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

[Federal Register Volume 86, Number 42 (Friday, March 5, 2021)]

[Notices]

[Pages 12901-12902]

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DEPARTMENT OF AGRICULTURE

Animal and Plant Health Inspection Service

[Docket No. APHIS-2021-0004]

Notice of Request for Revision to and Extension of Approval of an Information Collection; Control of Chronic Wasting Disease

AGENCY: Animal and Plant Health Inspection Service, USDA.

ACTION: Revision to and extension of approval of an information collection; comment request.

SUMMARY: In accordance with the Paperwork Reduction Act of 1995, this notice announces the Animal and Plant Health Inspection Service's intention to request a revision to and extension of approval of an information collection associated with the regulations for the control of chronic wasting disease in farmed and captive cervid herds.

DATES: We will consider all comments that we receive on or before May 4, 2021.

ADDRESSES: You may submit comments by either of the following methods: Federal eRulemaking Portal: Go to www.regulations.gov.

Enter APHIS-2021-0004 in the Search field. Select the Documents tab, then select the Comment button in the list of documents.

Postal Mail/Commercial Delivery: Send your comment to Docket No. APHIS-2021-0004, Regulatory Analysis and Development, PPD, APHIS, Station 3A-03.8, 4700 River Road Unit 118, Riverdale, MD 20737- 1238.

[[Page 12902]]

Supporting documents and any comments we receive on this docket may be viewed at regulations.gov or in our reading room, which is located in room 1620 of the USDA South Building, 14th Street and Independence Avenue SW, Washington, DC. Normal reading room hours are 8 a.m. to 4:30 p.m., Monday through Friday, except holidays. To be sure someone is there to help you, please call (202) 799-7039 before coming.

FOR FURTHER INFORMATION CONTACT: For information on the regulations related to the control of chronic wasting disease in farmed or captive cervid herds, contact Dr. Jennifer L. Siembieda, Ruminant Health Center (Cervid Health), Strategy and Policy, Veterinary Services, 2150 Centre Ave, Building B, MS 2E6, Fort Collins, CO 80526-8117; (970) 494-7412; Jennifer.L.Siembieda@usda.gov. For more detailed information on the information collection, contact Mr. Joseph Moxey, APHIS' Information Collection Coordinator, at (301) 851-2533.

SUPPLEMENTARY INFORMATION:

Title: Control of Chronic Wasting Disease.

OMB Control Number: 0579-0189.

Type of Request: Revision to and extension of approval of an information collection.

Abstract: Under the Animal Health Protection Act (7 U.S.C. 8301 et seq.), the Animal and Plant Health Inspection Service (APHIS) of the U.S. Department of Agriculture is authorized, among other things, to protect the health of the United States' livestock and poultry populations by preventing the introduction and interstate spread of serious diseases and pests of livestock and for eradicating such diseases from the United States when feasible.

Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy of cervids (elk, deer, and moose) typified by chronic weight loss leading to death. The presence of CWD in cervids causes significant economic and market losses to U.S. producers. In an effort to control and limit the spread of this disease in the United States, APHIS created a cooperative, voluntary Federal-State-private sector CWD Herd Certification Program designed to identify farmed or captive herds infected with CWD. The program is designed to identify farmed or captive herds infected with CWD and provide for the management of these herds in a way that will reduce the risk of spreading CWD. APHIS' Veterinary Services manages the CWD Herd Certification Program.

Owners of farmed or captive elk, deer, and moose herds who choose to participate in the Herd Certification Program need to follow program requirements for animal identification, testing, herd management, and movement of animals into and from herds. The regulations for this program are in 9 CFR part 55. Part 55 also contains the regulations that authorize the payment of indemnity for the voluntary depopulation of CWD-positive, CWD-exposed, or CWD-suspect captive cervids. APHIS also established requirements in 9 CFR part 81 for the interstate movement of deer, elk, and moose to prevent movement that could pose a risk of spreading CWD.

The Herd Certification Program and the indemnity program entail the use of information collection activities such as an APHIS Veterinary Services appraisal and indemnity claim form; sample collections and laboratory submissions, testing, and reporting; APHIS Veterinary Services State application for chronic wasting disease herd certification program approval, renewal, or reinstatement; memoranda of understanding between APHIS and participating States; herd or premises plans; annual reports; State reviews; epidemiological investigations and reporting of out-of-State traces to affected States; reports of cervid suspects, escapes, disappearances, and deaths; inspections and inventories; a letter to appeal suspension, cancellation, or change in status; farmed, captive, and wild cervid identification; interstate certificates of veterinary inspection; surveillance data; inspection reports; cooperative agreements; laboratory worksheets; and recordkeeping.

We are asking the Office of Management and Budget (OMB) to approve our use of these information collection activities, as described, for an additional 3 years.

The purpose of this notice is to solicit comments from the public (as well as affected agencies) concerning our information collection. These comments will help us:

(1) Evaluate whether the collection of information is necessary for the proper performance of the functions of the Agency, including whether the information will have practical utility;

(2) Evaluate the accuracy of our estimate of the burden of the collection of information, including the validity of the methodology and assumptions used;

(3) Enhance the quality, utility, and clarity of the information to be collected; and

(4) Minimize the burden of the collection of information on those who are to respond, through use, as appropriate, of automated, electronic, mechanical, and other collection technologies; e.g., permitting electronic submission of responses.

Estimate of burden: The public burden for this collection of information is estimated to average 4 hours per response.

Respondents: State animal health officials, laboratories, accredited veterinarians, and businesses managing farmed, captured, or wild cervid herds.

Estimated annual number of respondents: 9,053.

Estimated annual number of responses per respondent: 9.

Estimated annual number of responses: 78,128.

Estimated total annual burden on respondents: 322,546 hours. (Due to averaging, the total annual burden hours may not equal the product of the annual number of responses multiplied by the reporting burden per response.)

All responses to this notice will be summarized and included in the request for OMB approval. All comments will also become a matter of public record.

Done in Washington, DC, this 1st day of March 2021. Mark Davidson, Acting Administrator, Animal and Plant Health Inspection Service. [FR Doc. 2021-04511 Filed 3-4-21; 8:45 am] BILLING CODE 3410-34-P

https://www.govinfo.gov/content/pkg/FR-2021-03-05/html/2021-04511.htm

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.

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***> 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 ;

***> 1st and foremost your biggest problem is 'VOLUNTARY'!

"APHIS created a cooperative, voluntary Federal-State-private sector CWD Herd Certification Program designed to identify farmed or captive herds infected with CWD."

key word failure is 'voluntary'.

WE know for a fact now that voluntary does NOT WORK!

AS with the BSE 589.2001 FEED REGULATIONS (see , another colossal failure, and proven to be a sham, 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.
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Ryan Soulard, a wildlife biologist and privately-owned Cervidae coordinator for the DNR, noted that captive deer escaping into the wild is an all-too-common occurrence.

‘‘Last November [2017], over a dozen facilities reported losing deer,” Soulard said.

Keep in mind that’s just the number of facilities that reported losing deer in one month – not the number of deer they lost. Many times, facility owners/managers don’t know how many deer escape. Other times, facilities don’t know deer have gotten away or they choose to not report deer escapees over concerns of being penalized for losing animals.

Let’s take a closer look at the problem of deer permanently escaping from captive facilities. More specifically, we’ll consider recent trends in the number of captive deer escaping, the penalties for not reporting cervid escapees, what state officials are doing about the problem and what more can be done to keep farm-raised deer out of the wild...

http://www.michiganoutofdoors.com/desperado-deer-the-persistent-problem-of-captive-deer-running-wild/

TEXAS BREEDER DEER ESCAPEE WITH CWD IN THE WILD, or so the genetics would show?

OH NO, please tell me i heard this wrong, a potential Texas captive escapee with cwd in the wild, in an area with positive captive cwd herd?

apparently, no ID though. tell me it ain't so please...

23:00 minute mark

"Free Ranging Deer, Dr. Deyoung looked at Genetics of this free ranging deer and what he found was, that the genetics on this deer were more similar to captive deer, than the free ranging population, but he did not see a significant connection to any one captive facility that he analyzed, so we believe, Ahhhhhh, this animal had some captive ahhh, whatnot."

https://youtu.be/aoPDeGL6mpQ?t=1384

TEXAS CWD STRAIN

77. Assessing chronic wasting disease strain differences in free-ranging cervids across the United States

Kaitlyn M. Wagnera, Caitlin Ott-Connb, Kelly Strakab, Bob Dittmarc, Jasmine Battend, Robyn Piercea, Mercedes Hennessya, Elizabeth Gordona, Brett Israela, Jenn Ballarde and Mark D Zabela

aPrion Research Center at Colorado State University; bMichigan Department of Natural Resources; cTexas Parks and Wildlife Department; dMissouri Department of Conservation, 5. Arkansas Game and Fish Commission

CONTACT Kaitlyn M. Wagner miedkait@rams.colostate.edu

ABSTRACT

Background/Introduction: Chronic wasting disease (CWD) is an invariably fatal prion disease affecting captive and free-ranging cervids, including white-tailed deer, mule deer, moose, elk, and reindeer. Since the initial description of the disease in the 1960's, CWD has spread to 23 states, 3 Canadian Provinces, South Korea, Norway and, most recently, Finland. While some outbreaks of CWD were caused by transport of infected animals from endemic regions, the origin of CWD in other epizootics is unclear and has not been characterized. Previous studies have shown that there are two distinct strains of CWD. However, the continuous spread and the unclear origin of several outbreaks warrant continued surveillance and further characterization of strain diversity.

Materials and Methods: To address these knowledge gaps, we used biochemical tests to assess strain differences between CWD outbreaks in Michigan, Texas, Missouri, and Colorado, USA. Brain or lymph node samples were homogenized and digested in 50 µg/mL proteinase K (PK). These samples were then run on a Western blot to assess glycoform ratio and electrophoretic mobility. Texas samples were digested in 100 µg/mL PK. To assess conformational stability, brain or lymph node homogenates were incubated in increasing concentrations of guanidine hydrochloride from 0 M to 4 M in 0.5 M increments. Samples were then precipitated in methanol overnight, washed and PK digested in 50 µg/mL PK before slot blotting.

Results: Our results have found significant differences in glycoform ratio between CWD from Michigan and Colorado, but no differences were observed in conformational stability assays. Interestingly, when testing our CWD isolates from Texas to analyse electrophoretic mobility and glycoform ratio, we found that these samples did not exhibit the characteristic band shift when treated with PK, but PK resistant material remained. Additionally, results from our conformational stability assay demonstrate a unique profile of these Texas isolates. Testing of samples from Missouri is currently underway.

Conclusions: Thus far, our data indicate that there are strain differences between CWD circulating in Michigan and CWD in Colorado and provide important insight into CWD strain differences between two non-contiguous outbreaks. We have also identified a unique strain of CWD in Texas with biochemical strain properties not seen in any of our other CWD isolates. These results highlight the importance of continued surveillance to better understand this devastating disease. These results have important implications for CWD emergence, evolution and our understanding of prion strain heterogeneity on the landscape.

https://www.tandfonline.com/doi/full/10.1080/19336896.2019.1615197

SUNDAY, APRIL 14, 2019

Chronic Wasting Disease TSE Prion Strains everything in Texas is bigger, better, and badder

The disease devastating deer herds may also threaten human health

Scientists are exploring the origins of chronic wasting disease before it becomes truly catastrophic.

Rae Ellen Bichell

Image credit: David Parsons/Istock

April 8, 2019

Wagner and Zabel have suggested a possible answer: Perhaps, they say, there is not just one chronic wasting disease, but rather a bunch of different strains of it. And those different strains could be emerging at different times across the globe.

One day in late February, in their laboratory in Fort Collins, Colorado, Wagner and Zabel compared the prions from the brains of CWD-infected deer in Texas with those of elk in Colorado. They want to know if the proteins were all mangled in the same way, or not. “If they are different, this would suggest that we have different strain properties, which is evidence as we're building our case that we might have multiple strains of CWD circulating in the U.S.,” says Wagner.

Step one is to see if they’re equally easy to destroy using a chemical called guanidine. The shape of a prion dictates everything, including the way it interacts with an animal’s cells and the ease with which chemicals can unfold it.

‘‘Moment of truth,” said Wagner, as she and Zabel huddled around a computer, waiting for results to come through. When they did, Zabel was surprised.

‘‘Wow,” he said. “Unlike anything we've seen before.”

The prions from the Texas deer were a lot harder to destroy than the ones from the Colorado elk. In fact, the guanidine barely damaged them at all. “We’ve never seen that before in any prion strain, which means that it has a completely different structure than we've ever seen before,” says Zabel. And that suggests that it might be a very different kind of chronic wasting disease. The researchers ran the same test on another Texas deer, with the same results.

Now, these are only the preliminary results from a few animals. Wagner and Zabel have a lot more experiments to do. But if future tests come to the same conclusion, it would support their hypothesis that there are multiple strains of chronic wasting disease out there, all with different origins. That, in turn, could mean that this disease will become even trickier to manage than it already is.

And, Zabel adds, there’s something else. “If it's still evolving, it may still evolve into a form that could potentially, eventually affect humans,” he says.

Zabel is not the only one worried about that possibility.

OSTERHOLM, THE EPIDEMIOLOGIST from Minnesota, is also concerned. He directs the Center for Infectious Disease Research and Policy at the University of Minnesota, and is serving a one-year stint as a “Science Envoy for Health Security” with the U.S. State Department. In February, he told Minnesota lawmakers that when it comes to chronic wasting disease, we are playing with fire. “You are going to hear from people that this is not going to be a problem other than a game farm issue. You're going to hear from people that it's not going to transmit to people, and I hope they're right, but I wouldn't bet on it,” he said. “And if we lose this one and haven’t done all we can do, we will pay a price.”

If that wasn't warning enough, he added: "Just remember what happened in England."

https://www.hcn.org/articles/wildlife-the-disease-devastating-deer-herds-may-also-threaten-human-health-science/view

SUNDAY, APRIL 14, 2019

Chronic Wasting Disease TSE Prion Strains everything in Texas is bigger, better, and badder

https://chronic-wasting-disease.blogspot.com/2019/04/chronic-wasting-disease-tse-prion.html

Volume 23, Number 9—September 2017

Research Letter Chronic Wasting Disease Prion Strain Emergence and Host Range Expansion

***Thus, emergent CWD prion strains may have higher zoonotic potential than common strains.

https://wwwnc.cdc.gov/eid/article/23/9/16-1474_article

214. Identification of novel CWD strains

Debbie McKenziea, Camilo Duque Velasqueza, Allen Herbsta, Elizabeth Triscotta, Jacques van der Merwea, Samia Hannaouib, Leonardo Corteza, Sara Amidiana, Valerie Sima, Holger Willea, Hermann Schatzlb, Sabine Gilchb and Judd Aikena

aCentre for Prions and Protein Folding Diseases, University of Alberta, Edmonton, AB, Canada; bCalgary Prion Centre, University of Calgary, Calgary, AB, Canada

CONTACT Debbie McKenzie Debbie.mckenzie@ualberta.ca

ABSTRACT

Chronic wasting disease is a set of prion diseases infecting captive and wild cervids. As different CWD agents are transmitted between different cervid species and between the same species having different Prnp polymorphisms, novel CWD strains can be generated. These new strains can exhibit different biochemical properties as well as different conformers. Identification of novel CWD strains is critically important as the different strains may vary in their host ranges, increasing the potential for transmission to economically important species as well as zoonotic transmission.

A rate limiting step in the characterization of CWD strains is the identification of deer samples that potentially contain novel strains. Much of the strain generation likely occurs during early passage between different Prnp genotypes (of the same or different species). Traditionally these differences have been identified following passages into rodent models (strains of lab mice, transgenic mice expressing different Prnp sequences and/or hamsters). Incubation periods can be long and the number of potential isolates is high making transmission experiments a slow, tedious, and expensive process. To streamline the process, we have identified a panel of in vitro analyses to help target samples for further characterization. For some isolates, potential new strains have been identified by western blot analysis, others by cervid cell assay, changes in the Prnp sequence, folding and/or aggregation differences as well as ability to seed reactions in RT-QuIC. Isolates of interest are then further characterized by transmission in a variety of different tg mouse lines, wild-type mice, hamsters, and voles. Using these criteria, five potential CWD strains have been identified.

https://www.tandfonline.com/doi/full/10.1080/19336896.2019.1615197

TEXAS CWD 148 connected to deer breeding facilities and release sites OF 213 confirmed to date

TUESDAY, MARCH 02, 2021

Texas Confirms CWD TSE Prion in 213 white-tailed deer, mule deer, red deer and elk to date, 148 connected to deer breeding facilities and release sites

https://chronic-wasting-disease.blogspot.com/2021/03/texas-confirms-cwd-tse-prion-in-213.html

FRIDAY, DECEMBER 20, 2019

Texas TAHC, Administrative Code, Title 4, Part 2, Chapter 40, Chronic Wasting Disease Amendments Open For Comment beginning December 20, 2019 thru January 20, 2020 Terry Singeltary Comments Submission

http://chronic-wasting-disease.blogspot.com/2019/12/texas-tahc-administrative-code-title-4.html

Hundreds Of Escapes From State Deer Farms Reported Since 2013

Data From DATCP, DNR Show Wide Discrepancies In Total Number Of Escapes

By Rich Kremer

Published: Friday, December 14, 2018, 1:00pm

State records show hundreds of animals have escaped from Wisconsin deer and elk farms over the last five years, raising concerns about the potential spread of chronic wasting disease and highlighting discrepancies in the way state agencies track such incidents.

Data obtained from the Wisconsin Department of Agriculture, Trade and Consumer Protection under the state's open records law show the agency recorded 181 escapes from deer farms and hunting preserves between November 2013 and November 2018. Meanwhile, the state Department of Natural Resources recorded 331 escapes from 2013 through Dec. 10 of this year

https://www.wpr.org/hundreds-escapes-state-deer-farms-reported-2013

In the four-year period that ended last December, 247 deer escaped from Minnesota deer farms, state records show. Forty-six were never found.

‘‘The deer farms have gotten a pass," said John Zanmiller, spokesman for Bluffland Whitetails Association. "They shouldn't be allowed to operate at the public's expense."

https://www.startribune.com/hunting-preserves-and-the-spread-of-cwd-into-minnesota-s-wild-deer-populations/563287612/

Captive white-tailed deer industry—Current status and growing threat

Kip P. Adams Brian P. Murphy Matthew D. Ross

First published: 28 March 2016 https://doi.org/10.1002/wsb.627Citations: 5

ABSTRACT

In 2012, 10 states in the United States debated legislation to introduce or expand captive white-tailed deer (Odocoileus virginianus) breeding operations. Because this was far more than any prior year, we surveyed 37 state wildlife agencies in the eastern contiguous United States to determine the scope and intensity of captive operations and state-specific requirements. Thirty-seven states provided some data. Twenty of 32 states reported 5–1,332 breeding facilities holding >140,000 white-tailed deer; 20 of 29 states reported 1–150 shooting preserves holding >25,000 white-tailed deer. Captive white-tailed deer were classified as wildlife in 12 of 22 states (55%), livestock in 8 states (36%), and game animals in 2 states (9%). Only 5 of 21 states (24%) had minimum acreage requirements for breeding facilities, but 13 of 20 states (65%) did for shooting preserves. Four of 16 states (25%) had a minimum release time before deer could be shot in a preserve. Six of 21 states (29%) had stocking density requirements, with 6 of 25 states (24%) listing habitat requirements for captive white-tailed deer facilities. Seventeen of 23 states (74%) required external tagging, and 24 of 27 states (89%) allowed consumption of white-tailed deer killed in shooting preserves. We believe the captive white-tailed deer industry undermines the North American Model of Wildlife Conservation, threatens the health of wild deer, and undermines the public's perception of hunting. A better

understanding of this growing industry will help managers safeguard free-ranging white-tailed deer populations and the future of deer hunting. © 2016 The Wildlife Society.

https://wildlife.onlinelibrary.wiley.com/doi/abs/10.1002/wsb.627

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see; Infectious agent of sheep scrapie may persist in the environment for at least 16 to 21 years.

