Comments, DNR <dnrcomments@wv.gov>

Re: Deer baiting and feeding

1 message

Comments, DNR <dnrcomments@wv.gov> Mon, Jul 27, 2026 at 10:45 AM
To: James Britton

Good Morning Mr. Britton,

The West Virginia Division of Natural Resources would like to thank you for submitting your public comments for the proposed rule changes to 58CSR69 – Wildlife Disease Management.

Your comment submitted in support of a full ban on baiting in the state will be recorded for public record. I do want to note; this rule does not institute a full ban on baiting across the state. This rule is limited to the regulation of baiting and feeding in CWD containment areas.

On Wed, Jul 1, 2026 at 8:20 PM James Britton

> I support a full ban of deer feeding in WV. Countless studies have shown feeding creates unnatural congregation of deer which promotes disease spreading. It is time WV act before CWD finds its way into every county and it is then too late.

Comments, DNR <dnrcomments@wv.gov>

Re: Please do not adopt these restrictions.

1 message

Comments, DNR <dnrcomments@wv.gov> Mon, Jul 27, 2026 at 10:51 AM
To: alfie[redacted]

Good Morning,

The West Virginia Division of Natural Resources would like to thank you for submitting your public comment for the proposed rule changes to 58CSR69 – Wildlife Disease Management. Your comment submitted in opposition of the rule changes will be recorded for public record.

On Thu, Jul 2, 2026 at 8:49 AM a[redacted]

Sent from my Galaxy

Comments, DNR <dnrcomments@wv.gov>

Re: Chronic Wasting Disease CWD TSE PrP Singeltary Submission WV DNR

1 message

Comments, DNR <dnr[redacted]> Mon, Jul 27, 2026 at 11:25 AM
To: TERRY SINGELTA[redacted]
Cc: dnr.wildlife@wv.go[redacted]ov

Good Morning Mr. Singletary,

The West Virginia Division of Natural Resources would like to thank you for submitting comments to the agency. It appears your comments are related to a USDA Aphis rule and not the WV Division of Natural Resources 58CSR69 – Wildlife Disease Management rule. I have, however, shared your email with our Wildlife Disease Specialist and it will be recorded for public record.

On Thu, Jul 2, 2026 at 9:30 AM TERRY SINGELTAR[redacted] wrote:

> Chronic Wasting Disease CWD TSE PrP Singeltary Submission WV DNR
>
> Control of Chronic Wasting Disease OMB Control Number: 0579-0189 APHIS-2021-0004 Singeltary Submission
>
> Greetings APHIS et al, i would kindly like to comment on Control of Chronic Wasting Disease OMB Control Number: 0579-0189 APHIS-2021-0004.
>
> Greetings APHIS et al, i would kindly like to comment on Control of Chronic Wasting Disease OMB Control Number: 0579-0189 APHIS-2021-0004.
>
> ***> 1st and foremost your biggest problem is 'VOLUNTARY'! AS with the BSE 589.2001 FEED REGULATIONS, especially since it is still voluntary with cervid, knowing full well that cwd and scrapie will transmit to pigs by oral route. VOLUNTARY DOES NOT WORK! all animal products should be banned and be made mandatory, and the herd certification program should be mandatory, or you don't move cervid. IF THE CWD HERD CERTIFICATION IS NOT MANDATORY, it will be another colossal tse prion failure from the start.
>
> ***> 2nd USA should declare a Declaration of Extraordinary Emergency due to CWD, and all exports of cervid and cervid products must be stopped internationally, and there should be a ban of interstate movement of cervid, until a live cwd test is available.
>
> ***> 3rd Captive Farmed cervid ESCAPEES should be made mandatory to report immediately, and strict regulations for those suspect cwd deer that just happen to disappear. IF a cervid escapes and is not found, that farm should be indefinitely shut down, all movement, until aid MIA cervid is found, and if not ever found, that farm shut down permanently.
>
> ***> 4th Captive Farmed Cervid, INDEMNITY, NO MORE Federal indemnity program, or what i call, ENTITLEMENT PROGRAM for game farm industry. NO MORE BAIL OUTS FROM TAX PAYERS. if the captive industry can't buy insurance to protect not only themselves, but also their customers, and especially the STATE, from Chronic Wasting Disease CWD TSE Prion or what some call mad deer disease and harm therefrom, IF they can't afford to buy that insurance that will cover all of it, then they DO NOT GET A PERMIT to have a game farm for anything. This CWD TSE Prion can/could/has caused property values to fall from some reports in some places. roll the dice, how much is a state willing to lose?
>
> ***> 5th QUARANTINE OF ALL FARMED CAPTIVE, BREEDERS, URINE, ANTLER, VELVET, SPERM, OR ANY FACILITY, AND THEIR PRODUCTS, that has been confirmed to have Chronic Wasting Disease CWD TSE Prion, the QUARANTINE should be for 21 years due to science showing what scrapie can do. 5 years is NOT near long enough. see; Infectious agent of sheep scrapie may persist in the environment for at least 16 to 21 years.
>
> ***> 6th America BSE 589.2001 FEED REGULATIONS CWD TSE Prion
>
> ***> 7TH TRUCKING TRANSPORTING CERVID CHRONIC WASTING DISEASE TSE PRION VIOLATING THE LACEY ACT

***> 8TH ALL CAPTIVE FARMING CERVID OPERATIONS MUST BE INSURED TO PAY FOR ANY CLEAN UP OF CWD AND QUARANTINE THERE FROM FOR THE STATE, NO MORE ENTITLEMENT PROGRAM FOR CERVID GAME FARMING PAY TO PLAY FOR CWD TSE PRION OFF THE TAX PAYERS BACK.

***> 9TH ANY STATE WITH DOCUMENTED CWD, INTERSTATE, NATIONAL, AND INTERNATIONAL MOVEMENT OF ALL CERVID, AND ALL CERVID PRODUCTS MUST BE HALTED!

***> 10TH BAN THE SALE OF STRAW BRED BUCKS AND ALL CERVID SEMEN AND URINE PRODUCTS

***> 11th ALL CAPTIVE FARMED CERVID AND THEIR PRODUCTS MUST BE CWD TSE PRION TESTED ANNUALLY AND BEFORE SALE FOR CWD TSE PRION

SEE FULL SCIENCE REFERENCES AND REASONINGS ;

snip…see full text;

Control of Chronic Wasting Disease OMB Control Number: 0579-0189APHIS-2021-0004 Singeltary Submission

https://www.regulations.gov/comment/APHIS-2021-0004-0002

https://downloads.regulations.gov/APHIS-2021-0004-0002/attachment_1.pdf

Docket No. APHIS-2018-0011 Chronic Wasting Disease Herd Certification

https://www.regulations.gov/document/APHIS-2018-0011-0003

https://downloads.regulations.gov/APHIS-2018-0011-0003/attachment_1.pdf

APHIS Indemnity Regulations [Docket No. APHIS-2021-0010] RIN 0579-AE65 Singeltary Comment Submission

Comment from Singeltary Sr., Terry

Posted by the Animal and Plant Health Inspection Service on Sep 8, 2022

https://www.regulations.gov/comment/APHIS-2021-0010-0003

https://downloads.regulations.gov/APHIS-2021-0010-0003/attachment_1.pdf

Docket No. FDA-2003-D-0432 (formerly 03D-0186) Use of Material from Deer and Elk in Animal Feed

PUBLIC SUBMISSION

Comment from Terry Singeltary Sr.

Posted by the Food and Drug Administration on May 17, 2016 Comment

Docket No. FDA-2003-D-0432 (formerly 03D-0186) Use of Material from Deer and Elk in Animal Feed Singeltary Submission

https://www.regulations.gov/comment/FDA-2003-D-0432-0011

https://www.regulations.gov/docket/FDA-2003-D-0432

FRIDAY, JUNE 26, 2026

Historical and contemporary chronic wasting disease prions from the western United States demonstrate similar strain properties

(see Texas updated cwd cases, May, June 2026…terry)

https://chronic-wasting-disease.blogspot.com/2026/06/historical-and-contemporary-chronic.html

https://prpsc.proboards.com/thread/222/cwd-western-demonstrate-similar-strain

***> CWD TSE PrP Environmental Transmission

So, this is what we leave our children and grandchildren?

***> 2026 CHRONIC WASTING DISEASE CWD TSE PRION

***> 2026 ENVIRONMENTAL FACTORS FOR CHRONIC WASTING DISEASE CWD TSE Prion

Title: Horizontal transmission of chronic wasting disease in reindeer

Submitted to: Emerging Infectious Diseases Publication Type: Peer Reviewed Journal Publication Acceptance Date: 8/29/2016 Publication Date: 12/1/2016

Interpretive Summary: Chronic wasting disease (CWD) is a fatal neurodegenerative disease that occurs in farmed and wild cervids (deer and elk) of North America and was recently diagnosed in a single free-ranging reindeer (Rangifer tarandus tarandus) in Norway. CWD is a transmissible spongiform encephalopathy (TSE) that is caused by infectious proteins called prions that are resistant to various methods of decontamination and environmental degradation. Little is known about the susceptibility of or potential for transmission amongst reindeer. In this experiment, we tested the susceptibility of reindeer to CWD from various sources (elk, mule deer, or white-tailed deer) after intracranial inoculation and tested the potential for infected reindeer to transmit to non-inoculated animals by co-housing or housing in adjacent pens. Reindeer were susceptible to CWD from elk, mule deer, or white-tailed deer sources after experimental inoculation. Most importantly, non-inoculated reindeer that were co-housed with infected reindeer or housed in pens adjacent to infected reindeer but without the potential for nose-to-nose contact also developed evidence of CWD infection. This is a major new finding that may have a great impact on the recently diagnosed case of CWD in the only remaining free-ranging reindeer population in Europe as our findings imply that horizontal transmission to other reindeer within that herd has already occurred. Further, this information will help regulatory and wildlife officials developing plans to reduce or eliminate CWD and cervid farmers that want to ensure that their herd remains CWD-free, but were previously unsure of the potential for reindeer to transmit CWD.

https://www.ars.usda.gov/research/publications/publication/?seqNo115=328261

https://wwwnc.cdc.gov/eid/article/22/12/16-0635_article

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5189146/

Chronic wasting disease (CWD) prion detection in environmental and biological samples from a taxidermy site and nursing facility, and instruments used in surveillance activities

Available online 9 April 2025

Highlights

• CWD prions were identified in a taxidermy and deer nursing facility.

• Contaminated samples included waters, soils, dermestid beetles, domestic flies and a dumpster.

• Surgical instruments used to collect deer samples can get contaminated with CWD prions.

• Some of the infectious particles are readily released from surgical instruments when washed.

• Our results suggest that taxidermy practices actively contribute in the spreading of CWD.

Snip…

In summary, the information provided in this report demonstrate how anthropogenic activities, specifically taxidermy practices, animal processing, and rehabilitation of CWD susceptible species, may facilitate CWD transmission through the environmental dissemination of CWD prions. This study, along with future research efforts characterizing the overall level of infectivity, provides relevant information on managing CWD and to control its rapid geographic expansion. …

https://www.sciencedirect.com/science/article/abs/pii/S0048969725009544

Chronic wasting disease detection in environmental and biological samples from a taxidermy site

Results: The PMCA analysis demonstrated CWD seeding activity in some of the components of this facility, including insects involved in head processing, soils, and a trash dumpster.

Conclusions: Different areas of this property were used for various taxidermy procedures. We were able to detect the presence of prions in

i) soils that were in contact with the heads of dead animals, ii) insects involved in the cleaning of skulls, and iii) an empty dumpster where animal carcasses were previously placed.

This is the first report demonstrating that swabbing is a helpful method to screen for prion infectivity on surfaces potentially contaminated with CWD. These findings are relevant as this swabbing and amplification strategy may be used to evaluate the disease status of other free-ranging and captive settings where there is a concern for CWD transmissions, such as at feeders and water troughs with CWD-exposed properties. This approach could have substantial implications for free-ranging cervid surveillance as well as in epidemiological investigations of CWD.

Prion 2022 Conference abstracts: pushing the boundaries

https://www.tandfonline.com/doi/full/10.1080/19336896.2022.2091286

Artificial mineral sites that pre-date endemic chronic wasting disease become prion hotspots

The Ames Research and Educational Center property, centrally located within the CWD zone of southwest Tennessee, contains 49 historical mineral supplementation sites that were decommissioned in 2012. Here, we demonstrate that 32 of the 49 (65%) mineral sites within Ames established prior to the regional CWD outbreak, serve as foci of environmental PrPCWD contamination. Detection of PrPCWD in soils from these artificial mineral sites was dependent on site-specific management efforts. Soil physical properties were very similar across sites and no correlation between PrPCWD detection and soil physical properties was found. The detection of PrPCWD in soils at attractant sites within an endemic CWD zone significantly advances our understanding of environmental PrPCWD accumulation dynamics, providing valuable information for advancing adaptive CWD management approaches.

https://intcwdsympo.files.wordpress.com/2023/06/final-agenda-with-abstracts.pdf

Shedding of Chronic Wasting Disease Prions in Multiple Excreta Throughout Disease Course in White-tailed Deer

Conclusions: These studies demonstrate: (a) CWD prion excretion occurs throughout infection; (2) PRNP genotype (GG≫GS/NT) influences the excreta shedding; and (3) detection sensitivity in excreta can vary with different RT-QuIC protocols. These results provide a more complete perspective of prion shedding in deer during the course of CWD infection.

Prion 2022 Conference abstracts: pushing the boundaries

https://www.tandfonline.com/doi/full/10.1080/19336896.2022.2091286

"Additionally, we have determined that prion seeding activity is retained for at least fifteen years at a contaminated site following attempted remediation."

***> 15 YEARS!!!

Detection of prions in soils contaminated by multiple routes

Results: We are able to detect prion seeding activity at multiple types of environmental hotspots, including carcass sites, contaminated captive facilities, and scrapes (i.e. urine and saliva). Differences in relative prion concentration vary depending on the nature and source of the contamination. Additionally, we have determined that prion seeding activity is retained for at least fifteen years at a contaminated site following attempted remediation.

Conclusions: Detection of prions in the environment is of the utmost importance for controlling chronic wasting disease spread. Here, we have demonstrated a viable method for detection of prions in complex environmental matrices. However, it is quite likely that this method underestimates the total infectious prion load in a contaminated sample, due to incomplete recovery of infectious prions. Further refinements are necessary for accurate quantification of prions in such samples, and to account for the intrinsic heterogeneities found in the broader environment.

Funded by: Wisconsin Department of Natural Resources

Meeting-book-final-version prion 2023 Prion 2023 Congress Organizing Committee and the NeuroPrion Association, we invite you to join us for the International Conference Prion2023 from 16-20 October 2023 in Faro, Portugal.

https://prion2023.org/wp-content/uploads/2023/10/Meeting-book-final-version2.pdf

https://web.archive.org/web/20250828201533/https://prion2023.org/wp-content/uploads/2023/10/Meeting-book-final-version2.pdf

https://www.researchgate.net/profile/Syed-Zahid-Shah/publication/378314391_Meeting-book-final-version_prion_2023/links/65d44dad28b7720cecdca95f/Meeting-book-final-version-prion-2023.pdf

***> Infectious agent of sheep scrapie may persist in the environment for at least 16 years

***> Nine of these recurrences occurred 14–21 years after culling, apparently as the result of environmental contamination, but outside entry could not always be absolutely excluded.

JOURNAL OF GENERAL VIROLOGY Volume 87, Issue 12

Infectious agent of sheep scrapie may persist in the environment for at least 16 years Free

https://www.microbiologyresearch.org/content/journal/jgv/10.1099/vir.0.82011-0

Rapid recontamination of a farm building occurs after attempted prion removal

First published: 19 January 2019 https://doi.org/10.1136/vr.105054

The data illustrates the difficulty in decontaminating farm buildings from scrapie, and demonstrates the likely contribution of farm dust to the recontamination of these environments to levels that are capable of causing disease. snip...

This study clearly demonstrates the difficulty in removing scrapie infectivity from the farm environment. Practical and effective prion decontamination methods are still urgently required for decontamination of scrapie infectivity from farms that have had cases of scrapie and this is particularly relevant for scrapie positive goatherds, which currently have limited genetic resistance to scrapie within commercial breeds.24 This is very likely to have parallels with control efforts for CWD in cervids.

https://bvajournals.onlinelibrary.wiley.com/doi/abs/10.1136/vr.105054

***>This is very likely to have parallels with control efforts for CWD in cervids.

https://pubmed.ncbi.nlm.nih.gov/30602491/

Characterizing Hydrological Transport Pathways of Chronic Wasting Disease in the Environment

Authors Anu Wille, Diana L Karwan, Stuart Siegfried Lichtenberg, Madeline Grunklee, Gage Rowden, Victoria Ferguson-Kramer, Marc D Schwabenlander, Tiffany M Wolf, Peter A Larsen Publication date 2024/6 Journal Water Science Conference (WaterSciCon24 Pages 110-04

Description Chronic wasting disease (CWD) is a fatal neurodegenerative prion disease found in deer, moose, and elk. Cases of CWD in Minnesota have risen considerably over the last few years, raising wildlife, environmental, and public health concerns.

