

Docket No. APHIS–2023–0027 Notice of Request for Revision to and Extension of Approval of an Information Collection; National Veterinary Services Laboratories; Bovine Spongiform Encephalopathy Surveillance Program Singeltary Submission

Federal Register / Vol. 88, No. 83 / Monday, May 1, 2023 / Notices

DEPARTMENT OF AGRICULTURE

Animal and Plant Health Inspection Service

[Docket No. APHIS–2023–0027]

Notice of Request for Revision to and Extension of Approval of an Information Collection; National Veterinary Services Laboratories; Bovine Spongiform Encephalopathy Surveillance Program

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 National Veterinary Services Laboratories diagnostic support for the bovine spongiform encephalopathy surveillance program.

DATES: We will consider all comments that we receive on or before June 30, 2023.

ADDRESSES: You may submit comments by either of the following methods:

- Federal eRulemaking Portal: Go to [www.regulations.gov](http://www.regulations.gov). Enter APHIS– 2023–0027 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–2023–0027, Regulatory Analysis and Development, PPD, APHIS, Station 3A–03.8, 4700 River Road, Unit 118, Riverdale, MD 20737–1238.

Supporting documents and any comments we receive on this docket may be viewed at [www.regulations.gov](http://www.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 to prevent the introduction of bovine spongiform encephalopathy into the United States, contact Dr. Christina Loiacono, Coordinator, National Animal Health Laboratory Network, Veterinary Services, APHIS, USDA, 1920 Dayton Road, Ames, IA 50010; (515) 231–2515; [christina.m.loiacono@usda.gov](mailto:christina.m.loiacono@usda.gov). For information on the information collection process, contact Mr. Joseph Moxey, APHIS' Paperwork Reduction Act Coordinator, at (301) 851–2483; [joseph.moxey@usda.gov](mailto:joseph.moxey@usda.gov).

SUPPLEMENTARY INFORMATION:

Title: National Veterinary Services Laboratories; Bovine Spongiform Encephalopathy Surveillance Program. OMB Control Number: 0579-0409. 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 (USDA) is authorized, among other things, to carry out activities to detect, control, and eradicate pests and diseases of livestock within the United States. APHIS' National Veterinary Services Laboratories (NVSL) safeguard U.S. animal health and contribute to public health by ensuring that timely and accurate laboratory support is provided by their nationwide animal health diagnostic system.

USDA complies with the standard set by the World Organization for Animal Health (WOAH) for bovine spongiform encephalopathy (BSE) surveillance. This compliance is critical for maintaining our BSE-risk status with the WOAH. Our BSE surveillance program requires information collection activities, such as completing the USDA BSE Surveillance Submission form and the USDA BSE Surveillance Data Collection form. 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 0.159 hours per response.

Respondents: Slaughter establishments, offsite collection facilities for condemned slaughter cattle, rendering 3D/4D facilities, State animal health personnel, veterinary diagnostic laboratories, and accredited veterinarians.

Estimated annual number of respondents: 178.

Estimated annual number of responses per respondent: 121.

Estimated annual number of responses: 21,568.

Estimated total annual burden on respondents: 3,421 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 24th day of April 2023.

Michael Watson, Acting Administrator, Animal and Plant Health Inspection Service. [FR Doc. 2023–09140 Filed 4–28–23; 8:45 am] BILLING CODE 3410–34–P

<https://www.govinfo.gov/content/pkg/FR-2023-05-01/pdf/2023-09140.pdf>

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#### FOR FURTHER INFORMATION CONTACT:

For information on the regulations to prevent the introduction of bovine spongiform encephalopathy into the United States, contact Dr. Christina Loiacono, Coordinator, National Animal Health Laboratory Network, Veterinary Services, APHIS, USDA, 1920 Dayton Road, Ames, IA 50010; (515) 231-2515;

[christina.m.loiacono@usda.gov](mailto:christina.m.loiacono@usda.gov).

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Done in Washington, DC, this 24th day of April 2023.

Michael Watson,

Acting Administrator, Animal and Plant Health Inspection Service.

[FR Doc. 2023–09140 Filed 4–28–23; 8:45 am]

BILLING CODE 3410–34–P

<https://www.federalregister.gov/documents/2023/05/01/2023-09140/notice-of-request-for-revision-to-and-extension-of-approval-of-an-information-collection-national>

Docket No. APHIS–2023–0027 Notice of Request for Revision to and Extension of Approval of an Information Collection; National Veterinary Services Laboratories; Bovine Spongiform Encephalopathy Surveillance Program Singeltary Submission

Greetings again APHIS et al,

I would kindly like to again, post my concern or urgency, on why said information is so critical, and why the 3 year extension is so critical, especially today, with the recent mad cow cases in the UK, Switzerland, Brazil, Spain, and The Netherlands all atypical BSE cases, and the fact the OIE is so concerned with the recent science about atypical L-type BSE and atypical H-type BSE, both of which can transmit orally, (see OIE BSE atypical in my reference materials), new outbreak of a new Prion disease in a new livestock species, i.e. the camel. The fact Chronic Wasted Disease CWD TSE Prion of Cervid, is spreading across the USA, with no stopping it in the near future, now with 10 different strains, a spillover into cattle or sheep would be devastating, and the ramifications of human zoonosis there from, has great concern throughout the scientific community. The fact that the USA BSE feed ban was and is a joke today (see why, with the fact that CWD positive deer could enter the food/feed chain for other ruminants and what the DEFRA says), how the BSE surveillance and testing has failed us so terribly bad to date, by testing only 25k bovines a year for BSE, you will not find BSE until it's too late, again. THIS is all why INFORMATION COLLECTION is so vital for BSE and all human and animal Transmissible Spongiform Encephalopathy TSE Prion disease.

"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:"

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The question should be, what will the burden be, if WE DON'T COLLECT SAID INFORMATIONS ON BSE, and we find ourselves again facing a BSE epidemic?

I want to bring your attention too, and emphasize;

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

I remember that infamous TEXAS MAD COW that instead of a 48 turnaround at Weybridge, said suspect positive, was declared NEGATIVE, until an Act of Congress and the Honorable Phyllis Fong overrode Texas negative test, sent that BSE sample to Weybridge, and 6 MONTHS LATER ON A 48 HOUR TURNAROUND (BSE REDBOOKS), that BSE sample was CONFIRMED POSITIVE (see history in my references).

Let's not kid ourselves, the BSE ENHANCED BSE SURVEILLANCE efforts way back was a total failure, that's why it was shut down, too many atypical BSE cases were showing up.

ONLY by the Grace of God, have we not had a documented BSE outbreak, that and the fact the USDA et al are only testing 25K cattle for BSE, a number too low to find mad cow disease from some 28.9 million beef cows in the United States as of Jan. 1, 2023, down 4% from last year. The number of milk cows in the United States increased to 9.40 million. U.S. calf crop was estimated at 34.5 million head, down 2% from 2021. Jan 31, 2023.

ALL it would take is one BSE positive, yet alone a handful of BSE cases, this is why the Enhanced BSE was shut down, and the BSE testing shut down to 25k, and the BSE GBRs were replaced with BSE MRRs, after the 2003 Christmas Mad cow, the cow that stole Christmas, making it legal to trade BSE, imo.

THE world was set back to square one with the BSE Minimal Risk Regions, from the BSE GBRs.

WE must enhance our BSE Surveillance and BSE Testing, and the FDA PART 589 TSE PRION FEED BAN must be revised to include Cervid by-products and SRM, and it should be made MANDATORY, AND THIS SHOULD BE WELL DOCUMENTED with information collection.

said 'burden' cost, will be a heavy burden to bear, if we fail with Bovine Spongiform Encephalopathy BSE TSE Prion disease, that is why this information collection is so critical...

Singeltary References

DEFRA

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](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)

\*\*\* PLEASE SEE THIS URGENT UPDATE ON CWD AND FEED ANIMAL PROTEIN \*\*\*

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

PLoS One. 2020; 15(8): e0237410. Published online 2020 Aug 20. doi: 10.1371/journal.pone.0237410 PMCID: PMC7446902 PMID: 32817706

Very low oral exposure to prions of brain or saliva origin can transmit chronic wasting disease

Nathaniel D. Denkers, Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Validation, Visualization, Writing – review & editing,#1 Clare E. Hoover, Conceptualization, Data curation, Investigation, Writing – original draft, Writing – review & editing,#2 Kristen A. Davenport, Conceptualization, Data curation, Investigation, Writing – review & editing,#3 Davin M. Henderson, Conceptualization, Data curation, Investigation, Methodology,#1 Erin E. McNulty, Data curation, Investigation, Methodology, Writing – review & editing,#1 Amy V. Nalls, Conceptualization, Investigation, Methodology, Writing – review & editing,#1 Candace K. Mathiason, Conceptualization, Funding acquisition, Investigation,

Supervision, Writing – review & editing,<sup>1</sup> and Edward A. Hoover, Conceptualization, Data curation, Funding acquisition, Supervision, Writing – review & editing<sup>1,\*</sup> Byron Caughey, Editor Author information Article notes Copyright and License information Disclaimer This article has been corrected.

See PLoS One. 2021 June 10; 16(6): e0253356. Associated Data Data Availability Statement

## Abstract

The minimum infectious dose required to induce CWD infection in cervids remains unknown, as does whether peripherally shed prions and/or multiple low dose exposures are important factors in CWD transmission. With the goal of better understand CWD infection in nature, we studied oral exposures of deer to very low doses of CWD prions and also examined whether the frequency of exposure or prion source may influence infection and pathogenesis. We orally inoculated white-tailed deer with either single or multiple divided doses of prions of brain or saliva origin and monitored infection by serial longitudinal tissue biopsies spanning over two years. We report that oral exposure to as little as 300 nanograms (ng) of CWD-positive brain or to saliva containing seeding activity equivalent to 300 ng of CWD-positive brain, were sufficient to transmit CWD disease. This was true whether the inoculum was administered as a single bolus or divided as three weekly 100 ng exposures. However, when the 300 ng total dose was apportioned as 10, 30 ng doses delivered over 12 weeks, no infection occurred. While low-dose exposures to prions of brain or saliva origin prolonged the time from inoculation to first detection of infection, once infection was established, we observed no differences in disease pathogenesis. These studies suggest that the CWD minimum infectious dose approximates 100 to 300 ng CWD-positive brain (or saliva equivalent), and that CWD infection appears to conform more with a threshold than a cumulative dose dynamic.

snip...

In conclusion, we have attempted to model and better understand CWD infection relative to natural exposure. The results demonstrate: (a) that the minimum CWD oral infectious dose is vastly lower than historical studies used to establish infection; (b) that a direct relationship exists between dose and incubation time to first prion replication detection in tonsils, irrespective of genotype; (c) that a difference was not discernible between brain vs. saliva source prions in ability to establish infection or in resultant disease course; and (d) that the CWD infection process appears to conform more to a threshold dose than an accumulative dose dynamic.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7446902/>

Bovine spongiform encephalopathy: the effect of oral exposure dose on attack rate and incubation period in cattle

G. A. H. Wells,<sup>1</sup> T. Konold,<sup>1</sup> M. E. Arnold,<sup>1</sup> A. R. Austin,<sup>1</sup> S. A. C. Hawkins,<sup>1</sup> M. Stack,<sup>1</sup> M. M. Simmons,<sup>1</sup> Y. H. Lee,<sup>2</sup> D. Gavier-Wide'n,<sup>3</sup> M. Dawson<sup>1</sup> 4 and J. W. Wilesmith<sup>1</sup> 1  
Correspondence G. A. H. Wells

g.a.h.wells@vla.defra.gsi.gov.uk

1 Veterinary Laboratories Agency, Woodham Lane, New Haw, Addlestone, Surrey KT15 3NB, UK

2 National Veterinary Research and Quarantine Service, Anyang, Republic of Korea

3 National Veterinary Institute (SVA), SE-75189 Uppsala, Sweden

Received 27 July 2006

Accepted 18 November 2006

The dose–response of cattle exposed to the bovine spongiform encephalopathy (BSE) agent is an important component of modelling exposure risks for animals and humans and thereby, the modulation of surveillance and control strategies for BSE. In two experiments calves were dosed orally with a range of amounts of a pool of brainstems from BSE-affected cattle. Infectivity in the pool was determined by end-point titration in mice. Recipient cattle were monitored for clinical disease and, from the incidence of pathologically confirmed cases and their incubation periods (IPs), the attack rate and IP distribution according to dose were estimated. The dose at which 50 % of cattle would be clinically affected was estimated at 0.20 g brain material used in the experiment, with 95 % confidence intervals of 0.04–1.00 g. The IP was highly variable across all dose groups and followed a log-normal distribution, with decreasing mean as dose increased. There was no evidence of a threshold dose at which the probability of infection became vanishingly small, with 1/15 (7 %) of animals affected at the lowest dose (1 mg).

snip...

## DISCUSSION

The study has demonstrated that disease in cattle can be produced by oral exposure to as little as 1 mg brain homogenate (100.4 RIII mouse i.c./i.p. ID50 units) from clinically affected field cases of BSE and that the limiting dose for infection of calves is lower than this exposure...

snip...end

<https://www.microbiologyresearch.org/docserver/fulltext/jgv/88/4/1363.pdf?expires=1623186112&id=id&accname=guest&checksum=AE3A4C280431A05B70DE66DEC2E841B4>

<https://www.microbiologyresearch.org/content/journal/jgv/10.1099/vir.0.82421-0#tab2>

P04.27

Experimental BSE Infection of Non-human Primates: Efficacy of the Oral Route

Holznagel, E1; Yutzy, B1; Deslys, J-P2; Lasmzas, C2; Pocchiari, M3; Ingrosso, L3; Bierke, P4; Schulz-Schaeffer, W5; Motzkus, D6; Hunsmann, G6; Lwer, J1 1Paul-Ehrlich-Institut,

Germany; 2Commissariat Energie Atomique, France; 3Istituto Superiore di Sanit, Italy; 4Swedish Institute for Infectious Disease control, Sweden; 5Georg August University, Germany; 6German Primate Center, Germany

#### Background:

In 2001, a study was initiated in primates to assess the risk for humans to contract BSE through contaminated food. For this purpose, BSE brain was titrated in cynomolgus monkeys.

#### Aims:

The primary objective is the determination of the minimal infectious dose (MID50) for oral exposure to BSE in a simian model, and, by in doing this, to assess the risk for humans. Secondly, we aimed at examining the course of the disease to identify possible biomarkers.

#### Methods:

Groups with six monkeys each were orally dosed with lowering amounts of BSE brain: 16g, 5g, 0.5g, 0.05g, and 0.005g. In a second titration study, animals were intracerebrally (i.c.) dosed (50, 5, 0.5, 0.05, and 0.005 mg).

#### Results:

In an ongoing study, a considerable number of high-dosed macaques already developed simian vCJD upon oral or intracerebral exposure or are at the onset of the clinical phase. However, there are differences in the clinical course between orally and intracerebrally infected animals that may influence the detection of biomarkers.

#### Conclusions:

Simian vCJD can be easily triggered in cynomolgus monkeys on the oral route using less than 5 g BSE brain homogenate. The difference in the incubation period between 5 g oral and 5 mg i.c. is only 1 year (5 years versus 4 years). However, there are rapid progressors among orally dosed monkeys that develop simian vCJD as fast as intracerebrally inoculated animals.

The work referenced was performed in partial fulfilment of the study 'BSE in primates' supported by the EU (QLK1-2002-01096).

<http://www.prion2007.com/pdf/Prion%20Book%20of%20Abstracts.pdf>

<https://youtu.be/Vtt1kAVDhDQ>

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3647490/>

<http://cjdisa.com/wp-content/uploads/2017/06/PRION-2017-Summary-for-CJDISA.pdf>

[https://downloads.regulations.gov/APHIS-2018-0087-0002/attachment\\_1.pdf](https://downloads.regulations.gov/APHIS-2018-0087-0002/attachment_1.pdf)

#### 1.3. Determination of the Minimal Infectious BSE Dose in Non-human Primates

In a concerted European effort involving 5 laboratories including ours, the BSE-macaque model was then used to evaluate the minimal amount of BSE-infected material necessary to induce vCJD in primates. Results so far show that 5g of infectious BSE cattle brain is sufficient to induce the disease in all recipient animals by the oral route, with 500 mg yielding an incomplete attack rate<sup>10,11</sup>). The ID<sub>50</sub> of BSE cattle brain is 200 mg for cattle<sup>12</sup>). These results suggest a low species barrier between cattle and non-human primates.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6989170/>

look at the table and you'll see that as little as 1 mg (or 0.001 gm) caused 7% (1 of 14) of the cows to come down with BSE;

#### Risk of oral infection with bovine spongiform encephalopathy agent in primates

Corinne Ida Lasmzas, Emmanuel Comoy, Stephen Hawkins, Christian Herzog, Franck Mouthon, Timm Konold, Frdric Auvr, Evelynne Correia, Nathalie Lescoutra-EtcheGARAY, Nicole Sals, Gerald Wells, Paul Brown, Jean-Philippe Deslys

**Summary** The uncertain extent of human exposure to bovine spongiform encephalopathy (BSE)--which can lead to variant Creutzfeldt-Jakob disease (vCJD)--is compounded by incomplete knowledge about the efficiency of oral infection and the magnitude of any bovine-to-human biological barrier to transmission. We therefore investigated oral transmission of BSE to non-human primates. We gave two macaques a 5 g oral dose of brain homogenate from a BSE-infected cow. One macaque developed vCJD-like neurological disease 60 months after exposure, whereas the other remained free of disease at 76 months. On the basis of these findings and data from other studies, we made a preliminary estimate of the food exposure risk for man, which provides additional assurance that existing public health measures can prevent transmission of BSE to man.

snip...

#### BSE bovine brain inoculum

100 g 10 g 5 g 1 g 100 mg 10 mg 1 mg 01 mg 001 mg

Primate (oral route)\* 1/2 (50%)

Cattle (oral route)\* 10/10 (100%) 7/9 (78%) 7/10 (70%) 3/15 (20%) 1/15 (7%) 1/15 (7%)

RIII mice (ic ip route)\* 17/18 (94%) 15/17 (88%) 1/14 (7%)

#### PrPres biochemical detection

The comparison is made on the basis of calibration of the bovine inoculum used in our study with primates against a bovine brain inoculum with a similar PrPres concentration that was

inoculated into mice and cattle.<sup>8</sup> \*Data are number of animals positive/number of animals surviving at the time of clinical onset of disease in the first positive animal (%). The accuracy of

bioassays is generally judged to be about plus or minus 1 log. ic ip=intracerebral and int\*\*\*ritoneal.