***> 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

Gudmundur Georgsson1, Sigurdur Sigurdarson2, Paul Brown3

First Published: 01 December 2006 https://doi.org/10.1099/vir.0.82011-0 ABSTRACT In 1978, a rigorous programme was implemented to stop the spread of, and subsequently eradicate, sheep scrapie in Iceland. Affected flocks were culled, premises were disinfected and, after 2–3 years, restocked with lambs from scrapie-free areas. Between 1978 and 2004, scrapie recurred on 33 farms. 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. Of special interest was one farm with a small, completely self-contained flock where scrapie recurred 18 years after culling, 2 years after some lambs had been housed in an old sheep-house that had never been disinfected. Epidemiological investigation established with near certitude that the disease had not been introduced from the outside and it is concluded that the agent may have persisted in the old sheep-house for at least 16 years.

Gudmundur Georgsson,1 Sigurdur Sigurdarson2 and Paul Brown3

Correspondence

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1 Institute for Experimental Pathology, University of Iceland, Keldur v/vesturlandsveg, IS-112 Reykjavı´k, Iceland

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Received 7 March 2006 Accepted 6 August 2006

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http://www.microbiologyresearch.org/docserver/fulltext/jgv/87/12/3737.pdf?expires=1540908280&id=id&accname=guest&checksum=ED0572E1E5B272C100A32212A3E3761A

2018 - 2019

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

Rapid recontamination of a farm building occurs after attempted prion removal

http://dx.doi.org/10.1136/vr.105054

Kevin Christopher Gough, BSc (Hons), PhD1, Claire Alison Baker, BSc (Hons)2, Steve Hawkins, MIBiol3, Hugh Simmons, BVSc, MRCVS, MBA, MA3, Timm Konold, DrMedVet, PhD, MRCVS3 and Ben Charles Maddison, BSc (Hons), PhD2

Abstract

The transmissible spongiform encephalopathy scrapie of sheep/goats and chronic wasting disease of cervids are associated with environmental reservoirs of infectivity.

Preventing environmental prions acting as a source of infectivity to healthy animals is of major concern to farms that have had outbreaks of scrapie and also to the health management of wild and farmed cervids.

Here, an efficient scrapie decontamination protocol was applied to a farm with high levels of environmental contamination with the scrapie agent.

Post-decontamination, no prion material was detected within samples taken from the farm buildings as determined using a sensitive in vitro replication assay (sPMCA).

A bioassay consisting of 25 newborn lambs of highly susceptible prion protein genotype VRQ/VRQ introduced into this decontaminated barn was carried out in addition to sampling and analysis of dust samples that were collected during the bioassay.

Twenty-four of the animals examined by immunohistochemical analysis of lymphatic tissues were scrapie-positive during the bioassay, samples of dust collected within the barn were positive by month 3.

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...

As in the authors' previous study,12 the decontamination of this sheep barn was not effective at removing scrapie infectivity, and despite the extra measures brought into this study (more effective chemical treatment and removal of sources of dust) the overall rates of disease transmission mirror previous results on this farm. With such apparently effective decontamination (assuming that at least some sPMCA seeding ability is coincident with infectivity), how was infectivity able to persist within the environment and where does infectivity reside? Dust samples were collected in both the bioassay barn and also a barn subject to the same decontamination regime within the same farm (but remaining unoccupied). Within both of these barns dust had accumulated for three months that was able to seed sPMCA, indicating the accumulation of scrapie-containing material that was independent of the presence of sheep that may have been incubating and possibly shedding low amounts of infectivity.

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 scrapiepositive 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.

Acknowledgements The authors thank the APHA farm staff, Tony Duarte, Olly Roberts and Margaret Newlands for preparation of the sheep pens and animal husbandry during the study. The authors also thank the APHA pathology team for RAMALT and postmortem examination.

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Competing interests None declared.

https://veterinaryrecord.bmj.com/content/early/2019/01/02/vr.105054.long

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

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

Saturday, January 5, 2019

Rapid recontamination of a farm building occurs after attempted prion removal

https://prionprp.blogspot.com/2019/01/rapid-recontamination-of-farm-building.html

The effectiveness of on-farm decontamination methods for scrapie - SE1865

Description

Scrapie infectivity persists on farms where infected animals have been removed1. Recently we have demonstrated that it is possible to detect environmental scrapie contamination biochemically using serial Protein Misfolding Cyclic Amplification (sPMCA)2, allowing the monitoring of scrapie infectivity on farm premises. Ongoing Defra study SE1863 has compared pen decontamination regimes on a scrapie-infected farm by both sheep bioassay and sPMCA. For bioassay, scrapie-free genetically susceptible lambs were introduced into pens decontaminated using distinct methodologies, all pens contained scrapie-positive lambs within 1 year.

Remarkably this included lambs housed within a pen which had been jet washed/chloros treated, followed by regalvanisation/ replacement of all metalwork and painting of all other surfaces.

We have recently demonstrated using sPMCA, that material collected on swabs from vertical surfaces at heights inaccessible to sheep within a barn on the same scrapie affected farm contained scrapie prions (unpublished observations). We hypothesise that scrapie prions are most likely to have been deposited in these areas by bioaerosol movement. We propose that this bioaerosol movement contributes to scrapie transmission within the barn, and could account for the sheep that became positive within the pen containing re-galvanised/new metalwork and repainted surfaces (project SE1863). It is proposed that a thorough decontamination that would minimise prion-contaminated dust, both within the building and its immediate vicinity, is likely to increase the effectiveness of current methods for decontaminating farm buildings following outbreaks of scrapie. The proposed study builds on our previous data and will thoroughly investigate the potential for farm building scrapie-contamination via the bioaerosol route, a previously unrecognised route for dissemination of scrapie infectivity. This route could lead to the direct infection of healthy animals and/or indirect transmission of disease via contamination of surfaces within animal pens. The proposed study would analyse material collected using air samplers set up within “scrapie-infected” barns and their immediate vicinity, to confirm that prion containing material can be airborne within a scrapie infected farm environment. The study would incorporate a biochemical assessment of different surface decontamination methods, in order to demonstrate the best methodology and then the analysis of air and surface samples after a complete building decontamination to remove sources of dust and surface bound prions from both the building and its immediate vicinity. Analysis of such surface and air samples collected before and after treatment would measure the reduction in levels of infectivity. It is envisaged that the biochemical demonstration of airborne prions and the effective reduction in such prion dissemination would lead to a sheep bioassay experiment that would be conducted after a full farm decontamination. This would fully assess the effectiveness of an optimised scrapie decontamination strategy.

This study will contribute directly to Defra policy on best practice for on-farm decontamination after outbreaks of scrapie; a situation particularly relevant to decontamination after scrapie cases on goat farms where no genetic resistance to scrapie has currently been identified, and where complete decontamination is essential in order to stop recurrence of scrapie after restocking.

Objective

Phase 1

- Determine the presence and relative levels of airborne prions on a scrapie infected farm.

- Evaluate different pen surface decontamination procedures.

Phase 2

- Determine the presence of any airborne prions in a barn after a full decontamination.

Phase 3

- Further assess the efficacy of the decontamination procedure investigated in phase 2 by sheep bioassay.

Time-Scale and Cost

From: 2012

To: 2016

Cost: £326,784

Contractor / Funded Organisations

A D A S UK Ltd (ADAS)

Keywords Animals Fields of Study Animal Health

http://randd.defra.gov.uk/Default.aspx?FromSearch=Y&Location=None&Menu=Menu&Module=More&Paging=10&ProjectID=18479&Publisher=1&SearchText=SE1865&SortOrder=Asc&SortString=ProjectCode#Description

The Effectiveness of on-Farm Decontamination Methods for Scrapie

Institutions ADAS

Start date 2012

End date 2016

Objective Phase 1

Determine the presence and relative levels of airborne prions on a scrapie infected farm. Evaluate different pen surface decontamination procedures.

Phase 2

Determine the presence of any airborne prions in a barn after a full decontamination.

Phase 3

Further assess the efficacy of the decontamination procedure investigated in phase 2 by sheep bioassay.

More information

Scrapie infectivity persists on farms where infected animals have been removed1. Recently we have demonstrated that it is possible to detect environmental scrapie contamination biochemically using serial Protein Misfolding Cyclic Amplification (sPMCA)2, allowing the monitoring of scrapie infectivity on farm premises. Ongoing Defra study SE1863 has compared pen decontamination regimes on a scrapie-infected farm by both sheep bioassay and sPMCA. For bioassay, scrapie-free genetically susceptible lambs were introduced into pens decontaminated using distinct methodologies, all pens contained scrapie-positive lambs within 1 year. Remarkably this included lambs housed within a pen which had been jet washed/chloros treated, followed by regalvanisation/replacement of all metalwork and painting of all other surfaces.

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This study will contribute directly to Defra policy on best practice for on-farm decontamination after outbreaks of scrapie; a situation particularly relevant to decontamination after scrapie cases on goat farms where no genetic resistance to scrapie has currently been identified, and where complete decontamination is essential in order to stop recurrence of scrapie after restocking.

Funding Source

Department for Environment, Food and Rural Affairs

Project source

View this project

Project number

SE1865

Categories

Foodborne Disease

Policy and Planning

https://fsrio.nal.usda.gov/fsrio/research-projects/effectiveness-farm-decontamination-methods-scrapie

Circulation of prions within dust on a scrapie affected farm

Kevin C Gough1 , Claire A Baker2 , Hugh A Simmons3 , Steve A Hawkins3 and Ben C Maddison2*

Abstract

Prion diseases are fatal neurological disorders that affect humans and animals. Scrapie of sheep/goats and Chronic Wasting Disease (CWD) of deer/elk are contagious prion diseases where environmental reservoirs have a direct link to the transmission of disease. Using protein misfolding cyclic amplification we demonstrate that scrapie PrPSc can be detected within circulating dusts that are present on a farm that is naturally contaminated with sheep scrapie. The presence of infectious scrapie within airborne dusts may represent a possible route of infection and illustrates the difficulties that may be associated with the effective decontamination of such scrapie affected premises.

snip...

Discussion We present biochemical data illustrating the airborne movement of scrapie containing material within a contaminated farm environment. We were able to detect scrapie PrPSc within extracts from dusts collected over a 70 day period, in the absence of any sheep activity. We were also able to detect scrapie PrPSc within dusts collected within pasture at 30 m but not at 60 m distance away from the scrapie contaminated buildings, suggesting that the chance of contamination of pasture by scrapie contaminated dusts decreases with distance from contaminated farm buildings. PrPSc amplification by sPMCA has been shown to correlate with infectivity and amplified products have been shown to be infectious [14,15]. These experiments illustrate the potential for low dose scrapie infectivity to be present within such samples. We estimate low ng levels of scrapie positive brain equivalent were deposited per m2 over 70 days, in a barn previously occupied by sheep affected with scrapie. This movement of dusts and the accumulation of low levels of scrapie infectivity within this environment may in part explain previous observations where despite stringent pen decontamination regimens healthy lambs still became scrapie infected after apparent exposure from their environment alone [16]. The presence of sPMCA seeding activity and by inference, infectious prions within dusts, and their potential for airborne dissemination is highly novel and may have implications for the spread of scrapie within infected premises. The low level circulation and accumulation of scrapie prion containing dust material within the farm environment will likely impede the efficient decontamination of such scrapie contaminated buildings unless all possible reservoirs of dust are removed. Scrapie containing dusts could possibly infect animals during feeding and drinking, and

respiratory and conjunctival routes may also be involved. It has been demonstrated that scrapie can be efficiently transmitted via the nasal route in sheep [17], as is also the case for CWD in both murine models and in white tailed deer [18-20].

The sources of dust borne prions are unknown but it seems reasonable to assume that faecal, urine, skin, parturient material and saliva-derived prions may contribute to this mobile environmental reservoir of infectivity. This work highlights a possible transmission route for scrapie within the farm environment, and this is likely to be paralleled in CWD which shows strong similarities with scrapie in terms of prion dissemination and disease transmission. The data indicate that the presence of scrapie prions in dust is likely to make the control of these diseases a considerable challenge.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4397813/

THE 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.

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 with.

***> that's what's so worrisome about Iatrogenic mode of transmission, a simple autoclave will not kill this TSE prion agent.

1: J Neurol Neurosurg Psychiatry 1994 Jun;57(6):757-8

***> Transmission of Creutzfeldt-Jakob disease to a chimpanzee by electrodes contaminated during neurosurgery.

Gibbs CJ Jr, Asher DM, Kobrine A, Amyx HL, Sulima MP, Gajdusek DC.

Laboratory of Central Nervous System Studies, National Institute of

Neurological Disorders and Stroke, National Institutes of Health,

Bethesda, MD 20892.

Stereotactic multicontact electrodes used to probe the cerebral cortex of a middle aged woman with progressive dementia were previously implicated in the accidental transmission of Creutzfeldt-Jakob disease (CJD) to two younger patients. The diagnoses of CJD have been confirmed for all three cases. More than two years after their last use in humans, after three cleanings and repeated sterilisation in ethanol and formaldehyde vapour, the electrodes were implanted in the cortex of a chimpanzee. Eighteen months later the animal became ill with CJD. This finding serves to re-emphasise the potential danger posed by reuse of instruments contaminated with the agents of spongiform encephalopathies, even after scrupulous attempts to clean them.

PMID: 8006664 [PubMed - indexed for MEDLINE]

https://www.ncbi.nlm.nih.gov/pubmed/8006664?dopt=Abstract

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/

Detection of protease-resistant cervid prion protein in water from a CWD-endemic area

https://www.ncbi...nlm.nih.gov/pmc/articles/PMC2802782/pdf/prion0303_0171.pdf

A Quantitative Assessment of the Amount of Prion Diverted to Category 1 Materials and Wastewater During Processing

http://onlinelibrary.wiley.com/doi/10.1111/j.1539-6924.2012.01922.x/abstract

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

Back around 2000, 2001, or so, I was corresponding with officials abroad during the bse inquiry, passing info back and forth, and some officials from here inside USDA aphis FSIS et al. In fact helped me get into the USA 50 state emergency BSE conference call way back. That one was a doozy. But I always remember what “deep throat” I never knew who they were, but I never forgot;

Some unofficial information from a source on the inside looking out -

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

http://scrapie-usa.blogspot.com/2018/04/scrapie-transmits-to-pigs-by-oral-route.html

Infectivity surviving ashing to 600*C is (in my opinion) degradable but infective. based on Bown & Gajdusek, (1991), landfill and burial may be assumed to have a reduction factor of 98% (i.e. a factor of 50) over 3 years. CJD-infected brain-tissue remained infectious after storing at room-temperature for 22 months (Tateishi et al, 1988). Scrapie agent is known to remain viable after at least 30 months of desiccation (Wilson et al, 1950). and pastures that had been grazed by scrapie-infected sheep still appeared to be contaminated with scrapie agent three years after they were last occupied by sheep (Palsson, 1979).

http://europa.eu.int/comm/food/fs/sc/ssc/out58_en.pdf

Dr. Paul Brown Scrapie Soil Test BSE Inquiry Document

https://web.archive.org/web/20090505211734/http://www.bseinquiry.gov.uk/files/sc/Seac07/tab03.pdf

Using in vitro Prion replication for high sensitive detection of prions and prionlike proteins and for understanding mechanisms of transmission.

Claudio Soto Mitchell Center for Alzheimer's diseases and related Brain disorders, Department of Neurology, University of Texas Medical School at Houston.

Prion and prion-like proteins are misfolded protein aggregates with the ability to selfpropagate to spread disease between cells, organs and in some cases across individuals. I n T r a n s m i s s i b l e s p o n g i f o r m encephalopathies (TSEs), prions are mostly composed by a misfolded form of the prion protein (PrPSc), which propagates by transmitting its misfolding to the normal prion protein (PrPC). The availability of a procedure to replicate prions in the laboratory may be important to study the mechanism of prion and prion-like spreading and to develop high sensitive detection of small quantities of misfolded proteins in biological fluids, tissues and environmental samples. Protein Misfolding Cyclic Amplification (PMCA) is a simple, fast and efficient methodology to mimic prion replication in the test tube. PMCA is a platform technology that may enable amplification of any prion-like misfolded protein aggregating through a seeding/nucleation process. In TSEs, PMCA is able to detect the equivalent of one single molecule of infectious PrPSc and propagate prions that maintain high infectivity, strain properties and species specificity. Using PMCA we have been able to detect PrPSc in blood and urine of experimentally infected animals and humans affected by vCJD with high sensitivity and specificity. Recently, we have expanded the principles of PMCA to amplify amyloid-beta (Aβ) and alphasynuclein (α-syn) aggregates implicated in Alzheimer's and Parkinson's diseases, respectively. Experiments are ongoing to study the utility of this technology to detect Aβ and α-syn aggregates in samples of CSF and blood from patients affected by these diseases.

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***>>> Recently, we have been using PMCA to study the role of environmental prion contamination on the horizontal spreading of TSEs. These experiments have focused on the study of the interaction of prions with plants and environmentally relevant surfaces. Our results show that plants (both leaves and roots) bind tightly to prions present in brain extracts and excreta (urine and feces) and retain even small quantities of PrPSc for long periods of time. Strikingly, ingestion of prioncontaminated leaves and roots produced disease with a 100% attack rate and an incubation period not substantially longer than feeding animals directly with scrapie brain homogenate. Furthermore, plants can uptake prions from contaminated soil and transport them to different parts of the plant tissue (stem and leaves). Similarly, prions bind tightly to a variety of environmentally relevant surfaces, including stones, wood, metals, plastic, glass, cement, etc. Prion contaminated surfaces efficiently transmit prion disease when these materials were directly injected into the brain of animals and strikingly when the contaminated surfaces were just placed in the animal cage. These findings demonstrate that environmental materials can efficiently bind infectious prions and act as carriers of infectivity, suggesting that they may play an important role in the horizontal transmission of the disease.

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Since its invention 13 years ago, PMCA has helped to answer fundamental questions of prion propagation and has broad applications in research areas including the food industry, blood bank safety and human and veterinary disease diagnosis.

source reference Prion Conference 2015 abstract book

Grass Plants Bind, Retain, Uptake, and Transport Infectious Prions

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SUMMARY

Prions are the protein-based infectious agents responsible for prion diseases. Environmental prion contamination has been implicated in disease transmission. Here, we analyzed the binding and retention of infectious prion protein (PrPSc) to plants. Small quantities of PrPSc contained in diluted brain homogenate or in excretory materials (urine and feces) can bind to wheat grass roots and leaves. Wild-type hamsters were efficiently infected by ingestion of prion-contaminated plants. The prion-plant interaction occurs with prions from diverse origins, including chronic wasting disease. Furthermore, leaves contaminated by spraying with a prion-containing preparation retained PrPSc for several weeks in the living plant. Finally, plants can uptake prions from contaminated soil and transport them to aerial parts of the plant (stem and leaves). These findings demonstrate that plants can efficiently bind infectious prions and act as carriers of infectivity, suggesting a possible role of environmental prion contamination in the horizontal transmission of the disease.

INTRODUCTION

snip...

DISCUSSION

This study shows that plants can efficiently bind prions contained in brain extracts from diverse prion infected animals, including CWD-affected cervids. PrPSc attached to leaves and roots from wheat grass plants remains capable of seeding prion replication in vitro. Surprisingly, the small quantity of PrPSc naturally excreted in urine and feces from sick hamster or cervids was enough to efficiently contaminate plant tissue. Indeed, our results suggest that the majority of excreted PrPSc is efficiently captured by plants' leaves and roots. Moreover, leaves can be contaminated by spraying them with a prion-containing extract, and PrPSc remains detectable in living plants for as long as the study was performed (several weeks). Remarkably, prion contaminated plants transmit prion disease to animals upon ingestion, producing a 100% attack rate and incubation periods not substantially longer than direct oral administration of sick brain homogenates.

Finally, an unexpected but exciting result was that plants were able to uptake prions from contaminated soil and transport them to aerial parts of the plant tissue. Although it may seem farfetched that plants can uptake proteins from the soil and transport it to the parts above the ground, there are already published reports of this phenomenon (McLaren et al., 1960; Jensen and McLaren, 1960;Paungfoo-Lonhienne et al., 2008). The high resistance of prions to degradation and their ability to efficiently cross biological barriers may play a role in this process. The mechanism by which plants bind, retain, uptake, and transport prions is unknown. We are currently studying the way in which prions interact with plants using purified, radioactively labeled PrPSc to determine specificity of the interaction, association constant, reversibility, saturation, movement, etc.