Infectious prions, such as those causing CWD, enter the environment through bodily fluids or decomposing carcasses of infected individuals and can persist for at least fifteen years in soil and water.

Previous studies have shown strong prion sorption to various mineral particles in soils.

Through field observation and laboratory experimentation, we observed that prions readily partition to the particulate fraction of environmental waters, suggesting that hydrological transport of prions is likely sediment-facilitated.

To effectively contain the spread of CWD through the environment, it is imperative to predict prion transport times and pathways in the context of specific landscape and …

Scholar articles Characterizing Hydrological Transport Pathways of Chronic Wasting Disease in the Environment

A Wille, DL Karwan, SS Lichtenberg, M Grunklee… - Water Science Conference (WaterSciCon24, 2024 Related articles

https://scholar.google.com/citations?view_op=view_citation&hl=en&user=cVqWACIAAAAJ&cstart=20&

pagesize=80&citation_for_view=cVqWACIAAAAJ:0EnyYjriUFMC

Characterizing Hydrological Transport Pathways of Chronic Wasting Disease in the Environment

Wille, Anu search by orcid.svg ; Karwan, Diana L. search by orcid.svg ; Lichtenberg, Stuart Siegfried ;Grunklee, Madeline ; Rowden, Gage ; Ferguson-Kramer, Victoria ;Schwabenlander, Marc D. ; Wolf, Tiffany M. ; Larsen, Peter A.

https://ui.adsabs.harvard.edu/abs/2024wsc..conf11004W/abstract

https://agu.confex.com/agu/hydrology24/meetingapp.cgi/Paper/1502243

Characterizing Hydrological Transport Pathways of Chronic Wasting Disease in the Environment

Wille, Anu search by orcid ; Karwan, Diana L. search by orcid ; Lichtenberg, Stuart Siegfried ; Grunklee, Madeline ; Rowden, Gage ; Ferguson-Kramer, Victoria ; Schwabenlander, Marc D. ; Wolf, Tiffany M. ; Larsen, Peter A.

Abstract Chronic wasting disease (CWD) is a fatal neurodegenerative prion disease found in deer, moose, and elk. Cases of CWD in Minnesota have risen considerably over the last few years, raising wildlife, environmental, and public health concerns. Infectious prions, such as those causing CWD, enter the environment through bodily fluids or decomposing carcasses of infected individuals and can persist for at least fifteen years in soil and water. Previous studies have shown strong prion sorption to various mineral particles in soils. Through field observation and laboratory experimentation, we observed that prions readily partition to the particulate fraction of environmental waters, suggesting that hydrological transport of prions is likely sediment-facilitated. To effectively contain the spread of CWD through the environment, it is imperative to predict prion transport times and pathways in the context of specific landscape and watershed conditions. Our purpose is to characterize the hydrological transport of prions through watersheds at multiple scales. Through spatial analysis, we mapped surface flow pathways from CWD hotspots in Minnesota to identify how they overlap with major rivers and regions of high soil erosion. Based on in-stream measurements and sediment characterization, we used empirical equations to predict sediment mobilization conditions and transport rates in CWD-contaminated regions with diverse flow regimes. In order to model subsurface prion transport, we began conducting flow-through column experiments testing various flow rates and soil matrices.

Publication: Water Science Conference (WaterSciCon24), held in St. Paul, Minnesota, 24-27 June 2024, Session: Learning from observations / Catchment and Critical Zone Science - Understanding Ecosystems through Monitoring, Analysis, and Experimentation III Oral (Coupled Workshop: 231), id. 110-04. Pub Date: June 2024

https://ui.adsabs.harvard.edu/abs/2024wsc..conf11004W/abstract

I remember what “deep throat” told me about Scrapie back around 2001, during early days of my BSE investigation, after my Mom died from hvCJD, I never forgot, and it seems it’s come to pass;

***> Confidential!!!!

***> As early as 1992-3 there had been long studies conducted on small pastures containing scrapie infected sheep at the sheep research station associated with the Neuropathogenesis Unit in Edinburgh, Scotland. Whether these are documented...I don't know. But personal recounts both heard and recorded in a daily journal indicate that leaving the pastures free and replacing the topsoil completely at least 2 feet of thickness each year for SEVEN years....and then when very clean (proven scrapie free) sheep were placed on these small pastures.... the new sheep also broke out with scrapie and passed it to offspring. I am not sure that TSE contaminated ground could ever be free of the agent!! A very frightening revelation!!!

---end personal email---end...tss

and so it seems…

Chronic Wasting Disease CWD TSE Prion

THE CWD TSE Prion aka mad cow type disease is not your normal pathogen.

The TSE prion disease survives ashing to 600 degrees celsius, that’s around 1112 degrees farenheit.

You cannot cook the TSE prion disease out of meat. In fact new data now shows that exposure to high temperatures used to cook the meat increased the availability of prions for in vitro amplification.

you can take the ash and mix it with saline and inject that ash into a mouse, and the mouse will go down with TSE.

Prion Infected Meat-and-Bone Meal Is Still Infectious after Biodiesel Production as well.

the TSE prion agent also survives Simulated Wastewater Treatment Processes.

IN fact, you should also know that the TSE Prion agent will survive in the environment for years, if not decades.

you can bury it and it will not go away.

The TSE agent is capable of infected your water table i.e. Detection of protease-resistant cervid prion protein in water from a CWD-endemic area.

it's not your ordinary pathogen you can just cook it out and be done

New studies on the heat resistance of hamster-adapted scrapie agent: Threshold survival after ashing at 600°C suggests an inorganic template of replication

http://www.pnas.org/content/97/7/3418.full

Prion Infected Meat-and-Bone Meal Is Still Infectious after Biodiesel Production

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2493038/

Rapid assessment of bovine spongiform encephalopathy prion inactivation by heat treatment in yellow grease produced in the industrial manufacturing process of meat and bone meals

https://bmcvetres.biomedcentral.com/track/pdf/10.1186/1746-6148-9-134.pdf

THURSDAY, FEBRUARY 28, 2019

BSE infectivity survives burial for five years with only limited spread

https://link.springer.com/content/pdf/10.1007%2Fs00705-019-04154-8.pdf

Chronic wasting disease prions on deer feeders and wildlife visitation to deer feeding areas

First published: 10 February 2025

Snip…

Finally, we swabbed 19 feeders in 2 areas where CWD was newly detected, finding prion contamination on swabs from 4 feeders. We show that deer feeders in free-ranging populations with high CWD prevalence become contaminated with CWD prions quickly, becoming a potential site of exposure of deer to CWD prions. Our results also demonstrate the ability to find evidence of prion contamination on deer feeders, even in areas where CWD is newly detected.

Snip…

We found that supplemental feeding increased the risk of exposure to CWD prions due to contamination of feeders, increased deer visitation, and increased deer-to-deer contact.

The 12-fold increase in deer visitation to feeders compared to mast trees and 2-fold increase compared to food plots demonstrates increased risk for direct disease spread.

https://wildlife.onlinelibrary.wiley.com/doi/10.1002/jwmg.70000

***> CWD Transmission To, Cattle, Pigs, Sheep, Primates, oh my!

Transmission of the chronic wasting disease agent from elk to cattle after oronasal exposure

Justin Greenlee, Jifeng Bian, Zoe Lambert, Alexis Frese, and Eric Cassmann Virus and Prion Research Unit, National Animal Disease Center, USDA-ARS, Ames, IA, USA

Aims: The purpose of this study was to determine the susceptibility of cattle to chronic wasting disease agent from elk.

Materials and Methods: Initial studies were conducted in bovinized mice using inoculum derived from elk with various genotypes at codon 132 (MM, LM, LL). Based upon attack rates, inoculum (10% w/v brain homogenate) from an LM132

elk was selected for transmission studies in cattle. At approximately 2 weeks of age, one wild type steer (EE211) and one steer with the E211K polymorphism (EK211) were fed 1 mL of brain homogenate in a quart of milk replacer while another 1 mL was instilled intranasally. The cattle were examined daily for clinical signs for the duration of the experiment. One steer is still under observation at 71 months post-inoculation (mpi).

Results: Inoculum derived from MM132 elk resulted in similar attack rates and incubation periods in mice expressing wild type or K211 bovine PRNP, 35% at 531 days post inoculation (dpi) and 27% at 448 dpi, respectively. Inoculum from LM132 elk had a slightly higher attack rates in mice: 45% (693 dpi) in wild type cattle PRNP and 33% (468) in K211 mice. Inoculum from LL132 elk resulted in the highest attack rate in wild type bovinized mice (53% at 625 dpi), but no K211 mice were affected at >700 days. At approximately 70 mpi, the EK211 genotype steer developed clinical signs suggestive of prion disease, depression, low head carriage, hypersalivation, and ataxia, and was necropsied. Enzyme immunoassay (IDEXX) was positive in brainstem (OD=4.00, but non-detect in retropharyngeal lymph nodes and palatine tonsil. Immunoreactivity was largely limited to the brainstem, midbrain, and cervical spinal cord with a pattern that was primarily glia-associated.

Conclusions: Cattle with the E211K polymorphism are susceptible to the CWD agent after oronasal exposure of 0.2 g of infectious material.

https://prion2023.org/wp-content/uploads/2023/10/Meeting-book-final-version2.pdf

https://web.archive.org/web/20250828201533/https://prion2023.org/wp-content/uploads/2023/10/Meeting-book-final-version2.pdf

https://www.researchgate.net/profile/Syed-Zahid-Shah/publication/378314391_Meeting-book-final-version_prion_2023/links/65d44dad28b7720cecdca95f/Meeting-book-final-version-prion-2023.pdf

***> cwd to pigs

Chronic wasting disease prions in cervids and wild pigs in North America Preliminary Outbreak Assessment DEFRA 26 January 2026

DEFRA 26 January 2026 Department for Environment, Food and Rural Affairs

Preliminary Outbreak Assessment

Chronic wasting disease prions in cervids and wild pigs in North America

26 January 2026

Disease report

Chronic wasting disease (CWD) is a fatal neurodegenerative disease of cervids, such as deer, elk, moose and reindeer. It is caused by prions – infectious proteins that cause normal cellular prion proteins to misfold (CIDRAP, 2025). The disease is widespread in captive and free-ranging cervids in North America (Figure 1). For the first time, CWD prions have also been detected in the tissues of wild pigs (Sus scrofa) caught in CWD-affected areas of the USA (Soto et al. 2025). This discovery emerged from a study designed to investigate potential interactions between wild pigs and CWD prions, as wild pigs often coexist with cervids, which can shed prions into the environment. The following assessment discusses the epidemiology of CWD in North America and the detection of CWD prions in wild pigs. It also considers the potential implications for Great Britain.

Figure 1. Distribution of CWD in cervids in North America as of 11 April 2025 (USGS, 2025).Department for Environment, Food and Rural Affairs

Situation assessment

CWD is considered one of the most important cervid diseases due to its capacity for infectious spread, high mortality rate and associated socio-economic impacts on cervid farming and hunting-related industries (Kincheloe et al., 2021, CFSPH, 2024). The disease is always fatal, with no cure or vaccine (CFSPH, 2024).

CWD was first reported among captive cervids in the USA in the 1960s (Kincheloe et al., 2021). It has since been detected in captive and or free-ranging cervids in 36 US states and 5 Canadian provinces, as well as South Korea, Norway, Finland and Sweden (Silva, 2022, USGS, 2025). While the South Korean strains are thought to have originated from North America, the European strains appear to have emerged independently (Silva, 2022).

Transmission between cervids occurs by direct contact with infected animals or indirectly, through contact with a

contaminated environment, most likely via the oral route (Otero et al., 2021). The disease may also be vertically transmitted from doe to fawn (Nalls et al., 2013, Salariu et al., 2015). Environmental contamination occurs when infected animals shed infectious prions in various secretions and excretions, such as urine, faeces and saliva (Otero et al., 2021). It can also occur when infected carcasses decompose and release prions into the surrounding soil and vegetation (Miller et al., 2004). The minimum number of CWD prions required to cause infection in cervids is unknown but appears to be low (Denkers et al., 2020).

The disease is difficult to control, as infected animals can also be subclinical for months or years. During this time, they can shed CWD prions, which can remain infectious in the environment for at least 2 years (Miller et al., 2004, CFSPH, 2024). Diagnosis usually relies on post-mortem tests, which may fail to identify infected animals during the early stages of the disease (CFSPH, 2024, CIDRAP, 2025). Control efforts are further hampered by lack of evidence to inform effective CWD management and control strategies (Uehlinger et al., 2016, Mori et al., 2024).

CWD in North American cervids

CWD has been reported in a range of North American cervids, including white-tailed deer, mule deer, black-tailed deer, moose, wapiti, reindeer (captive) and red deer (captive) (EFSA BIOHAZ Panel, 2023). It was first reported in captive mule deer and black-tailed deer at research facilities in Colorado and Wyoming in the late 1960s (Otero et al., 2021). These animals were derived from wild populations. The disease was later identified in Rocky Mountain elk at these facilities and subsequently, in free-ranging populations of mule deer and elk in Wyoming and Colorado. The geographic expansion of CWD in North America is thought to reflect the commercial movement of subclinical animals and natural cervid migration (Otero et al., 2021). Epidemiological data suggest that the disease spread from the USA to Canada and then to South Korea through imports of infected cervids (Otero et al., 2021). A retrospective analysis revealed that, in 1978, a Colorado-born mule deer at Toronto Zoo in Ontario, Canada, died of CWD (Dubé et al., 2006). In 1996, the disease was detected in captive elk in Saskatchewan (Williams and Miller, 2002).

The disease

Department for Environment, Food and Rural Affairs

has since been detected in captive cervids in Alberta and Quebec and free-ranging cervids in Alberta, British Columbia, Manitoba and Saskatchewan (USGS, 2025). The origin of the outbreak in free-ranging Canadian cervids is unknown (Otero et al., 2021).

While the spread of CWD across North America is often described as 'rapid,' it has been suggested that this may reflect widening disease surveillance, rather than a 'real-time' indication of geographic spread. CWD epidemics appear to develop relatively slowly compared with other wildlife diseases (EFSA BIOHAZ Panel, 2023). Field and modelling data from North America suggest that it may take 15 to 20 years for CWD prevalence to reach 1% in free-ranging cervid populations, although more rapid transmission may occur in captive populations. The surveillance sensitivity in North America means that the disease may have been present for 10 years or more in some areas before it was detected (Miller et al., 2000).

The prevalence of CWD in affected populations or species varies across North America. In captive herds, prevalence may reach 100% over time, while in affected free-ranging populations, reported prevalence ranges from <1% to >30%. Most clinical cases are observed in cervids 2 to 7 years old, especially males, which is believed to be due to behavioural differences rather than differences in susceptibility between sexes (EFSA BIOHAZ Panel, 2023). At least 13 different risk factors may contribute towards CWD spread in North America, such as host genetics, high deer density or inappropriate disposal of deer carcasses and slaughter by-products (EFSA BIOHAZ Panel, 2019).

Approaches towards CWD control and surveillance in captive and free-ranging deer vary widely across North America within and between jurisdictions (CIDRAP, 2025). A summary of the measures in place in each US state and Canadian province is available from the CWD Alliance (2026), a coalition of wildlife conservation agencies, dedicated to providing accurate information on CWD and supporting strategies to minimise its impact on free-ranging cervids. Wildlife agencies rely on voluntary testing of hunted deer carcasses as the main mechanism for CWD surveillance and management, usually using post-mortem ELISA or immunohistochemistry methods (CIDRAP, 2025).

In the USA, Animal and Plant Health Inspection Service (APHIS) operates the CWD Herd Certification Programme (HCP) in collaboration with state and wildlife agencies. This is a voluntary scheme which aims to provide a consistent, national approach to controlling CWD in farmed cervids and preventing interstate spread by establishing control measures such as fencing, detailed record keeping and CWD testing of all cervids over 12 months old that die for any reason. The Canadian Food Inspection Agency (CFIA) operates a similar programme, the CWD Herd Certification Programme. As of December 2025, 28 states were participating in the USA's CWD HCP and 5 Canadian provinces and one Canadian territory were participating in the Canadian programme (USDA, 2025b, CFIA, 2025).

Control methods fall within three general categories: prevention, containment, and control and suppression. Prevention

and containment aim to prevent CWDDepartment for Environment, Food and Rural Affairs introduction into areas where it has not previously been reported and to limit its geographical spread once it has been introduced, respectively. Both tend to include regulatory measures such as bans on the movement of live cervids, cervid carcasses or specified risk materials. Control and suppression aim to stabilise or reduce infection rates within a herd or population through measures such as selective or random culling (EFSA BIOHAZ Panel, 2017).

