary Medical Science* 67: 753-759.
- Kong, Q.Z., S.H. Huang, W.Q. Zou et al. 2005. Chronic wasting disease of elk: transmissibility to humans examined by transgenic mouse models. *Journal of Neuroscience* 27:7944-1949.
- Li, J., S. Browning, S. P. Mahal, A.M. Oelschlegel, and C. Weismann. 2010. Darwinian evolution of prions in cell culture. *Science* 327:869–872.
- Maddison, B. C., C. A. Baker, H. C. Rees, L. A. Terry, L. Thorne, S. J. Bellworthy, G. C. Whitlam, and K. C. Gough. 2009. Prions are excreted in milk from clinically normal scrapie-exposed sheep. *Journal of Virology* 83:8293–8296. doi:10.1128/JVI.00051-09.
- Mathiason, C. K., S. A. Hays, J. Powers, J. Hayes-Klug, J. Langenberg, S. J. Dahmes, D. A. Osborn, K. V. Miller, R. J. Warren, G. L. Mason, and E. A. Hoover. 2009. Infectious prions in pre-clinical deer and transmission of chronic wasting disease solely by environmental exposure. *PLoS ONE* 4(6): e5916. doi:10.1371/journal.pone.0005916.
- Mathiason, C. K., J. G. Powers, and S. J. Dahmes, D. A Osborn, K. V. Miller, R. J. Warren, G. L.Mason, S. A. Hays, J. Hayes-Klug, D. M. Seeling, M. A. Wild, L. L. Wolfe, T. R. Spraker, M.W. Miller, C. J. Sigurdson, G. C. Telling, and E. A. Hoover. 2006. Infectious prions in the saliva and blood of deer with chronic wasting disease. *Science* 314:133–136.
- Miller, M. W., N. T. Hobbs, and S. J. Tavener. 2006. Dynamics of prion disease transmission in mule deer. *Ecological Applications* 16: 2208–2214.

- Miller, M.W., H. M. Swanson, L.L. Wolfe, F.G. Quartarone, S.L. Huwer, C.H. Southwick, and P.M. Lukacs. 2008. Lions and prions and deer demise. *PLoS ONE* 3(12): e4019. doi:10.1371/journal.pone.0004019
- Miller, M.W., and E.S. Williams. 2003. Horizontal prion transmission in mule deer. *Nature* 425:35–36.
- Miller, M.W., E.S. Williams, N.T. Hobbs, and L.L. Wolfe. 2004. Environmental sources of prion transmission in mule deer. *Emerging Infectious Diseases* 6:1003–1006.
- Murray, D.L., E.W. Cox, W.B. Ballard, H.A. Whitlaw, M.S. Lenarz, T.W. Custer, T. Barnett, and T.K. Fuller. 2006. Pathogens, nutritional deficiency, and climate influences on a declining moose population. *Wildlife Monographs* No. 166.
- Nichols, T.A., B. Pulford, A.C. Wyckoff, C. Meyerett, B. Michel, K. Gertig, E. A. Hoover, J.E. Jewell, G.C. Telling, and M.D. Zabel. 2009. Detection of protease-resistant cervid prion protein in water from a CWD-endemic area. *Prion* 3:3, 171-183.
- Northeast Association of Fish and Wildlife Agencies (NAFWA). 2006. Chronic wasting disease plan. 29 pp.
- O'Brien, D., P. Bernardi, S. Dubay, S. Mayhew, W. Moritz, and D. Puro. 2005. A risk-based audit of the captive/privately-owned cervid industry in Michigan. Michigan Department of Natural Resources Report Series Issue Report No. 1. 168 pp.
- Pybus, M. and Y.T. Hwang, editors. 2008. Chronic wasting disease workshop: response plans. Alberta Sustainable Resource Development, Fish and Wildlife Division and Saskatchewan Ministry of Environment, Fish and Wildlife Branch. 47 pp.
- Race, B., K. Meade-White, R. Race, and B. Chesebro. 2009. Prion infectivity in fat of deer with chronic wasting disease. *Journal of Virology* 83:9608–9610. doi:10.1128/JVI.01127-09.
- Safar, J. G., P. Lessard, G. Tamguney, Y. Freyman, C. Deering, F. Letessier, S. J. DeArmond, and S. B. Prusiner. 2008. Transmission and detection of prions in feces. *Journal of Infectious Diseases* 198:81–89.
- Seeger, H., M. Heikenwalder, N. Zeller, J. Kranich, P. Schwarz, A. Gaspert, B. Seifert, G. Miele, and A. Aguzzi. 2005. Coincident scrapie infection and nephritis lead to urinary prion excretion. *Science* 310:324–326.
- Sigurdson, C.J., and A. Aguzzi. 2007. Chronic wasting disease. *Molecular Basis of Disease* 1772:610-618.
- Sohn, H.J., J.H. Kim, K.S. Choi, J.J. Nah, Y.S. Joo, Y.H. Jean, S.W. Ahn, O.K. Kim, D.Y. Kim, and A. Balachandran. 2002. A case of chronic wasting disease in an elk imported to Korea from Canada. *Journal of Veterinary Medical Science* 64:855-858.
- Tamguney, G., M.W. Miller, L.L. Wolfe, T.M. Sirochman, D.V. Glidden, C. Plamer, A. Lemus, S.J. DeArmond, and S.B. Prusiner. 2009. Asymptomatic deer excrete infectious prions in faeces. *Nature*, doi:10.1038/nature08289.
- VanDeelen, T.R. 2003. Chronic wasting disease and the science in support of the ban on baiting and feeding deer. 7 pp.
- VerCauteren, K.C., M.J. Lavelle, G.E. Phillips, J.W. Fischer, and R.S. Stahl. 2009. Elk and deer use of mineral licks: implications for disease transmission (abstract). Page 114 *in* 3rd International Chronic Wasting Disease Symposium, July 22-24, 2009, Park City, Utah.
- Vercauteren, K.C., M.J. Lavelle, N.W. Seward, J.W. Fischer, and G.F. Phillips. 2007. Fence-line contact between wild and farmed white-tailed deer in Michigan: potential for disease transmission. *Journal of Wildlife Management* 71:1603-1606.
- Walsh, D.P., and M.W. Miller. 2010. A weighted surveillance approach for detecting chronic wasting disease foci. *Journal of Wildlife Diseases* 46:118–135.
- Williams, E. S. 2005. Chronic wasting disease. *Veterinary Pathology* 42:530–549.
- Williams, E.S., M.W. Miller, T.J. Kreeger, R.H. Kahn, and E.T. Thorne. 2002. Chronic wasting disease of deer and elk: a review with recommendations for management. *Journal of Wildlife Management* 66:551–563.
- Williams, E.S., and S. Young. 1980. Chronic wasting disease of captive mule deer: a spongiform encephalopathy. *Journal of Wildlife Diseases* 16:89–98.
- Wolfe, L.L., W.M. Miller, and E.S. Williams. 2004. Feasibility of “test-and-cull” for managing chronic wasting disease in urban mule deer. *Wildlife Society Bulletin* 32:500-505.