Epidemiological studies have shown numerous instances of scrapie or CWD recurrence upon reintroduction of animals on pastures previously exposed to prion-infected animals. Indeed, reappearance of scrapie has been documented following fallow periods of up to 16 years (Georgsson et al., 2006), and pastures were shown to retain infectious CWD prions for at least 2 years after exposure (Miller et al., 2004). It is likely that the environmentally mediated transmission of prion diseases depends upon the interaction of prions with diverse elements, including soil, water, environmental surfaces, various invertebrate animals, and plants.

However, since plants are such an important component of the environment and also a major source of food for many animal species, including humans, our results may have far-reaching implications for animal and human health. Currently, the perception of the risk for animal-to-human prion transmission has been mostly limited to consumption or exposure to

contaminated meat; our results indicate that plants might also be an important vector of transmission that needs to be considered in risk assessment.

https://www.cell.com/cell-reports/pdf/S2211-1247%2815%2900437-4.pdf

***> CONGRESSIONAL ABSTRACTS PRION CONFERENCE 2018

P69 Experimental transmission of CWD from white-tailed deer to co-housed reindeer

Mitchell G (1), Walther I (1), Staskevicius A (1), Soutyrine A (1), Balachandran A (1)

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Chronic wasting disease (CWD) continues to be detected in wild and farmed cervid populations of North America, affecting predominantly white-tailed deer, mule deer and elk. Extensive herds of wild caribou exist in northern regions of Canada, although surveillance has not detected the presence of CWD in this population. Oral experimental transmission has demonstrated that reindeer, a species closely related to caribou, are susceptible to CWD. Recently, CWD was detected for the first time in Europe, in wild Norwegian reindeer, advancing the possibility that caribou in North America could also become infected. Given the potential overlap in habitat between wild CWD-infected cervids and wild caribou herds in Canada, we sought to investigate the horizontal transmissibility of CWD from white-tailed deer to reindeer.

Two white-tailed deer were orally inoculated with a brain homogenate prepared from a farmed Canadian white-tailed deer previously diagnosed with CWD. Two reindeer, with no history of exposure to CWD, were housed in the same enclosure as the white-tailed deer, 3.5 months after the deer were orally inoculated. The white-tailed deer developed clinical signs consistent with CWD beginning at 15.2 and 21 months post-inoculation (mpi), and were euthanized at 18.7 and 23.1 mpi, respectively. Confirmatory testing by immunohistochemistry (IHC) and western blot demonstrated widespread aggregates of pathological prion protein (PrPCWD) in the central nervous system and lymphoid tissues of both inoculated white-tailed deer. Both reindeer were subjected to recto-anal mucosal associated lymphoid tissue (RAMALT) biopsy at 20 months post-exposure (mpe) to the white-tailed deer. The biopsy from one reindeer contained PrPCWD confirmed by IHC. This reindeer displayed only subtle clinical evidence of disease prior to a rapid decline in condition requiring euthanasia at 22.5 mpe. Analysis of tissues from this reindeer by IHC revealed widespread PrPCWD deposition, predominantly in central nervous system and lymphoreticular tissues. Western blot molecular profiles were similar between both orally inoculated white-tailed deer and the CWD positive reindeer. Despite sharing the same enclosure, the other reindeer was RAMALT negative at 20 mpe, and PrPCWD was not detected in brainstem and lymphoid tissues following necropsy at 35 mpe. Sequencing of the prion protein gene from both reindeer revealed differences at several codons, which may have influenced susceptibility to infection.

Natural transmission of CWD occurs relatively efficiently amongst cervids, supporting the expanding geographic distribution of disease and the potential for transmission to previously naive populations. The efficient horizontal transmission of CWD from white-tailed deer to reindeer observed here highlights the potential for reindeer to become infected if exposed to other cervids or environments infected with CWD.

SOURCE REFERENCE 2018 PRION CONFERENCE ABSTRACT

Research Project: TRANSMISSION, DIFFERENTIATION, AND PATHOBIOLOGY OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES Location: Virus and Prion Research

Title: Horizontal transmission of chronic wasting disease in reindeer

Author

item MOORE, SARAH - ORISE FELLOW item KUNKLE, ROBERT item WEST GREENLEE, MARY - IOWA STATE UNIVERSITY item Nicholson, Eric item RICHT, JUERGEN item HAMIR, AMIRALI item WATERS, WADE item Greenlee, Justin

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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.

Technical Abstract: Chronic wasting disease (CWD) is a naturally-occurring, fatal prion disease of cervids. Reindeer (Rangifer tarandus tarandus) are susceptible to CWD following oral challenge, and CWD was recently reported in a free-ranging reindeer of Norway. Potential contact between CWD-affected cervids and Rangifer species that are free-ranging or co-housed on farms presents a potential risk of CWD transmission. The aims of this study were to 1) investigate the transmission of CWD from white-tailed deer (Odocoileus virginianus; CWDwtd), mule deer (Odocoileus hemionus; CWDmd), or elk (Cervus elaphus nelsoni; CWDelk) to reindeer via the intracranial route, and 2) to assess for direct and indirect horizontal transmission to non-inoculated sentinels. Three groups of 5 reindeer fawns were challenged intracranially with CWDwtd, CWDmd, or CWDelk. Two years after challenge of inoculated reindeer, non-inoculated negative control reindeer were introduced into the same pen as the CWDwtd inoculated reindeer (direct contact; n=4) or into a pen adjacent to the CWDmd inoculated reindeer (indirect contact; n=2). Experimentally inoculated reindeer were allowed to develop clinical disease. At death/euthanasia a complete necropsy examination was performed, including immunohistochemical testing of tissues for disease-associated CWD prion protein (PrPcwd). Intracranially challenged reindeer developed clinical disease from 21 months post-inoculation (months PI). PrPcwd was detected in 5 out of 6 sentinel reindeer although only 2 out of 6 developed clinical disease during the study period (< 57 months PI). We have shown that reindeer are susceptible to CWD from various cervid sources and can transmit CWD to naïve reindeer both directly and indirectly.

TITLE: PATHOLOGICAL FEATURES OF CHRONIC WASTING DISEASE IN REINDEER AND DEMONSTRATION OF HORIZONTAL TRANSMISSION

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

*** DECEMBER 2016 CDC EMERGING INFECTIOUS DISEASE JOURNAL CWD HORIZONTAL TRANSMISSION

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

ORIGINAL RESEARCH ARTICLE

Front. Vet. Sci., 14 September 2015 | https://doi.org/10.3389/fvets.2015.00032

Objects in contact with classical scrapie sheep act as a reservoir for scrapie transmission

imageTimm Konold1*, imageStephen A. C. Hawkins2, imageLisa C. Thurston3, imageBen C. Maddison4, imageKevin C. Gough5, imageAnthony Duarte1 and imageHugh A. Simmons1

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Classical scrapie is an environmentally transmissible prion disease of sheep and goats. Prions can persist and remain potentially infectious in the environment for many years and thus pose a risk of infecting animals after re-stocking. In vitro studies using serial protein misfolding cyclic amplification (sPMCA) have suggested that objects on a scrapie-affected sheep farm could contribute to disease transmission. This in vivo study aimed to determine the role of field furniture (water troughs, feeding troughs, fencing, and other objects that sheep may rub against) used by a scrapie-infected sheep flock as a vector for disease transmission to scrapie-free lambs with the prion protein genotype VRQ/VRQ, which is associated with high susceptibility to classical scrapie. When the field furniture was placed in clean accommodation, sheep became infected when exposed to either a water trough (four out of five) or to objects used for rubbing (four out of seven). This field furniture had been used by the scrapie-infected flock 8 weeks earlier and had previously been shown to harbor scrapie prions by sPMCA. Sheep also became infected (20 out of 23) through exposure to contaminated field furniture placed within pasture not used by scrapie-infected sheep for 40 months, even though swabs from this furniture tested negative by PMCA. This infection rate decreased (1 out of 12) on the same paddock after replacement with clean field furniture. Twelve grazing sheep exposed to field furniture not in contact with scrapie-infected sheep for 18 months remained scrapie free. The findings of this study highlight the role of field furniture used by scrapie-infected sheep to act as a reservoir for disease re-introduction although infectivity declines considerably if the field furniture has not been in contact with scrapie-infected sheep for several months. PMCA may not be as sensitive as VRQ/VRQ sheep to test for environmental contamination.

snip...

Discussion

Classical scrapie is an environmentally transmissible disease because it has been reported in naïve, supposedly previously unexposed sheep placed in pastures formerly occupied by scrapie-infected sheep (4, 19, 20).

Although the vector for disease transmission is not known, soil is likely to be an important reservoir for prions (2) where – based on studies in rodents – prions can adhere to minerals as a biologically active form (21) and remain infectious for more than 2 years (22).

Similarly, chronic wasting disease (CWD) has re-occurred in mule deer housed in paddocks used by infected deer 2 years earlier, which was assumed to be through foraging and soil consumption (23).

Our study suggested that the risk of acquiring scrapie infection was greater through exposure to contaminated wooden, plastic, and metal surfaces via water or food troughs, fencing, and hurdles than through grazing.

Drinking from a water trough used by the scrapie flock was sufficient to cause infection in sheep in a clean building.

Exposure to fences and other objects used for rubbing also led to infection, which supported the hypothesis that skin may be a vector for disease transmission (9).

The risk of these objects to cause infection was further demonstrated when 87% of 23 sheep presented with PrPSc in lymphoid tissue after grazing on one of the paddocks, which contained metal hurdles, a metal lamb creep and a water trough in contact with the scrapie flock up to 8 weeks earlier, whereas no infection had been demonstrated previously in sheep grazing on this paddock, when equipped with new fencing and field furniture.

When the contaminated furniture and fencing were removed, the infection rate dropped significantly to 8% of 12 sheep, with soil of the paddock as the most likely source of infection caused by shedding of prions from the scrapie-infected sheep in this paddock up to a week earlier.

This study also indicated that the level of contamination of field furniture sufficient to cause infection was dependent on two factors: stage of incubation period and time of last use by scrapie-infected sheep.

Drinking from a water trough that had been used by scrapie sheep in the predominantly pre-clinical phase did not appear to cause infection, whereas infection was shown in sheep drinking from the water trough used by scrapie sheep in the later stage of the disease.

It is possible that contamination occurred through shedding of prions in saliva, which may have contaminated the surface of the water trough and subsequently the water when it was refilled.

Contamination appeared to be sufficient to cause infection only if the trough was in contact with sheep that included clinical cases.

Indeed, there is an increased risk of bodily fluid infectivity with disease progression in scrapie (24) and CWD (25) based on PrPSc detection by sPMCA.

Although ultraviolet light and heat under natural conditions do not inactivate prions (26), furniture in contact with the scrapie flock, which was assumed to be sufficiently contaminated to cause infection, did not act as vector for disease if not used for 18 months, which suggest that the weathering process alone was sufficient to inactivate prions.

PrPSc detection by sPMCA is increasingly used as a surrogate for infectivity measurements by bioassay in sheep or mice.

In this reported study, however, the levels of PrPSc present in the environment were below the limit of detection of the sPMCA method, yet were still sufficient to cause infection of in-contact animals.

In the present study, the outdoor objects were removed from the infected flock 8 weeks prior to sampling and were positive by sPMCA at very low levels (2 out of 37 reactions).

As this sPMCA assay also yielded 2 positive reactions out of 139 in samples from the scrapie-free farm, the sPMCA assay could not detect PrPSc on any of the objects above the background of the assay.

False positive reactions with sPMCA at a low frequency associated with de novo formation of infectious prions have been reported (27, 28).

This is in contrast to our previous study where we demonstrated that outdoor objects that had been in contact with the scrapie-infected flock up to 20 days prior to sampling harbored PrPSc that was detectable by sPMCA analysis [4 out of 15 reactions (12)] and was significantly more positive by the assay compared to analogous samples from the scrapie-free farm.

This discrepancy could be due to the use of a different sPMCA substrate between the studies that may alter the efficiency of amplification of the environmental PrPSc.

In addition, the present study had a longer timeframe between the objects being in contact with the infected flock and sampling, which may affect the levels of extractable PrPSc.

Alternatively, there may be potentially patchy contamination of this furniture with PrPSc, which may have been missed by swabbing.

The failure of sPMCA to detect CWD-associated PrP in saliva from clinically affected deer despite confirmation of infectivity in saliva-inoculated transgenic mice was associated with as yet unidentified inhibitors in saliva (29), and it is possible that the sensitivity of sPMCA is affected by other substances in the tested material.

In addition, sampling of amplifiable PrPSc and subsequent detection by sPMCA may be more difficult from furniture exposed to weather, which is supported by the observation that PrPSc was detected by sPMCA more frequently in indoor than outdoor furniture (12).

A recent experimental study has demonstrated that repeated cycles of drying and wetting of prion-contaminated soil, equivalent to what is expected under natural weathering conditions, could reduce PMCA amplification efficiency and extend the incubation period in hamsters inoculated with soil samples (30).

This seems to apply also to this study even though the reduction in infectivity was more dramatic in the sPMCA assays than in the sheep model.

Sheep were not kept until clinical end-point, which would have enabled us to compare incubation periods, but the lack of infection in sheep exposed to furniture that had not been in contact with scrapie sheep for a longer time period supports the hypothesis that prion degradation and subsequent loss of infectivity occurs even under natural conditions.

In conclusion, the results in the current study indicate that removal of furniture that had been in contact with scrapie-infected animals should be recommended, particularly since cleaning and decontamination may not effectively remove scrapie infectivity (31), even though infectivity declines considerably if the pasture and the field furniture have not been in contact with scrapie-infected sheep for several months. As sPMCA failed to detect PrPSc in furniture that was subjected to weathering, even though exposure led to infection in sheep, this method may not always be reliable in predicting the risk of scrapie infection through environmental contamination.

These results suggest that the VRQ/VRQ sheep model may be more sensitive than sPMCA for the detection of environmentally associated scrapie, and suggest that extremely low levels of scrapie contamination are able to cause infection in susceptible sheep genotypes.

Keywords: classical scrapie, prion, transmissible spongiform encephalopathy, sheep, field furniture, reservoir, serial protein misfolding cyclic amplification

http://journal.frontiersin.org/article/10.3389/fvets.2015.00032/full

***> 6th America BSE 589.2001 FEED REGULATIONS CWD TSE Prion

so far, we have been lucky. to date, with the science at hand, no cwd transmitted to cattle, that has been documented, TO DATE, WITH THE SCIENCE AT HAND, it's not to say it has not already happened, just like with zoonosis of cwd i.e. molecular transmission studies have shown that cwd transmission to humans would look like sporadic cjd, NOT nvCJD or what they call now vCJD. the other thing is virulence and or horizontal transmission. this is very concerning with the recent fact of what seems to be a large outbreak of a new tse prion disease in camels in Africa. there is much concern now with hay, straw, grains, and such, with the cwd tse prion endemic countries USA, Canada. what is of greatest concern is the different strains of cwd, and the virulence there from? this thing (cwd) keeps mutating to different strains, and to different species, the bigger the chance of one of these strains that WILL TRANSMIT TO CATTLE OR HUMANS, and that it is documented (i believe both has already occured imo with scienct to date). with that said, a few things to ponder, and i am still very concerned with, the animal feed. we now know from transmission studies that cwd and scrapie will transmit to pigs by oral routes. the atypical bse strains will transmit by oral routes. i don't mean to keep kicking a mad cow, just look at the science;

***> cattle, pigs, sheep, cwd, tse, prion, oh my!

***> In contrast, cattle are highly susceptible to white-tailed deer CWD and mule deer CWD in experimental conditions but no natural CWD infections in cattle have been reported (Sigurdson, 2008; Hamir et al., 2006).

Sheep and cattle may be exposed to CWD via common grazing areas with affected deer but so far, appear to be poorly susceptible to mule deer CWD (Sigurdson, 2008). In contrast, cattle are highly susceptible to white-tailed deer CWD and mule deer CWD in experimental conditions but no natural CWD infections in cattle have been reported (Sigurdson, 2008; Hamir et al., 2006). It is not known how susceptible humans are to CWD but given that the prion can be present in muscle, it is likely that humans have been exposed to the agent via consumption of venison (Sigurdson, 2008). Initial experimental research suggests that human susceptibility to CWD is low and there may be a robust species barrier for CWD transmission to humans (Sigurdson, 2008), however the risk appetite for a public health threat may still find this level unacceptable.

https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/733407/DEFRA_QRA_TSE_in_cervids_June2018_v1.pdf

http://chronic-wasting-disease.blogspot.com/2012/08/susceptibility-of-cattle-to-agent-of.html

Friday, December 14, 2012

DEFRA U.K. What is the risk of Chronic Wasting Disease CWD being introduced into Great Britain? A Qualitative Risk Assessment October 2012

snip.....

In the USA, under the Food and Drug Administration's BSE Feed Regulation (21 CFR 589.2000) most material (exceptions include milk, tallow, and gelatin) from deer and elk is prohibited for use in feed for ruminant animals. With regards to feed for non-ruminant animals, under FDA law, CWD positive deer may not be used for any animal feed or feed ingredients. For elk and deer considered at high risk for CWD, the FDA recommends that these animals do not enter the animal feed system. However, this recommendation is guidance and not a requirement by law. Animals considered at high risk for CWD include:

1) animals from areas declared to be endemic for CWD and/or to be CWD eradication zones and

2) deer and elk that at some time during the 60-month period prior to slaughter were in a captive herd that contained a CWD-positive animal.

Therefore, in the USA, materials from cervids other than CWD positive animals may be used in animal feed and feed ingredients for non-ruminants.

The amount of animal PAP that is of deer and/or elk origin imported from the USA to GB can not be determined, however, as it is not specified in TRACES.

It may constitute a small percentage of the 8412 kilos of non-fish origin processed animal proteins that were imported from US into GB in 2011.

Overall, therefore, it is considered there is a __greater than negligible risk___ that (nonruminant) animal feed and pet food containing deer and/or elk protein is imported into GB.

There is uncertainty associated with this estimate given the lack of data on the amount of deer and/or elk protein possibly being imported in these products.

snip.....

36% in 2007 (Almberg et al., 2011). In such areas, population declines of deer of up to 30 to 50% have been observed (Almberg et al., 2011). In areas of Colorado, the prevalence can be as high as 30% (EFSA, 2011). The clinical signs of CWD in affected adults are weight loss and behavioural changes that can span weeks or months (Williams, 2005). In addition, signs might include excessive salivation, behavioural alterations including a fixed stare and changes in interaction with other animals in the herd, and an altered stance (Williams, 2005). These signs are indistinguishable from cervids experimentally infected with bovine spongiform encephalopathy (BSE). Given this, if CWD was to be introduced into countries with BSE such as GB, for example, infected deer populations would need to be tested to differentiate if they were infected with CWD or BSE to minimise the risk of BSE entering the human food-chain via affected venison. snip..... The rate of transmission of CWD has been reported to be as high as 30% and can approach 100% among captive animals in endemic areas (Safar et al., 2008).

snip.....

In summary, in endemic areas, there is a medium probability that the soil and surrounding environment is contaminated with CWD prions and in a bioavailable form. In rural areas where CWD has not been reported and deer are present, there is a greater than negligible risk the soil is contaminated with CWD prion. snip..... In summary, given the volume of tourists, hunters and servicemen moving between GB and North America, the probability of at least one person travelling to/from a CWD affected area and, in doing so, contaminating their clothing, footwear and/or equipment prior to arriving in GB is greater than negligible... For deer hunters, specifically, the risk is likely to be greater given the increased contact with deer and their environment. However, there is significant uncertainty associated with these estimates.

snip.....