Despite control efforts, CWD has continued to spread among captive and free- ranging cervids in North America, with increasing prevalence in affected areas (Uehlinger et al., 2016, CFSPH, 2024). Eradicating CWD from North America appears infeasible due to its extent of geographic spread and epidemiological characteristics, such as environmental persistence (EFSA BIOHAZ Panel, 2017).

CWD in wild pigs in the USA

Wild pigs are an invasive population in the USA, especially in the south (Figure 2). They comprise escaped domestic swine, Eurasian wild boar and hybrids of the two (Smyser et al., 2020). Wild pigs frequently coexist with cervids in areas where CWD is endemic and may be exposed to CWD prions through rooting in contaminated soil, scavenging deer carcasses and predation on fawns. These ecological interactions provide multiple routes by which wild pigs could encounter prions from infected deer (Soto et al. 2025).

Under experimental conditions, domestic pigs can become infected with CWD by oral and intracerebral routes, suggesting that wild pigs might also be susceptible. Domestic pigs rarely develop clinical signs of CWD but accumulate prions in the lymphoid tissues in their heads and gut, suggesting that, like cervids, they could shed the prions in saliva and faeces (Moore et al., 2017).

Against this background, Soto et al. (2025) investigated potential interactions between wild pigs and CWD prions. They analysed over 300 brain and lymph node samples from 178 wild pigs living across Arkansas and Texas, USA. The animals were captured by the United States Department of Agriculture (USDA) between 2020 and 2021. None of the pigs included in the study were reported to be displaying clinical signs of disease.

Using an ultra-sensitive laboratory method (protein misfolding cyclic amplification (PMCA)), the researchers identified CWD prions in up to 37% of the lymph node samples and 15% of brain samples. The lowest detection rates were in the Texas samples (below 16%), matching the lower CWD prevalence in the state's cervid population. These findings indicate that wild pigs are naturally exposed to CWD prions in areas where the disease is present (Soto et al., 2025).

When intracerebrally inoculated with tissues from wild pigs, a small proportion of mice expressing deer prion protein developed subclinical prion infection. No transmission was detected in mice expressing pig prion protein. This suggests that wild pig tissues only contain low levels of infectious prions and that wild pigs are relatively resistant to natural infection. However, they could still contribute to CWD transmission, influencing its epidemiology, geographic distribution and interspecies spread (Soto et al., 2025).Department for Environment, Food and Rural Affairs While their exact role and importance in CWD transmission is unclear, wild pigs have considerable home ranges in North America (1.1 to 5.32 km on average), which may increase when food is scarce. This mobility could complicate efforts to control the disease if they play a role in its transmission (Soto et al., 2025).

The USDA's APHIS does not currently conduct active surveillance for CWD in wild pigs (USDA, 2025a).

Figure 2. Geographic distribution of wild pigs (purple) in the USA as of 27 January 2025, comprising escaped domestic pigs, Eurasian wild boar and hybrids of the two (adapted from USDA, 2026). Yellow (Texas) and green (Arkansas) circles indicate the states where CWD prions were detected in wild pig tissues.

Department for Environment, Food and Rural Affairs

Implications for Great Britain

CWD is a notifiable animal disease in Great Britain, but no cases have ever been reported (Defra and APHA, 2018, CIDRAP, 2025).

The introduction of CWD into Great Britain's cervid population could have devastating socio-economic and animal welfare impacts, resulting in marked population declines, as seen in the USA (Miller et al., 2008). There could also be significant losses to cervid farming, hunting and rural tourism industries, as well as significant costs associated with controlling the spread of the disease. The UK venison market alone is worth an estimated £100 million (Scotland Food and Drink, 2018).

There are several discrete wild pig populations in Great Britain, including wild boar and feral pigs. The largest known population is in the Forest of Dean in Gloucestershire, with an estimated 583 wild boar as of 2025/2026, although Forestry England (2025) aims to reduce the number to 400 to protect other species, such as plants and insects.

Pockets of wild boar and feral pigs exist in other parts of the country, but their exact numbers are unknown (Mathews et al., 2018). The potential impact of CWD introduction into Great Britain's wild pig population is uncertain because their role in disease transmission remains unclear. While they appear to be relatively resistant to natural CWD infection and disease, they could potentially contribute towards the maintenance and spread of CWD in Great Britain's cervid population (Soto et al., 2025).

To reduce the risk of CWD introduction, Great Britain suspended the import of live cervids and high-risk cervid products in June 2023, including urine hunting lures, from all countries where CWD has been reported. Fresh cervid meat, excluding offal and spinal cord, can only be imported into Great Britain from CWD-affected countries if it has tested negative for CWD using an approved diagnostic method, such as immunohistochemistry, and originates from an area where CWD has not been reported or officially suspected in the last 3 years (Defra and APHA, 2026).

The current risk of CWD prions being introduced into Great Britain's wild pig or cervid population ranges from very low (event is very rare but cannot be excluded) to negligible (event is so rare it does not merit consideration). This is based on the risk of incursion tool, developed by Roberts et al., (2011). It is also supported by a recent Defra and APHA (2025) risk assessment. While this assessment identified a few theoretical entry pathways, such as contaminated equipment, that could not be fully assessed due to limited data, there is no definitive evidence that they have ever resulted in the introduction of CWD into a new area.

Detection of CWD prions in wild pigs in the USA is unlikely to affect Great Britain's CWD risk level, as the USA is not approved to export live wild pigs to Great Britain (Defra, 2025). Import of infected wild pig meat or wild pig by-products from the USA could theoretically introduce CWD prions into Great Britain, but the risk of this is also very low. To date, CWD prions have only been reported in lymph node and brain tissue samples in wild pigs, at levels too low to cause disease in mouse models

Department for Environment, Food and Rural Affairs

(Soto et al., 2025). However, their presence in other tissues cannot be excluded. The USA is approved to export wild pig meat and certain wild pig by-products to Great Britain, excluding offal, minced meat and germplasm (Defra, 2025), but there appears to be limited trade in these commodities.

It is difficult to quantify the exact amount of wild pig meat exported to Great Britain, as available trade data does not always distinguish between meat of wild and domestic pigs. However, based on HMRC data, the last known export of non- domestic pig meat from the USA to Great Britain was in 2013 (4,881 kg).

Conclusion

CWD has continued to spread among captive and free-ranging cervids in North America since it was first detected in the 1960s. The finding of CWD prions in wild pigs in the USA suggests they could contribute towards transmission of the disease, influencing its epidemiology, geographic distribution and interspecies spread. However, further research is needed to confirm this. CWD has never been reported in Great Britain and the current risk of CWD prions being introduced into Great Britain's wild pig or cervid population ranges from very low to negligible.

Readers are reminded to be vigilant for signs of CWD. Information on how to spot the disease can be found here. Suspected cases must be reported immediately to the Defra Rural Services Helpline on 03000 200 301. In Wales, call 0300 303 8268. In Scotland, contact your local Field Services Office. Failure to do so is an offence. We will continue to monitor the situation.

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References

snip...see;

https://assets.publishing.service.gov.uk/media/697a3b013c71d838df6bd413/CWD_Prions_in_Cervids_and_Wild_Pigs_in_North_America.pdf

APHIS USDA Captive CWD Herds Update by State March 2026 Update

CHRONIC WASTING DISEASE CASES

Date of Index Case Confirmation Index Case State County Species Herd Type HCP Enrolled HCP Certified Number of Animals Herd Status

3/5/2026 2 YR Male PA Huntingdon WTD Elk Sika Hunt No No 100+ Quarantine

3/5/2026 1.5 YR Male TX Mason WTD Breeder No No 73 Quarantine

3/5/2026 2.5 YR Female TX Medina WTD Breeder No No 90 Quarantine

2/20/2026 2.75 YR Male MI Calhoun WTD Breeder Yes Yes 24 Quarantine

2/17/2026 2.5 YR Male KS Osage Axis Breeder No No 21 Quarantine

2/12/2026 3.5 YR TX Duval WTD Hunt No No ukn Quarantine

1/22/2026 3 YR Male PA Butler Elk Breeding Yes Yes 30 Quarantine 12/16/2025 Adults Male PA Indiana WTD elk Red deer Fallow Hunt No No ukn Quarantine

12/15/2025 3 YR Male PA Warren WTD Breeder No No >100 Quarantine

12/15/2025 4.5 YR Male PA Lycoming WTD ELK Hunt No No >100 Quarantine

12/15/2025 2.5 YR Male PA Juniata WTD Hobby No No 4 Quarantine 11/1/2025 ukn TX Limestone WTD Hunt No No 132 Quarantine

10/27/2025 3 YR Male WI Richland WTD Breeder Yes Yes ukn Depopulated

10/9/2025 2 YR Female TX Duvall WTD Breeder No No 94+ Quarantine

10/9/2025 2 YR Male PA Franklin WTD Breeder No No 23 Quarantine

Updated April 2026 CHRONIC WASTING DISEASE CASES

10/8/2025 3.5 YR Male PA Huntingdon WTD Hobby No No 2 Quarantine

10/8/2025 3 YR Male WI Portage WTD Fallow Hunt No No 132 Quarantine

9/26/2025 8.5 YR Female TX Navaro WTD Breeder No No 650 Quarantine

9/16/2025 3 YR Female PA Dauphin WTD Breeder Yes Yes 85 Quarantine

9/5/2025 3 YR Male TX Duvall WTD Breeder No No 107+ Quarantine

8/6/2025 Adult Female PA Fulton WTD Breeder No No 14 Quarantine

7/21/2025 4 YR Female PA Bedford WTD Breeder No No 34 Quarantine

6/3/2025 11 YR Female PA Blair WTD Breeder No No 45 Quarantine

6/3/2025 8 YR Female PA Bedford WTD Breeder No No 6 Quarantine

5/16/2025 5.5 YR Female WI Rock WTD Breeder No No ~46 Quarantine

5/14/2025 3 YR Female UT Weber Elk Hunt No No ukn Quarantine

4/30/2025 4.5 YR Male PA Jefferson WTD Hunt No No 36 Depopulated

Updated April 2026 CHRONIC WASTING DISEASE CASES

4/18/2025 10+ YR Ukn TX Zavala WTD Hunt No No 190 Quarantine

4/9/2025 6 YR Male MI Montcalm WTD Breeder No No 86 Quarantine

3/28/2025 3.5 YR Male PA Huntingdon WTD Hobby No No 2 Quarantine

3/28/2025 3.5 YR Female PA Wayne Red Deer Hunt No no 31 Depopulated

2/26/2025 1.5 YR Male TX Kauffman WTD Breeder Yes Yes 400 Quarantine

2/26/2025 3.5 Yr Male PA Lancaster WTD Breeder Yes Yes 105 Quarantine

2/21/2025 4 YR Male CO Montrose Elk Hunt No No 97 Quarantine

2/21/2025 7 YR Female MI Osceola WTD Hunt No No 201 Quarantine

2/10/2025 3.5 YR Male PA Perry WTD Hunt No No 15 Quarantine

1/7/2025 4 YR Female CO Mesa Elk Hunt No No 217 Quarantine

1/7/2025 2 YR Male UT Duchesne Elk Hunt No No 0 No animals

12/30/2024 4 YR Female ID Jefferson Elk Breeder No No 197 Quarantine

Updated April 2026

Updated March 2026

https://www.aphis.usda.gov/sites/default/files/status-of-captive-herds.pdf

https://chronic-wasting-disease.blogspot.com/2026/04/aphis-usda-captive-cwd-herds-update-by.html

APHIS Announces Funding to Support Chronic Wasting Disease Control and Prevention

Washington, D.C., May 15, 2026—The U.S. Department of Agriculture's Animal and Plant Health Inspection Service (APHIS) will provide approximately $12 million to support efforts by States and Tribal governments, research institutions, and universities to control and prevent chronic wasting disease (CWD) in wild and farmed cervids (e.g., deer, elk).

"Chronic wasting disease poses a serious threat to U.S. wildlife and agriculture. This funding reflects our commitment to working collaboratively with States, Tribes, and research partners to develop innovative solutions and protect the health of our nation's cervid populations," said Dr. Alan Huddleston, Acting U.S. Chief Veterinary Officer.

APHIS will competitively fund the most promising projects that develop innovative tools or methods, support State and Tribal CWD control programs at the local level and provide indemnity payments to cervid owners with pending claims. This includes:

Approximately $6 million to support critical projects to control and prevent CWD in farmed cervids,

Approximately $5.5 million to support research and management of CWD in wild cervids, and

Approximately $500,000 to support CWD prevention and management on Tribal lands.

CWD is an infectious, degenerative disease of cervids that causes brain cells to die, ultimately leading to the death of the affected animal. The incubation period can be lengthy, and infected animals may look healthy until the end stages of the disease, making it difficult to distinguish affected animals from healthy animals. Animals infected with CWD can transmit the disease to other animals during the "silent" incubation period. The disease has spread widely and the limited number of tools, as well as their efficacy, impacts the ability to effectively control the disease.

Cooperative agreement funding in previous years has resulted in the development and implementation of predictive genetics to assist farmed cervid owners in breeding for less susceptible deer, the removal of CWD-positive farmed cervid herds, increased diagnostic capabilities, increased CWD surveillance in wild cervid populations, hunter and public education, and carcass disposal options to reduce spread of CWD.

This year's investment will allow State departments of agriculture, State animal health agencies, State departments of wildlife or natural resources, federally recognized Native American Tribal governments and organizations, and research institutions and universities to further develop and implement CWD research, management, and response activities.

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https://www.usda.gov/about-usda/news/press-releases/2026/05/15/aphis-announces-funding-support-chronic-wasting-disease-control-and-prevention

Farmed Cervid Chronic Wasting Disease Management and Response Activities Funding Opportunity FY 2026 Last

Modified: May 15, 2026

https://www.aphis.usda.gov/funding/cwd/farmed-cervid-cwd-funding-opportunity-2026

Farmed Cervid Chronic Wasting Disease Management and Response Activities 2025 Cooperative Agreements

https://www.aphis.usda.gov/sites/default/files/2025-cwd-spending-plan.pdf

USDA EXPLANATORY NOTES ANIMAL AND PLANT HEALTH INSPECTION SERVICE 2025-2014 CHRONIC WASTING DISEASE CWD TSE CERVID

2025 USDA EXPLANATORY NOTES – ANIMAL AND PLANT HEALTH INSPECTION SERVICE

In 2023, eight percent of the farmed cervids in the HCP were tested for CWD at APHIS and State laboratories.

Of the 303,242 farmed cervids tested in 2023, APHIS confirmed 22 new CWD positive farmed cervid herds.

APHIS provided Federal indemnity to depopulate one of the newly identified positive herds and approved an indemnity payment for a second positive herd which will be provided in 2024 once depopulation occurs. The remaining infected herds are under State quarantines.

https://www.usda.gov/sites/default/files/documents/22-APHIS-2025-ExNotes.pdf

2024 USDA EXPLANATORY NOTES – ANIMAL AND PLANT HEALTH INSPECTION SERVICE

Cervids

In 2022, 7 percent of the 285,589 farmed cervids in the HCP participating states were tested for CWD at State and APHIS laboratories.

APHIS confirmed 23 new CWD positive farmed cervid herds.

APHIS provided Federal indemnity to depopulate nine of the newly identified positive herds in 2022. The remaining infected herds are under State quarantines. APHIS determines the use of Federal indemnity payments within the CWD program on a case-by-case basis.

https://www.usda.gov/sites/default/files/documents/23-2024-APHIS.pdf

2023 USDA EXPLANATORY NOTES – ANIMAL AND PLANT HEALTH INSPECTION SERVICE

Cervids

Currently, 28 States participate in the national CWD HCP. In FY 2021, more than 20,502 farmed cervids were tested for CWD at State and APHIS laboratories.

As a result, APHIS identified 35 new CWD positive farmed cervid herds.

APHIS provided Federal indemnity to depopulate nine of the newly identified deer herds in FY 2021. The remaining infected herds are under State quarantines. APHIS determines the use of Federal indemnity payments within the CWD program on a case-by-case basis.

https://www.usda.gov/sites/default/files/documents/23-2023-APHIS.pdf

2022 USDA EXPLANATORY NOTES – ANIMAL AND PLANT HEALTH INSPECTION SERVICE

Cervids

Currently, 28 States participate in the national CWD HCP. In FY 2020, more than 11,182 farmed cervids were tested for CWD at State and APHIS laboratories.

As a result, APHIS identified 22 new CWD positive farmed cervid herds.

APHIS provided Federal indemnity to depopulate 15 of the 22 newly identified deer herds in FY 2020.

Four additional farmed cervid herds that were identified as CWD positive herds in FY 2019, were indemnified in FY

2020.

