WEDNESDAY, APRIL 25, 2018

Docket No. APHIS-2018-0012 Concurrence With OIE Risk Designations for Bovine Spongiform Encephalopathy Singeltary Submission

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Greetings again APHIS et al,

i would kindly like to submit my comments regarding Docket No. APHIS-2018-0012 Concurrence With OIE Risk Designations for Bovine Spongiform Encephalopathy.

[Federal Register Volume 83, Number 79 (Tuesday, April 24, 2018)] [Notices] [Page 17789] From the Federal Register Online via the Government Publishing Office [www.gpo.gov] [FR Doc No: 2018-08430]

DEPARTMENT OF AGRICULTURE

Animal and Plant Health Inspection Service

[Docket No. APHIS-2018-0012]

Concurrence With OIE Risk Designations for
Bovine Spongiform Encephalopathy

AGENCY: Animal and Plant Health Inspection Service, USDA.

ACTION: Notice.

SUMMARY: We are advising the public of our preliminary concurrence with the World Organization for Animal Health's (OIE) bovine spongiform encephalopathy (BSE) risk designations for four regions. The OIE recognizes these regions as being of negligible risk for BSE. We are taking this action based on our review of information supporting the OIE's risk designations for these regions.

DATES: We will consider all comments that we receive on or before June 25, 2018.

ADDRESSES: You may submit comments by either of the following methods: Federal eRulemaking Portal: Go to http://www.regulations.gov/#!docketDetail;D=APHIS-2018-0012. Postal Mail/Commercial Delivery: Send your comment to Docket No. APHIS-2018-0012, 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

http://www.regulations.gov/#!docketDetail;D=APHIS-2018-0012

or in our reading room, which is located in room 1141 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: Dr. Rebecca Gordon, Senior Staff Veterinarian, Regionalization Evaluation Services, National Import Export Services, VS, APHIS, 920 Main Campus Drive, Suite

ABOUT ME

TERRY S. SINGELTARY SR.
BACLIFF, TEXAS, UNITED STATES

My mother was murdered by what I call corporate and political homicide i.e. FOR PROFIT! she died from a rare phenotype of CJD i.e. the Heidenhain Variant of Creutzfeldt Jakob Disease i.e. sporadic, simply meaning from unknown route and source. I have simply been trying to validate her death DOD 12/14/97 with the truth. There is a route, and there is a source. There are many here in the USA. WE must make CJD and all human TSE, of all age groups 'reportable' Nationally and Internationally, with a written CJD questionnaire asking real questions pertaining to route and source of this agent. Friendly fire has the potential to play a huge role in the continued transmission of this agent via the medical, dental, and surgical arena. We must not flounder any longer. ...TSS

VIEW MY COMPLETE PROFILE

PREVIOUS POSTS

TAHC TEXAS CHRONIC WASTING DISEASE CWD TSE PRION S...
Michigan-Sportsman.com, Dr. Jim Brauker or tornado...
MINNESOTA STATE AUDITORS Board of Animal Health ha...
Use of environmental sites by mule deer: a proxy f...
Theodore Roosevelt Conservation Partnership Chroni...
Wisconsin CWD detection in a wild deer in Eau Clai...
Chronic wasting disease is a threat to Wyoming's d...
Chronic wasting disease: Bambi vs. the prion
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200, Raleigh, NC 27606; (919) 855-7741.

SUPPLEMENTARY INFORMATION: The regulations in 9 CFR part 92 subpart B, ``Importation of Animals and Animal Products; Procedures for Requesting BSE Risk Status Classification With Regard To Bovines'' (referred to below as the regulations), set forth the process by which the Animal and Plant Health Inspection Service (APHIS) classifies regions for bovine spongiform encephalopathy (BSE) risk. Section 92.5 of the regulations provides that all countries of the world are considered by APHIS to be in one of three BSE risk categories: Negligible risk, controlled risk, or undetermined risk. These risk categories are defined in Sec. 92.1. Any region that is not classified by APHIS as presenting either negligible risk or controlled risk for BSE is considered to present an undetermined risk. The list of those regions classified by APHIS as having either negligible risk or controlled risk can be accessed on the APHIS website at.

https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/animal-and-animal-product-import-information/import-live-animals/ct_bovine_spongiform_encephalopathy

The list can also be obtained by writing to APHIS at National Import Export Services, 4700 River Road, Unit 38, Riverdale, MD 20737.

Under the regulations, APHIS may classify a region for BSE in one of two ways.

One way is for regions that have not received a risk classification from the World Organization for Animal Health (OIE) to request classification by APHIS.

The other way is for APHIS to concur with the classification given to a country or region by the OIE.

If the OIE has classified a region as either BSE negligible risk or BSE controlled risk, APHIS will seek information to support concurrence with the OIE classification. This information may be publicly available information, or APHIS may request that regions supply the same information given to the OIE. APHIS will announce in the Federal Register, subject to public comment, its intent to concur with an OIE classification.

In accordance with this process, we are giving notice in this document that APHIS intends to concur with the OIE risk classifications of the following regions:

Regions of negligible risk for BSE: Croatia, Poland, Northern Ireland (region of United Kingdom), and Scotland (region of United Kingdom).

The OIE recommendations regarding each of the above regions can be viewed at .

http://www.oie.int/animal-health-in-the-world/official-disease-status/bse/list-of-bse-risk-status/

The conclusions of the OIE scientific commission for these regions can be viewed at:

Croatia: **http://www.oie.int/fileadmin/Home/eng/Internationa_Standard_Setting/docs/pdf/SCAD/A_SCAD_Feb2014.pdf** (page 88).

Poland: **http://www.oie.int/fileadmin/Home/eng/Internationa_Standard_Setting/docs/pdf/SCAD/A_SCAD_Feb2017.pdf** (page 71).

Northern Ireland: **http://www.oie.int/fileadmin/Home/eng/Internationa_Standard_Setting/docs/pdf/SCAD/A_SCAD_Feb2017.pdf** (page 74).

Scotland: **http://www.oie.int/fileadmin/Home/eng/Internationa_Standard_Setting/docs/pdf/SCAD/A_SCAD_Feb2017.pdf** (page 76).

After reviewing any comments we receive, we will announce our final determination regarding the BSE classification of these countries in the Federal Register, along with a discussion of and response to pertinent issues raised by commenters. If APHIS recognizes a region as either negligible risk or controlled risk for BSE, the Agency will include that region on the list of regions of negligible risk or controlled risk for BSE,

as applicable, that is available to the public on the Agency's website at.

https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/animal-and-animal-product-import-information/import-live-animals/ct_bovine_spongiform_encephalopathy

Authority: 7 U.S.C. 1622 and 8301-8317; 21 U.S.C. 136 and 136a; 31 U.S.C. 9701; 7 CFR 2.22, 2.80, and 371.4.

Done in Washington, DC, this 18th day of April 2018. Kevin Shea, Administrator, Animal and Plant Health Inspection Service. [FR Doc. 2018-08430 Filed 4-23-18; 8:45 am] BILLING CODE 3410-34-P

https://www.gpo.gov/fdsys/pkg/FR-2018-04-24/html/2018-08430.htm

Docket No. APHIS-2018-0012 Concurrence With OIE Risk Designations for Bovine Spongiform Encephalopathy Singeltary Submission

Greetings again APHIS et al,

i would kindly like to submit my comments regarding Docket No. APHIS-2018-0012 Concurrence With OIE Risk Designations for Bovine Spongiform Encephalopathy.

i would kindly like to address several issues regarding BSE and other strains of Transmissible Spongiform Encephalopathy TSE Prion disease, due to the potential ramifications of the new outbreak of Camel TSE Prion disease (Mad Camel Disease) in Algeria, and imports and exports there from, the recent findings that Scrapie of sheep and goats and Chronic Wasting Disease of Cervid will transmit to PIGS by the ORAL route, and the recent findings of the Macaque studies, and oral transmission of CWD and Scrapie TSE Prion transmission to the macaque, one of the closets monkey's to the human species, and remember, transmission studies to humans from TSE Prion are illegal. So, there is much new science and must concern here, and in my opinion, the BSE MRR i.e. Minimal Risk Region is a disaster that is not waiting to happen, but has already happened, by allowing the legal trading of the TSE Prion across the globe.

I once again urge strenuously that the BSE MRR policy be withdrawn and abolished, and that the BSE GBR Risk Assessments be implemented once again, to include ALL TSE PRION DISEASE IN EVERY SPECIES. The ruminant and mammalian feed bans have failed us terrible, the BSE surveillance in the USA failed terribly, and the BSE testing program was a failure as well, and all proven to be so by the GAO and the OIG. The massive, copious amounts of banned Bovine Spongiform Encephalopathy BSE animal feeds for ruminants rule, 21 CFR 589.200 i.e. mad cow feed, is so great still to this day, some 2 plus decades post Bovine Spongiform Encephalopathy BSE animal feeds for ruminants rule, 21 CFR 589.200 i.e. mad cow feed of August 1997, that there should be an emergency meeting to address the mad cow feed ban immediately, considering the recent findings of cwd and scrapie transmitting to pigs.

there should also be a DECLARATION OF EMERGENCY DECLARED OF A FOREIGN ANIMAL DISEASE FROM THE USA, i.e. CWD TSE Prion, and this declaration should go out to the world, as there was back On July 14, 2000, USDA issued a declaration of extraordinary emergency to acquire the sheep. but those test were wrong, and a decade later after FOIA request after request, turns out those sheep from Belgium never had any TSE Prion disease. long story, but what is the difference here, especially since we are dealing with Chronic Wasting Disease CWD TSE Prion, and the fact now that not only has CWD been exported from North America to South Korea, and to Norway, but now Finland has confirmed it's first case of Chronic Wasting Disease CWD TSE Prion. So, where does the 'BUCK' stop?

why has this 'DECLARATION OF EXTRAORDINARY EMERGENCY FOR FOREIGN ANIMAL DISEASE OF THE United States of America USA' due to Chronic Wasting Disease CWD Transmissible Spongiform Encephalopathy TSE Prion disease, not already been declared, and why has not a Intercontinental, International, Interstate movements of cervid BAN not already been put in place, especially since the recent findings of oral transmission studies with the Macaque, in relations with oral transmission of muscle meat with cwd, and oral transmission of cwd to the pig?

do we just continue to truck, ship, or fly this CWD TSE Prion all around the globe, just to save the industry?

see; August 15, 2000 OIG case # NY-3399-56 REDACTED, VT

"Enclosed is OIG's notification that they have scheduled an investigation of the following individual. REDACTED is alleged to have provided possibly inaccurate test results involving diseased sheep. However, because the results were determined to be inconclusive, no actual violation was actually committed."

http://foiamadsheepmadrivervalley.blogspot.com/

I believe that before addressing BSE TSE Prion related issues with Croatia, Poland, Northern Ireland, Scotland, that APHIS/USDA/FSIS/FDA et al should clean up there own back yards. Much BSE TSE Risk factor still reside right here in the USA and North America.

> "One way is for regions that have not received a risk classification from the World Organization for Animal Health (OIE) to request classification by APHIS" <

IN my honest opinion, this is the same as letting the wolf guard the hen house.

My Submission of Scientific Facts as follows, and why i believe that the BSE MRR is a terribly failed policy, that only allows the continued spread of the TSE Prion agent, and why THE BSE MRR MUST BE REPEALED IMMEDIATELY!

NEW
TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHY TSE PRION DISEASE (MAD CAMEL DISEASE) IN A NEW SPECIES

NEW OUTBREAK OF
TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHY TSE PRION DISEASE IN A NEW SPECIES

Subject: Prion Disease in Dromedary Camels, Algeria

Our identification of this prion disease in a geographically widespread livestock species requires urgent enforcement of surveillance and assessment of the potential risks to human and animal health.

https://wwwnc.cdc.gov/eid/article/24/6/17-2007_article

http://camelusprp.blogspot.com/2018/04/tse-prion-disease-in-dromedary-camels.html

> IMPORTS AND EXPORTS <

http://camelusprp.blogspot.com/2018/04/dromedary-camels-algeria-prion-mad.html

2017 USAHA RESOLUTION

RESOLUTION NUMBER: 1 Combined with 6, 13, 16, and 22 APPROVED

SUBJECT MATTER: Adequate Funding for Prevention, Diagnosis, and Response for Foreign Animal Disease Outbreaks

http://www.usaha.org/upload/Resolution/2017/Resolution_1_6_13_16_22_FAD_Sup.pdf

http://camelusprp.blogspot.com/2018/04/genetic-variation-of-prion-protein-gene.html

Scrapie Transmits To Pigs By Oral Route, what about the terribly flawed USA tse prion feed ban?

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

Location: Virus and Prion Research 2017 Annual Report

1a. Objectives (from AD-416): 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.

1b. Approach (from AD-416): 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.

3. 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.

4. 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 (PrPSc) 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 PrPSc 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 Hwang, S., Greenlee, J.J., Nicholson, E.M. 2017. Use of bovine recombinant prion protein and real-time quaking-induced conversion to detect cattle transmissible mink encephalopathy prions and discriminate classical and atypical L- and H-type
bovine spongiform encephalopathy. PLoS One. 12(2):e0172391. Moore, S., Kunkle, R., Greenlee, M., Nicholson, E., Richt, J., Hamir, A., Waters, W., Greenlee, J. 2016. Horizontal transmission of chronic wasting disease in reindeer. Emerging Infectious Diseases. 22(12):2142-2145. doi:10.3201/eid2212.160635. Moore, S.J., West Greenlee, M.H., Smith, J.D., Vrentas, C.E., Nicholson, E.M., Greenlee, J.J. 2016. A comparison of classical and H-type
bovine spongiform encephalopathy associated with E211K prion protein polymorphism in wild type and EK211 cattle following intracranial inoculation. Frontiers in Veterinary Science. 3:78. Greenlee, J.J., Kunkle, R.A., Smith, J.D., West Greenlee, M.H. 2016. Scrapie in swine: a diagnostic challenge. Food Safety. 4(4):110-114. Kondru, N., Manne, S., Greenlee, J., West Greenlee, H., Anantharam, V., Halbur, P., Kanthasamy, A., Kanthasamy, A. 2017. Integrated organotypic slice cultures and RT-QuIC (OSCAR) assay: implications for translational discovery in
protein misfolding diseases. Scientific Reports. 7:43155. doi:10.1038/srep43155. Mammadova, N., Ghaisas, S., Zenitsky, G., Sakaguchi, D.S., Kanthasamy, A.G., Greenlee, J.J., West Greenlee, M.H. 2017. Lasting retinal injury in a mouse model of blast-induced

trauma. American Journal of Pathology. 187(7):1459-1472. doi:10.1016/j.ajpath.2017.03.005.

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

> 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. <***

FRIDAY, APRIL 20, 2018

Scrapie Transmits To Pigs By Oral Route, what about the terribly flawed USA tse prion feed ban?

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

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

TUESDAY, APRIL 24, 2018

ARS Research atypical Nor98 and Michigan Scrapie, CWD, CJD and mad cow feed

Research Project: Genetic Impact and Improved Diagnostics for Sheep and Goat Transmissible Spongiform Encephalopathies

http://nor-98.blogspot.com/2018/04/ars-research-atypical-nor98-and.html

TUESDAY, APRIL 17, 2018

Chronic wasting disease: Bambi vs. the prion

Research Project: Immunodiagnostics to Detect Prions and Other Important Animal Pathogens

Location: Produce Safety and Microbiology Research

http://chronic-wasting-disease.blogspot.com/2018/04/chronic-wasting-disease-bambi-vs-prion.html

> CWD TO PIGS <

Research Project: TRANSMISSION, DIFFERENTIATION, AND PATHOBIOLOGY OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES

Location: Virus and Prion Research

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

Author item Moore, Sarah item Kunkle, Robert item Kondru, Naveen item Manne, Sireesha item Smith, Jodi item Kanthasamy, Anumantha item West Greenlee, M item Greenlee, Justin

Submitted to: Prion Publication Type: Abstract Only Publication Acceptance Date: 3/15/2017 Publication Date: N/A Citation: N/A Interpretive Summary:

Technical Abstract: Aims: Chronic wasting disease (CWD) is a naturally-occurring, fatal neurodegenerative disease of cervids. We previously demonstrated that disease-associated prion protein (PrPSc) can be detected in the brain and retina from pigs challenged intracranially or orally with the CWD agent. In that study, neurological signs consistent

with prion disease were observed only in one pig: an intracranially challenged pig that was euthanized at 64 months post-challenge. The purpose of this study was to use an antigen-capture immunoassay (EIA) and real-time quaking-induced conversion (QuIC) to determine whether PrPSc is present in lymphoid tissues from pigs challenged with the CWD agent.

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

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

This study demonstrates that PrPSc accumulates in lymphoid tissues from pigs challenged intracranially or orally with the CWD agent, and can be detected as early as 4 months after challenge.

CWD-infected pigs rarely develop clinical disease and if they do, they do so after a long incubation period. This raises the possibility that CWD-infected pigs could shed prions into their environment long before they develop clinical disease.

Furthermore, lymphoid tissues from CWD-infected pigs could present a potential source of CWD infectivity in the animal and human food chains.

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

CONFIDENTIAL

EXPERIMENTAL PORCINE SPONGIFORM ENCEPHALOPATHY

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

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

snip...see much more here ;

WEDNESDAY, APRIL 05, 2017

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

http://chronic-wasting-disease.blogspot.com/2017/04/disease-associated-prion-protein.html

WEDNESDAY, APRIL 05, 2017

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

http://chronic-wasting-disease.blogspot.com/2017/04/disease-associated-prion-protein.html

> CATTLE ARE HIGHLY SUSCEPTIBLE TO WHITE-TAILED DEER CWD AND MULE DEER CWD <

***> cattle are highly susceptible to white-tailed deer CWD and mule deer CWD

***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/qra-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

THE Aug. 1997 mad cow feed ban was/is a joke, BSE surveillance also was proven to be terribly flawed, along with BSE testing, shown to be flawed as well.

ALSO, WHAT ABOUT CWD TRANSMITTING TO PIGS AS WELL, AND MAD CAMEL DISEASE NOW, BIG OUTBREAK, NOT SPONTANEOUS, WHAT ABOUT THAT, and the feed ban concern there as well? AND what about Scrapie transmission to the Macaque recently. seems the tse prion poker continue to goes up. very worrying.

P.97 : Scrapie transmits to white-tailed deer by the oral route and has a molecular profile similar to chronic wasting disease and distinct from the scrapie inoculum

Justin Greenlee1, S JO Moore1, Jodi Smith1, M Heather WestGreenlee2 and Robert Kunkle1

1National Animal Disease Center; Ames, IA USA

2Iowa State University; Ames, IA USA

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

***In summary, this work demonstrates that WTD are susceptible to the agent of scrapie, 2 distinct molecular profiles of PrPSc are present in the tissues of affected deer, and inoculum of either profile readily passes to deer.

https://www.tandfonline.com/doi/full/10.1080/19336896.2015.1033248

*** After a natural route of exposure, 100% of WTD were susceptible to scrapie.

PO-039: A comparison of scrapie and chronic wasting disease in white-tailed deer Justin Greenlee, Jodi Smith, Eric Nicholson US Dept. Agriculture; Agricultural Research Service, National Animal Disease Center; Ames, IA USA

http://www.landesbioscience.com/journals/prion/03-Prion6-2-Transmission-and-strains.pdf

White-tailed deer are susceptible to the agent of sheep scrapie by intracerebral inoculation

snip...

It is unlikely that CWD will be eradicated from free-ranging cervids, and the disease is likely to continue to spread geographically [10]. However, the potential that white-tailed deer may be susceptible to sheep scrapie by a natural route presents an additional confounding factor to halting the spread of CWD. This leads to the additional speculations that

1) infected deer could serve as a reservoir to infect sheep with scrapie offering challenges to scrapie eradication efforts and

2) CWD spread need not remain geographically confined to current endemic areas, but could occur anywhere that sheep with scrapie and susceptible cervids cohabitate.

This work demonstrates for the first time that white-tailed deer are susceptible to sheep scrapie by intracerebral inoculation with a high attack rate and that the disease that results has similarities to CWD. These experiments will be repeated with a more natural

route of inoculation to determine the likelihood of the potential transmission of sheep scrapie to white-tailed deer. If scrapie were to occur in white-tailed deer, results of this study indicate that it would be detected as a TSE, but may be difficult to differentiate from CWD without in-depth biochemical analysis.

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3199251/?tool=pubmed

http://chronic-wasting-disease.blogspot.com/2011/10/white-tailed-deer-are-susceptible-to.html

2012

PO-039: A comparison of scrapie and chronic wasting disease in white-tailed deer

Justin Greenlee, Jodi Smith, Eric Nicholson US Dept. Agriculture; Agricultural Research Service, National Animal Disease Center; Ames, IA USA

snip...

The results of this study suggest that there are many similarities in the manifestation of CWD and scrapie in WTD after IC inoculation including early and widespread presence of PrPSc in lymphoid tissues, clinical signs of depression and weight loss progressing to wasting, and an incubation time of 21-23 months. Moreover, western blots (WB) done on brain material from the obex region have a molecular profile similar to CWD and distinct from tissues of the cerebrum or the scrapie inoculum. However, results of microscopic and IHC examination indicate that there are differences between the lesions expected in CWD and those that occur in deer with scrapie: amyloid plaques were not noted in any sections of brain examined from these deer and the pattern of immunoreactivity by IHC was diffuse rather than plaque-like.

