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



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

Greetings again FDA and Mr. Pritchett et al,

I would kindly like to comment on ;

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

#158

Guidance for Industry

Use of Material from Deer and Elk in Animal Feed

This version of the guidance replaces the version made available September15, 2003.

This document has been revised to update the docket number, contact information, and standard disclosures. Submit comments on this guidance at any time.

Submit electronic comments to <http://www.regulations.gov>. Submit written comments to the Division of Dockets Management (HFA-305), Food and Drug Administration, 5630 Fishers Lane, Rm. 1061, Rockville, MD 20852. All comments should be identified with the Docket No. FDA-2003-D-0432 (formerly 03D-0186).

For further information regarding this guidance, contact Burt Pritchett, Center for Veterinary Medicine (HFV-222), Food and Drug Administration, 7519 Standish Place, Rockville, MD 20855, 240-402-6276, E-mail: burt.pritchett@fda.hhs.gov.

Additional copies of this guidance document may be requested from the Policy and Regulations Staff (HFV-6), Center for Veterinary Medicine, Food and Drug Administration, 7519 Standish Place, Rockville, MD 20855, and may be viewed on the Internet at either <http://www.fda.gov/AnimalVeterinary/default.htm> or <http://www.regulations.gov>.

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

V I E W M Y C O M P L E T E P R O F I L E

P R E V I O U S P O S T S

Michigan confirms additional CWD-positive free-ran...

Wisconsin CWD sample survey 2015 confirms 290 case...

WYOMING RIDE EM COWBOY HELICOPTER WRANGLING RAMBO ...

Arkansas confirms second case of Chronic Wasting D...

Oklahoma Chronic Wasting Disease CWD of Deer and E...

Wyoming Game and Fish Department confirmed chronic...

Missouri 2015-2016 CWD Surveillance Summary to Dat...

Social affiliation and contact patterns among whit...

Kansas Chronic Wasting Disease CWD TSE Prion 53 ca...

TEXAS Hartley County Mule Deer Tests Positive for ...



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Contains Nonbinding Recommendations

2

Guidance for Industry Use of Material from Deer and Elk in Animal Feed

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

I. Introduction

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

In general, FDA's guidance documents do not establish legally enforceable responsibilities. Instead, guidances describe the Agency's current thinking on a topic and should be viewed only as recommendations, unless specific regulatory or statutory requirements are cited. The use of the word should in Agency guidances means that something is suggested or recommended, but not required.

II. Background

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

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

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

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

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

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

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

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

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.

<http://web.archive.org/web/20061003022720/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.

<http://web.archive.org/web/20030526212610/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...

<http://collections.europarchive.org/tna/20090505194948/http://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...

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>

please see further ;

REFERENCE MATERIALS

*** 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>

*** 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 about 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.

<http://collections.europarchive.org/tna/20080102193705/http://www.bseinquiry.gov.uk/files/mb/m11b/tab01.pdf>

PL1

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 self-propagate to spread disease between cells, organs and in some cases across individuals. In Transmissible spongiform encephalopathies (TSEs), prions are mostly composed by a misfolded form of the prion protein (PrP^{Sc}), which propagates by transmitting its misfolding to the normal prion protein (PrP^C). 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 PrP^{Sc} and propagate prions that maintain high infectivity, strain properties and species specificity. Using PMCA we have been able to detect PrP^{Sc} 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 (A β) and α -synuclein (α -syn) aggregates implicated in Alzheimer's and Parkinson's diseases, respectively. Experiments are ongoing to study the utility of this technology to detect A β and α -syn aggregates in samples of CSF and blood from patients affected by these diseases.

***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.

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>

see ;

<http://www.tandfonline.com/doi/pdf/10.4161/pri.28467>

<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](http://www.cell.com/cell-reports/pdfExtended/S2211-1247(15)00437-4)

Wednesday, December 16, 2015

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

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

Timm Konold^{1*}, Stephen A. C. Hawkins², Lisa C. Thurston³, Ben C. Maddison⁴, Kevin C. Gough⁵, Anthony Duarte¹ and Hugh A. Simmons¹

¹ Animal Sciences Unit, Animal and Plant Health Agency Weybridge, Addlestone, UK, ² Pathology Department, Animal and Plant Health Agency Weybridge, Addlestone, UK, ³ Surveillance and Laboratory Services, Animal and Plant Health Agency Penrith, Penrith, UK, ⁴ ADAS UK, School of Veterinary Medicine and Science, University of Nottingham, Sutton Bonington, UK, ⁵ School of Veterinary Medicine and Science, University of Nottingham, Sutton Bonington, UK

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

snip...

Discussion

Classical scrapie is an environmentally transmissible disease because it has been reported in naïve, supposedly previously unexposed sheep placed in pastures formerly occupied by scrapie-infected sheep (4, 19, 20). Although the vector for disease transmission is not known, soil is likely to be an important reservoir for prions (2) where – based on studies in rodents – prions can adhere to minerals as a biologically active form (21) and remain infectious for more than 2 years (22). Similarly, chronic wasting disease (CWD) has re-occurred in mule deer housed in paddocks used by infected deer 2 years earlier, which was assumed to be through foraging and soil consumption (23).

Our study suggested that the risk of acquiring scrapie infection was greater through exposure to contaminated wooden, plastic, and metal surfaces via water or food troughs, fencing, and hurdles than through grazing. Drinking from a water trough used by the scrapie flock was sufficient to cause infection in sheep in a clean building. Exposure to fences and other objects used for rubbing also led to infection, which supported the hypothesis that skin may be a vector for disease transmission (9). The risk of these objects to cause infection was further demonstrated when 87% of 23 sheep presented with PrPSc in lymphoid tissue after grazing on one of the paddocks, which contained metal hurdles, a metal lamb creep and a water trough in contact with the scrapie flock up to 8 weeks earlier, whereas no infection had been demonstrated previously in sheep grazing on this paddock, when equipped with new fencing and field furniture. When the contaminated furniture and fencing were removed, the infection rate dropped significantly to 8% of 12 sheep, with soil of the paddock as the most likely source of infection caused by shedding of prions from the scrapie-infected sheep in this paddock up to a week earlier.

This study also indicated that the level of contamination of field furniture sufficient to cause infection was dependent on two factors: stage of incubation period and time of last use by scrapie-infected sheep. Drinking from a water trough that had been used by scrapie sheep in the predominantly pre-clinical phase did not appear to cause infection, whereas infection was shown in sheep drinking from the water trough used by scrapie sheep in the later stage of the disease. It is possible that contamination occurred through shedding of prions in saliva, which may have contaminated the surface of the water trough and subsequently the water when it was refilled. Contamination appeared to be sufficient to cause infection only if the trough was in contact with sheep that included clinical cases. Indeed, there is an increased risk of bodily fluid infectivity with disease progression in scrapie (24) and CWD (25) based on PrPSc detection by sPMCA. Although ultraviolet light and heat under natural conditions do not inactivate prions (26), furniture in contact with the scrapie flock, which was assumed to be sufficiently contaminated to cause infection, did not act as vector for disease if not used for 18 months, which suggest that the weathering process alone was sufficient to inactivate prions.

PrPSc detection by sPMCA is increasingly used as a surrogate for infectivity measurements by bioassay in sheep or mice. In this reported study, however, the levels of PrPSc present in the environment were below the limit of detection of the sPMCA method, yet were still sufficient to cause infection of in-contact animals. In the present study, the outdoor objects were removed from the infected flock 8 weeks prior to sampling and were positive by sPMCA at very low levels (2 out of 37 reactions). As this sPMCA assay also yielded 2 positive reactions out of 139 in samples from the scrapie-free farm, the sPMCA assay could not detect PrPSc on any of the objects above the background of the assay. False positive reactions with sPMCA at a low frequency associated with de novo formation of infectious prions have been reported (27, 28). This is in contrast to our previous study where we demonstrated that outdoor objects that had been in contact with the scrapie-infected flock up to 20 days prior to sampling harbored PrPSc that was detectable by sPMCA analysis [4 out of 15 reactions (12)] and was significantly more positive by the assay compared to analogous samples from the scrapie-free farm. This discrepancy could be due to the use of a different sPMCA substrate between the studies that may alter the efficiency of amplification of the environmental PrPSc. In addition, the present study had a longer timeframe between the objects being in contact with the infected flock and sampling, which may affect the levels of extractable PrPSc. Alternatively, there may be potentially patchy contamination of this furniture with PrPSc, which may have been missed by swabbing. The failure of sPMCA to detect CWD-associated PrP in saliva from clinically affected deer despite confirmation of infectivity in saliva-inoculated transgenic mice was associated with as yet unidentified inhibitors in saliva (29), and it is possible that the sensitivity of sPMCA is affected by other substances in the tested material. In addition, sampling of amplifiable PrPSc and subsequent detection by sPMCA may be more difficult from furniture exposed to weather, which is supported by the observation that PrPSc was detected by sPMCA more frequently in indoor than outdoor furniture (12). A recent experimental study has demonstrated that repeated cycles of drying and wetting of prion-contaminated soil, equivalent to what is expected under natural weathering conditions, could reduce PMCA amplification efficiency and extend the incubation period in

hamsters inoculated with soil samples (30). This seems to apply also to this study even though the reduction in infectivity was more dramatic in the sPMCA assays than in the sheep model. Sheep were not kept until clinical end-point, which would have enabled us to compare incubation periods, but the lack of infection in sheep exposed to furniture that had not been in contact with scrapie sheep for a longer time period supports the hypothesis that prion degradation and subsequent loss of infectivity occurs even under natural conditions.

In conclusion, the results in the current study indicate that removal of furniture that had been in contact with scrapie-infected animals should be recommended, particularly since cleaning and decontamination may not effectively remove scrapie infectivity (31), even though infectivity declines considerably if the pasture and the field furniture have not been in contact with scrapie-infected sheep for several months. As sPMCA failed to detect PrP^{Sc} 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>

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

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

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

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

***with features similar to some reported for human cases of

sporadic CJD, albeit requiring fourfold long incubation than BSE. Scrapie, as recently evoked in humanized mice (Cassard, 2014),

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

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

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

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thus questioning the origin of human sporadic cases

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

***Our study demonstrates susceptibility of adult cattle to oral transmission of classical BSE. ***

***our findings suggest that possible transmission risk of H-type BSE to sheep and human. ***

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 (PrP^{Sc}) of typical and atypical BSEs (L-type and H-type) and typical scrapie to convert normal prion protein (PrP^C) from bovine, ovine, and human to proteinase K-resistant PrP^{Sc}-like form (PrP^{Res}) 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 PrP^C in combination with PrP^{Sc} seed from typical and atypical BSE- or typical scrapie-infected brain homogenates from native host species. In the conventional PMCA, the conversion of PrP^C to PrP^{Res} was observed only when the species of PrP^C source and PrP^{Sc} seed matched. However, in the PMCA with supplements (digitonin, synthetic polyA and heparin), both bovine and ovine PrP^C were converted by PrP^{Sc} 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.

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

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

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PRION 2015 CONFERENCE FT. COLLINS CWD RISK FACTORS TO HUMANS

*** LATE-BREAKING ABSTRACTS PRION 2015 CONFERENCE ***

O18

Zoonotic Potential of CWD Prions

Liuting Qing¹, Ignazio Cali^{1,2}, Jue Yuan¹, Shenghai Huang³, Diane Kofskey¹, Pierluigi Gambetti¹, Wenquan Zou¹, Qingzhong Kong¹
¹Case Western Reserve University, Cleveland, Ohio, USA, ²Second University of Naples, Naples, Italy, ³Encore Health Resources, Houston, Texas, USA

*** These results indicate that the CWD prion has the potential to infect human CNS and peripheral lymphoid tissues and that there might be asymptomatic human carriers of CWD infection.

=====

***These results indicate that the CWD prion has the potential to infect human CNS and peripheral lymphoid tissues and that there

might be asymptomatic human carriers of CWD infection.***

=====

P.105: RT-QuIC models trans-species prion transmission

Kristen Davenport, Davin Henderson, Candace Mathiason, and
Edward Hoover Prion Research Center; Colorado State University;
Fort Collins, CO USA

Conversely, FSE maintained sufficient BSE characteristics to more
efficiently convert bovine rPrP than feline rPrP. Additionally,
human rPrP was competent for conversion by CWD and fCWD.

***This insinuates that, at the level of protein:protein
interactions, the barrier preventing transmission of CWD to
humans is less robust than previously estimated.

=====

***This insinuates that, at the level of protein:protein
interactions, the barrier preventing transmission of CWD to
humans is less robust than previously estimated.***

=====

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

*** PRICE OF CWD TSE PRION POKER GOES UP 2014 ***

Transmissible Spongiform Encephalopathy TSE PRION update
January 2, 2014

*** chronic wasting disease, there was no absolute barrier to
conversion of the human prion protein.

*** Furthermore, the form of human PrPres produced in this in
vitro assay when seeded with CWD, resembles that found in the
most common human prion disease, namely sCJD of the MM1
subtype.

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

<http://chronic-wasting-disease.blogspot.com/2014/01/molecular-barriers-to-zoonotic.html>

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

<https://www.landesbioscience.com/journals/prion/article/28124/?nocache=112223249>

*** 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

now, let's see what the authors said about this casual link, personal communications years ago. 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>

CJD is so rare in people under age 30, one case in a billion (leaving out medical mishaps), that four cases under 30 is "very high," says Colorado neurologist Bosque. "Then, if you add these other two from Wisconsin [cases in the newspaper], six cases of CJD in people associated with venison is very, very high." Only now, with Mary Riley, there are at least seven, and possibly eight, with Steve, her dining companion. "It's not critical mass that matters," however, Belay says. "One case would do it for me." The chance that two people who know each other would both contract CJD, like the two Wisconsin sportsmen, is so unlikely, experts say, it would happen only once in 140 years.

Given the incubation period for TSEs in humans, it may require another generation to write the final chapter on CWD in Wisconsin. "Does chronic wasting disease pass into humans? We'll be able to answer that in 2022," says Race. Meanwhile, the state has become part of an immense experiment.

https://www.organicconsumers.org/old_articles/madcow/killer123103.php

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 ***

<http://zoomify.uzh.ch:8080/zoomify/videos/video-004/video-004.html>

Envt.07:

Pathological Prion Protein (PrPTSE) in Skeletal Muscles of Farmed and Free Ranging White-Tailed Deer Infected with Chronic Wasting Disease

***The presence and seeding activity of PrPTSE in skeletal muscle from CWD-infected cervids suggests prevention of such tissue in the human diet as a precautionary measure for food safety, pending on further clarification of whether CWD may be transmissible to humans.

<http://www.landesbioscience.com/journals/prion/Prion5-Supp-PrionEnvironment.pdf?nocache=1333529975>

Prions in Skeletal Muscles of Deer with Chronic Wasting Disease
Rachel C. Angers^{1,*}, Shawn R. Browning^{1,*}, Tanya S. Seward²,
Christina J. Sigurdson⁴, Michael W. Miller⁵, Edward A. Hoover⁴,
Glenn C. Telling^{1,2,3}, § snip...

Abstract The emergence of chronic wasting disease (CWD) in deer and elk in an increasingly wide geographic area, as well as the interspecies transmission of bovine spongiform encephalopathy to humans in the form of variant Creutzfeldt Jakob disease, have

raised concerns about the zoonotic potential of CWD. Because meat consumption is the most likely means of exposure, it is important to determine whether skeletal muscle of diseased cervids contains prion infectivity. 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://www.sciencemag.org/content/311/5764/1117.long>

*****CJD REPORT 1994 increased risk for consumption of veal and venison and lamb*****

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

Consumption of venison and veal was much less widespread among both cases and controls. For both of these meats there was evidence of a trend with increasing frequency of consumption being associated with increasing risk of CJD. (not nvCJD, but sporadic CJD...tss)

These associations were largely unchanged when attention was restricted to pairs with data obtained from relatives. ...

Table 9 presents the results of an analysis of these data.

There is **STRONG** evidence of an association between "regular" veal eating and risk of CJD ($p = .0.01$).

Individuals reported to eat veal on average at least once a year appear to be at **13 TIMES THE RISK** of individuals who have never eaten veal.

There is, however, a very wide confidence interval around this estimate. There is no strong evidence that eating veal less than once per year is associated with increased risk of CJD ($p = 0.51$).

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** ($p = 0.04$).

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 ;

<http://collections.europarchive.org/tna/20090505194948/http://bseinquiry.gov.uk/files/yb/1994/08/00004001.pdf>

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 independent 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 independently of the the Department.

The statistical results regarding 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 programme.

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>

*** 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).***

<https://www.landesbioscience.com/journals/prion/article/28124/?nocache=112223249>

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

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

Authors

item Comoy, Emmanuel - item Mikol, Jacqueline - item
Luccantoni-Freire, Sophie - item Correia, Evelyne - item
Lescoutra-Etcheagaray, Nathalie - item Durand, Valérie - item
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Submitted to: Scientific Reports Publication Type: Peer Reviewed
Journal Publication Acceptance Date: May 28, 2015 Publication
Date: June 30, 2015 Citation: Comoy, E.E., Mikol, J., Luccantoni-
Freire, S., Correia, E., Lescoutra-Etcheagaray, N., Durand, V.,
Dehen, C., Andreoletti, O., Casalone, C., Richt, J.A., Greenlee, J.J.,
Baron, T., Benestad, S., Brown, P., Deslys, J. 2015. Transmission of
scrapie prions to primate after an extended silent incubation
period. Scientific Reports. 5:11573.

Interpretive Summary: The transmissible spongiform encephalopathies (also called prion diseases) are fatal neurodegenerative diseases that affect animals and humans. The agent of prion diseases is a misfolded form of the prion protein that is resistant to breakdown by the host cells. Since all mammals express prion protein on the surface of various cells such as neurons, all mammals are, in theory, capable of replicating prion diseases. One example of a prion disease, bovine spongiform encephalopathy (BSE; also called mad cow disease), has been shown to infect cattle, sheep, exotic ungulates, cats, non-human primates, and humans when the new host is exposed to feeds or foods contaminated with the disease agent. The purpose of this study was to test whether non-human primates (cynomolgous macaque) are susceptible to the agent of sheep scrapie.

***After an incubation period of approximately 10 years a macaque developed progressive clinical signs suggestive of neurologic disease.

***Upon postmortem examination and microscopic examination of tissues, there was a widespread distribution of lesions consistent with a transmissible spongiform encephalopathy.

***This information will have a scientific impact since it is the first study that demonstrates the transmission of scrapie to a non-human primate with a close genetic relationship to humans.

***This information is especially useful to regulatory officials and those involved with risk assessment of the potential transmission of animal prion diseases to humans.

Technical Abstract:

Classical bovine spongiform encephalopathy (c-BSE) is an animal prion disease that also causes variant Creutzfeldt-Jakob disease in humans. Over the past decades, c-BSE's zoonotic potential has been the driving force in establishing extensive protective measures for animal and human health.

***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

SPONTANEOUS TRANSMISSIBLE SPONGIFORM
ENCEPHALOPATHY TSE PRION AKA MAD COW TYPE DISEASE ???

*** We describe the transmission of spongiform encephalopathy in a non-human primate inoculated 10 years earlier with a strain of sheep c-scrapie. Because of this extended incubation period in a facility in which other prion diseases are under study, we are obliged to consider two alternative possibilities that might explain its occurrence. We first considered the possibility of a sporadic origin (like CJD in humans). Such an event is extremely improbable because the inoculated animal was 14 years old when the clinical signs appeared, i.e. about 40% through the expected natural lifetime of this species, compared to a peak age incidence of 60–65 years in human sporadic CJD, or about 80% through their expected lifetimes.