The remaining infected herds are under State quarantines.

https://www.usda.gov/sites/default/files/documents/22APHIS2022Notes.pdf

2021 USDA EXPLANATORY NOTES – ANIMAL AND PLANT HEALTH INSPECTION SERVICE

In FY 2019 APHIS tested more than 11,000 farmed cervids for CWD.

As a result, APHIS identified 17 new CWD positive farmed cervid herds.

APHIS provided Federal indemnity to depopulate 7 of the 17 newly identified deer herds in FY 2019. The remaining infected herds found in FY 2019 are under State quarantines.

https://www.usda.gov/sites/default/files/documents/20aphis2021notes.pdf

2020 USDA EXPLANATORY NOTES – ANIMAL AND PLANT HEALTH INSPECTION SERVICE

Cervids

In FY 2018, APHIS identified 15 new CWD positive farmed cervid herds (14 deer herds and 1 reindeer herd).

The reindeer herd in Illinois was the first confirmed case of CWD in a reindeer in North America.

APHIS provided Federal indemnity to depopulate seven of the 15 newly identified deer herds in FY 2018.

The Agency also provided funding for the test and removal of 161 high risk animals that were in close proximity to reactors.

The remaining herds in FY 2018 are under State quarantines.

The Agency determines the use of Federal indemnities within the CWD program on a case-by-case basis. 20-59

https://www.usda.gov/sites/default/files/documents/20aphis2020notes.pdf

2019 President's Budget Animal and Plant Health Inspection Service

Cervids

APHIS' voluntary national CWD Herd Certification Plan (HCP) helps States, Tribes, and the cervid industry control CWD in farmed cervids by allowing the interstate movement only from certified herds.

Currently, 28 States participate in the national CWD HCP and the program tested 23,053 farmed cervids for CWD.

In FY 2017, eight new CWD positive farmed corvid herds were identified– one white-tail deer in Iowa, one white-tail deer herd in Minnesota, one white-tail and mule deer herd in Minnesota, one white-tail and sika deer herd in Michigan, three white-tail deer herds in Pennsylvania, and one white-tail deer herd in Texas.

APHIS provided Federal indemnity to depopulate the Iowa herd, the white-tail deer herd in Minnesota, one herd in Pennsylvania and the Texas herd. The State depopulated the Michigan herd. The remaining herds are under State quarantines. One Texas herd used Federal indemnity to remove and test select, high-risk animals to inform the epidemiological investigation and to evaluate the performance of ante-mortem tests.

The Agency determines the use of Federal indemnities within the CWD program on a case-by-case basis.

https://www.usda.gov/sites/default/files/documents/20aphis2019notes.pdf

2018 President's Budget Animal and Plant Health Inspection Service

Cervids

APHIS' voluntary national CWD Herd Certification Plan (HCP) helps States, Tribes, and the cervid industry control CWD in farmed cervids by allowing the interstate movement only from certified herds considered to be low risk.

Currently, 29 States participate in the national CWD HCP.

In FY 2016, the program tested 14,503 farmed cervids for CWD and identified seven new CWD positive farmed cervid herds – two white-tail deer herds in Texas, three white-tail deer herds in Wisconsin, one elk herd in Colorado and one elk herd in Iowa. The elk herd in Colorado was depopulated without Federal indemnity and the rest of the herds are under State quarantines. One Texas herd used Federal indemnity to remove and test select animals to inform the epidemiological investigation and to evaluate 20-72 the performance of ante-mortem tests.

The use of Federal indemnities within the CWD program is determined on a case-by-case basis. APHIS is also conducting several pilot projects related to new technologies. In FY 2016, the Agency

https://www.usda.gov/sites/default/files/documents/20aphisexnotes2018.pdf

2017 Explanatory Notes Animal and Plant Health Inspection Service

Cervids

APHIS' voluntary national CWD Herd Certification Plan (HCP) helps States, Tribes, and the cervid industry control CWD in farmed cervids by allowing the interstate movement only from certified herds considered to be low risk.

Currently, 30 States participate in the national CWD HCP: 29 have Approved Status and 1 has Provisional Approved Status. States that meet the CWD HCP requirements have Approved Status and States that do not meet CWD HCP program requirements but have developed a work plan and time frame with APHIS to complete those requirements have Provisional Approved Status.

In FY 2015, the program tested approximately 20,000 farmed cervids for CWD and identified eight new CWD positive farmed white-tailed deer herds – one in Utah, one in Pennsylvania, two in Ohio, two in Wisconsin, and two in Texas.

APHIS depopulated five of these herds (Pennsylvania, Utah, and one each in Wisconsin, Texas, and Ohio). Six elk herds in Colorado, four elk herds in Nebraska, one white-tailed deer herd in Wisconsin and one white-tailed deer herd in Texas remained in quarantine at the end of FY 2015.

APHIS also provided indemnity for and was the lead agency for the depopulation and disposal of four large CWD infected farmed cervid herds in Pennsylvania, Ohio, Utah, and Texas. In cooperation with the National Agricultural Statistics Service, APHIS conducted the first national study of the U.S. farmed-cervid industry in FY 2015. The study provides baseline industry statistics, a description of production practices and challenges, producer-reported disease occurrences, and an overview of health management and biosecurity practices.

https://www.usda.gov/sites/default/files/documents/20aphis2017notes.pdf

2016 Explanatory Notes Animal and Plant Health Inspection Service

In FY 2014, the program tested approximately 20,000 farmed cervids for CWD.

Two new CWD positive farmed white-tailed deer herds were identified – one in Pennsylvania and one in Wisconsin.

The program depopulated the PA herd and two additional CWD positive herds in quarantine since FY 2012 in Iowa (white-tailed deer herd) and Minnesota (red deer herd).

Six elk herds in Colorado, four elk herds in Nebraska, and one white-tailed deer in Wisconsin remained in quarantine at the end of FY 2014.

There also are numerous CWD exposed herds that are epidemiologically linked to CWD positive herds that remain in State quarantine pending completion of the epidemiology investigations.

snip…

APHIS provided indemnity for and was the lead agency for the depopulation and disposal of two large CWD infected farmed cervid herds in Iowa and Minnesota.

APHIS also provided indemnity for and assisted with the 20-80 appraisal and depopulation of a CWD infected farmed cervid herd in Pennsylvania.

APHIS also provided assistance to States with outbreak investigation, assessment of risk posed by infected or exposed animals, development of herd plans and continues to develop strategies for the purpose of controlling and managing

CWD in farmed cervids.

https://www.usda.gov/sites/default/files/documents/20aphis2016notes.pdf

2015 Explanatory Notes Animal and Plant Health Inspection Service

In FY 2013, the program tested approximately 18,100 farmed cervids for CWD, a fatal, degenerative disease that affects the central nervous system and lymphoid system of cervids. Through this routine surveillance, no new CWD cases were reported in farmed cervids in FY 2013. The last CWD positive herd was reported in FY 2012 in an Iowa white tail deer herd. Twelve positive herds remain (seven elk herds in Colorado, three elk herds in Nebraska, one white tail deer herd in Iowa, and one red deer herd in Minnesota).

https://www.usda.gov/sites/default/files/documents/20aphis2015notes.pdf

2014 Explanatory Notes Animal and Plant Health Inspection Service

In 2007, the cervid industry in the United States included 5,600 deer farms and 1,900 elk farms with an economic value of $894 million that supported nearly 30,000 jobs.

APHIS' main cervid activities are testing approximately 15,000 captive cervids for tuberculosis each year and supporting the chronic wasting disease (CWD) herd certification program (HCP). The joint tuberculosis (TB) and brucellosis proposed rule will represent significant changes to TB activities in captive cervids when implemented; it proposes to bring cervids into the regulatory program for brucellosis, as requested by stakeholders. As a result, surveillance will be enhanced and the number of captive cervids that are tested for TB annually is expected to increase. In 2014, APHIS will implement a survey to evaluate the effectiveness and impact of new APHIS regulations on the industry. Additionally, approval for a new diagnostic test for TB in captive cervids is expected to occur by FY 2013. The CWD HCP allows participating States to enroll herd owners

18-26

to meet minimum Federal standards to achieve and maintain a herd certification status. APHIS approves State applications for the national voluntary CWD herd certification program, conducts periodic reviews to ensure compliance, and supports confirmatory testing of presumptive CWD cases.

snip…

Without continued program funding, there would be a reduced preparedness, surveillance, and response to equine/cervid health issues that could increase the likelihood of disease spread resulting in larger and more serious disease outbreaks, lack of national standards leading to a patchwork of State level requirements which diminish interstate commerce, and loss in international credibility regarding U.S. animal health status.

Approximately 59 percent of the Equine and Cervid Health funding will be used for salaries and benefits, less than 1 percent for cooperative agreements and programmatic contracts, and the remaining supports normal operating costs such as travel, supplies, rent and utilities.

Reduce lower priority program activities (-$1.295 million) APHIS will reduce lower priority equine and cervid program activities in 2014, including eliminating Federal contributions for addressing CWD.

CWD is a degenerative neurological illness affecting elk and deer (cervids) in North America.

***> APHIS has determined that continued efforts to manage CWD are not practical and therefore considers this to be a low priority for the Agency.

***> On August 13, 2012, the rule that established uniform standards for a voluntary Federal-State cooperative CWD HCP and interstate movement requirements became effective. Implementation of the interstate movement of cervids was implemented in December 2012.

***> Many States have herd certification programs in place, and the incidence of CWD detections in farmed cervids is decreasing.

***> With the regulatory framework in place, continued APHIS activity, while useful, is no longer essential.

Stakeholders can continue to carry on program activities. The success of the voluntary HCP is based upon cooperation and shared responsibility among the Federal government and State and local interests. However, since these are local or regional disease spread issues, State and local governments are better positioned to take a more active role and to

better anticipate and plan for local or regional needs. APHIS will continue to conduct higher priority equine and cervid health activities and address concerns when identified. APHIS will reassign staff years to other Equine and Cervid Health activities as practical and reduce the overall staff years by eliminating the positions when vacancies arise. Reduction in Agency-level operating expenses (-$66,000) A reduction of $66,000 is requested for this line item related to Agency-level cost savings measures and operating efficiencies. Please refer to second paragraph on page 18-18.

18-27

Pay Increase (+$15,000)

An increase of $15,000 for pay costs which includes $3,000 for annualization of the FY 2013 pay raise and $12,000 for the anticipated FY 2014 pay raise.

https://www.usda.gov/sites/default/files/documents/18aphis2014notes.pdf

***> APHIS has determined that continued efforts to manage CWD are not practical and therefore considers this to be a low priority for the Agency.

***> On August 13, 2012, the rule that established uniform standards for a voluntary Federal-State cooperative CWD HCP and interstate movement requirements became effective. Implementation of the interstate movement of cervids was implemented in December 2012.

***> Many States have herd certification programs in place, and the incidence of CWD detections in farmed cervids is decreasing.

***> With the regulatory framework in place, continued APHIS activity, while useful, is no longer essential.

***> The occurrence of CWD must be viewed against the contest of the locations in which it occurred. It was an incidental and unwelcome complication of the respective wildlife research programmes. Despite its subsequent recognition as a new disease of cervids, therefore justifying direct investigation, no specific research funding was forthcoming. The USDA viewed it as a wildlife problem and consequently not their province!" page 26.

https://web.archive.org/web/20060307063531/http://www.bseinquiry.gov.uk/files/mb/m11b/tab01.pdf

FRIDAY, MAY 01, 2026

Oklahoma House Bill 3270 FAILED, Great News for Hunters and Wildlife

https://chronic-wasting-disease.blogspot.com/2026/05/oklahoma-house-bill-3270-failed-great.html

***> CWD to sheep, Scrapie to Cervid

Chronic Wasting Disease CWD vs Scrapie TSE Prion

https://www.ars.usda.gov/research/publications/publication/?seqNo115=410511

Detection of infectivity in orally inoculated pigs using mouse bioassay raises the possibility that naturally exposed pigs could act as a reservoir of CWD infectivity. Currently, swine rations in the U.S. could contain animal derived components including materials from deer or elk. In addition, feral swine could be exposed to infected carcasses in areas where CWD is present in wildlife populations. The current feed ban in the U.S. is based exclusively on keeping tissues from TSE infected cattle from entering animal feeds. These results indicating the susceptibility of pigs to CWD, coupled with the limitations of the current feed ban, indicates that a revision of the feed ban may be necessary to protect swine production and potentially human health.

https://www.ars.usda.gov/research/publications/publication/?seqNo115=353091

https://www.ars.usda.gov/research/project/?accnNo=432011&fy=2017

https://www.ars.usda.gov/research/publications/publication/?seqNo115=337105

https://www.ars.usda.gov/research/publications/publication/?seqNo115=326166

> However, at 51 months of incubation or greater, 5 animals were positive by one or more diagnostic methods. Furthermore, positive bioassay results were obtained from all inoculated groups (oral and intracranial; market weight and end of study) suggesting that swine are potential hosts for the agent of scrapie. <

*** Although the current U.S. feed ban is based on keeping tissues from TSE infected cattle from contaminating animal feed, swine rations in the U.S. could contain animal derived components including materials from scrapie infected sheep and goats. These results indicating the susceptibility of pigs to sheep scrapie, coupled with the limitations of the current feed ban, indicates that a revision of the feed ban may be necessary to protect swine production and potentially human health. <***

***> Results: PrPSc was not detected by EIA and IHC in any RPLNs. All tonsils and MLNs were negative by IHC, though the MLN from one pig in the oral <6 month group was positive by EIA. PrPSc was detected by QuIC in at least one of the lymphoid tissues examined in 5/6 pigs in the intracranial <6 months group, 6/7 intracranial >6 months group, 5/6 pigs in the oral <6 months group, and 4/6 oral >6 months group. Overall, the MLN was positive in 14/19 (74%) of samples examined, the RPLN in 8/18 (44%), and the tonsil in 10/25 (40%).

***> Conclusions: This study demonstrates that PrPSc accumulates in lymphoid tissues from pigs challenged intracranially or orally with the CWD agent, and can be detected as early as 4 months after challenge. CWD-infected pigs rarely develop clinical disease and if they do, they do so after a long incubation period. This raises the possibility that CWD-infected pigs could shed prions into their environment long before they develop clinical disease. Furthermore, lymphoid tissues from CWD-infected pigs could present a potential source of CWD infectivity in the animal and human food chains.

https://www.ars.usda.gov/research/publications/publication/?seqNo115=353091

https://www.ars.usda.gov/research/project/?accnNo=432011&fy=2017

Research Project: Elucidating the Pathobiology and Transmission of Transmissible Spongiform Encephalopathies

Location: Virus and Prion Research

Title: Differentiation of scrapie from chronic wasting disease in white-tailed deer

snip…

Accomplishments

1. 01 Determined that white-tailed deer (WTD) infected with scrapie from sheep can transmit the disease to other deer under conditions mimicking natural exposure. It has long been suggested that prion disease in deer (chronic wasting disease (CWD)) was caused by the prion agent from sheep. The prion disease that affects sheep, scrapie, has been recognized for hundreds of years. However, chronic wasting disease, a similar disease found in WTD, has only been recognized since the 1960s. ARS researchers in Ames, Iowa, showed that white-tailed deer sick with scrapie from sheep can infect other deer under conditions mimicking natural exposure. Furthermore, this work shows that CWD is difficult to differentiate from WTD infected with scrapie. WTD scrapie prions accumulate in the lymphoreticular system in a manner similar to CWD, meaning that environmental contamination may occur through feces, saliva, and other body fluids of scrapie affected WTD as has been shown for CWD. The presence of WTD infected with scrapie could confound mitigation efforts for chronic wasting disease. This information informs regulatory officials, the farmed cervid industry, and officials tasked with protecting animal health such as state Departments of Agriculture, Natural Resources, or Parks and Wildlife with regard to a disease similar to CWD but arising from sheep scrapie that could be present in WTD that have contact with scrapie affected sheep and/or goats.

https://www.ars.usda.gov/research/project/?accnNo=440677&fy=202

Chronic Wasting Disease CWD vs Scrapie TSE Prion

Volume 30, Number 8—August 2024

Research

Scrapie Versus Chronic Wasting Disease in White-Tailed Deer

Zoe J. Lambert1, Jifeng Bian, Eric D. Cassmann, M. Heather West Greenlee, and Justin J. Greenlee

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Abstract

White-tailed deer are susceptible to scrapie (WTD scrapie) after oronasal inoculation with the classical scrapie agent from sheep. Deer affected by WTD scrapie are difficult to differentiate from deer infected with chronic wasting disease (CWD). To assess the transmissibility of the WTD scrapie agent and tissue phenotypes when further passaged in white-tailed deer, we oronasally inoculated wild-type white-tailed deer with WTD scrapie agent. We found that WTD scrapie and CWD agents were generally similar, although some differences were noted. The greatest differences were seen in bioassays of cervidized mice that exhibited significantly longer survival periods when inoculated with WTD scrapie agent than those inoculated with CWD agent. Our findings establish that white-tailed deer are susceptible to WTD scrapie and that the presence of WTD scrapie agent in the lymphoreticular system suggests the handling of suspected cases should be consistent with current CWD guidelines because environmental shedding may occur.

snip...