Table 1: Comparison of transmission rates in primates and cattle infected orally with similar BSE brain inocula

Published online January 27, 2005

<http://www.thelancet.com/journal/journal.isa>

It is clear that the designing scientists must

also have shared Mr Bradley's surprise at the results because all the dose

levels right down to 1 gram triggered infection.

<http://web.archive.org/web/20090506002904/http://www.bseinquiry.gov.uk/files/ws/s145d.pdf>

6. It also appears to me that Mr Bradley's answer (that it would take less than say 100 grams) was probably given with the benefit of hindsight; particularly if one considers that later in the same answer Mr Bradley expresses his surprise that it could take as little of 1 gram of brain to cause BSE by the oral route within the same species. This information did not become available until the "attack rate" experiment had been completed in 1995/96. This was a titration experiment designed to ascertain the infective dose. A range of dosages was used to ensure that the actual result was within both a lower and an upper limit within the study and the designing scientists would not have expected all the dose levels to trigger infection.

The dose ranges chosen by the most informed scientists at that time ranged from 1 gram to three times one hundred grams.

It is clear that the designing scientists must have also shared Mr Bradley's surprise at the results because all the dose levels right down to 1 gram triggered infection.

<http://web.archive.org/web/20090506004507/http://www.bseinquiry.gov.uk/files/ws/s147f.pdf>

Notice of Request To Renew an Approved Information Collection: Specified Risk Materials  
DOCKET NUMBER Docket No. FSIS-2022-0027 Singeltary Submission

Greetings FSIS, USDA, et al,

Thank you kindly for allowing the public to comment on ;

(a) whether the proposed collection of information is necessary for the proper performance of FSIS' functions, including whether the information will have practical utility;



(b) the accuracy of FSIS' estimate of the burden of the proposed collection of information, including the validity of the method and assumptions used;

(c) ways to enhance the quality, utility, and clarity of the information to be collected; and

(d) ways to minimize the burden of the collection of information, including through the use of appropriate automated, electronic, mechanical, or other technological collection techniques, or other forms of information technology.

I will be commenting mostly on a, b, and c, because d, is wanting to minimize the burden of collection, and i do not think that is possible if "These statutes mandate that FSIS protect the public by verifying that meat, poultry, and egg products are safe, wholesome, and properly labeled and packaged.", is truly the intent of these statutes, and i would kindly like to explain why, and why it is so critical that these Specified Risk Materials SRM TSE Prion Statues are so important for public health, and WHY there is an urgent need to enhance them, considering the risk factors of Chronic Wasting Disease CWD TSE Prion in Cervid.

THIS collection of SRM materials information should be done all the time, year after year, and ending it EVER would be foolish, imo, not scientific, and will lead to future risk to public health, if you consider just how bad USDA/FSIS/APHIS/FDA failed so badly with the FDA PART 589 TSE PRION FEED BAN, the SRM REMOVAL, THE BSE SURVEILLANCE AND TESTING PROGRAMS, THEY FAILED ALL OF THEM TERRIBLY IMO, AND BY CONTINUING TO INSIST ON TESTING 25K CATTLE FOR BSE IS A DISASTER WATING TO HAPPEND IMO!

## SPECIFIED RISK MATERS

Specified Risk Materials SRMs, are the most high risk infectious materials, organs, of a cow that is infected with Bovine Spongiform Encephalopathy, Transmissible Spongiform Encephalopathy, BSE TSE Prion. the atypical BSE strains are, like atypical L-type BSE are more infectious that the typical C-type BSE. Also, Science of the BSE TSE has evolved to show that there are more infectious tissues and organs than previously thought. I wish to kindly post all this evidence, as to show you why this information collection of SRMs are so vital to public safety, and why they should be enhanced for cattle, cervid, sheep, and goats, oh, and not to forget the new livestock prion disease in camel, the Camel Prion Disease CPD.

ONE other thing, you must remember, SCIENCE AND TRANSMISSION STUDIES have now shown that CWD and Scrapie can transmit to PIGS by Oral route. This should be included in any enhancement of the SRM or FDA PART 589 TSE PRION FEED ban.

NOT to forget Zoonosis of all of the above, i will post the latest science to date at the bottom of the attached files.

Thank You, terry

<https://www.regulations.gov/comment/FSIS-2022-0027-0002>

Singeltary further comments in attachment;



Specified Risk Materials DOCKET NUMBER Docket No. FSIS-2022-0027 Singeltary Submission Attachment [https://downloads.regulations.gov/FSIS-2022-0027-0002/attachment\\_1.pdf](https://downloads.regulations.gov/FSIS-2022-0027-0002/attachment_1.pdf) Monday, December 5, 2022 Notice of Request To Renew an Approved Information Collection: Specified Risk Materials DOCKET NUMBER Docket No. FSIS-2022-0027 Singeltary Submission

<https://specifiedriskmaterial.blogspot.com/2022/12/notice-of-request-to-renew-approved.html>

SEE MAD COW FEED VIOLATIONS AFER MAD COW FEED VIOLATIONS ;

<http://chronic-wasting-disease.blogspot.com/2016/03/docket-no-fda-2003-d-0432-formerly-03d.html>

## PUBLIC SUBMISSION

Use of Electronic Identification Eartags as Official Identification in Cattle and Bison APHIS-2021-0020-0001 Singeltary Submission

Comment from Singeltary Sr., Terry

Posted by the Animal and Plant Health Inspection Service on Mar 21, 2023

<https://www.regulations.gov/comment/APHIS-2021-0020-0999>

SEE ADDITIONAL COMMENTS IN ATTACHMENT;

[https://downloads.regulations.gov/APHIS-2021-0010-0003/attachment\\_1.pdf](https://downloads.regulations.gov/APHIS-2021-0010-0003/attachment_1.pdf)

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

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://www.regulations.gov/comment/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)

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

Comment from Singeltary Sr., Terry

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

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

[https://downloads.regulations.gov/APHIS-2021-0010-0003/attachment\\_1.pdf](https://downloads.regulations.gov/APHIS-2021-0010-0003/attachment_1.pdf)

PDF]Freas, William TSS SUBMISSION File Format: PDF/Adobe Acrobat -

Page 1. J Freas, William From: Sent: To: Subject: Terry S. Singeltary

Sr. [flounder@wt.net] Monday, January 08,2001 3:03 PM freas ...

[http://web.archive.org/web/20170301223601/https://www.fda.gov/OHRMS/DOCKETS/AC/01/slides/3681s2\\_09.pdf](http://web.archive.org/web/20170301223601/https://www.fda.gov/OHRMS/DOCKETS/AC/01/slides/3681s2_09.pdf)

2. WE have a new Prion disease outbreak, in a new Livestock species, in Africa, and apparently, it's fairly large.

Monday, November 14, 2022

Prion Diseases in Dromedary Camels (CPD) 2022 Review

<https://camelusprp.blogspot.com/2022/11/prion-diseases-in-dromedary-camels-cpd.html>

3. PIGS

Title: Prion infectivity detected in swine challenged with chronic wasting disease via the intracerebral or oral route

Author item MOORE, S - Orise Fellow item Kunkle, Robert item SMITH, JODI - Iowa State University item WEST-GREENLEE, M - Iowa State University item Greenlee, Justin Submitted to: Prion Publication Type: Abstract Only Publication Acceptance Date: 4/4/2016 Publication Date: N/A Citation: N/A

Interpretive Summary:

Technical Abstract: Chronic wasting disease (CWD) is a naturally-occurring, fatal neurodegenerative disease of North American cervids. The potential for swine to serve as a host for the agent of chronic wasting disease is unknown. In the US, feeding of ruminant by-products to ruminants is prohibited, but feeding of ruminant materials to swine, mink, and poultry still occurs. In addition, scavenging of CWD-affected cervid carcasses by feral pigs presents a potential risk for CWD exposure. The purpose of this study was to investigate the susceptibility of swine to the CWD agent following oral or intracranial experimental challenge. At 8 weeks 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). At death a complete necropsy examination was performed, including testing of tissues for misfolded prion protein (PrP<sup>Cwd</sup>) by western blotting (WB), enzyme-linked immunosorbent assay (ELISA), and immunohistochemistry (IHC). None of the pigs developed clinical signs consistent with prion disease. Four >6 month intracranially challenged pigs (survival times 45-73 mpc) were positive by ELISA, two were also positive by WB, and one was positive by IHC. One >6 month orally challenged pig (64 mpc) was positive by ELISA. To further investigate the potential for infectivity, brain tissue from selected pigs was bioassayed in mice expressing porcine PRNP. Tissue from the two WB-positive >6 month intracranially challenged pigs produced positive bioassay results, albeit with low attack rates and variable incubation periods. Interestingly, bioassay of material from the longest surviving >6 month orally challenged pig (72 mpc), which was negative for PrP<sup>Cwd</sup> by all other tests, produced a positive bioassay result. Bioassay of material from additional animals is currently underway. This study demonstrates that pigs can serve as potential hosts for CWD, although with low attack rates and scant PrP<sup>Cwd</sup> accumulation.

Detection of infectivity in orally challenged pigs using mouse bioassay raises the possibility that naturally exposed pigs act as a reservoir of CWD infectivity, even though affected pigs do not develop overt clinical signs or readily detectable PrP<sup>Cwd</sup>.

<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:

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>

"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."

ARS researchers at Ames, Iowa conducted this experiment to test the susceptibility of swine to U.S. scrapie isolates by intracranial and oral inoculation. Necropsies were done on a subset of animals at approximately 6 months post inoculation (PI): the time the pigs were expected to reach market weight. Remaining pigs were maintained and monitored for clinical signs of transmissible spongiform encephalopathies (TSE) until study termination at 80 months PI or when removed due to intercurrent disease. Brain samples were examined by multiple diagnostic approaches, and for a subset of pigs in each inoculation group, bioassay in mice expressing porcine prion protein. At 6 months PI, no evidence of scrapie infection was noted by any diagnostic method. 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.

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

see full report;

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

2017 Annual Report

## Objectives

Objective 1: Investigate the mechanisms of protein misfolding in prion disease, including the genetic determinants of misfolding of the prion protein and the environmental influences on protein misfolding as it relates to prion diseases. Subobjective 1.A: Investigate the differences in the unfolded state of wild-type and disease associated prion proteins to better understand the mechanism of misfolding in genetic prion disease. Subobjective 1.B: Investigate the influence of metal ions on the misfolding of the prion protein in vitro to determine if environmental exposure to metal ions may alter disease progression. Objective 2: Investigate the pathobiology of prion strains in natural hosts, including the influence of prion source genotype on interspecies transmission and the pathobiology of atypical transmissible spongiform encephalopathies (TSEs). Subobjective 2.A: Investigate the pathobiology of atypical TSEs. Subobjective 2.B: Investigate the influence of prion source genotype on interspecies transmission. Objective 3: Investigate sampling methodologies for antemortem detection of prion disease, including the utility of blood sampling as a means to assess prion disease status of affected animals and the utility of environmental sampling for monitoring herd prion disease status. Subobjective 3.A: Investigate the utility of blood sampling as a means to assess prion disease status of affected animals. Subobjective 3.B: Investigate the utility of environmental sampling for monitoring herd prion disease status.

## Approach



The studies will focus on three animal transmissible spongiform encephalopathy (TSE) agents found in the United States: bovine spongiform encephalopathy (BSE); scrapie of sheep and goats; and chronic wasting disease (CWD) of deer, elk, and moose. The research will address sites of protein folding and misfolding as it relates to prion disease, accumulation of misfolded protein in the host, routes of infection, and ante mortem diagnostics with an emphasis on controlled conditions and natural routes of infection. Techniques used will include spectroscopic monitoring of protein folding/misfolding, clinical exams, histopathology, immunohistochemistry, and biochemical analysis of proteins. The enhanced knowledge gained from this work will help understand the underlying mechanisms of prion disease and mitigate the potential for unrecognized epidemic expansions of these diseases in populations of animals that could either directly or indirectly affect food animals.

## Progress Report

All 8 project plan milestones for FY17 were fully met. Research efforts directed toward meeting objective 1 of our project plan center around the production of recombinant prion protein from either bacteria or mammalian tissue culture systems and collection of thermodynamic data on the folding of the recombinant prion protein produced. Both bacterial and mammalian expression systems have been established. Thermodynamic data addressing the denatured state of wild-type and a disease associated variant of bovine prion protein has been collected and a manuscript is in preparation. In research pertaining to objective 2, all studies have been initiated and animals are under observation for the development of clinical signs. The animal studies for this objective are long term and will continue until onset of clinical signs. In vitro studies planned in parallel to the animals studies have similarly been initiated and are ongoing. Objective 3 of the project plan focuses on the detection of disease associated prion protein in body fluids and feces collected from a time course study of chronic wasting disease inoculated animals. At this time samples are being collected as planned and methods for analysis are under development.

## Accomplishments

1. Showed that swine are potential hosts for the scrapie agent. A naturally occurring prion disease has not been recognized in swine, but the agent of bovine spongiform encephalopathy does transmit to swine by experimental routes. Swine are thought to have a robust species barrier when exposed to the naturally occurring prion diseases of other species, but the susceptibility of swine to the agent of sheep scrapie has not been thoroughly tested. ARS researchers at Ames, Iowa conducted this experiment to test the susceptibility of swine to U.S. scrapie isolates by intracranial and oral inoculation. Necropsies were done on a subset of animals at approximately 6 months post inoculation (PI): the time the pigs were expected to reach market weight. Remaining pigs were maintained and monitored for clinical signs of transmissible spongiform encephalopathies (TSE) until study termination at 80 months PI or when removed due to intercurrent disease. Brain samples were examined by multiple diagnostic approaches, and for a subset of pigs in each inoculation group, bioassay in mice expressing porcine prion protein. At 6 months PI, no evidence of scrapie infection was noted by any diagnostic method. 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.

2. Determined that pigs naturally exposed to chronic wasting disease (CWD) may act as a reservoir of CWD infectivity. Chronic wasting disease is a naturally occurring, fatal, neurodegenerative disease of cervids. The potential for swine to serve as a host for the agent of CWD disease is unknown. The purpose of this study was to investigate the susceptibility of swine to the CWD agent following experimental oral or intracranial inoculation. Pigs were assigned to 1 of 3 groups: intracranially inoculated; orally inoculated; or non-inoculated. At market weight age, half of the pigs in each group were tested ('market weight' groups). The remaining pigs ('aged' groups) were allowed to incubate for up to 73 months post inoculation (MPI). Tissues collected at necropsy were examined for disease-associated prion protein (PrP<sup>Sc</sup>) by multiple diagnostic methods. Brain samples from selected pigs were bioassayed in mice expressing porcine prion protein. Some pigs from each inoculated group were positive by one or more tests. Bioassay was positive in 4 out of 5 pigs assayed. Although only small amounts of PrP<sup>Sc</sup> were detected using sensitive methods, this study demonstrates that pigs can serve as hosts for CWD. Detection of infectivity in orally inoculated pigs using mouse bioassay raises the possibility that naturally exposed pigs could act as a reservoir of CWD infectivity. Currently, swine rations in the U.S. could contain animal derived components including materials from deer or elk. In addition, feral swine could be exposed to infected carcasses in areas where CWD is present in wildlife populations. The current feed ban in the U.S. is based exclusively on keeping tissues from TSE infected cattle from entering animal feeds. These results indicating the susceptibility of pigs to CWD, coupled with the limitations of the current feed ban, indicates that a revision of the feed ban may be necessary to protect swine production and potentially human health.

3. Developed a method for amplification and discrimination of the 3 forms of BSE in cattle. The prion protein (PrP) is a protein that is the causative agent of transmissible spongiform encephalopathies (TSEs). The disease process involves conversion of the normal cellular PrP to a pathogenic misfolded conformation. This conversion process can be recreated in the lab using a misfolding amplification process known as real-time quaking induced conversion (RT-QuIC). RT-QuIC allows the detection of minute amounts of the abnormal infectious form of the prion protein by inducing misfolding in a supplied substrate. Although RT-QuIC has been successfully used to detect pathogenic PrP with substrates from a variety of host species, prior to this work bovine prion protein had not been proven for its practical uses for RT-QuIC. We demonstrated that prions from transmissible mink encephalopathy (TME) and BSE-infected cattle can be detected with using bovine prion proteins with RT-QuIC, and developed an RT-QuIC based approach to discriminate different forms of BSE. This rapid and robust method, both to detect and discriminate BSE types, is of importance as the economic implications for different types of BSE vary greatly.

## Review Publications

snip...

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

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>

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>

CONFIDENTIAL

EXPERIMENTAL PORCINE SPONGIFORM ENCEPHALOPATHY

LINE TO TAKE

3. If questions on pharmaceuticals are raised at the Press conference, the suggested line to take is as follows:-

"There are no medicinal products licensed for use on the market which make use of UK-derived porcine tissues with which any hypothetical "high risk" 'might be associated. The results of the recent experimental work at the CSM will be carefully examined by the CSM's Working Group on spongiform encephalopathy at its next meeting.

DO Hagger RM 1533 MT Ext 3201

<http://web.archive.org/web/20030822054419/www.bseinquiry.gov.uk/files/yb/1990/09/21009001.pdf>

While this clearly is a cause for concern we should not jump to the conclusion that this means that pigs will necessarily be infected by bone and meat meal fed by the oral route as is the case with cattle. ...

<http://web.archive.org/web/20031026000118/www.bseinquiry.gov.uk/files/yb/1990/08/23004001.pdf>

we cannot rule out the possibility that unrecognised subclinical spongiform encephalopathy could be present in British pigs though there is no evidence for this: only with parenteral/implantable pharmaceuticals/devices is the theoretical risk to humans of sufficient concern to consider any action.

<http://web.archive.org/web/20030822031154/www.bseinquiry.gov.uk/files/yb/1990/09/10007001.pdf>

May I, at the outset, reiterate that we should avoid dissemination of papers relating to this experimental finding to prevent premature release of the information. ...

<http://web.archive.org/web/20030822052332/www.bseinquiry.gov.uk/files/yb/1990/09/11005001.pdf>

3. It is particularly important that this information is not passed outside the Department, until Ministers have decided how they wish it to be handled. ...

<http://web.archive.org/web/20030822052438/www.bseinquiry.gov.uk/files/yb/1990/09/12002001.pdf>

But it would be easier for us if pharmaceuticals/devices are not directly mentioned at all. ...

<http://web.archive.org/web/20030518170213/www.bseinquiry.gov.uk/files/yb/1990/09/13004001.pdf>

Our records show that while some use is made of porcine materials in medicinal products, the only products which would appear to be in a hypothetically "higher risk" area are the adrenocorticotrophic hormone for which the source material comes from outside the United Kingdom, namely America China Sweden France and Germany. The products are manufactured by Ferring and Armour. A further product, "Zenoderm Corium implant" manufactured by Ethicon, makes use of porcine skin - which is not considered to be a "high risk" tissue, but one of its uses is described in the data sheet as "in dural replacement". This product is sourced from the United Kingdom.....