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.

>>> 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>

Tuesday, December 16, 2014

*** Evidence for zoonotic potential of ovine scrapie prions

Hervé Cassard,¹ n1 Juan-Maria Torres,² n1 Caroline Lacroux,¹ Jean-Yves Douet,¹ Sylvie L. Benestad,³ Frédéric Lantier,⁴ Séverine Lugan,¹ Isabelle Lantier,⁴ Pierrette Costes,¹ Naima Aron,¹ Fabienne Reine,⁵ Laetitia Herzog,⁵ Juan-Carlos Espinosa,² Vincent Beringue⁵, & Olivier Andréoletti¹, Affiliations Contributions Corresponding author Journal name: Nature Communications Volume: 5, Article number: 5821 DOI: doi:10.1038/ncomms6821 Received 07 August 2014 Accepted

Abstract

Although Bovine Spongiform Encephalopathy (BSE) is the cause of variant Creutzfeldt Jakob disease (vCJD) in humans, the zoonotic potential of scrapie prions remains unknown. Mice genetically engineered to overexpress the human prion protein (tgHu) have emerged as highly relevant models for gauging the capacity of prions to transmit to humans. These models can propagate human prions without any apparent transmission barrier and have been used to confirm the zoonotic ability of BSE. Here we show that a panel of sheep scrapie prions transmit to several tgHu mice models with an efficiency comparable to that of cattle BSE. ***The serial transmission of different scrapie isolates in these mice led to the propagation of prions that are phenotypically identical to those causing sporadic CJD (sCJD) in humans. ***These results demonstrate that scrapie prions have a zoonotic potential and raise new questions about the possible link between animal and human prions.

Subject terms: Biological sciences • Medical research At a glance

<http://www.nature.com/ncomms/2014/141216/ncomms6821/full/ncomms6821.html>

see more here ;

<http://www.nature.com/ncomms/2014/141216/ncomms6821/extref/ncomms6821-s1.pdf>

The serial transmission of different scrapie isolates in these mice led to the propagation of prions that are phenotypically identical to those causing sporadic CJD (sCJD) in humans.

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

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

5. A positive result from a chimpanzee challenged severely 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

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

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

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

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

snip...

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

PMID: 6997404

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

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

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

snip...

76/10.12/4.6

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

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

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

Gibbs CJ Jr, Gajdusek DC.

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

Transmission of Scrapie to the Cynomolgus Monkey (*Macaca fascicularis*)

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

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

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

<http://www.nature.com/nature/journal/v236/n5341/abs/236073ao.html>

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 1 Paul-Ehrlich-Institut, Germany; 2Commissariat à l'Énergie Atomique, France; 3Istituto 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 vCJD 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-Etcheagaray, 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, ie 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>

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/o3-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;

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 (PrP^{Sc}) 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 PrP^{Sc} 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*)

Christina J. Sigurdson¹, Elizabeth S. Williams², Michael W. Miller³, Terry R. Spraker^{1,4}, Katherine I. O'Rourke⁵ and Edward A. Hoover¹

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, USA² 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

Author for correspondence: Edward Hoover. Fax +1 970 491 0523. e-mail ehoover@lamar.colostate.edu

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 PrP^{res}, 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 PrP res 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*)

Gordon B. Mitchell¹, Christina J. Sigurdson^{2,3}, Katherine I. O'Rourke⁴, James Algire¹, Noel P. Harrington¹, Ines Walther¹, Terry R. Spraker⁵, Aru Balachandran^{1*}

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+\(PLoS+ONE+Alerts%3A+New+Articles\)#](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...

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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

<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/doc>

<http://webarchive.nationalarchives.gov.uk/20130402151656/http://archive.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/bse/cor-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 reply ; Molecular, Biochemical and Genetic Characteristics of BSE in Canada Singeltary reply ;

<http://www.plosone.org/annotation/listThread.action?sessionId=635CE9094E0EA15D5362B7D7B809448C?root=7143>

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

Discussion: The C, L and H type BSE cases in Canada exhibit molecular characteristics similar to those described for classical and atypical BSE cases from Europe and Japan.

*** 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.neuropriion.org/resources/pdf_docs/conferences/prion2009/prion2009_bookofabstracts.pdf

atypical spontaneous BSE in France LOL

FRANCE STOPS TESTING FOR MAD COW DISEASE BSE, and here's why, to many spontaneous events of mad cow disease \$\$\$

***so 20 cases of atypical BSE in France, compared to the remaining 40 cases in the remaining 12 Countries, divided by the remaining 12 Countries, about 3+ cases per country, besides Frances 20 cases. you cannot explain this away with any spontaneous BSe. ...TSS

Sunday, October 5, 2014

France stops BSE testing for Mad Cow Disease

<http://transmissiblespongiformencephalopathy.blogspot.com/2014/10/france-stops-bse-testing-for-mad-cow.html>

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

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 EMC1 Terry S. Singeltary Sr. Vol #: 1

Date: Fri, 16 May 2003 11:47:37 0500 EMC1 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>

PLEASE SEE FULL TEXT SUBMISSION ;

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

Subject: DOCKET-- 03D-0186 -- FDA Issues Draft Guidance on Use
of Material From Deer and Elk in Animal Feed; Availability

Date: Fri, 16 May 2003 11:47:37 -0500

From: "Terry S. Singeltary Sr." T

o: fdadockets@oc.fda.gov

Greetings FDA,

i would kindly like to comment on;

Docket 03D-0186

FDA Issues Draft Guidance on Use of Material From Deer and Elk
in Animal Feed; Availability

Several factors on this apparent voluntary proposal disturbs me

greatly, please allow me to point them out;

1. MY first point is the failure of the partial ruminant-to-ruminant feed ban of 8/4/97. this partial and voluntary feed ban of some ruminant materials being fed back to cattle is terribly flawed. without the _total_ and _mandatory_ ban of all ruminant materials being fed back to ruminants including cattle, sheep, goat, deer, elk and mink, chickens, fish (all farmed animals for human/animal consumption), this half ass measure will fail terribly, as in the past decades...

2. WHAT about sub-clinical TSE in deer and elk? with the recent findings of deer fawns being infected with CWD, how many could possibly be sub-clinically infected. until we have a rapid TSE test to assure us that all deer/elk are free of disease (clinical and sub-clinical), we must ban not only documented CWD infected deer/elk, but healthy ones as well. it this is not done, they system will fail...

3. WE must ban not only CNS (SRMs specified risk materials), but ALL tissues. recent new and old findings support infectivity in the rump or ass muscle. wether it be low or high, accumulation will play a crucial role in TSEs.

4. THERE are and have been for some time many TSEs in the USA. TME in mink, Scrapie in Sheep and Goats, and unidentified TSE in USA cattle. all this has been proven, but the TSE in USA cattle has been totally ignored for decades. i will document this data below in my references.

5. UNTIL we ban all ruminant by-products from being fed back to ALL ruminants, until we rapid TSE test (not only deer/elk) but cattle in sufficient numbers to find (1 million rapid TSE test in USA cattle annually for 5 years), any partial measures such as the ones proposed while ignoring sub-clinical TSEs and not rapid TSE testing cattle, not closing down feed mills that continue to violate the FDA's BSE feed regulation (21 CFR 589.2000) and not making freely available those violations, will only continue to spread these TSE mad cow agents in the USA. I am curious what we will call a phenotype in a species that is mixed with who knows how many strains of scrapie, who knows what strain or how many strains of TSE in USA cattle, and the CWD in deer and elk (no telling how many strains there), but all of this has been rendered for animal feeds in the USA for decades. it will get interesting once someone starts looking in all species, including humans here in the USA, but this has yet to happen...

6. IT is paramount that CJD be made reportable in every state (especially "sporadic" cjd), and that a CJD Questionnaire must be issued to every family of a victim of TSE. only checking death certificates will not be sufficient. this has been proven as well (see below HISTORY OF CJD – CJD QUESTIONNAIRE)

7. WE must learn from our past mistakes, not continue to make the same mistakes...

REFERENCES

Six white-tailed deer fawns test positive for CWD

MADISON -- Six fawns in the area of south central Wisconsin where chronic wasting disease has been found in white-tailed deer have tested positive for the disease, according to Department of Natural Resources wildlife health officials. These are the youngest wild white-tailed deer detected with chronic wasting disease (CWD) to date.

Approximately 4,200 fawns, defined as deer under 1 year of age, were sampled from the eradication zone over the last year. The majority of fawns sampled were between the ages of 5 to 9 months, though some were as young as 1 month. Two of the six fawns with CWD detected were 5 to 6 months old. All six of the positive fawns were taken from the core area of the CWD eradication zone where the highest numbers of positive deer have been identified.

snip...

<http://www.dnr.state.wi.us/org/caer/ce/news/on/2003/on20030513.htm#art4>

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Issued: Monday, 28 August 2000

NEW EVIDENCE OF SUB-CLINICAL PRION INFECTION: IMPORTANT RESEARCH FINDINGS RELEVANT TO CJD AND BSE

A team of researchers led by Professor John Collinge at the Medical Research Council Prion Unit¹ report today in the Proceedings of the National Academy of Sciences, on new evidence for the existence of a 'sub-clinical' form of BSE in mice which was unknown until now.

The scientists took a closer look at what is known as the 'species barrier' - the main protective factor which limits the ability of prions² to jump from one species to infect another. They found the mice had a 'sub-clinical' form of disease where they carried high levels of infectivity but did not develop the clinical disease during their normal lifespan. The idea that individuals can carry a disease and show no clinical symptoms is not new. It is commonly seen in conventional infectious diseases.

Researchers tried to infect laboratory mice with hamster prions³ called Sc²³⁷ and found that the mice showed no apparent signs of disease. However, on closer inspection they found that the mice had high levels of mouse prions in their brains. This was surprising because it has always been assumed that hamster prions could not cause the disease in mice, even when injected directly into the brain.

In addition the researchers showed that this new sub-clinical infection could be easily passed on when injected into healthy mice and hamsters.

The height of the species barrier varies widely between different combinations of animals and also varies with the type or strain of

prions. While some barriers are quite small (for instance BSE easily infects mice), other combinations of strain and species show a seemingly impenetrable barrier. Traditionally, the particular barrier studied here was assumed to be robust.

Professor John Collinge said: "These results have a number of important implications. They suggest that we should re-think how we measure species barriers in the laboratory, and that we should not assume that just because one species appears resistant to a strain of prions they have been exposed to, that they do not silently carry the infection. This research raises the possibility, which has been mentioned before, that apparently healthy cattle could harbour, but never show signs of, BSE.

"This is a timely and unexpected result, increasing what we know about prion disease. These new findings have important implications for those researching prion disease, those responsible for preventing infected material getting into the food chain and for those considering how best to safeguard health and reduce the risk that theoretically, prion disease could be contracted through medical and surgical procedures."

ISSUED FRIDAY 25 AUGUST UNDER EMBARGO. PLEASE NOTE THAT THE EMBARGO IS SET BY THE JOURNAL.

FOR FURTHER INFORMATION CONTACT THE MRC PRESS OFFICE ON 020 7637 6011 (OFFICE HOURS) OR 07818 428297 OR 0385 774357 (OUT-OF-OFFICE-HOURS) OR PROFESSOR JOHN COLLINGE ON 020 7594 3760. PLEASE NOTE THAT OWING TO TRAVEL COMMITMENTS PROFESSOR COLLINGE WILL ONLY BE AVAILABLE UNTIL 16.30 ON FRIDAY 25 AUGUST AND CONTACTABLE AGAIN ON MONDAY 28 AUGUST VIA THE MRC PRESS OFFICE. DR ANDREW HILL (A CO-AUTHOR ON THE PAPER) FROM THE DEPARTMENT OF PATHOLOGY AT THE UNIVERSITY OF MELBOURNE WILL BE AVAILABLE ON 00 61 3 8344 3995 (DURING OFFICE HOURS) OR 00 61 3 9443 0009 (OUT-OF-OFFICE HOURS). PLEASE NOTE THAT AUSTRALIA IS TEN HOURS AHEAD OF UK TIME.

NOTES FOR EDITORS

Professor Collinge is a consultant neurologist and Director of the newly formed MRC Prion Unit based at The Imperial College School of Medicine at St Mary's Hospital. He is also a member of the UK Government's Spongiform Encephalopathy Advisory Committee (SEAC). The MRC prion unit is was set up in 1999, and its work includes molecular genetic studies of human prion disease and transgenic modelling of human prion diseases.

Prions are unique infectious agents that cause fatal brain diseases such as Creutzfeldt-Jakob disease (CJD) in humans and scrapie and BSE (mad cow disease) in animals. In some circumstances prions from one species of animals can infect another and it is clear that BSE has done this to cause the disease variant CJD in the UK and France. It remains unclear how large an epidemic of variant CJD will occur over the years ahead.

The strain of prion used here to infect the mice is the Sc237 strain (also known as 263K) which infects hamsters, and until now was assumed not to infect mice.

This research was funded by the Medical Research Council and

The Medical Research Council (MRC) is a national organisation funded by the UK tax-payer. Its business is medical research aimed at improving human health; everyone stands to benefit from the outputs. The research it supports and the scientists it trains meet the needs of the health services, the pharmaceutical and other health-related industries and the academic world. MRC has funded work which has led to some of the most significant discoveries and achievements in medicine in the UK. About half of the MRC's expenditure of Â£345 million is invested in over 50 of its Institutes and Units, where it employs its own research staff. The remaining half goes in the form of grant support and training awards to individuals and teams in universities and medical schools.

The Wellcome Trust is the world's largest medical research charity with a spend of some Â£600 million in the current financial year 1999/2000. The Wellcome Trust supports more than 5,000 researchers, at 400 locations, in 42 different countries to promote and foster research with the aim of improving human and animal health. As well as funding major initiatives in the public understanding of science, the Wellcome Trust is the country's leading supporter of research into the history of medicine.

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Contact the MRC

http://www.mrc.ac.uk/index/public_interest/public-press_office/public-press_releases_2000/public-mrc-43-00.htm

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

Christina J. Sigurdson¹, Elizabeth S. Williams², Michael W. Miller³, Terry R. Spraker^{1,4}, Katherine I. O'Rourke⁵ and Edward A. Hoover¹

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, USA² 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

Author for correspondence: Edward Hoover. Fax +1 970 491 0523. e-mail ehoover@lamar.colostate.edu

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 PrP^{res}, 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. PrP^{res} 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 PrP^{res} staining was detected in lymphoid tissue of three control fawns receiving a control brain inoculum, nor was PrP^{res} detectable in neural tissue of any fawn. PrP^{res}-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 PrP^{res} after oral exposure to an inoculum containing CWD prions. In the earliest post-exposure period, CWD PrP^{res} 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 PrP^{res} 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>

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now, just what is in that deer feed? _ANIMAL PROTEIN_

Subject: MAD DEER/ ELK DISEASE AND POTENTIAL SOURCES

Date: Sat, 25 May 2002 18:41:46 -0700

From: "Terry S. Singeltary Sr." Reply-To: BSE-L To: BSE-L

snip...

animal protein

<http://www.surefed.com/deer.htm>

BODE'S GAME FEED SUPPLEMENT #400 A RATION FOR DEER
NET WEIGHT 50 POUNDS 22.6 KG.

snip...

animal protein

<http://www.bodefeed.com/prod7.htm>

Ingredients

Grain Products, Plant Protein Products, Processed Grain By-
Products, Forage Products, Roughage Products 15%, Molasses
Products, __Animal Protein Products__, Monocalcium
Phosphate, Dicalcium Pyosphate, Salt, Calcium Carbonate,
Vitamin A Acetate with D-activated Animal Sterol (source of
Vitamin D₃), Vitamin E Supplement, Vitamin B₁₂ Supplement,
Riboflavin Supplement, Niacin Supplement, Calcium Pantothenate,
Choline Chloride, Folic Acid, Menadione Sodium Bisulfite
Complex, Pyridoxine Hydrochloride, Thiamine Mononitrate, d-
Biotin, Manganous Oxide, Zinc Oxide, Ferrous Carbonate, Calcium
Iodate, Cobalt Carbonate, Dried Saccharomyces Berevisiae
Fermentation Solubles, Cellulose gum, Artificial Flavors added.

<http://www.bodefeed.com/prod6.htm>

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MORE ANIMAL PROTEIN PRODUCTS FOR DEER

Bode's #1 Game Pellets A RATION FOR DEER F3153

GUARANTEED ANALYSIS Crude Protein (Min) 16% Crude Fat
(Min) 2.0% Crude Fiber (Max) 19% Calcium (Ca) (Min) 1.25%
Calcium (Ca) (Max) 1.75% Phosphorus (P) (Min) 1.0% Salt (Min)
.30% Salt (Max) .70%

Ingredients

Grain Products, Plant Protein Products, Processed Grain By-Products, Forage Products, Roughage Products, 15% Molasses Products, ___Animal Protein Products___, Monocalcium Phosphate, Dicalcium Phosphate, Salt, Calcium Carbonate, Vitamin A Acetate with D-activated Animal Sterol (source of Vitamin D₃) Vitamin E Supplement, Vitamin B₁₂ Supplement, Riboflavin Supplement, Niacin Supplement, Calcium Pantothenate, Choline Chloride, Folic Acid, Menadione Sodium Bisulfite Complex, Pyridoxine Hydrochloride, Thiamine Mononitrate, e - Biotin, Manganous Oxide, Zinc Oxide, Ferrous Carbonate, Calcium Iodate, Cobalt Carbonate, Dried Saccharomyces Cerevisiae Fermentation Solubles, Cellulose gum, Artificial Flavors added.

FEEDING DIRECTIONS Feed as Creep Feed with Normal Diet

<http://www.bodefeed.com/prod8.htm>

INGREDIENTS

Grain Products, Roughage Products (not more than 35%), Processed Grain By-Products, Plant Protein Products, Forage Products, ___Animal Protein Products___, L-Lysine, Calcium Carbonate, Salt, Monocalcium/Dicalcium Phosphate, Yeast Culture, Magnesium Oxide, Cobalt Carbonate, Basic Copper Chloride, Manganese Sulfate, Manganous Oxide, Sodium Selenite, Zinc Sulfate, Zinc Oxide, Sodium Selenite, Potassium Iodide, Ethylenediamine Dihydriodide, Vitamin E Supplement, Vitamin A Supplement, Vitamin D₃ Supplement, Mineral Oil, Mold Inhibitor, Calcium Lignin Sulfonate, Vitamin B₁₂ Supplement, Menadione Sodium Bisulfite Complex, Calcium Pantothenate, Riboflavin, Niacin, Biotin, Folic Acid, Pyridoxine Hydrochloride, Mineral Oil, Chromium Tripicolinate

DIRECTIONS FOR USE

Deer Builder Pellets is designed to be fed to deer under range conditions or deer that require higher levels of protein. Feed to deer during gestation, fawning, lactation, antler growth and pre-rut, all phases which require a higher level of nutrition. Provide adequate amounts of good quality roughage and fresh water at all times.

http://www.profilenutrition.com/Products/Specialty/deer_builder_pellets.html

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DEPARTMENT OF HEALTH & HUMAN SERVICES PUBLIC HEALTH
SERVICE FOOD AND DRUG ADMINISTRATION

April 9, 2001 WARNING LETTER

01-PHI-12 CERTIFIED MAIL RETURN RECEIPT REQUESTED

Brian J. Raymond, Owner Sandy Lake Mills 26 Mill Street P.O. Box
117 Sandy Lake, PA 16145 PHILADELPHIA DISTRICT

Tel: 215-597-4390

Dear Mr. Raymond:

Food and Drug Administration Investigator Gregory E. Beichner conducted an inspection of your animal feed manufacturing operation, located in Sandy Lake, Pennsylvania, on March 23, 2001, and determined that your firm manufactures animal feeds including feeds containing prohibited materials. The inspection found significant deviations from the requirements set forth in Title 21, code of Federal Regulations, part 589.2000 - Animal Proteins Prohibited in Ruminant Feed. The regulation is intended to prevent the establishment and amplification of Bovine Spongiform Encephalopathy (BSE). Such deviations cause products being manufactured at this facility to be misbranded within the meaning of Section 403(f), of the Federal Food, Drug, and Cosmetic Act (the Act).