The potential for zoonoses of cervid-derived PrPSc is still not well understood (6,18,45–47); however, interspecies transmission can increase host range and zoonotic potential (48–50). Therefore, to protect herds and the food supply, suspected cases of WTD scrapie should be handled the same as cases of CWD.

https://wwwnc.cdc.gov/eid/article/30/8/24-0007_article

Western blots done on samples from the brainstem, cerebellum, and lymph nodes of scrapie-infected WTD have a molecular profile similar to CWD and distinct from western blots of samples from the cerebral cortex, retina, or the original sheep scrapie inoculum. WTD are susceptible to the agent of scrapie from sheep and differentiation from CWD may be difficult.

https://www.ars.usda.gov/research/publications/publication/?seqNo115=336834

Component 6: Transmissible Spongiform Encephalopathies

Sheep scrapie agent can infect white-tailed deer after oronasal exposure.

The origin of chronic wasting disease (CWD) is not known, but it has many similarities to the sheep prion disease called scrapie. It has long been hypothesized that CWD arose through transmission of sheep scrapie to deer. ARS researchers in Ames, Iowa, conducted research to determine if scrapie derived from sheep could be transmitted to white-tailed deer. The deer inoculated with sheep scrapie developed clinical signs and the abnormal prion protein could be detected in a wide range of tissues. These results indicate that deer may be susceptible to sheep scrapie if exposed to the disease in natural or agricultural settings. In addition, several strong similarities between CWD in white-tailed deer and the experimental cases of scrapie in white-tailed deer suggests that it would be difficult to distinguish scrapie from CWD in deer or identify scrapie if a case occurs. This information should be considered by deer farmers for keeping their herds free from prion diseases.

https://www.ars.usda.gov/ARSUserFiles/np103/AnnualReports/NP103%20FY2023%20Annual%20Report_Final.pdf

It has long been hypothesized that CWD arose through transmission of sheep scrapie to deer. ARS researchers in Ames, Iowa, conducted research to determine if scrapie derived from sheep could be transmitted to white-tailed deer. The deer inoculated with sheep scrapie developed clinical signs and the abnormal prion protein could be detected in a wide range of tissues. These results indicate that deer may be susceptible to sheep scrapie if exposed to the disease in natural or agricultural settings. In addition, several strong similarities between CWD in white-tailed deer and the experimental cases of scrapie in white-tailed deer suggests that it would be difficult to distinguish scrapie from CWD in deer or identify scrapie if a case occurs. This information should be considered by deer farmers for keeping their herds free from prion diseases.

https://www.ars.usda.gov/ARSUserFiles/np103/AnnualReports/NP103%20FY2023%20Annual%20Report_Final.pdf

Scrapie, Humans, Zoonotic, what if?

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Transmission of scrapie prions to primate after an extended silent incubation period

*** In complement to the recent demonstration that humanized mice are susceptible to scrapie, we report here the first observation of direct transmission of a natural classical scrapie isolate to a macaque after a 10-year incubation period. Neuropathologic examination revealed all of the features of a prion disease: spongiform change, neuronal loss, and accumulation of PrPres throughout the CNS.

*** This observation strengthens the questioning of the harmlessness of scrapie to humans, at a time when protective

measures for human and animal health are being dismantled and reduced as c-BSE is considered controlled and being eradicated.

*** Our results underscore the importance of precautionary and protective measures and the necessity for long-term experimental transmission studies to assess the zoonotic potential of other animal prion strains.

http://www.ars.usda.gov/research/publications/publications.htm?SEQ_NO_115=313160

Moreover, sporadic disease has never been observed in breeding colonies or primate research laboratories, most notably among hundreds of animals over several decades of study at the National Institutes of Health25, and in nearly twenty older animals continuously housed in our own facility.

Even if the prevailing view is that sporadic CJD is due to the spontaneous formation of CJD prions, it remains possible that its apparent sporadic nature may, at least in part, result from our limited capacity to identify an environmental origin.

https://www.nature.com/articles/srep11573

https://www.ars.usda.gov/research/publications/publication/?seqNo115=361032

O.05: Transmission of prions to primates after extended silent incubation periods: Implications for BSE and scrapie risk assessment in human populations

*** We recently observed the direct transmission of a natural classical scrapie isolate to macaque after a 10-year silent incubation period,

***with features similar to some reported for human cases of sporadic CJD, albeit requiring fourfold long incubation than BSE. Scrapie, as recently evoked in humanized mice (Cassard, 2014),

***is the third potentially zoonotic PD (with BSE and L-type BSE),

***thus questioning the origin of human sporadic cases.

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PRION 2015 CONFERENCE

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5019500/

PRION 2016 TOKYO

Saturday, April 23, 2016

SCRAPIE WS-01: Prion diseases in animals and zoonotic potential 2016

Prion. 10:S15-S21. 2016 ISSN: 1933-68961933-690X

WS-01: Prion diseases in animals and zoonotic potential

***Transmission data also revealed that several scrapie prions propagate in HuPrP-Tg mice with efficiency comparable to that of cattle BSE. While the efficiency of transmission at primary passage was low, subsequent passages resulted in a highly virulent prion disease in both Met129 and Val129 mice.

***Transmission of the different scrapie isolates in these mice leads to the emergence of prion strain phenotypes that showed similar characteristics to those displayed by MM1 or VV2 sCJD prion.

***These results demonstrate that scrapie prions have a zoonotic potential and raise new questions about the possible link between animal and human prions.

http://www.tandfonline.com/doi/abs/10.1080/19336896.2016.1163048?journalCode=kprn20

Title: Transmission of scrapie prions to primate after an extended silent incubation period)

*** In complement to the recent demonstration that humanized mice are susceptible to scrapie, we report here the first observation of direct transmission of a natural classical scrapie isolate to a macaque after a 10-year incubation period. Neuropathologic examination revealed all of the features of a prion disease: spongiform change, neuronal loss, and

accumulation of PrPres throughout the CNS.

*** This observation strengthens the questioning of the harmlessness of scrapie to humans, at a time when protective measures for human and animal health are being dismantled and reduced as c-BSE is considered controlled and being eradicated.

*** Our results underscore the importance of precautionary and protective measures and the necessity for long-term experimental transmission studies to assess the zoonotic potential of other animal prion strains.

http://www.ars.usda.gov/research/publications/publications.htm?SEQ_NO_115=313160

*** Grant Agreement number: 222887 ***

*** Project acronym: PRIORITY ***

*** Project title: Protecting the food chain from prions: shaping European priorities through basic and applied research Funding ***

Scheme: Large-scale integrating project Period covered: from Oct. 1, 2009 to Sept. 30, 2014

Name of the scientific representative of the project's co-ordinator1, Title and Organisation: Jesús R. Requena, Ph.D., Associate Professor, Department of medicine, University of Santiago de Compostela, Spàin. Tel: 34-881815464 Fax: 34-881815403 E-mail: jesus.requena@usc.es

Project website¡ Error! Marcador no definido. address: www.prionpriority.eu

PRIORITY, PROJECT FINAL REPORT

*** 14) Concluding that atypical scrapie can transmit to Humans and that its strain properties change as it transmits between species ***

snip...

http://cordis.europa.eu/docs/results/222/222887/final1-priority-final-report.pdf

> CWD TSE PrP Humans, what if? <

BRIEF RESEARCH REPORT

Public Health, 23 June 2026 Sec. Infectious Diseases: Epidemiology and Prevention Volume 14 - 2026 | https://doi.org/10.3389/fpubh.2026.1847770

Sporadic Creutzfeldt-Jakob disease following venison exposure in a chronic wasting disease-endemic region: a zoonotic surveillance perspective

Eithan Kotkowski .jpeg Eithan Kotkowski 1,2* J Jonathan Trout 1,3 M Matthew Roberts 1,4,5 M Michael Tabet 6 C Carlayne Jackson 1 Sarah Horn .jpeg Sarah Horn 1 1. Department of Neurology, UT Health San Antonio, San Antonio, TX, United States 2. Research Imaging Institute, UT Health San Antonio, San Antonio, TX, United States

Abstract Background: Creutzfeldt-Jakob disease (CJD) is a rare, rapidly progressive prion disease with established zoonotic transmission only in its variant form. Chronic wasting disease (CWD), a prion disease affecting cervid populations in North America, continues to expand geographically, raising public health concerns regarding potential interspecies transmission.

Methods: We describe the clinical course, diagnostic evaluation, and public health investigation of a patient with confirmed sporadic CJD and long-term venison exposure from CWD-endemic regions in Louisiana. Clinical data, neuroimaging, electroencephalography, cerebrospinal fluid (CSF) biomarkers, neuropathology, and epidemiologic findings were reviewed.

Case report: A 72-year-old lifelong hunter developed rapidly progressive dementia with startle myoclonus and characteristic MRI findings of cortical ribboning and basal ganglia diffusion restriction. CSF biomarkers (RT-QuIC, total tau, 14-3-3) and postmortem neuropathology confirmed sporadic CJD, MM1 subtype. During hospitalization, the patient developed posterior reversible encephalopathy syndrome (PRES), complicating radiographic interpretation. Given extensive cervid exposure and reports of similar illness among hunting peers from the same lodge, the case was reported to public health authorities. Given extensive cervid exposure and reports of similar illness among hunting

peers from the same lodge, the apparent clustering of suspected prion disease prompted epidemiologic investigation and public health review.

Conclusion: This case highlights diagnostic and public health challenges posed by prion disease in individuals with relevant zoonotic exposure histories. Although no evidence supporting confirmed CWD-to-human transmission was identified, the apparent clustering of suspected prion disease within a shared exposure network appropriately prompted epidemiologic investigation, structured exposure assessment, and public health review.

snip…

Concern for zoonotic transmission arose after the neurology team learned that other individuals from the same hunting lodge had experienced similarly rare neurologic symptoms. Public health investigation identified one additional individual from the lodge with reported positive CSF prion biomarkers but without neuro-pathologic confirmation, precluding definitive classification. This investigation allowed exclusion of alternative neurodegenerative explanations within a suspected cluster and informed appropriate classification within national prion surveillance frameworks. Geographic and temporal analyses supported classification as sporadic CJD.

https://www.frontiersin.org/journals/public-health/articles/10.3389/fpubh.2026.1847770/full

***> CWD to Primates and Humans, what if?

So, this is what we leave our children and grandchildren?

> CWD TSE Prion Zoonotic Zoonosis Humans, What if? <

CDC CWD TSE Prion Update 2024

KEY POINTS

Chronic wasting disease affects deer, elk and similar animals in the United States and a few other countries.

The disease hasn't been shown to infect people.

However, it might be a risk to people if they have contact with or eat meat from animals infected with CWD.

https://www.cdc.gov/chronic-wasting/about/index.html

see archived url link;

https://web.archive.org/web/20240617224730/https://www.cdc.gov/chronic-wasting/about/index.html

Prions in Muscles of Cervids with Chronic Wasting Disease, Norway

Volume 31, Number 2—February 2025

Research

Prions in Muscles of Cervids with Chronic Wasting Disease, Norway

Snip…

In summary, the results of our study indicate that prions are widely distributed in peripheral and edible tissues of cervids in Norway, including muscles. This finding highlights the risk of human exposure to small amounts of prions through handling and consuming infected cervids.

Appendix

https://wwwnc.cdc.gov/eid/article/31/2/24-0903-app1.pdf

https://wwwnc.cdc.gov/eid/article/31/2/24-0903_article

Volume 31, Number 2—February 2025

Dispatch

Detection of Chronic Wasting Disease Prions in Raw, Processed, and Cooked Elk Meat, Texas, USA

Snip…

Of note, our data show that exposure to high temperatures used to cook the meat increased the availability of prions for in vitro amplification. Considering the potential implications in food safety and public health, we believe that the findings described in this study warrant further research. Our results suggest that although the elk meat used in this study resisted different manipulations involved in subsequent consumption by humans, their zoonotic potential was limited. Nevertheless, even though no cases of CWD transmission to human have been reported, the potential for human infection is still unclear and continued monitoring for zoonotic potential is warranted.

https://wwwnc.cdc.gov/eid/article/31/2/24-0906_article

Detection of chronic wasting disease prions in processed meats

Results: Our results show positive prion detection in all the samples analyzed using deer and elk substrates. Surprisingly, cooked meats displayed increased seeding activities. This data suggests that CWD-prions are available to people even after meats are processed and cooked.

Conclusions: These results suggest CWD prions are accessible to humans through meats, even after processing and cooking. Considering the fact that these samples were collected from already processed specimens, the availability of CWD prions to humans is probably underestimated.

"Our results show positive prion detection in all the samples analyzed using deer and elk substrates. Surprisingly, cooked meats displayed increased seeding activities."

Meeting-book-final-version prion 2023 Prion 2023 Congress Organizing Committee and the NeuroPrion Association, we invite you to join us for the International Conference Prion2023 from 16-20 October 2023 in Faro, Portugal.

https://prion2023.org/wp-content/uploads/2023/10/Meeting-book-final-version2.pdf

https://web.archive.org/web/20250828201533/https://prion2023.org/wp-content/uploads/2023/10/Meeting-book-final-version2.pdf

https://www.researchgate.net/profile/Syed-Zahid-Shah/publication/378314391_Meeting-book-final-version_prion_2023/links/65d44dad28b7720cecdca95f/Meeting-book-final-version-prion-2023.pdf

DETECTION OF CHRONIC WASTING DISEASE PRIONS IN PROCESSED MEATS.

In this study, we analyzed different processed meats derived from a pre-clinical, CWD-positive free-ranging elk. Products tested included filets, sausages, boneless steaks, burgers, ham steaks, seasoned chili meats, and spiced meats. CWD-prion presence in these products were assessed by PMCA using deer and elk substrates. Our results show positive prion detection in all products. To confirm the resilience of CWD-prions to traditional cooking methods, we grilled and boiled the meat products and evaluated them for any remnant PMCA seeding activity. Results confirmed the presence of CWD-prions in these meat products suggesting that infectious particles may still be available to people even after cooking. Our results strongly suggest ongoing human exposure to CWD-prions and raise significant concerns of zoonotic transmission through ingestion of CWD contaminated meat products.

Products tested included filets, sausages, boneless steaks, burgers, ham steaks, seasoned chili meats, and spiced meats.

CWD-prion presence in these products were assessed by PMCA using deer and elk substrates.

Our results show positive prion detection in all products.

Results confirmed the presence of CWD-prions in these meat products suggesting that infectious particles may still be available to people even after cooking.

Our results strongly suggest ongoing human exposure to CWD-prions and raise significant concerns of zoonotic transmission through ingestion of CWD contaminated meat products.

https://intcwdsympo.files.wordpress.com/2023/06/final-agenda-with-abstracts.pdf?force_download=true

Transmission of prion infectivity from CWD-infected macaque tissues to rodent models demonstrates the zoonotic potential of chronic wasting disease.

Further passage to cervidized mice revealed transmission with a 100% attack rate.

Our findings demonstrate that macaques, considered the best model for the zoonotic potential of prions, were infected upon CWD challenge, including the oral one.

The disease manifested as atypical in macaques and initial transgenic mouse transmissions, but with infectivity present at all times, as unveiled in the bank vole model with an unusual tissue tropism.

Epidemiologic surveillance of prion disease among cervid hunters and people likely to have consumed venison contaminated with chronic wasting disease

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https://intcwdsympo.files.wordpress.com/2023/06/final-agenda-with-abstracts.pdf?force_download=true

Fortuitous generation of a zoonotic cervid prion strain

Aims: Whether CWD prions can infect humans remains unclear despite the very substantial scale and long history of human exposure of CWD in many states or provinces of USA and Canada. Multiple in vitro conversion experiments and in vivo animal studies indicate that the CWD-to-human transmission barrier is not unbreakable. A major long-term public health concern on CWD zoonosis is the emergence of highly zoonotic CWD strains. We aim to address the question of whether highly zoonotic CWD strains are possible.

Materials and Methods: We inoculated several sCJD brain samples into cervidized transgenic mice (Tg12), which were intended as negative controls for bioassays of brain tissues from sCJD cases who had potentially been exposed to CWD. Some of the Tg12mice became infected and their brain tissues were further examined by Western blot as well as serial passages in humanized or cervidized mice.

Results: Passage of sCJDMM1 in transgenic mice expressing elk PrP (Tg12) resulted in a "cervidized" CJD strain that we termed CJDElkPrP. We observed 100% transmission of the original CJDElkPrP in transgenic mice expressing human PrP. We passaged CJDElkPrP two more times in the Tg12mice. We found that such second and third passage CJDElkPrP prions retained 100% transmission rate in the humanized mice, despite that the natural elk CWD isolates and CJDElkPrP share the same elk PrP sequence. In contrast, we and others found zero or poor transmission of natural elk CWD isolates in humanized mice.