<http://web.archive.org/web/20030822054419/www.bseinquiry.gov.uk/files/yb/1990/09/21009001.pdf>

4. RACCOONS, Beavers, Rodents, Mountain Lions, Pumas, Wolves

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

Paulina Soto<sup>a,b</sup>, J. Hunter Reed<sup>c</sup>, Mitch Lockwood<sup>c</sup>, and Rodrigo Morales<sup>a,b</sup>

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Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy affecting captive and free-ranging cervids (e.g., mule deer, white-tailed deer, elk, reindeer, and moose). Nowadays, CWD is widely distributed in North America. It is suggested that CWD spreads due to direct animal contact or through exposure to contaminated environments previously inhabited by infected animals. CWD may also be spread through the movement of infected animals and carcasses. Taxidermy practices involve processing deer tissues (or whole animal carcasses). In many cases, the CWD status of processed animals is unknown. This can generate risks of disease spread and transmission. Taxidermy practices include different steps involving physical, chemical, and biological procedures. Without proper tissue handling or disposal practices, taxidermist facilities may become a focus of prion infectivity.

**Aims:** In this study, we evaluated the presence of infectious prions in a taxidermy facility believed to be exposed to CWD. Detection was performed using the Protein Misfolding Cyclic Amplification (PMCA) technique in biological and inert environmental samples.

**Methods:** We collected biological and environmental samples (plants, soils, insects, excreta, and others) from a taxidermy facility, and we tested these samples using the PMCA technique. In addition, we swabbed different surfaces possibly exposed to CWD-infected animals. For the PMCA reaction, we directly used a swab piece or 10 µL of 20% w/v homogenized samples.

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

**Conclusions:** Different areas of this property were used for various taxidermy procedures. We were able to detect the presence of prions in i) soils that were in contact with the heads of dead animals, ii) insects involved in the cleaning of skulls, and iii) an empty dumpster where animal carcasses were previously placed. This is the first report demonstrating that swabbing is a helpful method to screen for prion infectivity on surfaces potentially contaminated with CWD. These findings are relevant as this swabbing and amplification strategy may be used to evaluate the disease status of other free-ranging and captive settings where there is a concern for CWD transmissions, such as at feeders and water troughs with CWD-exposed properties. This approach could have substantial implications for free-ranging cervid surveillance as well as in epidemiological investigations of CWD.

Funded by: USDA Grant number: AP20VSSPRS00C143 PRION 2022 ABSTRACTS, AND A BIG THANK YOU TO On behalf of the Prion2020/2022 Congress Organizing Committee and the NeuroPrion Association, we heartily invite you to join us for the International Conference Prion2020/2022 from 13.-16. September 2022 in Göttingen.

Prion 2022 Conference abstracts: pushing the boundaries

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

Volume 28, Number 4—April 2022

Research

Increased Attack Rates and Decreased Incubation Periods in Raccoons with Chronic Wasting Disease  
Passed through Meadow Voles

S. Jo Moore, Christina M. Carlson, Jay R. Schneider, Christopher J. Johnson, and Justin J. Greenlee  
Comments to Author Author affiliations: US Department of Agriculture, Ames, Iowa, USA (S.J. Moore, J.J. Greenlee); US Geological Survey National Wildlife Health Center, Madison, Wisconsin, USA (C.M. Carlson, J.R. Schneider, C.J. Johnson).

## Abstract

Chronic wasting disease (CWD) is a naturally-occurring neurodegenerative disease of cervids. Raccoons (*Procyon lotor*) and meadow voles (*Microtus pennsylvanicus*) have previously been shown to be susceptible to the CWD agent. To investigate the potential for transmission of the agent of CWD from white-tailed deer to voles and subsequently to raccoons, we intracranially inoculated raccoons with brain homogenate from a CWD-affected white-tailed deer (CWDWtd) or derivatives of this isolate after it had been passaged through voles 1 or 5 times. We found that passage of the CWDWtd isolate through voles led to a change in the biologic behavior of the CWD agent, including increased attack rates and decreased incubation periods in raccoons. A better understanding of the dynamics of cross-species transmission of CWD prions can provide insights into how these infectious proteins evolve in new hosts.

snip...

Prion diseases of free-ranging animals do not exist in isolation. Meadow voles and raccoons are widespread in North America, and their habitat ranges overlap with those of CWD-affected white-tailed deer and other cervids. Therefore, a substantial potential for exposure of these or other off-target species to CWD infectivity in the environment exists. We have demonstrated that CWDWtd from a GS96 white-tailed deer transmitted readily to raccoons. Passage of this isolate through voles followed by intracranial inoculation of raccoons with vole-derived inoculum resulted in disease with different biologic characteristics and neuropathology than the original CWDWtd isolate. These results provide strong evidence for the emergence of a novel strain of CWD after passage in meadow voles and raccoons. Therefore, interspecies transmission of CWD prions between cervids and noncervid species that share the same habitat might represent a confounding factor in CWD-management programs. In addition, passage of CWD prions through off-target species might represent a source of novel CWD strains with unknown biologic characteristics, including zoonotic potential. Characterization of the biologic behavior of CWD isolates after cross-species transmission will help us develop more effective management strategies for CWD-affected populations.

[https://wwwnc.cdc.gov/eid/article/28/4/21-0271\\_article](https://wwwnc.cdc.gov/eid/article/28/4/21-0271_article)

## Susceptibility of Beavers to Chronic Wasting Disease

Allen Herbst 1 2 3, Serene Wohlgemuth 2 4, Jing Yang 2 4, Andrew R Castle 2 4, Diana Martinez Moreno 2 5, Alicia Otero 6, Judd M Aiken 2 3, David Westaway 2 4, Debbie McKenzie 2 5

Affiliations expand

PMID: 35625395 PMCID: PMC9137852 DOI: 10.3390/biology11050667

## Abstract

Chronic wasting disease (CWD) is a contagious, fatal, neurodegenerative prion disease of cervids. The expanding geographical range and rising prevalence of CWD are increasing the risk of pathogen transfer and spillover of CWD to non-cervid sympatric species. As beavers have close contact with environmental and food sources of CWD infectivity, we hypothesized that they may be susceptible to CWD prions. We evaluated the susceptibility of beavers to prion diseases by challenging transgenic mice expressing beaver prion protein (tgBeaver) with five strains of CWD, four isolates of rodent-adapted prions and one strain of Creutzfeldt-Jakob disease. All CWD strains transmitted to the tgBeaver mice, with attack rates highest from moose CWD and the 116AG and H95+ strains of deer CWD. Mouse-, rat-, and especially hamster-adapted prions were also transmitted with complete attack rates and short incubation periods. We conclude that the beaver prion protein is an excellent substrate for sustaining prion replication and that beavers are at risk for CWD pathogen transfer and spillover.

Keywords: beavers; chronic wasting disease; prions; wildlife diseases.

<https://pubmed.ncbi.nlm.nih.gov/35625395/>

FRIDAY, MARCH 24, 2023

Mountain lions, Wolves, Coyotes, could help stop the spread of CWD TSE Prion in deer, WHERE STUPID MEETS THE ROAD!

<https://transmissiblespongiformencephalopathy.blogspot.com/2023/03/mountain-lions-wolves-coyotes-could.html>

## 5. RODENTS

Chronic Wasting Disease (CWD) Susceptibility of Several North American Rodents That Are Sympatric with Cervid CWD Epidemics

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AUTHORS INFO & AFFILIATIONS

DOI: <https://doi.org/10.1128/JVI.00560-09>

## ABSTRACT

Chronic wasting disease (CWD) is a highly contagious always fatal neurodegenerative disease that is currently known to naturally infect only species of the deer family, Cervidae. CWD epidemics are occurring in free-ranging cervids at several locations in North America, and other wildlife species are certainly being exposed to infectious material. To assess the potential for transmission, we intracerebrally inoculated four species of epidemic-sympatric rodents with CWD. Transmission was efficient in all species; the onset of disease was faster in the two vole species than the two *Peromyscus* spp. The results for inocula prepared from CWD-positive deer with or without CWD-resistant genotypes were similar. Survival times were substantially shortened upon second passage, demonstrating adaptation. Unlike all other known prion protein sequences for cricetid rodents that possess asparagine at position 170, our red-backed voles expressed serine and refute previous suggestions that a serine in this position substantially reduces susceptibility to CWD. Given the scavenging habits of these rodent species, the apparent persistence of CWD prions in the environment,

and the inevitable exposure of these rodents to CWD prions, our intracerebral challenge results indicate that further investigation of the possibility of natural transmission is warranted.

snip...

In light of our findings, the possibility of natural transmission to rodents cannot be dismissed. This is concerning because of a TSE's ability to change its properties and host affinities after being passaged (4). Cannibalism and scavenging are common among small rodents, and small rodents are a very important food source for many predators and scavengers. Small rodent tissue also enters the domestic livestock and human food chain by accidental inclusion in grain and forage. Further investigation of these species as potential hosts, bridge species, and reservoirs of CWD is warranted. Even in its natural cervid hosts, the mechanisms of natural transmission and infection of CWD are not well understood. However, the ability to support amplification of PrPd would seem to be a prerequisite, which all of our rodent species have demonstrated. We have initiated studies to examine the susceptibility of these rodent species via more natural routes of infection.

<https://journals.asm.org/doi/reader/10.1128/JVI.00560-09>

<https://journals.asm.org/doi/10.1128/JVI.00560-09>

\*\*\*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, however, suggests that human susceptibility to CWD is low and there may be a robust species barrier for CWD transmission to humans (Sigurdson, 2008). It is apparent, though, that CWD is affecting wild and farmed cervid populations in endemic areas with some deer populations decreasing as a result.

SNIP...

[https://www.gov.uk/government/uploads/system/uploads/attachment\\_data/file/514401/gra-chronic-wasting-disease.pdf](https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/514401/gra-chronic-wasting-disease.pdf)

price of prion poker goes up for cwd to cattle;

Monday, April 04, 2016

\*\*\* Limited amplification of chronic wasting disease prions in the peripheral tissues of intracerebrally inoculated cattle \*\*\*

<http://chronic-wasting-disease.blogspot.com/2016/04/limited-amplification-of-chronic.html>

\*\*\*> Chronic wasting disease: a cervid prion infection looming to spillover

Alicia Otero, Camilo Duque Velásquez, Judd Aiken & Debbie McKenzie

Veterinary Research volume 52, Article number: 115 (2021)



<https://veterinaryresearch.biomedcentral.com/articles/10.1186/s13567-021-00986-y>

repeating our same failures, over and over again...8. 21 CFR Part 589.2000

8. 21 CFR Part 589.2000 Failed Mad Cow Feed Ban in USA (these are just a few examples of 100s i have filed...terry)

#### BANNED MAD COW FEED IN COMMERCE IN ALABAMA

Date: September 6, 2006 at 7:58 am PST PRODUCT

- a) EVSRC Custom dairy feed, Recall # V-130-6;
- b) Performance Chick Starter, Recall # V-131-6;
- c) Performance Quail Grower, Recall # V-132-6;
- d) Performance Pheasant Finisher, Recall # V-133-6.

CODE None RECALLING FIRM/MANUFACTURER Donaldson & Hasenbein/dba J&R Feed Service, Inc., Cullman, AL, by telephone on June 23, 2006 and by letter dated July 19, 2006. Firm initiated recall is complete.

#### REASON

Dairy and poultry feeds were possibly contaminated with ruminant based protein.

VOLUME OF PRODUCT IN COMMERCE 477.72 tons

#### DISTRIBUTION AL

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<http://www.fda.gov/bbs/topics/enforce/2006/ENF00968.html>

[http://web.archive.org/web/20080229052729/http://www.fda.gov/bbs/topics/enforce/2006/ENF00968.h  
tml](http://web.archive.org/web/20080229052729/http://www.fda.gov/bbs/topics/enforce/2006/ENF00968.html)

PRODUCT Bulk custom dairy pre-mixes,

Recall # V-120-6 CODE None RECALLING FIRM/MANUFACTURER Ware Milling Inc., Houston, MS, by telephone on June 23, 2006. Firm initiated recall is complete. REASON Possible contamination of dairy animal feeds with ruminant derived meat and bone meal.

VOLUME OF PRODUCT IN COMMERCE 350 tons

DISTRIBUTION AL and MS

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## PRODUCT

- a) Tucker Milling, LLC Tm 32% Sinking Fish Grower, #2680-Pellet, 50 lb. bags, Recall # V-121-6;
- b) Tucker Milling, LLC #31120, Game Bird Breeder Pellet, 50 lb. bags, Recall # V-122-6;
- c) Tucker Milling, LLC #31232 Game Bird Grower, 50 lb. bags, Recall # V-123-6;
- d) Tucker Milling, LLC 31227-Crumble, Game Bird Starter, BMD Medicated, 50 lb bags, Recall # V-124-6;
- e) Tucker Milling, LLC #31120, Game Bird Breeder, 50 lb bags, Recall # V-125-6;
- f) Tucker Milling, LLC #30230, 30 % Turkey Starter, 50 lb bags, Recall # V-126-6;
- g) Tucker Milling, LLC #30116, TM Broiler Finisher, 50 lb bags, Recall # V-127-6

CODE All products manufactured from 02/01/2005 until 06/20/2006 RECALLING FIRM/MANUFACTURER Recalling Firm: Tucker Milling LLC, Guntersville, AL, by telephone and visit on June 20, 2006, and by letter on June 23, 2006. Manufacturer: H. J. Baker and Brothers Inc., Stamford, CT. Firm initiated recall is ongoing.

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

VOLUME OF PRODUCT IN COMMERCE 7,541-50 lb bags

DISTRIBUTION AL, GA, MS, and TN

END OF ENFORCEMENT REPORT FOR AUGUST 9, 2006

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<http://www.fda.gov/bbs/topics/ENFORCE/2006/ENF00964.html>

<http://web.archive.org/web/20070223174152/http://www.fda.gov/bbs/topics/ENFORCE/2006/ENF00964.html>

Subject: MAD COW FEED RECALL AL AND FL VOLUME OF PRODUCT IN COMMERCE 125 TONS Products manufactured from 02/01/2005 until 06/06/2006

Date: August 6, 2006 at 6:16 pm PST 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

END OF ENFORCEMENT REPORT FOR AUGUST 2, 2006

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<http://www.fda.gov/bbs/topics/enforce/2006/ENF00963.html>

[http://web.archive.org/web/20060821195949/http://www.fda.gov/bbs/topics/enforce/2006/ENF00963.h  
tml](http://web.archive.org/web/20060821195949/http://www.fda.gov/bbs/topics/enforce/2006/ENF00963.html)

MAD COW FEED RECALL USA EQUALS 10,878.06 TONS NATIONWIDE Sun Jul 16, 2006 09:22  
71.248.128.67

RECALLS AND FIELD CORRECTIONS: VETERINARY MEDICINE -- CLASS II

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## PRODUCT

- a) PRO-LAK, bulk weight, Protein Concentrate for Lactating Dairy Animals, Recall # V-079-6;
  - b) ProAmino II, FOR PREFRESH AND LACTATING COWS, net weight 50lb (22.6 kg), Recall # V-080-6;
  - c) PRO-PAK, MARINE & ANIMAL PROTEIN CONCENTRATE FOR USE IN ANIMAL FEED, Recall # V-081-6;
  - d) Feather Meal, Recall # V-082-6 CODE
- a) Bulk
  - b) None
  - c) Bulk
  - d) Bulk

RECALLING FIRM/MANUFACTURER H. J. Baker & Bro., Inc., Albertville, AL, by telephone on June 15, 2006 and by press release on June 16, 2006. Firm initiated recall is ongoing.

## REASON

Possible contamination of animal feeds with ruminant derived meat and bone meal.

VOLUME OF PRODUCT IN COMMERCE 10,878.06 tons

DISTRIBUTION Nationwide

END OF ENFORCEMENT REPORT FOR July 12, 2006

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<http://www.fda.gov/bbs/topics/enforce/2006/ENF00960.html>

<http://web.archive.org/web/20070223180551/http://www.fda.gov/bbs/topics/enforce/2006/ENF00960.html>

10,000,000+ LBS. of PROHIBITED BANNED MAD COW FEED I.E. BLOOD LACED MBM IN COMMERCE USA 2007

Date: March 21, 2007 at 2:27 pm PST

RECALLS AND FIELD CORRECTIONS: VETERINARY MEDICINES -- CLASS II

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## PRODUCT

Bulk cattle feed made with recalled Darling's 85% Blood Meal, Flash Dried, Recall # V-024-2007

## CODE

Cattle feed delivered between 01/12/2007 and 01/26/2007

## RECALLING FIRM/MANUFACTURER

Pfeiffer, Arno, Inc, Greenbush, WI. by conversation on February 5, 2007.

Firm initiated recall is ongoing.

## REASON

Blood meal used to make cattle feed was recalled because it was cross- contaminated with prohibited bovine meat and bone meal that had been manufactured on common equipment and labeling did not bear cautionary BSE statement.

## VOLUME OF PRODUCT IN COMMERCE

42,090 lbs.

## DISTRIBUTION

WI

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## PRODUCT

Custom dairy premix products: MNM ALL PURPOSE Pellet, HILLSIDE/CDL Prot- Buffer Meal, LEE, M.-CLOSE UP PX Pellet, HIGH DESERT/ GHC LACT Meal, TATARKA, M CUST PROT Meal, SUNRIDGE/CDL PROTEIN Blend, LOURENZO, K PVM DAIRY Meal, DOUBLE B DAIRY/GHC LAC Mineral, WEST PIONT/GHC CLOSEUP Mineral, WEST POINT/GHC LACT Meal, JENKS, J/COMPASS PROTEIN Meal, COPPINI - 8# SPECIAL DAIRY Mix, GULICK, L-LACT Meal (Bulk), TRIPLE J - PROTEIN/LACTATION, ROCK CREEK/GHC MILK Mineral, BETTENCOURT/GHC S.SIDE MK-MN, BETTENCOURT #1/GHC MILK MINR, V&C DAIRY/GHC LACT Meal, VEENSTRA, F/GHC LACT Meal, SMUTNY, A- BYPASS ML W/SMARTA, Recall # V-025-2007

## CODE

The firm does not utilize a code - only shipping documentation with commodity and weights identified.

## RECALLING FIRM/MANUFACTURER

Rangen, Inc, Buhl, ID, by letters on February 13 and 14, 2007. Firm initiated recall is complete.