*** After a natural route of exposure, 100% of WTD were susceptible to scrapie.

Deer developed clinical signs of wasting and mental depression and were necropsied from 28 to 33 months PI. Tissues from these deer were positive for PrPSc by IHC and WB. Similar to IC inoculated deer, samples from these deer exhibited two different molecular profiles: samples from obex resembled CWD whereas those from cerebrum were similar to the original scrapie inoculum. On further examination by WB using a panel of antibodies, the tissues from deer with scrapie exhibit properties differing from tissues either from sheep with scrapie or WTD with CWD. Samples from WTD with CWD or sheep with scrapie are strongly immunoreactive when probed with mAb P4, however, samples from WTD with scrapie are only weakly immunoreactive. In contrast, when probed with mAb's 6H4 or SAF 84, samples from sheep with scrapie and WTD with CWD are weakly immunoreactive and samples from WTD with scrapie are strongly positive. This work demonstrates that WTD are highly susceptible to sheep scrapie, but on first passage, scrapie in WTD is differentiable from CWD.

http://www.landesbioscience.com/journals/prion/03-Prion6-2-Transmission-and-strains.pdf

2011

*** After a natural route of exposure, 100% of white-tailed deer were susceptible to scrapie.

http://www.usaha.org/Portals/6/Reports/2011/report-cwal-2011.pdf

-----Original Message-----
From: Terry Singeltary <**flounder9@verizon.net**>
To: Tracy.A.Nichols <**Tracy.A.Nichols@aphis.usda.gov**>
Sent: Fri, Mar 30, 2018 12:51 pm
Subject: Docket No. APHIS-2018-0011 Chronic Wasting Disease Herd Certification Program Standards Singeltary Submission March 30, 2018

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

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

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

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

FOREIGN ANIMAL DISEASE' declared in the USA way back On July 10, 2000, several sheep from the flock tested positive for a TSE, a class of degenerative neurological diseases that is characterized by a very long incubation period and a 100 percent mortality rate in infected sheep. Two of the better known varieties of TSE are scrapie in sheep and BSE in cattle. On July 14, 2000, USDA issued a declaration of extraordinary emergency to acquire the sheep. but those test were wrong, and a decade later after FOIA request after request, turns out those sheep from Belgium never had any TSE Prion disease. long story, but what is the difference here, especially since we are dealing with Chronic Wasting Disease CWD TSE Prion, and the fact now that not only has CWD been exported from North America to South Korea, and to Norway, but now Finland has confirmed it's first case of Chronic Wasting Disease CWD TSE Prion. So, where does the 'BUCK' stop? why has this 'DECLARATION OF EXTRAORDINARY EMERGENCY FOR FOREIGN ANIMAL DISEASE OF THE United States of America USA' due to Chronic Wasting Disease CWD Transmissible Spongiform Encephalopathy TSE Prion disease, not already been declared, and why has not a Intercontinental, International, Interstate movements of cervid BAN not already been put in place, especially since the recent findings of oral transmission studies with the Macaque, in relations with oral transmission of muscle meat with cwd, and oral transmission of cwd to the pig? do we just continue to truck, ship, or fly this CWD TSE Prion all around the globe, just to save the industry? see; August 15, 2000 OIG case # NY-3399-56 REDACTED, VT "Enclosed is OIG's notification that they have scheduled an investigation of the following individual. REDACTED is alleged to have provided possibly inaccurate test results involving diseased sheep. However, because the results were determined to be inconclusive, no actual violation was actually committed."

http://foiamadsheepmadrivervalley.blogspot.com/

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

3. INDEMNITY, NO MORE Federal indemnity program, or what i call, ENTITLEMENT PROGRAM for game farm industry. NO MORE BAIL OUTS FROM TAX PAYERS. if the captive industry can't buy insurance to protect not only themselves, but also their customers, and especially the STATE, from Chronic Wasting Disease CWD TSE Prion or what some call mad deer disease and harm therefrom, IF they can't afford to buy that insurance that will cover all of it, then they DO NOT GET A PERMIT to have a game farm for anything. This CWD TSE Prion can/could/has caused property values to fall from some reports in some places. roll the dice, how much is a state willing to lose?

4. QUARANTINE OF ALL CAPTIVE, BREEDERS, URINE, ANTLER, VELVET, SPERM, OR ANY FACILITY that has been confirmed to have Chronic Wasting Disease CWD TSE Prion, the QUARANTINE should be for 21 years due to science showing what scrapie can do. 5 years is NOT enough. see; Infectious agent of sheep scrapie may persist in the environment for at least 16 years
Gudmundur Georgsson,1 Sigurdur Sigurdarson2 and Paul Brown3Correspondence Gudmundur Georgsson **ggeorgs@hi.is1** Institute for Experimental Pathology, University of Iceland, Keldur v/vesturlandsveg, IS-112 Reykjav1´k, Iceland2 Laboratory of the Chief Veterinary Officer, Keldur, Iceland3 Bethesda, Maryland, USAReceived 7 March 2006Accepted 6 August 2006In 1978, a rigorous programme was implemented to stop the spread of, and subsequently eradicate, sheep scrapie in Iceland. Affected flocks were culled, premises were disinfected and, after 2–3 years, restocked with lambs from scrapie-free areas. Between 1978 and 2004, scrapie recurred on 33 farms. Nine of these recurrences occurred 14–21 years after culling, apparently as the result of environmental contamination, but outside entry could not always be absolutely excluded. Of special interest was one farm with a small, completely self-contained flock where scrapie recurred 18 years after culling, 2 years after some lambs had been housed in an old sheephouse that had never been disinfected. Epidemiological investigation established with near certitude that the disease had not been introduced from the outside and it is concluded that the agent may have persisted in the old sheep-house for at least 16 years.

http://www.microbiologyresearch.org/docserver/fulltext/jgv/87/12/3737.pdf?expires=1521907990&id=id&accname=guest&checksum=51DB085BD612A0603240F09E29D4AADD

Survival of Scrapie virus after 3 years interment

Paul Brown, D. Carleton Gajdusek

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

Back around 2000, 2001, or so, I was corresponding with officials abroad during the bse inquiry, passing info back and forth, and some officials from here inside USDA aphis FSIS et al. In fact helped me get into the USA 50 state emergency BSE conference

call way back. That one was a doozy. But I always remember what "deep throat" I never knew who they were, but I never forgot;

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

Confidential!!!!

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

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

5. DESCRIBING APHIS' intent to amend the regulations to define susceptible species based on scientific evidence of natural infection or experimental infections through natural routes and adding the genera Rangifer and Muntiacus to the list of susceptible species...

snip...see full text;

*** APHIS USDA CFIA CWD TSE Prion Herd Certifications Update ***

FRIDAY, MARCH 30, 2018

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

Terry S. Singeltary Sr., Bacliff, Texas USA
77518 **flounder9@verizon.net** Attachments (1) Docket No. APHIS-2018-0011 Chronic Wasting Disease Herd Certification Program Standards Singeltary View Attachment:View as format pdf

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

https://www.regulations.gov/contentStreamer?documentId=APHIS-2018-0011-0003&attachmentNumber=1&contentType=pdf

https://www.regulations.gov/docketBrowser?rpp=25&so=DESC&sb=commentDueDate&po=0&dct=PS&D=APHIS-2018-0011

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

WEDNESDAY, OCTOBER 4, 2017

*** EFSA Scientific Report on the Assessment of the Geographical BSE-Risk (GBR) of the United States of America (USA) a review 2017 ***

http://bseusa.blogspot.com/2017/10/efsa-scientific-report-on-assessment-of.html

TUESDAY, AUGUST 8, 2017

Concurrence With OIE Risk Designations for Bovine Spongiform Encephalopathy [Docket No. APHIS-2016-0092]

http://animalhealthreportpriontse.blogspot.com/2017/08/concurrence-with-oie-risk-designations.html

I urge everyone to watch this video closely...terry

*** you can see video here and interview with Jeff's Mom, and scientist telling you to test everything and potential risk factors for humans ***

https://histodb11.usz.ch/Images/videos/video-004/video-004.html

ONE DECADE POST MAD COW FEED BAN OF AUGUST

1997…2007

ONE DECADE POST MAD COW FEED BAN OF AUGUST 1997…2007

2007

10,000,000 POUNDS 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.

2007

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

RECALLS AND FIELD CORRECTIONS: VETERINARY MEDICINES -- CLASS II 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

PAGE NOT FOUND

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

ALABAMA MAD COW FEED IN COMMERCE 2006

RECALLS AND FIELD CORRECTIONS: VETERINARY MEDICINE -- CLASS II

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PRODUCT

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

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

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

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

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

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

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

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

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

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

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

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

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

CODE

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

RECALLING FIRM/MANUFACTURER

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

REASON

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

VOLUME OF PRODUCT IN COMMERCE

125 tons

DISTRIBUTION

AL and FL

PRODUCT

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

CODE

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

RECALLING FIRM/MANUFACTURER

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

REASON

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

VOLUME OF PRODUCT IN COMMERCE

27,694,240 lbs

DISTRIBUTION

MI

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PRODUCT

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

CODE

None

RECALLING FIRM/MANUFACTURER

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

REASON

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

VOLUME OF PRODUCT IN COMMERCE

?????

DISTRIBUTION

KY

END OF ENFORCEMENT REPORT FOR AUGUST 2, 2006

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

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PRODUCT

Bulk Whole Barley, Recall # V-256-2009

CODE

No code or lot number.

RECALLING FIRM/MANUFACTURER

Mars Petcare US, Clinton, OK, by telephone on May 21, 2009. Firm initiated recall is complete.

REASON

Product may have contained prohibited materials without cautionary statement on the label.

VOLUME OF PRODUCT IN COMMERCE

208,820 pounds

DISTRIBUTION

TX

END OF ENFORCEMENT REPORT FOR AUGUST 26, 2009

###

https://www.fda.gov/Safety/Recalls/EnforcementReports/ucm180348.htm

Subject: MAD COW FEED RECALL KY VOLUME OF PRODUCT IN COMMERCE ?????

Date: August 6, 2006 at 6:19 pm PST

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

CODE None

RECALLING FIRM/MANUFACTURER Burkmann Feeds LLC, Glasgow, KY, by letter on July 14, 2006.

Firm initiated recall is ongoing. REASON Custom made feeds contain ingredient called Pro-Lak, which may contain ruminant derived meat and bone meal.

VOLUME OF PRODUCT IN COMMERCE ?????

DISTRIBUTION KY

END OF ENFORCEMENT REPORT FOR AUGUST 2, 2006

###

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

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 ruminent derived meat and bone meal..

VOLUME OF PRODUCT IN COMMERCE 10,878.06 tons

DISTRIBUTION Nationwide

END OF ENFORCEMENT REPORT FOR July 12, 2006

###

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

Subject: MAD COW FEED BAN WARNING LETTER ISSUED MAY 17, 2006

Date: June 27, 2006 at 7:42 am PST Public Health Service Food and Drug Administration

New Orleans District 297 Plus Park Blvd. Nashville, TN 37217

Telephone: 615-781-5380 Fax: 615-781-5391

May 17, 2006

WARNING LETTER NO.. 2006-NOL-06

FEDERAL EXPRESS OVERNIGHT DELIVERY

Mr. William Shirley, Jr., Owner Louisiana.DBA Riegel By-Products 2621 State Street Dallas, Texas 75204

Dear Mr. Shirley:

On February 12, 17, 21, and 22, 2006, a U.S. Food & Drug Administration (FDA) investigator inspected your rendering plant, located at 509 Fortson Street, Shreveport, Louisiana. The inspection revealed significant deviations from the requirements set forth in Title 21, Code of Federal Regulations, Part 589.2000 [21 CFR 589.2000], Animal Proteins Prohibited in Ruminant Feed. This regulation is intended to prevent the establishment and amplification of Bovine Spongiform Encephalopathy (BSE). You failed to follow the requirements of this regulation; products being manufactured and distributed by your facility are misbranded within the meaning of Section 403(a)(1) [21 USC 343(a)(1)] of the Federal Food, Drug, and Cosmetic Act (the Act).

Our investigation found you failed to provide measures, including sufficient written procedures, to prevent commingling or cross-contamination and to maintain sufficient written procedures [21 CFR 589.2000(e)] because:

You failed to use clean-out procedures or other means adequate to prevent carryover of protein derived from mammalian tissues into animal protein or feeds which may be used for ruminants. For example, your facility uses the same equipment to process mammalian and poultry tissues. However, you use only hot water to clean the cookers between processing tissues from each species. You do not clean the auger, hammer mill, grinder, and spouts after processing mammalian tissues.

You failed to maintain written procedures specifying the clean-out procedures or other means to prevent carryover of protein derived from mammalian tissues into feeds which may be used for ruminants.

As a result . the poultry meal you manufacture may contain protein derived from mammalian tissues prohibited in ruminant feed. Pursuant to 21 CFR 589.2000(e)(1)(i), any products containing or may contain protein derived from mammalian tissues must be labeled, "Do not feed to cattle or other ruminants." Since you failed to label a product which may contain protein derived from mammalian tissues with the required cautionary statement. the poultry meal is misbranded under Section 403(a)(1) [21 USC 343(a)(1)] of the Act.

This letter is not intended as an all-inclusive list of violations at your facility. As a manufacturer of materials intended for animal feed use, you are responsible for ensuring your overall operation and the products you manufacture and distribute are in compliance with the law. You should take prompt action to correct these violations, and you should establish a system whereby violations do not recur. Failure to promptly correct these violations may result in regulatory action, such as seizure and/or injunction, without further notice.

You should notify this office in writing within 15 working days of receiving this letter, outlining the specific steps you have taken to bring your firm into compliance with the law. Your response should include an explanation of each step taken to correct the violations and prevent their recurrence. If corrective action cannot be completed within 15 working days, state the reason for the delay and the date by which the corrections will be completed. Include copies of any available documentation demonstrating corrections have been made.

Your reply should be directed to Mark W. Rivero, Compliance Officer, U.S. Food and Drug Administration, 2424 Edenborn Avenue, Suite 410, Metairie, Louisiana 70001. If you have questions regarding any issue in this letter, please contact Mr. Rivero at (504) 219-8818, extension 103.

Sincerely,

/S

Carol S. Sanchez Acting District Director New Orleans District

http://www.fda.gov/foi/warning_letters/g5883d.htm

PLEASE NOTE, THE FDA URLS FOR OLD WARNING LETTERS ARE OBSOLETE AND DO NOT WORK IN MOST CASES.. I LOOKED UP THE OLD ONE ABOVE AND FOUND IT, BUT HAVE NOT DONE THAT FOR THE OTHERS TO FOLLOW. THE DATA IS VALID THOUGH!

Subject: MAD COW PROTEIN IN COMMERCE USA 2006 RECALL UPDATE

From: "Terry S. Singeltary Sr." <[log in to unmask]>

Reply-To: SAFETY <[log in to unmask]>

Date: Mon, 9 Oct 2006 14:10:37 -0500

Subject: MAD COW FEED RECALL USA SEPT 6, 2006 1961.72 TONS

IN COMMERCE AL, TN, AND WV

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

_____

snip...

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

Subject: MAD COW FEED RECALLS ENFORCEMENT REPORT FOR AUGUST 9, 2006 KY, LA, MS, AL, GA, AND TN 11,000+ TONS

Date: August 16, 2006 at 9:19 am PST RECALLS AND FIELD CORRECTIONS: VETERINARY MEDICINE - CLASS II

_____

snip...

_____

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

_____

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

###

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

###

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

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 ruminent derived meat and bone meal.

VOLUME OF PRODUCT IN COMMERCE 10,878.06 tons

DISTRIBUTION Nationwide

END OF ENFORCEMENT REPORT FOR July 12, 2006

###

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

Product Details

Product Description:

CalDensity Black Label, CalDensity White Label with HA, packaged in white plastic 5, 15, 25, 40, 60 lb pails with plastic liner and white plastic lid. Reason for Recall:

During an FDA inspection it was found that the CalDensity Black label and CalDensity White Label with HA product containers did not include the precautionary statement DO NOT FEED TO CATTLE OR OTHER RUMINANTS

Product Quantity: 50,935 lbs

Recall Number: V-209-2012

Code Information: 042009, 051009, 061209, 071509, 091009, 011510, 030310, 031610, 052610, 092410, 120110, 011211, 020111, 030911, 050111, 071111 & 090111. Classification: Class II Event Details

Event ID: 61880

Voluntary / Mandated:

Voluntary: Firm Initiated

Product Type:

Veterinary

Initial Firm Notification of Consignee or Public:

E-Mail

Status:

Terminated

Distribution Pattern:

Nationwide distribution: AL, AR, AZ, CA, CO, FL, GA, IA, ID, IL, KY, LA, MD, MI, MN, MO, MS, NC, NE, NJ, NM, NY, OH, OK, PA, SC, TX, UT, VA, WA & WV. No shipments were made to foreign countries including Canada.

Recalling Firm:

Process Managers LLC

485 Gawthrope Dr

Winchester, KY 40391-8910

United States

Recall Initiation Date:

1/6/2012

Center Classification Date:

9/7/2012

Date Terminated:

1/24/2014

https://www.accessdata.fda.gov/scripts/ires/index.cfm#tabNav_advancedSearch

Product Details

Product Description:

Regular Chicken 50# Ingredients: Corn, Wheat, Oats, Oyster shells, Medium Grit, CCC, ADS, Plant Protein Products, Animal Protein Products, Processed Grain By-Products, Roughage Products, Animal Fat procession with DHA, etc

Reason for Recall:

During an FDA sample collection, the firms 50# Regular Chicken Feed was found to contain mammalian protein. The label does not contain the warning statement.

Product Quantity:

5400lbs (50lb bags)

Recall Number:

V-137-2013

Code Information:

8/6/2012

Classification:

Class III

Event Details

Event ID:

63743

Voluntary / Mandated:

Voluntary: Firm Initiated

Product Type:

Veterinary

Initial Firm Notification of Consignee or Public:

Other

Status:

Terminated

Distribution Pattern:

Midland MI area only.

Recalling Firm:

Cohoons Elevator Inc.

802 Townsend St

Midland, MI 48640-5362

United States

Recall Initiation Date:

11/21/2012

Center Classification Date:

2/8/2013

Date Terminated:

2/12/2013

https://www.accessdata.fda.gov/scripts/ires/index.cfm#tabNav_advancedSearch

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

FDA continues to consider materials from deer and elk NOT considered at high risk for CWD to be acceptable for use in NON-RUMINANT animal feeds in accordance with current agency regulations, 21 CFR 589.2000.

Deer and elk not considered at high risk include:

(1) deer and elk from areas not declared by State officials to be endemic for CWD and/or to be CWD eradication zones; and

(2) deer and elk that were not at some time during the 60-month period immediately before the time of slaughter in a captive herd that contained a CWD-positive animal.

https://www.fda.gov/downloads/animalveterinary/guidancecomplianceenforcement/guidanceforindustry/ucm052506.pdf

2017 Section 21 C.F.R. 589.2000, Animal Proteins Prohibited in Ruminant Feed

Subject: MICHIGAN FDA PART 589 -- SUBSTANCES PROHIBITED FROM USE IN ANIMAL FOOD OR FEED VIOLATIONS OFFICIAL ACTION INDICATED OAI UPDATE BREACH APRIL 4, 2017

MICHIGAN FDA PART 589 -- SUBSTANCES PROHIBITED FROM USE IN ANIMAL FOOD OR FEEDVIOLATIONS OFFICIAL ACTION INDICATED OAI UPDATE BREACH APRIL 4, 2017

FDA BSE/Ruminant Feed Inspections Firms Inventory

11998 DET-DO MI 48846-847 OPR 4/4/2017 OAI

http://www.accessdata.fda.gov/scripts/BSEInspect/bseinspections.csv

NAI = NO ACTION INDICATED

OAI = OFFICIAL ACTION INDICATED

VAI = VOLUNTARY ACTION INDICATED

RTS = REFERRED TO STATE

OAI (Official Action Indicated) when inspectors find significant objectionable conditions or practices and believe that regulatory sanctions are warranted to address the establishment's lack of compliance with the regulation. An example of an OAI classification would be findings of manufacturing procedures insufficient to ensure that ruminant feed is not contaminated with prohibited material. Inspectors will promptly re-inspect facilities classified OAI after regulatory sanctions have been applied to determine whether

the corrective actions are adequate to address the objectionable conditions...end...TSS

TUESDAY, APRIL 18, 2017

*** EXTREME USA FDA PART 589 TSE PRION FEED LOOP HOLE STILL EXIST, AND PRICE OF POKER GOES UP ***

http://usdameatexport.blogspot.com/2017/04/extreme-usa-fda-part-589-tse-prion-feed.html

TUESDAY, JANUARY 17, 2017

FDA PART 589 -- SUBSTANCES PROHIBITED FROM USE IN ANIMAL FOOD OR FEEDVIOLATIONS OFFICIAL ACTION INDICATED OAI UPDATE 2016 to 2017 BSE TSE PRION

http://bovineprp.blogspot.com/2017/01/fda-part-589-substances-prohibited-from.html

FY 2016 Inspectional Observation Summaries

4132 21 CFR 589.2000(d)(1) Protein blenders, feed manufacturers, distributors Products that contain or may contain prohibited material fail to bear the caution statement, "Do not feed to cattle or other ruminants." Specifically, *** 2

4131 21 CFR 589.2000(c)(1)(i) Renderers Products that contain or may contain prohibited material fail to bear a label containing the caution statement, "Do not feed to cattle or other ruminants." Specifically, *** 1

https://www.fda.gov/ICECI/EnforcementActions/ucm531890.htm#Veterinary Medicine

FY 2015 Inspectional Observation Summaries

4132 21 CFR 589.2000(d)(1) Protein blenders, feed manufacturers, distributors Products that contain or may contain prohibited material fail to bear the caution statement, "Do not feed to cattle or other ruminants." Specifically, *** 2

https://www.fda.gov/ICECI/Inspections/ucm481432.htm#Veterinary Medicine

FY 2014 Inspectional Observation Summaries

4146 21 CFR 589.2000(e)(1) Written clean-out procedures Failure to maintain written clean-out procedures to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, *** 2

4131 21 CFR 589.2000(c)(1)(i) Renderers Products that contain or may contain prohibited material fail to bear a label containing the caution statement, "Do not feed to cattle or other ruminants." Specifically, *** 1

4132 21 CFR 589.2000(d)(1) Protein blenders, feed manufacturers, distributors Products that contain or may contain prohibited material fail to bear the caution statement, "Do not feed to cattle or other ruminants." Specifically, *** 1

4145 21 CFR 589.2000(e)(1) Use of clean-out procedures Failure to use clean-out procedures or other means adequate to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, *** 1

https://www.fda.gov/iceci/inspections/ucm424098.htm#Veterinary Medicine

FY 2013 Inspectional Observation Summaries

4131 21 CFR 589.2000(c)(1)(i) 5 Renderers Products that contain or may contain prohibited material fail to bear a label containing the caution statement, "Do not feed to cattle or other ruminants." Specifically, ***

4132 21 CFR 589.2000(d)(1) 5 Protein blenders, feed manufacturers, distributors Products that contain or may contain prohibited material fail to bear the caution statement, "Do not feed to cattle or other ruminants." Specifically, ***

4145 21 CFR 589.2000(e)(1) 1 Use of clean-out procedures Failure to use clean-out procedures or other means adequate to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, ***