Our investigation found failure to label your swine feed with the required cautionary statement "Do Not Feed to cattle or other Ruminants" The FDA suggests that the statement be distinguished by different type-size or color or other means of highlighting the statement so that it is easily noticed by a purchaser.

In addition, we note that you are using approximately 140 pounds of cracked corn to flush your mixer used in the manufacture of animal feeds containing prohibited material. This flushed material is fed to wild game including deer, a ruminant animal. Feed material which may potentially contain prohibited material should not be fed to ruminant animals which may become part of the food chain.

The above is not intended to be an all-inclusive list of deviations from the regulations. As a manufacturer of materials intended for animal feed use, you are responsible for assuring that your overall operation and the products you manufacture and distribute are in compliance with the law. We have enclosed a copy of FDA's Small Entity Compliance Guide to assist you with complying with the regulation... blah, blah, blah...

http://www.fda.gov/foi/warning_letters/g1115d.pdf

snip...end

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

Date: Thu, 15 May 2003 19:15:01 -0500

Subject: FDA ANNOUNCES USE OF MATERIAL FROM DEER AND ELK IN ANIMAL FEED ARE AT HIGH RISK OF CWD

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

Reply-To: Bovine Spongiform Encephalopathy <[log in to unmask]>

Date: Thu, 15 May 2003 19:15:01 -0500

Content-Type: text/plain

Parts/Attachments: Parts/Attachments text/plain (249 lines)

Bovine Spongiform Encephalopathy <[log in to unmask]>

FDA Talk Paper

To: 34 May 15, 2003

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

FDA Issues Draft Guidance on Use of Material From Deer and Elk in Animal Feed

The Food and Drug Administration (FDA) is announcing the availability of a draft guidance for industry entitled "Use of Material From Deer and Elk in Animal Feed." When finalized, this draft guidance will describe FDA's recommendations regarding the use in all animal feed of all material from deer and elk that are positive, or at high risk, for Chronic Wasting Disease (CWD).

The potential risks from CWD to humans or non-cervid animals such as poultry and swine are not well understood. However, because of recent recognition that CWD is spreading rapidly in white-tailed deer, and because CWD's route of transmission is poorly understood, FDA is recommending that any material from CWD-positive animals, or deer and elk considered to be at high risk for CWD, not be used in any animal feed or feed ingredients. High risk deer and elk are those from 1) areas declared by state officials to be endemic for CWD and/or to be CWD eradication zones and 2) those that at some time during the 60-month period before the time of slaughter were part of a captive herd with a CWD-positive animal.

CWD is a neurological (brain) disease of farmed and wild deer and the elk that belong in the cervidae animal family (cervids). Only deer and elk are known to be susceptible to CWD by natural transmission. The disease has been found in farmed and wild mule deer, white-tailed deer, North American elk, and farmed black-tailed deer. CWD belongs to a family of animal and human diseases called transmissible spongiform encephalopathies (TSEs). These include bovine spongiform encephalopathy (BSE or "mad cow" disease) in cattle; scrapie in sheep and goats; and classical and variant Creutzfeldt-Jakob diseases (CJD and vCJD) in humans. TSEs are very rare, but are always fatal.

There is no known treatment for these diseases, and there is no vaccine to prevent them. In addition, although validated postmortem diagnostic tests are available, there are no validated diagnostic tests for CWD that can be used to test live animals for the disease.

FDA's guidance documents are not regulations and are not mandatory. They set forth voluntary recommendations from FDA. They do not create or confer any rights for, or on, any person and do not operate to bind FDA or the public. An alternate method may be used as long as it satisfies the requirements of applicable statutes and regulations.

This draft guidance is being distributed for comment purposes only. Written comments on the draft guidance may be submitted to the Dockets Management Branch (HFA-305), Food and Drug Administration, 5630 Fishers Lane, Room 1061, Rockville, MD 20852. Electronic comments may be submitted to <http://www.accessdata.fda.gov/scripts/oc/dockets/comments/commentdocket.cfm>. Comments should be identified with the full title of the draft guidance and Docket number 03D-0186. Written comments on the draft guidance may be submitted at any time; however, comments should be submitted by June 16, 2003, to ensure their adequate consideration in preparation of the final document.

Additional information on the draft guidance document may be found in the May 16, 2003, Federal Register and from Dr. Burt Pritchett, Center for Veterinary Medicine (HFV-222) Food and Drug Administration, 7500 Standish Place, Rockville, MD 20855, 301-827-0177. E-mail: [log in to unmask]. The draft guidance is posted at

<http://www.fda.gov/cvm/guidance/dguide158.pdf>.

<http://www.fda.gov/bbs/topics/ANSWERS/2003/ANS01220.html>

CONTAINS NON-BINDING RECOMMENDATIONS

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Guidance for Industry Use of Material from Deer and Elk in Animal Feed DRAFT GUIDANCE

This draft guidance is being distributed for comment purposes only. Comments and suggestions regarding this draft guidance should be sent to the Dockets Management Branch (HFA-305), Food and Drug Administration, 5630 Fishers Lane, Room 1061,

Rockville, MD 20852. Comments may also be submitted electronically on the Internet at <http://www.fda.gov/dockets/ecomments>. Once on this Internet site, select "[03D-0186][Use of Material from Deer and Elk in Animal Feed]" and follow the directions. All written comments should be identified with Docket No. 03D-0186. For questions regarding this draft document, contact Burt Pritchett, Center for Veterinary Medicine (HFV-222), Food and Drug Administration, 7500 Standish Place, Rockville, MD 20855, 301-827-0177. E-mail: [log in to unmask] Additional copies of this draft guidance document may be requested from the Communications Staff (HFV-12), Center for Veterinary Medicine, Food and Drug Administration, 7500 Standish Place, Rockville, MD 20855, and may be viewed on the Internet at <http://www.fda.gov/cvm>.

U.S. Department of Health and Human Services Food and Drug Administration Center for Veterinary Medicine May 14, 2003
CONTAINS NON-BINDING RECOMMENDATIONS

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Guidance for Industry¹ Use of Material from Deer and Elk in Animal Feed I.

Introduction

FDA's guidance documents, including this guidance, do not establish legally enforceable responsibilities. Instead, guidances describe the Agency's current thinking on a topic and should be viewed only as recommendations, unless specific regulatory or statutory requirements are cited. The use of the word "should" in Agency guidances means that something is suggested or recommended, but not required. Under FDA's BSE feed regulation (21 CFR 589.2000) most material from deer and elk is prohibited for use in feed for ruminant animals. This draft guidance document describes FDA's recommendations regarding the use in all animal feed of all material from deer and elk that are positive for Chronic Wasting Disease (CWD) or are considered at high risk for CWD. The potential risks from CWD to humans or non-cervid animals such as poultry and swine are not well understood. However, because of recent recognition that CWD is spreading rapidly in white-tailed deer, and because CWD's route of transmission is poorly understood, FDA is making recommendations regarding the use in animal feed of rendered materials from deer and elk that are CWD-positive or that are at high risk for CWD. II. Background CWD is a neurological (brain) disease of farmed and wild deer and elk that belong in the animal family cervidae (cervids). Only deer and elk are known to be susceptible to CWD by natural transmission. The disease has been found in farmed and wild mule deer, white-tailed deer, North American elk, and in farmed black-tailed deer. CWD belongs to a family of animal and human diseases called transmissible spongiform encephalopathies.¹ This draft guidance has been prepared by the Division of Animal Feeds in the Center for Veterinary Medicine (CVM) at the Food and Drug Administration. This draft guidance, when finalized, will represent the Food and Drug Administration's current thinking on the use of material from deer and elk in animal feed. It does not create or confer any rights for or on any person and does not operate to bind FDA or the public. You can use an alternative approach if the approach satisfies the requirements of applicable statutes or regulations. If you want to discuss an alternative approach, contact the FDA staff responsible for implementing this guidance. If you cannot identify the appropriate FDA staff, call the appropriate number listed on the title page of this guidance.

(TSEs). These include bovine spongiform encephalopathy (BSE or “mad cow” disease) in cattle; scrapie in sheep and goats; and classical and variant Creutzfeldt-Jakob diseases (CJD and vCJD) in humans. There is no known treatment for these diseases, and there is no vaccine to prevent them. In addition, although validated postmortem diagnostic tests are available, there are no validated diagnostic tests for CWD that can be used to test for the disease in live animals.

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

IV. Use in animal feed of material from deer and elk considered at high risk for CWD. Deer and elk considered at high risk for CWD include:

(1) animals from areas declared by State officials to be endemic for CWD and/or to be CWD eradication zones; and

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

FDA recommends that materials from deer and elk considered at high risk for CWD no longer be entered into the animal feed system. Under present circumstances, FDA is not recommending that feed made from deer and elk from a non-endemic area be recalled if a State later declares the area endemic for CWD or a CWD eradication zone. In addition, at this time, FDA is not recommending that feed made from deer and elk believed to be from a captive herd that contained no CWD-positive animals be recalled if that herd is subsequently found to contain a CWD-positive animal. V. Use in animal feed of material from deer and elk NOT considered at high risk for CWD. FDA continues to consider materials from deer and elk NOT considered at high risk for CWD to be acceptable for use in NON-RUMINANT animal feeds in accordance with current agency regulations, 21 CFR 589.2000. Deer and elk not considered at high risk include:

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

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

DAY LATE AND DOLLAR SHORT, they should have done this 30 years ago before they fed all the cattle this recipe for TSEs. and what about those cattle, what will we call this phenotype mixed up of scrapie, cwd and UTSEA in cattle (unidentified TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHY agent) in the USA?

i got a name for it, but cannot post to this list.

no wonder we don't test humans or cattle for tse's in the USA...

i just want to go out and slap some one, but i should "DELAY" myself. i am in one of those moods. so i will not be posting for a short/long? period of time, so i can get my diplomatic composure back, may wet a hook, or maybe "DELAY" that for a moment or two and just slap someone...

disgusted again in Bacliff, TEXAS USA...

Terry S. Singeltary Sr.

<http://mailhost.rz.uni-karlsruhe.de/warc/bse-1.html>

Subject: MAD COW FEED BAN WARNING LETTER USA 2003

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

Reply-To: Bovine Spongiform Encephalopathy <[log in to unmask]>

Date: Tue, 20 May 2003 08:39:31 -0500

Content-Type: text/plain

Parts/Attachments: Parts/Attachments text/plain (181 lines)

Bovine Spongiform Encephalopathy <[log in to unmask]>

Public Health Service Food and Drug Administration

Minneapolis District Office Central Region 212 Third Avenue South
Minneapolis, MN 55401 Telephone: (612) 334-4100 FAX: (612)
334-4134

May 6, 2003

WARNING LETTER CERTIFIED MAIL RETURN RECEIPT
REQUESTED Refer to MIN 03 - 20

Steve L. Denk President Barr Animal Foods A Division of Barr
Enterprises, Inc. W7276 Chickadee Road Greenwood, Wisconsin
54437

Dear Mr. Denk:

On April 8, 2003, an investigator from the Food and Drug Administration (FDA) inspected your rendering and animal feed manufacturing operation located at W7276 Chickadee Road, Greenwood, WI. This inspection found significant deviations from the requirements set forth in Title 21, Code of Federal Regulations, Part 589.2000, "Animal Proteins Prohibited in Ruminant Feed" (21 CFR 589.2000). The regulation is intended to prevent the establishment and amplification of Bovine Spongiform Encephalopathy (BSE). Under 21 CFR 589.2000(g)(2), such deviations cause products being manufactured and/or distributed by this facility to be deemed misbranded within the meaning of Section 403(a)(1) of the Federal Food, Drug and Cosmetic Act (the Act), and these products may not be lawfully introduced, or delivered for introduction, into interstate commerce.

Products that contain or may contain protein derived from mammalian tissues and are intended for use in animal feed must be labeled with the cautionary statement, "Do not feed to cattle or other ruminants." This is required by 21 CFR 589.2000(c)(1)(i). The FDA suggests the statement be distinguished by different type size or color, or other means of highlighting the statement so that it is easily noticed by a purchaser. Our inspection found that you are not labeling your 50-pound blocks of frozen beef and bulk loads of beef bone chips and rendering waste, which are intended for animal feed, with that caution statement. As a result, these products are misbranded within the meaning of Section 403(a)(1) of the Act.

The above is not intended to be an all-inclusive list of deviations from the regulations. As a renderer and manufacturer of materials intended for animal feed use, you are responsible for ensuring that your overall operation and the products you manufacture and distribute are in compliance with the law. We have enclosed a copy of the FDA's Small Entity Compliance Guide Nos. 67 and 68 to assist you with complying with the regulation.

You should take prompt action to correct these violations and you should establish a system whereby such violations do not recur. Failure to promptly correct these violations may result in regulatory action without further notice. These actions include, but are not limited to, seizure and/or injunction.

It is necessary for you to take action on this matter now. Please provide this office a written response within 15 working days of receipt of this letter with the steps you have taken to bring your firm into compliance with the law. Your response should include an explanation of each step being taken to correct the violations

and prevent their recurrence. If corrective action cannot be taken within 15 working days, state the reason for the delay and the date by which the corrections will be completed. Please include copies of any available documentation demonstrating that corrections have been made.

Your reply should be directed to Compliance Officer Timothy G. Philips at the address indicated on the letterhead.

Sincerely,

/s/

David R. Yost for W. Charles Becoat

Minneapolis District

http://www.fda.gov/foi/warning_letters/g4000d.htm

Greetings List Members,

what a surprise to see this posting of warning letter for feeding ruminants to ruminants in the USA, simply astonishing. my question is, if they cannot stop this practice of feeding cows to cows in the USA in 2003, after some 6 years (since 8/4/97 partial and voluntary), how in the world do they plan on enforcing a ban on deer/elk products from entering the feed market?

FDA Issues Draft Guidance on Use of Material From Deer and Elk in Animal Feed

<http://www.fda.gov/bbs/topics/ANSWERS/2003/ANSo1220.html>

TSS SUBMISSION TO FEDERAL DOCKET ON DRAFT GUIDANCE ON USE OF MATERIAL FROM DEER AND ELK IN ANIMAL FEED

(if this url does not change again? it will be posted here for viewing for those interested...TSS)

snip...end...TSS

Draft Guidance for Industry on Ensuring Safety of Animal Feed Maintained and Fed On-Farm; Availability FDA-2014-D-1180-0001 Singeltary Submission Comment Period Closed Jun 3 2015, at 11:59 PM ET

*** See attached file(s) No documents available. Attachments
View All (1) Guidance for Industry Ensuring Safety of Animal Feed
Maintained and Fed On-Farm Terry Singeltary Comment View
Attachment:

<http://www.regulations.gov/#!documentDetail;D=FDA-2014-D-1180-0003>

Sunday, April 5, 2015

*** Guidance for Industry Ensuring Safety of Animal Feed
Maintained and Fed On-Farm Draft Guidance FDA-2014-D-1180

<http://madcowfeed.blogspot.com/2015/04/guidance-for-industry-ensuring-safety.html>

*** Docket No. APHIS-2007-0127 Scrapie in Sheep and Goats
Terry Singeltary Sr. Submission ***

Monday, November 16, 2015

*** Docket No. APHIS-2007-0127 Scrapie in Sheep and Goats
Terry Singeltary Sr. Submission ***

<http://www.regulations.gov/#!documentDetail;D=APHIS-2007-0127-0032>

Draft Guidance for Industry on Ensuring Safety of Animal Feed
Maintained and Fed On-Farm; Availability

203 entitled "Ensuring Safety of Animal Feed Maintained and
Fed On-Farm."

<http://www.regulations.gov/#!documentDetail;D=FDA-2014-D-1180-0001>

Terry S. Singeltary Sr. submission ;

<http://www.regulations.gov/#!documentDetail;D=FDA-2014-D-1180-0003>

Docket No. APHIS-2014-0107 Bovine Spongiform
Encephalopathy; Importation of Animals and Animal Products
Singeltary Submission

Posted: 12/30/2014 ID: APHIS-2014-0107-0001

<http://www.regulations.gov/#!documentDetail;D=APHIS-2014-0107-0003>

Notice: Environmental Impact Statements; Availability, etc.:
Animal Carcass Management

Document ID: APHIS-2013-0044-0001 Docket ID: APHIS-2013-0044 Comment ID: APHIS-2013-0044-0002

<http://www.noticeandcomment.com/APHIS-2013-0044-0002-fcod-365217.aspx>

(APHIS) Notice: Agency Information Collection Activities;
Proposals, Submissions, and Approvals:

Chronic Wasting Disease Herd Certification Program Agency
Information Collection Activities; Proposals, Submissions, and
Approvals:

Chronic Wasting Disease Herd Certification Program (Document
ID APHIS-2011-0032-0001)

<http://www.regulations.gov/#!documentDetail;D=APHIS-2011-0032-0002>

Comment from Terry S Singletary Sr

Subject: BSE; MRR; IMPORTATION OF LIVE BOVINES AND
PRODUCTS DERIVED FROM BOVINES [Docket No. APHIS-2006-
0041] RIN 0579-AC01

Date: January 9, 2007 at 9:08 am PST [Federal...

Public Submission Posted: 01/09/2007

ID: APHIS-2006-0041-0006 Agency: APHIS RIN: 0579-AC01
Docket ID: APHIS-2006-0041

Organization: na Submitter Name: Terry Singletary Sr.

<https://www.regulations.gov/#!documentDetail;D=APHIS-2006-0041-0006>

<https://www.regulations.gov/contentStreamer?documentId=APHIS-2006-0041-0006&attachmentNumber=1&disposition=attachment&contentType=msw8>

<https://view.officeapps.live.com/op/view.aspx?src=https%3A%2F%2Fwww.regulations.gov%2FcontentStreamer%3FdocumentId%3DAPHIS-2006-0041-0006%26attachmentNumber%3D1%26disposition%3Dattachment%26contentType%3Dmsw8>

Owens, Julie

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

Sent: Monday, July 24, 2006 1:09 PM

To: FSIS RegulationsComments

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

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

FSIS, USDA, REPLY TO SINGELTARY

http://www.fsis.usda.gov/PDF/BSE_Risk_Assess_Response_Public_Comments.pdf

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

Sent: Thursday, September 08, 2005 6:17 PM

To: fsis.regulationscomments@fsis.usda.gov

Subject: [Docket No. 03-025IFA] FSIS Prohibition of the Use of Specified Risk Materials for Human Food and Requirements for the Disposition of Non-Ambulatory Disabled Cattle

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

<http://www.regulations.gov/#!documentDetail;D=APHIS-2006-0118-0100>

Monday, January 08,2001

PDF]Freas, William TSS SUBMISSION File Format: PDF/Adobe Acrobat - Page 1. J Freas, William From: Sent: To: Subject: Terry S. Singeltary Sr. [[log in to unmask]] Monday, January 08,2001 3:03 PM freas ...

http://www.fda.gov/ohrms/dockets/ac/01/slides/3681s2_09.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

The infectious agents responsible for transmissible spongiform encephalopathy (TSE) are notoriously resistant to most physical and chemical methods used for inactivating pathogens, including heat. It has long been recognized, for example, that boiling is ineffective and that higher temperatures are most efficient when combined with steam under pressure (i.e., autoclaving). As a means of decontamination, dry heat is used only at the extremely high temperatures achieved during incineration, usually in excess of 600°C. It has been assumed, without proof, that incineration totally inactivates the agents of TSE, whether of human or animal origin.