#### 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.

#### VOLUME OF PRODUCT IN COMMERCE

9,997,976 lbs.

#### DISTRIBUTION

ID and NV

#### END OF ENFORCEMENT REPORT FOR MARCH 21, 2007

<http://www.fda.gov/Safety/Recalls/EnforcementReports/2007/ucm120446.htm>

<http://web.archive.org/web/20091104111717/http://www.fda.gov/Safety/Recalls/EnforcementReports/2007/ucm120446.htm>

WEDNESDAY, MARCH 29, 2023

The use of animal by-products in a circular bioeconomy: Time for a TSE road map 3?

<https://transmissiblespongiformencephalopathy.blogspot.com/2023/03/the-use-of-animal-by-products-in.html>

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

[Federal Register Volume 84, Number 116 (Monday, June 17, 2019)] [Notices] [Pages 28001-28002]  
From the Federal Register Online via the Government Publishing Office [www.gpo.gov] [FR Doc No: 2019-12654]

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#### DEPARTMENT OF AGRICULTURE

Animal and Plant Health Inspection Service

[Docket No. APHIS-2018-0087]

Concurrence With OIE Risk Designation for Bovine Spongiform Encephalopathy

[https://downloads.regulations.gov/APHIS-2018-0087-0002/attachment\\_1.pdf](https://downloads.regulations.gov/APHIS-2018-0087-0002/attachment_1.pdf)

# WAHIS, WOAH, OIE, REPORT United Kingdom Bovine Spongiform Encephalopathy Atypical H-Type

United Kingdom - Bovine spongiform encephalopathy - Immediate notification

<https://wahis.woah.org/#/in-review/4977>

Total BSE Cases 181,137

General statistics on BSE cases in Great Britain

Total farms 36199 n/a

Total Cases 181137 n/a

Dairy Farms 22423 61.94

Suckler Farms 10240 28.29

Mixed Farms 2136 5.90

Not Recorded 1400 3.87

Dairy Cases 146123 80.67

Suckler cases 22015 12.15

Mixed Cases 10637 5.87

Not Recorded 2362 1.30

Purchased Cases 59155 32.66

Homebred Cases 120744 66.66

Not Recorded 1238 0.68 Confirmed dairy herd incidence 62.1%

Confirmed suckler herd incidence 17.8%

Confirmed total herd incidence 38.7%

Youngest confirmed case 20 Months

Oldest confirmed case 22 Years Data valid to 30 November 2022\*

\*These figures will only be updated when new data is reported

[https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment\\_data/file/1122885/Overview\\_Of\\_Great\\_Britain\\_2022\\_11\\_30\\_V5.0.ods](https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1122885/Overview_Of_Great_Britain_2022_11_30_V5.0.ods)

## Overview of BSE Cases in Great Britain

[https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment\\_data/file/1122885/Overview\\_Of\\_Great\\_Britain\\_2022\\_11\\_30\\_V5.0.ods](https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1122885/Overview_Of_Great_Britain_2022_11_30_V5.0.ods)

## Age and related statistics

[https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment\\_data/file/1122892/AgeAndRelatedStats\\_2022\\_11\\_30\\_V6.0.ods](https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1122892/AgeAndRelatedStats_2022_11_30_V6.0.ods)

## BSE cases born after the reinforced feed ban (BARB) in the UK

[https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment\\_data/file/1122893/BARB\\_tables\\_2022\\_11\\_30.ods](https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1122893/BARB_tables_2022_11_30.ods)

Details These documents provide statistics on the number of cases of TSE disease found through the active and passive disease surveillance of cattle in the United Kingdom.

Cases of TSE disease identified in cattle from passive surveillance in United Kingdom have been recorded since 1986. The UK carried out limited active surveillance in cattle from 1999 to 2001. The European Union active surveillance programme started in July 2001.

The UK carries out active disease surveillance for bovine spongiform encephalopathy (BSE) in cattle. The testing programme includes cattle over 48 months of age which:

die or are killed other than for human consumption (fallen cattle) are emergency slaughtered or show certain abnormalities at ante-mortem inspection These age thresholds apply to cattle born in the United Kingdom or in other EU member states except Bulgaria and Romania. For cattle born elsewhere the age thresholds are 24 months for fallen cattle or emergency slaughtered cattle, and 30 months for healthy fallen cattle.

Passive disease surveillance takes place when an animal with clinical signs suspicious of a TSE disease is reported to Animal and Plant Health Agency (APHA), and further investigation determines whether the animal was affected by BSE or scrapie.

<https://www.gov.uk/government/publications/cattle-tse-surveillance-statistics>

ODD to me, all these mad cow cases showing up about the same time???

WAHIS, WOAAH, OIE, REPORT Switzerland Bovine Spongiform Encephalopathy Atypical L-Type

Switzerland Bovine Spongiform Encephalopathy Atypical L-Type

Switzerland - Bovine spongiform encephalopathy - Immediate notification

<https://wahis.woah.org/#/in-review/4962>

BRAZIL BSE START DATE 2023/01/18



BRAZIL BSE CONFIRMATION DATE 2023/02/22

BRAZIL BSE END DATE 2023/03/03

<https://wahis.woah.org/#/in-review/4918>

SPAIN BSE START DATE 2023/01/21

SPAIN BSE CONFIRMATION DATE 2023/02/03

SPAIN BSE END DATE 2023/02/06

<https://wahis.woah.org/#/in-review/4888>

NETHERLANDS BSE START DATE 2023/02/01

NETHERLANDS BSE CONFIRMATION DATE 2023/02/01

NETHERLANDS BSE END DATE 2023/03/13

<https://wahis.woah.org/#/in-review/4876>

NOW before you go off and start repeating BSE TSE Prion science that is almost 50 years old, let's be perfectly clear what science is saying today, and especially what the WAHIS/WOAH/OIE et al are saying about the atypical BSE strains... OIE Conclusions on transmissibility of atypical BSE among cattle

Given that cattle have been successfully infected by the oral route, at least for L-BSE, it is reasonable to conclude that atypical BSE is potentially capable of being recycled in a cattle population if cattle are exposed to contaminated feed. In addition, based on reports of atypical BSE from several countries that have not had C-BSE, it appears likely that atypical BSE would arise as a spontaneous disease in any country, albeit at a very low incidence in old cattle. In the presence of livestock industry practices that would allow it to be recycled in the cattle feed chain, it is likely that some level of exposure and transmission may occur. As a result, since atypical BSE can be reasonably considered to pose a potential background level of risk for any country with cattle, the recycling of both classical and atypical strains in the cattle and broader ruminant populations should be avoided.

[https://www.oie.int/fileadmin/SST/adhocreports/Bovine%20spongiform%20encephalopathy/AN/A\\_AhG\\_BSEsurv\\_RiskAss\\_Mar2019.pdf](https://www.oie.int/fileadmin/SST/adhocreports/Bovine%20spongiform%20encephalopathy/AN/A_AhG_BSEsurv_RiskAss_Mar2019.pdf)

Annex 7 (contd) AHG on BSE risk assessment and surveillance/March 2019

34 Scientific Commission/September 2019

### 3. Atypical BSE

The Group discussed and endorsed with minor revisions an overview of relevant literature on the risk of atypical BSE being recycled in a cattle population and its zoonotic potential that had been prepared

ahead of the meeting by one expert from the Group. This overview is provided as Appendix IV and its main conclusions are outlined below. With regard to the risk of recycling of atypical BSE, recently published research confirmed that the L-type BSE prion (a type of atypical BSE prion) may be orally transmitted to calves<sup>1</sup>. In light of this evidence, and the likelihood that atypical BSE could arise as a spontaneous disease in any country, albeit at a very low incidence, the Group was of the opinion that it would be reasonable to conclude that atypical BSE is potentially capable of being recycled in a cattle population if cattle were to be exposed to contaminated feed. Therefore, the recycling of atypical strains in cattle and broader ruminant populations should be avoided.

#### 4. Definitions of meat-and-bone meal (MBM) and greaves

[http://web.oie.int/downld/PROC2020/A\\_SCAD\\_Sept2019.pdf](http://web.oie.int/downld/PROC2020/A_SCAD_Sept2019.pdf)

Consumption of L-BSE-contaminated feed may pose a risk for oral transmission of the disease agent to cattle.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5324790/>

Thus, it is imperative to maintain measures that prevent the entry of tissues from cattle possibly infected with the agent of L-BSE into the food chain.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3310119/>

#### "H-TYPE BSE AGENT IS TRANSMISSIBLE BY THE ORONASAL ROUTE"

This study demonstrates that the H-type BSE agent is transmissible by the oronasal route. These results reinforce the need for ongoing surveillance for classical and atypical BSE to minimize the risk of potentially infectious tissues entering the animal or human food chains.

<https://www.ars.usda.gov/research/publications/publication/?seqNo115=353094>

\*\*\*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 Health<sup>25</sup>, 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 (atypical animal prion strains, sporadic CJD summing 80% of human prion cases).

Non-human primate models provided the first evidences supporting the transmissibility 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.

=====

\*\*\*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.

=====

PRION 2015 CONFERENCE

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

\*\*\*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 print/ 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](http://www.ars.usda.gov/research/publications/publications.htm?SEQ_NO_115=313160)

SO, WHO'S UP FOR SOME MORE TSE PRION POKER, WHO'S ALL IN \$\$\$

SO, ATYPICAL SCRAPIE ROUGHLY HAS 50 50 CHANCE ATYPICAL SCRAPIE IS CONTAGIOUS, AS NON-CONTAGIOUS, TAKE YOUR PICK, BUT I SAID IT LONG AGO WHEN USDA OIE ET AL MADE ATYPICAL SCRAPIE A LEGAL TRADING COMMODITY, I SAID YOUR PUTTING THE CART BEFORE THE HORSE, AND THAT'S EXACTLY WHAT THEY DID, and it's called in Texas, TEXAS TSE PRION HOLDEM POKER, WHO'S ALL IN \$\$ \$

\*\*\*> AS is considered more likely (subjective probability range 50–66%) that AS is a non-contagious, rather than a contagious, disease.

SNIP...SEE;

THURSDAY, JULY 8, 2021

EFSA Scientific report on the analysis of the 2-year compulsory intensified monitoring of atypical scrapie

<https://efsa.onlinelibrary.wiley.com/doi/10.2903/j.efsa.2021.6686>

<https://efsa.onlinelibrary.wiley.com/doi/full/10.2903/j.efsa.2021.6686>

<https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2021.6686>

<https://efsaopinionbseanimalprotein.blogspot.com/2021/07/efsa-scientific-report-on-analysis-of.html>

PAST US MAD COW CASES AND TRACEABILITY PROBLEMS, WHAT'S IT GOING TO TAKE?

AUG. 11, 2017

\*\*\*>Assuming no other factors influenced the levels of correct diagnosis and that the numbers estimated for 1997 to 1999 were a true representation of the potential under-diagnosis of the entire epidemic up until 1999, then the total number of missed cases positive for BSE could have been in the region of 5,500.

\*\*\*>As a result, using more sensitive diagnostic assays, we were able to diagnose BSE positive cattle from the years 1997-1999 inclusive that were originally negative by vacuolation. From these data we have estimated that approximately 3% of the total suspect cases submitted up until the year 1999 were mis-diagnosed.

YOU know, Confucius is confused again LOL, i seem to have remembered something in line with this here in the USA...

USDA did not test possible mad cows

By Steve Mitchell

United Press International

Published 6/8/2004 9:30 PM

WASHINGTON, June 8 (UPI) -- The U.S. Department of Agriculture claims it tested 500 cows with signs of a brain disorder for mad cow disease last year, but agency documents obtained by United Press International show the agency tested only half that number.

<http://madcowtesting.blogspot.com/2007/10/bse-base-mad-cow-testing-texas-usa-and.html>

<http://www.usda.gov/oig/webdocs/50601-10-KC.pdf>

"These 9,200 cases were different because brain tissue samples were preserved with formalin, which makes them suitable for only one type of test--immunohistochemistry, or IHC."

THIS WAS DONE FOR A REASON!

THE IHC test has been proven to be the LEAST LIKELY to detect BSE/TSE in the bovine, and these were probably from the most high risk cattle pool, the ones the USDA et al, SHOULD have been testing. ...TSS

TEXAS 2ND MAD COW THAT WAS COVERED UP, AFTER AN ACT OF CONGRESS, AND CALLS FROM TSE PRION SCIENTIST AROUND THE GLOBE, THIS 2ND MAD COW IN TEXAS WAS CONFIRMED

THE USDA MAD COW FOLLIES POSITIVE TEST COVER UP

JOHANNIS SECRET POSTIVE MAD COW TEST THAT WERE IGNORED

OIG AND THE HONORABLE FONG CONFIRMS TEXAS MAD AFTER AN ACT OF CONGRESS 7 MONTHS LATER

TEXAS MAD COW

THEY DID FINALLY TEST AFTER SITTING 7+ MONTHS ON A SHELF WHILE GW BORE THE BSE MRR POLICY, i.e. legal trading of all strains of TSE. now understand, i confirmed this case 7 months earlier to the TAHC, and then, only after i contacted the Honorable Phyllis Fong and after an act of Congress, this animal was finally confirmed ;

During the course of the investigation, USDA removed and tested a total of 67 animals of interest from the farm where the index animal's herd originated. All of these animals tested negative for BSE. 200 adult animals of interest were determined to have left the index farm. Of these 200, APHIS officials determined that 143 had gone to slaughter, two were found alive (one was determined not to be of interest because of its age and the other tested negative), 34 are presumed dead, one is known dead and 20 have been classified as untraceable. In addition to the adult animals, APHIS was looking for two calves born to the index animal. Due to record keeping and identification issues, APHIS had to trace 213 calves. Of these 213 calves, 208 entered feeding and slaughter channels, four are presumed to have entered feeding and slaughter channels and one calf was untraceable.

[http://www.usda.gov/wps/portal/!ut/p/\\_s.7\\_0\\_A/7\\_0\\_1OB?contentidonly=true&contentid=2005/08/0336.xml](http://www.usda.gov/wps/portal/!ut/p/_s.7_0_A/7_0_1OB?contentidonly=true&contentid=2005/08/0336.xml)

NEW URL LINK;

[http://web.archive.org/web/20090424121101/http://www.usda.gov/wps/portal/!ut/p/\\_s.7\\_0\\_A/7\\_0\\_1OB?contentidonly=true&contentid=2005/08/0336.xml](http://web.archive.org/web/20090424121101/http://www.usda.gov/wps/portal/!ut/p/_s.7_0_A/7_0_1OB?contentidonly=true&contentid=2005/08/0336.xml)

Executive Summary In June 2005, an inconclusive bovine spongiform encephalopathy (BSE) sample from November 2004, that had originally been classified as negative on the immunohistochemistry test, was confirmed positive on SAF immunoblot (Western blot). The

U.S. Department of Agriculture (USDA) identified the herd of origin for the index cow in Texas; that identification was confirmed by DNA analysis. USDA, in close cooperation with the Texas Animal Health Commission (TAHC), established an incident command post (ICP) and began response activities according to USDA's BSE Response Plan of September 2004. Response personnel removed at-risk cattle and cattle of interest (COI) from the index herd, euthanized them, and tested them for BSE; all were negative. USDA and the State extensively traced all at-risk cattle and COI that left the index herd. The majority of these animals entered rendering and/or slaughter channels well before the investigation began. USDA's response to the Texas finding was thorough and effective.

snip...

**Trace Herd 3** The owner of Trace Herd 3 was identified as possibly having received an animal of interest. The herd was placed under hold order on 7/27/05. The herd inventory was conducted on 7/28/05. The animal of interest was not present within the herd, and the hold order was released on 7/28/05. The person who thought he sold the animal to the owner of Trace Herd 3 had no records and could not remember who else he might have sold the cow to. Additionally, a search of GDB for all cattle sold through the markets by that individual did not result in a match to the animal of interest. The animal of interest traced to this herd was classified as untraceable because all leads were exhausted.

**Trace Herd 4** The owner of Trace Herd 4 was identified as having received one of the COI through an order buyer. Trace Herd 4 was placed under hold order on 7/29/05. A complete herd inventory was conducted on 8/22/05 and 8/23/05. There were 233 head of cattle that were examined individually by both State and Federal personnel for all man-made identification and brands. The animal of interest was not present within the herd. Several animals were reported to have died in the herd sometime after they arrived on the premises in April 2005. A final search of GDB records yielded no further results on the eartag of interest at either subsequent market sale or slaughter. With all leads having been exhausted, this animal of interest has been classified as untraceable. The hold order on Trace Herd 4 was released on 8/23/05.

**Trace Herd 5** The owner of Trace Herd 5 was identified as having received two COI and was placed under hold order on 8/1/05. Trace Herd 5 is made up of 67 head of cattle in multiple pastures. During the course of the herd inventory, the owner located records that indicated that one of the COI, a known birth cohort, had been sold to Trace Herd 8 where she was subsequently found alive. Upon completion of the herd inventory, the other animal of interest was not found within the herd. A GDB search of all recorded herd tests conducted on Trace Herd 5 and all market sales by the owner failed to locate the identification tag of the animal of interest and she was subsequently classified as untraceable due to all leads having been exhausted. The hold order on Trace Herd 5 was released on 8/8/05.

**Trace Herd 6** The owner of Trace Herd 6 was identified as possibly having received an animal of interest and was placed under hold order on 8/1/05. This herd is made up of 58 head of cattle on two pastures. A herd inventory was conducted and the animal of interest was not present within the herd. The owner of Trace Herd 6 had very limited records and was unable to provide further information on where the cow might have gone after he purchased her from the livestock market. A search of GDB for all cattle sold through the markets by that individual did not result in a match to the animal of interest. Additionally, many of the animals presented

for sale by the owner of the herd had been re-tagged at the market effectually losing the traceability of the history of that animal prior to re-tagging. The animal of interest traced to this herd was classified as untraceable due to all leads having been exhausted. The hold order on Trace Herd 6 was released on 8/3/05.

Trace Herd 7 The owner of Trace Herd 7 was identified as having received an animal of interest and was placed under hold order on 8/1/05. Trace Herd 7 contains 487 head of cattle on multiple pastures in multiple parts of the State, including a unit kept on an island. The island location is a particularly rough place to keep cattle and the owner claimed to have lost 22 head on the island in 2004 due to liver flukes. Upon completion of the herd inventory, the animal of interest was not found present within Trace Herd 7. A GDB search of all recorded herd tests conducted on Trace Herd 7 and all market sales by the owner failed to locate the identification tag of the animal of interest. The cow was subsequently classified as untraceable. It is quite possible though that she may have died within the herd, especially if she belonged to the island unit. The hold order on Trace Herd 7 was released on 8/8/05.