Conclusions: Our data indicate that highly zoonotic cervid prion strains are not only possible but also can retain zoonotic potential after serial passages in cervids, suggesting a very significant and serious long-term risk of CWD zoonosis given that the broad and continuing spread of CWD prions will provide fertile grounds for the emergence of zoonotic CWD strains over time.

https://prion2023.org/wp-content/uploads/2023/10/Meeting-book-final-version2.pdf

https://web.archive.org/web/20250828201533/https://prion2023.org/wp-content/uploads/2023/10/Meeting-book-final-version2.pdf

https://www.researchgate.net/profile/Syed-Zahid-Shah/publication/378314391_Meeting-book-final-version_prion_2023/links/65d44dad28b7720cecdca95f/Meeting-book-final-version-prion-2023.pdf

Transmission of cervid prions to humanized mice demonstrates the zoonotic potential of CWD

Samia Hannaoui1 · Irina Zemlyankina1 · Sheng Chun Chang1 · Maria Immaculata Arifn1 · Vincent Béringue2 · Debbie McKenzie3 · Hermann M. Schatzl1 · Sabine Gilch1

Received: 24 May 2022 / Revised: 5 August 2022 / Accepted: 7 August 2022

Abstract

Prions cause infectious and fatal neurodegenerative diseases in mammals. Chronic wasting disease (CWD), a prion disease of cervids, spreads efficiently among wild and farmed animals. Potential transmission to humans of CWD is a growing concern due to its increasing prevalence. Here, we provide evidence for a zoonotic potential of CWD prions, and its probable signature using mice expressing human prion protein (PrP) as an infection model. Inoculation of these mice with deer CWD isolates resulted in atypical clinical manifestation with prion seeding activity and efficient transmissible infectivity in the brain and, remarkably, in feces, but without classical neuropathological or Western blot appearances of prion diseases. Intriguingly, the protease-resistant PrP in the brain resembled that found in a familial human prion disease and was transmissible upon second passage. Our results suggest that CWD might infect humans, although the transmission barrier is likely higher compared to zoonotic transmission of cattle prions. Notably, our data suggest a different clinical presentation, prion signature, and tissue tropism, which causes challenges for detection by current diagnostic assays. Furthermore, the presence of infectious prions in feces is concerning because if this occurs in humans, it is a source for human-to-human transmission. These findings have strong implications for public health and CWD management.

Keywords Chronic wasting disease · CWD · Zoonotic potential · Prion strains · Zoonotic prions

HIGHLIGHTS OF THIS STUDY

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Our results suggest that CWD might infect humans, although the transmission barrier is likely higher compared to zoonotic transmission of cattle prions. Notably, our data suggest a different clinical presentation, prion signature, and tissue tropism, which causes challenges for detection by current diagnostic assays. Furthermore, the presence of infectious prions in feces is concerning because if this occurs in humans, it is a source for human-to-human transmission. These findings have strong implications for public health and CWD management.

In this study, we evaluated the zoonotic potential of CWD using a transgenic mouse model overexpressing human M129-PrPC (tg650 [12]). We inoculated tg650mice intracerebrally with two deer CWD isolates, Wisc-1 and 116AG [22, 23, 27, 29]. We demonstrate that this transgenic line was susceptible to infection with CWD prions and displayed a distinct leading clinical sign, an atypical PrPSc signature and unusual fecal shedding of infectious prions. Importantly, these prions generated by the human PrP transgenic mice were transmissible upon passage. Our results are the first evidence of a zoonotic risk of CWD when using one of the most common CWD strains, Wisc-1/CWD1 for infection. We demonstrated in a human transgenic mouse model that the species barrier for transmission of CWD to humans is not absolute. The fact that its signature was not typical raises the questions whether CWD would manifest in humans as a subclinical infection, whether it would arise through direct or indirect transmission including an intermediate host, or a silent to uncovered human-to-human transmission, and whether current detection techniques will be suffcient to unveil its presence.

Our findings strongly suggest that CWD should be regarded as an actual public health risk. Here, we use humanized mice to show that CWD prions can cross the species barrier to humans, and remarkably, infectious prions can be excreted in feces.

Our results indicate that if CWD crosses the species-barrier to humans, it is unlikely to resemble the most common forms of human prion diseases with respect to clinical signs, tissue tropism and PrPSc signature. For instance, PrPSc in variable protease-sensitive prionopathy (VPSPr), a sporadic form of human prion disease, and in the genetic form Gerstmann-Sträussler-Scheinker syndrome (GSS) is defined by an atypical PK-resistant PrPSc fragment that is non-glycosylated and truncated at both C- and N-termini, with a molecular weight between 6 and 8 kDa [24, 44–46]. These biochemical features are unique and distinctive from PrPSc (PrP27-30) found in most other human or animal prion disease. The atypical PrPSc signature detected in brain homogenate of tg650 mice #321 (1st passage) and #3063 (2nd passage), and the 7–8 kDa fragment (Figs. 2, 4) are very similar to that of GSS, both in terms of migration profile and the N-terminal cleavage site.

CWD in humans might remain subclinical but with PrPSc deposits in the brain with an unusual morphology that does not resemble the patterns usually seen in different prion diseases (e.g., mouse #328; Fig. 3), clinical with untraceable abnormal PrP (e.g., mouse #327) but still transmissible and uncovered upon subsequent passage (e.g., mouse #3063; Fig. 4), or prions have other reservoirs than the usual ones, hence the presence of infectivity in feces (e.g., mouse #327) suggesting a potential for human-to-human transmission and a real iatrogenic risk that might be unrecognizable.

“suggesting a potential for human-to-human transmission and a real iatrogenic risk that might be unrecognizable.”

==========

Supplementary Information The online version contains supplementary material available at

https://doi.org/10.1007/s00401-022-02482-9

snip...see full text;

https://link.springer.com/article/10.1007/s00401-022-02482-9

https://link.springer.com/content/pdf/10.1007/s00401-022-02482-9.pdf

Transmission of Cervid Prions to Humanized Mice Demonstrates the Zoonotic Potential of CWD

Samia Hannaouia, Irina Zemlyankinaa, Sheng Chun Changa, Maria Immaculata Arifina, Vincent Béringueb, Debbie McKenziec, Hermann M. Schatzla, and Sabine Gilcha

Results: Here, we provide the strongest evidence supporting the zoonotic potential of CWD prions, and their possible phenotype in humans. Inoculation of mice expressing human PrPCwith deer CWD isolates (strains Wisc-1 and 116AG) resulted in atypical clinical manifestations in > 75% of the mice, with myoclonus as leading clinical sign. Most of tg650brain homogenates were positive for seeding activity in RT-QuIC. Clinical disease and presentation was transmissible to tg650mice and bank voles. Intriguingly, protease-resistant PrP in the brain of tg650 mice resembled that found in a familial human prion disease and was transmissible upon passage. Abnormal PrP aggregates upon infection with Wisc-1 were detectable in thalamus, hypothalamus, and midbrain/pons regions.

Unprecedented in human prion disease, feces of CWD-inoculated tg650 mice harbored prion seeding activity and infectious prions, as shown by inoculation of bank voles and tg650 with fecal homogenates.

Conclusions: This is the first evidence that CWD can infect humans and cause disease with a distinctive clinical presentation, signature, and tropism, which might be transmissible between humans while current diagnostic assays might fail to detect it. These findings have major implications for public health and CWD-management.

https://www.tandfonline.com/doi/full/10.1080/19336896.2022.2091286

Macaque tissues to rodent models demonstrates the zoonotic potential of chronic wasting disease.

Samia Hannaoui1,2, Ginny Cheng1,2, Wiebke Wemheuer3, Walter Schulz-Schaeffer3, Sabine Gilch1,2, Hermann Schatzl1,2
1University of Calgary, Calgary, Canada. 2Calgary Prion Research Unit, Calgary, Canada. 3Institute of Neuropathology, Medical Faculty, Saarland University, Homburg/Saar, Germany

Snip…

***> Further passage to cervidized mice revealed transmission with a 100% attack rate.

***> Our findings demonstrate that macaques, considered the best model for the zoonotic potential of prions, were infected upon CWD challenge, including the oral one.

****> The disease manifested as atypical in macaques and initial transgenic mouse transmissions, but with infectivity present at all times, as unveiled in the bank vole model with an unusual tissue tropism.

***> Epidemiologic surveillance of prion disease among cervid hunters and people likely to have consumed venison contaminated with chronic wasting disease

=====

https://intcwdsympo.files.wordpress.com/2023/06/final-agenda-with-abstracts.pdf?force_download=true

18. Zoonotic potential of moose-derived chronic wasting disease prions after adaptation in intermediate species

Tomás Barrioa, Jean-Yves Doueta, Alvina Huora, Séverine Lugana, Naïma Arona, Hervé Cassarda, Sylvie L. Benestadb, Juan Carlos Espinosac, Juan María Torresc, Olivier Andréolettia

aUnité Mixte de Recherche de l'Institut National de Recherche pour l'Agriculture, l'Alimentation et l'Environnement 1225 Interactions Hôtes-Agents Pathogènes, École Nationale Vétérinaire de Toulouse, 31076 Toulouse, France; bNorwegian Veterinary Institute, P.O. Box 64, NO-1431 Ås, Norway; cCentro de Investigación en Sanidad Animal (CISA-INIA), 28130, Valdeolmos,

Madrid, Spain

Aims: Chronic wasting disease (CWD) is an emerging prion disease in Europe. To date, cases have been reported in three Nordic countries and in several species, including reindeer (Rangifer tarandus), moose (Alces alces) and red deer (Cervus elaphus). Cumulating data suggest that the prion strains responsible for the European cases are distinct from those circulating in North America. The biological properties of CWD prions are still poorly documented, in particular their spillover and zoonotic capacities. In this study, we aimed at characterizing the interspecies transmission potential of Norwegian moose CWD isolates.

Materials and Methods: For that purpose, we performed experimental transmissions in a panel of transgenic models expressing the PrPC sequence of various species.

Results: On first passage, one moose isolate propagated in the ovine PrPC-expressing model (Tg338). After adaptation in this host, moose CWD prions were able to transmit in mice expressing either bovine or human PrPC with high efficacy.

Conclusions: These results suggest that CWD prions can acquire enhanced zoonotic properties following adaptation in an intermediate species.

Funding

Grant number: AAPG2020 EU-CWD, ICRAD2020 TCWDE, NRC2022 NorCWD

Acknowledgement

https://www.tandfonline.com/doi/full/10.1080/19336896.2024.2424058

" After adaptation in this host, moose CWD prions were able to transmit in mice expressing either bovine or human PrPC with high efficacy."

"Also, we do not claim that "no-one has ever been infected with prion disease from eating venison." Our conclusion stating that we found no strong evidence of CWD transmission to humans in the article you quoted or in any other forum is limited to the patients we investigated."

*** now, let's see what the authors said about this casual link, personal communications years ago, and then the latest on the zoonotic potential from CWD to humans from the TOKYO PRION 2016 CONFERENCE.

see where it is stated NO STRONG evidence. so, does this mean there IS casual evidence ????

"Our conclusion stating that we found no strong evidence of CWD transmission to humans"

Subject: CWD aka MAD DEER/ELK TO HUMANS ???

Date: September 30, 2002 at 7:06 am PST

From: "Belay, Ermias"

To: Cc: "Race, Richard (NIH)" ; ; "Belay, Ermias"

Sent: Monday, September 30, 2002 9:22 AM

Subject: RE: TO CDC AND NIH - PUB MED- 3 MORE DEATHS - CWD - YOUNG HUNTERS

Dear Sir/Madam, In the Archives of Neurology you quoted (the abstract of which was attached to your email), we did not say CWD in humans will present like variant CJD.. That assumption would be wrong. I encourage you to read the whole article and call me if you have questions or need more clarification (phone: 404-639-3091).

Also, we do not claim that "no-one has ever been infected with prion disease from eating venison." Our conclusion stating that we found no strong evidence of CWD transmission to humans in the article you quoted or in any other forum is limited to the patients we investigated.

Ermias Belay, M.D. Centers for Disease Control and Prevention

Subject: TO CDC AND NIH - PUB MED- 3 MORE DEATHS - CWD - YOUNG HUNTERS

Sunday, November 10, 2002 6:26 PM .....snip.....end.....TSS

-----Original Message-----

From:

Sent: Sunday, September 29, 2002 10:15 AM

To: rr26k@nih.gov; rrace@niaid.nih.gov; ebb8@CDC.GOV

Subject: TO CDC AND NIH - PUB MED- 3 MORE DEATHS - CWD - YOUNG HUNTERS

Sunday, November 10, 2002 6:26 PM .....snip.....end.....TSS

Thursday, April 03, 2008

A prion disease of cervids: Chronic wasting disease 2008 1: Vet Res. 2008 Apr 3;39(4):41 A prion disease of cervids: Chronic wasting disease Sigurdson CJ.

snip...

*** twenty-seven CJD patients who regularly consumed venison were reported to the Surveillance Center, however there have been no unusual or novel prion subtypes that might indicate the appearance of a new prion strain [7, 41].

snip... full text ;

https://www.vetres.org/articles/vetres/abs/2008/04/v08092/v08092.html

https://chronic-wasting-disease.blogspot.com/2008/04/prion-disease-of-cervids-chronic.html

"regular venison eating associated with a 9 FOLD INCREASE IN RISK OF CJD"

Subject: Re: DEER SPONGIFORM ENCEPHALOPATHY SURVEY & HOUND STUDY

Date: Fri, 18 Oct 2002 23:12:22 +0100

From: Steve Dealler Reply-To: Bovine Spongiform Encephalopathy Organization: Netscape Online member

To: BSE-L@ …

######## Bovine Spongiform Encephalopathy <BSE-L@UNI-KARLSRUHE.DE> #########

Dear Terry,

An excellent piece of review as this literature is desparately difficult to get back from Government sites. What happened with the deer was that an association between deer meat eating and sporadic CJD was found in about 1993. The evidence was not great but did not disappear after several years of asking CJD cases what they had eaten. I think that the work into deer disease largely stopped because it was not helpful to the UK industry...and no specific cases were reported.

Well, if you dont look adequately like they are in USA currenly then you wont find any!

Steve Dealler

########## http://mailhost.rz.uni-karlsruhe.de/warc/bse-l.html ############

Subject: DEER SPONGIFORM ENCEPHALOPATHY SURVEY & HOUND STUDY

From: "Terry S. Singeltary Sr." <flounder@WT.NET>

Reply To: Bovine Spongiform Encephalopathy <BSE-L@UNI-KARLSRUHE.DE>

Date: Thu, 17 Oct 2002 17:04:51 -0700

snip...

"The association between venison eating and risk of CJD shows similar pattern, with regular venison eating associated with a 9 FOLD INCREASE IN RISK OF CJD (p = 0.04)."

CREUTZFELDT JAKOB DISEASE SURVEILLANCE IN THE UNITED KINGDOM THIRD ANNUAL REPORT AUGUST 1994

snip...see full report ;

http://web.archive.org/web/20090506050043/http://www.bseinquiry.gov.uk/files/yb/1994/08/00004001.pdf

http://web.archive.org/web/20090506050007/http://www.bseinquiry.gov.uk/files/yb/1994/10/00003001.pdf

http://web.archive.org/web/20090506050244/http://www.bseinquiry.gov.uk/files/yb/1994/07/00001001.pdf

Stephen Dealler is a consultant medical microbiologist deal@airtime.co.uk BSE Inquiry Steve Dealler Management In Confidence BSE: Private Submission of Bovine Brain Dealler

snip...end

########## http://mailhost.rz.uni-karlsruhe.de/warc/bse-l.html ############

*** These results would seem to suggest that CWD does indeed have zoonotic potential, at least as judged by the compatibility of CWD prions and their human PrPC target. Furthermore, extrapolation from this simple in vitro assay suggests that if zoonotic CWD occurred, it would most likely effect those of the PRNP codon 129-MM genotype and that the PrPres type would be similar to that found in the most common subtype of sCJD (MM1).***

http://www.tandfonline.com/doi/full/10.4161/pri.28124?src=recsys

http://www.tandfonline.com/doi/pdf/10.4161/pri.28124?needAccess=true

https://wwwnc.cdc.gov/eid/article/20/1/13-0858_article

Two Hunters from the Same Lodge Afflicted with Sporadic CJD: Is Chronic Wasting Disease to Blame?

(P7-13.002) Jonathan Trout, Matthew Roberts, Michel Tabet, Eithan Kotkowski, and Sarah HornAUTHORS INFO & AFFILIATIONS April 9, 2024 issue 102 (17_supplement_1) https://doi.org/10.1212/WNL.0000000000204407

Abstract Publication History Information & Authors Metrics & Citations Share Abstract

Objective:

This study presents a cluster of Creutzfeldt-Jakob disease (CJD) cases after exposure to chronic wasting disease (CWD)-infected deer, suggestive of potential prion transmission from CWD-infected deer to humans.