Therefore, it is considered that farmed and park deer may have a higher probability of exposure to CWD transferred to the environment than wild deer given the restricted habitat range and higher frequency of contact with tourists and returning GB residents.

snip.....

https://web.archive.org/web/20170404125557/http://webarchive.nationalarchives.gov.uk/20130822084033/http://www.defra.gov.uk/animal-diseases/files/qra_chronic-wasting-disease-121029.pdf

***> READ THIS VERY, VERY, CAREFULLY, AUGUST 1997 MAD COW FEED BAN WAS A SHAM, AS I HAVE STATED SINCE 1997! 3 FAILSAFES THE FDA ET AL PREACHED AS IF IT WERE THE GOSPEL, IN TERMS OF MAD COW BSE DISEASE IN USA, AND WHY IT IS/WAS/NOT A PROBLEM FOR THE USA, and those are;

BSE TESTING (failed terribly and proven to be a sham)

BSE SURVEILLANCE (failed terribly and proven to be a sham)

BSE 589.2001 FEED REGULATIONS (another colossal failure, and proven to be a sham)

these are facts folks. trump et al just admitted it with the feed ban.

see;

FDA Reports on VFD Compliance

John Maday

August 30, 2019 09:46 AM VFD-Form 007 (640x427)

Before and after the current Veterinary Feed Directive rules took full effect in January, 2017, the FDA focused primarily on education and outreach. (John Maday) Before and after the current Veterinary Feed Directive (VFD) rules took full effect in January, 2017, the FDA focused primarily on education and outreach to help feed mills, veterinarians and producers understand and comply with the requirements. Since then, FDA has gradually increased the number of VFD inspections and initiated enforcement actions when necessary. On August 29, FDA released its first report on inspection and compliance activities. The report, titled “Summary Assessment of Veterinary Feed Directive Compliance Activities Conducted in Fiscal Years 2016 – 2018,” is available online.

https://www.fda.gov/media/130382/download

SUNDAY, SEPTEMBER 1, 2019

***> FDA Reports on VFD Compliance

https://bovineprp.blogspot.com/2019/09/fda-reports-on-vfd-compliance.html
THURSDAY, SEPTEMBER 26, 2019

Veterinary Biologics Guideline 3.32E: Guideline for minimising the risk of introducing transmissible spongiform encephalopathy prions and other infectious agents through veterinary biologics

https://bovineprp.blogspot.com/2019/09/veterinary-biologics-guideline-332e.html

U.S.A. 50 STATE BSE MAD COW CONFERENCE CALL Jan. 9, 2001

Subject: BSE--U.S. 50 STATE CONFERENCE CALL Jan. 9, 2001

Date: Tue, 9 Jan 2001 16:49:00 -0800

From: "Terry S. Singeltary Sr."

Reply-To: Bovine Spongiform Encephalopathy

To: BSE-L@uni-karlsruhe.de

snip...

[host Richard Barns] and now a question from Terry S. Singeltary of CJD Watch.

[TSS] yes, thank you, U.S. cattle, what kind of guarantee can you give for serum or tissue donor herds?

[no answer, you could hear in the back ground, mumbling and 'we can't. have him ask the question again.]

[host Richard] could you repeat the question?

[TSS] U.S. cattle, what kind of guarantee can you give for serum or tissue donor herds?

[not sure whom ask this] what group are you with?

[TSS] CJD Watch, my Mom died from hvCJD and we are tracking CJD world-wide.

[not sure who is speaking] could you please disconnect Mr. Singeltary

[TSS] you are not going to answer my question?

[not sure whom speaking] NO

snip...see full archive and more of this;

http://tseac.blogspot.com/2011/02/usa-50-state-bse-mad-cow-conference.html

Contains Nonbinding Recommendations

2

Guidance for Industry

Use of Material from Deer and Elk

in Animal Feed

This guidance represents the current thinking of the Food and Drug Administration (FDA or Agency) on this topic. It does not establish any rights for any person and is not binding on FDA or the public. You can use an alternative approach if it satisfies the requirements of the applicable statutes and regulations. To discuss an alternative approach, contact the FDA office responsible for this guidance as listed on the title page.

I. Introduction

Under FDA's BSE feed regulation (21 CFR 589.2000) most material from deer and elk is prohibited for use in feed for ruminant animals. This guidance document describes FDA's recommendations regarding the use in all animal feed of all material from deer and elk that are positive for Chronic Wasting Disease (CWD) or are considered at high risk for CWD. The potential risks from CWD to humans or non-cervid animals such as poultry and swine are not well understood. However, because of recent recognition that CWD is spreading rapidly in white-tailed deer, and because CWD's route of transmission is poorly understood, FDA is making recommendations regarding the use in animal feed of rendered materials from deer and elk that are CWD-positive or that are at high risk for CWD.

In general, FDA's guidance documents do not establish legally enforceable responsibilities. Instead, guidances describe the Agency's current thinking on a topic and should be viewed only as recommendations, unless specific

regulatory or statutory requirements are cited. The use of the word should in Agency guidances means that something is suggested or recommended, but not required.

II. Background

CWD is a neurological (brain) disease of farmed and wild deer and elk that belong in the animal family cervidae (cervids). Only deer and elk are known to be susceptible to CWD by natural transmission. The disease has been found in farmed and wild mule deer, white-tailed deer, North American elk, and in farmed black-tailed deer. CWD belongs to a family of animal and human diseases called transmissible spongiform encephalopathies (TSEs). These include bovine spongiform encephalopathy (BSE or “mad cow” disease) in cattle; scrapie in sheep and goats; and classical and variant Creutzfeldt-Jakob diseases (CJD and vCJD) in humans. There is no known treatment for these diseases, and there is no vaccine to prevent them. In addition, although validated postmortem diagnostic tests are available, there are no validated diagnostic tests for CWD that can be used to test for the disease in live animals.

Contains Nonbinding Recommendations

3

III. Use in animal feed of material from CWD-positive deer and elk

Material from CWD-positive animals may not be used in any animal feed or feed ingredients. Pursuant to Sec. 402(a)(5) of the Federal Food, Drug, and Cosmetic Act, animal feed and feed ingredients containing material from a CWD-positive animal would be considered adulterated. FDA recommends that any such adulterated feed or feed ingredients be recalled or otherwise removed from the marketplace.

IV. Use in animal feed of material from deer and elk considered at high risk for CWD Deer and elk considered at high risk for CWD include: (1) animals from areas declared by State officials to be endemic for CWD and/or to be CWD eradication zones; and (2) deer and elk that at some time during the 60-month period immediately before the time of slaughter were in a captive herd that contained a CWD-positive animal.

FDA recommends that materials from deer and elk considered at high risk for CWD no longer be entered into the animal feed system. Under present circumstances, FDA is not recommending that feed made from deer and elk from a non-endemic area be recalled if a State later declares the area endemic for CWD or a CWD eradication zone. In addition, at this time, FDA is not recommending that feed made from deer and elk believed to be from a captive herd that contained no CWD-positive animals be recalled if that herd is subsequently found to contain a CWD-positive animal.

V. Use in animal feed of material from deer and elk NOT considered at high risk for CWD

FDA continues to consider materials from deer and elk NOT considered at high risk for CWD to be acceptable for use in NON-RUMINANT animal feeds in accordance with current agency regulations, 21 CFR 589.2000. Deer and elk not considered at high risk include: (1) deer and elk from areas not declared by State officials to be endemic for CWD and/or to be CWD eradication zones; and (2) deer and elk that were not at some time during the 60-month period immediately before the time of slaughter in a captive herd that contained a CWD-positive animal.

Sunday, March 20, 2016

Docket No. FDA-2003-D-0432 (formerly 03D-0186) Use of Material from Deer and Elk in Animal Feed ***UPDATED MARCH 2016*** Singeltary Submission

http://www.fda.gov/downloads/AnimalVeterinary/GuidanceComplianceEnforcement/GuidanceforIndustry/UCM052506.pdf?source=govdelivery&utm_medium=email&utm_source=govdelivery

https://www.reginfo.gov/public/do/DownloadDocument?objectID=70082300

2003D-0186 Guidance for Industry: Use of Material From Deer and Elk In Animal Feed

EMC 1 Terry S. Singeltary Sr.

Vol #: 1

http://www.fda.gov/ohrms/dockets/dailys/03/Jun03/060903/060903.htm

http://www.fda.gov/ohrms/dockets/dailys/01/Oct01/101501/101501.htm

CWD AND SCRAPIE TRANSMITS BY ORAL ROUTES TO PIGS, PRICE OF TSE PRION POKER GOES UP!

2021 Transmissible Spongiform Encephalopathy TSE Prion End of Year Report 2020

CJD FOUNDATION VIRTUAL CONFERENCE CJD Foundation Research Grant Recipient Reports Panel 2 Nov 3, 2020

zoonotic potential of PMCA-adapted CWD PrP 96SS inoculum

https://youtu.be/VfazuR7cjMc?t=1992

4 different CWD strains, and these 4 strains have different potential to induce any folding of the human prion protein.

https://youtu.be/VfazuR7cjMc?t=2019

> PIGS, WILD BOAR, CWD <

***> POPULATIONS OF WILD BOARS IN THE UNITED STATES INCREASING SUPSTANTUALLY AND IN MANY AREAS WE CAN SEE A HIGH DENSITY OF WILD BOARS AND HIGH INCIDENT OF CHRONIC WASTING DISEASE

HYPOTHOSIS AND SPECIFIC AIMS

HYPOTHOSIS

BSE, SCRAPIE, AND CWD, EXPOSED DOMESTIC PIGS ACCUMULATE DIFFERENT QUANTITIES AND STRAINS OF PRIONS IN PERIPHERAL TISSUES, EACH ONE OF THEM WITH PARTICULAR ZOONOTIC POTENTIALS

https://youtu.be/VfazuR7cjMc

Final Report – CJD Foundation Grant Program A.

Project Title: Systematic evaluation of the zoonotic potential of different CWD isolates. Principal Investigator: Rodrigo Morales, PhD.

https://cjdfoundation.org/sites/default/files/grant-downloads/Final%20Report%20-%20CJD%20Foundation%20-%20Morales.pdf

Systematic evaluation of the zoonotic potential of different CWD isolates. Rodrigo Morales, PhD Assistant Professor Protein Misfolding Disorders lab Mitchell Center for Alzheimer's disease and Related Brain Disorders Department of Neurology University of Texas Health Science Center at Houston Washington DC. July 14th, 2018

Conclusions and Future Directions • We have developed a highly sensitive and specific CWD-PMCA platform to be used as a diagnostic tool. • Current PMCA set up allow us to mimic relevant prion inter-species transmission events. • Polymorphic changes at position 96 of the prion protein apparently alter strain properties and, consequently, the zoonotic potential of CWD isolates. • Inter-species and inter-polymorphic PrPC → PrPSc conversions further increase the spectrum of CWD isolates possibly present in nature. • CWD prions generated

in 96SS PrPC substrate apparently have greater inter-species transmission potentials. • Future experiments will explore the zoonotic potential of CWD prions along different adaptation scenarios, including inter-species and inter-polymorphic.

https://cjdfoundation.org/files/Conf2018/Rodrigo%20Morales%202018.pdf

https://www.youtube.com/watch?v=CzQKemJRBIE&list=PLGXRDfDPg57yTYvn6tifH13NrSiLjv1d7&index=7&t=0s

Research Project: TRANSMISSION, DIFFERENTIATION, AND PATHOBIOLOGY OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES Location: Virus and Prion Research

Title: Disease-associated prion protein detected in lymphoid tissues from pigs challenged with the agent of chronic wasting disease

Author item MOORE, SARAH - Orise Fellow item Kunkle, Robert item KONDRU, NAVEEN - Iowa State University item MANNE, SIREESHA - Iowa State University item SMITH, JODI - Iowa State University item KANTHASAMY, ANUMANTHA - Iowa State University item WEST GREENLEE, M - Iowa State University item Greenlee, Justin Submitted to: Prion Publication Type: Abstract Only Publication Acceptance Date: 3/15/2017 Publication Date: N/A Citation: N/A Interpretive Summary:

Technical Abstract: Aims: Chronic wasting disease (CWD) is a naturally-occurring, fatal neurodegenerative disease of cervids. We previously demonstrated that disease-associated prion protein (PrPSc) can be detected in the brain and retina from pigs challenged intracranially or orally with the CWD agent. In that study, neurological signs consistent with prion disease were observed only in one pig: an intracranially challenged pig that was euthanized at 64 months post-challenge. The purpose of this study was to use an antigen-capture immunoassay (EIA) and real-time quaking-induced conversion (QuIC) to determine whether PrPSc is present in lymphoid tissues from pigs challenged with the CWD agent.

Methods: At two months of age, crossbred pigs were challenged by the intracranial route (n=20), oral route (n=19), or were left unchallenged (n=9). At approximately 6 months of age, the time at which commercial pigs reach market weight, half of the pigs in each group were culled (<6 month challenge groups). The remaining pigs (>6 month challenge groups) were allowed to incubate for up to 73 months post challenge (mpc). The retropharyngeal lymph node (RPLN) was screened for the presence of PrPSc by EIA and immunohistochemistry (IHC). The RPLN, palatine tonsil, and mesenteric lymph node (MLN) from 6-7 pigs per challenge group were also tested using EIA and QuIC.

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=337105

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

Research Project: Pathobiology, Genetics, and Detection of Transmissible Spongiform Encephalopathies Location: Virus and Prion Research

Title: The agent of chronic wasting disease from pigs is infectious in transgenic mice expressing human PRNP

Author item MOORE, S - Orise Fellow item Kokemuller, Robyn item WEST-GREENLEE, M - Iowa State University item BALKEMA-BUSCHMANN, ANNE - Friedrich-Loeffler-institut item GROSCHUP, MARTIN - Friedrich-Loeffler-institut item Greenlee, Justin Submitted to: Prion Publication Type: Abstract Only Publication Acceptance Date: 5/10/2018 Publication Date: 5/22/2018 Citation: Moore, S.J., Kokemuller, R.D., West-Greenlee, M.H., Balkema-Buschmann, A., Groschup, M.H., Greenlee, J.J. 2018. The agent of chronic wasting disease from pigs is infectious in transgenic mice expressing human PRNP. Prion 2018, Santiago de Compostela, Spain, May 22-25, 2018. Paper No. WA15, page 44.

Interpretive Summary:

Technical Abstract: We have previously shown that the chronic wasting disease (CWD) agent from white-tailed deer can be transmitted to domestic pigs via intracranial or oral inoculation although with low attack rates and restricted PrPSc accumulation. The objective of this study was to assess the potential for cross-species transmission of pig-passaged CWD using bioassay in transgenic mice. Transgenic mice expressing human (Tg40), bovine (TgBovXV) or porcine (Tg002) PRNP were inoculated intracranially with 1% brain homogenate from a pig that had been intracranially inoculated with a pool of CWD from white-tailed deer. This pig developed neurological clinical signs, was euthanized at 64 months post-inoculation, and PrPSc was detected in the brain. Mice were monitored daily for clinical signs of disease until the end of the study. Mice were considered positive if PrPSc was detected in the brain using an enzyme immunoassay (EIA). In transgenic mice expressing porcine prion protein the average incubation period was 167 days post-inoculation (dpi) and 3/27 mice were EIA positive (attack rate = 11%). All 3 mice were found dead and clinical signs were not noted prior to death. One transgenic mouse expressing bovine prion protein was euthanized due to excessive scratching at 617 dpi and 2 mice culled at the end of the study at 700 dpi were EIA positive resulting in an overall attack rate of 3/16 (19%). None of the transgenic mice expressing human prion protein that died or were euthanized up to 769 dpi were EIA positive and at study end point at 800 dpi 2 mice had positive EIA results (overall attack rate = 2/20 = 10%). The EIA optical density (OD) readings for all positive mice were at the lower end of the reference range (positive mice range, OD = 0.266-0.438; test positive reference range, OD = 0.250-4.000). To the authors' knowledge, cervid-derived CWD isolates have not been successfully transmitted to transgenic mice expressing human prion protein. The successful transmission of pig-passaged CWD to Tg40 mice reported here suggests that passage of the CWD agent through pigs results in a change of the transmission characteristics which reduces the transmission barrier of Tg40 mice to the CWD agent. If this biological behavior is recapitulated in the original host species, passage of the CWD agent through pigs could potentially lead to increased pathogenicity of the CWD agent in humans.

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

cwd scrapie pigs oral routes

> 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

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

Research Project: TRANSMISSION, DIFFERENTIATION, AND PATHOBIOLOGY OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES Location: Virus and Prion Research

Title: Scrapie transmits to white-tailed deer by the oral route and has a molecular profile similar to chronic wasting disease

Author

item Greenlee, Justin item Moore, S - Orise Fellow item Smith, Jodi - Iowa State University item Kunkle, Robert item West Greenlee, M - Iowa State University Submitted to: American College of Veterinary Pathologists Meeting Publication Type: Abstract Only Publication Acceptance Date: 8/12/2015 Publication Date: N/A Citation: N/A

Interpretive Summary:

Technical Abstract: The purpose of this work was to determine susceptibility of white-tailed deer (WTD) to the agent of sheep scrapie and to compare the resultant PrPSc to that of the original inoculum and chronic wasting disease (CWD). We inoculated WTD by a natural route of exposure (concurrent oral and intranasal (IN); n=5) with a US scrapie isolate. All scrapie-inoculated deer had evidence of PrPSc accumulation. PrPSc was detected in lymphoid tissues at preclinical time points, and deer necropsied after 28 months post-inoculation had clinical signs, spongiform encephalopathy, and widespread distribution of PrPSc in neural and lymphoid tissues. Western blotting (WB) revealed PrPSc with 2 distinct molecular profiles. WB on cerebral cortex had a profile similar to the original scrapie inoculum, whereas WB of brainstem, cerebellum, or lymph nodes revealed PrPSc with a higher profile resembling CWD. Homogenates with the 2 distinct profiles from WTD with clinical scrapie were further passaged to mice expressing cervid prion protein and intranasally to sheep and WTD. In cervidized mice, the two inocula have distinct incubation times. Sheep inoculated intranasally with WTD derived scrapie developed disease, but only after inoculation with the inoculum that had a scrapie-like profile. The WTD study is ongoing, but deer in both inoculation groups are positive for PrPSc by rectal mucosal biopsy. In summary, this work demonstrates that WTD are susceptible to the agent of scrapie, two distinct molecular profiles of PrPSc are present in the tissues of affected deer, and inoculum of either profile readily passes to deer.

https://www.ars.usda.gov/research/publications/publication/?seqNo115=317901

223. Scrapie in white-tailed deer: a strain of the CWD agent that efficiently transmits to sheep?

Justin J. Greenleea, Robyn D. Kokemullera, S. Jo Moorea and Heather West Greenleeb

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ABSTRACT

Scrapie is a transmissible spongiform encephalopathy of sheep and goats that is associated with widespread accumulation of abnormal prion protein (PrPSc) in the central nervous and lymphoid tissues. Chronic wasting disease (CWD) is the natural prion disease of cervid species, and the tissue distribution of PrPSc in affected cervids is similar to scrapie in sheep. There are several lines of evidence that suggest that multiple strains of CWD exist, which may affect the agent's potential to transmit to hosts of the same or different species. We inoculated white-tailed deer with the scrapie agent from ARQ/ARQ sheep, which resulted in 100% attack rates by either the

intracranial or oronasal route of inoculation. When examining tissues from the brainstems or lymphoid tissues by traditional diagnostic methods such as immunohistochemistry or western blots, it is difficult to differentiate tissues from deer infected with scrapie from those infected with CWD. However, there are several important differences between tissues from scrapie-infected white-tailed deer (WTD scrapie) and those infected with CWD (WTD CWD). First, there are different patterns of PrPSc deposition in the brains of infected deer: brain tissues from deer with WTD scrapie had predominantly particulate and stellate immunoreactivity whereas those from deer with WTD-CWD had large aggregates and plaque-like deposits. Secondly, the incubation periods of WTD scrapie isolates are longer than CWD isolates in mice expressing cervid prion protein. Most notably, the transmission potential of these two isolates back to sheep is distinctly different. Attempts to transmit various CWD isolates to sheep by the oral or oronasal routes have been unsuccessful despite observation periods of up to 7 years. However, WTD scrapie efficiently transmitted back to sheep by the oronasal route. Upon transmission back to sheep, the WTD scrapie isolate exhibited different phenotypic properties when compared to the sheep receiving the original sheep scrapie inoculum including different genotype susceptibilities, distinct PrPSc deposition patterns, and much more rapid incubation periods in transgenic mice expressing the ovine prion protein. The scrapie agent readily transmits between sheep and deer after oronasal exposure. This could confound the identification of CWD strains in deer and the eradication of scrapie from sheep.

https://www.tandfonline.com/doi/full/10.1080/19336896.2019.1615197

HERE IS A FIND EXAMPLE OF ONE MAD COW FEED BAN WARNING LETTER, 9 YEARS POST MAD COW FEED BAN...

e) "Big Jim's" BBB Deer Ration, Big Buck Blend, Recall # V-104-6;

f) CO-OP 40% Hog Supplement Medicated Pelleted, Tylosin 100 grams/ton, 50 lb. bag, Recall # V-105-6;

g) Pig Starter Pell II, 18% W/MCDX Medicated 282020, Carbadox -- 0.0055%, Recall # V-106-6;

ALABAMA MAD COW FEED IN COMMERCE 2006

RECALLS AND FIELD CORRECTIONS: VETERINARY MEDICINE -- CLASS II

PRODUCT

a) CO-OP 32% Sinking Catfish, Recall # V-100-6;

b) Performance Sheep Pell W/Decox/A/N, medicated, net wt. 50 lbs, Recall # V-101-6;

c) Pro 40% Swine Conc Meal -- 50 lb, Recall # V-102-6;

d) CO-OP 32% Sinking Catfish Food Medicated, Recall # V-103-6;

e) "Big Jim's" BBB Deer Ration, Big Buck Blend, Recall # V-104-6;

f) CO-OP 40% Hog Supplement Medicated Pelleted, Tylosin 100 grams/ton, 50 lb. bag, Recall # V-105-6;

g) Pig Starter Pell II, 18% W/MCDX Medicated 282020, Carbadox -- 0.0055%, Recall # V-106-6;

h) CO-OP STARTER-GROWER CRUMBLES, Complete Feed for Chickens from Hatch to 20 Weeks, Medicated, Bacitracin Methylene Disalicylate, 25 and 50 Lbs, Recall # V-107-6;

i) CO-OP LAYING PELLETS, Complete Feed for Laying Chickens, Recall # 108-6;

j) CO-OP LAYING CRUMBLES, Recall # V-109-6;

k) CO-OP QUAIL FLIGHT CONDITIONER MEDICATED, net wt 50 Lbs, Recall # V-110-6;

l) CO-OP QUAIL STARTER MEDICATED, Net Wt. 50 Lbs, Recall # V-111-6;

m) CO-OP QUAIL GROWER MEDICATED, 50 Lbs, Recall # V-112-6

CODE

Product manufactured from 02/01/2005 until 06/06/2006

RECALLING FIRM/MANUFACTURER

Alabama Farmers Cooperative, Inc., Decatur, AL, by telephone, fax, email and visit on June 9, 2006. FDA initiated recall is complete.