[http://www.aphis.usda.gov/lpa/issues/bse/epi-updates/bse\\_final\\_epidemiology\\_report.pdf](http://www.aphis.usda.gov/lpa/issues/bse/epi-updates/bse_final_epidemiology_report.pdf)

NEW URL LINK;

[http://web.archive.org/web/20060315165436/http://www.aphis.usda.gov/lpa/issues/bse/epi-updates/bse\\_final\\_epidemiology\\_report.pdf](http://web.archive.org/web/20060315165436/http://www.aphis.usda.gov/lpa/issues/bse/epi-updates/bse_final_epidemiology_report.pdf)

Owner and Corporation Plead Guilty to Defrauding Bovine Spongiform Encephalopathy (BSE) Surveillance Program

An Arizona meat processing company and its owner pled guilty in February 2007 to charges of theft of Government funds, mail fraud, and wire fraud. The owner and his company defrauded the BSE Surveillance Program when they falsified BSE Surveillance Data Collection Forms and then submitted payment requests to USDA for the services. In addition to the targeted sample population (those cattle that were more than 30 months old or had other risk factors for BSE), the owner submitted to USDA, or caused to be submitted, BSE obex (brain stem) samples from healthy USDA-inspected cattle. As a result, the owner fraudulently received approximately \$390,000. Sentencing is scheduled for May 2007.

snip...

4 USDA OIG SEMIANNUAL REPORT TO CONGRESS FY 2007 1st Half

<http://www.usda.gov/oig/webdocs/sarc070619.pdf>

NEW URL LINK;

[http://web.archive.org/web/20080922232504/http://www.usda.gov/oig/webdocs/sarc070619.p  
df](http://web.archive.org/web/20080922232504/http://www.usda.gov/oig/webdocs/sarc070619.pdf)

Audit Report Animal and Plant Health Inspection Service Bovine Spongiform Encephalopathy (BSE) Surveillance Program – Phase II and Food Safety and Inspection Service Controls

## Over BSE Sampling, Specified Risk Materials, and Advanced Meat Recovery Products - Phase III

UNITED STATES DEPARTMENT OF AGRICULTURE OFFICE OF INSPECTOR GENERAL  
Washington, D.C. 20250 January 25, 2006 REPLY TO ATTN OF: 50601-10-KC TO: W. Ron DeHaven Administrator Animal and Plant Health Inspection Service Barbara Masters Administrator Food Safety and Inspection Service ATTN: William J. Hudnall Deputy Administrator Marketing Regulatory Program Business Services William C. Smith Assistant Administrator Office of Program Evaluation, Enforcement, and Review FROM: Robert W. Young /s/ Assistant Inspector General for Audit SUBJECT: Animal and Plant Health Inspection Service - Bovine Spongiform Encephalopathy (BSE) Surveillance Program - Phase II and Food Safety and Inspection Service - Controls Over BSE Sampling, Specified Risk Materials, and Advanced Meat Recovery Products - Phase III This report presents the results of our audit of the enhanced BSE surveillance program and controls over specified risk materials and advanced meat recovery products. Your written response to the official draft report, dated January 20, 2006, is included as exhibit G with excerpts of the response and the Office of Inspector General's (OIG) position incorporated into the Findings and Recommendations section of the report, where applicable. We accept the management decisions for all recommendations. Please follow your agency's internal procedures in forwarding documentation for final action to the Office of the Chief Financial Officer (OCFO). We are providing a separate memorandum to the agencies and OCFO that provides specific information on the actions to be completed to achieve final action. We appreciate your timely response and the cooperation and assistance provided to our staff during the audit USDA/OIG-A/50601-10-KC/ Page i

### Executive Summary

Animal and Plant Health Inspection Service - Bovine Spongiform Encephalopathy (BSE) Surveillance Program - Phase II and Food Safety and Inspection Service - Controls Over BSE Sampling, Specified Risk Materials, and Advanced Meat Recovery Products - Phase III

**Results in Brief** This report evaluates elements of the interlocking safeguards in place to protect United States (U.S.) beef from Bovine Spongiform Encephalopathy, widely known as BSE or "mad cow disease." Since 1990, the U.S. Department of Agriculture (USDA), Animal and Plant Health Inspection Service (APHIS), has led a multi-agency effort to monitor and prevent BSE from entering the food supply. After discovering a BSE-positive cow in December 2003, APHIS expanded its BSE surveillance program. To further protect the food supply, USDA banned materials identified as being at risk of carrying BSE (specified risk materials (SRM)), such as central nervous system tissue. As part of this effort, USDA's Food Safety and Inspection Service (FSIS) required beef slaughter and processing facilities to incorporate controls for handling such materials into their operational plans. Onsite FSIS inspectors also inspect cattle for clinical signs in order to prevent diseased animals from being slaughtered for human consumption. To evaluate the effectiveness of the safeguards, we assessed APHIS' implementation of the expanded surveillance program, as well as FSIS' controls to prevent banned SRMs from entering the food supply.

In June 2004, APHIS implemented its expanded surveillance program; participation by industry in this surveillance program is voluntary. As of May 2005, over 350,000 animals were

sampled and tested for BSE. To date, two animals tested positive for BSE; one tested positive after implementation of the expanded surveillance program.

USDA made significant efforts to implement the expanded BSE surveillance program. Much needed to be done in a short period of time to establish the necessary processes, controls, infrastructure, and networks to assist in this effort. In addition, extensive outreach and coordination was undertaken with other Federal, State, and local entities, private industry, and laboratory and veterinary networks. This report provides an assessment as to the progress USDA made in expanding its surveillance effort and the effectiveness of its controls and processes. This report also discusses the limitations of its program and data in assessing the prevalence of BSE in the U.S. herd.

snip...

40 ELISA test procedures require two additional (duplicate) tests if the initial test is reactive, before final interpretation. If either of the duplicate tests is reactive, the test is deemed inconclusive.

41 Protocol for BSE Contract Laboratories to Receive and Test Bovine Brain Samples and Report Results for BSE Surveillance Standard Operating Procedure (SOP), dated October 26, 2004.

42 The NVSL conducted an ELISA test on the original material tested at the contract laboratory and on two new cuts from the sample tissue.

43 A visual examination of brain tissue by a microscope.

44 A localized pathological change in a bodily organ or tissue.

SNIP...

PLEASE SEE FLAMING EVIDENCE THAT THE USDA ET AL COVERED UP MAD COW DISEASE IN TEXAS ;

PAGE 43;

Section 2. Testing Protocols and Quality Assurance Controls

snip...

FULL TEXT 130 PAGES

<http://www.usda.gov/oig/webdocs/50601-10-KC.pdf>

NEW URL LINK;

<http://web.archive.org/web/20080920075242/http://www.usda.gov/oig/webdocs/50601-10-KC.pdf>



Comments on technical aspects of the risk assessment were then submitted to FSIS.

Comments were received from Food and Water Watch, Food Animal Concerns Trust (FACT), Farm Sanctuary, R-CALF USA, Linda A Detwiler, and Terry S. Singeltary.

This document provides itemized replies to the public comments received on the 2005 updated Harvard BSE risk assessment. Please bear the following points in mind:

[http://www.fsis.usda.gov/PDF/BSE\\_Risk\\_Assess\\_Response\\_Public\\_Comments.pdf](http://www.fsis.usda.gov/PDF/BSE_Risk_Assess_Response_Public_Comments.pdf)

NEW URL LINK;

[http://web.archive.org/web/20090626021608/http://www.fsis.usda.gov/PDF/BSE\\_Risk\\_Assess\\_Response\\_Public\\_Comments.pdf](http://web.archive.org/web/20090626021608/http://www.fsis.usda.gov/PDF/BSE_Risk_Assess_Response_Public_Comments.pdf)

Owens, Julie From: Terry S. Singeltary Sr. [flounder9@verizon.net]

Sent: Monday, July 24, 2006 1:09 PM To: FSIS RegulationsComments

Subject: [Docket No. FSIS-2006-0011] FSIS Harvard Risk Assessment of Bovine Spongiform Encephalopathy (BSE) Page 1 of 98 8/3/2006

Greetings FSIS, I would kindly like to comment on the following ;

<http://www.fsis.usda.gov/OPPDE/Comments/2006-0011/2006-0011-1.pdf>

NEW URL LINK;

<http://web.archive.org/web/20080921202102/http://www.fsis.usda.gov/OPPDE/Comments/2006-0011/2006-0011-1.pdf>

Suppressed peer review of Harvard study October 31, 2002.

October 31, 2002 Review of the Evaluation of the Potential for Bovine Spongiform Encephalopathy in the United States Conducted by the Harvard Center for Risk Analysis, Harvard School of Public Health and Center for Computational Epidemiology, College of Veterinary Medicine, Tuskegee University Final Report Prepared for U.S. Department of Agriculture Food Safety and Inspection Service Office of Public Health and Science Prepared by RTI Health, Social, and Economics Research Research Triangle Park, NC 27709 RTI Project Number 07182.024

[http://www.fsis.usda.gov/oa/topics/BSE\\_Peer\\_Review.pdf](http://www.fsis.usda.gov/oa/topics/BSE_Peer_Review.pdf)

NEW URL LINK;

[http://web.archive.org/web/20050308184249/http://www.fsis.usda.gov/oa/topics/BSE\\_Peer\\_Review.pdf](http://web.archive.org/web/20050308184249/http://www.fsis.usda.gov/oa/topics/BSE_Peer_Review.pdf)

Sunday, February 14, 2010

[Docket No. FSIS-2006-0011] FSIS Harvard Risk Assessment of Bovine Spongiform Encephalopathy (BSE)

<http://bseusa.blogspot.com/2010/02/docket-no-fsis-2006-0011-fsis-harvard.html>

snip...SEE FULL TEXT;

BSE research project final report 2005 to 2008 SE1796 SID5

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

TUESDAY, MAY 31, 2022

USA Bovine Spongiform Encephalopathy BSE: description of typical and atypical cases

<https://bovineprp.blogspot.com/2022/05/usa-bovine-spongiform-encephalopathy.html>

TUESDAY, SEPTEMBER 07, 2021

Atypical Bovine Spongiform Encephalopathy BSE OIE, FDA 589.2001 FEED REGULATIONS, and Ingestion Therefrom

<https://bse-atypical.blogspot.com/2021/09/atypical-bovine-spongiform.html>

TUESDAY, SEPTEMBER 13, 2022

BSE pathogenesis in the ileal Peyer's patches and the central and peripheral nervous system of young cattle 8 months post oral BSE challenge

<https://bovineprp.blogspot.com/2022/09/bse-pathogenesis-in-ileal-peyers.html>

TUESDAY, SEPTEMBER 07, 2021

Atypical Bovine Spongiform Encephalopathy BSE OIE, FDA 589.2001 FEED REGULATIONS, and Ingestion Therefrom

<https://bse-atypical.blogspot.com/2021/09/atypical-bovine-spongiform.html>

Bovine Spongiform Encephalopathy BSE TSE Prion Origin USA

<https://bovineprp.blogspot.com/2021/10/bovine-spongiform-encephalopathy-bse.html>

WEDNESDAY, JANUARY 12, 2022

Bovine Spongiform Encephalopathy BSE TSE Prion Origin USA, what if?

<https://bovineprp.blogspot.com/2022/01/bovine-spongiform-encephalopathy-bse.html>

PLOS ONE Journal

\*\*\* Singeltary reply ; Molecular, Biochemical and Genetic Characteristics of BSE in Canada  
Singeltary reply ;

<https://journals.plos.org/plosone/article/comment?id=10.1371/annotation/4f9be886-69fe-4c7c-922b-85b0ecbe6d53>

IBNC Tauopathy or TSE Prion disease, it appears, no one is sure

Terry S. Singeltary Sr., 03 Jul 2015 at 16:53 GMT

\*\*\*however in 1 C-type challenged animal, Prion 2015 Poster Abstracts S67 PrPsc was not detected using rapid tests for BSE.

\*\*\*Subsequent testing resulted in the detection of pathologic lesion in unusual brain location and PrPsc detection by PMCA only.

\*\*\* IBNC Tauopathy or TSE Prion disease, it appears, no one is sure \*\*\*

<http://www.plosone.org/annotation/listThread.action?root=86610>

MONDAY, SEPTEMBER 19, 2022

589.2001 BSE TSE regulations which prohibits the use of high-risk cattle material in feed for all animal species 2022

<https://animalhealthreportpriontse.blogspot.com/2022/09/5892001-bse-tse-regulations-which.html>

SATURDAY, SEPTEMBER 24, 2022

Transmission of CH1641 in cattle

<https://transmissiblespongiformencephalopathy.blogspot.com/2022/09/transmission-of-ch1641-in-cattle.html>

FRIDAY, APRIL 1, 2022

USDA TAKES THE C OUT OF COOL, what's up with that?

<https://naiscoolyes.blogspot.com/2022/04/usda-takes-c-out-of-cool-whats-up-with.html>

MONDAY, JUNE 6, 2022

APHIS USDA History Highlight: APHIS Combats Bovine Spongiform Encephalopathy  
Published Jun 1, 2022

<https://bovineprp.blogspot.com/2022/06/aphis-usda-history-highlight-aphis.html>

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>

WEDNESDAY, DECEMBER 8, 2021

Importation of Sheep, Goats, and Certain Other Ruminants AGENCY: Animal APHIA, USDA, FINAL RULE [Docket No. APHIS–2009–0095] RIN 0579–AD10

<https://animalhealthreportpriontse.blogspot.com/2021/12/importation-of-sheep-goats-and-certain.html>

WEDNESDAY, MARCH 24, 2021

USDA Animal and Plant Health Inspection Service 2020 IMPACT REPORT BSE TSE Prion Testing and Surveillance MIA

<https://animalhealthreportpriontse.blogspot.com/2021/03/usda-animal-and-plant-health-inspection.html>

SUNDAY, MARCH 21, 2021

Investigation Results of Texas Cow That Tested Positive for Bovine Spongiform Encephalopathy (BSE) Aug. 30, 2005 Singeltary's Review 2021

<https://animalhealthreportpriontse.blogspot.com/2021/03/investigation-results-of-texas-cow-that.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>

THURSDAY, JANUARY 23, 2020

USDA Consolidates Regulations for NAHLN Laboratory Testing USDA Animal and Plant Health Inspection Service

sent this bulletin at 01/23/2020 02:15 PM EST

<http://madcowusda.blogspot.com/2020/01/usda-consolidates-regulations-for-nahln.html>

WEDNESDAY, APRIL 24, 2019

USDA Announces Atypical Bovine Spongiform Encephalopathy Detection Aug 29, 2018 A Review of Science 2019

<https://bse-atypical.blogspot.com/2019/04/usda-announces-atypical-bovine.html>

Saturday, July 23, 2016

BOVINE SPONGIFORM ENCEPHALOPATHY BSE TSE PRION SURVEILLANCE, TESTING, AND SRM REMOVAL UNITED STATE OF AMERICA UPDATE JULY 2016

<http://bovineprp.blogspot.com/2016/07/bovine-spongiform-encephalopathy-bse.html>

Tuesday, July 26, 2016

Atypical Bovine Spongiform Encephalopathy BSE TSE Prion UPDATE JULY 2016

<http://bse-atypical.blogspot.com/2016/07/atypical-bovine-spongiform.html>

Monday, June 20, 2016

Specified Risk Materials SRMs BSE TSE Prion Program

<http://specifiedriskmaterial.blogspot.com/2016/06/specified-risk-materials-srms-bse-tse.html>

\*\*\* PLEASE SEE THIS URGENT UPDATE ON CWD AND FEED ANIMAL PROTEIN \*\*\*

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

SEE MAD COW FEED VIOLATIONS AFER MAD COW FEED VIOLATIONS ;

<http://chronic-wasting-disease.blogspot.com/2016/03/docket-no-fda-2003-d-0432-formerly-03d.html>

Tuesday, April 19, 2016

Docket No. FDA-2013-N-0764 for Animal Feed Regulatory Program Standards Singeltary Comment Submission

## BSE REDBOOK

### Preliminary Notification

The director of NVSL is responsible for immediately notifying the APHIS, Veterinary Services (VS) deputy administrator when tests suggest a presumptive diagnosis of BSE. Once NVSL has made a presumptive diagnosis of BSE, APHIS and FSIS field activities will also be initiated. APHIS will receive notification (either confirming or not confirming NVSL's diagnosis) from the United Kingdom anywhere between 24 and 96 hours. (The international animal health community has recognized the United Kingdom's Central Veterinary Laboratory {CVL} as the world's reference laboratory for diagnosing BSE. Other countries, including Belgium, France, Ireland, Luxembourg, the Netherlands, Portugal, and Switzerland, have all sent samples to this lab to confirm their first case of BSE).

snip...

### BSE Response Team

The BSE Response Team will complete the informational memorandum for the Secretary. The Team will prepare the letter to the Office of International Epizootics (OIE), the international animal health organization, for signature by the APHIS, VS Deputy Administrator. OIE requires that all countries submit official notification within 24 hours of confirming a diagnosis of BSE. The BSE Response Team and the office of the APHIS, VS Deputy Administrator would coordinate a teleconference to inform all APHIS regional directors and AVIC'S. The BSE Response Team and the office of the FSIS, OPHS Deputy Administrator would coordinate a teleconference to inform all regional and field FSIS offices. The BSE Response Team would coordinate a teleconference to notify other Federal agencies. The BSE Response Team would coordinate a teleconference to notify key industry/consumer representatives. The BSE Response Team and APHIS International Services would notify foreign embassies. The BSE Response Team would establish a toll-free 800 telephone line for industry representatives, reporters, and the public. The BSE Response Team would coordinate with APHIS Legislative and Public Affairs and USDA office of Communications to issue a press release the day the diagnosis is confirmed. The press release would announce a press conference to be held the morning after the diagnosis is confirmed.....

THE END

From: Terry S. Singeltary Sr. (216-119-138-126.ipset18.wt.net)  
Subject: Hunkering down in the APHIS BSE Situation Room...  
Date: February 14, 2000 at 9:04 am PST

Subject: hunkering down in the APHIS BSE Situation Room  
Date: Wed, 12 May 1999 01:55:54 -0800  
From: tom Reply-To: Bovine Spongiform Encephalopathy  
To: BSE-L@uni-karlsruhe.de

i am looking now a bizarre Oct 98 internal USDA publication describing a james bond-type US effort to control media should the long-anticipated first case of BSE in the US be admitted.

'Players' on the 27 member BSE Response Team are to be flown in from all over the country to a BSE Headquarters 'situation room' apparently an underground bunker in Riverdale, Maryland under the command of the Assistant Secretary of Marketing.