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

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

Histochemical analysis of hamster brains inoculated with the solid residue showed typical spongiform degeneration and vacuolation. Re-inoculation of these brains into a new cohort of hamsters led to onset of clinical scrapie symptoms within 75 days, suggesting that the specific infectivity of the prion protein was not changed during the biodiesel process. The biodiesel reaction cannot be considered a viable prion decontamination method for MBM, although we observed increased survival time of hamsters and reduced infectivity greater than 6 log orders in the solid MBM residue. Furthermore, results from our study compare for the first time prion detection by Western Blot versus an infectivity bioassay for analysis of biodiesel reaction products. We could show that biochemical analysis alone is insufficient for detection of prion infectivity after a biodiesel process.

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

Detection of protease-resistant cervid prion protein in water from

The data presented here demonstrate that sPMCA can detect low levels of PrPCWD in the environment, corroborate previous biological and experimental data suggesting long term persistence of prions in the environment^{2,3} and imply that PrPCWD accumulation over time may contribute to transmission of CWD in areas where it has been endemic for decades. This work demonstrates the utility of sPMCA to evaluate other environmental water sources for PrPCWD, including smaller bodies of water such as vernal pools and wallows, where large numbers of cervids congregate and into which prions from infected animals may be shed and concentrated to infectious levels.

<http://www.landesbioscience.com/journals/prion/NicholsPRION3-3.pdf>

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

Keywords: Abattoir; bovine spongiform encephalopathy; QRA; scrapie; TSE

In this article the development and parameterization of a quantitative assessment is described that estimates the amount of TSE infectivity that is present in a whole animal carcass (bovine spongiform encephalopathy [BSE] for cattle and classical/atypical scrapie for sheep and lambs) and the amounts that subsequently fall to the floor during processing at facilities that handle specified risk material (SRM). BSE in cattle was found to contain the most oral doses, with a mean of 9864 BOID₅₀s (310, 38840) in a whole carcass compared to a mean of 1851 OOID₅₀s (600, 4070) and 614 OOID₅₀s (155, 1509) for a sheep infected with classical and atypical scrapie, respectively. Lambs contained the least infectivity with a mean of 251 OOID₅₀s (83, 548) for classical scrapie and 1 OOID₅₀s (0.2, 2) for atypical scrapie. The highest amounts of infectivity falling to the floor and entering the drains from slaughtering a whole carcass at SRM facilities were found to be from cattle infected with BSE at rendering and large incineration facilities with 7.4 BOID₅₀s (0.1, 29), intermediate plants and small incinerators with a mean of 4.5 BOID₅₀s (0.1, 18), and collection centers, 3.6 BOID₅₀s (0.1, 14). The lowest amounts entering drains are from lambs infected with classical and atypical scrapie at intermediate plants and atypical scrapie at collection centers with a mean of 3×10^{-7} OOID₅₀s (2×10^{-8} , 1×10^{-6}) per carcass. The results of this model provide key inputs for the model in the companion paper published here.

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

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

<http://collections.europearchive.org/tna/20080102193705/http://www.bseinquiry.gov.uk/files/mb/m11b/tab01.pdf>

10 years post mad cow feed ban August 1997

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

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

RECALLS AND FIELD CORRECTIONS: VETERINARY MEDICINES -
- CLASS II

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

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- BY PASS ML W/SMARTA, Recall # V-025-2007

CODE

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

RECALLING FIRM/MANUFACTURER

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

REASON

Products manufactured from bulk feed containing blood meal that was cross contaminated with prohibited meat and bone meal and the labeling did not bear cautionary BSE statement.

VOLUME OF PRODUCT IN COMMERCE

9,997,976 lbs.

DISTRIBUTION

ID and NV

END OF ENFORCEMENT REPORT FOR MARCH 21, 2007

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

NEW URL LINK ;

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

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>

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 FEED VIOLATIONS OFFICIAL ACTION
INDICATED OAI UPDATE DECEMBER 2014 BSE TSE PRION

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

2006 WAS A BANNER YEARS TOO FOR MAD COW PROTEIN IN
COMMERCE IN USA.

a few examples below ;

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

PRODUCT a) Dairy feed, custom, Recall # V-134-6; b) Custom Dairy Feed with Monensin, Recall # V-135-6. CODE None. Bulk product RECALLING FIRM/MANUFACTURER Recalling Firm: Burkmann Feed, Greeneville, TN, by Telephone beginning on June 28, 2006. Manufacturer: H. J. Baker & Bro., Inc., Albertville, AL. Firm initiated recall is complete.

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

VOLUME OF PRODUCT IN COMMERCE 1,484 tons DISTRIBUTION TN and WV

<http://www.fda.gov/bbs/topics/enforce/2...00968.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

PRODUCT Bulk custom made dairy feed, Recall # V-115-6 CODE None RECALLING FIRM/MANUFACTURER Hiseville Feed & Seed Co., Hiseville, KY, by telephone and letter on or about July 14, 2006. FDA 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 Approximately 2,223 tons
DISTRIBUTION KY

PRODUCT Bulk custom made dairy feed, Recall # V-116-6 CODE
None RECALLING FIRM/MANUFACTURER Rips Farm Center,
Tollesboro, KY, by telephone and letter on July 14, 2006. FDA
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 1,220 tons DISTRIBUTION
KY

PRODUCT Bulk custom made dairy feed, Recall # V-117-6 CODE
None RECALLING FIRM/MANUFACTURER Kentwood Co-op,
Kentwood, LA, by telephone on June 27, 2006. FDA initiated
recall is completed.

REASON Possible contamination of animal feed ingredients,
including ingredients that are used in feed for dairy animals, with
ruminant derived meat and bone meal.

VOLUME OF PRODUCT IN COMMERCE 40 tons DISTRIBUTION LA
and MS

PRODUCT Bulk Dairy Feed, Recall V-118-6 CODE None
RECALLING FIRM/MANUFACTURER Cal Maine Foods, Inc.,
Edwards, MS, by telephone on June 26, 2006. FDA initiated recall
is complete.

REASON Possible contamination of animal feed ingredients,
including ingredients that are used in feed for dairy animals, with
ruminant derived meat and bone meal.

VOLUME OF PRODUCT IN COMMERCE 7,150 tons DISTRIBUTION
MS

PRODUCT Bulk custom dairy pre-mixes, Recall # V-119-6 CODE
None RECALLING FIRM/MANUFACTURER Walthall County Co-
op, Tylertown, MS, by telephone on June 26, 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 87 tons DISTRIBUTION MS

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

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<http://www.fda.gov/bbs/topics/ENFORCE/2...00964.html>

Subject: MAD COW FEED RECALL MI MAMMALIAN PROTEIN
VOLUME OF PRODUCT IN COMMERCE 27,694,240 lbs

Date: August 6, 2006 at 6:14 pm PST PRODUCT Bulk custom dairy feeds 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

END OF ENFORCEMENT REPORT FOR AUGUST 2, 2006

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<http://www.fda.gov/bbs/topics/enforce/2...00963.html>

ENFORCEMENT REPORT FOR AUGUST 2, 2006 please note, considering .005 grams is lethal, I do not know how much of this 125 TONS of banned mad cow protein was part of the ;

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

bbbut, this was about 10 years post mad cow feed ban from 1997. 10 years later, and still feeding banned mad cow protein to cervids??? considering that .005 gram is lethal to several bovines, and we know that the oral consumption of CWD tainted products is very efficient mode of transmission of CWD.

Subject: MAD COW FEED RECALL AL AND FL VOLUME OF
PRODUCT IN COMMERCE 125 TONS

Products manufactured from 02/01/2005 until 06/06/2006
Date: August 6, 2006 at 6:16 pm PST PRODUCT a) CO-OP 32% Sinking Catfish, Recall # V-100-6; b) Performance Sheep Pell W/Decox/A/N, medicated, net wt. 50 lbs, Recall # V-101-6; c) Pro 40% Swine Conc Meal -- 50 lb, Recall # V-102-6; d) CO-OP 32% Sinking Catfish Food Medicated, Recall # V-103-6; ***e) "Big Jim's" BBB Deer Ration, Big Buck Blend, Recall # V-104-6; f) CO-OP 40% Hog Supplement Medicated Pelleted, Tylosin 100 grams/ton, 50

lb. bag, Recall # V-105-6; g) Pig Starter Pell II, 18% W/MCDX Medicated 282020, Carbadox -- 0.0055%, Recall # V-106-6; h) CO-OP STARTER-GROWER CRUMBLES, Complete Feed for Chickens from Hatch to 20 Weeks, Medicated, Bacitracin Methylene Disalicylate, 25 and 50 Lbs, Recall # V-107-6; i) CO-OP LAYING PELLETS, Complete Feed for Laying Chickens, Recall # 108-6; j) CO-OP LAYING CRUMBLES, Recall # V-109-6; k) CO-OP QUAIL FLIGHT CONDITIONER MEDICATED, net wt 50 Lbs, Recall # V-110-6; l) CO-OP QUAIL STARTER MEDICATED, Net Wt. 50 Lbs, Recall # V-111-6; m) CO-OP QUAIL GROWER MEDICATED, 50 Lbs, Recall # V-112-6 CODE Product manufactured from 02/01/2005 until 06/06/2006 RECALLING FIRM/MANUFACTURER Alabama Farmers Cooperative, Inc., Decatur, AL, by telephone, fax, email and visit on June 9, 2006. FDA initiated recall is complete.

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

VOLUME OF PRODUCT IN COMMERCE 125 tons DISTRIBUTION AL and FL END OF ENFORCEMENT REPORT FOR AUGUST 2, 2006
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<http://www.fda.gov/Safety/Recalls/EnforcementReports/2006/ucm120413.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

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<http://www.fda.gov/bbs/topics/enforce/2...00963.html>

CJD WATCH MESSAGE BOARD TSS 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 ruminant derived meat and bone meal.

VOLUME OF PRODUCT IN COMMERCE 10,878.06 tons

DISTRIBUTION Nationwide

END OF ENFORCEMENT REPORT FOR July 12, 2006

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<http://www.fda.gov/bbs/topics/enforce/2...00960.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

'ANIMAL PROTEIN' SEARCH 9/9/02

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Darling International, Inc. 5/07/02 Seattle District Office Animal Proteins Prohibited in Ruminant Feed/Misbranded [PDF] [HTML]

All American Feed & Tractor 4/01/02 Seattle District Office Animal Proteins Prohibited in Ruminant Feed/Adulterated [PDF] [HTML]

Tyson Foods 2/12/02 Seattle District Office Animal Proteins Prohibited in Ruminant Feed/Misbranded [PDF] [HTML]

The Feed Bucket 12/11/01 Atlanta District Office Animal Proteins Prohibited in Ruminant Feed/Adulterated/Misbranded [PDF] [HTML]

Finlayson Ag Center 11/08/01 Minneapolis District Office Animal Proteins Prohibited in Ruminant Feed/Adulterated [PDF] [HTML]

Dixon Feeds, Inc. 10/24/01 Seattle District Office Animal Proteins Prohibited in Ruminant Feed/Adulterated [PDF] [HTML]

Buckeye Feed Mills, Inc. 9/20/01 Cincinnati District Office Animal Proteins Prohibited in Ruminant Feed/Adulterated/Misbranded [PDF] [HTML]

Wilcox Farms, Inc. 9/14/01 Seattle District Office Animal Proteins Prohibited in Ruminant Feed [PDF] [HTML]

[http://www.accessdata.fda.gov/scripts/wlcfm/full_text.cfm?
full_text=animal+protein&Search=Search](http://www.accessdata.fda.gov/scripts/wlcfm/full_text.cfm?full_text=animal+protein&Search=Search)

now, compare search on 8/8/01...tss

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'ANIMAL PROTEIN' SEARCH 8/8/01

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Date: Tue, 28 Aug 2001 11:13:43 -0700 Reply-

To: BSE-L Sender: Bovine Spongiform Encephalopathy BSE-L

From: "Terry S. Singeltary Sr."

Subject: MAD COW FEED BAN WARNING LETTERS U.S.A. AUGUST
8, 2001

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

Seattle District Pacific Region 22201 23rd Drive SE Bothell, WA
98021-4421

Telephone: 426-486-8788 FAX: 426-483-4996

August 8, 2001

VIA CERTIFIED MAIL RETURN RECEIPT REQUESTED

In reply refer to Warning Letter SEA 01-75

William W. Himmelspach, Owner 22195 S.W. 78th Tualatin,
Oregon 97062

WARNING LETTER

Dear Mr. Himmelspach:

An investigation at your animal feed manufacturing operation located at 22195 S.W. 78th Tualatin, Oregon 97062, conducted by a Food and Drug Administration investigator on July 12, 2001, found significant deviations from the requirements set forth in Title 21, Code of Federal Regulations, Part 589.2000 - Animal Proteins Prohibited in Ruminant Feed. The regulation is intended to prevent the establishment and amplification of Bovine Spongiform Encephalopathy (BSE). Such deviations cause products being manufactured at this facility to be adulterated within the meaning of Section 402(a)(2)(C), and 402(a)(4) of the Federal Food, Drug and Cosmetic Act (the Act).

Our investigation found a failure to separate the receipt, processing, and storage of the product containing prohibited material from non-prohibited material; failure to establish a written system, including clean-out and flushing procedures, to avoid commingling and cross-contamination of common equipment; and failure to maintain records sufficient to track the materials throughout the receipt, processing, and distribution of your products.

In addition, our investigation found a failure to label your products with the required cautionary statement "Do Not Feed to Cattle or Other Ruminants," Your pig feeds, containing prohibited materials, were not labeled with the cautionary statement, and you reuse poly-tote bags for ruminant feed and pig feed, where the bags could become contaminated with prohibited material. The FDA suggests the statement be distinguished by different type size or color or other means of highlighting the statement so that it is easily noticed by a purchaser.

The above is not intended to be an all-inclusive list of deviations from the regulations. As a manufacturer of materials intended for animal feed use, you are responsible for assuring that your overall operation and the products you manufacture and distribute are in compliance with

William W. Himmelspach Tualatin, Oregon Re: Warning Letter SEA 01-75 Page 2

your overall operation and the products you manufacture and distribute are in compliance with the law. We have enclosed a copy of the FDA's Small Entity Compliance Guide to assist you with complying with the regulation.

You should take prompt action to correct these violations, and you should establish a system whereby such violations do not recur. Failure to promptly correct these violations may result in regulatory action without further notice, such as seizure and/or injunction.

You should notify this office in writing within 15 working days of receipt of this letter, of the steps you have taken to bring your firm into compliance with the law. Your response should include an explanation of each step being taken to correct the violations, and prevent their recurrence. If corrective action cannot be completed in 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 that corrections have been made.

Your reply should be directed to the Food and Drug Administration, Attention: Bruce Williamson, Compliance Officer. If you have any questions please contact Mr. Williamson at (425) 483-4976.

Sincerely,

Charles M. Breen District Director

Enclosure; Form FDA 483 Small Entity Compliance Guide

http://www.fda.gov/foi/warning_letters/g1619d.pdf

Warning Letters Index - Search Form Results Company Name Date Issued Issuing Office

Subject

File Adrian Elevator, Inc. 5/03/01 Minneapolis District Office
Animal Proteins Prohibited in Ruminant Feed

View File Alaska Garden and Pet Supply, Inc. 4/27/01 Seattle
District Office Animal Proteins Prohibited in Ruminant Feed

View File Bryan Enterprises 2/20/01 Cincinnati District Office
Feed Mill/Animal Proteins Prohibited in Ruminant
Feed/Adulterated

View File Carrollton Farmers Exchange 7/12/01 Cincinnati
District Office Animal Proteins Prohibited in Ruminant Feed

View File Centerburg Mill and General Store, Inc 3/23/01
Cincinnati District Office Animal Proteins Prohibited in Ruminant
Feed

View File Centerburg Mill and General Store, Inc. 5/23/01
Cincinnati District Office Animal Proteins Prohibited in Ruminant
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View File Central Ohio Farmers Cooperative, Inc. 5/24/01
Cincinnati District Office Animal Protein Prohibited in Ruminant
Feed

View File Champaign Landmark, Inc. 3/05/01 Cincinnati District
Office Animal Proteins Prohibited in Ruminant Feed/Misbranded

View File Countryline Co-Op, Inc. 5/14/01 Cincinnati District
Office Animal Proteins Prohibited in Ruminant Feed

View File Dorset Milling 4/16/01 Cincinnati District Office Animal
Proteins Prohibited in Ruminant Feed

View File Earl B. Olson Feed Mill 4/23/01 Minneapolis District
Office Animal Proteins Prohibited in Ruminant Feed

View File Faler Feed Store, Inc. 3/21/01 Cincinnati District Office
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View File Farmers Mill & Elevator Company 3/30/01 Atlanta
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View File Farnam Companies, Inc. 7/20/01 Kansas City District
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View File Greeley Elevator Company 4/04/01 Denver District
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View File Hartville Elevator Company, Inc. 2/22/01 Cincinnati
District Office Feed Mill/Animal Proteins Prohibited in Ruminant
Feed/Adulterated

View File Himmelspace, William W. 8/08/01 Seattle District Office
Animal Proteins Prohibited in Ruminant Feed

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Animal Protein Prohibited in Ruminant Feed

[View File Medina Landmark, Inc. 3/23/01 Cincinnati District Office Animal Proteins Prohibited in Ruminant Feed](#)

[View File Minister Farmers Cooperative Exchange, Inc. 4/10/01 Cincinnati District Office Animal Proteins Prohibited in Ruminant Feed/Feed Mill](#)

[View File Peco Foods, Inc. 2/23/01 New Orleans District Office CGMP Requirements for Medicated Feeds/Animal Proteins Prohibited in Ruminant Feed](#)

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[View File River Valley Co-Op 5/22/01 Cincinnati District Office Animal Proteins Prohibited in Ruminant Feed](#)

[View File Round Lake Farmers Coop. 5/30/01 Minneapolis District Office Animal Proteins Prohibited in Ruminant Feed](#)

[View File Rudy, Inc. 3/22/01 Cincinnati District Office Animal Proteins Prohibited in Ruminant Feed](#)

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[View File Shields Feed and Supply Company 3/07/01 New Orleans District Office Animal Proteins Prohibited in Ruminant Feed](#)

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View File Z & W Mill, Inc. 3/27/01 Denver District Office Animal
Proteins Prohibited in Ruminant Feed

View File

<http://63.75.126.221/scripts/wlcfm/resultswl.cfm>

(TYPE IN 'ANIMAL PROTEIN')

we must not forget the ANIMAL PROTEIN FED TO DEER/ELK.
those warning letters were stopped long ago;

Subject: MAD DEER/ELK DISEASE AND POTENTIAL SOURCES
Date: Sat, 25 May 2002 18:41:46 -0700 From: "Terry S. Singeltary
Sr." Reply-To: BSE-L To: BSE-L

8420-20.5% Antler Developer For Deer and Game in the wild
Guaranteed Analysis Ingredients / Products Feeding Directions

snip...

animal protein

<http://www.surefed.com/deer.htm>

BODES GAME FEED SUPPLEMENT #400 A RATION FOR DEER
NET WEIGHT 50 POUNDS 22.6 KG.

snip...