REASON

Animal and fish feeds which were possibly contaminated with ruminant based protein not labeled as "Do not feed to ruminants".

VOLUME OF PRODUCT IN COMMERCE

125 tons

DISTRIBUTION

AL and FL

PRODUCT

Bulk custom dairy feds manufactured from concentrates, Recall # V-113-6

CODE

All dairy feeds produced between 2/1/05 and 6/16/06 and containing H. J. Baker recalled feed products.

RECALLING FIRM/MANUFACTURER

Vita Plus Corp., Gagetown, MI, by visit beginning on June 21, 2006. Firm initiated recall is complete.

REASON

The feed was manufactured from materials that may have been contaminated with mammalian protein.

VOLUME OF PRODUCT IN COMMERCE

27,694,240 lbs

DISTRIBUTION

MI

PRODUCT

Bulk custom made dairy feed, Recall # V-114-6

CODE

None

RECALLING FIRM/MANUFACTURER

Burkmann Feeds LLC, Glasgow, KY, by letter on July 14, 2006. Firm initiated recall is ongoing.

REASON

Custom made feeds contain ingredient called Pro-Lak, which may contain ruminant derived meat and bone meal.

VOLUME OF PRODUCT IN COMMERCE

?????

DISTRIBUTION

KY

END OF ENFORCEMENT REPORT FOR AUGUST 2, 2006

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http://data.nber.org/fda/enforcement-report/2006/ucm120413.htm

oral transmission of cwd to cervid is easily transmitted, and now we know cwd will transmit to pigs orally.

IT IS PARAMONT THAT THE FDA PART 589 TSE PRION FEED BAN LOOP HOLE STILL ALLOWING CERVID TO BE FED ANIMAL PROTEIN, SHOULD BE BANNED IMMEDIATELY ASAP!

***> 7TH TRUCKING TRANSPORTING CERVID CHRONIC WASTING DISEASE TSE PRION VIOLATING THE LACEY ACT

TUESDAY, JANUARY 21, 2020

***> 2004 European Commission Chronic wasting disease AND TISSUES THAT MIGHT CARRY A RISK FOR HUMAN FOOD AND ANIMAL FEED CHAINS REPORT UPDATED 2020

SNIP...

In summary, lateral transmission, compounded by animal movements is the most important factor in the spread of CWD. Indirect transmission via environmental contamination may play a role in natural dynamics and persistence of the disease and thus exacerbates epidemics and may present an obstacle to eradicating CWD from infected premises. This possibility and other epidemiological uncertainties also present significant obstacles to eradicating CWD from wildlife.

SEE;

https://ec.europa.eu/food/sites/food/files/safety/docs/sci-com_ssc_out324_en.pdf

PLEASE SEE HISTORY OF TEXAS TRUCKING CWD TSE PRION DISEASE AT THE BOTTOM OF MY SUBMISSION, TOO LONG TO POST HERE.

MONDAY, MARCH 05, 2018

TRUCKING AROUND AND SPREADING CHRONIC WASTING DISEASE CWD TSE PRION VIA MOVEMENT OF CERVID AND TRANSPORTATION VEHICLES

http://chronic-wasting-disease.blogspot.com/2018/03/trucking-around-and-spreading-chronic.html

SATURDAY, JULY 09, 2016

Texas Intrastate – within state movement of all Cervid or Trucking Chronic Wasting Disease CWD TSE Prion Moratorium

http://chronic-wasting-disease.blogspot.com/2016/07/texas-intrastate-within-state-movement.html

***> 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

TUESDAY, DECEMBER 31, 2019

In Vitro detection of Chronic Wasting Disease (CWD) prions in semen and reproductive tissues of white tailed deer bucks (Odocoileus virginianus

SUNDAY, AUGUST 02, 2015

TEXAS CWD, Have you been ThunderStruck, deer semen, straw bred bucks, super ovulation, and the potential TSE Prion connection, what if?

https://chronic-wasting-disease.blogspot.com/2019/12/in-vitro-detection-of-chronic-wasting.html

SUNDAY, FEBRUARY 16, 2020

***> Jerking for Dollars, Are Texas Politicians and Legislators Masturbating Deer For Money, and likely spreading CWD TSE Prion?

https://chronic-wasting-disease.blogspot.com/2020/02/jerking-for-dollars-are-texas.html

CAPTIVE FARMED CERVID SHOULD BE BANNED imo, TO SAVE THE WILD CERVID HERDS, OR AT LEAST, THE USDA/APHIS CERVID HERD CERTIFICATION PROGRAM SHOULD BE MADE MANDATORY, WITH REAL CWD TSE PRION CHANGES THAT CAN BE MADE AND THEN STRICTLY ENFORCED. CATERING TO THE CAPTIVE FARMED CERVID INDUSTRY IS HELPING THE SPREAD OF CWD TSE PRION, AND THIS MUST BE STOPPED, NO MORE VOLUNTARY ACTIONS, THEY DO NOT WORK!

WEDNESDAY, MARCH 13, 2019

CWD, TSE, PRION, MATERNAL mother to offspring, testes, epididymis, seminal fluid, and blood
Subject: Prion 2019 Conference

See full Prion 2019 Conference Abstracts

https://www.tandfonline.com/doi/full/10.1080/19336896.2019.1615197

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

https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/nvap/NVAP-Reference-Guide/Control-and-Eradication/Chronic-Wasting-Disease

179. PrPCWD detection in CWD-infected TgElk mice model using RT-QUIC

Hyun Joo Sohn, Kyung Je Park, In Soon Roh, Hyo Jin Kim, Hae Eun Kang

Foreign animal disease division, Animal and Plant Quarantine Agency, Gimcheon, Gyeongsangbuk-do, Korea

ABSTRACT

Introduction: Chronic wasting disease (CWD) is the only prion disease affecting free-ranging animals, reported in North America, South Korea, and Norway. Unlike in most other prion disease CWD agents are shed in blood, urine, and faeces which most likely contribute to the horizontal transmission between cervid species. The developments of amplification-based seeding assays have been instrumental in the detection of low levels of prions in clinical samples. Using real-time quaking-induced conversion (RT-QUIC), we established an ultrasensitive detection method for PrPCWD in the urine from CWD-infected sequentially sampled transgenic mice overexpressing elk prion protein (TgElk mice). In addtion, RT-QUIC was performed in the kidney and brain of theses mice model to trace abnormal prion.

Materials and Methods: 44 brain and kidney, urine samples from sequentially collected from CWD-infected TgElk mice (TgElk CWD) were stored at −80°C. In brain and kidney, 10% (w/v) homogenate was prepared in 0.9% sterilized saline. In urine 100 µL of each sample was mixed with 10 µL 2.8% sodium phosphotungustic acid (NaPTA) and incubated for 1hr at 37°C with shaking at 1,350 rpm. Samples were centrifuged for 30 min at 16,100 g. The pellet was resuspended in 10 µL of 0.1% SDS/PBS for 30 min at 55°C. RT-QUIC reactions were set up in 96-well clear bottom optic plates and consisted of 98 µL RT-QUIC buffer [final concentrations of 1XPBS, 1 mM EDTA, 10 µM Thioflavin, 300 mM NaCl buffer and 0.1 mg/ml recombinant Syrian hamster recombinant protein (23–231), and 2 µL of sample. The RT-QUIC assay was performed on a FLUOstar Omega fluorescence plate reader that was preheated to 42°C for 60 h with 90 s shaking at 700 rpm followed by 1 min incubation.

Results: Five randomly selected mice were sequentially culled on every 15 days from 30dpi to 120dpi during CWD infected TgElk mice reached terminal stage. Rough hair coats among clinical signs were showed from 90 dpi. PrPCWD in the brain in TgElk CWD was detectable persistently from early stages (30dpi), and in the kidney PrPCWD was also detectable in clinical and terminal stages (90 dpi and 120dpi). PrPCWD in the urine in TgElk CWD reached the highest levels at 120dpi. NaPTA/RT-QUIC was applied to measure PrPCWD in urine samples collected on every 15 days from 30dpi to 120dpi when CWD infected TgElk mice reached terminal stage. PrPCWD in the urine in TgElk CWD reached the highest levels at 90dpi. PrPCWD was also detectable in late and terminal stages (120dpi).

Conclusions: We demonstrate that CWD prions can be detected by RT-QUIC or NaPTA/RT-QUIC in the brain, kidney and urine of TgElk mice at the early and terminal stages of disease. Based on these data, we suggest that PrPCWD is excreted into only urine until 90 dpi and then slowly accumulated in kidney. Our results can be used in designing future study of CWD pathogenesis in TgElk mice.

References

Henderson DM et al., Rapid antemortem detection of CWD prions in deer saliva. PLOS one. 2013:e74377 1–12 [Google Scholar] Nagooka K, Yoshika M, Shimozaki N. et al., Sensitive detection of scrapie prion protein in soil. Biochem Biophys Res Commun. 2010;397:626–630. [Google Scholar]

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252. RT-QuIC as an antemortem diagnostic tool to detect chronic wasting disease in deer skin

Natália C. Ferreiraa, Jorge M. Charcob, Michael A. Metricka, Christina D. Orrua, Andrew G. Hughsona, Joaquín Castillaa, Michael W. Millerc and Byron Caugheya

aLaboratory of Persistent Viral Diseases, Rocky Mountain Laboratories, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Hamilton, MT, USA; 2 CIC bioGUNE, Derio (Bizkaia), Spain; 3Colorado Division of Parks and Wildlife, Wildlife Health Program, Fort Collins, Colorado, USA

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ABSTRACT

Chronic wasting disease (CWD) is a fatal prion disease which affects cervids. This disease has an asymptomatic incubation time of 2–4 years, and during this period they can shed prions through saliva, feces, urine and placental tissue, contaminating the environment. Indeed, CWD highly contagious between cervids and shedding from live, infected animals likely contributes to its rapid spread. So far, CWD has been reported at least in 24 states in the US, as well as Canada, South Korea and Norway. To date, there is no evidence of CWD transmission to humans. However, in infected areas, deer population can drop as much as ~25 percent. Currently, there are two tests approved to diagnose CWD: immunohistochemistry and ELISA. However, these tests are applied postmortem and tissue types approved are the medial retropharyngeal lymph nodes and a specific region of brainstem (obex). Here we describe our efforts to adapt the real time quaking-induced conversion (RT-QuIC) as a diagnostic tool to detect PrPCWD in deer ear skin. Our initial analysis of a blinded panel of 50 samples yielded 82% sensitivity and 75% specificity. We are working to improve the conditions and performance of this assay, given that it might be useful for antemortem CWD diagnostics and surveillance.

https://www.tandfonline.com/doi/full/10.1080/19336896.2019.1615197

33. Detection of CWD prions in third eyelids of deer and elk

Sarah K. Coopera, Clare E. Hoovera,b, Davin M. Hendersona, Nathaniel D. Denkersa, Candace K. Mathiasona and Edward A. Hoovera

aPrion Research Center, Department of Microbiology, Immunology, and Pathology, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, CO, USA; bAstraZeneca, Waltham, NJ, USA

CONTACT Sarah K. Cooper scooper2@rams.colostate.edu

ABSTRACT

Background: The increasing prevalence of CWD globally, makes critical the development of fast, cost-effective methods to detect the disease in hunter-harvested deer and assist wildlife population disease management. Based in part on the demonstration of PrPSc in the third eyelid lymphoid follicle in sheep with scrapie, we explored the third eyelid for its potential as a non-invasive and easily accessible lymphoid tissue that could be sampled without special anatomical training antemortem and postmortem for PrPCWD detection by real-time quaking induced conversion (RT-QuIC) and immunohistochemistry (IHC).

Methods: We compared RT-QuIC detection sensitivity in the third eyelid to the retropharyngeal lymph node and obex region of the brain from experimentally CWD inoculated white-tailed deer and naturally occurring subclinical elk using IHC. We examined symptomatic and asymptomatic white-tailed deer inoculated with CWD(+) and CWD(-) brain homogenate or saliva via the per os or aerosol route. In addition, we examined samples from asymptomatic, naturally exposed elk which were culled due to being RAMALT biopsy positive or suspect by RT-QuIC.

Results: We identified prion seeding activity in third eyelids in 24 out of 25 (96%) samples from terminal, experimentally CWD-infected deer and found RT-QuIC positivity in the third eyelid as early as 1 month after experimental exposure to CWD. In addition, we identified prion seeding activity in third eyelids in 17 out of 25 (68%) samples asymptomatic, naturally exposed elk. By contrast, IHC detected prion deposition in third eyelid lymphoid follicles in 5 of 10 deer (50%). We show that RT-QuIC can be more sensitive for CWD prion detection in the third eyelid compared to IHC.

Conclusions: RT-QuIC testing on the third eyelid is a rapid and sensitive means for post-mortem detection of CWD with potential to augment CWD diagnostics and hunter testing compliance.

Funding

Supported by NIH R01-NS-061902, P01-AI-077774, F30-ODO-118,143, T32-OD0-10,437

https://www.tandfonline.com/doi/full/10.1080/19336896.2019.1615197

https://www.maes.umn.edu/chronicwastingdisease

Transmissible Spongiform Encephalopathies in exotic species

In exotic species, the last one was in 2007.

SPECIES No. DATES AFFECTED

Ankole cow 2 1991, 95

Bison 1 1996

Cheetah 5 1992 – 98

Eland 6 1989 – 95

Gemsbok 1 1987

Kudu 6 1989 – 92

Asian Leopard Cat1 1 2005

Lion 5 1998 - 2007

Nyala 1 1986

Ocelot 3 1994 – 99

Oryx 2 1989, 92

Puma 3 1992 – 95

Tiger 3 1995 – 99

Data valid to 30 September 2019

1Felis (Prionailurus) bengalensis.

https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/840096/pub-tse-stats-exotic.pdf

ZOO ANIMALS AND TSE PRION DISEASE

The 82 zoo animals with BSE:

Id TSE Genus Species Subsp Birth Origin Death Place of Death

654 x Microcebus murinus - 1997 U.Montpellier 1998 U.Montpellier

656 x Microcebus murinus - 1997 U.Montpellier 1998 U.Montpellier

481 + Eulemur fulvus mayottensis 1974 Madagascar 1992 Montpellier zoo

474 + Eulemur fulvus mayottensis 1974 Madagascar 1990 Montpellier zoo

584 - Eulemur fulvus mayottensis 1984 Montpellier 1991 Montpellier zoo

455 + Eulemur fulvus mayottensis 1983 Montpellier 1989 Montpellier zoo

- + Eulemur fulvus mayottensis 1988 Montpellier 1992 Montpellier zoo

- + Eulemur fulvus mayottensis 1995 Montpellier 1996 Montpellier zoo

- + Eulemur fulvus albifrons 1988 Paris 1992 Montpellier zoo

- + Eulemur fulvus albifrons 1988 Paris 1990 Montpellier zoo

- + Eulemur fulvus albifrons 1988 Paris 1992 Montpellier zoo

456 + Eulemur fulvus albifrons 1988 Paris 1990 Montpellier zoo

586 + Eulemur mongoz - 1979 Madagascar 1998 Montpellier zoo

- p Eulemur mongoz - 1989 Mulhouse 1991 Montpellier zoo

- p Eulemur mongoz - 1989 Mulhouse 1990 Montpellier zoo

- p Eulemur macaco - 1986 Montpellier 1996 Montpellier zoo

- p Lemur catta - 1976 Montpellier 1994 Montpellier zoo

- p Varecia variegata variegata 1985 Mulhouse 1990 Montpellier zoo

- p Varecia variegata variegata 1993 xxx 1994 Montpellier zoo

455 + Macaca mulatta - 1986 Ravensden UK 1992 Montpellier zoo

- p Macaca mulatta - 1986 Ravensden UK 1993 Montpellier zoo

- p Macaca mulatta - 1988 Ravensden UK 1991 Montpellier zoo

- p Saimiri sciureus - 1987 Frejus France 1990 Frejus zoo

700 pc eulemur hybrid - - Besancon zoo 1998 Besancon zoo

701 pc eulemur hybrid - - Besancon zoo 1998 Besancon zoo

702 pc eulemur hybrid - - Besancon zoo 1998 Besancon zoo

703 pc eulemur hybrid - - Besancon zoo 1998 Besancon zoo

704 pc eulemur hybrid - - Besancon zoo 1998 Besancon zoo

705 pc eulemur hybrid - - Besancon zoo 1998 Besancon zoo

706 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

707 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

708 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

709 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

710 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

711 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

712 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

713 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

714 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

715 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

716 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

717 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

x p genus species - - Lille zoo 1996 Lille zoo

y p genus species - - Lille zoo 1996 Lille zoo

z p genus species - - Lille zoo 1996 Lille zoo

1 + Actinonyx jubatus cheetah 1986 Marwell zoo 1991 Pearle Coast AU

Duke + Actinonyx jubatus cheetah 1984 Marwell zoo 1992 Colchester zoo? UK

Saki + Actinonyx jubatus cheetah 1986 Marwell zoo 1993 unknown UK

Mich + Actinonyx jubatus cheetah 1986 Whipsnade 1993 Whipsnade UK

Fr1 + Actinonyx jubatus cheetah 1987 Whipsnade 1997 Safari de Peaugres FR

Fr2 + Actinonyx jubatus cheetah 1991 Marwell zoo 1997 Safari de Peaugres Fr

xx + Actinonyx jubatus cheetah 19xx xxx zoo 199x Fota zoo IR

yy + Actinonyx jubatus cheetah 19xx yyy zoo 1996+ yyyy zoo UK

zz + Actinonyx jubatus cheetah 19xx zzz zoo 1996+ yyyy zoo UK

aaa + Felis concolor puma 1986 Chester zoo 1991 Chester zoo UK

yy + Felis concolor puma 1980 yyy zoo 1995 yyyy zoo UK

zz + Felis concolor puma 1978 zzz zoo 1995 zzzz zoo UK

xxx + Felis pardalis ocelot 1987 xxx 1994 Chester zoo UK

zzz + Felis pardalis ocelot 1980 zzz 1995 zzzz zoo UK

85 + Felis catus cat 1990+ various 1999+ various UK LI NO

19 + Canis familia. dog 1992+ various 1999+ various UK

Fota + Panthera tigris tiger 1981 xxx zoo 1995 xxxx zoo UK

yy + Panthera tigris tiger 1983 yyy zoo 1998 yyyy zoo UK

Lump + Panthera leo lion 1986 Woburn SP 1998 Edinburgh zoo UK [since 1994]