Authentic press releases are already prepared and ready to go out after a few specifics have been filled in. They are spelled out in a separate document, the BSE Red Book, aka BSE Emergency Disease Guidelines.

Aphis' National Veterinary Services Laboratories (NVSL) activates team assembly. From the time a bovine brain sample is submitted, it takes 14-18 days to confirm a diagnosis of BSE. In the first 10-13 days, NVSL have enough information to determine the need for additional tests. If a provisional BSE diagnosis is made, the sample is 'hand-carried' (are they going to tell the airline and customs?) to the Central Veterinary Laboratory in England for confirmation, where they are expecting a 24 to 96 hour turn-around.

I guess that means we can get the white tiger brain analyzed by Friday despite the 22 year delay to date. Maybe we could throw in a few cougar brains from NE Colorado too.

A Team Member is designated to silently monitor this listserve and [www.mad-cow.org](http://www.mad-cow.org) (among others) -- for what, it doesn't say. The Freedom of Information Act request from the East Coast consumer group turned up numerous top-secret USDA downloads from that site and Dealer's.

After 24 hours of secret briefings for 'select industry and trading partners' (to allow them to take positions on the commodities markets opposite the 'non-select' industry and trading partners?), a press conference will be held the next day.

There are plans to trace the cow, its lineage, its herdmates, the renderer, traceout of product, buyout of herd, farm of origin, to get the state involved to quarantine the herd (pre-arranged for all 50 states), expectations for trade bans, notification of OIE within 24 hours, media 800 numbers, spokespersons and backups, notify CDC, FDA, NIH, and many other commendable activities. The Flow Chart is a sight to behold, I will try to scan it in tomorrow.

In short, that cow is going to be toast by the time the public first hears about it.

The Plan does not speak to the scenario in which the CVL says, yes, this is bovine spongiform encephalopathy all right but it is one of your strains, not ours. Invoking their Absence of Evidence is Evidence of Absence principle, there may be no perceived need for public disclosure in this case.

USDA is caught completely unprepared if BSE first turns up in a US zoo animal. These animals could easily be diagnosed outside the "system" and be the subject of a publicity-seeking lab press release. I think this is a more likely scenario because the US has likely imported many thousands of zoo animals with advanced infections from Britain and France and there has been zero monitoring. Unlike with downer cows, anyone with the right



colleagues can get ahold of a fallen zoo animal. Zoo animals enter the food chain in some cases after being rendered.

Another scenario would be some stock market speculator obtaining the Red Book and issuing a flurry of bogus but authentic-looking press releases that included bogus 800 and hacked USDA web links. The press here is so lazy and so accustomed to putting out public relation handouts as news that the objectives would be accomplished for a few hour (or days, depending on the Response Team's paralysis vis-a-vis off-flow chart events). Some people think a practise run for this happened in the Indiana case a year or two back.

The first case of nvCJD in an American will also be a public relations fiasco. In the dim bulb of the public mind, any American with mad cow disease would have gotten it from eating meat here. USDA has no way to prove that the victim acquired it on a three week trip to England in 1987. This will sound lame even to the press. All CJD is synonymous with mad cow disease in the public perception; the more often the different kinds are explained, the more their suspicions are aroused. The first case of nvCJD in an American will simply validate what they already know and just be viewed as an overdue admission from the government.

tom

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From: Terry S. Singeltary Sr. (216-119-130-102.ipset10.wt.net)  
Subject: When a case of B.S.E. is found in the U.S/Response to Disease outbreak...'redbook'  
Date: March 13, 2000 at 10:13 am PST

BSE Red Book 2.1-26

5.0 Response to Disease Outbreak

snip...see full report of From: Terry S. Singeltary Sr. (216-119-130-102.ipset10.wt.net) Subject: When a case of B.S.E. is found in the U.S/Response to Disease outbreak...'redbook' Date: March 13, 2000 at 10:13 am PST

<https://bseusa.blogspot.com/2008/08/qualitative-analysis-of-bse-risk.html>

Thursday, April 6, 2023

WOAH OIE CHAPTER 11.4 . BOVINE SPONGIFORM ENCEPHALOPATHY Article 11.4.1.

<https://woahoie.blogspot.com/2023/04/woah-oie-chapter-114-bovine-spongiform.html>

2023

The risk of CJD increases with age; the 2016–2020 average annual rate in the United States was about 5 cases per million in persons 55 years of age or older.

<https://www.cdc.gov/prions/cjd/occurrence-transmission.html>

Singeltary 1999

US scientists develop a possible test for BSE

BMJ 1999; 319 doi: <https://doi.org/10.1136/bmj.319.7220.1312b> (Published 13 November 1999)

Cite this as: BMJ 1999;319:1312

15 November 1999

Terry S Singeltary

NA

medically retired

Rapid Response:

Re: vCJD in the USA \* BSE in U.S.

In reading the recent article in the BMJ about the potential BSE tests being developed in the U.S. and Bart Van Everbroeck reply. It does not surprise me, that the U.S. has been concealing vCJD. There have been people dying from CJD, with all the symptoms and pathological findings that resemble U.K. vCJD for some time. It just seems that when there is one found, they seem to change the clirical classification of the disease, to fit their agenda. I have several autopsies, stating kuru type amyloid plaques, one of the victims was 41 years of age. Also, my Mom died a most hideous death, Heidenhain Variant Creutzfeldt Jakob disease.

Her symptoms resemble that of all the U.K. vCJD victims. She would jerk so bad at times, it would take 3 of us to hold her down, while she screamed "God, what's wrong with me, why can't I stop this." 1st of symptoms to death, 10 weeks, she went blind in the first few weeks. But, then they told me that this was just another strain of sporadic CJD. They can call it what ever they want, but I know what I saw, and what she went through. Sporadic, simply means, they do not know.

My neighbors Mom also died from CJD. She had been taking a nutritional supplement which contained the following;

vacuum dried bovine BRAIN, bone meal, bovine EYE, veal bone, bovine liver powder, bovine adrenal, vacuum dried bovine kidney, and vacuum dried porcine stomach. As I said, this woman taking these nutritional supplements, died from CJD.

The particular batch of pills that was located, in which she was taking, was tested. From what I have heard, they came up negative, for the prion protein. But, in the same breath, they said their testing, may not have been strong enough to pick up the infectivity. Plus, she had been taking these type pills for years, so, could it have come from another batch?

CWD is just a small piece of a very big puzzle. I have seen while deer hunting, deer, squirrels and birds, eating from cattle feed troughs where they feed cattle, the high protein cattle by products, at least up until Aug. 4, 1997.

So why would it be so hard to believe that this is how they might become infected with a TSE. Or, even by potentially infected land. It's been well documented that it could be possible, from scrapie. Cats becoming infected with a TSE. Have you ever read the ingredients on the labels of cat and dog food? But, they do not put these tissues from these animals in pharmaceuticals, cosmetics, nutritional supplements, hGH, hPG, blood products, heart valves, and the many more products that come from bovine, ovine, or porcine tissues and organs. So, as I said, this CWD would be a small piece of a very big puzzle. But, it is here, and it most likely has killed. You see, greed is what caused this catastrophe, rendering and feeding practices. But, once Pandora's box was opened, the potential routes of infection became endless.

No BSE in the U.S.A.? I would not be so sure of that considering that since 1990;

Since 1990 the U.S. has raised 1,250,880,700 cattle;

Since 1990 the U.S. has ONLY checked 8,881 cattle brains for BSE, as of Oct. 4, 1999;

There are approx. 100,000 DOWNER cattle annually in the U.S., that up until Aug. 4, 1997 went to the renders for feed;

Scrapie running rampant for years in the U.S., 950 infected FLOCKS, as of Aug. 1999;

Our feeding and rendering practices have mirrored that of the U.K. for years, some say it was worse. Everything from the downer cattle, to those scrapie infected sheep, to any roadkill, including the city police horse and the circus elephant went to the renders for feed and other products for consumption. Then they only implemented a partial feed ban on Aug. 4, 1997, but pigs, chickens, dogs, and cats, and humans were exempt from that ban. So they can still feed pigs and chickens those potentially TSE tainted by-products, and then they can still feed those by-products back to the cows. I believe it was Dr. Joe Gibbs, that said, the prion protein, can survive the digestinal track. So you have stopped nothing. It was proven in Oprah Winfrey's trial, that Cactus Cattle feeders, sent neurologically ill cattle, some with encephalopathy stamped on the dead slips, were picked up and sent to the renders, along with sheep carcasses. Speaking of autopsies, I have a stack of them, from CJD victims. You would be surprised of the number of them, who ate cow brains, elk brains, deer brains, or hog brains.

I believe all these TSE's are going to be related, and originally caused by the same greedy Industries, and they will be many. Not just the Renders, but you now see, that they are re-using medical devices that were meant for disposal. Some medical institutions do not follow proper auto- claving procedures (even Olympus has put out a medical warning on their endoscopes about CJD, and the fact you cannot properly clean these instruments from TSE's), and this is just one product. Another route of infection.

Regardless what the Federal Government in the U.S. says. It's here, I have seen it, and the longer they keep sweeping it under the rug and denying the fact that we have a serious

problem, one that could surpass aids (not now, but in the years to come, due to the incubation period), they will be responsible for the continued spreading of this deadly disease.

It's their move, it's CHECK, but once CHECKMATE has been called, how many thousands or millions, will be at risk or infected or even dead. You can't play around with these TSE's. I cannot stress that enough. They are only looking at body bags, and the fact the count is so low. But, then you have to look at the fact it is not a reportable disease in most states, misdiagnosis, no autopsies performed. The fact that their one-in-a- million theory is a crude survey done about 5 years ago, that's a joke, under the above circumstances. A bad joke indeed.....

The truth will come, but how many more have to die such a hideous death. It's the Government's call, and they need to make a serious move, soon. This problem, potential epidemic, is not going away, by itself.

Terry S. Singeltary Sr.

Bacliff, Texas 77518 USA

flounder@wt.net

Competing interests: No competing interests

<https://www.bmj.com/rapid-response/2011/10/28/re-vcjd-usa-bse-us>

Singeltary 2000

BMJ 2000; 320 doi: <https://doi.org/10.1136/bmj.320.7226.8/b> (Published 01 January 2000)  
Cite this as: BMJ 2000;320:8

02 January 2000 Terry S Singeltary retired

Rapid Response:

U.S. Scientist should be concerned with a CJD epidemic in the U.S., as well...

In reading your short article about 'Scientist warn of CJD epidemic' news in brief Jan. 1, 2000. I find the findings in the PNAS old news, made famous again. Why is the U.S. still sitting on their butts, ignoring the facts? We have the beginning of a CJD epidemic in the U.S., and the U.S. Gov. is doing everything in it's power to conceal it.

The exact same recipe for B.S.E. existed in the U.S. for years and years. In reading over the Qualitative Analysis of BSE Risk Factors-1, this is a 25 page report by the USDA:APHIS:VS. It could have been done in one page. The first page, fourth paragraph says it all;

"Similarities exist in the two countries usage of continuous rendering technology and the lack of usage of solvents, however, large differences still remain with other risk factors which greatly reduce the potential risk at the national level."

Then, the next 24 pages tries to down-play the high risks of B.S.E. in the U.S., with nothing more than the cattle to sheep ratio count, and the geographical locations of herds and flocks. That's all the evidence they can come up with, in the next 24 pages.

Something else I find odd, page 16;

"In the United Kingdom there is much concern for a specific continuous rendering technology which uses lower temperatures and accounts for 25 percent of total output. This technology was \_originally\_ designed and imported from the United States. However, the specific application in the production process is \_believed\_ to be different in the two countries."

A few more factors to consider, page 15;

"Figure 26 compares animal protein production for the two countries. The calculations are based on slaughter numbers, fallen stock estimates, and product yield coefficients. This approach is used due to variation of up to 80 percent from different reported sources. At 3.6 million tons, the United States produces 8 times more animal rendered product than the United Kingdom."

"The risk of introducing the BSE agent through sheep meat and bone meal is more acute in both relative and absolute terms in the United Kingdom (Figures 27 and 28). Note that sheep meat and bone meal accounts for 14 percent, or 61 thousand tons, in the United Kingdom versus 0.6 percent or 22 thousand tons in the United States. For sheep greater than 1 year, this is less than one-tenth of one percent of the United States supply."

"The potential risk of amplification of the BSE agent through cattle meat and bone meal is much greater in the United States where it accounts for 59 percent of total product or almost 5 times more than the total amount of rendered product in the United Kingdom."

Considering, it would only take \_one\_ scrapie infected sheep to contaminate the feed. Considering Scrapie has run rampant in the U.S. for years, as of Aug. 1999, 950 scrapie infected flocks. Also, Considering only one quarter spoonful of scrapie infected material is lethal to a cow.

Considering all this, the sheep to cow ration is meaningless. As I said, it's 24 pages of B.S.e.

To be continued...

Terry S. Singeltary Sr. Bacliff, Texas USA

Competing interests: No competing interests

<https://www.bmj.com/rapid-response/2011/10/28/us-scientist-should-be-concerned-cjd-epidemic-us-well>

Singeltary 2001

Diagnosis and Reporting of Creutzfeldt-Jakob Disease

Singeltary, Sr et al. JAMA.2001; 285: 733-734. Vol. 285 No. 6, February 14, 2001 JAMA

## Diagnosis and Reporting of Creutzfeldt-Jakob Disease

To the Editor:

In their Research Letter, Dr Gibbons and colleagues<sup>1</sup> reported that the annual US death rate due to Creutzfeldt-Jakob disease (CJD) has been stable since 1985. These estimates, however, are based only on reported cases, and do not include misdiagnosed or preclinical cases. It seems to me that misdiagnosis alone would drastically change these figures. An unknown number of persons with a diagnosis of Alzheimer disease in fact may have CJD, although only a small number of these patients receive the postmortem examination necessary to make this diagnosis. Furthermore, only a few states have made CJD reportable. Human and animal transmissible spongiform encephalopathies should be reportable nationwide and internationally..

Terry S. Singeltary, Sr Bacliff, Tex

1. Gibbons RV, Holman RC, Belay ED, Schonberger LB. Creutzfeldt-Jakob disease in the United States: 1979-1998. JAMA. 2000;284:2322-2323.

<http://jama.jamanetwork.com/article.aspx?articleid=1031186>

Singeltary 2003

doi:10.1016/S1473-3099(03)00715-1 Copyright © 2003 Published by Elsevier Ltd. Newsdesk

Tracking spongiform encephalopathies in North America

Xavier Bosch

Available online 29 July 2003.

Volume 3, Issue 8, August 2003, Page 463

Volume 3, Number 8 01 August 2003

Newsdesk

Tracking spongiform encephalopathies in North America

Xavier Bosch

My name is Terry S Singeltary Sr, and I live in Bacliff, Texas. I lost my mom to hvCJD (Heidenhain variant CJD) and have been searching for answers ever since. What I have found is that we have not been told the truth. CWD in deer and elk is a small portion of a much bigger problem.

49-year-old Singeltary is one of a number of people who have remained largely unsatisfied after being told that a close relative died from a rapidly progressive dementia compatible with spontaneous Creutzfeldt-Jakob disease (CJD). So he decided to gather hundreds of documents on transmissible spongiform encephalopathies (TSE) and realised that if Britons could get variant CJD from bovine spongiform encephalopathy (BSE), Americans might get a similar disorder from chronic wasting disease (CWD) the relative of mad cow disease seen among deer and elk in the USA. Although his feverish search did not lead him to the smoking gun linking CWD to a similar disease in North American people, it did uncover a largely disappointing situation.

Singeltary was greatly demoralised at the few attempts to monitor the occurrence of CJD and CWD in the USA. Only a few states have made CJD reportable. Human and animal TSEs should be reportable nationwide and internationally, he complained in a letter to the Journal of the American Medical Association (JAMA 2003; 285: 733). I hope that the CDC does not continue to expect us to still believe that the 85% plus of all CJD cases which are sporadic are all spontaneous, without route or source.

Until recently, CWD was thought to be confined to the wild in a small region in Colorado. But since early 2002, it has been reported in other areas, including Wisconsin, South Dakota, and the Canadian province of Saskatchewan. Indeed, the occurrence of CWD in states that were not endemic previously increased concern about a widespread outbreak and possible transmission to people and cattle.

To date, experimental studies have proven that the CWD agent can be transmitted to cattle by intracerebral inoculation and that it can cross the mucous membranes of the digestive tract to initiate infection in lymphoid tissue before invasion of the central nervous system. Yet the plausibility of CWD spreading to people has remained elusive.

Part of the problem seems to stem from the US surveillance system. CJD is only reported in those areas known to be endemic foci of CWD. Moreover, US authorities have been criticised for not having performed enough prionic tests in farm deer and elk.

Although in November last year the US Food and Drug Administration issued a directive to state public-health and agriculture officials prohibiting material from CWD-positive animals from being used as an ingredient in feed for any animal species, epidemiological control and research in the USA has been quite different from the situation in the UK and Europe regarding BSE.

Getting data on TSEs in the USA from the government is like pulling teeth, Singeltary argues. You get it when they want you to have it, and only what they want you to have.

Norman Foster, director of the Cognitive Disorders Clinic at the University of Michigan (Ann Arbor, MI, USA), says that current surveillance of prion disease in people in the USA is inadequate to detect whether CWD is occurring in human beings; adding that, the cases that we know about are reassuring, because they do not suggest the appearance of a new variant of CJD in the USA or atypical features in patients that might be exposed to CWD. However, until we establish a system that identifies and analyses a high proportion of suspected prion disease cases we will not know for sure. The USA should develop a system modelled on that established in the UK, he points out.



Ali Samii, a neurologist at Seattle VA Medical Center who recently reported the cases of three hunters two of whom were friends who died from pathologically confirmed CJD, says that at present there are insufficient data to claim transmission of CWD into humans; adding that [only] by asking [the questions of venison consumption and deer/elk hunting] in every case can we collect suspect cases and look into the plausibility of transmission further. Samii argues that by making both doctors and hunters more aware of the possibility of prions spreading through eating venison, doctors treating hunters with dementia can consider a possible prion disease, and doctors treating CJD patients will know to ask whether they ate venison.

CDC spokesman Ermias Belay says that the CDC will not be investigating the [Samii] cases because there is no evidence that the men ate CWD-infected meat. He notes that although the likelihood of CWD jumping the species barrier to infect humans cannot be ruled out 100% and that [we] cannot be 100% sure that CWD does not exist in humans& the data seeking evidence of CWD transmission to humans have been very limited.