__animal protein__

<http://www.bodefeed.com/prod7.htm>

Ingredients

Grain Products, Plant Protein Products, Processed Grain By-
Products, Forage Products, Roughage Products 15%, Molasses
Products, __Animal Protein Products__, snip...

<http://www.bodefeed.com/prod6.htm>

=====

MORE ANIMAL PROTEIN PRODUCTS FOR DEER

Bode's #1 Game Pellets A RATION FOR DEER F3153

GUARANTEED ANALYSIS Crude Protein (Min) 16% Crude Fat
(Min) 2.0% snip...

Ingredients

Grain Products, Plant Protein Products, Processed Grain By-
Products, Forage Products, Roughage Products, 15% Molasses
Products, __Animal Protein Products__, Monocalcium
Phosphate, Dicalcium Phosphate, Salt, snip...

FEEDING DIRECTIONS Feed as Creep Feed with Normal Diet

<http://www.bodefeed.com/prod8.htm>

INGREDIENTS

Grain Products, Roughage Products (not more than 35%), Processed Grain By-Products, Plant Protein Products, Forage Products, ___Animal Protein Products___, L-Lysine, Calcium Carbonate, Salt, Monocalcium/Dicalcium snip...

DIRECTIONS FOR USE

Deer Builder Pellets is designed to be fed to deer under range conditions or deer that require higher levels of protein. Feed to deer during gestation, fawning, lactation, antler growth and pre-rut, all phases which require a higher level of nutrition. Provide adequate amounts of good quality roughage and fresh water at all times.

http://www.profilenutrition.com/Pro...er_pellets.html

DEPARTMENT OF HEALTH & HUMAN SERVICES PUBLIC HEALTH
SERVICE FOOD AND DRUG ADMINISTRATION

April 9, 2001 WARNING LETTER

01-PHI-12 CERTIFIED MAIL RETURN RECEIPT REQUESTED

Brian J. Raymond, Owner Sandy Lake Mills 26 Mill Street P.O. Box
117 Sandy Lake, PA 16145 PHILADELPHIA DISTRICT

Tel: 215-597-4390

Dear Mr. Raymond:

Food and Drug Administration Investigator Gregory E. Beichner conducted an inspection of your animal feed manufacturing operation, located in Sandy Lake, Pennsylvania, on March 23, 2001, and determined that your firm manufactures animal feeds including feeds containing prohibited materials. The inspection found significant deviations from the requirements set forth in Title 21, code of Federal Regulations, part 589.2000 - Animal Proteins Prohibited in Ruminant Feed. The regulation is intended to prevent the establishment and amplification of Bovine Spongiform Encephalopathy (BSE). Such deviations cause products being manufactured at this facility to be misbranded within the meaning of Section 403(f), of the Federal Food, Drug, and Cosmetic Act (the Act).

Our investigation found failure to label your swine feed with the required cautionary statement "Do Not Feed to cattle or other Ruminants" The FDA suggests that the statement be distinguished by different type-size or color or other means of highlighting the statement so that it is easily noticed by a purchaser.

In addition, we note that you are using approximately 140 pounds of cracked corn to flush your mixer used in the manufacture of animal feeds containing prohibited material. This flushed material is fed to wild game including deer, a ruminant animal. Feed material which may potentially contain prohibited material should not be fed to ruminant animals which may become part of the food chain.

The above is not intended to be an all-inclusive list of deviations from the regulations. As a manufacturer of materials intended for animal feed use, you are responsible for assuring that your overall operation and the products you manufacture and distribute are in compliance with the law. We have enclosed a copy of FDA's Small Entity Compliance Guide to assist you with complying with the regulation... blah, blah, blah...

http://www.fda.gov/foi/warning_letters/g1115d.pdf

=====

Subject: USA BSE/TSE RUMINANT-TO-RUMINANT FEED BAN VIOLATIONS "cover-up"

From: "Terry S. Singeltary Sr."

Date: Mon, 2 Dec 2002 11:17:40 -0600 To: BSE-L

Greetings List members,

i have tried to inquire about the USA BSE/TSE feed ban violations with no luck via USDA/APHIS. since about april or may of 2002, the warning letters have ceased to be posted publicly, and at the site CVM and Ruminant feed inspections site url, they have not been updated either. it seems to me the new administration has taken away all rights for the public to view these violations.

where are they now being posted ???

you can hide it, but it will not make it go away.

would/could the USDA/APHIS whom lurk on this list, please comment?

<http://www.fda.gov/cvm/efoi/InspectionListDescriptionforHP.htm>

GBR risk assessment of BSE should be changed to all TSEs.

USA GBR II should be changed to GBR III immediately!

TSS

Terry S. Singeltary Sr. wrote:

> X-Virus-Scanner: Found to be clean > Message-ID: <[log in to
unmask]> > Date: Tue, 20 May 2003 08:39:31 -0500 > Reply-To:
Bovine Spongiform Encephalopathy Sender: Bovine Spongiform
Encephalopathy

> > From: "Terry S. Singeltary Sr." <[log in to unmask]> > Subject:
MAD COW FEED BAN WARNING LETTER USA 2003 > >
Bovine Spongiform Encephalopathy > > Public Health
Service Food and Drug Administration > > Minneapolis District
Office Central Region 212 Third Avenue South > Minneapolis, MN
55401 Telephone: (612) 334-4100 FAX: (612) 334-4134 > > May
6, 2003 > > WARNING LETTER CERTIFIED MAIL RETURN
RECEIPT REQUESTED Refer to MIN 03 > - 20 > > Steve L. Denk
President Barr Animal Foods A Division of Barr > Enterprises, Inc.
W7276 Chickadee Road Greenwood, Wisconsin 54437 > > Dear
Mr. Denk: > > On April 8, 2003, an investigator from the Food and
Drug > Administration (FDA) inspected your rendering and
animal feed > manufacturing operation located at W7276
Chickadee Road, Greenwood, > WI. This inspection found
significant deviations from the requirements > set forth in Title 21,
Code of Federal Regulations, Part 589.2000, > "Animal Proteins
Prohibited in Ruminant Feed" (21 CFR 589.2000). The >
regulation is intended to prevent the establishment and
amplification > of Bovine Spongiform Encephalopathy (BSE).
Under 21 CFR > 589.2000(g)(2), such deviations cause products
being manufactured > and/or distributed by this facility to be
deemed misbranded within the > meaning of Section 403(a)(1) of
the Federal Food, Drug and Cosmetic > Act (the Act), and these
products may not be lawfully introduced, or > delivered for
introduction, into interstate commerce. > > Products that contain
or may contain protein derived from mammalian > tissues and are
intended for use in animal feed must be labeled with > the
cautionary statement, "Do not feed to cattle or other ruminants." >
This is required by 21 CFR 589.2000(c)(1)(i). The FDA suggests
the > statement be distinguished by different type size or color, or
other > means of highlighting the statement so that it is easily
noticed by a > purchaser. Our inspection found that you are not
labeling your > 50-pound blocks of frozen beef and bulk loads of
beef bone chips and > rendering waste, which are intended for
animal feed, with that caution > statement. As a result, these
products are misbranded within the > meaning of Section 403(a)
(1) of the Act. > > The above is not intended to be an all-inclusive
list of deviations > from the regulations. As a renderer and
manufacturer of materials > intended for animal feed use, you are
responsible for ensuring that > your overall operation and the
products you manufacture and distribute > are in compliance with
the law. We have enclosed a copy of the FDA's > Small Entity
Compliance Guide Nos. 67 and 68 to assist you with > complying
with the regulation. > > You should take prompt action to correct
these violations and you > should establish a system whereby
such violations do not recur. > Failure to promptly correct these
violations may result in regulatory > action without further
notice. These actions include, but are not > limited to, seizure
and/or injunction. > > It is necessary for you to take action on
this matter now. Please > provide this office a written response
within 15 working days of > receipt of this letter with the steps you
have taken to bring your > firm into compliance with the law.
Your response should include an > explanation of each step being
taken to correct the violations and > prevent their recurrence. If
corrective action cannot be taken within > 15 working days, state
the reason for the delay and the date by which > the corrections
will be completed. Please include copies of any > available
documentation demonstrating that corrections have been made.
> > Your reply should be directed to Compliance Officer Timothy
G. Philips > at the address indicated on the letterhead. > >
Sincerely, > > /s/ > > David R. Yost for W. Charles Becoat > >
Minneapolis District > >

http://www.fda.gov/foi/warning_letters/g4000d.htm > >
 Greetings List Members, > > what a surprise to see this posting of
 warning letter for feeding > ruminants to ruminants in the USA,
 simply astonishing. my question is, > if they cannot stop this
 practice of feeding cows to cows in the USA > in 2003, after some
 6 years (since 8/4/97 partial and voluntary), how > in the world
 do they plan on enforcing a ban on deer/elk products from >
 entering the feed market? > > FDA Issues Draft Guidance on Use of
 Material From Deer and Elk in > Animal Feed > >
<http://www.fda.gov/bbs/topics/ANSWERS/2003/ANS01220.html>
 > > > TSS > > RECALLS AND FIELD CORRECTIONS: VETERINARY
 MEDICINE- CLASS II > _____ >
 > PRODUCT > Unlabeled bulk "Cattle Feed" sold by weight to
 user/farmers who pick it > up at the firm. Product is a ruminant
 feed used to feed beef cattle. > Recall # V-046-3. > CODE >
 Product is bulk and uncoded. > RECALLING
 FIRM/MANUFACTURER > Zephyr Feed Company, Zephyrhills,
 FL., by letters on March 19, 2003 and > March 26, 2003. FDA
 initiated recall is ongoing. > REASON > Cattle feed was distributed
 to farmers that may contain prohibited > protein for ruminants. >
 VOLUME OF PRODUCT IN COMMERCE > 517,990 lbs. >
 DISTRIBUTION > FL. > >
<http://www.fda.gov/bbs/topics/enforce/2003/ENF00792.html>
 > > > RECALLS AND FIELD CORRECTIONS: VETERINARY
 MEDICINE--CLASS II > > _____ >
 PRODUCT > Red Rooster Booster, Super Gallo (brand), 60
 capsules. Recall # V-011-3. > CODE > All codes. > RECALLING
 FIRM/MANUFACTURER > Thomas Laboratories, Tolleson, AZ, by
 letters on or about November 8, > 2002. State initiated recall is
 ongoing. > REASON > Is not labeled "Do not feed to cattle or other
 ruminants" and contains a > bovine tissue derivative. > VOLUME
 OF PRODUCT IN COMMERCE > Unknown. > DISTRIBUTION >
 Nationwide. > > _____ > >
 PRODUCT > CATTLE FEED, Flock #999, Date: 12/5/02, Quantity
 8000, Load A, Feed > C205, Grower# Z001, Tag C100. Recall # V-
 012-3. > CODE > C-205, C-210, C-220, C-302, C-406 and all other
 codes manufactured and > distributed by Grove River Mills, Inc.,
 > RECALLING FIRM/MANUFACTURER > Grove River Mills Inc.,
 Pendergrass, GA, by telephone and letter on > December 9, 2002.
 Firm initiated recall is ongoing. > REASON > Cattle Feed
 contaminated with prohibited materials. > VOLUME OF PRODUCT
 IN COMMERCE > 235,668 lbs. > DISTRIBUTION > GA. > > END OF
 ENFORCEMENT REPORT FOR FEBRUARY 5, 2003 > >
<http://www.fda.gov/bbs/topics/enforce/2003/ENF00781.html>
 > > another description here; > > > FEBRUARY 2003 > >
 PRODUCT Red Rooster Booster, Super Gallo (brand), 60 capsules.
 > > CODE All codes. > RECALLING FIRM/MANUFACTURER
 Thomas Laboratories, Tolleson, AZ, > > REASON Is not labeled
 "Do not feed to cattle or other ruminants" and > contains a bovine
 tissue derivative. > VOLUME OF PRODUCT IN COMMERCE
 Unknown. > DISTRIBUTION Nationwide. > > > PRODUCT CATTLE
 FEED, Flock #999, Date: 12/5/02, Quantity 8000, Load A, > Feed
 C205, Grower# Z001, Tag C100. > > CODE C-205, C-210, C-220,
 C-302, C-406 and all other codes manufactured > and distributed
 by Grove River Mills, Inc., > RECALLING
 FIRM/MANUFACTURER Grove River Mills Inc., Pendergrass, GA,
 > > REASON Cattle Feed contaminated with prohibited materials.
 > VOLUME OF PRODUCT IN COMMERCE 235,668 lbs. >
 DISTRIBUTION GA. > >
<http://www.recalls.org/vet2003.html> > > Red Rooster
 Booster - 60 Capsules \$7.95 > 210-4610-C03 > > >
 Concentrated nutritional supplement for body building
 gamebirds. > > >
http://www.thomasveterinarydrug.com/mailorder/catalog/product_info.php?manufacturers_id=11&products_id=65 > > > interesting; >
 Bone Meal with D3 - 25 lb. Powder \$69.95 > 520-0210-P09 > > >
http://www.thomasveterinarydrug.com/mailorder/catalog/product_info.php?manufacturers_id=11&products_id=651 > > > >

Subject: MAD COW FEED BAN WARNING LETTER USA (a real hum
 dinger) !

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

Reply-To: Bovine Spongiform Encephalopathy <[log in to
 unmask]>

Date: Tue, 10 Jun 2003 07:43:21 -0500

Bovine Spongiform Encephalopathy <[log in to
unmask]> #####

Public Health Service Food and Drug Administration

New Orleans District Nashville Branch Office Plus Park Blvd.
Nashville, TN 37217 Tel: 615-781-6388 FAX: 615-781-6383

May 22, 2003

VIA FEDERAL EXPRESS OVERNIGHT DELIVERY

Mr. John F. Turner, Owner, Manager Millstone Agri Distributors
3721 E. Lamar Alexander Highway Maryville, TN 37804

Warning Letter No 03-NSV-16

Dear Mr. Turner:

An inspection of your animal feed manufacturing operation, located at Maryville, Tennessee conducted by a U.S. Food and Drug Administration investigator on February 13, 2003, found significant deviations from the requirements set forth in Title 21, Code of Federal Regulations (21 CFR.), Part 589.2000 - Animal Proteins Prohibited in Ruminant Feed. The regulation is intended to prevent the establishment and amplification of Bovine Spongiform Encephalopathy (BSE). Because you failed to follow this rule, products you manufactured and distributed are adulterated within the meaning of Sections 402(a)(2)(C) and 402(a)(4) of the Federal Food, Drug, and Cosmetic Act (the Act) since they contain an unsafe food additive and were prepared, packed, or held under insanitary conditions . . . whereby [they] may have been rendered injurious to health. Feed you manufactured also was misbranded within the meaning of Section 403(a)(1) of the Act because of your failure to follow this rule.

Our investigation found the following violations of 21 C.F.R. 589.2000:

1. Failure to separate the receipt, processing, and storage of products containing prohibited material from products not containing prohibited material [21 C.F.R. 589.2000(e)(1)(iv)];
2. Failure to establish written procedures, including clean-out and flushing procedures, to avoid commingling and cross-contamination of common equipment [21 C.F.R. 589.2000(e)(1)(iii)(B)];

3. Failure to maintain records sufficient to track prohibited materials throughout the receipt, processing, and distribution of your products [21 C.F.R. 589.2000(c)(1)(ii)];

4. Failure to provide for measures to avoid commingling or cross-contamination of feeds intended for ruminants and feeds intended for nonruminants that may contain prohibited materials [21 C.F.R. 589.2000(c)(1)(iii)]. Specifically, our investigation found that the ruminant product 10% Beef Conditioned was formulated primarily with screenings and fines derived from previously manufactured non-ruminant products, Premium Rooster Kicker in particular, that contain or may contain prohibited material. Such deviations cause the ruminant product 10% Beef Conditioner being manufactured at this facility to be adulterated within the meaning of Sections 402(a)(2)(C) and 402(a)(4) of the Act;

5. Failure to label your non-ruminant products with the required cautionary statement Do not Feed to Cattle or Other Ruminants [21 C.F.R. 589.2000(c)(1)(ii)]. Our investigation specifically found that dog food containing prohibited material was added as an ingredient to your product Premium Rooster Kicker. The failure of these feeds to bear the required BSE warning statement causes them to be misbranded within the meaning of Section 403(f) of the Act.

The above is not intended to be an all-inclusive list of deviations from the regulations. As a manufacturer of materials intended for animal feed use, you are responsible for assuring that your overall operation and the products you manufacture and distribute are in compliance with the law. We have enclosed a copy of the FDA's Small Entity Compliance Guide to assist you with complying with the regulations.

You should take prompt action to correct these violations, and you should establish a system whereby such violations do not recur. Failure to promptly correct these violations may result in regulatory action without further notice, such as seizure and/or injunction. You should notify this office in writing within 15 working days of receipt of this letter of the steps you have taken to bring your firm into compliance with the law. Your response should include an explanation of each step being taken to correct the violations, and prevent their recurrence. If corrective action cannot be completed in 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 that corrections have been made.

Your reply should be directed to the attention of Joseph E. Hayes, Compliance Officer, U.S. Food and Drug Administration, 297 Plus Park Boulevard, Nashville, TN 37217.

Sincerely,

/s/ Carl E. Draper Director, New Orleans District Office

http://www.fda.gov/foi/warning_letters/g4056d.htm

Subject: USDA OIG SEMI ANNUAL 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>

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 flounder on 03 Jul 2015 at 16:53 GMT

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

Conclusions We demonstrated that the agent of L-BSE can be transmitted by the oral route from cattle to mouse lemurs. As expected, orally inoculated animals survived longer than IC-inoculated animals. Orally inoculated lemurs had less severe clinical signs and symptoms, with no evidence of motor dysfunction. It was previously suggested that the agent of L-BSE might be involved in the foodborne transmission of a prion disease in mink (11,12), a species in which several outbreaks of transmissible mink encephalopathy had been identified, notably in the United States (13).

Our study clearly confirms, experimentally, the potential risk for interspecies oral transmission of the agent of L-BSE. In our model, this risk appears higher than that for the agent of classical BSE, which could only be transmitted to mouse lemurs after a first passage in macaques (14). We report oral transmission of the L-BSE agent in young and adult primates. Transmission by the IC route has also been reported in young macaques (6,7). A previous study of L-BSE in transgenic mice expressing human PrP suggested an absence of any transmission barrier between cattle and humans for this particular strain of the agent of BSE, in contrast to findings for the agent of classical BSE (9). Thus, it is imperative to maintain measures that prevent the entry of tissues from cattle possibly infected with the agent of L-BSE into the food chain.