1 + Taurotragus oryx eland 1987 Port Lympne 1989 Port Lympne zoo UK

Moll + Taurotragus oryx eland 1989 xx UK 1991 not Port Lympne UK

Nedd + Taurotragus oryx eland 1989 xx UK 1991 not Port Lympne UK

Elec + Taurotragus oryx eland 1990 xx UK 1992 not Port Lympne Uk

Daph p Taurotragus oryx eland 1988 xx UK 1990 not Port Lympne UK

zzz + Taurotragus oryx eland 1991 zz UK 1994 zzz UK

yyy + Taurotragus oryx eland 1993 yy UK 1995 yyy UK

Fran p Tragelaphus strepsi. kudu 1985 London zoo 1987 London zoo UK

Lind + Tragelaphus strepsi. kudu 1987 London zoo 1989 London zoo UK

Karl + Tragelaphus strepsi. kudu 1988 London zoo 1990 London zoo UK

Kaz + Tragelaphus strepsi. kudu 1988 London zoo 1991 London zoo UK

Bamb pc Tragelaphus strepsi. kudu 1988 London zoo 1991 London zoo UK

Step - Tragelaphus strepsi. kudu 1984 London zoo 1991 London zoo UK

346 pc Tragelaphus strepsi. kudu 1990 London zoo 1992 London zoo UK

324 + Tragelaphus strepsi. kudu 1989 Marwell zoo 1992 London zoo UK

xxx + Tragelaphus angasi nyala 1983 Marwell zoo 1986 Marwell zoo UK

yy + Oryx gazella gemsbok 1983 Marwell zoo 1986 Marwell zoo UK

zz + Oryx gazella gemsbok 1994+ zzz zoo 1996+ zzzz zoo UK

xx + Oryx dammah scim oryx 1990 xxxx zoo 1993 Chester zoo UK

yy + Oryx leucoryx arab oryx 1986 Zurich zoo 1991 London zoo UK

yy + Bos taurus ankole cow 1987 yyy zoo 1995 yyyy zoo UK

zz + Bos taurus ankole cow 1986 zzz zoo 1991 zzzz zoo UK

xx + Bison bison Eu bison 1989 xxx zoo 1996 xxxx zoo UK

http://www.mad-cow.org/may99_zoo_news.html

http://www.mad-cow.org/99feb_cwd_special.html#fff

http://www.mad-cow.org/00/aug00_late_news.html#ggg

http://www.mad-cow.org/00/aug00_last_news.html#fff

http://www.pnas.org/content/96/7/4046.full ;

THURSDAY, DECEMBER 19, 2019

TSE surveillance statistics exotic species and domestic cats Update December 2019

https://transmissiblespongiformencephalopathy.blogspot.com/2019/12/tse-surveillance-statistics-exotic.html

120. Detection of CWD prion seeding activity in faeces demonstrates both consistent shedding and potential for environmental monitoring

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ABSTRACT

Background: Chronic wasting disease (CWD) is spreading in susceptible cervid populations in North America, Korea, and Europe. Environmental contamination and exposure to prions is considered to be a significant factor in horizontal CWD transmission. Currently disease surveillance is limited by the necessary presence and testability of infected animals. Previous studies have shown that excreta from CWD infected deer and elk contains prion seeding activity. The detection of CWD through excreta, specifically faeces, could be beneficial in passive monitoring of CWD prevalence in endemic and emerging habitats.

Methods: Longitudinal collections of faeces from low-dose CWD inoculated deer were evaluated by real-time quaking induced conversion (RT-QuIC). To emulate common environmental weathering conditions, CWD-positive faecal samples were dried and exposed to UV light and prion seeding activity was evaluated by RT-QuIC before and after treatment. In addition, faeces from premises known to contain either CWD positive or negative cervids were collected and tested in RT-QuIC for prion seeding activity in a blinded experiment.

Results: CWD prion seeding activity was detected by RT-QuIC in faeces of five out of six low-dose CWD inoculated 96GG deer. The RT-QuIC detectable prion shedding in these deer corresponded with detection of PrPCWD by IHC in rectoanal mucosa-associated lymphoid tissue (RAMALT) biopsy and prion shedding was detected during both the pre-symptomatic and symptomatic stages of disease. Prion seeding activity was little affected by drying and exposure to ultraviolet light as both rectal collected and dried faeces showed prion seeding activity. Faeces collected from the pens containing CWD positive vs. negative deer correlated with presence vs. absence prion seeding activity, respectively.

Conclusion: These studies demonstrate that low-dose inoculated deer shed prion seeding activity in faeces through much of their disease course. Further, the prion seeding activity in faeces was still detectable after exposure to UV light and desiccation. These findings suggest that environmental monitoring of CWD via landscape faecal deposits may be possible as a means for monitoring CWD in populations of free-ranging deer and elk.

https://www.tandfonline.com/doi/full/10.1080/19336896.2019.1615197

172. Establishment of PrP^{CWD} extraction and detection methods in the farm soil

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ABSTRACT

Introduction: Transmissible spongiform encephalopathy (TSE) is a fatal neurodegenerative disorder, which is so-called as prion diseases due to the causative agents (PrP^{Sc}). TSEs are believed to be due to the template-directed accumulation of disease-associated prion protein, generally designated PrP^{Sc}. Chronic wasting disease (CWD) is the prion disease that is known spread horizontally. CWD has confirmed last in Republic of Korea in 2016 since first outbreak of CWD in 2001. The environmental reservoirs mediate the transmission of this disease. The significant levels of infectivity have been detected in the saliva, urine, and faeces of TSE-infected animals. Soil can serve as a stable reservoir for infectious prion proteins. We found that PrP^{CWD} can be extracted and detected in CWD contaminated soil which has kept at room temperature until 4 years after 0.001 ~ 1% CWD exposure and natural CWD-affected farm soil through PBS washing and sPMCAb.

Materials and Methods: Procedure of serial PMCAb. CWD contaminated soil which has kept at room temperature (RT) for 1 ~ 4 year after 0.001%~1% CWD brain homogenates exposure for 4 months collected 0.14 g. The soil was collected by the same method once of year until 4 year after stop CWD exposure. We had conducted the two steps. There are two kinds of 10 times washing step and one amplification step. The washing step was detached PrP^{Sc} from contaminated soil by strong vortex with maximum rpm. We harvest supernatant every time by 10 times. As the other washing step, the Washed soil was made by washing 10 times soil using slow rotator and then harvest resuspended PBS for removing large impurity material. Last step was prion amplification step for detection of PrP^{CWD} in soil supernatant and the washed soil by

sPMCAb. Normal brain homogenate (NBH) was prepared by homogenization of brains with glass dounce in 9 volumes of cold PBS with TritonX-100, 5 mM EDTA, 150 mM NaCl and 0.05% Digitonin (sigma) plus Complete mini protease inhibitors (Roche) to a final concentration of 5%(w/v) NBHs were centrifuged at 2000 g for 1 min, and supernatant removed and frozen at −70 C for use. CWD consisted of brain from natural case in Korea and was prepared as 10%(w/v) homogenate. Positive sample was diluted to a final dilution 1:1000 in NBH, with serial 3:7 dilutions in NBH. Sonication was performed with a Misonix 4000 sonicator with amplitude set to level 70, generating an average output of 160W with two teflon beads during each cycle. One round consisted of 56 cycles of 30 s of sonication followed 9 min 30 s of 37°C incubation. **Western Blotting (WB) for PrP^{Sc} detection**. The samples (20 µL) after each round of amplification were mixed with proteinase K (2 mg/ml) and incubated 37°C for 1 h. Samples were separated by SDS-PAGE and transferred onto PVDF membrane. After blocking, the membrane was incubated for 1 h with 1st antibody S1 anti rabbit serum (APQA, 1:3000) and developed with enhanced chemiluminescence detection system. **Results**: We excluded from first to third supernatant in view of sample contamination. It was confirmed abnormal PrP amplification in all soil supernatants from fourth to tenth. From 0.01% to 1% contaminated washed soils were identified as abnormal prions. 0.001% contaminated washed soil did not show PrP specific band (Fig 1). The soil was collected by the same method once of year until 4 year after stop CWD exposure. After sPMCAb, there were no PrP^{CWD} band in from second to fourth year 0.001% washed soil. but It was confirmed that the abnormal prion was amplified in the washing supernatant which was not amplified in the washed soil. we have decided to use soil supernatant for soil testing (Fig. 2). After third rounds of amplification, PrP^{Sc} signals observed in three out of four sites from CWD positive farm playground. No signals were observed in all soil samples from four CWD negative farm (Fig. 3).
Conclusions: Our studies showed that PrP^{CWD} persist in 0.001% CWD contaminated soil for at least 4 year and natural CWD-affected farm soil. When cervid reintroduced into CWD outbreak farm, the strict decontamination procedures of the infectious agent should be performed in the environment of CWD-affected cervid habitat.
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186. Serial detection of hematogenous prions in CWD-infected deer

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CONTACT Amy V. Nalls amy.nalls@colostate.edu
ABSTRACT
Blood contains the infectious agent associated with prion disease affecting several mammalian species, including humans, cervids, sheep, and cattle. It has been confirmed that sufficient prion agent is present in the blood of both symptomatic and asymptomatic carriers to initiate the amyloid templating and accumulation process that results in this fatal neurodegenerative disease. Yet, to date, the ability to detect blood-borne prions by *in vitro* methods remains difficult.

We have capitalized on blood samples collected from longitudinal chronic wasting disease (CWD) studies in the native white-tailed deer host to examine hematogenous prion load in blood collected minutes, days, weeks and months post exposure. Our work has focused on refinement of the amplification methods RT-QuIC and PMCA. We demonstrate enhanced *in vitro* detection of amyloid seeding activity (prions) in blood cell fractions harvested from deer orally-exposed to 300 ng CWD positive brain or saliva. These findings permit assessment of the role hematogenous prions play in the pathogenesis of CWD and provide tools to assess the same for prion diseases of other mammalian species.
https://www.tandfonline.com/doi/full/10.1080/19336896.2019.1615197

Considering the oral secretion of prions, saliva from CWD-infected deer was shown to transmit disease to other susceptible naïve deer when harvested from the animals in both the prions in the saliva and blood of deer with chronic wasting disease.

and preclinical stages69

of infection, albeit within relatively large volumes of saliva (50 ml). In sheep with preclinical, natural scrapie infections, sPMCA facilitated the detection of PrP^{Sc} within buccal swabs throughout most of the incubation period of the disease with an apparent peak in prion secretion around the mid-term of disease progression.70

The amounts of prion present in saliva are likely to be low as indicated by CWD-infected saliva producing prolonged incubation periods and incomplete attack rates within the transgenic mouse bioassay.41

snip...

Indeed, it has also been shown that the scrapie and CWD prions are excreted in urine, feces and saliva and are likely to be excreted from skin. While levels of prion within these excreta/secreta are very low, they are produced throughout long periods of preclinical disease as well as clinical disease. Furthermore, the levels of prion in such materials are likely to be increased by concurrent inflammatory conditions affecting the relevant secretory organ or site. Such dissemination of prion into the environment is very likely to facilitate the repeat exposure of flockmates to low levels of the disease agent, possibly over years.

snip...

Given the results with scrapie-contaminated milk and CWD-contaminated saliva, it seems very likely that these low levels of prion in different secreta/excreta are capable of transmitting disease upon prolonged exposure, either through direct animal-to-animal contact or through environmental reservoirs of infectivity.

https://www.tandfonline.com/doi/full/10.4161/pri.4.4.13678

the other part, these tissues and things in the body then shed or secrete prions which then are the route to other animals into the environment, so in particular, the things, the secretions that are infectious are salvia, feces, blood and urine. so pretty much anything that comes out of a deer is going to be infectious and potential for transmitting disease.

https://www.youtube.com/watch?v=bItnEElzuKo&index=6&list=PL7ZG8MkruQh3wI96XQ8_EymytO828rGxj

HUNTERS, CWD TSE PRION, THIS SHOULD A WAKE UP CALL TO ALL OF YOU GUTTING AND BONING OUT YOUR KILL IN THE FIELD, AND YOUR TOOLS YOU USE...

* 1: J Neurol Neurosurg Psychiatry 1994 Jun;57(6):757-8
Transmission of Creutzfeldt-Jakob disease to a chimpanzee by electrodes contaminated during neurosurgery.

Gibbs CJ Jr, Asher DM, Kobrine A, Amyx HL, Sulima MP, Gajdusek DC.

Laboratory of Central Nervous System Studies, National Institute of

Neurological Disorders and Stroke, National Institutes of Health,

Bethesda, MD 20892.

Stereotactic multicontact electrodes used to probe the cerebral cortex of a middle aged woman with progressive dementia were previously implicated in the accidental transmission of Creutzfeldt-Jakob disease (CJD) to two younger patients. The diagnoses of CJD have been confirmed for all three cases. More than two years after their last use in humans, after three cleanings and repeated sterilisation in ethanol and formaldehyde vapour, the electrodes were implanted in the cortex of a chimpanzee. Eighteen months later the animal became ill with CJD. This finding serves to re-emphasise the potential danger posed by reuse of instruments contaminated with the agents of spongiform encephalopathies, even after scrupulous attempts to clean them.

PMID: 8006664 [PubMed - indexed for MEDLINE]

http://jnnp.bmj.com/content/57/6/757.long
Monday, November 30, 2020

CAMEL PRION DISEASE OR MAD CAMEL DISASE

***>Tunisia has become the second country after Algeria to detect a case of CPD within a year

https://camelusprp.blogspot.com/2020/11/tunisia-has-become-second-country-after.html

> However, to date, no CWD infections have been reported in people.

key word here is ‘reported’. science has shown that CWD in humans will look like sporadic CJD. SO, how can one assume that CWD has not already transmitted to humans? they can’t, and it’s as simple as that. from all recorded science to date, CWD has already transmitted to humans, and it’s being misdiagnosed as sporadic CJD. …terry

*** LOOKING FOR CWD IN HUMANS AS nvCJD or as an ATYPICAL CJD, LOOKING IN ALL THE WRONG PLACES $$$ ***

*** 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

Chronic Wasting Disease CWD TSE Prion aka mad deer disease zoonosis

We hypothesize that:

(1) The classic CWD prion strain can infect humans at low levels in the brain and peripheral lymphoid tissues;

(2) The cervid-to-human transmission barrier is dependent on the cervid prion strain and influenced by the host (human) prion protein (PrP) primary sequence;

(3) Reliable essays can be established to detect CWD infection in humans; and

(4) CWD transmission to humans has already occurred. We will test these hypotheses in 4 Aims using transgenic (Tg) mouse models and complementary in vitro approaches.

http://grantome.com/grant/NIH/R01-NS088604-04

ZOONOTIC CHRONIC WASTING DISEASE CWD TSE PRION UPDATE

Prion 2017 Conference

First evidence of intracranial and peroral transmission of Chronic Wasting Disease (CWD) into Cynomolgus macaques: a work in progress Stefanie Czub1, Walter Schulz-Schaeffer2, Christiane Stahl-Hennig3, Michael Beekes4, Hermann Schaetzl5 and Dirk Motzkus6 1

University of Calgary Faculty of Veterinary Medicine/Canadian Food Inspection Agency; 2Universitatsklinikum des Saarlandes und Medizinische Fakultat der Universitat des Saarlandes; 3 Deutsches Primaten Zentrum/Goettingen; 4 Robert-Koch-Institut Berlin; 5 University of Calgary Faculty of Veterinary Medicine; 6 presently: Boehringer Ingelheim Veterinary Research Center; previously: Deutsches Primaten Zentrum/Goettingen

This is a progress report of a project which started in 2009. 21 cynomolgus macaques were challenged with characterized CWD material from white-tailed deer (WTD) or elk by intracerebral (ic), oral, and skin exposure routes. Additional blood transfusion experiments are supposed to assess the CWD contamination risk of human blood product. Challenge materials originated from symptomatic cervids for ic, skin scarification and partially per oral routes (WTD brain). Challenge material for feeding of muscle derived from preclinical WTD and from preclinical macaques for blood transfusion experiments. We have confirmed that the

CWD challenge material contained at least two different CWD agents (brain material) as well as CWD prions in muscle-associated nerves.

Here we present first data on a group of animals either challenged ic with steel wires or per orally and sacrificed with incubation times ranging from 4.5 to 6.9 years at postmortem. Three animals displayed signs of mild clinical disease, including anxiety, apathy, ataxia and/or tremor. In four animals wasting was observed, two of those had confirmed diabetes. All animals have variable signs of prion neuropathology in spinal cords and brains and by supersensitive IHC, reaction was detected in spinal cord segments of all animals. Protein misfolding cyclic amplification (PMCA), real-time quaking-induced conversion (RT-QuiC) and PET-blot assays to further substantiate these findings are on the way, as well as bioassays in bank voles and transgenic mice.

At present, a total of 10 animals are sacrificed and read-outs are ongoing. Preclinical incubation of the remaining macaques covers a range from 6.4 to 7.10 years. Based on the species barrier and an incubation time of > 5 years for BSE in macaques and about 10 years for scrapie in macaques, we expected an onset of clinical disease beyond 6 years post inoculation.

PRION 2017 DECIPHERING NEURODEGENERATIVE DISORDERS

PRION 2018 CONFERENCE

Oral transmission of CWD into Cynomolgus macaques: signs of atypical disease, prion conversion and infectivity in macaques and bio-assayed transgenic mice

Hermann M. Schatzl, Samia Hannaoui, Yo-Ching Cheng, Sabine Gilch (Calgary Prion Research Unit, University of Calgary, Calgary, Canada) Michael Beekes (RKI Berlin), Walter Schulz-Schaeffer (University of Homburg/Saar, Germany), Christiane Stahl-Hennig (German Primate Center) & Stefanie Czub (CFIA Lethbridge).

To date, BSE is the only example of interspecies transmission of an animal prion disease into humans. The potential zoonotic transmission of CWD is an alarming issue and was addressed by many groups using a variety of in vitro and in vivo experimental systems. Evidence from these studies indicated a substantial, if not absolute, species barrier, aligning with the absence of epidemiological evidence suggesting transmission into humans. Studies in non-human primates were not conclusive so far, with oral transmission into new-world monkeys and no transmission into old-world monkeys. Our consortium has challenged 18 Cynomolgus macaques with characterized CWD material, focusing on oral transmission with muscle tissue. Some macaques have orally received a total of 5 kg of muscle material over a period of 2 years.

After 5-7 years of incubation time some animals showed clinical symptoms indicative of prion disease, and prion neuropathology and PrPSc deposition were detected in spinal cord and brain of some euthanized animals. PrPSc in immunoblot was weakly detected in some spinal cord materials and various tissues tested positive in RT-QuIC, including lymph node and

spleen homogenates. To prove prion infectivity in the macaque tissues, we have intracerebrally inoculated 2 lines of transgenic mice, expressing either elk or human PrP. At least 3 TgElk mice, receiving tissues from 2 different macaques, showed clinical signs of a progressive prion disease and brains were positive in immunoblot and RT-QuIC. Tissues (brain, spinal cord and spleen) from these and pre-clinical mice are currently tested using various read-outs and by second passage in mice. Transgenic mice expressing human PrP were so far negative for clear clinical prion disease (some mice >300 days p.i.). In parallel, the same macaque materials are inoculated into bank voles.