<http://www.thelancet.com/journals/laninf/article/PIIS1473309903007151/fulltext>

Singeltary 2003

January 28, 2003; 60 (2) VIEWS & REVIEWS

RE-Monitoring the occurrence of emerging forms of Creutzfeldt-Jakob disease in the United States Terry S. Singeltary, retired (medically)

Published March 26, 2003

26 March 2003

Terry S. Singeltary, retired (medically) CJD WATCH

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

<http://n.neurology.org/content/re-monitoring-occurrence-emerging-forms-creutzfeldt-jakob-disease-united-states>

Singeltary 2007

The Pathological Protein: Mad Cow, Chronic Wasting, and Other Deadly Prion Diseases

by Philip Yam

"Answering critics like Terry Singeltary, who feels that the US undercounts CJD, Schonberger \_conceded\_ that the current surveillance system has errors but stated that most of the errors will be confined to the older population"...

Revisiting Sporadic CJD

It's not hard to get Terry Singeltary going. "I have my conspiracy theories," admitted the 49-year-old Texan.<sup>1</sup> Singeltary is probably the nation's most relentless consumer advocate when it comes to issues in prion diseases. He has helped families learn about the sickness and coordinated efforts with support groups such as CJD Voice and the CJD Foundation. He has also connected with others who are critical of the American way of handling the threat of prion diseases. Such critics include Consumers Union's Michael Hansen, journalist John Stauber, and Thomas Pringle, who used to run the voluminous [www.madcow.org](http://www.madcow.org) Web site. These three lend their expertise to newspaper and magazine stories about prion diseases, and they usually argue that

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prions represent more of a threat than people realize, and that the government has responded poorly to the dangers because it is more concerned about protecting the beef industry than people's health.

Singeltary has similar inclinations, but unlike these men, he doesn't have the professional credentials behind him. He is an 11th-grade dropout, a machinist who retired because of a neck injury sustained at work. But you might not know that from the vast stores of information in his mind and on his hard drive. Over the years, he has provided unacknowledged help to reporters around the globe, passing on files to such big-time players as The New York Times, Newsweek, and USA Today. His networking with journalists, activists, and concerned citizens has helped medical authorities make contact with suspected CJD victims. He has kept scientists informed with his almost daily posting of news items and research abstracts on electronic newsgroups, including the bulletin board on [www.vegsource.com](http://www.vegsource.com) and the BSE-listserv run out of the University of Karlsruhe, Germany. His combative, blunt, opinionated style sometimes borders on obsessive ranting that earns praise from some officials and researchers but infuriates others—especially when he repeats his conviction that "the government has lied to us, the feed industry has lied to us—all over a buck." As evidence, Singeltary cites the USDA's testing approach, which targets downer cows and examined 19,900 of them in 2002. To him, the USDA should test 1 million cattle, because the incidence of BSE may be as low as one in a million, as it was in some European countries. That the U.S. does not, he thinks, is a sign that the government is really not interested in finding mad cows because of fears of an economic disaster.

Singeltary got into the field of transmissible spongiform encephalopathy in 1997, just after his mother died of sporadic CJD. She had an especially aggressive version—the Heidenhain variant—that first causes the patient to go blind and then to deteriorate rapidly. She died just ten weeks after her symptoms began. Singeltary, who said he had watched his grandparents die of cancer, considered her death by CJD to be much, much worse: “It’s something you never forget.” Her uncontrollable muscle twitching became so bad “that it took three of us to hold her one time,” Singeltary recalled. “She did everything but levitate in bed and spin her head.” Doctors originally diagnosed Alzheimer’s disease, but a postmortem neuropathological exam demanded by Singeltary revealed the true nature of her death.

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Classifying a disease as “sporadic” is another way for doctors to say they don’t know the cause. Normal prion proteins just turn rogue in the brain for no apparent reason. The term “sporadic” is often particularly hard for the victims’ families to accept, especially when the patient was previously in robust health. Maybe it was something in the water, they wonder, or in the air, or something they ate—the same questions CJD researchers tried to answer decades ago. The names “sporadic CJD” and “variant CJD” also confuse the public and raise suspicions that U.S. authorities are hiding something when they say there have been no native variant CJD cases in the country.

Singeltary suspected an environmental cause in his mother’s demise—a feeling reinforced a year later when a neighbor died of sporadic CJD. For years, the neighbor had been taking nutritional supplements that contained cow brain extracts. Researchers from the National Institutes of Health collected samples of the supplement, Singeltary recounted, and inoculated suspensions into mice. The mice remained healthy—which only means that those supplement samples tested were prion-free.

Scientists have made several attempts during the past few decades to find a connection between sporadic CJD and the environment. Often, these studies take the form of asking family members about CJD victims—their diet, occupation, medical history, hobbies, pets, and so forth—and comparing them with non-CJD subjects. Such case-control CJD studies have produced some intriguing—and sometimes contradictory—results. In 1985, Carleton Gajdusek and his NIH colleagues reported a correlation between CJD and eating a lot of roast pork, ham, hot dogs, and lamb, as well as rare meats and raw oysters.<sup>2</sup> Yet they also recognized that the findings were preliminary and that more studies were needed.

Following up, Robert Will of the U.K. National CJD Surveillance Unit and others pooled this data with those from two other case-control studies on CJD (one from Japan and one from the U.K.). In particular, they figured the so-called odds ratio—calculated by dividing the frequency of a possible factor in the patient group by the frequency of the factor in the control group. An odds ratio greater than 1 means that the factor may be significant. In their study, Will and his collaborators found an increase of CJD in people who have worked as health professionals (odds ratio of 1.5) and people who have had contact with cows

(1.7) and sheep (1.6). Unfortunately, those connections were not statistically significant: The numbers of pooled patients (117) and control subjects (333) were so small that the researchers felt the odds ratios needed to reach 2.5 to 8 (depending on the assumptions) before they could be deemed statistically significant. The only statistically significant correlations they found were between CJD and a family history of either CJD (19.1) or other psychotic disease (9.9), although the latter might simply be correlated because psychotic disease may be an early symptom of undiagnosed CJD.<sup>3</sup> In contrast with earlier findings, the team concluded that there was no association between sporadic CJD and the consumption of organ meats, including brains (0.6).

Although these case-control studies shed a certain amount of light on potential risk factors for CJD, it's impossible to draw firm conclusions. Obtaining data that produces statistically meaningful results can be difficult because of the rarity of CJD and hence the shortage of subjects. Human memory is quite fragile, too, so patients' families may not accurately recall the lifestyle and dietary habits of their loved ones over the course of a decade or more. Consequently, researchers must cope with data that probably contain significant biases. In a review paper on CJD, Joe Gibbs of the NIH and Richard T. Johnson of Johns Hopkins University concluded that "the absence of geographic differences in incidence is more convincing evidence against major dietary factors, since large populations eschew pork and some consume no meat or meat products." A CJD study of lifelong vegetarians, they proposed, could produce some interesting data.<sup>4</sup>

The inconclusive results of case-control studies do not completely rule out the environment as a possible cause of CJD. "Dr. Prusiner's theory does fit much of the data of spontaneous generation of [malformed] PrP somewhere in the brain," Will remarked—that is, the idea that sporadic CJD just happens by itself falls within the realm of the prion theory. Still, "it's very odd, if you look at all the forms of human prion diseases there are, all of them are transmissible in the laboratory and could be due to some sort of infectious agent."<sup>5</sup> One of the great difficulties, he explained, is that "given that this is a disease of an extraordinarily long incubation period, are we really confident that we can exclude childhood exposure that is transmitted from person to person, as people move around? It's difficult to be sure about that." There might be a "carrier state" that leaves people healthy yet still able to

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infect others. If so, "you would never be able to identify what's causing the spread of the disease," concluded Will, who hasn't stopped looking for a possible environmental link. He has some preliminary data based on studies that trace CJD victims' lives well before the time symptoms began—up to 70 years; they suggest some degree of geographic clustering, but no obvious candidates for a source of infection.

### A Case for Undercounting

The difficulty in establishing causal links in sporadic prion diseases—if there are any in the first place—underlines the importance of thorough surveillance. The U.K. has an active program, and when a victim of CJD is reported, one of Robert Will's colleagues visits and questions the victim's family. "No one has looked for CJD systematically in the U.S.," the NIH's Paul Brown noted. "Ever."<sup>6</sup> The U.S., through the Centers for Disease Control and Prevention, has generally maintained a more passive system, collecting information from

death certificates from the National Center for Health Statistics. Because CJD is invariably fatal, mortality data is considered to be an effective means of tabulating cases. The CDC assessed the accuracy of such data by comparing the numbers with figures garnered through an active search in 1996: Teams covering five regions of the U.S. contacted the specialists involved and reviewed medical records for CJD cases between 1991 and 1995. Comparing the actively garnered data with the death certificate information showed that “we miss about 14 percent,” said CDC epidemiologist Lawrence Schonberger. “That’s improving. Doctors are becoming more knowledgeable,” thanks to increased scientific and media attention given to prion diseases.<sup>7</sup>

The active surveillance study of 1996, however, only looked at cases in which physicians attributed the deaths to CJD. Misdiagnosed patients or patients who never saw a neurologist were not tabulated— thus CJD may be grossly underreported. Many neurological ailments share symptoms, especially early on. According to various studies, autopsies have found that CJD is misdiagnosed as other ills, such as dementia or Alzheimer’s disease, 5 to 13 percent of the time. The CDC finds that around 50,000 Americans die from Alzheimer’s each year

#### Laying Odds 227

(about 4 million have the disease, according to the Alzheimer’s Association). Therefore, one could argue that thousands of CJD cases are being missed. (On the flip side, CJD could be mistakenly diagnosed as Alzheimer’s disease or dementia, but the number of CJD patients is so small that they wouldn’t dramatically skew the statistics for other neurological ills.)

In part to address the issue of misdiagnosis, CJD families have asked the CDC to place the disease on the national list of officially notifiable illnesses, which tends to include more contagious conditions such as AIDS, tuberculosis, hepatitis, and viral forms of encephalitis. Currently, only some states impose this requirement. CDC officials have discounted the utility of such an approach, arguing that it would duplicate the mortality data, which is more accurate than early diagnoses of CJD, anyway. Moreover, mandatory reporting of CJD cases does not necessarily guarantee the end to missed cases.<sup>8</sup>

One clue suggests that the passive system is undercounting CJD in the U.S.: racial difference. The number of black CJD victims is about 38 percent that of white victims. Rather than sporadic CJD being a one-in-a-million lottery, it’s more like one-in-2.5-million for African Americans. Access to medical care might be one reason. Schonberger recounted that the CDC had asked other countries with substantial black populations to submit CJD figures for comparison but found that the surveillance in those countries was inadequate. “We haven’t been able to find any comparable literature on this issue, so it’s still up in the air,” Schonberger said. On the other hand, Alzheimer’s disease is more common among black people than whites, with an estimated higher prevalence ranging from 14 percent to almost 100 percent, according to a February 2002 report by the Alzheimer’s Association. Are some black CJD cases being misdiagnosed as Alzheimer’s?

Answering critics like Terry Singeltary, who feels that the U.S. undercounts CJD, Schonberger conceded that the current surveillance system has errors but stated that most of the errors will be confined to the older population. As Schonberger pointed out, no doctor would misdiagnose a 30-year-old CJD patient as having Alzheimer’s. The average age of the first 100 variant CJD victims was 29; should the epidemiology of vCJD change—if older people



start coming down with it—then there would be problems. “The adequacy of our overall CJD surveillance would be greatly reduced should the proportion of older individuals affected by variant CJD substantially increase,” Schonberger explained.<sup>9</sup>

SNIP...SEE FULL TEXT;

<http://ndl.ethernet.edu.et/bitstream/123456789/38386/1/516.pdf>

"The average age of the first 100 variant CJD victims was 29; should the epidemiology of vCJD change—if older people start coming down with it—then there would be problems. “The adequacy of our overall CJD surveillance would be greatly reduced should the proportion of older individuals affected by variant CJD substantially increase,” Schonberger explained.<sup>9</sup>"

<http://ndl.ethernet.edu.et/bitstream/123456789/38386/1/516.pdf>

THE PATHOLOGICAL PROTEIN by Philip Yam

[https://www.amazon.com/s?k=THE+PATHOLOGICAL+PROTEIN&i=stripbooks&crd=LA5IKGMF6PI9&srefix=the+pathological+protein%2Cstripbooks%2C96&ref=nb\\_sb\\_noss\\_1](https://www.amazon.com/s?k=THE+PATHOLOGICAL+PROTEIN&i=stripbooks&crd=LA5IKGMF6PI9&srefix=the+pathological+protein%2Cstripbooks%2C96&ref=nb_sb_noss_1)

Singeltary Submission SEAC 2007

SEAC SPONGIFORM ENCEPHALOPATHY ADVISORY COMMITTEE Minutes of the 99th meeting held on 14th December 2007 Singeltary Submission

This was 22 years to the day Mom died from the Heidenhain Variant of Creutzfeldt Jakob Disease i.e. hvCJD, when i made this submission to SEAC and this was their reply to my questions of concern about cjd in the USA, my how things have changed...terry

SEAC SPONGIFORM ENCEPHALOPATHY ADVISORY COMMITTEE Minutes of the 99th meeting held on 14th December 2007

ITEM 8 – PUBLIC QUESTION AND ANSWER SESSION 40. The Chair explained that the purpose of the question and answer session was to give members of the public an opportunity to ask questions related to the work of SEAC. Mr Terry Singeltary (Texas, USA) had submitted a question prior to the meeting, asking: “With the Nor-98 now documented in five different states so far in the USA in 2007, and with the two atypical BSE H-base cases in Texas and Alabama, with both scrapie and chronic wasting disease (CWD) running rampant in the USA, is there any concern from SEAC with the rise of sporadic CJD in the USA from “unknown phenotype”, and what concerns if any, in relations to blood donations, surgery, optical, and dental treatment, do you have with these unknown atypical phenotypes in both humans and animals in the USA? Does it concern SEAC, or is it of no concern to SEAC? Should it concern USA animal and human health officials?”

41. A member considered that this question appeared to be primarily related to possible links between animal and human TSEs in the USA. There is no evidence that sCJD is increasing in the USA and no evidence of any direct link between TSEs and CJD in the USA. Current evidence does not suggest that CWD is a significant risk to human health. There are

unpublished data from a case of human TSE in the USA that are suggestive of an apparently novel form of prion disease with distinct molecular characteristics. However, it is unclear whether the case had been further characterised, if it could be linked to animal TSEs or if other similar cases had been found in the USA or elsewhere. In relation to the possible public health implications of atypical scrapie, H-type BSE and CWD, research was being conducted to investigate possible links and surveillance was in place to detect any changes in human TSEs. Although possible links between these diseases and human TSEs are of concern and require research, there is no evidence to suggest immediate public health action is warranted. The possible human health risks from classical scrapie had been discussed earlier in the meeting. Members noted that there are effective channels of discussion and collaboration on research between USA and European groups. Members agreed it is important to keep a watching brief on new developments on TSEs.

<http://web.archive.org/web/20091010132933/http://www.seac.gov.uk/minutes/99.pdf>

Singeltary 2009

Monitoring the occurrence of emerging forms of Creutzfeldt-Jakob disease in the United States 2003 revisited 2009

August 10, 2009

Greetings,

I would like to submit a review of past CJD surveillance in the USA, and the urgent need to make all human TSE in the USA a reportable disease, in every state, of every age group, and to make this mandatory immediately without further delay. The ramifications of not doing so will only allow this agent to spread further in the medical, dental, surgical arena's. North America seems to have the most species with documented Transmissible Spongiform Encephalopathy's, most all of which have been rendered and fed back to food producing animals and to humans for years. If you look at the statistics, sporadic CJD seems to be rising in the USA, and has been, with atypical cases of the sCJD. I find deeply disturbing in the year of 2009, that Human Transmissible Spongiform Encephalopathy of any strain and or phenotype, of all age groups, and I stress all age groups, because human TSE's do not know age, and they do not know borders. someone 56 years old, that has a human TSE, that has surgery, can pass this TSE agent on i.e. friendly fire, and or passing it forward, and there have been documented nvCJD in a 74 year old. Remembering also that only sporadic CJD has been documented to transmit via iatrogenic routes, until recently with the 4 cases of blood related transmission, of which the origin is thought to be nvCJD donors. However most iatrogenic CJD cases are nothing more than sporadic CJD, until the source is proven, then it becomes iatrogenic. An oxymoron of sorts, because all sporadic CJD is, are multiple forms, or strains, or phenotypes of Creutzfeldt Jakob Disease, that the route and source and species have not been confirmed and or documented. When will the myth of the UKBSEnvCJD only theory be put to bed for good. This theory in my opinion, and the following there from, as the GOLD STANDARD, has done nothing more than help spread this agent around the globe. Politics and money have caused the terrible consequences to date, and the fact that TSEs are a slow incubating death, but a death that is 100% certain for those that are exposed and live long enough to go clinical. once clinical, there is no recourse, to date. But, while sub-clinical, how many can one exposed human infect? Can humans exposed to CWD and



scrapie strains pass it forward as some form of sporadic CJD in the surgical and medical arenas? why must we wait decades and decades to prove this point, only to expose millions needlessly, only for the sake of the industries involved? would it not have been prudent from the beginning to just include all TSE's, and rule them out from there with transmission studies and change policies there from, as opposed to doing just the opposite? The science of TSE's have been nothing more than a political circus since the beginning, and for anyone to still believe in this one strain, one group of bovines, in one geographical location, with only one age group of human TSE i.e. nvCJD myth, for anyone to believe this today only enhances to spreading of these human and animal TSE's. This is exactly why we have been in this quagmire.

The ones that believe that there is a spontaneous CJD in 85%+ of all cases of human TSE, and the ones that do not believe that cattle can have this same phenomenon, are two of the same, the industry, and so goes the political science aspect of this tobacco and or asbestos scenario i.e. follow the money. I could go into all angles of this man made nightmare, the real facts and science, for instance, the continuing rendering technology and slow cooking with low temps that brewed this stew up, and the fact that THE USA HAD THIS TECHNOLOGY FIRST AND SHIPPED IT TO THE U.K. SOME 5 YEARS BEFORE THE U.S. STARTED USING THE SAME TECHNOLOGY, to save on fuel cost. This is what supposedly amplified the TSE agent via sheep scrapie, and spread via feed in the U.K. bovine, and other countries exporting the tainted product. BUT most everyone ignores this fact, and the fact that the U.S. has been recycling more TSE, from more species with TSEs, than any other country documented, but yet, it's all spontaneous, and the rise in sporadic CJD in the U.S. is a happenstance of bad luck ??? I respectfully disagree. To top that all off, the infamous BSE-FIREWALL that the USDA always brags about was nothing more than ink on paper, and I can prove this. YOU can ignore it, but this is FACT (see source, as late as 2007, in one recall alone, some 10,000,000 MILLION POUNDS OF BANNED MAD COW FEED WENT OUT INTO COMMERCE TO BE FED OUT, and most was never recovered. This was banned blood laced, meat and bone meal. 2006 was a banner year for banned mad cow protein going into commerce in the U.S. (see source of FDA feed ban warning letter below). I stress that the August 4, 1997 USA mad cow feed ban and this infamous BSE firewall, was nothing more than ink on paper, it was never enforceable.