4146 21 CFR 589.2000(e)(1) 1 Written clean-out procedures Failure to maintain written clean-out procedures to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, ***

FY 2012 Inspectional Observation Summaries

4131 21 CFR 589.2000(c)(1)(i) 5 Renderers Products that contain or may contain prohibited material fail to bear a label containing the caution statement, "Do not feed to cattle or other ruminants." Specifically, ***

4132 21 CFR 589.2000(d)(1) 4 Protein blenders, feed manufacturers, distributors Products that contain or may contain prohibited material fail to bear the caution statement, "Do not feed to cattle or other ruminants." Specifically, ***

https://www.fda.gov/ICECI/Inspections/ucm326984.htm

FY 2011 Inspectional Observation Summaries

4132 21 CFR 589.2000(d)(1) 5 Protein blenders, feed manufacturers, distributors Products that contain or may contain prohibited material fail to bear the caution statement, "Do not feed to cattle or other ruminants."Specifically, ***

4131 21 CFR 589.2000(c)(1)(i) 4 Renderers Products that contain or may contain prohibited material fail to bear a label containing the caution statement, "Do not feed to cattle or other ruminants."Specifically, ***

4146 21 CFR 589.2000(e)(1) 1 Written clean-out procedures Failure to maintain written clean-out procedures to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, ***

https://www.fda.gov/ICECI/Inspections/ucm327135.htm#vet

FY 2010 Inspectional Observation Summaries

4131 21 CFR 589.2000(c)(1)(i) 3 Renderers Products that contain or may contain prohibited material fail to bear a label containing the caution statement, "Do not feed to cattle or other ruminants." Specifically, *** 4132 21 CFR 589.2000(d)(1) 3 Protein blenders, feed manufacturers, distributors Products that contain or may contain prohibited material fail to bear the caution statement, "Do not feed to cattle or other ruminants." Specifically, ***

4146 21 CFR 589.2000(e)(1) 1 Written clean-out procedures Failure to maintain written clean-out procedures to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, ***

https://www.fda.gov/ICECI/Inspections/ucm255532.htm

FY 2009 Inspectional Observation Summaries

4132 21 CFR 589.2000(d)(1) 10 Protein blenders, feed manufacturers, distributors Products that contain or may contain prohibited material fail to bear the caution statement, "Do not feed to cattle or other ruminants." Specifically, ***

4146 21 CFR 589.2000(e)(1) 4 Written clean-out procedures Failure to maintain written clean-out procedures to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, ***

4145 21 CFR 589.2000(e)(1) 3 Use of clean-out procedures Failure to use clean-out procedures or other means adequate to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, ***

https://www.fda.gov/ICECI/Inspections/ucm255534.htm

FY 2008 Inspectional Observation Summaries

4132 21 CFR 589.2000(d)(1) 7 Protein blenders, feed manufacturers, distributors Products that contain or may contain prohibited material fail to bear the caution statement, "Do not feed to cattle or other ruminants." Specifically, ***

4145 21 CFR 589.2000(e)(1) 1 Use of clean-out procedures Failure to use clean-out procedures or other means adequate to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, *** 4146 21 CFR 589.2000(e)(1) 1 Written clean-out procedures Failure to maintain written clean-out procedures to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, ***

https://www.fda.gov/ICECI/Inspections/ucm255535.htm

FY 2007 Inspectional Observation Summaries

4132 21 CFR 589.2000(d)(1) 3 Protein blenders, feed manufacturers, distributors Products that contain or may contain prohibited material fail to bear the caution statement, "Do not feed to cattle or other ruminants." Specifically, ***

4146 21 CFR 589.2000(e)(1) 3 Written clean-out procedures Failure to maintain written clean-out procedures to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, ***

4131 21 CFR 589.2000(c)(1)(i) 2 Renderers Products that contain or may contain prohibited material fail to bear a label containing the caution statement, "Do not feed to cattle or other ruminants." Specifically, ***

4145 21 CFR 589.2000(e)(1) 1 Use of clean-out procedures Failure to use clean-out procedures or other means adequate to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, ***

https://www.fda.gov/ICECI/Inspections/ucm255536.htm

FY 2006 Inspectional Observation Summaries

4132 21 CFR 589.2000(d)(1) 6 Protein blenders, feed manufacturers, distributors Products that contain or may contain prohibited material fail to bear the caution statement, "Do not feed to cattle or other ruminants."Specifically, ***

4146 21 CFR 589.2000(e)(1) 5 Written clean-out procedures Failure to maintain written clean-out procedures to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, ***

4145 21 CFR 589.2000(e)(1) 4 Use of clean-out procedures Failure to use clean-out procedures or other means adequate to prevent carryover of protein derived from mammalian tissues to animal protein or feeds that may be used for ruminants. Specifically, ***

4131 21 CFR 589.2000(c)(1)(i) 2 Renderers Products that contain or may contain prohibited material fail to bear a label containing the caution statement, "Do not feed to cattle or other ruminants." Specifically, ***

https://www.fda.gov/ICECI/Inspections/ucm255537.htm

Sunday, September 27, 2015

*** TEXAS CONFIRMATION OF BOVINE SPONGIFORM ENCEPHALOPATHY BSE TSE PRION IN ONE SAMPLE OF SORGHUM DDGS OUT OF 168 DG SAMPLES ***

i questioned this article and it was retracted, see;

https://www.ncbi.nlm.nih.gov/pubmed/26408135

Dear Terry S. Singeltary Sr. Thank for your interest and concern about our published article entitled “Evaluation of Selected Nutrients and Contaminants in Distillers Grains from Ethanol Production in Texas”. I should apologize you and others that there were some errors and misleading statements in this article due to inappropriate terminology. The statement you were concerned about was corrected to "One sorghum DDGS out of 168 DG samples was contaminated with animal protein prohibited for use in ruminant feed and was channeled to poultry feed." We

requested the journal editor to correct some errors and the relevant statements, or to withdraw the article from the journal. Again I sincerely apologize for any confusion and inconvenience this may cause. Thanks. best wishes, Kyung-Min Kyung-Min Lee, Ph. D. Research Scientist Office of the Texas State Chemist Texas A&M AgriLife Research P.O. Box 3160, College Station, TX 77841-3160

https://bovineprp.blogspot.com/2015/09/texas-confirmation-of-bovine-spongiform.html

HOWEVER, "One sorghum DDGS out of 168 DG samples was contaminated with animal protein prohibited for use in ruminant feed and was channeled to poultry feed." COULD LEAD TO 'Bovine Spongiform Encephalopathy', IMO...terry

*** 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

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

https://www.regulations.gov/#!documentDetail;D=FDA-2003-D-0432-0011

17 years post mad cow feed ban August 1997

Monday, October 26, 2015

FDA PART 589 -- SUBSTANCES PROHIBITED FROM USE IN ANIMAL FOOD OR FEED VIOLATIONS OFFICIAL ACTION INDICATED OIA UPDATE October 2015

http://madcowusda.blogspot.com/2015/10/fda-part-589-substances-prohibited-from.html

Tuesday, December 23, 2014

FDA PART 589 -- SUBSTANCES PROHIBITED FROM USE IN ANIMAL FOOD OR FEEDVIOLATIONS OFFICIAL ACTION INDICATED OAI UPDATE DECEMBER 2014 BSE TSE PRION

http://madcowusda.blogspot.com/2014/12/fda-part-589-substances-prohibited-from.html

16 years post mad cow feed ban August 1997 2013

Sunday, December 15, 2013

FDA PART 589 -- SUBSTANCES PROHIBITED FROM USE IN ANIMAL FOOD OR FEED VIOLATIONS OFFICIAL ACTION INDICATED OIA UPDATE DECEMBER 2013 UPDATE

http://madcowusda.blogspot.com/2013/12/fda-part-589-substances-prohibited-from.html

Saturday, August 29, 2009

FOIA REQUEST FEED RECALL 2009 Product may have contained prohibited materials Bulk Whole Barley, Recall # V-256-2009

http://madcowfeed.blogspot.com/2009/08/foia-request-feed-recall-2009-product.html

Friday, September 4, 2009

FOIA REQUEST ON FEED RECALL PRODUCT 429,128 lbs. feed for

ruminant animals may have been contaminated with prohibited material Recall # V-258-2009

http://madcowfeed.blogspot.com/2009/09/foia-request-on-feed-recall-product.html

Thursday, March 19, 2009

MILLIONS AND MILLIONS OF POUNDS OF MAD COW FEED IN COMMERCE USA WITH ONGOING 12 YEARS OF DENIAL NOW, WHY IN THE WORLD DO WE TO TALK ABOUT THIS ANYMORE $$$

http://madcowfeed.blogspot.com/2009/03/millions-and-millions-of-pounds-of-mad.html

http://madcowusda.blogspot.com/2009/10/cvm-annual-report-fiscal-year-2008.html

DOSE RATE ORAL TRANSMISSION TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHY TSE PRION

THIS SHOULD BE VERY IMPORTANT TO ALL CATTLE RANCHERS, BEEF PRODUCERS, AND OR DAIRY FARMERS

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

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

SNIP...

Greetings again FDA and Mr. Pritchett et al,

MY comments and source reference of sound science on this very important issue are as follows ;

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

I kindly wish to once again submit to Docket No. FDA-2003-D-0432 (formerly 03D-0186) Use of Material from Deer and Elk in Animal Feed.

Thank you kindly for allowing me to comment again, ...and again...and again, on a topic so important, why it is 'NON-BINDING' is beyond me.

this should have been finalized and made 'BINDING' or MANDATORY OVER A DECADE AGO.

but here lay the problem, once made 'BINDING' or 'MANDATORY', it is still nothing but ink on paper.

we have had a mad cow feed ban in place since August 1997, and since then, literally 100s of millions of pounds BANNED MAD COW FEED has been sent out to commerce and fed out (see reference materials).

ENFORCEMENT OF SAID BINDING REGULATIONS HAS FAILED US TOO MANY TIMES.

so, in my opinion, any non-binding or voluntary regulations will not work, and to state further, 'BINDING' or MANDATORY regulations will not work unless enforced.

with that said, we know that Chronic Wasting Disease CWD TSE Prion easily transmits to other cervid through the oral route.

the old transmission studies of BSE TSE floored scientist once they figured out what they had, and please don't forget about those mink that were fed 95%+ dead stock downer cow, that all came down with TME. please see ;

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

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

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

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

Evidence That Transmissible Mink Encephalopathy Results from

Feeding Infected Cattle

Over the next 8-10 weeks, approximately 40% of all the adult mink on the farm died from TME.

snip...

The rancher was a "dead stock" feeder using mostly (>95%) downer or dead dairy cattle...

https://web.archive.org/web/20090506002258/http://www.bseinquiry.gov.uk/files/mb/m09/tab05.pdf

To further complicate things, we now know that science has shown that plants and vegetables can uptake the TSE Prion, and that the Scrapie agent can still be infectious from soil 16 years later. a frightening thought with the CWD running rampant now in North America (please see source reference materials below).

IF we don't do this, we have failed, and the TSE Prion agent will continue to spread, as it is doing as we speak.

I strenuously once again urge the FDA and its industry constituents, to make it MANDATORY that all ruminant feed be banned to all ruminants, and this should include all cervids, as well as non-ruminants such as cats and dogs as well, as soon as possible for the following reasons...

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

Sunday, March 20, 2016

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

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

Research Project: TRANSMISSION, DIFFERENTIATION, AND PATHOBIOLOGY OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES

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

Authors

item Greenlee, Justin item Moore, S - item Smith, Jodi - item Kunkle, Robert item West Greenlee, M -

Submitted to: American College of Veterinary Pathologists

Meeting Publication Type:

Abstract Only Publication Acceptance Date: August 12, 2015
Publication Date: N/A

Technical Abstract: The purpose of this work was to determine susceptibility of white-tailed deer (WTD) to the agent of sheep scrapie and to compare the resultant PrPSc to that of the original inoculum and chronic wasting disease (CWD).

We inoculated WTD by a natural route of exposure (concurrent oral and intranasal (IN); n=5) with a US scrapie isolate.

All scrapie-inoculated deer had evidence of PrPSc accumulation.

PrPSc was detected in lymphoid tissues at preclinical time points, and deer necropsied after 28 months post-inoculation had clinical signs, spongiform encephalopathy, and widespread distribution of PrPSc in neural and lymphoid tissues.

Western blotting (WB) revealed PrPSc with 2 distinct molecular profiles.

WB on cerebral cortex had a profile similar to the original scrapie inoculum, whereas WB of brainstem, cerebellum, or lymph nodes revealed PrPSc with a higher profile resembling CWD.

Homogenates with the 2 distinct profiles from WTD with clinical scrapie were further passaged to mice expressing cervid prion protein and intranasally to sheep and WTD. In cervidized mice, the two inocula have distinct incubation times.

Sheep inoculated intranasally with WTD derived scrapie developed disease, but only after inoculation with the inoculum that had a scrapie-like profile.

The WTD study is ongoing, but deer in both inoculation groups are positive for PrPSc by rectal mucosal biopsy.

In summary, this work demonstrates that WTD are susceptible to the agent of scrapie, two distinct molecular profiles of PrPSc are present in the tissues of affected deer, and inoculum of either profile readily passes to deer.

http://www.ars.usda.gov/research/publications/publications.htm?SEQ_NO_115=317901

http://www.ars.usda.gov/research/publications/publications.htm?SEQ_NO_115=260467

White-tailed Deer are Susceptible to Scrapie by Natural Route of Infection

Jodi D. Smith, Justin J. Greenlee, and Robert A. Kunkle; Virus and Prion Research Unit, National Animal Disease Center, USDA-ARS

Interspecies transmission studies afford the opportunity to better understand the potential host range and origins of prion diseases. Previous experiments demonstrated that white-tailed deer are susceptible to sheep-derived scrapie by intracranial inoculation. The purpose of this study was to determine susceptibility of white-tailed deer to scrapie after a natural route of exposure. Deer (n=5) were inoculated by concurrent oral (30 ml) and intranasal (1 ml) instillation of a 10% (wt/vol) brain homogenate derived from a sheep clinically affected with scrapie. Non-inoculated deer were maintained as negative controls. All deer were observed daily for clinical signs. Deer were euthanized and necropsied when neurologic disease was evident, and tissues were examined for abnormal prion protein (PrPSc) by immunohistochemistry (IHC) and western blot (WB). One animal was euthanized 15 months post-inoculation (MPI) due to an injury. At that time, examination of obex and lymphoid tissues by IHC was positive, but WB of obex and colliculus were negative. Remaining deer developed clinical signs of wasting and mental depression and were necropsied from 28 to 33 MPI. Tissues from these deer were positive for scrapie by IHC and WB. Tissues with PrPSc immunoreactivity included brain, tonsil, retropharyngeal and mesenteric lymph nodes, hemal node, Peyer's patches, and spleen.

***>This work demonstrates for the first time that white-tailed deer are susceptible to sheep scrapie by potential natural routes of inoculation. In-depth analysis of tissues will be done to determine similarities between scrapie in deer after intracranial and oral/intranasal inoculation and chronic wasting disease resulting from similar routes of inoculation.

see full text ;

http://www.usaha.org/Portals/6/Reports/2010/report-cwal-2010.pdf

PO-039: A comparison of scrapie and chronic wasting disease in white-tailed deer

Justin Greenlee, Jodi Smith, Eric Nicholson US Dept. Agriculture; Agricultural Research Service, National Animal Disease Center; Ames, IA USA

http://www.landesbioscience.com/journals/prion/03-Prion6-2-Transmission-and-strains.pdf

White-tailed deer are susceptible to the agent of sheep scrapie by intracerebral inoculation

snip...

It is unlikely that CWD will be eradicated from free-ranging cervids, and the disease is likely to continue to spread geographically [10]. However, the potential that white-tailed deer may be susceptible to sheep scrapie by a natural route presents an additional confounding factor to halting the spread of CWD. This leads to the additional speculations that

1) infected deer could serve as a reservoir to infect sheep with scrapie offering challenges to scrapie eradication efforts and

2) CWD spread need not remain geographically confined to current endemic areas, but could occur anywhere that sheep with scrapie and susceptible cervids cohabitate.

This work demonstrates for the first time that white-tailed deer are susceptible to sheep scrapie by intracerebral inoculation with a high attack rate and that the disease that results has similarities to CWD. These experiments will be repeated with a more natural route of inoculation to determine the likelihood of the potential transmission of sheep scrapie to white-tailed deer. If scrapie were to occur in white-tailed deer, results of this study indicate that it would be detected as a TSE, but may be difficult to differentiate from CWD without in-depth biochemical analysis.

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3199251/?tool=pubmed

http://chronic-wasting-disease.blogspot.com/2011/10/white-tailed-deer-are-susceptible-to.html

2012

PO-039: A comparison of scrapie and chronic wasting disease in white-tailed deer

Justin Greenlee, Jodi Smith, Eric Nicholson US Dept. Agriculture; Agricultural Research Service, National Animal Disease Center; Ames, IA USA

snip...

The results of this study suggest that there are many similarities in the manifestation of CWD and scrapie in WTD after IC inoculation including early and widespread presence of PrPSc in lymphoid tissues, clinical signs of depression and weight loss progressing to wasting, and an incubation time of 21-23 months. Moreover, western blots (WB) done on brain material from the obex region have a molecular profile similar to CWD and distinct from tissues of the cerebrum or the scrapie inoculum. However, results of microscopic and IHC examination indicate that there are differences between the lesions expected in CWD and those that occur in deer with scrapie: amyloid plaques were not noted in any sections of brain examined from these deer and the pattern of immunoreactivity by IHC was diffuse rather than plaque-like.

*** After a natural route of exposure, 100% of WTD were susceptible to scrapie.

Deer developed clinical signs of wasting and mental depression and were necropsied from 28 to 33 months PI. Tissues from these deer were positive for PrPSc by IHC and WB. Similar to IC inoculated deer, samples from these deer exhibited two different molecular profiles: samples from obex resembled CWD whereas those from cerebrum were similar to the original scrapie inoculum. On further examination by WB using a panel of antibodies, the tissues from deer with scrapie exhibit properties differing from tissues either from sheep with scrapie or WTD with CWD. Samples from WTD with CWD or sheep with scrapie are strongly immunoreactive when probed with mAb P4, however, samples from WTD with scrapie are only weakly immunoreactive. In contrast, when probed with mAb's 6H4 or SAF 84, samples from sheep with scrapie and WTD with CWD are weakly immunoreactive and samples from WTD with scrapie are strongly positive. This work demonstrates that WTD are highly susceptible to sheep scrapie, but on first passage, scrapie in WTD is differentiable from CWD.

http://www.landesbioscience.com/journals/prion/03-Prion6-2-Transmission-and-strains.pdf

2011

*** After a natural route of exposure, 100% of white-tailed deer were susceptible to scrapie.

http://www.usaha.org/Portals/6/Reports/2011/report-cwal-2011.pdf

White-tailed Deer are Susceptible to Scrapie by Natural Route of Infection

Jodi D. Smith, Justin J. Greenlee, and Robert A. Kunkle; Virus and Prion Research Unit, National Animal Disease Center, USDA-ARS

Interspecies transmission studies afford the opportunity to better understand the potential host range and origins of prion diseases. Previous experiments demonstrated that white-tailed deer are susceptible to sheep-derived scrapie by intracranial inoculation. The purpose of this study was to determine susceptibility of white-tailed deer to scrapie after a natural route of exposure. Deer (n=5) were inoculated by concurrent oral (30 ml) and intranasal (1 ml) instillation of a 10% (wt/vol) brain homogenate derived from a sheep clinically affected with scrapie. Non-inoculated deer were maintained as negative controls. All deer were observed daily for clinical signs. Deer were euthanized and necropsied when neurologic disease was evident, and tissues were examined for abnormal prion protein (PrPSc) by immunohistochemistry (IHC) and western blot (WB). One animal was euthanized 15 months post-inoculation (MPI) due to an injury. At that time, examination of obex and lymphoid tissues by IHC was positive, but WB of obex and colliculus were negative. Remaining deer developed clinical signs of wasting and mental depression and were necropsied from 28 to 33 MPI. Tissues from these deer were positive for scrapie by IHC and WB. Tissues with PrPSc immunoreactivity included brain, tonsil, retropharyngeal and mesenteric lymph nodes, hemal node, Peyer's patches, and spleen. This work demonstrates for the first time that white-tailed deer are susceptible to sheep scrapie by potential natural routes of inoculation. In-depth analysis of tissues will be done to determine similarities between scrapie in deer after intracranial and oral/intranasal inoculation and chronic wasting disease resulting from similar routes of inoculation.

see full text ;

http://www.usaha.org/Portals/6/Reports/2010/report-cwal-2010.pdf

Oral transmission and early lymphoid tropism of chronic wasting disease PrPres in mule deer fawns (Odocoileus hemionus)

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Department of Pathology, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, CO 80523- 1671, USA1 Department of Veterinary Sciences, University of Wyoming, 1174 Snowy Range Road, University of Wyoming, Laramie, WY 82070, USA 2 Colorado Division of Wildlife, Wildlife Research Center, 317 West Prospect Road, Fort Collins, CO 80526-2097, USA3 Colorado State University Veterinary Diagnostic Laboratory, 300 West Drake Road, Fort Collins, CO 80523-1671, USA4 Animal Disease Research Unit, Agricultural Research Service, US Department of Agriculture, 337 Bustad Hall, Washington State University, Pullman, WA 99164-7030, USA5

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Mule deer fawns (Odocoileus hemionus) were inoculated orally with a brain homogenate prepared from mule deer with naturally occurring chronic wasting disease (CWD), a prion-induced transmissible spongiform encephalopathy.

Fawns were necropsied and examined for PrPres, the abnormal prion

protein isoform, at 10, 42, 53, 77, 78 and 80 days post-inoculation (p.i.) using an immunohistochemistry assay modified to enhance sensitivity.

PrPres was detected in alimentary-tract-associated lymphoid tissues (one or more of the following: retropharyngeal lymph node, tonsil, Peyer's patch and ileocaecal lymph node) as early as 42 days p.i. and in all fawns examined thereafter (53 to 80 days p.i.).

No PrPres staining was detected in lymphoid tissue of three control fawns receiving a control brain inoculum, nor was PrPres detectable in neural tissue of any fawn.

PrPres-specific staining was markedly enhanced by sequential tissue treatment with formic acid, proteinase K and hydrated autoclaving prior to immunohistochemical staining with monoclonal antibody F89/160.1.5.