Taken together, there is strong evidence of transmissibility of CWD orally into macaques and from macaque tissues into transgenic mouse models, although with an incomplete attack rate.

The clinical and pathological presentation in macaques was mostly atypical, with a strong emphasis on spinal cord pathology.
Our ongoing studies will show whether the transmission of CWD into macaques and passage in transgenic mice represents a form of non-adaptive prion amplification, and whether macaque-adapted prions have the potential to infect mice expressing human PrP.

The notion that CWD can be transmitted orally into both new-world and old-world non-human primates asks for a careful reevaluation of the zoonotic risk of CWD..

> The notion that CWD can be transmitted orally into both new-world and old-world non-human primates asks for a careful reevaluation of the zoonotic risk of CWD. <

https://prion2018.org/

READING OVER THE PRION 2018 ABSTRACT BOOK, LOOKS LIKE THEY FOUND THAT from this study ;

P190 Human prion disease mortality rates by occurrence of chronic wasting disease in freeranging cervids, United States

Abrams JY (1), Maddox RA (1), Schonberger LB (1), Person MK (1), Appleby BS (2), Belay ED (1) (1) Centers for Disease Control and Prevention (CDC), National Center for Emerging and Zoonotic Infectious Diseases, Atlanta, GA, USA (2) Case Western Reserve University, National Prion Disease Pathology Surveillance Center (NPDPSC), Cleveland, OH, USA..

SEEMS THAT THEY FOUND Highly endemic states had a higher rate of prion disease mortality compared to non-CWD
states.

AND ANOTHER STUDY;

P172 Peripheral Neuropathy in Patients with Prion Disease

Wang H(1), Cohen M(1), Appleby BS(1,2) (1) University Hospitals Cleveland Medical Center, Cleveland, Ohio (2) National Prion Disease Pathology Surveillance Center, Cleveland, Ohio..

IN THIS STUDY, THERE WERE autopsy-proven prion cases from the National Prion Disease Pathology Surveillance Center that were diagnosed between September 2016 to March 2017,

AND

included 104 patients. SEEMS THEY FOUND THAT The most common sCJD subtype was MV1-2 (30%), followed by MM1-2 (20%),

AND

THAT The Majority of cases were male (60%), AND half of them had exposure to wild game.

snip…

see more on Prion 2017 Macaque study from Prion 2017 Conference and other updated science on cwd tse prion zoonosis below…terry

https://prion2018.org/wp-content/uploads/2018/05/program.pdf

https://prion2018.org/

8. Even though human TSE-exposure risk through consumption of game from European cervids can be assumed to be minor, if at all existing, no final conclusion can be drawn due to the overall lack of scientific data. In particular the US data do not clearly exclude the possibility of human (sporadic or familial) TSE development due to consumption of venison. The Working Group thus recognizes a potential risk to consumers if a TSE would be present in European cervids. It might be prudent considering appropriate measures to reduce such a risk, e.g. excluding tissues such as CNS and lymphoid tissues from the human food chain, which would greatly reduce any potential risk for consumers. However, it is stressed that currently, no data regarding a risk of TSE infections from cervid products are available.

https://efsa.onlinelibrary.wiley.com/doi/full/10.2903/j.efsa.2018.5132

156. Screening and characterization of unusual sCJD cases in a CWD endemic state in the USA

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CONTACT Qingzhong Kong qxk2@case.edu

ABSTRACT

Background: Chronic wasting disease (CWD) has spread to 26 states in the USA and three provinces in Canada, and it has been detected recently in Norway and Finland. Potential CWD zoonosis is a serious public health concern. It is unclear whether CWD transmission to humans has already occurred. We aim to start to address this question by examining all available sCJD cases from a CWD endemic state in the USA.

Methods: Frozen brain tissues from all available sCJD cases archived in the National Prion Disease Pathology Surveillance Center from a US state that has been significantly impacted by CWD were sampled at five brain regions. These brain samples were subjected to detailed biochemical analysis to look for unusual patterns, characteristics, and/or distribution of PrPSc in comparison with sCJD samples from states that have not detected CWD. Unusual cases are further scrutinized for their clinical presentations, histopathological features, and history of cervid hunting and venison consumption.

Results and Conclusions: We have found some unusual sCJD cases in this CWD endemic state. We will report our preliminary findings on their features. Currently there is no convincing evidence to support a direct link to CWD for any of these unusual sCJD cases.

Disclaimer: The findings and conclusions in this report are those of the authors and do not necessarily represent the position of the National Prion Disease Pathology Surveillance Center.

https://www.tandfonline.com/doi/full/10.1080/19336896.2019.1615197

International Conference on Emerging Diseases, Outbreaks & Case Studies & 16th Annual Meeting on Influenza March 28-29, 2018 | Orlando, USA

Qingzhong Kong

Case Western Reserve University School of Medicine, USA

Zoonotic potential of chronic wasting disease prions from cervids

Chronic wasting disease (CWD) is the prion disease in cervids (mule deer, white-tailed deer, American elk, moose, and reindeer). It has become an epidemic in North America, and it has been detected in the Europe (Norway) since 2016. The widespread CWD and popular hunting and consumption of cervid meat and other products raise serious public health concerns, but questions remain on human susceptibility to CWD prions, especially on the potential difference in zoonotic potential among the various CWD prion strains. We have been working to address this critical question for well over a decade. We used CWD samples from various cervid species to inoculate transgenic mice expressing human or elk prion protein (PrP). We found infectious prions in the spleen or brain in a small fraction of CWD-inoculated transgenic mice expressing human PrP, indicating that humans are not completely resistant to CWD prions; this finding has significant ramifications on the public health impact of CWD prions. The influence of cervid PrP polymorphisms, the prion strain dependence of CWD-to-human transmission barrier, and the characterization of experimental human CWD prions will be discussed.

Speaker Biography Qingzhong Kong has completed his PhD from the University of Massachusetts at Amherst and Post-doctoral studies at Yale University. He is currently an Associate Professor of Pathology, Neurology and Regenerative Medicine. He has published over 50 original research papers in reputable journals (including Science Translational Medicine, JCI, PNAS and Cell Reports) and has been serving as an Editorial Board Member on seven scientific journals. He has multiple research interests, including public health risks of animal prions (CWD of cervids and atypical BSE of cattle), animal modeling of human prion diseases, mechanisms of prion replication and pathogenesis, etiology of sporadic Creutzfeldt-Jacob disease (CJD) in humans, normal cellular PrP in the biology and pathology of multiple brain and peripheral diseases, proteins responsible for the α-cleavage of cellular PrP, as well as gene therapy and DNA vaccination.

qxk2@case.edu

https://www.alliedacademies.org/conference-abstracts-files/zoonotic-potential-of-chronic-wasting-disease-prions-from.pdf

https://prionconference.blogspot.com/2018/02/prion-round-table-conference-2018-may.html

http://prionconference.blogspot.com/2018/02/prion-round-table-conference-2018-may.html

http://prionconference.blogspot.com/

SATURDAY, FEBRUARY 23, 2019

Chronic Wasting Disease CWD TSE Prion and THE FEAST 2003 CDC an updated review of the science 2019

https://chronic-wasting-disease.blogspot.com/2019/02/chronic-wasting-disease-cwd-tse-prion.html

TUESDAY, NOVEMBER 04, 2014

Six-year follow-up of a point-source exposure to CWD contaminated venison in an Upstate New York community: risk behaviours and health outcomes 2005–2011

Authors, though, acknowledged the study was limited in geography and sample size and so it couldn't draw a conclusion about the risk to humans. They recommended more study. Dr. Ermias Belay was the report's principal author but he said New York and Oneida County officials are following the proper course by not launching a study. "There's really nothing to monitor presently. No one's sick," Belay said, noting the disease's incubation period in deer and elk is measured in years. "

http://chronic-wasting-disease.blogspot.com/2014/11/six-year-follow-up-of-point-source.html

Transmission Studies

Mule deer transmissions of CWD were by intracerebral inoculation and compared with natural cases {the following was written but with a single line marked through it "first passage (by this route)}....TSS

resulted in a more rapidly progressive clinical disease with repeated episodes of synocopy ending in coma. One control animal became affected, it is believed through contamination of inoculum (?saline). Further CWD transmissions were carried out by Dick Marsh into ferret, mink and squirrel monkey. Transmission occurred in ALL of these species with the shortest incubation period in the ferret.

snip....

https://web.archive.org/web/20090506002237/http://www..bseinquiry.gov.uk/files/mb/m11b/tab01.pdf

Prion Infectivity in Fat of Deer with Chronic Wasting Disease▿

Brent Race#, Kimberly Meade-White#, Richard Race and Bruce Chesebro* + Author Affiliations

In mice, prion infectivity was recently detected in fat. Since ruminant fat is consumed by humans and fed to animals, we determined infectivity titers in fat from two CWD-infected deer. Deer fat devoid of muscle contained low levels of CWD infectivity and might be a risk factor for prion infection of other species.

http://jvi.asm.org/content/83/18/9608.full

Prions in Skeletal Muscles of Deer with Chronic Wasting Disease

Here bioassays in transgenic mice expressing cervid prion protein revealed the presence of infectious prions in skeletal muscles of CWD-infected deer, demonstrating that humans consuming or handling meat from CWD-infected deer are at risk to prion exposure.

http://science.sciencemag.org/content/311/5764/1117..long

*** 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"

From: TSS

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

-----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***,

snip... full text ;

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

> However, to date, no CWD infections have been reported in people.

sporadic, spontaneous CJD, 85%+ of all human TSE, just not just happen. never in scientific literature has this been proven.

if one looks up the word sporadic or spontaneous at pubmed, you will get a laundry list of disease that are classified in such a way;

sporadic = 54,983 hits https://www.ncbi.nlm.nih.gov/pubmed/?term=sporadic

spontaneous = 325,650 hits https://www.ncbi.nlm.nih.gov/pubmed/?term=spontaneous

key word here is 'reported'. science has shown that CWD in humans will look like sporadic CJD. SO, how can one assume that CWD has not already transmitted to humans? they can't, and it's as simple as that. from all recorded science to date, CWD has already transmitted to humans, and it's being misdiagnosed as sporadic CJD. ...terry

*** LOOKING FOR CWD IN HUMANS AS nvCJD or as an ATYPICAL CJD, LOOKING IN ALL THE WRONG PLACES $$$ ***

*** 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

*** IF CWD is not a risk factor for humans, then I guess the FDA et al recalled all this CWD tainted elk tenderloin (2009 Exotic Meats USA of San Antonio, TX) for the welfare and safety of the dead elk. ...tss
Exotic Meats USA Announces Urgent Statewide Recall of Elk Tenderloin Because It May Contain Meat Derived From An Elk Confirmed To Have Chronic Wasting Disease
Contact: Exotic Meats USA **1-800-680-4375**
FOR IMMEDIATE RELEASE -- February 9, 2009 -- Exotic Meats USA of San Antonio, TX is initiating a voluntary recall of Elk Tenderloin because it may contain meat derived from an elk confirmed to have Chronic Wasting Disease (CWD). The meat with production dates of December 29, 30 and 31, 2008 was purchased from Sierra Meat Company in Reno, NV. The infected elk came from Elk Farm LLC in Pine Island, MN and was among animals slaughtered and processed at USDA facility Noah's Ark Processors LLC.
Chronic Wasting Disease (CWD) is a fatal brain and nervous system disease found in elk and deer. The disease is caused by an abnormally shaped protein called a prion, which can damage the brain and nerves of animals in the deer family. Currently, it is believed that the prion responsible for causing CWD in deer and elk is not capable of infecting humans who eat deer or elk contaminated with the prion, but the observation of animal-to-human transmission of other prion-mediated diseases, such as bovine spongiform encephalopathy (BSE), has raised a theoretical concern regarding the transmission of CWD from deer or elk to humans. At the present time, FDA believes the risk of becoming ill from eating CWD-positive elk or deer meat is remote. However, FDA strongly advises consumers to return the product to the place of purchase, rather than disposing of it themselves, due to environmental concerns.
Exotic Meats USA purchased 1 case of Elk Tenderloins weighing 16.9 lbs. The Elk Tenderloin was sold from January 16 – 27, 2009. The Elk Tenderloins was packaged in individual vacuum packs weighing approximately 3 pounds each. A total of six packs of the Elk Tenderloins were sold to the public at the Exotic Meats USA retail store. Consumers who still have the Elk Tenderloins should return the product to Exotic Meats USA at 1003 NE Loop 410, San Antonio, TX 78209. Customers with concerns or questions about the Voluntary Elk Recall can call **1-800-680-4375**. The safety of our customer has always been and always will be our number one priority.
Exotic Meats USA requests that for those customers who have products with the production dates in question, do not consume or sell them and return them to the point of purchase. Customers should return the product to the vendor. The vendor should return it to the distributor and the distributor should work with the state to decide upon how best to dispose. If the consumer is disposing of the product he/she should consult with the local state EPA office.
#
RSS Feed for FDA Recalls Information11 [what's this?12]
http://www.fda.gov/Safety/Recalls/ArchiveRecalls/2009/ucm128543.htm
USGS Outstanding in the Field podcast, Episode 3: Chronic Wasting Disease - Oh, Deer (Credit: USGS)

https://prd-wret.s3.us-west-2.amazonaws.com/assets/palladium/production/s3fs-public/atoms/audio/20190628_3_oh_deerpodcast.mp3

https://www.usgs.gov/news/chronic-wasting-disease-can-science-save-our-dear-deer?qt-news_science_products=1#qt-news_science_products

TUESDAY, DECEMBER 29, 2020

Chronic Wasting Disease: Can Science Save Our Dear Deer?

https://chronic-wasting-disease.blogspot.com/2020/12/chronic-wasting-disease-can-science.html

FRIDAY, JULY 26, 2019

Chronic Wasting Disease in Cervids: Implications for Prion Transmission to Humans and Other Animal Species

https://chronic-wasting-disease.blogspot.com/2019/07/chronic-wasting-disease-in-cervids.html

USGS Outstanding in the Field podcast, Episode 3: Chronic Wasting Disease - Oh, Deer (Credit: USGS)

https://prd-wret.s3.us-west-2.amazonaws.com/assets/palladium/production/s3fs-public/atoms/audio/20190628_3_oh_deerpodcast.mp3

https://www.usgs.gov/news/chronic-wasting-disease-can-science-save-our-dear-deer?qt-news_science_products=1#qt-news_science_products

TUESDAY, DECEMBER 29, 2020

Chronic Wasting Disease: Can Science Save Our Dear Deer?

https://chronic-wasting-disease.blogspot.com/2020/12/chronic-wasting-disease-can-science.html

FRIDAY, JULY 26, 2019

Chronic Wasting Disease in Cervids: Implications for Prion Transmission to Humans and Other Animal Species

https://chronic-wasting-disease.blogspot.com/2019/07/chronic-wasting-disease-in-cervids.html

TUESDAY, JANUARY 21, 2020

***> 2004 European Commission Chronic wasting disease AND TISSUES THAT MIGHT CARRY A RISK FOR HUMAN FOOD AND ANIMAL FEED CHAINS REPORT UPDATED 2020

https://chronic-wasting-disease.blogspot.com/2020/01/2004-european-commission-chronic.html

CWD TSE PRION AND ZOONOTIC, ZOONOSIS, POTENTIAL

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@ References: <3daf5023 .4080804="" wt.net="">

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 ==========

https://caninespongiformencephalopathy.blogspot.com/2010/03/canine-spongiform-encephalopathy-aka.html

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...see full text;

MONDAY, FEBRUARY 25, 2019

***> MAD DOGS AND ENGLISHMEN BSE, SCRAPIE, CWD, CJD, TSE PRION A REVIEW 2019

https://bseinquiry.blogspot.com/2019/02/mad-dogs-and-englishmen-bse-scrapie-cwd.html

> In conclusion, sensory symptoms and loss of reflexes in Gerstmann-Sträussler-Scheinker syndrome can be explained by neuropathological changes in the spinal cord. We conclude that the sensory symptoms and loss of lower limb reflexes in Gerstmann-Sträussler-Scheinker syndrome is due to pathology in the caudal spinal cord. <

> The clinical and pathological presentation in macaques was mostly atypical, with a strong emphasis on spinal cord pathology.<

> The notion that CWD can be transmitted orally into both new-world and old-world non-human primates asks for a careful reevaluation of the zoonotic risk of CWD. <

> All animals have variable signs of prion neuropathology in spinal cords and brains and by supersensitive IHC, reaction was detected in spinal cord segments of all animals.<

> In particular the US data do not clearly exclude the possibility of human (sporadic or familial) TSE development due to consumption of venison. The Working Group thus recognizes a potential risk to consumers if a TSE would be present in European cervids." Scientific opinion on chronic wasting disease (II) <

https://familialcjdtseprion.blogspot.com/2019/02/cwd-gss-tse-prion-spinal-cord-confucius.html

TUESDAY, NOVEMBER 17, 2020

The European Union summary report on surveillance for the presence of transmissible spongiform encephalopathies (TSE) in 2019 First published 17 November 2020

https://efsaopinionbseanimalprotein.blogspot.com/2020/11/the-european-union-summary-report-on.html

FRIDAY, OCTOBER 30, 2020

Efficient transmission of US scrapie agent by intralingual route to genetically susceptible sheep with a low dose inoculum

https://scrapie-usa.blogspot.com/2020/10/efficient-transmission-of-us-scrapie.html

TUESDAY, JANUARY 12, 2021

Annual Scrapie Report Available for Fiscal Year 2020 USA October 1, 2019 to September 30, 2020

https://scrapie-usa.blogspot.com/2021/01/annual-scrapie-report-available-for.html

THURSDAY, JANUARY 7, 2021

Atypical Nor-98 Scrapie TSE Prion USA State by State Update January 2021

https://nor-98.blogspot.com/2021/01/atypical-nor-98-scrapie-tse-prion-usa.html

FRIDAY, FEBRUARY 12, 2021

Transmission of the atypical/Nor98 scrapie agent to Suffolk sheep with VRQ/ARQ, ARQ/ARQ, and ARQ/ARR genotypes

https://transmissiblespongiformencephalopathy.blogspot.com/2021/02/transmission-of-atypicalnor98-scrapie.html

WEDNESDAY, FEBRUARY 10, 2021

SENATORS URGE BIDEN TO WITHDRAW SHEEP IMPORT RULE DUE TO SCRAPIE TSE Prion CONCERNS

https://scrapie-usa.blogspot.com/2021/02/senators-urge-biden-to-withdraw-sheep.html

WEDNESDAY, FEBRUARY 03, 2021

Scrapie TSE Prion United States of America a Review February 2021 Singeltary et al

https://scrapie-usa.blogspot.com/2021/02/scrapie-tse-prion-united-states-of.html

TUESDAY, JANUARY 5, 2021

Exploration of genetic factors resulting in abnormal disease in cattle experimentally challenged with bovine spongiform encephalopathy

https://bovineprp.blogspot.com/2021/01/exploration-of-genetic-factors.html

2.3.2. New evidence on the zoonotic potential of atypical BSE and atypical scrapie prion strains

PLEASE NOTE;

2.3.2. New evidence on the zoonotic potential of atypical BSE and atypical scrapie prion strainsNo

Olivier Andreoletti, INRA Research Director, Institut National de la Recherche Agronomique (INRA) – École Nationale Vétérinaire de Toulouse (ENVT), invited speaker, presented the results of two recently published scientific articles of interest, of which he is co-author: 'Radical Change in Zoonotic Abilities of Atypical BSE Prion Strains as Evidenced by Crossing of Sheep Species Barrier in Transgenic Mice' (MarinMoreno et al., 2020) and 'The emergence of classical BSE from atypical/Nor98 scrapie' (Huor et al., 2019).