I propose that the current diagnostic criteria for human TSEs only enhances and helps the spreading of human TSE from the continued belief of the UKBSEnvCJD only theory in 2009. With all the science to date refuting it, to continue to validate this old myth, will only spread this TSE agent through a multitude of potential routes and sources i.e. consumption, medical i.e., surgical, blood, dental, endoscopy, optical, nutritional supplements, cosmetics etc. I propose as with Aguzzi, Asante, Collinge, Caughey, Deslys, Dormont, Gibbs, Gajdusek, Ironside, Manuelidis, Marsh, et al and many more, that the world of TSE Transmissible Spongiform Encephalopathy is far from an exact science, but there is enough proven science to date that this myth should be put to rest once and for all, and that we move forward with a new classification for human and animal TSE that would properly identify the infected species, the source species, and then the route. This would further have to be broken down to strain of species and then the route of transmission would further have to be broken down. Accumulation and Transmission are key to the threshold from sub- clinical to clinical disease, and key to all this, is to stop the amplification and transmission of this agent, the spreading of, no matter what strain. In my opinion, to continue with this myth that the U.K. strain of BSE one strain TSE in cows, and the nv/v CJD one strain TSE humans, and the one geographical

location source i.e. U.K., and that all the rest of human TSE are just one single strain i.e. sporadic CJD, a happenstance of bad luck that just happens due to a twisted protein that just twisted the wrong way, IN 85%+ OF ALL HUMAN TSEs, when to date there are 6 different phenotypes of sCJD, and growing per Gambetti et al, and that no other animal TSE transmits to humans ??? With all due respect to all Scientist that believe this, I beg to differ. To continue with this masquerade will only continue to spread, expose, and kill, who knows how many more in the years and decades to come. ONE was enough for me, My Mom, hvCJD i.e. Heidenhain Variant CJD, DOD 12/14/97 confirmed, which is nothing more than another mans name added to CJD, like CJD itself, Jakob and Creutzfeldt, or Gerstmann-Straussler-Scheinker syndrome, just another CJD or human TSE, named after another human. WE are only kidding ourselves with the current diagnostic criteria for human and animal TSE, especially differentiating between the nvCJD vs the sporadic CJD strains and then the GSS strains and also the FFI fatal familial insomnia strains or the ones that mimics one or the other of those TSE? Tissue infectivity and strain typing of the many variants of the human and animal TSEs are paramount in all variants of all TSE. There must be a proper classification that will differentiate between all these human TSE in order to do this. With the CDI and other more sensitive testing coming about, I only hope that my proposal will some day be taken seriously. ...

please see history, and the ever evolving TSE science to date ;

Saturday, June 13, 2009

Monitoring the occurrence of emerging forms of Creutzfeldt-Jakob disease in the United States 2003 revisited 2009

<https://journals.plos.org/plosone/article/comment?id=10.1371/annotation/04ce2b24-613d-46e6-9802-4131e2bfa6fd>

Singeltary 2010

Human Prion Diseases in the United States

Robert C. Holman ,Ermias D. Belay,Krista Y. Christensen,Ryan A. Maddox,Arialdi M. Minino,Arianne M. Folkema,Dana L. Haberling,Teresa A. Hammett,Kenneth D. Kochanek,James J. Sejvar,Lawrence B. Schonberger

Published: January 1, 2010

<https://doi.org/10.1371/journal.pone.0008521>

re-Human Prion Diseases in the United States

Posted by flounder on 01 Jan 2010 at 18:11 GMT

I kindly disagree with your synopsis for the following reasons ;

<https://journals.plos.org/plosone/article/comment?id=10.1371/annotation/04ce2b24-613d-46e6-9802-4131e2bfa6fd>

Singeltary 2014

Alzheimer's disease, iatrogenic transmission, what if?

let's not forget the elephant in the room. curing Alzheimer's would be a great and wonderful thing, but for starters, why not start with the obvious, lets prove the cause or causes, and then start to stop that. think iatrogenic, friendly fire, or the pass it forward mode of transmission. think medical, surgical, dental, tissue, blood, related transmission. think transmissible spongiform encephalopathy aka tse prion disease aka mad cow type disease...

Commentary: Evidence for human transmission of amyloid- $\beta$  pathology and cerebral amyloid angiopathy

<http://journals.plos.org/plosone/article/comment?id=info:doi/10.1371/annotation/933cc83a-a384-45c3-b3b2-336882c30f9d>

<http://journals.plos.org/plosone/article/comments?id=10.1371/journal.pone.0111492>

<http://journals.plos.org/plosone/article/comment?id=10.1371/annotation/933cc83a-a384-45c3-b3b2-336882c30f9d>

<https://www.frontiersin.org/articles/10.3389/fnagi.2016.00005/full>

\*\*\* Singeltary comment PLoS \*\*\*

Alzheimer's disease and Transmissible Spongiform Encephalopathy prion disease, Iatrogenic, what if ?

Posted by flounder on 05 Nov 2014 at 21:27 GMT

Alzheimer's disease and Transmissible Spongiform Encephalopathy prion disease, Iatrogenic, what if ?

## Background

Alzheimer's disease and Transmissible Spongiform Encephalopathy disease have both been around a long time, and was discovered in or around the same time frame, early 1900's. Both diseases are incurable and debilitating brain disease, that are in the end, 100% fatal, with the incubation/clinical period of the Alzheimer's disease being longer (most of the time) than the TSE prion disease. Symptoms are very similar, and pathology is very similar.

## Methods

Through years of research, as a layperson, of peer review journals, transmission studies, and observations of loved ones and friends that have died from both Alzheimer's and the TSE prion disease i.e. Heidenhain Variant Creutzfeldt Jakob Disease CJD.

## Results

I propose that Alzheimer's is a TSE disease of low dose, slow, and long incubation disease, and that Alzheimer's is Transmissible, and is a threat to the public via the many iatrogenic routes and sources. It was said long ago that the only thing that disputes this, is Alzheimer's disease transmissibility, or the lack of. The likelihood of many victims of Alzheimer's disease from the many different iatrogenic routes and modes of transmission as with the TSE prion disease.

## Conclusions

There should be a Global Congressional Science round table event set up immediately to address these concerns from the many potential routes and sources of the TSE prion disease, including Alzheimer's disease, and a emergency global doctrine put into effect to help combat the spread of Alzheimer's disease via the medical, surgical, dental, tissue, and blood arena's. All human and animal TSE prion disease, including Alzheimer's should be made reportable in every state, and Internationally, WITH NO age restrictions. Until a proven method of decontamination and autoclaving is proven, and put forth in use universally, in all hospitals and medical, surgical arena's, or the TSE prion agent will continue to spread. IF we wait until science and corporate politicians wait until politics lets science \_prove\_ this once and for all, and set forth regulations there from, we will all be exposed to the TSE Prion agents, if that has not happened already.

<http://www.plosone.org/annotation/listThread.action?root=82860>

MONDAY, APRIL 24, 2023

Prion Disease on the Rise in the U.S., Now the question is, why?

"5 cases per million in persons 55 years of age or older."

<https://creutzfeldt-jakob-disease.blogspot.com/2023/04/prion-disease-on-rise-in-us-now.html>

NATIONAL PRION DISEASE PATHOLOGY SURVEILLANCE CENTER SURVEILLANCE TABLES OF CASES EXAMINED January 11th, 2023

Tables of Cases Examined National Prion Disease Pathology Surveillance Center Cases Examined<sup>1</sup>

Updated quarterly.

Last updated on: January 11th, 2023

Year Total Neuropath Referrals<sup>2</sup> Prion Disease Sporadic Genetic Iatrogenic vCJD

1999 & earlier 383 232 202 27 3 0

2000 145 102 90 12 0 0

2001 209 118 110 8 0 0

2002 241 144 124 18 2 0

2003 259 160 137 21 2 0

2004 315 180 163 16 0 1<sup>3</sup>

2005 330 179 157 21 1 0

2006 365 179 159 17 1 2<sup>4</sup>

2007 374 210 191 19 0 0

2008 384 221 205 16 0 0

2009 397 231 210 20 1 0

2010 402 246 218 28 0 0

2011 392 238 214 24 0 0

2012 413 244 221 23 0 0

2013 416 258 223 34 1 0

2014 355 208 185 21 1 1<sup>5</sup>

2015 401 263 243 20 0 0

2016 395 277 248 29 0 0

2017 375 266 247 19 0 0

2018 308 221 202 18 1 0

2019 433 280 259 21 0 0

2020 366 252 227 24 1 0

2021 343 248 223 22 0 0

2022 307 199 165 13 0 0

TOTAL 83086 51567 46238 4919 14 4

Year CSF Only and RT-QulC Positive10

2015 241

2016 360

2017 406

2018 431

2019 538

2020 494

2021 516

2022 492

TOTAL 3478

1Listed based on the year of death or, if not available, on the year of referral;

2Cases with suspected prion disease for which brain tissue was submitted;

3Disease acquired in the United Kingdom;

4Disease acquired in the United Kingdom in one case and in Saudi Arabia in the other;

5Disease possibly acquired in a Middle Eastern or Eastern European country;

6Includes 25 cases in which the diagnosis is pending (1 from 2020, 2 from 2021 and 21 from 2022), and 20 inconclusive cases;

7Includes 24 (3 from 2021 and 21 from 2022) cases with type determination pending in which the diagnosis of vCJD has been excluded.

8The sporadic cases include 4504 cases of sporadic Creutzfeldt-Jakob disease (sCJD), 82 cases of Variably Protease-Sensitive Prionopathy (VPSPr) and 37 cases of sporadic Fatal Insomnia (sFI).

9Total does not include 301 Familial cases diagnosed by blood test only.

10Lists number of patients (deceased and alive) who have had a positive RT-QuIC and no neuropath examination.

For a downloadable PDF version of our quarterly table, please click the link below:

<https://case.edu/medicine/pathology/divisions/national-prion-disease-pathology-surveillance-center/surveillance/tables-cases-examined>

NPDPS C Table of Cases Examined

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Tables of Cases Examined | Pathology | School of Medicine | Case Western Reserve  
University [case.edu](http://case.edu)

National Prion Disease Pathology Surveillance Center Cases Examined<sup>1</sup> (September 20,  
2022) Year Total Neuropath Referrals<sup>2</sup> Prion Disease Sporadic Familial iCJD vCJD

1999 & earlier 383 232 202 27 3 0

2000 145 102 90 12 0 0

2001 209 118 110 8 0 0

2002 241 144 124 18 2 0

2003 259 160 137 21 2 0

2004 315 180 163 16 0 13

2005 328 179 157 21 1 0

2006 365 179 159 17 1 24

2007 374 210 191 19 0 0

2008 384 221 205 16 0 0

2009 397 231 210 20 1 0

2010 401 246 218 28 0 0

2011 392 238 214 24 0 0

2012 413 244 221 23 0 0

2013 416 258 223 34 1 0

2014 355 208 185 21 1 15

2015 401 263 243 20 0 0



2016 395 277 248 29 0 0

2017 375 266 247 19 0 0

2018 308 221 202 18 1 0

2019 434 281 259 22 0 0

2020 365 252 227 24 1 0

2021 343 248 223 22 0 0

2022 213 124 98 9 0 0

TOTAL 82116 50827 45568 4889 14 4

Year CSF Only & RT-QuIC Positive<sup>10</sup>

2015 140

2016 183

2017 227

2018 266

2019 311

2020 310

2021 341

2022 262

TOTAL 2040

1 Listed based on the year of death or, if not available, on year of referral;

2 Cases with suspected prion disease for which brain tissue and/or blood (in familial cases) were submitted;

3 Disease acquired in the United Kingdom;

4 Disease acquired in the United Kingdom in one case and in Saudi Arabia in the other;

5 Disease possibly acquired in a Middle Eastern or Eastern European country;

6 Includes 28 cases in which the diagnosis is pending (1 from 2020, 3 from 2021 and 24 from 2022), and 20 inconclusive cases;

7 Includes 20 (3 from 2021 and 17 from 2022) cases with type determination pending in which the diagnosis of vCJD has been excluded.

8 The sporadic cases include 4437 cases of sporadic Creutzfeldt-Jakob disease (sCJD), 82 cases of Variably Protease-Sensitive Prionopathy (VPSPr) and 37 cases of sporadic Fatal Insomnia (sFI).

9 Total does not include 300 Familial cases diagnosed by blood only. 10 Lists number of patients (deceased and alive) who have had a positive RT-QuIC and no neuropath examination.

<https://case.edu/medicine/pathology/sites/case.edu.pathology/files/2022-10/WebTable%20NPDPSC.pdf>

WEDNESDAY, JANUARY 25, 2023

Canada Creutzfeldt-Jakob disease surveillance system (CJDSS) report steady rise in cases as of January 2023 and STILL NO CASES REPORTED OF VPSPr CJD

<https://creutzfeldt-jakob-disease.blogspot.com/2023/01/canada-creutzfeldt-jakob-disease.html>

<https://vpspr.blogspot.com/>

NATIONAL PRION DISEASE PATHOLOGY SURVEILLANCE CENTER SURVEILLANCE TABLES OF CASES EXAMINED January 11th, 2023

<https://prionunitusaupdate.blogspot.com/2023/02/national-prion-disease-pathology.html>

Tuesday APRIL 05, 2022

Incidence of Creutzfeldt-Jakob Disease in the United States 1993-2014

[https://creutzfeldt-jakob-disease.blogspot.com/2022/04/incidence-of-creutzfeldt-jakob-disease\\_5.html](https://creutzfeldt-jakob-disease.blogspot.com/2022/04/incidence-of-creutzfeldt-jakob-disease_5.html)

ARS USDA Generation of human chronic wasting disease in transgenic mice

Publication Date: 9/26/2021

“We believe that our study provides the first evidence that CWD PrPSc is able to convert human PrPC into PrPSc in vitro and the CWD-derived human PrPSc mimics atypical sCJD subtypes in humanized Tg mice.”

<https://doi.org/10.1186/s40478-021-01262-y>

<https://www.ars.usda.gov/research/publications/publication/?seqNo115=382551>

SUNDAY, APRIL 9, 2023

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

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

<https://transmissiblespongiformencephalopathy.blogspot.com/2023/04/transmission-of-cervid-prions-to.html>

SEE A FEW HIGHLIGHTS;

"Here, we provide evidence for a zoonotic potential of CWD prions, and its probable signature using mice expressing human prion protein (PrP) as an infection model."

"Furthermore, the presence of infectious prions in feces is concerning because if this occurs in humans, it is a source for human-to-human transmission." "These findings have strong implications for public health and CWD management."

"We demonstrate that this transgenic line was susceptible to infection with CWD prions and displayed a distinct leading clinical sign, an atypical PrPSc signature and unusual fecal shedding of infectious prions."

"Importantly, these prions generated by the human PrP transgenic mice were transmissible upon passage."

Our results are the first evidence of a zoonotic risk of CWD when using one of the most common CWD strains, Wisc-1/CWD1 for "infection. "

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

"Indeed, such heterogeneity and distinct seeding activities and infectivity of abnormal PrP fragments was observed in VPSPr cases [20, 43]."

"This implies a high risk of exposure to this strain, e.g., through consumption or handling of infected carcasses, in contrast to rarer CWD strains, and therefore, an actual risk for human health."

"Fecal shedding of infectious prions, if it occurs in humans, is particularly concerning because of potential human-to-human transmission and adaptation of hCWD."

Overall, our findings suggest that CWD surveillance in humans should encompass a wider spectrum of tissues/organs tested and include new criteria in the diagnosis of potential patients.

\*\*\*> PLEASE NOTE;

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

snip...

"CWD in humans might remain subclinical but with PrPSc deposits in the brain with an unusual morphology that does not resemble the patterns usually seen in different prion diseases (e.g., mouse #328; Fig. 3), clinical with untraceable abnormal PrP (e.g., mouse #327) but still transmissible and uncovered upon subsequent passage (e.g., mouse #3063; Fig. 4), or prions have other reservoirs than the usual ones, hence the presence of infectivity in feces (e.g., mouse #327) suggesting a potential for human-to-human transmission and a real iatrogenic risk that might be unrecognizable. Here, humanized mice inoculated with CWD deer isolates had an atypical onset of the disease with myoclonus (93.75%), before presenting typical clinical signs, generating prions that presented with either atypical biochemical signature (#321 and #3063), shed in feces (#327), or were undetectable by the classical detection methods. The fact that we could not establish a strong correlation between disease manifestation in tg650 mice inoculated with Wisc-1- or 116AG-CWD and the presence of abnormal PrP (Western blot, IHC or RTQuIC) might be explained by the presence of heterogeneous prions in the brains of infected mice with different seeding properties in vitro. Indeed, such heterogeneity and distinct seeding activities and infectivity of abnormal PrP fragments was observed in VPSPr cases [20, 43]."

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

VPSPr, GSS, and CWD zoonosis, concerns there from, where did i hear this concern before?

1. 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>

FRIDAY, APRIL 07, 2023

\*\*\*> Case report: Two clusters of Creutzfeldt-Jakob disease cases within 1 year in West Michigan <\*\*\*

<https://creutzfeldt-jakob-disease.blogspot.com/2023/04/case-report-two-clusters-of-creutzfeldt.html>

TUESDAY, APRIL 11, 2023

Texas TAHC Chronic Wasting Disease Discovered in Deer Breeding Facilities in Frio and Hamilton Counties

<https://chronic-wasting-disease.blogspot.com/2023/04/texas-tahc-chronic-wasting-disease.html>

TUESDAY, MARCH 21, 2023

Texas CWD seven new cases three separate deer-breeding facilities in Zavala, Washington and Gonzales counties 471 confirmed to date

<https://chronic-wasting-disease.blogspot.com/2023/03/texas-cwd-seven-new-cases-three.html>

THURSDAY, APRIL 27, 2023

TEXAS REPUBLICAN SB 1372 TAXPAYERS TO PAY FOR GAME FARMS CWD DEPOPULATION

<https://chronic-wasting-disease.blogspot.com/2023/04/texas-republican-sb-1372-taxpayers-to.html>

Terry S. Singeltary Sr.