These results indicate that CWD PrP res can be detected in lymphoid tissues draining the alimentary tract within a few weeks after oral exposure to infectious prions and may reflect the initial pathway of CWD infection in deer.

The rapid infection of deer fawns following exposure by the most plausible natural route is consistent with the efficient horizontal transmission of CWD in nature and enables accelerated studies of transmission and pathogenesis in the native species.

snip...

These results indicate that mule deer fawns develop detectable PrPres after oral exposure to an inoculum containing CWD prions.

In the earliest post-exposure period, CWD PrPres was traced to the lymphoid tissues draining the oral and intestinal mucosa (i.e. the retropharyngeal lymph nodes, tonsil, ileal Peyer's patches and ileocaecal lymph nodes), which probably received the highest initial exposure to the inoculum. Hadlow et al. (1982) demonstrated scrapie agent in the tonsil, retropharyngeal and mesenteric lymph nodes, ileum and spleen in a 10-month-old naturally infected lamb by mouse bioassay.

Eight of nine sheep had infectivity in the retropharyngeal lymph node.

He concluded that the tissue distribution suggested primary infection via the gastrointestinal tract.

The tissue distribution of PrPres in the early stages of infection in the fawns is strikingly similar to that seen in naturally infected sheep with scrapie.

These findings support oral exposure as a natural route of CWD infection in deer and support oral inoculation as a reasonable exposure route for experimental studies of CWD.

snip...

http://vir.sgmjournals.org/cgi/content/full/80/10/2757

Journal of Wildlife Diseases, 42(3), 2006, pp. 640–645 # Wildlife Disease Association 2006

Oral Transmission of Chronic Wasting Disease in Captive Shira's Moose

Terry J. Kreeger,1,3 D. L. Montgomery,2 Jean E. Jewell,2 Will Schultz,1 and Elizabeth S. Williams2 1 Wyoming Game and Fish Department, 2362 Highway 34, Wheatland, Wyoming 82201, USA; 2 Department of Veterinary Sciences, University of Wyoming, Laramie, Wyoming 82071, USA; 3 Corresponding author (email: tkreeger@wildblue.net)

ABSTRACT: Three captive Shira's moose (Alces alces shirasi) were orally inoculated with a single dose (5 g) of whole-brain homogenate prepared from chronic wasting disease (CWD)– affected mule deer (Odocoileus hemionus).

All moose died of causes thought to be other than CWD.

Histologic examination of one female moose dying 465 days postinoculation revealed spongiform change in the neuropil, typical of transmissible spongiform encephalopathy.

Immunohistochemistry staining for the proteinase-resistant isoform of the prion protein was observed in multiple lymphoid and nervous tissues.

Western blot and enzyme-linked immunosorbent assays provided additional confirmation of CWD.

***>These results represent the first report of experimental CWD in moose.

Key words: Alces alces shirasi, chronic wasting disease, enzyme-linked immunosorbent assay, immunohistochemistry, moose, oral inoculation, prion, PrPCWD.

http://www.jwildlifedis.org/doi/pdf/10.7589/0090-3558-42.3.640

Experimental Oral Transmission of Chronic Wasting Disease to Reindeer (Rangifer tarandus tarandus)

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1 National and OIE Reference Laboratory for Scrapie and CWD, Canadian Food Inspection Agency, Ottawa Laboratory – Fallowfield, Ottawa, Ontario, Canada,

2 Departments of Pathology and Medicine, University of California, San Diego, La Jolla, California, United States of America, 3 Department of Pathology, Microbiology and Immunology, University of California, Davis, California, United States of America, 4 Animal Disease Research Unit, Agricultural Research Service, United States Department of Agriculture, Pullman, Washington, United States of America, 5 Veterinary Diagnostic Laboratory, Colorado State University, Fort Collins, Colorado, United States of America

Abstract

Chronic wasting disease (CWD), a transmissible spongiform encephalopathy of cervids, remains prevalent in North American elk, white-tailed deer and mule deer. A natural case of CWD in reindeer (Rangifer tarandus tarandus) has not been reported despite potential habitat overlap with CWD-infected deer or elk herds. This study investigates the experimental transmission of CWD from elk or white-tailed deer to reindeer by the oral route of inoculation. Ante-mortem testing of the three reindeer exposed to CWD from white-tailed deer identified the accumulation of pathological PrP (PrPCWD) in the recto-anal mucosa associated lymphoid tissue (RAMALT) of two reindeer at 13.4 months post-inoculation. Terminal CWD occurred in the two RAMALT-positive reindeer at 18.5 and 20 months post-inoculation while one other reindeer in the white-tailed deer CWD inoculum group and none of the 3 reindeer exposed to elk CWD developed disease. Tissue distribution analysis of PrPCWD in CWD-affected reindeer revealed widespread deposition in central and peripheral nervous systems, lymphoreticular tissues, the gastrointestinal tract, neuroendocrine tissues and cardiac muscle. Analysis of prion protein gene (PRNP) sequences in the 6 reindeer identified polymorphisms at residues 2 (V/M), 129 (G/S), 138 (S/N) and 169 (V/M).

>***These findings demonstrate that (i) a sub-population of reindeer are susceptible to CWD by oral inoculation implicating the potential for transmission to other Rangifer species, and (ii) certain reindeer PRNP polymorphisms may be protective against CWD infection.

This is the first evidence of CWD transmission to the sub-species Rangifer tarandus tarandus, implicating the potential for transmission to others in this genus. Current diagnostic tests, including antemortem RAMALT testing, appear capable of detecting CWD in Rangifer species and increased surveillance would be required to determine if natural transmission has indeed occurred. Additional studies are ongoing to chart the distribution of infectivity during the course of disease and determine the influence of PRNP polymorphisms in disease susceptibility.

http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0039055?utm_source=feedburner&utm_medium=feed&utm_campaign=Feed%3A+plosone%2FPLoSONE+(PLoS+ONE+Alerts%3A+New+Articles)#

Saturday, January 31, 2015

European red deer (Cervus elaphus elaphus) are susceptible to Bovine Spongiform Encephalopathy BSE by Oral Alimentary route

http://transmissiblespongiformencephalopathy.blogspot.com/2015/01/european-red-deer-cervus-elaphus.html

I strenuously once again urge the FDA and its industry constituents, to make it MANDATORY that all ruminant feed be banned to all ruminants, and this should include all cervids as soon as possible for the following reasons...

======

In the USA, under the Food and Drug Administrations 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.

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31 Jan 2015 at 20:14 GMT

*** Ruminant feed ban for cervids in the United States? ***

31 Jan 2015 at 20:14 GMT

see Singeltary comment ;

http://www.plosone.org/annotation/listThread.action?root=85351

EVEN MAFF UK SAYS THEY HAVE A HIGHER RISK FACTOR OF CWD FROM USA DUE TO FEED ;

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...

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

SEE THE DRASTIC REDUCTION OF CONFIRMED BSE CASES IN THE UK ONCE THE FEED BAN TOOK HOLD FROM THE TOP YEAR DOWN TO THE FIRST ZERO YEAR ;

1992 36680 SLAUGHTERED SUSPECTS IN WHICH BSE CONFIRMED

2013 0 0 0 0 0 0 0 0

http://webarchive.nationalarchives.gov.uk/20130402151656/http://www.defra.gov.uk/ahvla-en/files/pub-tse-stats-gboverview.pdf

http://webarchive.nationalarchives.gov.uk/20130402151656/http://archive.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/bse/docume

http://webarchive.nationalarchives.gov.uk/20130402151656/http://archive.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/bse/controls eradication/feedban-bornafterban.htm

http://webarchive.nationalarchives.gov.uk/20130402151656/http://www.defra.gov.uk/ahvla-en/disease-control/notifiable/bse/

http://webarchive.nationalarchives.gov.uk/20130402151656/http://www.defra.gov.uk/ahvla-en/science/tse/surveillance-stats/

Singeltary previous submission to DOCKET-- 03D-0186 -- FDA Issues Draft Guidance on Use of Material From Deer and Elk in Animal Feed; Availability

DOCKET-- 03D-0186 -- FDA Issues Draft Guidance on Use of Material From Deer and Elk in Animal Feed; Availability Fri, 16 May 2003 11:47:37 0500 EMC 1 Terry S. Singeltary Sr. Vol #: 1

Date: Fri, 16 May 2003 11:47:37 0500 EMC 1 Terry S. Singeltary Sr. Vol #: 1

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

http://www.fda.gov/ohrms/dockets/dailys/03/oct03/100203/100203.htm

http://madcowfeed.blogspot.com/2008/07/docket-03d-0186-fda-issues-draft.html

MONDAY, JANUARY 09, 2017

Oral Transmission of L-Type Bovine Spongiform Encephalopathy Agent among Cattle

CDC Volume 23, Number 2—February 2017

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

Volume 23, Number 2—February 2017

https://wwwnc.cdc.gov/eid/article/23/2/16-1416_article

http://bse-atypical.blogspot.com/2017/01/oral-transmission-of-l-type-bovine.html

***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.

P.86: Estimating the risk of transmission of BSE and scrapie to ruminants and humans by protein misfolding cyclic amplification

Morikazu Imamura, Naoko Tabeta, Yoshifumi Iwamaru, and Yuichi Murayama

National Institute of Animal Health; Tsukuba, Japan

To assess the risk of the transmission of ruminant prions to ruminants and humans at the molecular level, we investigated the ability of abnormal prion protein (PrPSc) of typical and atypical BSEs (L-type and H-type) and typical scrapie to convert normal prion protein (PrPC) from bovine, ovine, and human to proteinase K-resistant PrPSc-like form (PrPres) using serial protein misfolding cyclic amplification (PMCA).

Six rounds of serial PMCA was performed using 10% brain homogenates from transgenic mice expressing bovine, ovine or human PrPC in combination with PrPSc seed from typical and atypical BSE- or typical scrapie-infected brain homogenates from native host species. In the conventional PMCA, the conversion of PrPC to PrPres was observed only when the species of PrPC source and PrPSc seed matched. However, in the PMCA with supplements (digitonin, synthetic polyA and heparin), both bovine and ovine PrPC were converted by PrPSc from all tested prion strains. On the other hand, human PrPC was converted by PrPSc from typical and H-type BSE in this PMCA condition.

Although these results were not compatible with the previous reports describing the lack of transmissibility of H-type BSE to ovine and human transgenic mice, 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.

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

P.170: Potential detection of oral transmission of H type atypical BSE in cattle using in vitro conversion

***P.170: Potential detection of oral transmission of H type atypical BSE in cattle using in vitro conversion

Sandor Dudas, John G Gray, Renee Clark, and Stefanie Czub
Canadian Food Inspection Agency; Lethbridge, AB Canada

Keywords: Atypical BSE, oral transmission, RT-QuIC

The detection of bovine spongiform encephalopathy (BSE) has had a significant negative impact on the cattle industry worldwide. In response, governments took actions to prevent transmission and additional threats to animal health and food safety. While these measures seem to be effective for controlling classical BSE, the more recently discovered atypical BSE has presented a new challenge. To generate data for risk assessment and control measures, we have challenged cattle orally with atypical BSE to determine transmissibility and mis-folded prion (PrPSc) tissue distribution. Upon presentation of clinical symptoms, animals were euthanized and tested for characteristic histopathological changes as well as PrPSc deposition.

The H-type challenged animal displayed vacuolation exclusively in rostral brain areas but the L-type challenged animal showed no evidence thereof. To our surprise, neither of the animals euthanized, which were displaying clinical signs indicative of BSE, showed conclusive mis-folded prion accumulation in the brain or gut using standard molecular or immunohistochemical assays. To confirm presence or absence of prion infectivity, we employed an optimized real-time quaking induced conversion (RT-QuIC) assay developed at the Rocky Mountain Laboratory, Hamilton, USA.

Detection of PrPSc was unsuccessful for brain samples tests from the orally inoculated L type animal using the RT-QuIC. It is possible that these negative results were related to the tissue sampling locations or that type specific optimization is needed to detect PrPSc in this animal. We were however able to consistently detect the presence of mis-folded prions in the brain of the H-type inoculated animal. Considering the negative and inconclusive results with other PrPSc detection methods, positive results using the optimized RT-QuIC suggests the method is extremely sensitive for H-type BSE detection. This may be evidence of the first successful oral transmission of H type atypical BSE in cattle and additional investigation of samples from these animals are ongoing.

http://www.tandfonline.com/doi/pdf/10.4161/pri.29370

http://transmissiblespongiformencephalopathy.blogspot.com/2014/06/prion-2014-typical-and-atypical-bse-and.html

http://bse-atypical.blogspot.com/2016/05/a-comparison-of-classical-and-h-type.html

TEXAS OFFICIALS DEAD WRONG ON AMOUNT OF INFECTIVITY TO CAUSE A TSE PRION DISEASE ;

"FDA has determined that each animal could have consumed, at most and in total, five-and-one-half grams – approximately a quarter ounce — of prohibited material. These animals weigh approximately 600 pounds."

5.5 GRAMS OF INFECTIOUS PROHIBITED MAD COW FEED FOR EACH OF THE 1,222 ANIMALS (5.5 GRAMS X 1,222 ANIMALS) IS ENOUGH INFECTIOUS MAD COW FEED TO KILL A SMALL HERD OF COWS...TSS

U.S. Food and Drug Administration FDA News | Today the Food and Drug Administ…U.S. Food and Drug Administration FDA News

Today the Food and Drug Administration announced the results of tests taken on feed used at a Texas feedlot that was suspected of containing meat and bone meal from other domestic cattle — a violation of FDA's 1997 prohibition on using ruminant material in feed for other ruminants. Results indicate that a very low level of prohibited material was found in the feed fed to cattle.

FDA has determined that each animal could have consumed, at most and in total, five-and-one-half grams – approximately a quarter ounce — of prohibited material. These animals weigh approximately 600 pounds.

It is important to note that the prohibited material was domestic in origin (therefore not likely to contain infected material because there is no evidence of BSE in U.S. cattle), fed at a very low level, and fed only once. The potential risk of BSE to such cattle is therefore exceedingly low, even if the feed were contaminated.

According to Dr. Bernard Schwetz, FDA's Acting Principal Deputy Commissioner, "The challenge to regulators and industry is to keep this disease out of the United States. One important defense is to prohibit the use of any ruminant animal materials in feed for other ruminant animals. Combined with other steps, like U.S. Department of Agriculture's (USDA) ban on the importation of live ruminant animals from affected countries, these steps represent a series of protections, to keep American cattle free of BSE."

Despite this negligible risk, Purina Mills, Inc., is nonetheless announcing that it is voluntarily purchasing all 1,222 of the animals held in Texas and mistakenly fed the animal feed containing the prohibited material. Therefore, meat from those animals will not enter the human food supply. FDA believes any cattle that did not consume feed containing the prohibited material are unaffected by this incident, and should be handled in the beef supply clearance process as usual.

FDA believes that Purina Mills has behaved responsibly by first reporting the human error that resulted in the misformulation of the

animal feed supplement and then by working closely with State and Federal authorities.

This episode indicates that the multi-layered safeguard system put into place is essential for protecting the food supply and that continued vigilance needs to be taken, by all concerned, to ensure these rules are followed routinely.

FDA will continue working with USDA as well as State and local officials to ensure that companies and individuals comply with all laws and regulations designed to protect the U.S. food supply.

http://www.usmef.org/news-statistics/press-releases/us-food-and-drug-administration-fda-news-today-the-food-and-drug-administ-13375/

FOR IMMEDIATE RELEASE P01-05 January 30, 2001

Print Media: 301-827-6242 Consumer Inquiries: 888-INFO-FDA

FDA ANNOUNCES TEST RESULTS FROM TEXAS FEED LOT

Today the Food and Drug Administration announced the results of tests taken on feed used at a Texas feedlot that was suspected of containing meat and bone meal from other domestic cattle -- a violation of FDA's 1997 prohibition on using ruminant material in feed for other ruminants. Results indicate that a very low level of prohibited material was found in the feed fed to cattle.

FDA has determined that each animal could have consumed, at most and in total, five-and-one-half grams - approximately a quarter ounce -- of prohibited material. These animals weigh approximately 600 pounds.

It is important to note that the prohibited material was domestic in origin (therefore not likely to contain infected material because there is no evidence of BSE in U.S. cattle), fed at a very low level, and fed only once. The potential risk of BSE to such cattle is therefore exceedingly low, even if the feed were contaminated.

According to Dr. Bernard Schwetz, FDA's Acting Principal Deputy Commissioner, "The challenge to regulators and industry is to keep this disease out of the United States. One important defense is to prohibit the use of any ruminant animal materials in feed for other ruminant animals. Combined with other steps, like U.S. Department of Agriculture's (USDA) ban on the importation of live ruminant animals from affected countries, these steps represent a series of protections, to keep American cattle free of BSE."

Despite this negligible risk, Purina Mills, Inc., is nonetheless announcing that it is voluntarily purchasing all 1,222 of the animals held in Texas and mistakenly fed the animal feed containing the prohibited material. Therefore, meat from those animals will not enter the human food supply. FDA believes any cattle that did not consume feed containing the prohibited material are unaffected by this incident, and should be handled in the beef supply clearance process as usual.

FDA believes that Purina Mills has behaved responsibly by first reporting the human error that resulted in the misformulation of the animal feed supplement and then by working closely with State and Federal authorities.

This episode indicates that the multi-layered safeguard system put into place is essential for protecting the food supply and that continued vigilance needs to be taken, by all concerned, to ensure these rules are followed routinely.

FDA will continue working with USDA as well as State and local officials to ensure that companies and individuals comply with all laws and regulations designed to protect the U.S. food supply.

http://www.fda.gov/bbs/topics/news/2001/new00752.html

PRION 2009 CONGRESS BOOK OF ABSTRACTS

O.4.3

Spread of BSE prions in cynomolgus monkeys (Macaca fascicularis) after oral transmission

Edgar Holznagel1, Walter Schulz-Schaeffer2, Barbara Yutzy1, Gerhard Hunsmann3, Johannes Loewer1 1Paul-Ehrlich-Institut, Federal Institute for Sera and Vaccines, Germany; 2Department of Neuropathology, Georg-August University, Göttingen, Germany, 3Department of Virology and Immunology, German Primate Centre, Göttingen, Germany

Background: BSE-infected cynomolgus monkeys represent a relevant animal model to study the pathogenesis of variant Creutzfeldt-Jacob disease (vCJD).

Objectives: To study the spread of BSE prions during the asymptomatic phase of infection in a simian animal model.

Methods: Orally BSE-dosed macaques (n=10) were sacrificed at defined time points during the incubation period and 7 orally BSE-dosed macaques were sacrificed after the onset of clinical signs. Neuronal and non-neuronal tissues were tested for the presence of proteinase-K-resistant prion protein (PrPres) by western immunoblot and by paraffin-embedded tissue (PET) blot technique.

Results: In clinically diseased macaques (5 years p.i. + 6 mo.), PrPres deposits were widely spread in neuronal tissues (including the peripheral sympathetic and parasympathetic nervous system) and in

lymphoid tissues including tonsils. In asymptomatic disease carriers, PrPres deposits could be detected in intestinal lymph nodes as early as 1 year p.i., but CNS tissues were negative until 3 – 4 years p.i. Lumbal/sacral segments of the spinal cord and medulla oblongata were PrPres positive as early as 4.1 years p.i., whereas sympathetic trunk and all thoracic/cervical segments of the spinal cord were still negative for PrPres. However, tonsil samples were negative in all asymptomatic cases.

Discussion: There is evidence for an early spread of BSE to the CNS via autonomic fibres of the splanchnic and vagus nerves indicating that trans-synaptical spread may be a time-limiting factor for neuroinvasion. Tonsils were predominantly negative during the main part of the incubation period indicating that epidemiological vCJD screening results based on the detection of PrPres in tonsil biopsies may mostly tend to underestimate the prevalence of vCJD among humans.

http://www.prion2009.com/sites/default/files/Prion2009_Book_of_Abstracts.pdf

P04.27

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

Holznagel, E1; Yutzy, B1; Deslys, J-P2; Lasmézas, C2; Pocchiari, M3; Ingrosso, L3; Bierke, P4; Schulz-Schaeffer, W5; Motzkus, D6; Hunsmann, G6; Löwer, J1 1Paul-Ehrlich-Institut, Germany; 2Commissariat à l´Energie Atomique, France; 3Instituto Superiore di Sanità, 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 v CJD as fast as intracerebrally inoculated animals.

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

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

Simian vCJD can be easily triggered in cynomolgus monkeys on the oral route using less than 5 g BSE brain homogenate.

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

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 Lasmézas, Emmanuel Comoy, Stephen Hawkins, Christian Herzog, Franck Mouthon, Timm Konold, Frédéric Auvré, Evelyne Correia, Nathalie Lescoutra-Etchegaray, Nicole Salès, 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 0·1 mg 0·01 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.8 *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 intraperitoneal.

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

Calves were challenged by mouth with homogenised brain from confirmed cases of BSE. Some received 300g (3 doses of 100g), some 100g, 10g or 1g. They were then left to develop BSE, but were not subjected to the normal stresses that they might have encountered in a dairy herd. Animals in all four groups developed BSE. There has been a considerable spread of incubation period in some of the groups, but it appears as if those in the 1 and 10g challenge groups most closely fit the picture of incubation periods seen in the epidemic. Experiments in progress indicate that oral infection can occur in some animals with doses as low as 0.01g and 0.001g. .....

http://www.defra.gov.uk/animalh/bse/science-research/pathog.html#dose

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.

https://web.archive.org/web/20161031085054/http://web.archive.org/web/20061003022720/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.

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

2009 UPDATE ON ALABAMA AND TEXAS MAD COWS 2005 and 2006

http://bse-atypical.blogspot.com/2006/08/bse-atypical-texas-and-alabama-update.html

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

http://madcowtesting.blogspot.com/

let's take a closer look at this new prionpathy or prionopathy, and then let's look at the g-h-BSEalabama mad cow.