Background:

CJD is a rapidly progressive central nervous system disorder caused by misfolded prion proteins. CWD, a prion disease prevalent in North American deer, has raised concerns due to its possible link to CJD. Although no conclusive evidence of cross-species prion transmission exists, vigilance for such cases is crucial for public health.

Design/Methods:

Not applicable.

Results:

In 2022, a 72-year-old man with a history of consuming meat from a CWD-infected deer population presented with rapid-onset confusion and aggression. His friend, who had also eaten venison from the same deer population, recently died of CJD, raising concerns about a potential link between CWD and human prion disease. Despite aggressive symptomatic treatment of seizures and agitation, the patient's condition deteriorated and he died within a month of initial presentation. The diagnosis was confirmed postmortem as sporadic CJD with homozygous methionine at codon 129 (sCJDMM1). The patient's history, including a similar case in his social group, suggests a possible novel animal-to-human transmission of CWD. Based on non-human primate and mouse models, cross-species transmission of CJD is plausible. Due to the challenge of distinguishing sCJDMM1 from CWD without detailed prion protein characterization, it is not possible to definitively rule out CWD in these cases. Although causation remains unproven, this cluster emphasizes the need for further investigation into the potential risks of consuming CWD-infected deer and its implications for public health.

Conclusions:

Clusters of sporadic CJD cases may occur in regions with CWD-confirmed deer populations, hinting at potential cross-species prion transmission. Surveillance and further research are essential to better understand this possible association.

Disclosure: Mr. Trout has nothing to disclose. Dr. Roberts has nothing to disclose. Dr. Tabet has nothing to disclose. Dr. Kotkowski has nothing to disclose. Dr. Horn has received personal compensation in the range of $500-$4,999 for serving as a Consultant for Cala Trio. The institution of Dr. Horn has received research support from Alzheimer's Association.

https://www.neurology.org/doi/abs/10.1212/WNL.0000000000204407

TUESDAY, MAY 11, 2021

A Unique Presentation of Creutzfeldt-Jakob Disease in a Patient Consuming Deer Antler Velvet

Conclusion

We believe that our patient's case of CJD is highly suspicious for cervid etiology given the circumstances of the case as well as the strong evidence of plausibility reported in published literature. This is the first known case of CJD in a patient who had consumed deer antler velvet. Despite the confirmed diagnosis of CJD, a causal relationship between the patient's disease and his consumption of deer antler velvet cannot be definitively concluded.

Supplemental data including molecular tissue sample analysis and autopsy findings could yield further supporting evidence. Given this patient's clinical resemblance to CBD and the known histological similarities of CBD with CJD, clinicians should consider both diseases in the differential diagnosis of patients with a similarly esoteric presentation. Regardless of the origin of this patient's disease, it is clear that the potential for prion transmission from cervids to humans should be further investigated by the academic community with considerable urgency.

https://thescipub.com/pdf/ajidsp.2021.43.48.pdf

"We believe that our patient's case of CJD is highly suspicious for cervid etiology given the circumstances of the case as well as the strong evidence of plausibility reported in published literature. This is the first known case of CJD in a patient who had consumed deer antler velvet. Despite the confirmed diagnosis of CJD, a causal relationship between the patient's disease and his consumption of deer antler velvet cannot be definitively concluded."

https://thescipub.com/pdf/ajidsp.2021.43.48.pdf

CREUTZFELDT JAKOB DISEASE: A Unique Presentation of Creutzfeldt-Jakob Disease in a Patient Consuming Deer Antler Velvet

i was warning England and the BSE Inquiry about just this, way back in 1998, and was ask to supply information to the BSE Inquiry. for anyone that might be interested, see;

Singeltary submission to the BSE Inquiry on CJD and Nutritional Supplements 1998

ABOUT that deer antler spray and CWD TSE PRION... I have been screaming this since my neighbors mom died from cjd, and she had been taking a supplement that contained bovine brain, bovine eyeball, and other SRMs specified risk materials, the most high risk for mad cow disease. just saying...

I made a submission to the BSE Inquiry long ago during the BSE Inquiry days, and they seemed pretty interested.

Sender: "Patricia Cantos"

To: "Terry S Singeltary Sr. (E-mail)"

Subject: Your submission to the Inquiry

Date: Fri, 3 Jul 1998 10:10:05 +0100 3 July 1998

Mr Terry S Singeltary Sr. E-Mail: Flounder at wt.netRef: E2979

Dear Mr Singeltary, Thank you for your E-mail message of the 30th of June 1998 providing the Inquiry with your further comments. Thank you for offering to provide the Inquiry with any test results on the nutritional supplements your mother was taking before she died. As requested I am sending you our general Information Pack and a copy of the Chairman's letter. Please contact me if your system cannot read the attachments. Regarding your question, the Inquiry is looking into many aspects of the scientific evidence on BSE and nvCJD.

I would refer you to the transcripts of evidence we have already heard which are found on our internet site at ;

http://www.bse.org.uk.

Could you please provide the Inquiry with a copy of the press article you refer to in your e-mail? If not an approximate date for the article so that we can locate it? In the meantime, thank you for you comments. Please do not hesitate to contact me on...
snip...end...tss

everyone I tell this too gets it screwed up...MY MOTHER WAS NOT TAKING THOSE SUPPLEMENTS IPLEX (that I ever knew of). this was my neighbors mother that died exactly one year previously and to the day of sporadic CJD that was diagnosed as Alzheimer's at first. my mother died exactly a year later from the Heidenhain Variant of Creutzfeldt Jakob Disease hvCJD, and exceedingly rare strains of the ever growing sporadic CJD's. both cases confirmed. ...kind regards, terry

So, this is what we leave our children and grandchildren?

2001 Singeltary on CJD, Journal of American Medical Association

February 14, 2001

Diagnosis and Reporting of Creutzfeldt-Jakob Disease

Terry S. Singeltary, Sr

Author Affiliations

JAMA. 2001;285(6):733-734. doi:10-1001/pubs.JAMA-ISSN-0098-7484-285-6-jlt0214

To the Editor: In their Research Letter, Dr Gibbons and colleagues1 reported that the annual US death rate due to Creutzfeldt-Jakob disease (CJD) has been stable since 1985. These estimates, however, are based only on reported cases, and do not include misdiagnosed or preclinical cases. It seems to me that misdiagnosis alone would drastically change these figures. An unknown number of persons with a diagnosis of Alzheimer disease in fact may have CJD, although only a small number of these patients receive the postmortem examination necessary to make this diagnosis. Furthermore, only a few states have made CJD reportable. Human and animal transmissible spongiform encephalopathies should be reportable nationwide and internationally.

February 14, 2001

Diagnosis and Reporting of Creutzfeldt-Jakob Disease

Terry S. Singeltary, Sr

Author Affiliations

JAMA. 2001;285(6):733-734. doi:10-1001/pubs.JAMA-ISSN-0098-7484-285-6-jlt0214

https://jamanetwork.com/journals/jama/article-abstract/1031186

RE-Monitoring the occurrence of emerging forms of Creutzfeldt-Jakob disease in the United States 2003 Singeltary Journal of Neurology

26 MARCH 2003

RE-Monitoring the occurrence of emerging forms of Creutzfeldt-Jakob disease in the United States

Terry S. Singeltary, retired (medically)

I lost my mother to hvCJD (Heidenhain Variant CJD). I would like to comment on the CDC's attempts to monitor the occurrence of emerging forms of CJD. Asante, Collinge et al [1] have reported that BSE transmission to the 129-methionine genotype can lead to an alternate phenotype that is indistinguishable from type 2 PrPSc, the commonest sporadic CJD. However, CJD and all human TSEs are not reportable nationally. CJD and all human TSEs must be made reportable in every state and internationally. I hope that the CDC does not continue to expect us to still believe that the 85%+ of all CJD cases which are sporadic are all spontaneous, without route/source. We have many TSEs in the USA in both animal and man. CWD in deer/elk is spreading rapidly and CWD does transmit to mink, ferret, cattle, and squirrel monkey by intracerebral inoculation. With the known incubation periods in other TSEs, oral transmission studies of CWD may take much longer. Every victim/family of CJD/TSEs should be asked about route and source of this agent. To prolong this will only spread the agent and needlessly expose others. In light of the findings of Asante and Collinge et al, there should be drastic measures to safeguard the medical and surgical arena from sporadic CJDs and all human TSEs. I only ponder how many sporadic CJDs in the USA are type 2 PrPSc?

https://www.neurology.org/doi/10.1212/01.WNL.0000036913.87823.D6

2023

https://creutzfeldt-jakob-disease.blogspot.com/2023/09/professor-john-collinge-on-tackling.html

is this what we leave our Children and Grandchildren?

Terry S. Singeltary Sr.

23 June 2026

Sporadic Creutzfeldt-Jakob disease following venison exposure in a chronic wasting disease-endemic region: a zoonotic surveillance perspective

Concern for zoonotic transmission arose after the neurology team learned that other individuals from the same hunting lodge had experienced similarly rare neurologic symptoms.

https://chronic-wasting-disease.blogspot.com/2026/07/sporadic-creutzfeldt-jakob-disease.html

https://prpsc.proboards.com/thread/223/scjd-venison-exposure-cwd-region

Terry S. Singeltary Sr.

Sent from my iPad

Comments, DNR <dnrcomments@wv.gov>

Re: Public comment related to CWD management

1 message

[redacted] Mon, Jul 27, 2026 at 11:46 AM

Good Morning Eric,

The West Virginia Division of Natural Resources would like to thank you for submitting your public comment for the proposed rule changes to 58CSR69 – Wildlife Disease Management. Your comment submitted in opposition to the proposed rule changes will be recorded for public record.

I would also like to share the following information with you from the agency's Wildlife Disease Specialist regarding risks of feeder use versus food plots.

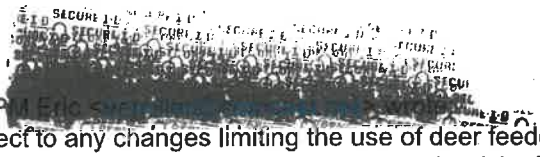
The most current available science indicates that feeders and food plots do not concentrate deer in the same way and thus do not present the same degree of risk of spreading disease. Two insightful peer-reviewed scientific papers have recently been published in the Journal of Wildlife Management, and both reach the same general conclusion: risks posed by deer feeders and by food plots are not the same. In the first publication, Huang et al. (2024) examined weekly deer visitation rates between feeders, food plots, and mast trees with visitation to feeders being more frequent than visitation to the other two. Additionally, deer to deer contact rates were higher at feeder sites than in food plots or at mast trees. The authors concluded that supplemental feeding of deer using feeders increased the risk of exposure to CWD due to a combination of feeder contamination, increased visitation, and increased deer-to-deer contact rates. Furthermore, feeders also contribute to unnatural contact, whether direct or indirect, between wildlife species such as deer and raccoons, which could have additional disease transmission risk implications (i.e., cross-species transmission risks, rabies, etc.). These results lend support to the restriction on feeding and baiting deer where CWD has been detected. Although in this publication food plots appeared to concentrate deer more than a natural stand of oak trees, concentration of deer around feeders was 2-fold higher than at food plots. In other words, the risks posed are not the same and are not interchangeable; while risk may be marginally elevated by food plots, this risk remains substantially less than that posed by a point source like a feeder. The environmental durability of prions also means that even following the removal of a feeder (or food from the feeder, leaving it empty), prion contamination and thus disease transmission potential persists and is spatially concentrated around a single point- the feeder- instead of being dispersed across a larger area as in a food plot, agricultural field, or forest.

The second peer-reviewed paper, published by Courtney et al. (2025), examined deer behaviors at baited sites as compared to food plots and natural foraging areas on the landscape to assess disease transmission risks. All contact types potentially linked to CWD transmission- direct (i.e., deer to deer), indirect (i.e., environmental), and self (e.g., grooming in which a deer could ingest contaminated soil) contacts- were all substantially more common at bait sites than food plots or in natural foraging areas. Additionally, the probability of encountering deer feces, known to be capable of containing infectious prions, was highest at bait sites and was also more concentrated in a central location. Deer are known to exhibit fidelity to locations where they have foraging success, and when these areas are very small or are individual points (as in the case of a feeder or a bait pile), repeat visitation likely carries enhanced risk of disease transmission. The authors, as in Huang et al. (2024), conclude that restrictions on baiting and/or feeding are advisable to reduce the risk of spreading CWD based on their results.

Please note that both publications are open access and you can find and read them free of charge using Google Scholar or another such search engine or database. While we understand your concerns relating to your own hunting, scientific evidence as summarized above suggests the risks posed by feeders or bait piles and food plots are not interchangeable: feeders and bait piles present distinctly higher risks of CWD transmission and concentration of both deer and potentially infectious materials.

It should also be noted that the proposed changes to 58CSR69 do not change the baiting and feeding restrictions that are currently in place in the CWD Containment Area. These proposed revisions do not prohibit feeding and baiting of deer statewide. The changes only clarify definitions and the language related to restrictions already in place applying only to the Containment Area. Whether or not you reside or hunt in the CWD Containment Area, the proposed changes would have no effect on your ability to conduct feeding or baiting. If you reside or hunt deer within the Containment Area, feeding and baiting would remain unlawful as they presently are under existing code. If you do not reside or hunt within the Containment Area, feeding and baiting of deer remains lawful to do (except on public lands).

We hope these comments have provided you with clarifying context and information. Thank you once again for submitting your comments to WVDNR regarding this proposed rule change.



On Thu, Jul 2, 2026 at 2:40 PM Edc [redacted]

> I would like to strongly object to any changes limiting the use of deer feeders unless food plots are also limited in the same way. Both concentrate animals and theoretically increase the risk of cwd. Not everyone has the money or the land and equipment required to manage food plots. Making deer feeders illegal would unfairly target lower income earners and small property owners and give those able to manage food plots the ability to draw deer off of properties that do not feature plots. Thank you for your consideration.
> Sent from my iPhone

Comments, DNR <dnrcomments@wv.gov>

Re: Deer feeding

1 message

Comments, [redacted] Mon, Jul 27, 2026 at 12:08 PM
To: Michael [redacted]

Good Afternoon Mr. Long,

The West Virginia Division of Natural Resources would like to thank you for submitting your public comment for the proposed rule changes to 58CSR69 – Wildlife Disease Management. Your comment submitted in opposition to the proposed rule changes will be recorded for public record.

We would like to share the following information related to deer forage in hopes of alleviating some of your concerns. Deer could not currently survive in urban environments if food wasn't available. They cannot live solely on corn and bird seed. Deer forage primarily consists of leaves, woody stems, forbs, and hard and soft mast. Annually deer consume over 100 different plant species and areas around residences generally have higher plant species richness compared to forested areas (Swihart et al 1995). Urban and suburban areas provide the necessary browse, forbs, and hard and soft mast that deer need. Suburban areas provide high quality habitat in the form of gardens, ornamental planting, and fertilized lawns (DeNicola et al.2000).

We hope these comments have provided you with clarifying context and information. Thank you once again for submitting your comments to WVDNR regarding this proposed rule change.

On Fri, Jul 3, 2026 at 9:37 AM Michael Long [redacted] wrote:

> So what do you propose the deer in town do when there is no forage for them to eat. Another overreaching government entity trying to tell me what I can do on my property. Don't Tread on Me

Comments, DNR <dnrcomments@wv.gov>

Re: CWD and restricted feeding proposal

1 message

Comments, DNR <dnrcomments@wv.gov>
To: RPaul Bennett

Mon, Jul 27, 2026 at 12:17 PM

Good Afternoon Mr. Bennett ,

The West Virginia Division of Natural Resources would like to thank you for submitting your public comment for the proposed rule changes to 58CSR69 – Wildlife Disease Management. Your comment submitted in opposition to the proposed rule changes will be recorded for public record.

I would like to take a moment to clarify what appears to be a misunderstanding of the proposed rule changes and has resulted in your opposition. The proposed changes to 58CSR69 does not establish a statewide baiting or feeding ban. The changes only clarify definitions and the language related to restrictions already in place applying only to the Containment Area. Whether or not you reside or hunt in the CWD Containment Area, the proposed changes would have no effect on your ability to conduct feeding or baiting. If you reside or hunt deer within the Containment Area, feeding and baiting would remain unlawful as they presently are under existing code. If you do not reside or hunt within the Containment Area, feeding and baiting of deer remains lawful to do (except on public lands).

We hope these comments have provided you with clarifying context and information. Thank you once again for submitting your comments to WVDNR regarding this proposed rule change.