Dr Mestre-Francés is an assistant professor at the École Pratique des Hautes Études. Her research focuses on neurodegenerative diseases (Alzheimer disease, prion diseases) in the nonhuman primate model *Microcebus murinus*.

http://wwwnc.cdc.gov/eid/article/18/1/11-1092_article.htm

*** Needless conflict ***

Nature 485, 279–280 (17 May 2012) doi:10.1038/485279b

Published online 16 May 2012

Terry S. Singeltary Sr. said:

I kindly wish to submit the following please ;

<http://www.nature.com/nature/journal/v485/n7398/full/485279b.html>

"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.

http://www.upi.com/Health_News/2006/03/15/Analysis-What-that-mad-cow-means/12841142465253/

Thursday, October 22, 2015

*** Former Ag Secretary Ann Veneman talks women in agriculture and we talk mad cow disease USDA and what really happened ***

<http://madcowusda.blogspot.com/2015/10/former-ag-secretary-ann-veneman-talks.html>

Thursday, January 14, 2016

*** EMERGING ANIMAL DISEASES Actions Needed to Better

GAO

http://animalhealthreportpriontse.blogspot.com/2016/01/emerging-animal-diseases-actions-needed_14.html

Wednesday, March 2, 2016

*** RANCHO He did not know that they were placing healthy cow heads next to suspect carcasses BSE/TSE Prion ***

<http://madcowusda.blogspot.com/2016/03/rancho-he-did-not-know-that-they-were.html>

Sunday, June 14, 2015

Larry's Custom Meats Inc. Recalls Beef Tongue Products That May Contain Specified Risk Materials BSE/TSE Prion

<http://madcowusda.blogspot.com/2015/06/larrys-custom-meats-inc-recalls-beef.html>

Thursday, June 12, 2014

Missouri Firm Recalls Ribeye and Carcass Products That May Contain Specified Risk Materials 4,012 pounds of fresh beef products because the dorsal root ganglia may not have been completely removed

<http://madcowusda.blogspot.com/2014/06/missouri-firm-recalls-ribeye-and.html>

Saturday, November 10, 2012

Wisconsin Firm Recalls Beef Tongues That May Contain Specified Risk Materials Nov 9, 2012 WI Firm Recalls Beef Tongues

<http://bseusa.blogspot.com/2012/11/wisconsin-firm-recalls-beef-tongues.html>

Saturday, July 23, 2011

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

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

Sunday, October 18, 2009

Wisconsin Firm Recalls Beef Tongues That Contain Prohibited
Materials SRM WASHINGTON, October 17, 2009

<http://madcowfeed.blogspot.com/2009/10/wisconsin-firm-recalls-beef-tongues.html>

Thursday, October 15, 2009

Nebraska Firm Recalls Beef Tongues That Contain Prohibited
Materials SRM WASHINGTON, Oct 15, 2009

<http://madcowfeed.blogspot.com/2009/10/nebraska-firm-recalls-beef-tongues-that.html>

Thursday, June 26, 2008

Texas Firm Recalls Cattle Heads That Contain Prohibited Materials

<http://madcowfeed.blogspot.com/2008/06/texas-firm-recalls-cattle-heads-that.html>

Tuesday, July 1, 2008

Missouri Firm Recalls Cattle Heads That Contain Prohibited
Materials SRMs

<http://madcowfeed.blogspot.com/2008/07/missouri-firm-recalls-cattle-heads-that.html>

Friday, August 8, 2008

Texas Firm Recalls Cattle Heads That Contain Prohibited Materials

SRMs 941,271 pounds with tonsils not completely removed

<http://madcowfeed.blogspot.com/2008/08/texas-firm-recalls-cattle-heads-that.html>

Saturday, April 5, 2008

SRM MAD COW RECALL 406 THOUSAND POUNDS CATTLE HEADS WITH TONSILS KANSAS

<http://cjdmadcowbaseoct2007.blogspot.com/2008/04/srm-mad-cow-recall-406-thousand-pounds.html>

Wednesday, April 30, 2008

Consumption of beef tongue: Human BSE risk associated with exposure to lymphoid tissue in bovine tongue in consideration of new research findings

<http://cjdmadcowbaseoct2007.blogspot.com/2008/04/consumption-of-beef-tongue-human-bse.html>

Wednesday, April 30, 2008

Consumption of beef tongue: Human BSE risk associated with exposure to lymphoid tissue in bovine tongue in consideration of new research findings

<http://cjdmadcowbaseoct2007.blogspot.com/2008/04/consumption-of-beef-tongue-human-bse.html>

Friday, October 15, 2010

BSE infectivity in the absence of detectable PrPSc accumulation in the tongue and nasal mucosa of terminally diseased cattle

<http://bseusa.blogspot.com/2010/10/bse-infectivity-in-absence-of.html>

SPECIFIED RISK MATERIALS SRMs

<http://madcowspontaneousnot.blogspot.com/2008/02/specified-risk-materials-srm.html>

seems USDA NSLP et al thought that it would be alright, to feed our children all across the USA, via the NSLP, DEAD STOCK DOWNER COWS, the most high risk cattle for mad cow type disease, and other dangerous pathogens, and they did this for 4 years, that was documented, then hid what they did by having a recall, one of the largest recalls ever, and they made this recall and masked the reason for the recall due to animal abuse (I do not condone animal abuse), not for the reason of the potential for these animals to have mad cow BSE type disease (or other dangerous and deadly pathogens). these TSE prion disease can lay dormant for 5, 10, 20 years, or longer, WHO WILL WATCH OUR CHILDREN FOR THE NEXT 5 DECADES FOR CJD ???

Saturday, September 21, 2013

Westland/Hallmark: 2008 Beef Recall A Case Study by The Food Industry Center January 2010 THE FLIM-FLAM REPORT

<http://downercattle.blogspot.com/2013/09/westlandhallmark-2008-beef-recall-case.html>

DID YOUR CHILD CONSUME SOME OF THESE DEAD STOCK DOWNER COWS, THE MOST HIGH RISK FOR MAD COW DISEASE ??? this recall was not for the welfare of the animals. ...tss you can check and see here ; (link now dead, does not work...tss)

http://www.fns.usda.gov/fns/safety/pdf/Hallmark-Westland_byState.pdf

see listings of schools from state to state, county to county, was your child exposed ;

try this link ;

<http://downercattle.blogspot.com/2013/09/school-food-authorities-affected-by.html>

<http://downercattle.blogspot.com/>

2015-2016

Friday, March 18, 2016

CFSAN Constituent Update: FDA Announces Final Rule on Bovine Spongiform Encephalopathy BSE MAD COW TSE PRION

Center for Food Safety and Applied Nutrition - Constituent Update

<http://bseusa.blogspot.com/2016/03/cfsan-constituent-update-fda-announces.html>

Thursday, February 25, 2016

U.S. Food & Drug Administration (FDA) FDA/CFSAN Cosmetics
Update: Cosmetics Program; Import and Domestic and
Transmissible Spongiform Encephalopathy TSE Prion Disease Risk
Factors

***WARNING TO ALL CONSUMERS AND COUNTRIES AROUND
THE WORLD***

***Note: FDA labs do not conduct BSE analysis and thus no
sampling guidance is issued for BSE. ***

<http://transmissiblespongiformencephalopathy.blogspot.com/2016/02/us-food-drug-administration-fda.html>

Tuesday, March 15, 2016

Docket No. FDA-2016-N-0321 Risk Assessment of Foodborne
Illness Associated with Pathogens from Produce Grown in Fields
Amended with Untreated Biological Soil Amendments of Animal
Origin; Request for Comments, Scientific Data, and Information
Singeltary Submission

<http://animalhealthreportpriontse.blogspot.com/2016/03/docket-no-fda-2016-n-0321-risk.html>

Friday, February 05, 2016

Report of the Committee on Wildlife Diseases FY 2015 CWD/TSE
PRION Detections in Farmed Cervids and Wild

<http://chronic-wasting-disease.blogspot.com/2016/02/report-of-committee-on-wildlife.html>

Saturday, February 6, 2016

*** Secretary's Advisory Committee on Animal Health; Meeting
[Docket No. APHIS-2016-0007] Singeltary Submission ***

<http://animalhealthreportpriontse.blogspot.com/2016/02/secretarys-advisory-committee-on-animal.html>

Sunday, January 17, 2016

Of Grave Concern Heidenhain Variant Creutzfeldt Jakob Disease

<http://creutzfeldt-jakob-disease.blogspot.com/2016/01/of-grave-concern-heidenhain-variant.html>

Saturday, February 13, 2016

The Risk of Prion Infection through Bovine Grafting Materials in dentistry

<http://bovineprp.blogspot.com/2016/02/the-risk-of-prion-infection-through.html>

In Confidence - Perceptions of unconventional slow virus diseases of animals in the USA - APRIL-MAY 1989 - GA H Wells

3. Prof. A. Robertson gave a brief account of BSE. The US approach was to accord it a very low profile indeed. Dr. A Thiermann showed the picture in the "Independent" with cattle being incinerated and thought this was a fanatical incident to be avoided in the US at all costs. ...

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

CANINE AND FELINE TSE PRION AND PET FOOD

Assessing Transmissible Spongiform Encephalopathy Species Barriers with an In Vitro Prion Protein Conversion Assay

Christopher J. Johnson¹, Christina M. Carlson², Aaron R. Morawski³, Alyson Manthei⁴, Neil R. Cashman⁵

¹USGS National Wildlife Health Center, ²Department of Soil Science, University of Wisconsin–Madison, ³Laboratory of Immunology, National Institute of Allergy and Infectious Diseases, National Institutes of Health, ⁴Meriel Veterinary Scholars Program, School of Veterinary Medicine, University of Wisconsin–Madison, ⁵Department of Neurology, University of British Columbia

Summary

Measuring the barrier to the interspecies transmission of prion diseases is challenging and typically involves animal challenges or biochemical assays. Here, we present an in vitro prion protein conversion assay with the ability to predict species barriers.

Date Published: 3/10/2015, Issue 97; doi: 10.3791/52522

Keywords: Medicine, Issue 97, Prion, species barrier, conversion, immunoblotting, transmissible spongiform encephalopathy, interspecies transmission Cite this Article

Johnson, C. J., Carlson, C. M., Morawski, A. R., Manthei, A., Cashman, N. R. Assessing Transmissible Spongiform Encephalopathy Species Barriers with an In Vitro Prion Protein Conversion Assay. *J. Vis. Exp.* (97), e52522, doi:10.3791/52522 (2015). Abstract

Studies to understanding interspecies transmission of transmissible spongiform encephalopathies (TSEs, prion diseases) are challenging in that they typically rely upon lengthy and costly in vivo animal challenge studies. A number of in vitro assays have been developed to aid in measuring prion species barriers, thereby reducing animal use and providing quicker results than animal bioassays. Here, we present the protocol for a rapid in vitro prion conversion assay called the conversion efficiency ratio (CER) assay. In this assay cellular prion protein (PrPC) from an uninfected host brain is denatured at both pH 7.4 and 3.5 to produce two substrates. When the pH 7.4 substrate is incubated with TSE agent, the amount of PrPC that converts to a proteinase K (PK)-resistant state is modulated by the original host's species barrier to the TSE agent. In contrast, PrPC in the pH 3.5 substrate is misfolded by any TSE agent. By comparing the amount of PK-resistant prion protein in the two substrates, an assessment of the host's species barrier can be made. We show that the CER assay correctly predicts known prion species barriers of laboratory mice and, as an example, show some preliminary results suggesting that bobcats (*Lynx rufus*) may be susceptible to white-tailed deer (*Odocoileus virginianus*) chronic wasting disease agent.

<http://www.jove.com/video/52522/assessing-transmissible-spongiform-encephalopathy-species-barriers>

>>> show some preliminary results suggesting that bobcats (*Lynx rufus*) may be susceptible to white-tailed deer (*Odocoileus virginianus*) chronic wasting disease agent.

AD.63: Susceptibility of domestic cats to chronic wasting disease

Amy V. Nalls,¹ Candace Mathiason,¹ Davis Seelig,² Susan Kraft,¹ Kevin Carnes,¹ Kelly Anderson,¹ Jeanette Hayes-Klug¹ and Edward A. Hoover¹

¹Colorado State University; Fort Collins, CO USA; ²University of Minnesota; Saint Paul, MN USA

Domestic and nondomestic cats have been shown to be susceptible to feline spongiform encephalopathy (FSE), almost certainly caused by consumption of bovine spongiform encephalopathy (BSE)-contaminated meat. Because domestic and free-ranging nondomestic felids scavenge cervid carcasses, including those in areas affected by chronic wasting disease (CWD), we evaluated the susceptibility of the domestic cat (*Felis catus*) to CWD infection experimentally. Cohorts of 5 cats each were inoculated either intracerebrally (IC) or orally (PO) with CWD-infected deer brain. At 40 and 42 mo post-inoculation, two IC-inoculated cats developed signs consistent with prion disease, including a stilted gait, weight loss, anorexia, polydipsia, patterned motor behaviors, head and tail tremors, and ataxia, and progressed to terminal disease within 5 mo. Brains from these two cats were pooled and inoculated into cohorts of cats by IC, PO, and intraperitoneal and subcutaneous (IP/SC) routes. Upon subpassage, feline-adapted CWD (FelCWD) was transmitted to all IC-inoculated cats with a decreased incubation period of 23 to 27 mo. FelCWD was detected in the brains of all the symptomatic cats by western blotting and immunohistochemistry and abnormalities were seen in magnetic resonance imaging, including multifocal T2 fluid attenuated inversion recovery (FLAIR) signal hyperintensities, ventricular size increases, prominent sulci, and white matter tract cavitation. Currently, 3 of 4 IP/SC and 2 of 4 PO inoculated cats have developed abnormal behavior patterns consistent with the early stage of feline CWD. These results demonstrate that CWD can be transmitted and adapted to the domestic cat, thus raising the issue of potential cervid-to-feline transmission in nature.

http://www.prion2013.ca/tiny_uploads/forms/Scientific-Program.pdf

www.landesbioscience.com

PO-081: Chronic wasting disease in the cat—Similarities to feline spongiform encephalopathy (FSE)

<http://www.landesbioscience.com/journals/prion/04-Prion6-2-Pathogenesis-and-pathology.pdf>

<http://chronic-wasting-disease.blogspot.com/2012/05/chronic-wasting-disease-cwd-prion2012.html>

[http://www.prion2011.ca/files/PRION_2011_-_Posters_\(May_5-11\).pdf](http://www.prion2011.ca/files/PRION_2011_-_Posters_(May_5-11).pdf)

<http://felinespongiformencephalopathyfse.blogspot.com/2011/08/susceptibility-of-domestic-cats-to-cwd.html>

PO-081: Chronic wasting disease in the cat—Similarities to feline spongiform encephalopathy (FSE)

<http://www.landesbioscience.com/journals/prion/04-Prion6-2-Pathogenesis-and-pathology.pdf>

[http://chronic-wasting-](http://chronic-wasting-disease.blogspot.com/2012/05/chronic-wasting-disease-cwd-prion2012.html)

[disease.blogspot.com/2012/05/chronic-wasting-disease-cwd-prion2012.html](http://chronic-wasting-disease-disease.blogspot.com/2012/05/chronic-wasting-disease-cwd-prion2012.html)

Thursday, May 31, 2012

CHRONIC WASTING DISEASE CWD PRION2012 Aerosol, Inhalation transmission, Scrapie, cats, species barrier, burial, and more

<http://chronic-wasting-disease.blogspot.com/2012/05/chronic-wasting-disease-cwd-prion2012.html>

Monday, August 8, 2011

Susceptibility of Domestic Cats to CWD Infection

<http://felinespongiformencephalopathyfse.blogspot.com/2011/08/susceptibility-of-domestic-cats-to-cwd.html>

Sunday, August 25, 2013

Prion2013 Chronic Wasting Disease CWD risk factors, humans, domestic cats, blood, and mother to offspring transmission

<http://chronic-wasting-disease.blogspot.com/2013/08/prion2013-chronic-wasting-disease-cwd.html>

Feline Spongiform Encephalopathy (FSE) FSE was first identified in the UK in 1990. Most cases have been reported in the UK, where the epidemic has been consistent with that of the BSE epidemic. Some other countries (e.g. Norway, Liechtenstein and France) have also reported cases.

Most cases have been reported in domestic cats but there have also been cases in captive exotic cats (e.g. Cheetah, Lion, Asian leopard cat, Ocelot, Puma and Tiger). The disease is characterised by progressive nervous signs, including ataxia, hyper-reactivity and behavioural changes and is fatal.

The chemical and biological properties of the infectious agent are identical to those of the BSE and vCJD agents. These findings support the hypothesis that the FSE epidemic resulted from the consumption of food contaminated with the BSE agent.

The FSE epidemic has declined as a result of tight controls on the disposal of specified risk material and other animal by-products.

References: Leggett, M.M. et al.(1990) A spongiform encephalopathy in a cat. Veterinary Record. 127. 586-588

Synge, B.A. et al. (1991) Spongiform encephalopathy in a Scottish cat. Veterinary Record. 129. 320

Wyatt, J. M. et al. (1991) Naturally occurring scrapie-like spongiform encephalopathy in five domestic cats. Veterinary Record. 129. 233.

Gruffydd-Jones, T. J. et al.. (1991) Feline spongiform encephalopathy. J. Small Animal Practice. 33. 471-476.

Pearson, G. R. et al. (1992) Feline spongiform encephalopathy: fibril and PrP studies. Veterinary Record. 131. 307-310.

Willoughby, K. et al. (1992) Spongiform encephalopathy in a captive puma (*Felis concolor*). Veterinary Record. 131. 431-434.

Fraser, H. et al. (1994) Transmission of feline spongiform encephalopathy to mice. Veterinary Record 134. 449.

Bratberg, B. et al. (1995) Feline spongiform encephalopathy in a cat in Norway. Veterinary Record 136. 444

Baron, T. et al. (1997) Spongiform encephalopathy in an imported cheetah in France. Veterinary Record 141. 270-271

Zanusso, G et al. (1998) Simultaneous occurrence of spongiform encephalopathy in a man and his cat in Italy. Lancet, V 352, N9134, OCT 3, Pp 1116-1117.

Ryder, S.J. et al. (2001) Inconsistent detection of PrP in extraneural tissues of cats with feline spongiform encephalopathy. Veterinary Record 146. 437-441

Kelly, D.F. et al. (2005) Neuropathological findings in cats with clinically suspect but histologically unconfirmed feline spongiform encephalopathy. Veterinary Record 156. 472-477.

<http://archive.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/bse/other/tse/index.htm#fse>

3 further cheetah cases have occurred, plus 1 lion, plus all the primates, and 20 additional house cats. Nothing has been published on any of these UK cases either. One supposes the problem here with publishing is that many unpublished cases were _born_ long after the feed "ban". Caught between a rock and a hard place: leaky ban or horizontal transmission (or both).

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

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

YOU explained that imported crushed heads were extensively used in the petfood industry ...

<http://web.archive.org/web/20060303042720/http://www.bseinquiry.gov.uk/files/yb/1989/04/14001001.pdf>

In particular I do not believe one can say that the levels of the scrapie agent in pet food are so low that domestic animals are not exposed...

<http://web.archive.org/web/20040301231838/http://www.bseinquiry.gov.uk/files/yb/1989/04/24003001.pdf>

<http://web.archive.org/web/20060303042732/http://www.bseinquiry.gov.uk/files/yb/1989/04/25001001.pdf>

on occasions, materials obtained from slaughterhouses will be derived from sheep affected with scrapie or cattle that may be incubating BSE for use in petfood manufacture...

<http://web.archive.org/web/20060303042739/http://www.bseinquiry.gov.uk/files/yb/1989/05/03007001.pdf>

*** Meldrum's notes on pet foods and materials used

<http://web.archive.org/web/20060303042745/http://www.bseinquiry.gov.uk/files/yb/1989/05/16001001.pdf>

*** BSE & Pedigree Petfoods ***

<http://web.archive.org/web/20060303042725/http://www.bseinquiry.gov.uk/files/yb/1989/05/16002001.pdf>

In 2003, Denver Post reporter Theo Stein interviewed scientists about CWD spreading through deer and elk in Colorado. Dr. Valerius Geist, who paradoxically has become a darling of anti-wolfers, made this assertion about the significance of wolves in containing CWD spread via proteins called prions.