This new prionopathy in humans? the genetic makeup is IDENTICAL to the g-h-BSEalabama mad cow, the only _documented_ mad cow in the world to date like this, ...wait, it get's better. this new prionpathy is killing young and old humans, with LONG DURATION from onset of symptoms to death, and the symptoms are very similar to nvCJD victims, OH, and the plaques are very similar in some cases too, bbbut, it's not related to the g-h-BSEalabama cow, WAIT NOW, it gets even better, the new human prionpathy that they claim is a genetic TSE, has no relation to any gene mutation in that family. daaa, ya think it could be related to that mad cow with the same genetic make-up ??? there were literally tons and tons of banned mad cow protein in Alabama in commerce, and none of it transmitted to cows, and the cows to humans there from ??? r i g h t $$$

ALABAMA MAD COW g-h-BSEalabama

In this study, we identified a novel mutation in the bovine prion protein gene (Prnp), called E211K, of a confirmed BSE positive cow from Alabama, United States of America. This mutation is identical to the E200K pathogenic mutation found in humans with a genetic form of CJD. This finding represents the first report of a confirmed case of BSE with a potential pathogenic mutation within the bovine Prnp gene. We hypothesize that the bovine Prnp E211K mutation most likely has caused BSE in "the approximately 10-year-old cow" carrying the E221K mutation.

http://www.plospathogens.org/article/info%3Adoi%2F10.1371%2Fjournal.ppat.1000156

http://www.plospathogens.org/article/fetchObjectAttachment.action?uri=info%3Adoi%2F10.1371%2Fjournal.ppat.1000156&representation=PDF

her healthy calf also carried the mutation (J. A. Richt and S. M. Hall PLoS Pathog. 4, e1000156; 2008).

This raises the possibility that the disease could occasionally be genetic in origin. Indeed, the report of the UK BSE Inquiry in 2000 suggested that the UK epidemic had most likely originated from such a mutation and argued against the scrapierelated assumption. Such rare potential pathogenic PRNP mutations could occur in countries at present considered to be free of BSE, such as Australia and New Zealand. So it is important to maintain strict surveillance for BSE in cattle, with rigorous enforcement of the ruminant feed ban (many countries still feed ruminant proteins to pigs). Removal of specified risk material, such as brain and spinal cord, from cattle at slaughter prevents infected material from entering the human food chain. Routine genetic screening of cattle for PRNP mutations, which is now available, could provide additional data on the risk to the public. Because the point mutation identified in the Alabama animals is identical to that responsible for the commonest type of familial (genetic) CJD in humans, it is possible that the resulting infective prion protein might cross the bovine–human species barrier more easily. Patients with vCJD continue to be identified. The fact that this is happening less often should not lead to relaxation of the controls necessary to prevent future outbreaks.

Malcolm A. Ferguson-Smith Cambridge University Department of Veterinary Medicine, Madingley Road, Cambridge CB3 0ES, UK e-mail: maf12@cam.ac.uk Jürgen A. Richt College of Veterinary Medicine, Kansas State University, K224B Mosier Hall, Manhattan, Kansas 66506-5601, USA

NATURE|Vol 457|26 February 2009

http://www.nature.com/nature/journal/v457/n7233/full/4571079b.html

THURSDAY, JULY 20, 2017

USDA OIE Alabama Atypical L-type BASE Bovine Spongiform Encephalopathy BSE animal feeds for ruminants rule, 21 CFR 589.200

https://www.aphis.usda.gov/aphis/newsroom/stakeholder-info/sa_by_date/sa-2017/sa-07/bse-alabama

http://agi.alabama.gov/s/press-release's/atypical-bse-in-alabama-cow

http://bovineprp.blogspot.com/2017/07/usda-oie-alabama-atypical-l-type-base.html

THURSDAY, JULY 20, 2017

USDA OIE Alabama Atypical L-type BASE Bovine Spongiform Encephalopathy BSE animal feeds for ruminants rule, 21 CFR 589.200

http://bovineprp.blogspot.com/2017/07/usda-oie-alabama-atypical-l-type-base.html

Thursday, June 23, 2011

Experimental H-type bovine spongiform encephalopathy characterized by plaques and glial- and stellate-type prion protein deposits

http://transmissiblespongiformencephalopathy.blogspot.com/2011/06/experimental-h-type-bovine-spongiform.html

Saturday, July 23, 2011

CATTLE HEADS WITH TONSILS, BEEF TONGUES, SPINAL CORD, SPECIFIED RISK MATERIALS (SRM's) AND PRIONS, AKA MAD COW DISEASE

http://transmissiblespongiformencephalopathy.blogspot.com/2011/07/cattle-heads-with-tonsils-beef-tongues.html

Saturday, November 6, 2010

TAFS1 Position Paper on Position Paper on Relaxation of the Feed Ban in the EU Berne, 2010 TAFS

INTERNATIONAL FORUM FOR TRANSMISSIBLE ANIMAL DISEASES AND FOOD SAFETY a non-profit Swiss Foundation

http://madcowfeed.blogspot.com/2010/11/tafs1-position-paper-on-position-paper.html

http://madcowusda.blogspot.com/2013/12/in-belly-of-beast.html

USDA BSE TSE PRION SURVEILLANCE AND TESTING

Office of Inspector General Semiannual Report to Congress FY 2007 - 2nd Half

Two Texas Companies Sentenced and Fined for Misbranding Meat Products In April 2007, two closely held and related Texas companies pled guilty in Federal court and were sentenced to 12 months of probation and ordered to pay $10,250 in fines for misbranding meat products. One of the companies sold adulterated meat products to a retail store in New Mexico. Additionally, portions of the invoices failed to properly and consistently identify the meat products as being from cattle more than 30 months old at time of slaughter. This information is required to be disclosed because of bovine spongiform encephalopathy (BSE, or "mad cow disease") concerns. No adulterated meat reached consumers.

http://www.usda.gov/oig/webdocs/sarc071212.pdf

THE USDA JUNE 2004 ENHANCED BSE SURVEILLANCE PROGRAM WAS TERRIBLY FLAWED;

CDC DR. PAUL BROWN TSE EXPERT COMMENTS 2006

In an article today for United Press International, science reporter Steve Mitchell writes:

Analysis: What that mad cow means

By STEVE MITCHELL UPI Senior Medical Correspondent

WASHINGTON, March 15 (UPI) -- The U.S. Department of Agriculture was quick to assure the public earlier this week that the third case of mad cow disease did not pose a risk to them, but what federal officials have not acknowledged is that this latest case indicates the deadly disease has been circulating in U.S. herds for at least a decade.

The second case, which was detected last year in a Texas cow and which USDA officials were reluctant to verify, was approximately 12 years old.

These two cases (the latest was detected in an Alabama cow) present a picture of the disease having been here for 10 years or so, since it is thought that cows usually contract the disease from contaminated feed they consume as calves. The concern is that humans can contract a fatal, incurable, brain-wasting illness from consuming beef products contaminated with the mad cow pathogen.

"The fact the Texas cow showed up fairly clearly implied the existence of other undetected cases," Dr. Paul Brown, former medical director of the National Institutes of Health's Laboratory for Central Nervous System Studies and an expert on mad cow-like diseases, told United Press International. "The question was, 'How many?' and we still can't answer that."

Brown, who is preparing a scientific paper based on the latest two mad cow cases to estimate the maximum number of infected cows that occurred in the United States, said he has "absolutely no confidence in USDA tests before one year ago" because of the agency's reluctance to retest the Texas cow that initially tested positive.

USDA officials finally retested the cow and confirmed it was infected seven months later, but only at the insistence of the agency's inspector general.

"Everything they did on the Texas cow makes everything they did before 2005 suspect," Brown said.

Despite this, Brown said the U.S. prevalence of mad cow, formally known as bovine spongiform encephalopathy, or BSE, did not significantly threaten human or cattle health.

"Overall, my view is BSE is highly unlikely to pose any important risk either in cattle feed or human feed," he said.

However, Jean Halloran of Consumers Union in Yonkers, N.Y., said consumers should be troubled by the USDA's secrecy and its apparent plan to dramatically cut back the number of mad cow tests it conducts.

"Consumers should be very concerned about how little we know about the USDA's surveillance program and the failure of the USDA to reveal really important details," Halloran told UPI. "Consumers have to be really concerned if they're going to cut back the program," she added.

Last year the USDA tested more than 300,000 animals for the disease, but it has proposed, even in light of a third case, scaling back the program to 40,000 tests annually.

"They seem to be, in terms of actions and policies, taking a lot more seriously the concerns of the cattle industry than the concerns of consumers," Halloran said. "It's really hard to know what it takes to get this administration to take action to protect the public."

The USDA has insisted that the safeguards of a ban on incorporating cow tissue into cattle feed (which is thought to spread the disease) and removal of the most infectious parts of cows, such as the brain and spinal cord, protect consumers. But the agency glosses over the fact that both of these systems have been revealed to be inadequately implemented.

The feed ban, which is enforced by the Food and Drug Administration, has been criticized by the Government Accountability Office in two reports, the most recent coming just last year. The GAO said the FDA's enforcement of the ban continues to have weaknesses that "undermine the nation's firewall against BSE."

USDA documents released last year showed more than 1,000 violations of the regulations requiring the removal of brains and spinal cords in at least 35 states, Puerto Rico and the Virgin Islands, with some plants being cited repeatedly for infractions. In addition, a violation of similar regulations that apply to beef exported to Japan is the reason why Japan closed its borders to U.S. beef in January six weeks after reopening them.

Other experts also question the adequacy of the USDA's surveillance system. The USDA insists the prevalence of mad cow disease is low, but the agency has provided few details of its surveillance program, making it difficult for outside experts to know if the agency's monitoring plan is sufficient.

"It's impossible to judge the adequacy of the surveillance system without having a breakdown of the tested population by age and risk status," Elizabeth Mumford, a veterinarian and BSE expert at Safe Food Solutions in Bern, Switzerland, a company that provides advice on reducing mad cow risk to industry and governments, told UPI.

"Everybody would be happier and more confident and in a sense it might be able to go away a little bit for (the USDA) if they would just publish a breakdown on the tests," Mumford added.

UPI requested detailed records about animals tested under the USDA's surveillance plan via the Freedom of Information Act in May 2004 but nearly two years later has not received any corresponding documents from the agency, despite a federal law requiring agencies to comply within 30 days. This leaves open the question of whether the USDA is withholding the information, does not have the information or is so haphazardly organized that it cannot locate it.

Mumford said the prevalence of the disease in U.S. herds is probably quite low, but there have probably been other cases that have so far gone undetected. "They're only finding a very small fraction of that low prevalence," she said.

Mumford expressed surprise at the lack of concern about the deadly disease from American consumers. "I would expect the U.S. public to be more concerned," she said.

Markus Moser, a molecular biologist and chief executive officer of Prionics, a Swiss firm that manufactures BSE test kits, told UPI one concern is that if people are infected, the mad cow pathogen could become "humanized" or more easily transmitted from person to person.

"Transmission would be much easier, through all kinds of medical procedures" and even through the blood supply, Moser said.

http://www.upi.com/ConsumerHealthDaily/view.php?StoryID=20060315-055557-1284r

http://www.upi.com/Science_News/2003/12/30/Mad-Cow-Linked-to-thousands-of-CJD-cases/UPI-47861072816318/

CDC - Bovine Spongiform Encephalopathy and Variant Creutzfeldt ... Dr. Paul Brown is Senior Research Scientist in the Laboratory of Central Nervous System ... Address for correspondence: Paul Brown, Building 36, Room 4A-05, ...

http://www.cdc.gov/ncidod/eid/vol7no1/brown..htm

PAUL BROWN COMMENT TO ME ON THIS ISSUE

Tuesday, September 12, 2006 11:10 AM

"Actually, Terry, I have been critical of the USDA handling of the mad cow issue for some years, and with Linda Detwiler and others sent lengthy detailed critiques and recommendations to both the USDA and the Canadian Food Agency." ....TSS

http://madcowtesting.blogspot.com/2009/07/mad-cow-cover-up-usa-masked-as-sporadic.html

Subject: USDA OIG SEMIANNUAL REPORT TO CONGRESS FY 2007 1st Half (bogus BSE sampling FROM HEALTHY USDA CATTLE)

Date: June 21, 2007 at 2:49 pm PST

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...

Topics that will be covered in ongoing or planned reviews under Goal 1 include:

soundness of BSE maintenance sampling (APHIS),

implementation of Performance-Based Inspection System enhancements for specified risk material (SRM) violations and improved inspection controls over SRMs (FSIS and APHIS),

snip...

The findings and recommendations from these efforts will be covered in future semiannual reports as the relevant audits and investigations are completed.

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

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

OR, what the Honorable Phyllis Fong of the OIG found ;

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

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

Report No. 50601-10-KC January 2006

Finding 2 Inherent Challenges in Identifying and Testing High-Risk Cattle Still Remain

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

TUESDAY, JANUARY 17, 2017

FDA PART 589 -- SUBSTANCES PROHIBITED FROM USE IN ANIMAL FOOD OR FEEDVIOLATIONS OFFICIAL ACTION INDICATED OAI UPDATE 2016 to 2017 BSE TSE PRION

http://bovineprp.blogspot.com/2017/01/fda-part-589-substances-prohibited-from.html

Thursday, November 16, 2017

Texas Natural Meats Recalls Beef Products Due To Possible Specified Risk Materials Contamination

http://specifiedriskmaterial.blogspot.com/2017/11/texas-natural-meats-recalls-beef.html

PAGE NOT FOUND

IF you will notice, archived information has now been deleted before 2008. please be aware, 2006 was a banner year for tons and tons of banned mad cow protein fed out into commerce. i have some archived, but not all. the mad cow feed ban by the FDA et al was and is nothing but ink on paper...terry

https://www.fda.gov/Safety/Recalls/EnforcementReports/Archived/default.htm

TUESDAY, JANUARY 17, 2017

FDA PART 589 -- SUBSTANCES PROHIBITED FROM USE IN ANIMAL FOOD OR FEEDVIOLATIONS OFFICIAL ACTION INDICATED OAI UPDATE 2016 to 2017 BSE TSE PRION

http://bovineprp.blogspot.com/2017/01/fda-part-589-substances-prohibited-from.html

http://bovineprp.blogspot.com/

FRIDAY, NOVEMBER 3, 2017

BSE MAD COW TSE PRION DISEASE PET FOOD FEED IN COMMERCE INDUSTRY VS TERRY S. SINGELTARY Sr. A REVIEW

"I have a neighbor who is a dairy farmer. He tells me that he knows of several farmers who feed their cattle expired dog food. These farmers are unaware of any dangers posed to their cattle from the pet food contents. For these farmers, the pet food is just another source of protein."

IN CONFIDENCE

http://madcowfeed.blogspot.com/2017/11/bse-mad-cow-tse-prion-disease-pet-food.html

2017

FRIDAY, DECEMBER 22, 2017

Detection of PrPBSE and prion infectivity in the ileal Peyer's patch of young calves as early as 2 months after oral challenge with classical bovine spongiform encephalopathy

http://bovineprp.blogspot.com/2017/12/detection-of-prpbse-and-prion.html

TUESDAY, JULY 18, 2017

***> USDA announces Alabama case of Bovine Spongiform Encephalopathy Alabama

http://bovineprp.blogspot.com/2017/07/usda-announces-alabama-case-of-bovine.html

THURSDAY, JULY 20, 2017

***> USDA OIE Alabama Atypical L-type BASE Bovine Spongiform Encephalopathy BSE animal feeds for ruminants rule, 21 CFR 589..200

http://bovineprp.blogspot.com/2017/07/usda-oie-alabama-atypical-l-type-base.html

SUNDAY, JULY 23, 2017

***> atypical L-type BASE Bovine Amyloidotic Spongiform Encephalopathy BSE TSE PRION

http://bse-atypical.blogspot.com/2017/07/atypical-l-type-base-bovine-amyloidotic.html

SUNDAY, JULY 23, 2017

***> Experimental Infection of Cattle With a Novel Prion Derived From Atypical H-Type Bovine Spongiform Encephalopathy

http://bse-atypical.blogspot.com/2017/07/experimental-infection-of-cattle-with.html

SPONTANEOUS ATYPICAL BOVINE SPONGIFORM ENCEPHALOPATHY

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

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

Wednesday, December 21, 2016

TRANSMISSION, DIFFERENTIATION, AND PATHOBIOLOGY OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES 2016 ANNUAL REPORT ARS RESEARCH

http://transmissiblespongiformencephalopathy.blogspot.com/2016/12/transmission-differentiation-and.html

Tuesday, September 06, 2016

A comparison of classical and H-type bovine spongiform encephalopathy associated with E211K prion protein polymorphism in wild type and EK211 cattle following intracranial inoculation

http://bse-atypical.blogspot.com/2016/09/a-comparison-of-classical-and-h-type.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

Thursday, December 08, 2016

USDA APHIS National Scrapie Eradication Program October 2016 Monthly Report Fiscal Year 2017 atypical NOR-98 Scrapie

http://scrapie-usa.blogspot.com/2016/12/usda-aphis-national-scrapie-eradication.html

UESDAY, JULY 18, 2017

MINK FARMING USA TRANSMISSIBLE MINK ENCEPHALOPATHY TSE PRION DISEASE SURVEILLANCE AND TESTING

http://transmissible-mink-encephalopathy.blogspot.com/2017/07/mink-farming-usa-transmissible-mink.html

http://transmissible-mink-encephalopathy.blogspot.com/

*** This supports the theory that the importation of BSE contaminated feedstuff is the source of C-type BSE in Canada.

*** It also suggests a similar cause or source for atypical BSE in these countries. ***

see page 176 of 201 pages...tss

http://www.neuroprion.org/resources/pdf_docs/conferences/prion2009/prion2009_bookofabstracts.pdf

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

http://www.plosone.org/annotation/listThread.action;jsessionid=635CE9094E0EA15D5362B7D7B809448C?root=7143

Wednesday, July 15, 2015

Additional BSE TSE prion testing detects pathologic lesion in unusual brain location and PrPsc by PMCA only, how many cases have we missed?

http://transmissiblespongiformencephalopathy.blogspot.com/2015/07/additional-bse-tse-prion-testing.html

***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 ***

Posted by Terry S. Singeltary Sr. on 03 Jul 2015 at 16:53 GMT

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

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

Emmanuel Comoy, Jacqueline Mikol, Valerie Durand, Sophie Luccantoni, Evelyne Correia, Nathalie Lescoutra, Capucine Dehen, and Jean-Philippe Deslys Atomic Energy Commission; Fontenay-aux-Roses, France

Prion diseases (PD) are the unique neurodegenerative proteinopathies reputed to be transmissible under field conditions since decades. The transmission of Bovine Spongiform Encephalopathy (BSE) to humans evidenced that an animal PD might be zoonotic under appropriate conditions. Contrarily, in the absence of obvious (epidemiological or experimental) elements supporting a transmission or genetic predispositions, PD, like the other proteinopathies, are reputed to occur spontaneously (atpical animal prion strains, sporadic CJD summing 80% of human prion cases). Non-human primate models provided the first evidences supporting the transmissibiity of human prion strains and the zoonotic potential of BSE. Among them, cynomolgus macaques brought major information for BSE risk assessment for human health (Chen, 2014), according to their phylogenetic proximity to humans and extended lifetime. We used this model to assess the zoonotic potential of other animal PD from bovine, ovine and cervid origins even after very long silent incubation periods.

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

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

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

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

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

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thus questioning the origin of human sporadic cases

==========

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

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

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

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

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

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

PRION 2016 TOKYO

Saturday, April 23, 2016

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

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

Taylor & Francis

Prion 2016 Animal Prion Disease Workshop Abstracts

WS-01: Prion diseases in animals and zoonotic potential

Juan Maria Torres a, Olivier Andreoletti b, J uan-Carlos Espinosa a. Vincent Beringue c. Patricia Aguilar a,

Natalia Fernandez-Borges a. and Alba Marin-Moreno a

"Centro de Investigacion en Sanidad Animal (CISA-INIA). Valdeolmos, Madrid. Spain; b UMR INRA -ENVT 1225 Interactions Holes Agents Pathogenes. ENVT. Toulouse. France: "UR892. Virologie Immunologie Moléculaires, Jouy-en-Josas. France

Dietary exposure to bovine spongiform encephalopathy (BSE) contaminated bovine tissues is considered as the origin of variant Creutzfeldt Jakob (vCJD) disease in human. To date, BSE agent is the only recognized zoonotic prion. Despite the variety of Transmissible Spongiform Encephalopathy (TSE) agents that have been circulating for centuries in farmed ruminants there is no apparent epidemiological link between exposure to ruminant products and the occurrence of other form of TSE in human like sporadic Creutzfeldt Jakob Disease (sCJD). However, the zoonotic potential of the diversity of circulating TSE agents has never been systematically assessed. The major issue in experimental assessment of TSEs zoonotic potential lies in the modeling of the 'species barrier', the biological phenomenon that limits TSE agents' propagation from a species to another. In the last decade, mice genetically engineered to express normal forms of the human prion protein has proved essential in studying human prions pathogenesis and modeling the capacity of TSEs to cross the human species barrier.

To assess the zoonotic potential of prions circulating in farmed ruminants, we study their transmission ability in transgenic mice expressing human PrPC (HuPrP-Tg). Two lines of mice expressing different forms of the human PrPC (129Met or 129Val) are used to determine the role of the Met129Val dimorphism in susceptibility/resistance to the different agents.

These transmission experiments confirm the ability of BSE prions to propagate in 129M- HuPrP-Tg mice and demonstrate that Met129 homozygotes may be susceptible to BSE in sheep or goat to a greater degree than the BSE agent in cattle and that these agents can convey molecular properties and neuropathological indistinguishable from vCJD. However homozygous 129V mice are resistant to all tested BSE derived prions independently of the originating species suggesting a higher transmission barrier for 129V-PrP variant.

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

why do we not want to do TSE transmission studies on chimpanzees $

5. A positive result from a chimpanzee challenged severly would likely create alarm in some circles even if the result could not be interpreted for man. I have a view that all these agents could be transmitted provided a large enough dose by appropriate routes was given and the animals kept long enough. Until the mechanisms of the species barrier are more clearly understood it might be best to retain that hypothesis.

snip...