On Thu, Jul 9, 2026 at 6:57 AM RPaul Bennett

> There are only 8 counties in WV with reported CWD cases …all counties localized to eastern area. Only one new reported area since 2023. I think that a total state mandated restriction of feeding is not warranted. I live in Webster County where Mast crops have been spotty over last few years…I have no doubt that feeding has saved many deer from starvation thru our very cold recent winters. I think that this idea would cause many hunters would reconsider hunting and lead to more deer increasing likihood of overpopulation and increased risk of CWD….the financial cost to WV would be significant with less hunters and licenses.
>
> Many of the ideas suggested by the DNR have had merit but this is not one of them in my opinion
>
> Respectfully, R. Paul Bennett
>
> Sent from Yahoo Mail for iPad

Comments, DNR <dnrcomments@wv.gov>

Re: Attn: Wendy Greene

1 message

Comments, DNR <dnrcomments@wv.gov> Mon, Jul 27, 2026 at 12:19 PM
To: Helanna [redacted]

Good Afternoon Ms. Nicol,

The West Virginia Division of Natural Resources would like to thank you for submitting your public comment for the proposed rule changes to 58CSR69 – Wildlife Disease Management. Your comment submitted in support of the proposed rule changes will be recorded for public record.

On Fri, Jul 10, 2026 at 1:00 PM Helanna Nicol [redacted]

July 10, 2026

Dear Deputy Director Greene,

I went hunting for the first time last year. I was forty-two years old. During early doe season I harvested my first doe while hunting with my significant other and my son. In the late doe season in December 2025, I went hunting again with my significant other. We did not use a hunting blind. We did not hunt over a bait pile. I missed a deer 25 yards in front of me, but I learned so much from that experience. I can't wait for the 2026 antlerless deer seasons!

I know there are many opinions on the topic of baiting and Chronic Wasting Disease. I know that Chronic Wasting Disease has proved to be quite fatal in Hampshire County. Now it seems that the Chronic Wasting Disease containment area has been expanded to Berkeley, Grant, Hardy, Jefferson, Mineral, and Morgan; that is the entire eastern panhandle of West Virginia!

According to the USDA's National Wildlife Research Center, Chronic Wasting Disease is spread through the bodily fluids of cervids. Chronic Wasting Disease is spread through urine, feces, saliva, and blood. Bait piles are petri dishes for Chronic Wasting Disease; unfortunately, we are not in a laboratory setting where the disease or specimen can be fully contained, so we have to use the best options at our disposal to try to slow it down.. Deer do not use discretion when they eat, defecate, and mark their territory… imagine the movie Outbreak! Research has shown there is a difference between a bait pile or feeder and the natural foods in the woods that are more dispersed when it comes to deer interacting with each other. Baiting and feeding are a risk to the health of the deer herd where CWD has been found, so I think it is sensible to restrict these things in those counties. You don't need a bait pile to hunt deer, there are plenty of other ways to have success without baiting.

As a child I can remember my dad hunting and sharing stories about hunting during his youth growing up in Grant County, where I also grew up on the same farm. I also remember my dad talking about the dismal deer population numbers in Grant County during the 1960s and 1970s. Hunting is part of our culture and heritage; it is a way of life for a lot of residents who live in West Virginia and venison is an important food to many West Virginians. Hunting allows us to preserve our proud cultural heritage as West Virginians. Providing for healthy deer herds with minimal disease is one of the best ways to protect the future of hunting in our state. I am asking our DNR commissioners and legislators to approve these

new regulations to slow the spread of diseases to help keep a part of heritage viable for hunters today and future hunters.

Sincerely,

Helanna Nicol

Comments, DNR <dnrcomments@wv.gov>

Re: Against banning baiting proposal

1 message

Comments, DNR <dnrcomments@wv.gov> Wed, Jul 29, 2026 at 10:34 AM
To: Carl Ringer

Dear Mr. Ringer,

Thank you for submitting your comments to the West Virginia Division of Natural Resources regarding the proposed 58CSR69, Wildlife Disease Management Rule.

Since your comment seemed to refer to the entire state, I want to clarify what may be a misunderstanding. The proposed changes to this rule are not changing the areas where baiting or feeding are currently restricted and currently permitted. If you currently live or hunt in an area of West Virginia where baiting is allowed, these proposed revisions do not affect you. If you live or hunt in an area where special regulations to reduce the human-facilitated spread of chronic wasting disease (CWD) are already in place, the revisions do not change that designated area. The intent of the proposed revision to this rule is to clarify language used in the definitions of baiting and feeding so the regulations are clearer and easier to understand. The proposed revision is not a proposal for a statewide ban on feeding or baiting deer. The counties in which baiting and feeding restrictions are currently in effect are Berkeley, Grant, Hampshire, Hardy, Jefferson, Mineral, Morgan, and Pendleton. The only statewide baiting restriction is a ban on baiting and feeding on public lands across the state.

At present, there is sound scientific support for restricting feeding and baiting - including mineral attractants - of deer where CWD is known to occur. Feeding and baiting, including the use of mineral attractants, cause deer to congregate in an unnatural manner at a single point. This unnatural congregation increases the risk of concentrated environmental contamination with infectious material and a greatly increased rate of close interactions among deer social or matrilineal family units that would not otherwise interact with any great frequency. In a peer-reviewed scientific publication from 2018, Plummer et al. found widespread CWD contamination at artificial mineral lick sites in Wisconsin. These mineral sites thus represent concentrated CWD exposure risks to any deer visiting the site and increased risks for cross-species exposure when livestock are attracted to those artificial mineral licks. Further, because minerals leach into the soil, removal of the mineral block is not adequate to immediately stop congregation of deer at that location; mineral blocks may thus represent one of the highest-risk forms of feeding and baiting. However, feeding and baiting with corn or other substances also carry substantial risk to deer.

In 2025, peer-reviewed publications by Huang et al. and Courtney et al. investigated the CWD transmission risks associated with feeding and baiting. Huang et al. found both visitation rates and deer to deer contact rates were substantially higher at feeders than in food plots or at naturally occurring oak mast trees and detected CWD accumulation at all feeder sites within 12 weeks of setup even in areas of suspected low disease prevalence. Courtney et al. found rates of three contact types (deer to deer, self, and environmental) related to disease transmission risks were highest at bait sites when compared to food plots and natural feeding areas. Deer observations were substantially more common at bait sites, as was the observation of fecal pellets in a concentrated area around the bait site. In summary, the risks posed by artificial feeding and baiting by humans substantially increases risks of CWD transmission through increased contact rates, environmental contamination with infectious material, and increased frequency of deer to deer contact outside of normal white-tailed deer social or matrilineal family units. The above referenced scientific publications are open access and are available to you free of charge via databases such

as Google Scholar should you wish to read them. The contention that restricting feeding and baiting has had "no effect" on slowing the spread of or increase in prevalence of CWD in other states is an unsubstantiated claim.

Chronic wasting disease represents a substantial threat to the long-term health and sustainability of free-ranging deer in areas where this disease occurs. Several studies in other states (e.g., Arkansas [Gaya et al. 2026], Colorado [Miller et al. 2008], Wyoming [Edmunds et al. 2016]) have found evidence of declining deer populations in areas where CWD has become endemic. West Virginia recently completed its own deer study (2021-26) involving three study areas, with CWD known to occur in only one of these areas: Hampshire County. Deer survival was significantly lower in Hampshire County than in the Barbour/Upshur and Mason/Jackson study areas, and end-stage CWD was the most common cause of mortality in Hampshire County. In the other study areas, hunter harvest was the leading cause of deer mortality. Furthermore, predation mortality of deer in Hampshire County was substantially higher than in the other two study areas, and many of the predator-killed Hampshire County deer were late-stage CWD positive animals thus suggesting the role of CWD in causing deer mortality is not strictly limited to the disease process itself. A similar phenomenon was documented by Miller et al. (2008) in Colorado mule deer, in which CWD positive animals were at far greater risk of predation than healthy deer.

The contention that "deer immune to CWD" are being released in Oklahoma is also unsubstantiated. It is true that several captive deer purportedly "less susceptible" to CWD have been released in Oklahoma. However, no genotype of deer has been found to be fully resistant or "immune" to CWD, and while genotypes may play a role in likelihood of infection or expected duration of survival post-infection, all known genotypes of white-tailed deer are susceptible to CWD. Further, the captive deer being released in Oklahoma have never been challenged with CWD to determine to what extent they may be "resistant" to disease. There are additional dangers in releasing such animals that have not been adequately studied using the best available science. It is possible highly genetically modified deer may induce changes in the current form of the infectious prion and thus increase the potential for the prion to cross species barriers. This is not speculation: prions are known to be capable of mutating in animals with atypical genotypes encoding for production of the normal cellular prion protein or following passaging through alternate hosts (e.g., Otero et al. 2023). Aside from the potential for unintended consequences of releasing these captive deer, the vast number of deer that would need to be released to make a significant effect on the frequency of deer Prnp genotypes at any meaningful scale makes this approach dubious, at best. At present, there seems to be far greater risk of net harm than net benefit stemming from this risk-laden approach which has not been properly validated using sound science.
The likelihood that restricting feeding and baiting in some areas of the state in response to the detection of CWD will "ruin the deer herd" is extraordinarily low. Carrying capacity for deer is predominantly dictated by the availability of naturally occurring food on the landscape.

Similarly, multiple U.S. states continue to boast large numbers of deer hunters despite restrictions on baiting, whether these restrictions are long-standing cultural norms engrained in the hunting community across the state or are more recent, localized regulations in response to disease detection. While there are numerous barriers to recruiting youth hunters that have been identified in several decades of study on this issue, the lack of ability to use bait for hunting has never been listed among the top-ranking barriers for youth hunter recruitment. Both deer and deer hunting are culturally, economically, and historically important to West Virginia and West Virginians. We are pleased you share a common value in our state's treasured deer resource and appreciate you taking the time to submit comments on the subject. We hope this additional information will be useful to you and will encourage you to conduct your own further research using reliable, peer-reviewed scientific studies on the subject of CWD, its effects on public trust cervid populations, and the likely role played by baiting and feeding in exacerbating outbreaks of CWD. The Division of Natural Resources is tasked with managing a sustainable population of white-tailed deer for

the benefit of all West Virginians, and CWD is a threat to the sustainability of those populations. Proposed regulation changes to try to slow the
spread or increase in prevalence of CWD are always bound to be controversial, but doing nothing and allowing disease to spread unabated is not an acceptable approach. We will continue to monitor ongoing scientific research and will continue to base any further recommendations for regulation changes on sound scientific guidance.

On Wed, Jul 15, 2026 at 10:47 PM Carl Ringer [redacted]

> I am highly against this proposal of (banning baiting in WV)!!! I have hunted several other states that have banned baiting. And absolutely nothing has changed with chronic wasting disease in there state from not baiting. I can't even believe WV is considering this and I am very disappointed in my state for considering this.
> I have watched other states wipe out all most there entire deer herds to supposedly prevent chronic wasting disease and for what? Oklahoma is the only state that is now trying to introduce deer that is immune to chronic wasting disease to those affected areas. Maybe try a positive approach instead of a negative approach that many states have tried unsuccessfully and absolutely ruined deer herds and a chance for our youth to get into hunting. Please reconsider this stupid proposal.

Comments, DNR <dnrcomments@wv.gov>

Re: Wildlife disease rules affecting deer feeding

1 message

Comments, DN 
To: Dwayne Hu

Wed, Jul 29, 2026 at 3:33 PM

Good Afternoon Mr. Hunter.

Thank you for submitting your comments to the West Virginia Division of Natural Resources regarding the proposed 58CSR69, Wildlife Disease Management Rule. Your comments will be recorded for public record.

This rule establishes regulations intended to reduce the role of humans in spreading chronic wasting disease (CWD) across the landscape and artificially increasing contact rates with both other deer and infectious material. The presence of CWD is caused by the movement of live deer and deer carcasses by humans. Carcass transport restrictions prevent the movement of high-risk parts out of areas where CWD is already known to occur. Improper disposal of these parts in areas where CWD is not known to occur may lead to deposition of infectious material on the landscape and thus potential transmission of CWD to cervids in those areas. Scientific evidence suggests environmental contamination with infectious material is sufficient to transmit CWD to healthy deer. Peer-reviewed publications by Miller et al. (2004) and Mathiason et al. (2009) demonstrate both decayed carcasses of CWD-positive animals and soil or surfaces contaminated by shedding of infectious material in saliva, urine, and feces can infect healthy deer. Further research by Grunklee et al. (2025) found environmental contamination with CWD caused by unlawful disposal of deer carcasses, demonstrating the risk for ongoing CWD transmission from environmental sources and carcass parts.

Feeding and baiting, including the use of mineral attractants, serve to congregate deer in an unnatural manner at a single point. This increases the risk of concentrated environmental contamination with infectious material and close interactions among deer social or matrilineal family units that would not otherwise interact with any great frequency. In a peer-reviewed scientific publication from 2018, Plummer et al. found widespread CWD contamination at
artificial mineral lick sites in Wisconsin. These mineral sites thus represent concentrated CWD exposure risks to any deer visiting the site, and also increased risks for cross-species exposure when livestock are attracted to those artificial mineral licks. Further, because minerals leach into the soil, removal of the mineral block is not adequate to immediately stop congregation of deer at that location; mineral blocks may thus represent one of the highest-risk forms of feeding and baiting. However, feeding and baiting with corn or other substances also carry substantial risk to deer.

In 2025, peer-reviewed publications by Huang et al. and Courtney et al. investigated the CWD transmission risks associated with feeding and baiting. Huang et al. found both visitation rates and deer to deer contact rates were substantially higher at feeders than in food plots or at naturally occurring oak mast trees and detected CWD accumulation at all feeder sites within 12 weeks of setup even in areas of suspected low disease prevalence. Courtney et al. found rates of three contact types (deer to deer, self, and environmental) related to disease transmission risks were highest at bait sites when compared to food plots and natural feeding areas. Deer observations were substantially more common at bait sites, as was the observation of fecal pellets in a concentrated area around the bait site. In summary, the risks posed by artificial feeding and baiting by humans substantially increases risks of CWD transmission through increased contact rates, environmental contamination with infectious material, and increased frequency of deer to deer contact outside of normal white-tailed deer social or matrilineal family units. The above

referenced scientific publications are open access and are available to you free of charge via databases such as Google Scholar should you wish to read them.

It is worth mentioning that CWD is not the only disease or parasite transmission risk posed by feeding and baiting. While the Wildlife Disease Management Rule is specifically intended, with respect to restrictions on feeding and baiting, to reduce human-associated risks of transmitting disease, elevated transmission risks for numerous other diseases and parasites of deer are associated with feeders, bait sites, and mineral blocks. Feeding can also increase environmental damage through browsing and preferential selection of plants by deer in proximity to feeders (e.g., Donier et al. 1997, Williamson 2000, Cooper et al. 2002). Further, because corn sold as "deer corn" is not subject to the same rigorous inspection standards as corn intended for use as human or livestock food, contamination of "deer corn" with fungal toxins such as aflatoxin is a relatively common finding. In some cases, contamination is so substantial that such "deer corn" would not be acceptable under U.S. Food and Drug Administration (FDA) guidelines to feed to any livestock. In 1998, Brown and Cooper (2006) found 40 of 100 bags of randomly selected, commercially available "deer corn" contained such levels of aflatoxin. Birds, including ruffed grouse and wild turkeys, are highly susceptible to acute toxicosis and death when exposed to some fungal toxins including aflatoxin, and widespread distribution of "deer corn" across the landscape may constitute a population health concern for these highly valued game birds.

In summary, feeding and baiting - while commonly conducted in West Virginia - is not a "zero risk" activity and is not exclusively beneficial to wildlife health. In fact, there are numerous examples of wildlife health risks posed by feeding and baiting and these are relatively well documented in peer-reviewed scientific publications authored by wildlife biologists and researchers. Wildlife in West Virginia is managed under the auspices of rigorous and objective scientific study, not strictly based on opinions or observations. As such, scientific evidence supports restricting feeding and baiting in areas in proximity of where CWD is known to occur among free-ranging deer to support the long-term viability of the deer population in the face of disease pressure. You should note that the proposed revisions to the Wildlife Disease Management Rule do not actually expand restrictions on feeding or baiting in the state but rather clarify language in the rule for the benefit of the public and law enforcement.

Both deer and deer hunting are culturally, economically, and historically important to West Virginia and West Virginians. We are pleased you share a common value in our state's treasured deer resource and appreciate you taking the time to submit comments on the subject.

On Sun, Jul 26, 2026 at 8:30 PM Dwayne Hunte[redacted]

> Hi,
>
> I have been hunting Ohio, West Virginia, Pennsylvania and Kentucky for many years. My experience tells me everywhere chronic wasting disease is present is in over populated areas of those states. I don't see minerals in a field or artificial feed on property as causing the issue but only helping create a healthy population that needs to be managed properly.
>
> Sincerely,
> Dwayne Hunter