"Wolves will certainly bring the disease to a halt," he said. "They will remove infected individuals and clean up carcasses that could transmit the disease."

Stein added that "Geist and Princeton University biologist Andrew Dobson theorize that killing off the wolf allowed CWD to take hold in the first place."

Wolves aren't alone. In a 2009 study titled "Mountain lions prey selectively on prion-infected mule deer," researchers in Colorado discovered that "adult mule deer killed by mountain lions were more likely to be prion-infected than were deer killed more randomly ... suggesting that mountain lions were selecting for infected individuals when they targeted adult deer."

http://www.jhnewsandguide.com/opinion/columnists/the_new_west_todd_wilkinson/dowolves-cougars-help-curb-diseases/article_cc4458co-08fe-55f2-ab4a-a1852d382352.html

NO, NO, NOT NO, BUT HELL KNOW !!!

PLEASE be careful what you ask for.

recently, canine spongiform encephalopathy has been confirmed.

I proved this in 2005, with a letter from MAFF/DEFRA et al confirming my suspicions of the 'hound study' way back. this was covered up. see documents below.

also, recently, cwd to the domestic cat is a great concern.

even though to date, as far as I am aware of, the cwd study on the mountain lion has not produced any confirmation yet, we already know that the feline species is highly susceptible to the TSE prion. domestic cats and the exotic zoo big cats.

so in my honest opinion, any program that would use wild animals to prey on other wild animals, as a tool to help curb CWD TSE prion disease, would only help enhance the spread of disease, and it would only help spread the disease to other species. ...TSS

Monday, February 14, 2011

THE ROLE OF PREDATION IN DISEASE CONTROL: A
COMPARISON OF SELECTIVE AND NONSELECTIVE REMOVAL ON
PRION DISEASE DYNAMICS IN DEER

NO, NO, NOT NO, BUT HELL NO !

Journal of Wildlife Diseases, 47(1), 2011, pp. 78-93 © Wildlife
Disease Association 2011

OR-09: Canine spongiform encephalopathy—A new form of animal prion disease

Monique David, Mourad Tayebi UT Health; Houston, TX USA

It was also hypothesized that BSE might have originated from an unrecognized sporadic or genetic case of bovine prion disease incorporated into cattle feed or even cattle feed contaminated with prion-infected human remains.¹ However, strong support for a genetic origin of BSE has recently been demonstrated in an H-type BSE case exhibiting the novel mutation E211K.² Furthermore, a specific prion protein strain causing BSE in cattle is believed to be the etiological agent responsible for the novel human prion disease, variant Creutzfeldt-Jakob disease (vCJD).³ Cases of vCJD have been identified in a number of countries, including France, Italy, Ireland, the Netherlands, Canada, Japan, US and the UK with the largest number of cases. Naturally occurring feline spongiform encephalopathy of domestic cats⁴ and spongiform encephalopathies of a number of zoo animals so-called exotic ungulate encephalopathies^{5,6} are also recognized as animal prion diseases, and are thought to have resulted from the same BSE-contaminated food given to cattle and humans, although and at least in some of these cases, a sporadic and/or genetic etiology cannot be ruled out. The canine species seems to display resistance to prion disease and no single case has so far been reported.^{7,8} Here, we describe a case of a 9 week old male Rottweiler puppy presenting neurological deficits; and histological examination revealed spongiform vacuolation characteristic of those associated with prion diseases.⁹ Initial biochemical studies using anti-PrP antibodies revealed the presence of partially proteinase K-resistant fragment by western blotting. Furthermore, immunohistochemistry revealed spongiform degeneration consistent with those found in prion disease and displayed staining for PrPSc in the cortex.

Of major importance, PrPSc isolated from the Rottweiler was able to cross the species barrier transmitted to hamster in vitro with PMCA and in vivo (one hamster out of 5). Furthermore, second in vivo passage to hamsters, led to 100% attack rate (n = 4) and animals displayed atypical lesion profile and shorter incubation period.

In this study, we show that the canine species might be sensitive to prion disease and that PrPSc isolated from a dog can be transmitted to dogs and hamsters in vitro using PMCA and in vivo to hamsters.

If our preliminary results are confirmed, the proposal will have a major impact on animal and public health and would certainly lead to implementing new control measures for 'canine spongiform encephalopathy' (CSE).

References 1. Colchester AC, Colchester NT. The origin of bovine spongiform encephalopathy: the human prion disease hypothesis. *Lancet* 2005; 366:856-61; PMID:16139661; [http://dx.doi.org/10.1016/S0140-6736\(05\)67218-2](http://dx.doi.org/10.1016/S0140-6736(05)67218-2).

2. Richt JA, Hall SM. BSE case associated with prion protein gene mutation. *PLoS Pathog* 2008; 4:e1000156; PMID:18787697;

<http://dx.doi.org/10.1371/journal.ppat.1000156>.

3. Collinge J. Human prion diseases and bovine spongiform encephalopathy (BSE). *Hum Mol Genet* 1997; 6:1699-705; PMID:9300662; <http://dx.doi.org/10.1093/hmg/6.10.1699>.

4. Wyatt JM, Pearson GR, Smerdon TN, Gruffydd-Jones TJ, Wells GA, Wilesmith JW. Naturally occurring scrapie-like spongiform encephalopathy in five domestic cats. *Vet Rec* 1991; 129:233-6; PMID:1957458; <http://dx.doi.org/10.1136/vr.129.11.233>.

5. Jeffrey M, Wells GA. Spongiform encephalopathy in a nyala (*Tragelaphus angasi*). *Vet Pathol* 1988; 25:398-9; PMID:3232315; <http://dx.doi.org/10.1177/030098588802500514>.

6. Kirkwood JK, Wells GA, Wilesmith JW, Cunningham AA, Jackson SI. Spongiform encephalopathy in an arabian oryx (*Oryx leucoryx*) and a greater kudu (*Tragelaphus strepsiceros*). *Vet Rec* 1990; 127:418-20; PMID:2264242.

7. Bartz JC, McKenzie DL, Bessen RA, Marsh RF, Aiken JM. Transmissible mink encephalopathy species barrier effect between ferret and mink: PrP gene and protein analysis. *J Gen Virol* 1994; 75:2947-53; PMID:7964604; <http://dx.doi.org/10.1099/0022-1317-75-11-2947>.

8. Lysek DA, Schorn C, Nivon LG, Esteve-Moya V, Christen B, Calzolari L, et al. Prion protein NMR structures of cats, dogs, pigs, and sheep. *Proc Natl Acad Sci U S A* 2005; 102:640-5; PMID:15647367; <http://dx.doi.org/10.1073/pnas.0408937102>.

9. Budka H. Neuropathology of prion diseases. *Br Med Bull* 2003; 66:121-30; PMID:14522854; <http://dx.doi.org/10.1093/bmb/66.1.121>.

<http://www.landesbioscience.com/journals/prion/01-Prion6-2-OralPresentations.pdf>

Monday, March 26, 2012

CANINE SPONGIFORM ENCEPHALOPATHY: A NEW FORM OF ANIMAL PRION DISEASE

<http://caninespongiformencephalopathy.blogspot.com/2012/03/canine-spongiform-encephalopathy-new.html>

Monday, March 8, 2010

Canine Spongiform Encephalopathy aka MAD DOG DISEASE

=====

2013

Strain characteristics of the in vitro-adapted rabbit and dog BSE agent remained invariable with respect to the original cattle BSE prion, suggesting that the naturally low susceptibility of rabbits and dogs to prion infections should not alter their zoonotic potential if these animals became infected with BSE.

=====

Neurobiology of Disease

Bovine Spongiform Encephalopathy Induces Misfolding of Alleged Prion-Resistant Species Cellular Prion Protein without Altering Its Pathobiological Features

Enric Vidal³, Natalia Fernández-Borges¹, Belén Pintado⁴, Montserrat Ordóñez³, Mercedes Márquez⁶, Dolors Fondevila^{5,6}, Juan María Torres⁷, Martí Pumarola^{5,6}, and Joaquín Castilla^{1,2} + Author Affiliations

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Author contributions: E.V., N.F.-B., and J.C. designed research; E.V., N.F.-B., B.P., M.O., M.M., D.F., and J.C. performed research; E.V., N.F.-B., B.P., and J.C. contributed unpublished reagents/analytic tools; E.V., N.F.-B., B.P., M.O., M.M., D.F., J.M.T., M.P., and J.C. analyzed data; E.V. and J.C. wrote the paper.

Abstract

Bovine spongiform encephalopathy (BSE) prions were responsible for an unforeseen epizootic in cattle which had a vast social, economic, and public health impact. This was primarily because BSE prions were found to be transmissible to humans. Other species were also susceptible to BSE either by natural infection (e.g., felids, caprids) or in experimental settings (e.g., sheep, mice). However, certain species closely related to humans, such as canids and leporids, were apparently resistant to BSE. In vitro prion amplification techniques (saPMCA) were used to successfully misfold the cellular prion protein (PrP_c) of these allegedly resistant species into a BSE-type prion protein. The biochemical and biological properties of the new prions generated in vitro after seeding rabbit and dog brain homogenates with classical BSE were studied. Pathobiological features of the resultant prion strains were determined after their inoculation into transgenic mice expressing bovine and human PrP^C. Strain characteristics of the in vitro-adapted rabbit and dog BSE agent remained invariable with respect to the original cattle BSE prion, suggesting that the naturally low susceptibility of rabbits and dogs to prion infections should not alter their zoonotic potential if these animals became infected with BSE. This study provides a sound basis for risk assessment regarding prion diseases in purportedly resistant species.

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<http://www.jneurosci.org/content/33/18/7778.short>

2005

DEFRA Department for Environment, Food & Rural Affairs

Area 307, London, SW1P 4PQ Telephone: 0207 904 6000 Direct line: 0207 904 6287 E-mail: h.mcdonagh.defra.gsi.gov.uk

GTN: FAX:

Mr T S Singeltary P.O. Box 42 Bacliff Texas USA 77518

21 November 2001

Dear Mr Singeltary

TSE IN HOUNDS

Thank you for e-mail regarding the hounds survey. I am sorry for the long delay in responding.

As you note, the hound survey remains unpublished. However the Spongiform Encephalopathy Advisory Committee (SEAC), the UK Government's independent Advisory Committee on all aspects related to BSE-like disease, gave the hound study detailed consideration at their meeting in January 1994. As a summary of this meeting published in the BSE inquiry noted, the Committee were clearly concerned about the work that had been carried out, concluding that there had clearly been problems with it, particularly the control on the histology, and that it was more or less inconclusive. However was agreed that there should be a re-evaluation of the pathological material in the study.

Later, at their meeting in June 95, The Committee re-evaluated the hound study to see if any useful results could be gained from it. The Chairman concluded that there were varying opinions within the Committee on further work. It did not suggest any further transmission studies and thought that the lack of clinical data was a major weakness.

Overall, it is clear that SEAC had major concerns about the survey as conducted. As a result it is likely that the authors felt that it would not stand up to peer review and hence it was never published. As noted above, and in the detailed minutes of the SEAC meeting in June 95, SEAC considered whether additional work should be performed to examine dogs for evidence of TSE infection. Although the Committee had mixed views about the merits of conducting further work, the Chairman noted that when the Southwood Committee made their recommendation to complete an assessment of possible spongiform disease in dogs, no TSEs had been identified in other species and hence dogs were perceived as a high risk population and worthy of study. However subsequent to the original recommendation, made in 1990, a number of other species had been identified with TSE (e.g. cats) so a study in hounds was less

critical. For more details see-
<http://www.bseinquiry.gov.uk/files/yb/1995/06/21005001.pdf>

As this study remains unpublished, my understanding is that the ownership of the data essentially remains with the original researchers. Thus unfortunately, I am unable to help with your request to supply information on the hound survey directly. My only suggestion is that you contact one of the researchers originally involved in the project, such as Gerald Wells. He can be contacted at the following address.

Dr Gerald Wells, Veterinary Laboratories Agency, New Haw,
Addlestone, Surrey, KT 15 3NB, UK

You may also wish to be aware that since November 1994 all suspected cases of spongiform encephalopathy in animals and

poultry were made notifiable. Hence since that date there has been a requirement for vets to report any suspect SE in dogs for further investigation. To date there has never been positive identification of a TSE in a dog.

I hope this is helpful

Yours sincerely 4

HUGH MCDONAGH BSE CORRESPONDENCE SECTION

=====

HOUND SURVEY

I am sorry, but I really could have been a co-signatory of Gerald's minute.

I do NOT think that we can justify devoting any resources to this study, especially as larger and more important projects such as the pathogenesis study will be quite demanding.

If there is a POLITICAL need to continue with the examination of hound brains then it should be passed entirely to the VI Service.

J W WILESMITH Epidemiology Unit 18 October 1991

Mr. R Bradley

cc: Mr. G A H Wells

<http://collections.europarchive.org/tna/20081106102318/http://www.bseinquiry.gov.uk/files/yb/1991/10/18001001.pdf>

3.3. Mr R J Higgins in conjunction with Mr G A Wells and Mr A C Scott would by the end of the year, identify the three brains that were from the "POSITIVE" end of the lesion spectrum.

<http://collections.europarchive.org/tna/20080103034308/http://www.bseinquiry.gov.uk/files/yb/1993/12/06001001.pdf>

TSE in dogs have not been documented simply because OF THE ONLY STUDY, those brain tissue samples were screwed up too. see my investigation of this here, and to follow, later follow up, a letter from defra, AND SEE SUSPICIOUS BRAIN TISSUE SAF's. ...TSS

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

TSE & HOUNDS

GAH WELLS (very important statement here...TSS)

HOUND STUDY

AS implied in the Inset 25 we must not _ASSUME_ that transmission of BSE to other species will invariably present pathology typical of a scrapie-like disease.

snip...

<http://web.archive.org/web/20010305222642/www.bseinquiry.gov.uk/files/yb/1991/01/04004001.pdf>

76 pages on hound study;

snip...

<http://web.archive.org/web/20030327022236/http://www.bseinquiry.gov.uk/files/sc/seac16/tabo4.pdf>

The spongiform changes were not pathognomonic (ie. conclusive proof) for prion disease, as they were atypical, being largely present in white matter rather than grey matter in the brain and spinal cord. However, Tony Scott, then head of electron microscopy work on TSEs, had no doubt that these SAFs were genuine and that these hounds therefore must have had a scrapie-like disease. I reviewed all the sections myself (original notes appended) and although the pathology was not typical, I could not exclude the possibility that this was a scrapie-like disorder, as white matter vacuolation is seen in TSEs and Wallerian degeneration was also present in the white matter of the hounds, another feature of scrapie.

38. I reviewed the literature on hound neuropathology, and discovered that micrographs and descriptive neuropathology from papers on 'hound ataxia' mirrored those in material from Robert Higgins' hound survey. Dr Tony Palmer (Cambridge) had done much of this work, and I obtained original sections from hound ataxia cases from him. This enabled me provisionally to conclude that Robert Higgins had in all probability detected hound ataxia, but also that hound ataxia itself was possibly a TSE. Gerald Wells confirmed in 'blind' examination of single restricted microscopic fields that there was no distinction between the white matter vacuolation present in BSE and scrapie cases, and that occurring in hound ataxia and the hound survey cases.

39. Hound ataxia had reportedly been occurring since the 1930's,

and a known risk factor for its development was the feeding to hounds of downer cows, and particularly bovine offal. Circumstantial evidence suggests that bovine offal may also be causal in FSE, and TME in mink. Despite the inconclusive nature of the neuropathology, it was clearly evident that this putative canine spongiform encephalopathy merited further investigation.

40. The inconclusive results in hounds were never confirmed, nor was the link with hound ataxia pursued. I telephoned Robert Higgins six years after he first sent the slides to CVL. I was informed that despite his submitting a yearly report to the CVO including the suggestion that the hound work be continued, no further work had been done since 1991. This was surprising, to say the very least.

41. The hound work could have provided valuable evidence that a scrapie-like agent may have been present in cattle offal long before the BSE epidemic was recognised. The MAFF hound survey remains unpublished.

Histopathological support to various other published MAFF experiments

42. These included neuropathological examination of material from experiments studying the attempted transmission of BSE to chickens and pigs (CVL 1991) and to mice (RVC 1994).

<http://www.bseinquiry.gov.uk/witness/htm/stato67.htm>

It was thought likely that at least some, and probably all, of the cases in zoo animals were caused by the BSE agent. Strong support for this hypothesis came from the findings of Bruce and others (1994) (Bruce, M.E., Chree, A., McConnell, I., Foster, J., Pearson, G. & Fraser, H. (1994) Transmission of bovine spongiform encephalopathy and scrapie to mice: strain variation and species barrier. Philosophical Transactions of the Royal Society B 343, 405-411: J/PTRSL/343/405), who demonstrated that the pattern of variation in incubation period and lesion profile in six strains of mice inoculated with brain homogenates from an affected kudu and the nyala, was similar to that seen when this panel of mouse strains was inoculated with brain from cattle with BSE. The affected zoo bovids were all from herds that were exposed to feeds that were likely to have contained contaminated ruminant-derived protein and the zoo felids had been exposed, if only occasionally in some cases, to tissues from cattle unfit for human consumption.

snip...

<http://www.bseinquiry.gov.uk/files/ws/s324.pdf>

NEW URL ;

<http://collections.europarchive.org/tna/20080102174910/http://www.bseinquiry.gov.uk/files/ws/s324.pdf>

Friday, March 8, 2013

Dogs may have been used to make Petfood and animal feed

<http://caninespongiformencephalopathy.blogspot.com/2013/03/dogs-may-have-been-used-to-make-petfood.html>

<http://caninespongiformencephalopathy.blogspot.com/>

<http://felinespongiformencephalopathyfse.blogspot.com/>

Tuesday, June 11, 2013

Weld County Bi-Products dba Fort Morgan Pet Foods 6/1/12
significant deviations from requirements in FDA regulations that
are intended to reduce the risk of bovine spongiform
encephalopathy (BSE) within the United States

<http://madcowfeed.blogspot.com/2013/06/weld-county-bi-products-dba-fort-morgan.html>

I lost my mother to the hvCJD and just made a promise, never
forget, and never let them forget, or we might all forget...
Wednesday, January 06, 2016

CREUTZFELDT JAKOB DISEASE SURVEILLANCE IN THE U.K.
23rd ANNUAL REPORT 2014 (published 18th November 2015)

<http://creutzfeldt-jakob-disease.blogspot.com/2016/01/creutzfeldt-jakob-disease-surveillance.html>

Saturday, December 12, 2015

CREUTZFELDT JAKOB DISEASE CJD TSE PRION REPORT
DECEMBER 14, 2015

<http://creutzfeldt-jakob-disease.blogspot.com/2015/12/creutzfeldt-jakob-disease-cjd-tse-prion.html>

Terry S. Singeltary Sr.

Evidence for human transmission of amyloid- β pathology and
cerebral amyloid angiopathy

07 02:27 AM

Terry S. Singeltary Sr. said:

re-Evidence for human transmission of amyloid- β pathology and cerebral amyloid angiopathy

2015-12-07 02:27 AM

Terry S. Singeltary Sr. said: 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)

<http://www.nature.com/nature/journal/v525/n7568/full/nature15369.html>

I would kindly like to comment on the Nature Paper, the Lancet reply, and the newspaper articles.

First, I applaud Nature, the Scientist and Authors of the Nature paper, for bringing this important finding to the attention of the public domain, and the media for printing said findings.