R. BRADLEY

https://web.archive.org/web/20170126051158/http://collections.europarchive.org/tna/20080102222950/http://www.bseinquiry.gov.uk/files/yb/1990/09/23001001.pdf

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

MONDAY, JUNE 12, 2017

Rethinking Major grain organizations opposition to CFIA's control zone approach to Chronic Wasting CWD TSE Prion Mad Deer Type Disease 2017 ?

http://chronic-wasting-disease.blogspot.com/2017/06/rethinking-major-grain-organizations.html

WEDNESDAY, MAY 17, 2017

*** Chronic Wasting Disease CWD TSE Prion aka Mad Deer Disease and the Real Estate Market Land Values ***

http://chronic-wasting-disease.blogspot.com/2017/05/chronic-wasting-disease-cwd-tse-prion.html

ZOONOTIC, ZOONOSIS, CHRONIC WASTING DISEASE CWD TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHY TSE PRION

10. ZOONOTIC, ZOONOSIS, CHRONIC WASTING DISEASE CWD TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHY TSE PRION AKA MAD DEER ELK DISEASE IN HUMANS, has it already happened, that should be the question...

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

EFSA Panel on Biological Hazards (BIOHAZ) Antonia Ricci Ana Allende Declan Bolton Marianne Chemaly Robert Davies Pablo Salvador Fernández Escámez ... See all authors

First published: 17 January 2018 **https://doi.org/10.2903/j.efsa.2018.5132** ;

also, see;

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

snip...

The tissue distribution of infectivity in CWD-infected cervids is now known to extend beyond CNS and lymphoid tissues. While the removal of these specific tissues from the food chain would reduce human dietary exposure to infectivity, exclusion from the food chain of the whole carcass of any infected animal would be required to eliminate human dietary exposure.

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

zoonosis zoonotic cervid tse prion cwd to humans, preparing for the storm

An alternative to modeling the species barrier is the cell-free conversion assay which points to CWD as the animal prion disease with the greatest zoonotic potential, after (and very much less than) BSE.116

https://www.tandfonline.com/doi/pdf/10.4161/pri.29237

To date there is no direct evidence that CWD has been or can be transmitted from animals to humans.

However, initial findings from a laboratory research project funded by the Alberta Prion Research Institute (APRI) and Alberta Livestock Meat Agency (ALMA), and led by a Canadian Food Inspection Agency (CFIA) scientist indicate that CWD has been transmitted to cynomolgus macaques (the non-human primate species most closely related to humans that may be used in research), through both the intracranial and oral routes of exposure.

Both infected brain and muscle tissues were found to transmit disease.

Health Canada's Health Products and Food Branch (HPFB) was asked to consider the impact of these findings on the Branch's current position on CWD in health products and foods.

Summary and Recommendation:

snip...

Health Portfolio partners were recently made aware of initial findings from a research project led by a CFIA scientist that have demonstrated that cynomolgus macaques can be infected via intracranial exposure and oral gavage with CWD infected muscle.

These findings suggest that CWD, under specific experimental conditions, has the potential to cross the human species barrier, including by enteral feeding of CWD infected muscle.

https://www.thetyee.ca/Documents/2017/06/24/Risk-Advisory-Opinion-CWD-2017.pdf

*** WDA 2016 NEW YORK ***

We found that CWD adapts to a new host more readily than BSE and that human PrP was unexpectedly prone to misfolding by CWD prions.

In addition, we investigated the role of specific regions of the bovine, deer and human PrP protein in resistance to conversion by prions from another species.

***We have concluded that the human protein has a region that confers unusual

susceptibility to conversion by CWD prions.

Student Presentations Session 2

The species barriers and public health threat of CWD and BSE prions

Ms. Kristen Davenport1, Dr. Davin Henderson1, Dr. Candace Mathiason1, Dr. Edward Hoover1 1Colorado State University

Chronic wasting disease (CWD) is spreading rapidly through cervid populations in the USA. Bovine spongiform encephalopathy (BSE, mad cow disease) arose in the 1980s because cattle were fed recycled animal protein.

These and other prion diseases are caused by abnormal folding of the normal prion protein (PrP) into a disease causing form (PrPd), which is pathogenic to nervous system cells and can cause subsequent PrP to misfold. CWD spreads among cervids very efficiently, but it has not yet infected humans. On the other hand, BSE was spread only when cattle consumed infected bovine or ovine tissue, but did infect humans and other species.

The objective of this research is to understand the role of PrP structure in cross-species infection by CWD and BSE. To study the propensity of each species' PrP to be induced to misfold by the presence of PrPd from verious species, we have used an in vitro system that permits detection of PrPd in real-time.

We measured the conversion efficiency of various combinations of PrPd seeds and PrP substrate combinations.

We observed the cross-species behavior of CWD and BSE, in addition to feline-adapted CWD and BSE. We found that CWD adapts to a new host more readily than BSE and that human PrP was unexpectedly prone to misfolding by CWD prions. In addition, we investigated the role of specific regions of the bovine, deer and human PrP protein in resistance to conversion by prions from another species.

***We have concluded that the human protein has a region that confers unusual susceptibility to conversion by CWD prions. CWD is unique among prion diseases in its rapid spread in natural populations. BSE prions are essentially unaltered upon passage to a new species, while CWD adapts to the new species. This adaptation has consequences for surveillance of humans exposed to CWD. Wildlife Disease Risk Communication Research Contributes to Wildlife Trust Administration Exploring perceptions about chronic wasting disease risks among wildlife and agriculture professionals and stakeholders

http://www.wda2016.org/uploads/5/8/6/1/58613359/wda_2016_conference_proceedings_low_res.pdf

CDC CWD 2018 TRANSMISSION

https://www.cdc.gov/prions/cwd/transmission.html

Transmissible Spongiform Encephalopathies

Spongiform Encephalopathy in Captive Wild ZOO BSE INQUIRY

https://web.archive.org/web/20090506001201/http://www.bseinquiry.gov.uk/files/mb/m09a/tab03.pdf

BSE INQUIRY

CJD9/10022

October 1994

Mr R.N. Elmhirst Chairman British Deer Farmers Association Holly Lodge Spencers Lane

BerksWell Coventry CV7 7 BZ

Dear Mr Elmhirst,

CREUTZFELDT-JAKOB DISEASE (CJD) SURVEILLANCE UNIT REPORT

Thank you for your recent letter concerning the publication of the third annual report from the CJD Surveillance Unit. I am sorry that you are dissatisfied with the way in which this report was published.

The Surveillance Unit is a completely independant outside body and the Department of Health is committed to publishing their reports as soon as they become available. In the circumstances it is not the practice to circulate the report for comment since the findings of the report would not be amended. In future we can ensure that the British Deer Farmers Association receives a copy of the report in advance of publication.

The Chief Medical Officer has undertaken to keep the public fully informed of the results of any research in respect of CJD. This report was entirely the work of the unit and was produced completely independantly of the the Department.

The statistical results reqarding the consumption of venison was put into perspective in the body of the report and was not mentioned at all in the press release. Media attention regarding this report was low key but gave a realistic presentation of the

statistical findings of the Unit. This approach to publication was successful in that consumption of venison was highlighted only once by the media ie. in the News at one television proqramme.

I believe that a further statement about the report, or indeed statistical links between CJD and consumption of venison, would increase, and quite possibly give damaging credence, to the whole issue. From the low key media reports of which I am aware it seems unlikely that venison consumption will suffer adversely, if at all.

http://web.archive.org/web/20030511010117/http://www.bseinquiry.gov.uk/files/yb/1994/10/00003001.pdf

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

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There is some evidence that risk of CJD INCREASES WITH INCREASING FREQUENCY OF LAMB EATING (p = 0.02).

The evidence for such an association between beef eating and CJD is weaker (p = 0.14). When only controls for whom a relative was interviewed are included, this evidence becomes a little STRONGER (p = 0.08).

snip...

It was found that when veal was included in the model with another exposure, the association between veal and CJD remained statistically significant (p = < 0.05 for all exposures), while the other exposures ceased to be statistically significant (p = > 0.05).

snip...

In conclusion, an analysis of dietary histories revealed statistical associations between various meats/animal products and INCREASED RISK OF CJD. When some account was taken of possible confounding, the association between VEAL EATING AND RISK OF CJD EMERGED AS THE STRONGEST OF THESE ASSOCIATIONS STATISTICALLY. ...

snip...

In the study in the USA, a range of foodstuffs were associated with an increased risk of CJD, including liver consumption which was associated with an apparent SIX-FOLD INCREASE IN THE RISK OF CJD. By comparing the data from 3 studies in relation to this particular dietary factor, the risk of liver consumption became non-significant with an odds ratio of 1.2 (PERSONAL COMMUNICATION, PROFESSOR A. HOFMAN. ERASMUS UNIVERSITY, ROTTERDAM). (???...TSS)

snip...see full report ;

https://web.archive.org/web/20170126073306/http://collections..europarchive.org/tna/20090505194948/http://bseinquiry.gov.uk/files/y

TUESDAY, SEPTEMBER 12, 2017

CDC Now Recommends Strongly consider having the deer or elk tested for CWD before you eat the meat

http://chronic-wasting-disease.blogspot.com/2017/09/cdc-now-recommends-strongly-consider.html

SATURDAY, JANUARY 27, 2018

CDC CHRONIC WASTING DISEASE CWD TSE PRION UPDATE REPORT USA JANUARY 2018

http://chronic-wasting-disease.blogspot.com/2018/01/cdc-chronic-wasting-disease-cwd-tse.html

Subject: CDC CHRONIC WASTING DISEASE CWD TSE PRION UPDATE REPORT USA JANUARY 2018

CHRONIC WASTING DISEASE CWD TSE PRION IS THE USA AND NORTH AMERICA'S MAD COW DISEASE.

THE USDA INC ET AL WORKED VERY HARD CONCEALING BSE TSE PRION IN CATTLE. they almost succeeded $$$

BUT CWD TSE PRION IN CERVIDS IS A DIFFERENT BEAST, THE COVER UP THERE, USDA INC COULD NOT CONTAIN.

SPORADIC CJD IS 85%+ OF ALL HUMAN TSE PRION DISEASE.

SPORADIC CJD HAS NOW BEEN LINKED TO TY PICAL AND ATY PICAL BSE, SCRAPIE, AND CWD.

SPORADIC/SPONTANEOUS TSE HAS NEVER BEEN PROVEN.

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

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

CDC CWD TSE PRION UPDATE USA JANUARY 2018

As of January 2018, CWD in free-ranging deer, elk and/or moose has been reported in at least 22 states in the continental United States, as well as two provinces in Canada. In addition, CWD has been reported in reindeer and moose in Norway, and a small number of imported cases have been reported in South Korea. The disease has also been found in farmed deer and elk. CWD was first identified in captive deer in the late 1960s in Colorado and in wild deer in 1981. By the 1990s, it had been reported in surrounding areas in northern Colorado and southern Wyoming. Since 2000, the area known to be affected by CWD in free-ranging animals has increased to at least 22 states, including states in the Midwest, Southwest, and limited areas on the East Coast.. It is possible that CWD may also occur in other states without strong animal surveillance systems, but that cases haven't been detected yet. Once CWD is established in an area, the risk can remain for a long time in the environment. The affected areas are likely to continue to expand. Nationwide, the overall occurrence of CWD in free-ranging deer and elk is relatively low. However, in several locations where the disease is established, infection rates may exceed 10 percent (1 in 10), and localized infection rates of more than 25 percent (1 in 4) have been reported. The infection rates among some captive deer can be much higher, with a rate of 79% (nearly 4 in 5) reported from at least one captive herd. As of January 2018, there were 186 counties in 22 states with reported CWD in free-ranging cervids.

Chronic Wasting Disease Among Free-Ranging Cervids by County, United States, January 2018

snip....

https://www.cdc.gov/prions/cwd/occurrence.html

*** 2017-2018 CWD TSE Prion UPDATE

cwd-2018-Map.jpg

https://www.cdc.gov/prions/cwd/occurrence.html

Prion 2017 Conference Abstracts CWD

2017 PRION CONFERENCE

First evidence of intracranial and peroral transmission of Chronic Wasting Disease (CWD) into Cynomolgus macaques: a work in progress

Stefanie Czub1, Walter Schulz-Schaeffer2, Christiane Stahl-Hennig3, Michael Beekes4, Hermann Schaetzl5 and Dirk Motzkus6
1

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

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

Here we present first data on a group of animals either challenged ic with steel wires or per orally and sacrificed with incubation times ranging from 4.5 to 6.9 years at postmortem. Three animals displayed signs of mild clinical disease, including anxiety, apathy, ataxia and/or tremor. In four animals wasting was observed, two of those had confirmed diabetes. All animals have variable signs of prion neuropathology in spinal cords and brains and by supersensitive IHC, reaction was detected in spinal cord segments

of all animals. Protein misfolding cyclic amplification (PMCA), real-time quaking-induced conversion (RT-QuiC) and PET-blot assays to further substantiate these findings are on the way, as well as bioassays in bank voles and transgenic mice.

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

PRION 2017 DECIPHERING NEURODEGENERATIVE DISORDERS

Subject: PRION 2017 CONFERENCE DECIPHERING NEURODEGENERATIVE DISORDERS VIDEO

PRION 2017 CONFERENCE DECIPHERING NEURODEGENERATIVE DISORDERS

*** PRION 2017 CONFERENCE VIDEO

https://www.youtube.com/embed/Vtt1kAVDhDQ

http://prion2017.org/programme/

TUESDAY, JUNE 13, 2017

PRION 2017 CONFERENCE ABSTRACT

First evidence of intracranial and peroral transmission of Chronic Wasting Disease (CWD) into Cynomolgus macaques: a work in progress

http://chronic-wasting-disease.blogspot.com/2017/06/prion-2017-conference-abstract-first.html

SATURDAY, JULY 29, 2017

Risk Advisory Opinion: Potential Human Health Risks from Chronic Wasting Disease CFIA, PHAC, HC (HPFB and FNIHB), INAC, Parks Canada, ECCC and AAFC

http://chronic-wasting-disease.blogspot.com/2017/07/risk-advisory-opinion-potential-human.html

*** The potential impact of prion diseases on human health was greatly magnified by the recognition that interspecies transfer of BSE to humans by beef ingestion resulted in vCJD. While changes in animal feed constituents and slaughter practices appear to have curtailed vCJD, there is concern that CWD of free-ranging deer and elk in the U.S. might also cross the species barrier. Thus, consuming venison could be a source of human prion disease. Whether BSE and CWD represent interspecies scrapie transfer or are newly arisen prion diseases is unknown. Therefore, the possibility of transmission of prion disease through other food animals cannot be ruled out. There is evidence that vCJD can be transmitted through blood transfusion. There is likely a pool of unknown size of asymptomatic individuals infected with vCJD, and there may be asymptomatic individuals infected with the CWD equivalent. These circumstances represent a potential threat to blood, blood products, and plasma supplies.

http://cdmrp.army.mil/prevfunded/nprp/NPRP_Summit_Final_Report.pdf

Transmission Studies

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

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

snip...

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

http://www.fsis.usda.gov/OPPDE/Comments/03-025IFA/03-025IFA-2.pdf

Prion Infectivity in Fat of Deer with Chronic Wasting Disease▿

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

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

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

Prions in Skeletal Muscles of Deer with Chronic Wasting Disease

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

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

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

see where it is stated NO STRONG evidence. so, does this mean there IS casual evidence ???? "Our conclusion stating that we found no strong evidence of CWD transmission to humans"

From: TSS (216-119-163-189.ipset45.wt.net)

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

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

From: "Belay, Ermias"

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

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

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

Dear Sir/Madam,

In the Archives of Neurology you quoted (the abstract of which was attached to your email), we did not say CWD in humans will present like variant CJD. That assumption would be wrong. I encourage you to read the whole article and call me if you have questions or need more clarification (phone: 404-639-3091). Also, we do not claim that "no-one has ever been infected with prion disease from eating venison." Our conclusion stating that we found no strong evidence of CWD transmission to humans in the article you quoted or in any other forum is limited to the patients we investigated.

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

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

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

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

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

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

Thursday, April 03, 2008

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

snip...

*** twenty-seven CJD patients who regularly consumed venison were reported to the Surveillance Center***,

snip... full text ;

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

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

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

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

*** These results would seem to suggest that CWD does indeed have zoonotic potential, at least as judged by the compatibility of CWD prions and their human PrPC target. Furthermore,

extrapolation from this simple in vitro assay suggests that if zoonotic CWD occurred, it would most likely effect those of the PRNP codon 129-MM genotype and that the PrPres type would be similar to that found in the most common subtype of sCJD (MM1).***

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

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

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

SEE; Travel History, Hunting, and Venison Consumption Related to Prion Disease Exposure, 2006-2007 FoodNet Population Survey

Monday, May 23, 2011

CDC Assesses Potential Human Exposure to Prion Diseases Travel Warning

Public release date: 23-May-2011

Contact: Francesca Costanzo **adajmedia@elsevier.com** 215-239-3249 Elsevier Health Sciences

CDC assesses potential human exposure to prion diseases Study results reported in the Journal of the American Dietetic Association Philadelphia, PA, May 23, 2011 – Researchers from the Centers for Disease Control and Prevention (CDC) have examined the potential for human exposure to prion diseases, looking at hunting, venison consumption, and travel to areas in which prion diseases have been reported in animals. Three prion diseases in particular – bovine spongiform encephalopathy (BSE or "Mad Cow Disease"), variant Creutzfeldt-Jakob disease (vCJD), and chronic wasting disease (CWD) – were specified in the investigation. The results of this investigation are published in the June issue of the Journal of the American Dietetic Association.

"While prion diseases are rare, they are generally fatal for anyone who becomes infected. More than anything else, the results of this study support the need for continued surveillance of prion diseases," commented lead investigator Joseph Y. Abrams, MPH, National Center for Emerging and Zoonotic Infectious Diseases, CDC, Atlanta."But it's also important that people know the facts about these diseases, especially since this study shows that a good number of people have participated in activities that may expose them to infection-causing agents."

Although rare, human prion diseases such as CJD may be related to BSE. Prion (proteinaceous infectious particles) diseases are a group of rare brain diseases that affect humans and animals. When a person gets a prion disease, brain function is impaired. This causes memory and personality changes, dementia, and problems with movement. All of these worsen over time. These diseases are invariably fatal. Since these diseases may take years to manifest, knowing the extent of human exposure to possible prion diseases could become important in the event of an outbreak.

CDC investigators evaluated the results of the 2006-2007 population survey conducted by the Foodborne Diseases Active Surveillance Network (FoodNet). This survey collects information on food consumption practices, health outcomes, and demographic characteristics of residents of the participating Emerging Infections Program sites. The survey was conducted in Connecticut, Georgia, Maryland, Minnesota, New Mexico, Oregon, and Tennessee, as well as five counties in the San Francisco Bay area, seven counties in the Greater Denver area, and 34 counties in western and northeastern New York.

Survey participants were asked about behaviors that could be associated with exposure to the agents causing BSE and CWD, including travel to the nine countries considered to be BSE-endemic (United Kingdom, Republic of Ireland, France, Portugal, Switzerland, Italy, the Netherlands, Germany, Spain) and the cumulative length of stay in each of those countries. Respondents were asked if they ever had hunted for deer or elk, and if that hunting had taken place in areas considered to be CWD-endemic (northeastern Colorado, southeastern Wyoming or southwestern Nebraska). They were also asked if they had ever consumed venison, the frequency of consumption, and whether the meat came from the wild.

The proportion of survey respondents who reported travel to at least one of the nine BSE endemic countries since 1980 was 29.5%. Travel to the United Kingdom was reported by 19.4% of respondents, higher than to any other BSE-endemic country. Among those who traveled, the median duration of travel to the United Kingdom (14 days) was longer than that of any other BSE-endemic country. Travelers to the UK were more likely to have spent at least 30 days in the country (24.9%) compared to travelers to any other BSE endemic country. The prevalence and extent of travel to the UK indicate that health concerns in the UK may also become issues for US residents.

The proportion of survey respondents reporting having hunted for deer or elk was 18.5% and 1.2% reported having hunted for deer or elk in CWD-endemic areas. Venison consumption was reported by 67.4% of FoodNet respondents, and 88.6% of those reporting venison consumption had obtained all of their meat from the wild. These findings reinforce the importance of CWD surveillance and control programs for wild deer and elk to reduce human exposure to the CWD agent. Hunters in CWD-endemic areas are advised to take simple precautions such as: avoiding consuming meat from sickly deer or elk, avoiding consuming brain or spinal cord tissues, minimizing the handling of brain and spinal cord tissues, and wearing gloves when field-dressing carcasses.

According to Abrams, "The 2006-2007 FoodNet population survey provides useful information should foodborne prion infection become an increasing public health concern in the future. The data presented describe the prevalence of important behaviors and their associations with demographic characteristics. Surveillance of BSE, CWD, and human prion diseases are critical aspects of addressing the burden of these diseases in animal populations and how that may relate to human health."

###

The article is "Travel history, hunting, and venison consumption related to prion disease exposure, 2006-2007 FoodNet population survey" by Joseph Y. Abrams, MPH; Ryan A. Maddox, MPH; Alexis R Harvey, MPH; Lawrence B. Schonberger, MD; and Ermias D. Belay, MD. It appears in the Journal of the American Dietetic Association, Volume 111, Issue 6 (June 2011) published by Elsevier.

In an accompanying podcast CDC's Joseph Y. Abrams discusses travel, hunting, and eating venison in relation to prion diseases. It is available at **http://adajournal.org/content/podcast**.

http://www.eurekalert.org/pub_releases/2011-05/ehs-cap051811.php

Thursday, May 26, 2011

Travel History, Hunting, and Venison Consumption Related to Prion Disease Exposure, 2006-2007 FoodNet Population Survey

Journal of the American Dietetic Association Volume 111, Issue 6 , Pages 858-863, June 2011.

Travel History, Hunting, and Venison Consumption Related to Prion Disease Exposure, 2006-2007 FoodNet Population Survey

Joseph Y. Abrams, MPH, Ryan A. Maddox, MPH , Alexis R. Harvey, MPH , Lawrence B. Schonberger, MD , Ermias D. Belay, MD

Accepted 15 November 2010. Abstract Full Text PDF References .