In the first experimental study, H-type and L-type BSE were inoculated into transgenic mice expressing all three genotypes of the human PRNP at codon 129 and into adapted into ARQ and VRQ transgenic sheep mice. The results showed the alterations of the capacities to cross the human barrier species (mouse model) and emergence of sporadic CJD agents in Hu PrP expressing mice: type 2 sCJD in homozygous TgVal129 VRQ-passaged L-BSE, and type 1 sCJD in homozygous TgVal 129 and TgMet129 VRQ-passaged H-BSE.

https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/sp.efsa.2020.EN-1946

WEDNESDAY, OCTOBER 28, 2020

***> EFSA Annual report of the Scientific Network on BSE-TSE 2020 Singeltary Submission

https://efsaopinionbseanimalprotein.blogspot.com/2020/10/efsa-annual-report-of-scientific.html

SUNDAY, OCTOBER 11, 2020

Bovine adapted transmissible mink encephalopathy is similar to L-BSE after passage through sheep with the VRQ/VRQ genotype but not VRQ/ARQ

https://transmissible-mink-encephalopathy.blogspot.com/2020/10/bovine-adapted-transmissible-mink.html

THURSDAY, SEPTEMBER 24, 2020

The emergence of classical BSE from atypical/ Nor98 scrapie

https://nor-98.blogspot.com/2020/09/the-emergence-of-classical-bse-from.html

FRIDAY, OCTOBER 23, 2020

Scrapie TSE Prion Zoonosis Zoonotic, what if?

https://transmissiblespongiformencephalopathy.blogspot.com/2020/10/scrapie-tse-prion-zoonosis-zoonotic.html

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

O.05: Transmission of prions to primates after extended silent incubation periods: Implications for BSE and scrapie risk assessment in human populations
Emmanuel Comoy, Jacqueline Mikol, Valerie Durand, Sophie Luccantoni, Evelyne Correia, Nathalie Lescoutra, Capucine Dehen, and Jean-Philippe Deslys Atomic Energy Commission; Fontenay-aux-Roses, France

Prion diseases (PD) are the unique neurodegenerative proteinopathies reputed to be transmissible under field conditions since decades. The transmission of Bovine Spongiform Encephalopathy (BSE) to humans evidenced that an animal PD might be zoonotic under appropriate conditions. Contrarily, in the absence of obvious (epidemiological or experimental) elements supporting a transmission or genetic predispositions, PD, like the other proteinopathies, are reputed to occur spontaneously (atpical animal prion strains, sporadic CJD summing

80% of human prion cases).

Non-human primate models provided the first evidences supporting the transmissibiity of human prion strains and the zoonotic potential of BSE. Among them, cynomolgus macaques brought major information for BSE risk assessment for human health (Chen, 2014), according to their phylogenetic proximity to humans and extended lifetime. We used this model to assess the zoonotic potential of other animal PD from bovine, ovine and cervid origins even after very long silent incubation periods.

*** 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.

We will present an updated panorama of our different transmission studies and discuss the implications of such extended incubation periods on risk assessment of animal PD for human health.

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thus questioning the origin of human sporadic cases

==========

***our findings suggest that possible transmission risk of H-type BSE to sheep and human. Bioassay will be required to determine whether the PMCA products are infectious to these animals.

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https://prion2015.files.wordpress.com/2015/05/prion2015abstracts.pdf

***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

PRION 2016 TOKYO

Saturday, April 23, 2016

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

Prion. 10:S15-S21. 2016 ISSN: 1933-6896 printl 1933-690X online

Taylor & Francis

Prion 2016 Animal Prion Disease Workshop Abstracts

WS-01: Prion diseases in animals and zoonotic potential

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

1: J Infect Dis 1980 Aug;142(2):205-8

Oral transmission of kuru, Creutzfeldt-Jakob disease, and scrapie to nonhuman primates.

Gibbs CJ Jr, Amyx HL, Bacote A, Masters CL, Gajdusek DC.

Kuru and Creutzfeldt-Jakob disease of humans and scrapie disease of sheep and goats were transmitted to squirrel monkeys (Saimiri sciureus) that were exposed to the infectious agents only by their nonforced consumption of known infectious tissues. The asymptomatic incubation period in the one monkey exposed to the virus of kuru was 36 months; that in the two monkeys exposed to the virus of Creutzfeldt-Jakob disease was 23 and 27 months, respectively; and that in the two monkeys exposed to the virus of scrapie was 25 and 32 months, respectively. Careful physical examination of the buccal cavities of all of the monkeys failed to reveal signs or oral lesions. One additional monkey similarly exposed to kuru has remained asymptomatic during the 39 months that it has been under observation.

snip...

The successful transmission of kuru, Creutzfeldt-Jakob disease, and scrapie by natural feeding to squirrel monkeys that we have reported provides further grounds for concern that scrapie-infected meat may occasionally give rise in humans to Creutzfeldt-Jakob disease.

PMID: 6997404

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=6997404&dopt=Abstract

Recently the question has again been brought up as to whether scrapie is transmissible to man. This has followed reports that the disease has been transmitted to primates. One particularly lurid speculation (Gajdusek 1977) conjectures that the agents of scrapie, kuru, Creutzfeldt-Jakob disease and transmissible encephalopathy of mink are varieties of a single "virus". The U.S. Department of Agriculture concluded that it could "no longer justify or permit scrapie-blood line and scrapie-exposed sheep and goats to be processed for human or animal food at slaughter or rendering plants" (ARC 84/77)" The problem is emphasised by the finding that some strains of scrapie produce lesions identical to the once which characterise the human dementias"

Whether true or not. the hypothesis that these agents might be transmissible to man raises two considerations. First, the safety of laboratory personnel requires prompt attention. Second, action such as the "scorched meat" policy of USDA makes the solution of the acrapie problem urgent if the sheep industry is not to suffer grievously.

snip...

76/10.12/4.6

http://web.archive.org/web/20010305223125/www.bseinquiry.gov.uk/files/yb/1976/10/12004001.pdf

Nature. 1972 Mar 10;236(5341):73-4.

Transmission of scrapie to the cynomolgus monkey (Macaca fascicularis).

Gibbs CJ Jr, Gajdusek DC.

Nature 236, 73 - 74 (10 March 1972); doi:10.1038/236073a0

Transmission of Scrapie to the Cynomolgus Monkey (Macaca fascicularis)

C. J. GIBBS jun. & D. C. GAJDUSEK

National Institute of Neurological Diseases and Stroke, National Institutes of Health, Bethesda, Maryland

SCRAPIE has been transmitted to the cynomolgus, or crab-eating, monkey (Macaca fascicularis) with an incubation period of more than 5 yr from the time of intracerebral inoculation of scrapie-infected mouse brain. The animal developed a chronic central nervous system degeneration, with ataxia, tremor and myoclonus with associated severe scrapie-like pathology of intensive astroglial hypertrophy and proliferation, neuronal vacuolation and status spongiosus of grey matter. The strain of scrapie virus used was the eighth passage in Swiss mice (NIH) of a Compton strain of scrapie obtained as ninth intracerebral passage of the agent in goat brain, from Dr R. L. Chandler (ARC, Compton, Berkshire).

http://www.nature.com/nature/journal/v236/n5341/abs/236073a0.html

http://scrapie-usa.blogspot.com/2010/04/scrapie-and-atypical-scrapie.html

Wednesday, February 16, 2011

IN CONFIDENCE

SCRAPIE TRANSMISSION TO CHIMPANZEES

IN CONFIDENCE

http://scrapie-usa.blogspot.com/2011/02/in-confidence-scrapie-transmission-to.html

MONDAY, DECEMBER 16, 2019

Chronic Wasting Disease CWD TSE Prion aka mad cow type disease in cervid Zoonosis Update

> "In particular the US data do not clearly exclude the possibility of human (sporadic or familial) TSE development due to consumption of venison. The Working Group thus recognizes a potential risk to consumers if a TSE would be present in European cervids." Scientific opinion on chronic wasting disease (II) <

What if?

https://chronic-wasting-disease.blogspot.com/2019/12/chronic-wasting-disease-cwd-tse-prion.html

DECEMBER 2020 TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHY TSE BSE, SCRAPIE, CWD, CPD, PPD, CJD END OF YEAR REPORTS

MONDAY, DECEMBER 14, 2020

Experimental oral transmission of chronic wasting disease to sika deer (Cervus nippon)

https://chronic-wasting-disease.blogspot.com/2020/12/experimental-oral-transmission-of.html

Sunday, January 10, 2021

APHIS Concurrence With OIE Risk Designation for Bovine Spongiform Encephalopathy [Docket No. APHIS-2018-0087] Singeltary Submission June 17, 2019

APHIS Concurrence With OIE Risk Designation for Bovine Spongiform Encephalopathy [Docket No. APHIS-2018-0087] Singeltary Submission

June 17, 2019

APHIS Concurrence With OIE Risk Designation for Bovine Spongiform Encephalopathy [Docket No. APHIS-2018-0087] Singeltary Submission

Greetings APHIS et al,

I would kindly like to comment on APHIS Concurrence With OIE Risk Designation for Bovine Spongiform Encephalopathy [Docket No. APHIS-2018-0087], and my comments are as follows, with the latest peer review and transmission studies as references of evidence.

THE OIE/USDA BSE Minimal Risk Region MRR is nothing more than free pass to import and export the Transmissible Spongiform Encephalopathy TSE Prion disease. December 2003, when the USDA et al lost it's supposedly 'GOLD CARD' ie BSE FREE STATUS (that was based on nothing more than not looking and not finding BSE), once the USA lost it's gold card BSE Free status, the USDA OIE et al worked hard and fast to change the BSE Geographical Risk Statuses i.e. the BSE GBR's, and replaced it with the BSE MRR policy, the legal tool to trade mad cow type disease TSE Prion Globally. The USA is doing just what the UK did, when they shipped mad cow disease around the world, except with the BSE MRR policy, it's now legal.

Also, the whole concept of the BSE MRR policy is based on a false pretense, that atypical BSE is not transmissible, and that only typical c-BSE is transmissible via feed. This notion that atypical BSE TSE Prion is an old age cow disease that is not infectious is absolutely false, there is NO science to show this, and on the contrary, we now know that atypical BSE will transmit by ORAL ROUTES, but even much more concerning now, recent science has shown that Chronic Wasting Disease CWD TSE Prion in deer and elk which is rampant with no stopping is sight in the USA, and Scrapie TSE Prion in sheep and goat, will transmit to PIGS by oral routes, this is our worst nightmare, showing even more risk factors for the USA FDA PART 589 TSE PRION FEED ban.

The FDA PART 589 TSE PRION FEED ban has failed terribly bad, and is still failing, since August 1997. there is tonnage and tonnage of banned potential mad cow feed that went into commerce, and still is, with one decade, 10 YEARS, post August 1997 FDA PART 589 TSE PRION FEED ban, 2007, with 10,000,000 POUNDS, with REASON, Products manufactured from bulk feed containing blood meal that was cross contaminated with prohibited meat and bone meal and the labeling did not bear cautionary BSE statement. you can see all these feed ban warning letters and tonnage of mad cow feed in commerce, year after year, that is not accessible on the internet anymore like it use to be, you can see history of the FDA failure August 1997 FDA PART 589 TSE PRION FEED ban here, but remember this, we have a new

outbreak of TSE Prion disease in a new livestock species, the camel, and this too is very worrisome.

WITH the OIE and the USDA et al weakening the global TSE prion surveillance, by not classifying the atypical Scrapie as TSE Prion disease, and the notion that they want to do the same thing with typical scrapie and atypical BSE, it's just not scientific.

WE MUST abolish the BSE MRR policy, go back to the BSE GBR risk assessments by country, and enhance them to include all strains of TSE Prion disease in all species. With Chronic Wasting CWD TSE Prion disease spreading in Europe, now including, Norway, Finland, Sweden, also in Korea, Canada and the USA, and the TSE Prion in Camels, the fact the the USA is feeding potentially CWD, Scrapie, BSE, typical and atypical, to other animals, and shipping both this feed and or live animals or even grains around the globe, potentially exposed or infected with the TSE Prion. this APHIS Concurrence With OIE Risk Designation for Bovine Spongiform Encephalopathy [Docket No. APHIS-2018-0087], under it's present definition, does NOT show the true risk of the TSE Prion in any country. as i said, it's nothing more than a legal tool to trade the TSE Prion around the globe, nothing but ink on paper.

AS long as the BSE MRR policy stays in effect, TSE Prion disease will continued to be bought and sold as food for both humans and animals around the globe, and the future ramifications from friendly fire there from, i.e. iatrogenic exposure and transmission there from from all of the above, should not be underestimated. ...

https://beta.regulations.gov/document/APHIS-2018-0087-0002

https://downloads.regulations.gov/APHIS-2018-0087-0002/attachment_1.pdf

https://downloads.regulations.gov/APHIS-2018-0087-0002/attachment_1.pdf

https://bovineprp.blogspot.com/2019/06/aphis-concurrence-with-oie-risk.html

WEDNESDAY, DECEMBER 23, 2020

***> BSE research project final report 2005 to 2008 SE1796 SID5

http://bovineprp.blogspot.com/2020/12/

MONDAY, NOVEMBER 30, 2020

***> REPORT OF THE MEETING OF THE OIE SCIENTIFIC COMMISSION FOR ANIMAL DISEASES Paris, 9–13 September 2019 BSE, TSE, PRION

see updated concerns with atypical BSE from feed and zoonosis...terry

https://animalhealthreportpriontse.blogspot.com/2020/11/report-of-meeting-of-oie-scientific.html

SUNDAY, OCTOBER 4, 2020

Cattle Meat and Offal Imported from the United States of America, Canada and Ireland to Japan (Prions) Food Safety Commission of Japan

https://animalhealthreportpriontse.blogspot.com/2020/10/cattle-meat-and-offal-imported-from.html

TUESDAY, SEPTEMBER 29, 2020

ISO's Updated 22442 Animal Tissue Standards — What Changed? TSE Prion!

https://animalhealthreportpriontse.blogspot.com/2020/09/isos-updated-22442-animal-tissue.html

THURSDAY, DECEMBER 17, 2020

THE MAD COW BSE TSE PRION THAT STOLE CHRISTMAS DECEMBER 2003, WHAT REALLY HAPPENED, A REVIEW 2020

https://bovineprp.blogspot.com/2020/12/the-mad-cow-bse-tse-prion-that-stole.html

THURSDAY, AUGUST 20, 2020

Why is USDA "only" BSE TSE Prion testing 25,000 samples a year?

https://animalhealthreportpriontse.blogspot.com/2020/08/why-is-usda-only-bse-tse-prion-testing.html

WEDNESDAY, OCTOBER 21, 2020

Human Prion Disease Surveillance in Washington State, 2006-2017

https://creutzfeldt-jakob-disease.blogspot.com/2020/10/human-prion-disease-surveillance-in.html

MONDAY, MARCH 08, 2021

OHIO SECOND POSITIVE CWD TISSUE SAMPLE IDENTIFIED IN WILD

https://chronic-wasting-disease.blogspot.com/2021/03/ohio-second-positive-cwd-tissue-sample.html

TUESDAY, MARCH 02, 2021

Texas Confirms CWD TSE Prion in 213 white-tailed deer, mule deer, red deer and elk to date, 148 connected to deer breeding facilities and release sites

https://chronic-wasting-disease.blogspot.com/2021/03/texas-confirms-cwd-tse-prion-in-213.html

THURSDAY, FEBRUARY 25, 2021

Texas AN ACT Sec. 43.370. relating to a deer breeding facility affected by chronic wasting disease H.B. 432

https://chronic-wasting-disease.blogspot.com/2021/02/texas-act-sec-43370-relating-to-deer.html

TUESDAY, FEBRUARY 23, 2021

TAHC SUMMARY MINUTES OF THE 407th COMMISSION MEETING September 22, 2020 Chronic Wasting Disease (CWD) TSE Prion

https://chronic-wasting-disease.blogspot.com/2021/02/tahc-summary-minutes-of-407th.html

TUESDAY, MARCH 02, 2021

Montana Special Southwest CWD Hunt so far 305 samples turn in, 52 testing suspect or positive, with 439 confirmed total to date

https://chronic-wasting-disease.blogspot.com/2021/03/montana-special-southwest-cwd-hunt-so.html

TUESDAY, FEBRUARY 23, 2021

IOWA DNR 2020 surveillance wild deer herd CWD 21 new positive deer and added two new counties

https://chronic-wasting-disease.blogspot.com/2021/02/iowa-dnr-2020-surveillance-wild-deer.html

MONDAY, FEBRUARY 22, 2021

Minnesota Nine more deer added to tally of CWD positive whitetails at Houston County farm

https://chronic-wasting-disease.blogspot.com/2021/02/minnesota-nine-more-deer-added-to-tally.html

THURSDAY, FEBRUARY 11, 2021

Virginia DWR CWD TSE Prion Detected for the first time in Rappahannock County

https://chronic-wasting-disease.blogspot.com/2021/02/virginia-dwr-cwd-tse-prion-detected-for.html

MONDAY, NOVEMBER 23, 2020

***> Chronic Wasting Disease CWD TSE Prion Cervid State by State and Global Update November 2020

https://chronic-wasting-disease.blogspot.com/2020/11/chronic-wasting-disease-cwd-tse-prion.html

FRIDAY, FEBRUARY 05, 2021

USA 50 STATE CWD TSE Prion UPDATE FEBRUARY 2021

https://chronic-wasting-disease.blogspot.com/2021/02/usa-50-state-cwd-tse-prion-update.html

Docket (APHIS-2018-0011) Document PUBLIC SUBMISSION

Comment from Terry Singeltary Sr.

Posted by the Animal and Plant Health Inspection Service on Mar 29, 2018

Document ID APHIS-2018-0011-0003

Tracking Number 1k2-92b5-zmw4

Docket No. APHIS-2018-0011 Chronic Wasting Disease Herd Certification Program Standards Singeltary Submission March 30, 2018

Greetings APHIS, USDA, Dr. Tracy Nichols, et al,

I wish to kindly submit my comments on the Docket No. APHIS-2018-0011 Chronic Wasting Disease Herd Certification Program Standards please. i have submitted online and sent a hard copy to Dr. Nichols via email. i know that my concern may not be the same concern as others, but ramifications from cwd tse prion can be long lasting, and science is still emerging. however, the science today warrants immediate and further actions be taken, especially about zoonosis potential for cwd tse prion, if it has not happened already. my comments, with reference materials, are as follows, and will be formatted in such a way, i will address issues by numbers 1-10, and under each one of my comments by each number, i will reference my comments with science to back up what i am stating/asking...thank you kindly, terry

1. I believe that immediately, there should be a 'DECLARATION OF EXTRAORDINARY EMERGENCY FOR FOREIGN ANIMAL DISEASE OF THE United States of America USA' due to Chronic Wasting Disease CWD Transmissible Spongiform Encephalopathy TSE Prion disease. All Intercontinental, International, Interstate movements of cervid should be banned immediately from the USA, and documented CWD TSE Prion Countries. ...snip...see full text Singeltary Submission for references.

2. Voluntary Chronic Wasting Disease Herd Certification Program should be made MANDATORY immediately, OR NO PERMIT TO FARM DEER OR ELK, PERIOD! you don't want to join, then fine, you don't farm cervid and or any product there from...see full text Singeltary Submission for references.

3. 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?...see full text Singeltary Submission for references.

4. QUARANTINE OF ALL CAPTIVE, BREEDERS, URINE, ANTLER, VELVET, SPERM, OR ANY FACILITY 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 enough. see; Infectious agent of sheep scrapie may persist in the environment for at least 16 years...snip...see full text Singeltary Submission for references.

PLEASE SEE Singeltary 5 - 10 comments, and full text file DOWNLOAD ON GOVERNMENT SITE, OR GO TO THIS URL LINK FOR FULL TEXT OF SINGELTARY SUBMISSION TO Docket No. APHIS-2018-0011 Chronic Wasting Disease Herd Certification Program Standards Singeltary Submission March 30, 2018, PLEASE SEE;

FRIDAY, MARCH 30, 2018

Docket No. APHIS-2018-0011 Chronic Wasting Disease Herd Certification Program Standards Singeltary Submission March 30, 2018

http://chronic-wasting-disease.blogspot.com/2018/03/docket-no-aphis-2018-0011-chronic.html

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

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

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