Abstract

The transmission of bovine spongiform encephalopathy (BSE) to human beings and the spread of chronic wasting disease (CWD) among cervids have prompted concerns about zoonotic transmission of prion diseases. Travel to the United Kingdom and other European countries, hunting for deer or elk, and venison consumption could result in the exposure of US residents to the agents that cause BSE and CWD. The Foodborne Diseases Active Surveillance Network 2006-2007 population survey was used to assess the prevalence of these behaviors among residents of 10 catchment areas across the United States. Of 17,372 survey respondents, 19.4% reported travel to the United Kingdom since 1980, and 29.5% reported travel to any of the nine European countries considered to be BSE-endemic since 1980. The proportion of respondents who had ever hunted deer or elk was 18.5%, and 1.2% had hunted deer or elk in a CWD–endemic area. More than two thirds (67.4%) reported having ever eaten deer or elk meat. Respondents who traveled spent more time in the United Kingdom (median 14 days) than in any other BSE-endemic country. Of the 11,635 respondents who had consumed venison, 59.8% ate venison at most one to two times during their year of highest consumption, and 88.6% had obtained all of their meat from the wild. The survey results were useful in determining the prevalence and frequency of behaviors that could be important factors for foodborne prion transmission.

http://www.adajournal.org/article/S0002-8223(11)00278-1/abstract

PLUS, THE CDC DID NOT PUT THIS WARNING OUT FOR THE WELL BEING OF THE DEER AND ELK ;

Thursday, May 26, 2011

Travel History, Hunting, and Venison Consumption Related to Prion Disease Exposure, 2006-2007 FoodNet Population Survey

Journal of the American Dietetic Association Volume 111, Issue 6 , Pages 858-863, June 2011.

http://transmissiblespongiformencephalopathy.blogspot.com/2011/05/travel-history-hunting-and-venison.html

NOR IS THE FDA recalling this CWD positive elk meat for the well being of the dead elk ;

Wednesday, March 18, 2009

Noah's Ark Holding, LLC, Dawson, MN RECALL Elk products contain meat derived from an elk confirmed to have CWD NV, CA, TX, CO, NY, UT, FL, OK RECALLS AND FIELD CORRECTIONS: FOODS CLASS II

http://chronic-wasting-disease.blogspot.com/2009/03/noahs-ark-holding-llc-dawson-mn-recall.html

THURSDAY, MARCH 08, 2018

Cervid, Wild Hogs, Coyotes, Wolves, Cats, Rodents, Gut Piles and Scavengers, A Potential Risk as Regards Disease Transmission CWD TSE Prion

http://chronic-wasting-disease.blogspot.com/2018/03/cervid-wild-hogs-coyotes-wolves-cats.html

the tse prion aka mad cow type disease is not your normal pathogen.

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

you cannot cook the TSE prion disease out of meat.

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

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

the TSE prion agent also survives Simulated Wastewater Treatment Processes.

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

you can bury it and it will not go away.

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

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

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

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

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

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

Laboratory of Central Nervous System Studies, National Institute of

Neurological Disorders and Stroke, National Institutes of Health,

Bethesda, MD 20892.

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

PMID: 8006664 [PubMed - indexed for MEDLINE]

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

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

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

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

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

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

Gudmundur Georgsson1, Sigurdur Sigurdarson2 and Paul Brown3

http://jgv.sgmjournals.org/content/87/12/3737.full

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

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

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

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

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

https://prion2015.files.wordpress.com/2015/05/programguide1.pdf

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

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

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

Production

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

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

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

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

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

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

http://transmissiblespongiformencephalopathy.blogspot.com/2013/07/rapid-assessment-of-bovine-spongiform.html

PPo4-4:

Survival and Limited Spread of TSE Infectivity after Burial

http://www.neuroprion.org/resources/pdf_docs/conferences/prion2010/prion_2010_programme.pdf

http://chronic-wasting-disease.blogspot.com/2010/09/cwd-prion-2010.html

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

In the present study, the outdoor objects were removed from the infected flock 8 weeks prior to sampling and were positive by

sPMCA at very low levels (2 out of 37 reactions).

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Wednesday, December 16, 2015

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

http://scrapie-usa.blogspot.com/2015/12/objects-in-contact-with-classical.html

161: Prion soil binding may explain efficient horizontal CWD transmission

Nathaniel Denkers1, Davin Henderson1, Shannon Bartelt-Hunt2, Jason Bartz3 and Edward Hoover1

1Colorado State University; Fort Collins, Colorado USA

2University of Nebraska-Lincoln; Omaha, Nebraska USA

3Creighton University; Omaha, Nebraska USA

Background Chronic wasting disease (CWD) is unique due to the facile spread in nature. The interaction of excreted CWD prions and soil is a hypothesized contributor in environmental transmission. The present study examines whether and to what degree CWD prions bind to silty clay loam (SCL) using an adapted version of real-time quaking-induced conversion (RT-QuIC) methodology.

Materials and Methods Varying amounts (50–3.12 mg) of SCL were incubated with 1 mL-serial dilutions of CWD (+), CWD (–), or no brain homogenate (BH). Samples were centrifuged, washed, diluted 1:10 in 0.1% SDS, and 2.5 uL seeded in RT-QuIC assays employing recombinant Syrian hamster prion PrP substrate. Multiple well replicates of sample and supernatant fractions were assayed for positive seeding

activity (recorded as thioflavin T fluorescence emission; 480 nm). Samples were considered positive if they crossed a threshold of 25,000. Reaction rates (RR) were calculated, averaged, and expressed as 1/RR.

Results Positive seeding activity was detected for most SCL samples incubated with CWD (+) BH dilutions. Higher SCL concentrations (50 mg) produced low fluorescent readings due to optical interference. Lower SCL concentrations (6.25 mg) produced minimal optical interference and removed the vast majority of seeding activity from CWD+ BH in a concentration-dependent manner; determined by seeding activity in residual BH supernatants. Control SCL and supernatants produced minimal false-positive reactions (8 of 240 replicates; 3.3%). We estimated the prion binding capacity of SCL to be 0.16 ng/mg.

Conclusion Silty clay loam exhibits highly efficient prion binding, inferring a durable environmental reservoir, and an efficient mechanism for indirect horizontal CWD transmission.

https://www.tandfonline.com/doi/full/10.1080/19336896.2015.1033248

TSE Scrapie, CWD, BSE, Prion, Soil

Clay content and pH: soil characteristic associations with the persistent presence of chronic wasting disease in northern Illinois

Sheena J. Dorak, Michelle L. Green, Michelle M. Wander, Marilyn O. Ruiz, Michael G. Buhnerkempe, Ting Tian, Jan E. Novakofski & Nohra E. Mateus-Pinilla

Scientific Reports volume 7, Article number: 18062 (2017)
doi:10.1038/s41598-017-18321-x

Download Citation

Ecological epidemiology Ecological modelling Infectious diseases Prions

Received: 21 August 2017

Accepted: 08 December 2017

Published online: 22 December 2017

Abstract

Environmental reservoirs are important to infectious disease transmission and persistence, but empirical analyses are relatively few. The natural environment is a reservoir for prions that cause chronic wasting disease (CWD) and influences the risk of transmission to susceptible cervids. Soil is one environmental component demonstrated to affect prion infectivity and persistence. Here we provide the first landscape predictive model for CWD based solely on soil characteristics. We built a boosted regression tree model to predict the probability of the persistent presence of CWD in a region of northern Illinois using CWD surveillance in deer and soils data. We evaluated the outcome for possible pathways by which soil characteristics may increase the probability of CWD transmission via environmental contamination. Soil clay content and pH were the most important predictive soil characteristics of the persistent presence of CWD. The results suggest that exposure to prions in the environment is greater where percent clay is less than 18% and soil pH is greater than 6.6. These characteristics could alter availability of prions immobilized in soil and contribute to the environmental risk factors involved in the epidemiological complexity of CWD infection in natural populations of white-tailed deer.

https://www.nature.com/articles/s41598-017-18321-x

Oral Transmissibility of Prion Disease Is Enhanced by Binding to Soil Particles

Author Summary

Transmissible spongiform encephalopathies (TSEs) are a group of incurable neurological diseases likely caused by a misfolded form of the prion protein. TSEs include scrapie in sheep, bovine spongiform encephalopathy (“mad cow” disease) in cattle, chronic wasting disease in deer and elk, and Creutzfeldt-Jakob disease in humans. Scrapie and chronic wasting disease are unique among TSEs because they can be transmitted between animals, and the disease agents appear to persist in environments previously inhabited by infected animals. Soil has been hypothesized to act as a reservoir of infectivity and to bind the infectious agent. In the current study, we orally dosed experimental animals with a common clay mineral, montmorillonite, or whole soils laden with infectious prions, and compared the transmissibility to unbound agent. We found that prions bound to montmorillonite and whole soils remained orally infectious, and, in most cases, increased the oral transmission of disease compared to the unbound agent. The results presented in this study suggest that soil may contribute to environmental spread of TSEs by increasing the transmissibility of small amounts of infectious agent in the environment.

https://www.aphis.usda.gov/emergency_response/downloads/tools/johnson et al prions in soil.pdf

tse prion soil

http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0058630

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3567181/pdf/ppat.1003113.pdf

http://www.nature.com/srep/2015/150210/srep08358/full/srep08358.html?WT.ec_id=SREP-639-20150217

http://www.cell.com/cell-reports/pdfExtended/S2211-1247(15)00437-4

cwd tse prion and soil, see more ;

http://chronic-wasting-disease.blogspot.com/2017/01/chronic-wasting-disease-cwd-tse-prion.html

Between 1996 and 2002, chronic wasting disease was diagnosed in 39 herds of farmed elk in Saskatchewan in a single epidemic. All of these herds were depopulated as part of the Canadian Food Inspection Agency's (CFIA) disease eradication program. Animals, primarily over 12 mo of age, were tested for the presence CWD prions following euthanasia. Twenty-one of the herds were linked through movements of live animals with latent CWD from a single infected source herd in Saskatchewan, 17 through movements of animals from 7 of the secondarily infected herds.

***The source herd is believed to have become infected via importation of animals from a game farm in South Dakota where CWD was subsequently diagnosed (7,4). A wide range in herd prevalence of CWD at the time of herd depopulation of these herds was observed. Within-herd transmission was observed on some farms, while the disease remained confined to the introduced animals on other farms.

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2081988/

spreading cwd around...tss

Chronic Wasting Disease (CWD) outbreaks and surveillance program in the Republic of Korea Chronic Wasting Disease (CWD) outbreaks and surveillance program in the Republic of Korea

Hyun-Joo Sohn, Yoon-Hee Lee, Min-jeong Kim, Eun-Im Yun, Hyo-Jin Kim, Won-Yong Lee, Dong-Seob Tark, In- Soo Cho, Foreign Animal Disease Research Division, National Veterinary Research and Quarantine Service, Republic of Korea

Chronic wasting disease (CWD) has been recognized as an important prion disease in native North America deer and Rocky mountain elks. The disease is a unique member of the transmissible spongiform encephalopathies (TSEs), which naturally affects only a few species. CWD had been limited to USA and Canada until 2000.

On 28 December 2000, information from the Canadian government showed that a total of 95 elk had been exported from farms with CWD to Korea.

These consisted of 23 elk in 1994 originating from the so-called "source farm" in Canada, and 72 elk in 1997, which had been held in pre export quarantine at the "source farm".

Based on export information of CWD suspected elk from Canada to Korea, CWD surveillance program was initiated by the Ministry of Agriculture and Forestry (MAF) in 2001.

All elks imported in 1997 were traced back, however elks imported in 1994 were impossible to identify.

CWD control measures included stamping out of all animals in the affected farm, and thorough cleaning and disinfection of the premises.

In addition, nationwide clinical surveillance of Korean native cervids, and improved measures to ensure reporting of CWD suspect cases were implemented.

*Total of 9 elks were found to be affected. CWD was designated as a notifiable disease under the Act for Prevention of Livestock Epidemics in 2002.

*Additional CWD cases - 12 elks and 2 elks - were diagnosed in 2004 and 2005.

*Since February of 2005, when slaughtered elks were found to be positive, all slaughtered cervid for human consumption at abattoirs were designated as target of the CWD surveillance program.

Currently, CWD laboratory testing is only conducted by National Reference Laboratory on CWD, which is the Foreign Animal Disease Division (FADD) of National Veterinary Research and Quarantine Service (NVRQS).

*In July 2010, one out of 3 elks from Farm 1 which were slaughtered for the human consumption was confirmed as positive.

*Consequently, all cervid - 54 elks, 41 Sika deer and 5 Albino deer - were culled and one elk was found to be positive.

Epidemiological investigations were conducted by Veterinary Epidemiology Division (VED) of NVRQS in collaboration with provincial veterinary services.

*Epidemiologically related farms were found as 3 farms and all cervid at these farms were culled and subjected to CWD diagnosis.

*Three elks and 5 crossbreeds (Red deer and Sika deer) were confirmed as positive at farm 2.

All cervids at Farm 3 and Farm 4 - 15 elks and 47 elks - were culled and confirmed as negative.

Further epidemiological investigations showed that these CWD outbreaks were linked to the importation of elks from Canada in 1994 based on circumstantial evidences.

*In December 2010, one elk was confirmed as positive at Farm 5.

*Consequently, all cervid - 3 elks, 11 Manchurian Sika deer and 20 Sika deer - were culled and one Manchurian Sika deer and seven Sika deer were found to be positive.

This is the first report of CWD in these sub-species of deer.

*Epidemiological investigations found that the owner of the Farm 2 in CWD outbreaks in July 2010 had co-owned the Farm 5.

*In addition, it was newly revealed that one positive elk was introduced from Farm 6 of Jinju-si Gyeongsang Namdo.

All cervid - 19 elks, 15 crossbreed (species unknown) and 64 Sika deer - of Farm 6 were culled, but all confirmed as negative.

: Corresponding author: Dr. Hyun-Joo Sohn (+82-31-467-1867, E-mail: **shonhj@korea.kr**) 2011 Pre-congress Workshop: TSEs in animals and their environment 5

http://www.prion2011.ca/files/2011TSEBookletV6Final.pdf

http://www.prion2011.ca/files/PRION_2011_-_Posters_(May_5-11).pdf

http://usdavskorea.blogspot.com/

http://chronic-wasting-disease.blogspot.com/2012/06/natural-cases-of-cwd-in-eight-sika-deer.html

Friday, May 13, 2011

Chronic Wasting Disease (CWD) outbreaks and surveillance program in the Republic of Korea

http://chronic-wasting-disease.blogspot.com/2011/05/chronic-wasting-disease-cwd-outbreaks.html

MONDAY, MARCH 05, 2018

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

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

to date. there is no cervid that has been documented to be totally resistant to cwd tse prion.

***at present. no cervid PrP allele conferring absolute resistance to prion infection has been identified.

P-145 Estimating chronic wasting disease resistance in cervids using real time quaking- induced conversion

Nicholas J Haley1, Rachel Rielinger2, Kristen A Davenport3, W. David Walter4, Katherine I O'Rourke5, Gordon Mitchell6, Juergen A Richt2 1 Department of Microbiology and Immunology, Midwestern University, United States; 2Department of Diagnostic Medicine and Pathobiology, Kansas State University; 3Prion Research Center; Colorado State University; 4U.S. Geological Survey, Pennsylvania Cooperative Fish and Wildlife Research Unit; 5Agricultural Research Service, United States Department of Agriculture; 6Canadian Food Inspection Agency, National and OIE Reference Laboratory for Scrapie and CWD

In mammalian species, the susceptibility to prion diseases is affected, in part, by the sequence of the host's prion protein (PrP). In sheep, a gradation from scrapie susceptible to resistant has been established both in vivo and in vitro based on the amino acids present at PrP positions 136, 154, and 171, which has led to global breeding programs to reduce the prevalence of scrapie in domestic sheep. In cervids, resistance is commonly characterized as a delayed progression of chronic wasting disease (CWD); at present, no cervid PrP allele conferring absolute resistance to prion infection has been identified. To model the susceptibility of various naturally-occurring and hypothetical cervid PrP alleles in vitro, we compared the amplification rates and efficiency of various CWD isolates in recombinant PrPC using real time quaking-induced conversion. We hypothesized that amplification metrics of these isolates in cervid PrP substrates would correlate to in vivo susceptibility - allowing susceptibility prediction for alleles found at 10 frequency in nature, and that there would be an additive effect of multiple resistant codons in hypothetical alleles. Our studies demonstrate that in vitro amplification metrics predict in vivo susceptibility, and that alleles with multiple codons, each influencing resistance independently, do not necessarily contribute additively to resistance. Importantly, we found that the white-tailed deer 226K substrate exhibited the slowest amplification rate among those evaluated, suggesting that further investigation of this allele and its resistance in vivo are warranted to determine if absolute resistance to CWD is possible.

***at present, no cervid PrP allele conferring absolute resistance to prion infection has been identified.

PRION 2016 CONFERENCE TOKYO

http://prion2016.org/dl/newsletter_03.pdf

"There are no known familial or genetic TSEs of animals, although polymorphisms in the PRNP gene of some species (sheep for example) may influence the length of the incubation period and occurrence of disease."

c) The commonest form of CJD occurs as a sporadic disease, the cause of which is unknown, although genetic factors (particularly the codon 129 polymorphism in the prion protein gene (PRNP)) influence disease susceptibility. The familial forms of human TSEs (see Box 1) appear to have a solely genetic origin and are closely associated with mutations or insertions in the PRNP gene. Most, but not all, of the familial forms of human TSEs have been transmitted experimentally to animals. There are no known familial or genetic TSEs of animals, although polymorphisms in the PRNP gene of some species (sheep for example) may influence the length of the incubation period and occurrence of disease.

https://www.gov.uk/government/uploa...nt_data/file/209755/Part_1_-_Introduction.pdf

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https://www.gov.uk/government/uploa...nt_data/file/209755/Part_1_-_Introduction.pdf

Subject: cwd genetic susceptibility

Genetic susceptibility to chronic wasting disease in free-ranging white-tailed deer: Complement component C1q and Prnp polymorphisms§

Julie A. Blanchong a, *, Dennis M. Heisey b , Kim T. Scribner c , Scot V. Libants d , Chad Johnson e , Judd M. Aiken e , Julia A. Langenberg f , Michael D. Samuel g

snip...

Identifying the genetic basis for heterogeneity in disease susceptibility or progression can improve our understanding of individual variation in disease susceptibility in both free-ranging and captive populations. What this individual variation in disease susceptibility means for the trajectory of disease in a population, however, is not straightforward. For example, the greater, but not complete, resistance to CWD in deer with at least one Serine (S) at amino acid 96 of the Prnp gene appears to be associated with slower progression of disease (e.g., Johnson et al., 2006; Keane et al., 2008a). If slower disease progression results in longer-lived, infected deer with longer periods of infectiousness, resistance may lead to increased disease transmission rates, higher prion concentrations in the environment, and increased prevalence, as has been observed in some captive deer herds (Miller et al., 2006; Keane et al., 2008a). Alternatively, if the slower progression of disease in resistant deer is not associated with longer periods of infectiousness, but might instead indicate a higher dose of PrPCWD is required for infection, transmission rates in the population could decline especially if, as in Wisconsin, deer suffer high rates of mortality from other sources (e.g., hunting). Clearly, determining the relationship between genetic susceptibility to infection, dose requirements, disease progression, and the period of PrPCWD infectiousness are key components for understanding the consequences of CWD to free-ranging populations.

http://
http://forest.wisc.edu/files/pdfs/samuel/2009%20blanchong%20et%20al%20genetic%20susceptibility%20chronic%20wasting.pdf

http://lib.dr.iastate.edu/cgi/viewcontent.cgi?article=1083&context=nrem_pubs

http://onlinelibrary.wiley.com/doi/10.2903/j.efsa.2017.4667/epdf

http://www.tandfonline.com/doi/full/10.1080/19336896.2015.1115179

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4964855/pdf/kprn-09-06-1115179.pdf

http://www.sciencedirect.com/science/article/pii/S1567134809001956?via=ihub

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4964855/

December 2014, Volume 36, Issue 6, pp 1049–1061 | Cite as

Mineral licks: motivational factors for visitation and accompanying disease risk at communal use sites of elk and deer

Authors Authors and affiliations Michael J. LavelleEmail authorGregory E. PhillipsJustin W. FischerPatrick W. BurkeNathan W. SewardRandal S. StahlTracy A. NicholsBruce A. WunderKurt C. VerCauteren 1. 2. 3. 4. Article First Online: 08 April 2014 258 Downloads 1 Citations

Abstract

Free-ranging cervids acquire most of their essential minerals through forage consumption, though occasionally seek other sources to account for seasonal mineral deficiencies. Mineral sources occur as natural geological deposits (i.e., licks) or as

anthropogenic mineral supplements. In both scenarios, these sources commonly serve as focal sites for visitation. We monitored 11 licks in Rocky Mountain National Park, north-central Colorado, using trail cameras to quantify daily visitation indices (DVI) and soil consumption indices (SCI) for Rocky Mountain elk (Cervus elaphus) and mule deer (Odocoileus hemionus) during summer 2006 and documented elk, mule deer, and moose (Alces alces) visiting licks. Additionally, soil samples were collected, and mineral concentrations were compared to discern levels that explain rates of visitation. Relationships between response variables; DVI and SCI, and explanatory variables; elevation class, moisture class, period of study, and concentrations of minerals were examined. We found that DVI and SCI were greatest at two wet, low-elevation licks exhibiting relatively high concentrations of manganese and sodium. Because cervids are known to seek Na from soils, we suggest our observed association of Mn with DVI and SCI was a likely consequence of deer and elk seeking supplemental dietary Na. Additionally, highly utilized licks such as these provide an area of concentrated cervid occupation and interaction, thus increasing risk for environmental transmission of infectious pathogens such as chronic wasting disease, which has been shown to be shed in the saliva, urine, and feces of infected cervids.

Keywords Cervus elaphus Chronic wasting disease Elk Geophagy Mineral lick Mule deer Odocoileus hemionus

https://rd.springer.com/article/10.1007/s10653-014-9600-0

Elk and Deer Use of Mineral Licks: Implications for Disease Transmission

Kurt C. VerCauteren1*, Michael J. Lavelle1, Gregory E. Phillips1, Justin W. Fischer1, and Randal S. Stahl1 1United States Department of Agriculture, Animal and Plant Health Inspection Service, Wildlife Services, National Wildlife Research Center, 4101 LaPorte Avenue, Fort Collins, CO 80521-2154, USA *Cooresponding author e-mail: **kurt.c.vercauteren@aphis.usda.gov**

North American cervids require and actively seek out minerals to satisfy physiological requirements. Minerals required by free-ranging cervids exist within natural and artificial mineral licks that commonly serve as focal sites for cervids. Ingestion of soils contaminated with the agent that causes chronic wasting disease (CWD) may result in risk of contracting CWD. Our objective was to evaluate the extent and nature of use of mineral licks by CWD-susceptible cervid species. We used animal-activated cameras to monitor use of 18 mineral licks between 1 June and 16 October 2006 in Rocky Mountain National Park, north-central Colorado. We also assessed mineral concentrations at mineral licks to evaluate correlations between visitation rates and site-specific characteristics. We collected > 400,000 images of which 991 included elk, 293 included deer, and 6 included moose. We documented elk and deer participating in a variety of potentially risky behaviors (e.g., ingesting soil, ingesting water, defecating, urinating) while at mineral licks. Results from the mineral analyses combined with camera data revealed that visitation was highest at sodium-rich mineral licks. Mineral licks may play a role in disease transmission by acting as sites of increased interaction as well as reservoirs for deposition, accumulation, and ingestion of disease agents.

http://www.cwd-info.org/pdf/3rd_CWD_Symposium_utah.pdf

http://chronic-wasting-disease.blogspot.com/2009/08/third-international-cwd-symposium-july.html

what does sound science and the prion Gods say…

Sunday, January 06, 2013

USDA TO PGC ONCE CAPTIVES ESCAPE

*** "it's no longer its business."

http://chronic-wasting-disease.blogspot.com/2013/01/usda-to-pgc-once-captives-escape-its-no.html

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

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

SHOOTING PENS (HIGH/LOW FENCE), CAPTIVE CERVID FARMING, BREEDING, SPERM MILLS, ANTLER MILLS, URINE MILLS, a petri dish for cwd tse prion disease…

*** Spraker suggested an interesting explanation for the occurrence of CWD. The deer pens at the Foot Hills Campus were built some 30-40 years ago by a Dr. Bob Davis. At or abut that time, allegedly, some scrapie work was conducted at this site. When deer were introduced to the pens they occupied ground that had previously been occupied by sheep.

https://web.archive.org/web/20170126060744/http://collections.europarchive.org/tna/20080102193705/http://www.bseinquiry.gov

COLORADO THE ORIGIN OF CHRONIC WASTING DISEASE CWD TSE PRION?

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IN CONFIDENCE, REPORT OF AN UNCONVENTIONAL SLOW VIRUS DISEASE IN ANIMALS IN THE USA 1989

http://webarchive.nationalarchives.gov.uk/20080102193705/http://www.bseinquiry.gov.uk/files/mb/m11b/tab01.pdf

THURSDAY, APRIL 19, 2018

Theodore Roosevelt Conservation Partnership Chronic Wasting

Disease CWD What You Can Do!

http://chronic-wasting-disease.blogspot.com/2018/04/theodore-roosevelt-conservation.html

THURSDAY, APRIL 05, 2018

Boone and Crocket Club B&C News Release CHRONIC WASTING DISEASE TSE Prion

http://chronic-wasting-disease.blogspot.com/2018/04/boone-and-crocket-club-b-news-release.html

SUNDAY, APRIL 8, 2018

Transmissible Spongiform Encephalopathy TSE Prion Disease Global Pandemic Urgent Update April 9, 2018

http://transmissiblespongiformencephalopathy.blogspot.com/2018/04/transmissible-spongiform-encephalopathy.html

TUESDAY, APRIL 10, 2018

Animal TSEs and public health: What remains of past lessons?

http://chronic-wasting-disease.blogspot.com/2018/04/animal-tses-and-public-health-what.html

SATURDAY, MARCH 10, 2018

Chronic Wasting Disease CWD TSE Prion Goes Global Finland Falls, Behind Norway and S. Korea

FINLAND REPORTS FIRST CASE OF CHRONIC WASTING DISEASE CWD TSE PRION IN A moose or European elk (Alces alces)

http://chronic-wasting-disease.blogspot.com/2018/03/finland-reports-first-case-of-chronic.html

WEDNESDAY, MARCH 28, 2018

The executioner in Nordfjella and Chronic Wasting Disease CWD TSE Prion Skrantesjuke

http://chronic-wasting-disease.blogspot.com/2018/03/the-executioner-in-nordfjella-and.html

TUESDAY, FEBRUARY 27, 2018

NORWAY CWD TSE PRION Skrantesjuke Nordfjella zone 1 Complete Eradication Complete

http://chronic-wasting-disease.blogspot.com/2018/02/norway-cwd-tse-prion-skrantesjuke.html

WEDNESDAY, MARCH 21, 2018

World Animal Organization (OIE) Appoints Veterinary Institute as first European reference laboratory for land animal health field of CWD or skrantesjuke scratch disease

http://chronic-wasting-disease.blogspot.com/2018/03/world-animal-organization-oie-appoints.html

THURSDAY, APRIL 19, 2018

Wisconsin CWD detection in a wild deer in Eau Claire County will result in a renewal of the baiting and feeding ban

http://chronic-wasting-disease.blogspot.com/2018/04/wisconsin-cwd-detection-in-wild-deer-in.html

FRIDAY, APRIL 13, 2018

WISCONSIN DATCP Resolutions target spread of chronic wasting disease depopulation of Copper Hills Hunting Preserve near Oulu has begun

http://chronic-wasting-disease.blogspot.com/2018/04/wisconsin-datcp-resolutions-target.html

Wisconsin Ag News Headlines

Rep. Milroy Wants More Action to Combat CWD in Wisconsin

Wisconsin Ag Connection - 03/26/2018

A member of the state Assembly's Natural Resources Committee is calling on members of Governor Scott Walker's cabinet to do more to fight the spread of Chronic Wasting Disease on Wisconsin's deer farms. In a letter to State Agriculture Secretary Sheila Harsdorf, Rep. Nick Milroy said he is concerned about the recent increase of captive deer and deer farms that have tested positive for CWD.

"It was reported that DATCP has placed the entire state under quarantine due to Emerald Ash Borer. We don't want to get to that point with deer," Milroy said. "Immediate action must be taken to identify, cull, and test any animal that has been transported prior to being placed under quarantine to any non-quarantined farm."

The South Range Democrat adds that if any of these farms are located outside of Wisconsin, the respective agencies from those states must be notified as soon as possible.

"We know that deer can be infected with CWD for years before showing signs of the disease," he said. "This is why it is so critical to test any deer that have been transported off of these farms even if the transfer occurred prior to being placed under quarantine. It is imperative that we get a handle on the spread of this disease between captive operations to protect private investments, taxpayers, and our wild deer herd."

Milroy wants DATCP to provide his office with data for the past five years as to how many deer have been transferred from farms that subsequently were quarantined for CWD and where those deer were transferred.

He has also written similar requests over the past month to Walker's office and DNR Secretary Dan Meyer.

http://www.wisconsinagconnection.com/story-state.php?Id=353&yr=2018

SATURDAY, MARCH 03, 2018

WISCONSIN CHRONIC WASTING DISEASE TSE Prion DNR Study Finds CWD-Infected Deer Die At 3 Times Rate Of Healthy Animals

http://chronic-wasting-disease.blogspot.com/2018/03/wisconsin-chronic-wasting-disease-tse.html

FRIDAY, FEBRUARY 16, 2018

Wisconsin Deer from Now-Quarantined PA Lancaster County Farm Tests Positive for Chronic Wasting Disease CWD TSE Prion

http://chronic-wasting-disease.blogspot.com/2018/02/wisconsin-deer-from-now-quarantined-pa.html

FRIDAY, JANUARY 26, 2018

WISCONSIN REPORTS 588 CWD TSE PRION POSITIVE CASES FOR 2017 WITH 4170 CASES CONFIRMED TO DATE

http://chronic-wasting-disease.blogspot.com/2018/01/wisconsin-reports-588-cwd-tse-prion.html

USA MAD DEER ROUNDUP

Feb. 16, 2018

Durkin: Stop private deer industry from trucking CWD across state

Patrick Durkin, For USA TODAY NETWORK-Wisconsin Published 10:13 a.m. CT Feb. 16, 2018

A Waupaca County captive-deer shooting preserve that discovered its first two cases of chronic wasting disease in October found 10 more CWD cases last fall, with 11 of the deer coming from a breeding facility in Iowa County — Wisconsin's most infected county.

Hunt's End Deer Ranch near Ogdensburg is one of 376 fenced deer farms in Wisconsin, according to the Department of Agriculture, Trade and Consumer Protection. Hunt's End bought the diseased deer from Windy Ridge Whitetails, a 15-acre, 110-deer breeding facility south of Mineral Point in Iowa County. Of Wisconsin's 4,175 CWD cases in wild deer, 2,261 (54 percent) are in Iowa County.

Since CWD's discovery in three wild deer shot during the November 2001 gun season, CWD has been detected on 18 Wisconsin deer farms, of which 11 were "depopulated." DATCP has identified 242 CWD cases in captive facilities the past 16 years.

The state's worst site remains the former Buckhorn Flats Game Farm near Almond in Portage County, where 80 deer tested positive for this always-fatal disease from 2002 to 2006. When the U.S. Department of Agriculture shot out the 70-acre pen in January 2006, 60 of the remaining 76 deer carried CWD, a nearly 80 percent infection rate.

The Department of Natural Resources bought the heavily contaminated site for $465,000 in 2011 and has kept it fenced and deer-free since.

The last time DATCP exterminated a captive herd was November 2015, when it killed 228 deer at Fairchild Whitetails, a 10-acre breeding facility in Eau Claire County, and paid its owner, Richard Vojtik, $298,770 in compensation. Tests revealed 34 of those deer carried CWD (15 percent), but two bucks had escaped earlier. Those bucks roamed five months before being shot and tested. They, too, had CWD.

Both operations were outside the endemic CWD region in southern Wisconsin; Buckhorn Flats by about 60 miles and Fairchild Whitetails by about 120. Wisconsin's four most active CWD outbreaks on deer farms are north of U.S. 10, and farther away from the endemic region — basically the DNR's Southern Farmlands district — which had 584 CWD cases 2017-18 and 4,148 since 2001.

Those businesses are:

• Wilderness Whitetails, near Eland in Marathon County: 68 CWD cases, including 43 in 2017-18. DATCP first reported CWD there in December 2013 in a 5-year-old buck shot by a facility client. The operation also found three cases in 2014, nine in 2015 and 12 in 2016.

The preserve held about 310 deer in its 351-acre pen last summer. Since beginning tests in 2002, the facility tested 373 deer before finding its first case 11 years later.

• Hunt's End, Waupaca County: 12 cases, all in 2017-18. The owners, Dusty and Mandy Reid, didn't detect CWD on the 84-acre shooting facility until two 4-year-old bucks tested positive last fall. DATCP announced those cases Oct. 20, and disclosed 10 additional cases in response to my open-records request in January.

Both Oct. 20 bucks originated from Windy Ridge Whitetails. Nine other bucks from Windy Ridge, owned by Steven and Marsh Bertram, tested positive for CWD after being shot by Hunt's End clients.

Now DATCP records covering the past five years showed Hunt's End acquired 31 deer from Windy Ridge, which also sent a combined 67 whitetails to nine other Wisconsin deer farms during that period.

Paul McGraw, DATCP's state veterinarian and administrator in animal health, quarantined three Hunt's End properties Oct. 20, but let its owners, continue selling hunts because "properly handled dead animals leaving the premises do not pose a disease risk."

McGraw also quarantined Windy Ridge, but the specifications let the

business move more deer to the Waupaca shooting facility. It made two more shipments to Hunt's End, the last occurring Nov. 13.

• Apple Creek Whitetails, Oconto County: 11 cases. Since discovering CWD in September 2016 in an 18-month-old doe killed inside the facility near Gillett, DATCP has identified 10 more cases, including three in 2017-18. The preserve held about 1,850 deer on 1,363 acres, and tested 466 in 2016. After first testing for CWD in 2009, the business processed 1,192 deer before finding its first case 18 months ago.

• Three Lakes Trophy Ranch, Oneida County: Nine cases. Since discovering CWD in December 2015 in a 3-year-old buck at Three Lakes, DATCP has identified eight more cases, including two in 2017-18. The preserve held about 545 whitetails on 570 acres.

Although the Hunt's End outbreak traces to Iowa County deer, Windy Ridge Whitetails sent even more deer, 42, to Vojtik's American Adventures Ranch near Fairchild with no documented problems. DATCP reports no CWD cases there, and Vojtik, who also owned the 10-acre Fairchild Whitetails breeding facility, said he hasn't bought Windy Ridge deer the past two years.

Vojtik said Wednesday that he and his clients shoot out his enclosure's herd of about 200 deer each year to reduce CWD risks. And because he's not in DATCP's herd-status program, he must only test 50 percent of deer dying there.

Meanwhile, Wilderness Whitetails tests all of its dead deer. It leads the state with 68 CWD cases, even though it has maintained a "closed herd" since opening its Eland facility in 2004, said its owner, Greg Flees, when reached Wednesday. Flees said all deer in the 351-acre facility were born there or came from his family's Portage County breeding pen, which began in the 1970s and has never had CWD.

Flees said the jump from 12 CWD cases in 2016 to 43 in 2017 is no mystery or surprise. "We shot more deer to lower our densities, so we found more CWD," he said. He thinks CWD was in the facility's soils when they enclosed it with an 8-foot-high fence 14 years ago, or it arrived in alfalfa bales brought in for feed.

Perhaps the bigger mystery is why DATCP allows any deer from Iowa County to be shipped anywhere. Windy Ridge Whitetails is one of eight captive-deer facilities in CWD-infected counties — Sauk, Dane, Iowa, Rock, Walworth and Richland — enrolled in DATCP's herd-status program, which allows deer transfers if facilities follow specified guidelines.

That won't change soon, either. In a letter Jan. 30 responding to my open records request, Paul Dedinsky, DATCP's chief legal counsel, wrote, "The Department is not proposing any rule changes to prohibit movement from CWD endemic areas."

No doubt Wisconsin's wild deer provide a vast, mostly undocumented pool for spreading CWD, but sick deer can only carry disease as far as they walk. With DATCP's approval, privately owned deer could spread CWD wherever they're trucked.

Patrick Durkin is a freelance writer who covers outdoors for USA TODAY NETWORK-Wisconsin. Email him at **patrickdurkin56@gmail.com**.

https://www.greenbaypressgazette.com/story/sports/outdoors/2018/02/16/durkin-stop-private-deer-industry-trucking-cwd-across-state/342532002/

FRIDAY, FEBRUARY 16, 2018

Wisconsin Stop private deer industry from trucking CWD across state

https://www.greenbaypressgazette.com/story/sports/outdoors/2018/02/16/durkin-stop-private-deer-industry-trucking-cwd-across-state/342532002/

Tuesday, December 20, 2011

CHRONIC WASTING DISEASE CWD WISCONSIN Almond Deer (Buckhorn Flats) Farm Update DECEMBER 2011

The CWD infection rate was nearly 80%, the highest ever in a North American captive

herd. RECOMMENDATION: That the Board approve the purchase of 80 acres of land for $465,000 for the Statewide Wildlife Habitat Program in Portage County and approve the restrictions on public use of the site.

SUMMARY:

http://dnr.wi.gov/about/nrb/2011/december/12-11-2b2.pdf

***>captive deer farmers breeders entitlement program, i.e. indemnity program, why?

how many states have $465,000., and can quarantine and purchase there from, each cwd said infected farm, but how many states can afford this for all the cwd infected cervid game ranch type farms, and why do tax payers have to pay for it ???

MONDAY, MARCH 26, 2018

Wisconsin Rep. Milroy Wants More Action to Combat CWD TSE Prion aka Mad Deer Disease

http://chronic-wasting-disease.blogspot.com/2018/03/wisconsin-rep-milroy-wants-more-action.html

WEDNESDAY, MARCH 07, 2018

Michigan DNR CWD National Perspective: Captive Herd Certification Program - Dr. Tracy Nichols

CURRENT STATUS OF CWD IN CAPTIVE CERVID HERDS IN 16 STATES AS OF MAY 2017

43 ELK HERDS

37 WTD HERDS

1 RED DEER HERD

6 MIX SPECIES HERDS

85 CWD-POSITIVE CAPTIVE HERDS

snip...see

http://chronic-wasting-disease.blogspot.com/2018/03/michigan-dnr-cwd-national-perspective.html

January 14, 2018

Michigan's Chronic Wasting Disease Working Group Recommendations Report to the Natural Resources Commission Prepared December 2017 CWD Confirmed Cases holding for now at 57 cases

http://www.michigan.gov/emergingdiseases/0,4579,7-186-81018_25806-357110--,00.html

http://chronic-wasting-disease.blogspot.com/2018/01/michigans-chronic-wasting-disease.html

INFORM: Cervid Health and States Indemnity FY 2015

USDA Animal and Plant Health Inspection Service sent this bulletin at 09/19/2014 05:22 PM EDT

Animal and Plant Health Inspection Service (APHIS), Veterinary Services (VS) received a total of $3 million in appropriated funding to support cervid health activities in fiscal year (FY) 2014, and made approximately $1.0 million of this funding available for indemnity of chronic wasting disease (CWD) positive, suspect, and exposed farmed cervids. All of the available FY2014 indemnity funding was used to depopulate three CWD-infected herds. However, several States have asked about the availability of Federal indemnity funds for CWD-exposed animals in the future.

VS plans to offer Federal indemnity for CWD-exposed cervids beginning in FY2015. Briefly, we will prioritize the highest risk CWD-exposed animals for indemnity based on the availability of funding. Any newly reported CWD-positive herds will be considered for indemnity as they are identified, based first on funding availability and secondly on the risk presented by the herd.

We will reassess our fiscal year funding on a quarterly basis so that providing indemnity for exposed animals does not exhaust available funding early in the fiscal year. By taking this fiscally cautious approach, we hope to provide indemnity for positive herds identified later in the fiscal year while removing high-risk animals from the landscape as soon as possible to minimize the risk for disease spread. Further, removal and testing of these exposed animals will provide a better understanding of the disease risk presented by these animals/herds.

VS plans to work with our State and industry stakeholders on the criteria to assess the risk and on the process through which States can request this indemnity. These will be finalized in a VS Guidance Document in the near future. We look forward to working with you to implement this process in the coming year.

***

http://content.govdelivery.com/accounts/USDAAPHIS/bulletins/d05806

SATURDAY, APRIL 21, 2018

MINNESOTA STATE AUDITORS Board of Animal Health has failed to enforce some laws relating to deer and elk farms A CWD TSE PRION GLOBAL UPDATE

http://chronic-wasting-disease.blogspot.com/2018/04/minnesota-state-auditors-board-of.html

SATURDAY, MARCH 03, 2018

Minnesota CWD All seven of the remaining white-tailed deer on farm Positive

http://chronic-wasting-disease.blogspot.com/2018/03/minnesota-cwd-all-seven-of-remaining.html

FRIDAY, APRIL 20, 2018

Use of environmental sites by mule deer: a proxy for relative risk of chronic wasting disease exposure and transmission

http://chronic-wasting-disease.blogspot.com/2018/04/use-of-environmental-sites-by-mule-deer.html

TUESDAY, DECEMBER 12, 2017

Creutzfeldt Jakob Disease CJD National Prion Disease Pathology Surveillance Center Cases Examined to December 14, 2017

http://creutzfeldt-jakob-disease.blogspot.com/2017/12/creutzfeldt-jakob-disease-cjd-national.html

Tuesday, December 12, 2017

Neuropathology of iatrogenic Creutzfeldt–Jakob disease and immunoassay of French cadaver-sourced growth hormone batches suggest possible transmission of tauopathy and long incubation periods for the transmission of Abeta pathology

http://tauopathies.blogspot.com/2017/12/neuropathology-of-iatrogenic.html

MONDAY, OCTOBER 02, 2017

Creutzfeldt Jakob Disease United States of America USA and United Kingdom UK Increasing and Zoonotic Pontential From Different Species

http://creutzfeldt-jakob-disease.blogspot.com/2017/10/creutzfeldt-jakob-disease-united-states.html

THURSDAY, AUGUST 17, 2017

*** Monitoring the occurrence of emerging forms of Creutzfeldt-Jakob disease in the United States revisited 2017

Singeltary et al

http://creutzfeldt-jakob-disease.blogspot.com/2017/08/monitoring-occurrence-of-emerging-forms.html

Tuesday, March 20, 2018

Variably protease-sensitive prionopathy (VPSPr), sporadic creutzfeldt jakob disease sCJD, the same disease, what if?

http://vpspr.blogspot.com/2018/03/variably-protease-sensitive-prionopathy.html

TUESDAY, MARCH 06, 2018

ZOONOSIS OF CHRONIC WASTING DISEASE CWD TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHY TSE PRION DISEASE, who makes the final call?

http://chronic-wasting-disease.blogspot.com/2018/03/zoonosis-of-chronic-wasting-disease-cwd.html

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

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

Tracking spongiform encephalopathies in North America

Xavier Bosch

Published: August 2003

DOI: http://dx.doi.org/10.1016/S1473-3099(03)00715-1

Summary;

"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://infection.thelancet.com/

http://www.thelancet.com/pdfs/journals/laninf/PIIS1473-3099(03)00715-1.pdf

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://www.neurology.org/content/60/2/176/reply#neurology_el_535

***> 2001 FDA CJD TSE Prion Singeltary Submission

http://www.fda.gov/ohrms/dockets/ac/01/slides/3681s2_09.pdf

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

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

2 January 2000 British Medical Journal U.S.

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

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

15 November 1999 British Medical Journal hvCJD in the USA * BSE in U.S.

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

Re-Evidence for human transmission of amyloid-β pathology and cerebral amyloid angiopathy

>>> The only tenable public line will be that "more research is required'' <<<

>>> possibility on a transmissible prion remains open<<<

O.K., so it's about 23 years later, so somebody please tell me, when is "more research is required'' enough time for evaluation ?

Re-Evidence for human transmission of amyloid-β pathology and cerebral amyloid angiopathy

Nature 525, 247?250 (10 September 2015) doi:10.1038/nature15369 Received 26 April 2015 Accepted 14 August 2015 Published online 09 September 2015 Updated online 11 September 2015 Erratum (October, 2015)

snip...see full Singeltary Nature comment here;

Alzheimer's disease

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-β 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

Self-Propagative Replication of Ab Oligomers Suggests Potential Transmissibility in Alzheimer Disease

*** Singeltary comment PLoS ***

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

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

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

wasted days and wasted nights...Freddy Fender

stupid is, as stupid does...Forest Gump

sometimes you can't fix stupid...Terry S. Singeltary Sr.

Terry S. Singeltary Sr.

Bacliff, Texas, USA 77518, Galveston Bay, on the bottom.

POSTED BY TERRY S. SINGELTARY SR. AT 3:53 PM

0 COMMENTS:

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