Secondly, it seems once again, politics is getting in the way possibly of more important Transmissible Spongiform Encephalopathy TSE Prion scientific findings. findings that could have great implications for human health, and great implications for the medical surgical arena. but apparently, the government peer review process, of the peer review science, tries to intervene again to water down said disturbing findings.

where have we all heard this before? it's been well documented via the BSE Inquiry. have they not learned a lesson from the last time?

we have seen this time and time again in England (and other Country's) with the BSE mad cow TSE Prion debacle.

That 'anonymous' Lancet editorial was disgraceful. The editor, Dick Horton is not a scientist.

The pituitary cadavers were very likely elderly and among them some were on their way to CJD or Alzheimer's. Not a bit unusual. Then the recipients who got pooled extracts injected from thousands of cadavers were 100% certain to have been injected

with both seeds. No surprise that they got both diseases going after thirty year incubations.

That the UK has a "system in place to assist science journalists" to squash embargoed science reports they find 'alarming' is pathetic.

Sounds like the journalists had it right in the first place:
'Alzheimer's may be a transmissible infection' in The Independent
to 'You can catch Alzheimer's' in The Daily Mirror or 'Alzheimer's bombshell" in The Daily Express.

if not for the journalist, the layperson would not know about these important findings.

where would we be today with sound science, from where we were 30 years ago, if not for the cloak of secrecy and save the industry at all cost mentality?

when you have a peer review system for science, from which a government constantly circumvents, then you have a problem with science, and humans die.

to date, as far as documented body bag count, with all TSE prion named to date, that count is still relatively low (one was too many in my case, Mom hvCJD), however that changes drastically once the TSE Prion link is made with Alzheimer's, the price of poker goes up drastically.

so, who makes that final decision, and how many more decades do we have to wait?

the iatrogenic mode of transmission of TSE prion, the many routes there from, load factor, threshold from said load factor to sub-clinical disease, to clinical disease, to death, much time is there to spread a TSE Prion to anywhere, but whom, by whom, and when, do we make that final decision to do something about it globally? how many documented body bags does it take? how many more decades do we wait? how many names can we make up for one disease, TSE prion?

Professor Collinge et al, and others, have had troubles in the past with the Government meddling in scientific findings, that might in some way involve industry, never mind human and or animal health.

FOR any government to continue to circumvent science for monetary gain, fear factor, or any reason, shame, shame on you.

in my opinion, it's one of the reasons we are at where we are at to date, with regards to the TSE Prion disease science i.e. money, industry, politics, then comes science, in that order.

greed, corporate, lobbyist there from, and government, must be

removed from the peer review process of sound science, it's bad enough having them in the pharmaceutical aspect of healthcare policy making, in my opinion.

my mother died from confirmed hvCJD, and her brother (my uncle) Alzheimer's of some type (no autopsy?). just made a promise, never forget, and never let them forget, before I do.

I kindly wish to remind the public of the past, and a possible future we all hopes never happens again. ...

[9. Whilst this matter is not at the moment directly concerned with the iatrogenic CJD cases from hgH, there remains a possibility of litigation here, and this presents an added complication. There are also results to be made available shortly (1) concerning a farmer with CJD who had BSE animals, (2) on the possible transmissibility of Alzheimer's and (3) a CMO letter on prevention of iatrogenic CJD transmission in neurosurgery, all of which will serve to increase media interest.]

<http://web.archive.org/web/20030714222309/www.bseinquiry.gov.uk/files/yb/1992/12/16005001.pdf>

<http://collections.europarchive.org/tna/20080102232842/http://www.bseinquiry.gov.uk/files/yb/1992/11/04001001.pdf>

snip...see full Singeltary Nature comment here;

<http://www.nature.com/nature/journal/v525/n7568/full/nature15369.html#/comments>

see Singeltary comments to Plos ;

Subject: 1992 IN CONFIDENCE TRANSMISSION OF ALZHEIMER
TYPE PLAQUES TO PRIMATES POSSIBILITY ON A
TRANSMISSIBLE PRION REMAINS OPEN

BSE101/1 0136

IN CONFIDENCE

CMO

From: . Dr J S Metiers DCMO

4 November 1992

1. Thank you for showing me Diana Dunstan's letter. I am glad that MRC have recognised the public sensitivity of these findings and intend to report them in their proper context. This hopefully will avoid misunderstanding and possible distortion by the media to portray the results as having more greater significance than the findings so far justify.

2. Using a highly unusual route of transmission (intra-cerebral injection) the researchers have demonstrated the transmission of a pathological process from two cases one of severe Alzheimer's disease the other of Gerstmann-Straussler disease to marmosets. However they have not demonstrated the transmission of either clinical condition as the "animals were behaving normally when killed". As the report emphasises the unanswered question is whether the disease condition would have revealed itself if the marmosets had lived longer. They are planning further research to see if the conditions, as opposed to the partial pathological process, is transmissible.

what are the implications for public health?

3. The route of transmission is very specific and in the natural state of things highly unusual. However it could be argued that the results reveal a potential risk, in that brain tissue from these two patients has been shown to transmit a pathological process. Should therefore brain tissue from such cases be regarded as potentially infective? Pathologists, morticians, neuro surgeons and those assisting at neuro surgical procedures and others coming into contact with "raw" human brain tissue could in theory be at risk. However, on a priori grounds given the highly specific route of transmission in these experiments that risk must be negligible if the usual precautions for handling brain tissue are observed.

1

92/11.4/1.1

BSE101/1 0137

4. The other dimension to consider is the public reaction. To some extent the GSS case demonstrates little more than the transmission of BSE to a pig by intra-cerebral injection. If other prion diseases can be transmitted in this way it is little surprise that some pathological findings observed in GSS were also transmissible to a marmoset. But the transmission of features of Alzheimer's pathology is a different matter, given the much greater frequency of this disease and raises the unanswered question whether some cases are the result of a transmissible prion. The only tenable public line will be that "more research is required" before that hypothesis could be evaluated. The possibility on a transmissible prion remains open. In the meantime MRC needs carefully to consider the range and sequence of studies needed to follow through from the preliminary observations in these two cases. Not a particularly comfortable message, but until we know more about the causation of Alzheimer's disease the total reassurance is not practical.

<http://collections.europarchive.org/tna/20081106170650/http://www.bseinquiry.gov.uk/files/yb/1992/11/04001001.pdf>

>>> 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 ?

Self-Propagative Replication of Ab Oligomers Suggests Potential Transmissibility in Alzheimer Disease

*** Singeltary comment PLoS ***

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

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

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

Sunday, November 22, 2015

*** Effect of heating on the stability of amyloid A (AA) fibrils and the intra- and cross-species transmission of AA amyloidosis
Abstract

Amyloid A (AA) amyloidosis is a protein misfolding disease characterized by extracellular deposition of AA fibrils. AA fibrils are found in several tissues from food animals with AA amyloidosis. For hygienic purposes, heating is widely used to inactivate microbes in food, but it is uncertain whether heating is sufficient to inactivate AA fibrils and prevent intra- or cross-species transmission. We examined the effect of heating (at 60 °C or 100 °C) and autoclaving (at 121 °C or 135 °C) on murine and bovine AA fibrils using Western blot analysis, transmission electron microscopy (TEM), and mouse model transmission experiments. TEM revealed that a mixture of AA fibrils and amorphous aggregates appeared after heating at 100 °C, whereas autoclaving at 135 °C produced large amorphous aggregates. AA fibrils retained antigen specificity in Western blot analysis when heated at 100 °C or autoclaved at 121 °C, but not when autoclaved at 135 °C. Transmissible pathogenicity of murine and bovine AA fibrils subjected to heating (at 60 °C or 100 °C) was significantly stimulated and resulted in amyloid deposition in mice. Autoclaving of murine AA fibrils at 121 °C or 135 °C significantly decreased amyloid deposition. Moreover, amyloid deposition in mice injected with murine AA fibrils was more severe than that in mice injected with bovine AA fibrils. Bovine AA fibrils autoclaved at 121 °C or 135 °C did not induce amyloid deposition in mice.

These results suggest that AA fibrils are relatively heat stable and that similar to prions, autoclaving at 135 °C is required to destroy the pathogenicity of AA fibrils. These findings may contribute to the prevention of AA fibril transmission through food materials to different animals and especially to humans.

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[http://www.tandfonline.com/doi/abs/10.3109/13506129.2015.1095735?
journalCode=iamy20](http://www.tandfonline.com/doi/abs/10.3109/13506129.2015.1095735?journalCode=iamy20)

[http://betaamyloidejd.blogspot.com/2015/11/effect-of-
heating-on-stability-of.html](http://betaamyloidejd.blogspot.com/2015/11/effect-of-heating-on-stability-of.html)

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

Gibbs CJ Jr, Asher DM, Koblitz 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.

[http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?
cmd=Retrieve&db=PubMed&list_uids=8006664&dopt=Abstract](http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=8006664&dopt=Abstract)

the warning shots fired over the bow of the boat that were never
heard ;

PITUITARY EXTRACT

This was used to help cows super ovulate. This tissue was
considered to be of greatest risk of containing BSE and
consequently transmitting the disease...

<http://collections.europarchive.org/tna/20090114081754/http://www.bseinquiry.gov.uk/files/yb/1988/06/08011001.pdf>

NON-LICENSED HUMAN TISSUE DEVICES WERE NOT
COMMERCIALY AVAILABLE

snip...

I was quite prepared to believe in unofficial pituitary hormones, also in the 1970's, whether as described by Dr. Little, or in other circumstances, for animal use.

snip...

The fact that there were jars of pituitaries (or extract) around on shelves is attested by the still potent 1943 pituitaries, described in Stockell Hartree et al. (J/RF/17/291) which had come from the lab. at Mill Hill. Having taken the trouble to collect them, they were not lightly thrown out...

<http://collections.europarchive.org/tna/20080102174454/http://www.bseinquiry.gov.uk/files/ws/s467bx.pdf>

3. The extraction is from a pool of pituitary glands collected from abbatoirs and the process used is unlikely to have any effect on the BSE agent. Hormones extracted from human pituitary glands have been responsible for a small number of Creutzfeldt Jacob disease in man.

<http://collections.europarchive.org/tna/20090505194948/http://bseinquiry.gov.uk/files/yb/1988/06/10001001.pdf>

SEE LOOPHOLE;

<http://collections.europarchive.org/tna/20090505194948/http://bseinquiry.gov.uk/files/yb/1988/06/13008001.pdf>

SEE LOOPHOLE SHOULD BE CLOSED;

<http://collections.europarchive.org/tna/20090505194948/http://bseinquiry.gov.uk/files/yb/1988/06/13010001.pdf>

<http://collections.europarchive.org/tna/20090505194948/http://bseinquiry.gov.uk/files/yb/1988/06/14006001.pdf>

snip...see at bottom;

Thursday, August 13, 2015

Iatrogenic CJD due to pituitary-derived growth hormone with genetically determined incubation times of up to 40 years

<http://creutzfeldt-jakob->

*** PRION2015 Alzheimer's disease ***

*** P.34: Preliminary study of Alzheimer's disease transmission to bank vole

Guido Di Donato¹, Geraldina Riccardi¹, Claudia D'Agostino¹, Flavio Torriani¹, Maurizio Pocchiari², Romolo Nonno¹, Umberto Agrimi¹, and Michele Angelo Di Bari¹

¹Department of Food Safety and Veterinary Public Health Istituto Superiore di Sanit a, Rome, Italy; ²Department of Cellular Biology and Neuroscience; Istituto Superiore di Sanit a, Rome, Italy

Extensive experimental findings indicate that prion-like mechanisms underly the pathogenesis of Alzheimer disease (AD). Transgenic mice have been pivotal for investigating prionlike mechanisms in AD, still these models have not been able so far to recapitulate the complex clinical-pathological features of AD. Here we aimed at investigating the potential of bank vole, a wild-type rodent highly susceptible to prions, in reproducing AD pathology upon experimental inoculation.

Voles were intracerebrally inoculated with brain homogenate from a familial AD patient. Animals were examined for the appearance of neurological signs until the end of experiment (800 d post-inoculation, d.p.i.). Brains were studied by immunohistochemistry for pTau Prion 2015 Poster Abstracts S29 (with AT180 and PHF-1 antibodies) and b-amyloid (4G8).

Voles didn't show an overt clinical signs, still most of them (11/16) were found pTau positive when culled for intercurrent disease or at the end of experiment. Interestingly, voles culled as early as 125 d.p.i. already showed pTau aggregates. Deposition of pTau was similar in all voles and was characterized by neuropil threads and coiled bodies in the alveus, and by rare neurofibrillary tangles in gray matter. Conversely, b-amyloid deposition was rather rare (2/16). Nonetheless, a single vole showed the contemporaneous presence of pTau in the alveus and a few Ab plaque-like deposits in the subiculum. Uninfected age-matched voles were negative for pTau and Ab.

*** These findings corroborate and extend previous evidences on the transmissibility of pTau and Ab aggregation. Furthermore, the observation of a vole with contemporaneous propagation of pTau and Ab is intriguing and deserves further studies.

=====

P.155: Quantitative real-time analysis of disease specific tau amyloid seeding activity

Davin Henderson and Edward Hoover Prion Research Center; College of Veterinary Medicine and Biomedical Sciences; Colorado

A leading hypothesis for the cause of neurodegenerative diseases is the templated misfolding of cellular proteins to an amyloid state. Spongiform encephalopathies were the first diseases discovered to be caused by a misfolded amyloid-rich protein. It is now recognized that the major human neurodegenerative diseases, including Alzheimer's disease (AD), Parkinson's disease (PD), and chronic traumatic encephalopathy (CTE), also are associated with amyloid formation. Moreover, AD and PD amyloids have been shown competent to transmit disease in experimental animal models, suggesting shared mechanisms with traditional prion diseases. Sensitive detection of prion disease has been advanced by in vitro amplification of low levels of disease specific amyloid seeds, e.g. serial protein misfolding amplification (sPMCA), amyloid seeding (ASA) and real-time quaking induced conversion (RT-QuIC), thereby replicating the disease process in vitro. In addition, measurement of the amyloid formation rate can estimate the level of disease-associated seed by using methods analogous to quantitative polymerase chain reaction (qPCR). In the present work, we apply these principles to show that seeding activity of in vitro generated amyloid tau and AD brain amyloid tau can be readily detected and quantitated.

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P.83: Gerstmann-StrCausler-Scheinker disease with F198S mutation: Selective propagation of PrPSc and pTau upon inoculation in bank vole

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Gerstmann-StrCausler-Scheinker disease with F198S mutation (GSS-F198S) is characterized by the presence of PrP amyloid plaques as well as neurofibrillary tangles with abnormally-phosphorylated tau protein (pTau) in the brain. The relationship between tau protein and PrP in the pathogenesis of GSS-F198S is unknown. In a previous study, we inoculated intracerebrally 2 GSS-F198S cases in 2 lines of voles carrying either methionine (Bv109M) or isoleucine (Bv109I) at codon 109 of PrP. GSS-F198S transmitted rather efficiently to Bv109I, but not to Bv109M.

Here we investigated the presence of pTau, as assessed by immunohistochemistry with anti-pTau antibodies AT180 and PHF-1, in the same voles previously inoculated with GSSF198S. Among these voles, most Bv109I showed clinical signs after short survival times (>150 d.p.i.) and were positive for PrPSc. The remaining Bv109I and all Bv109M survived for longer times without showing prion-related pathology or detectable PrPSc. All Bv109I which were previously found PrPSc-positive,

were immunonegative for pTau deposition. In contrast, pTau deposition was detected in 16/20 voles culled without clinical signs after long survival times (225–804 d.p.i.). pTau deposition was characterized by neuropil threads and coiled bodies in the alveus, and was similar in all voles analyzed.

These findings highlight that pTau from GSS-F198S can propagate in voles. Importantly, pTau propagation was independent from PrPSc, as pTau was only found in PrPSc-negative voles surviving longer than 225 d.p.i. Thus, selective transmission of PrPSc and pTau proteinopathies from GSS-F198S can be accomplished by experimental transmission in voles.

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I3 A β Strains and Alzheimer's Disease

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An essential early event in the development of Alzheimer's disease is the misfolding and aggregation of A β . Enigmatically, despite the extensive deposition of human-sequence A β in the aging brain, nonhuman primates do not develop the full pathologic or cognitive phenotype of Alzheimer's disease, which appears to be unique to humans. In addition, some humans with marked A β accumulation in the brain retain their cognitive abilities, raising the question of whether the pathogenicity of A β is linked to the molecular features of the misfolded protein. I will present evidence for strain-like molecular differences in aggregated A β between humans and nonhuman primates, and among end-stage Alzheimer patients. I will also discuss a case of Alzheimer's disease with atypical A β deposition to illustrate heterogeneity in the molecular architecture of A β assemblies, and how this variability might influence the nature of the disease. As in the case of prion diseases, strain-like variations in the molecular architecture of A β could help to explain the phenotypic variability in Alzheimer's disease, as well as the distinctively human susceptibility to the disorder.

This research was conducted in collaboration with Harry LeVine, Rebecca Rosen, Amarallys Cintron, David Lynn, Yury Chernoff, Anil Mehta and Mathias Jucker and colleagues. Supported by AG040589, RR165/OD11132, AG005119, NS077049, the CART Foundation and MetLife.

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I5 Pathogenic properties of synthetically generated prions

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Synthetically generating prions with bacterially expressed recombinant prion protein (recPrP) strongly supports the prion hypothesis. Yet, it remains unclear whether the pathogenic properties of synthetically generated prions (rec-Prion) fully recapitulate those of naturally occurring prions. A series of analyses including intracerebral and intraperitoneal transmissions of rec-Prion in wild-type mice were performed to determine the characteristics of rec-Prion induced diseases. Results from these analyses demonstrated that the rec-Prion exhibits the same pathogenic properties with naturally occurring prions, including a titratable infectivity that can be determined by endpoint titration assays, capability of transmitting prion disease via routes other than the direct intra-cerebral inoculation, causing ultra-structural lesions that are specific to prion disease, and sharing a similar manner of visceral dissemination and neuroinvasion with naturally occurring scrapie and chronic wasting disease. These findings confirmed that the disease caused by rec-Prion in wild-type mice is bona fide prion disease or transmissible spongiform encephalopathies, and the rec-Prion contains similar pathogenic properties as naturally occurring prions.

P16 Transmissible protein toxins in neurodegenerative disease

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Amyotrophic lateral sclerosis (ALS) is an obvious example of neurodegenerative disease that seems to spread along anatomical pathways. The spread of symptoms from the site of onset (e.g. limb) to the respiratory musculature drives the rate of disease progression. In cognitive disorders, such as Alzheimer's disease, one can find similarly find evidence of spreading dysfunction and pathology. One mechanism to account for this spread of disease from one neural structure to another is by evoking prion-like propagation of a toxic misfolded protein from cell to cell. Recent studies in animals that model aspects of Alzheimer's Disease, Parkinson's Disease, and Tauopathy, have bolstered the arguments in favor of prion-like, although in most of these models the mice do not develop overt "clinical" symptoms. Recently, Jacob Ayers demonstrated that the symptoms of ALS can be transmitted from a strain of mice that expresses mutant SOD1-G93A at high levels to a second transgenic strain that expresses mutant SOD1 at low, nontoxic, levels. This model showed many prion-like features including evidence of host-adaptation (earlier and more penetrant disease upon second passage). Interestingly, homogenates from paralyzed mice expressing the G37R variant of SOD1 transmitted poorly, a finding suggestive that different SOD1 variants may exhibit strain-like properties. These "inducible" models of human neurodegenerative disease enable the generation of models that do not require extraordinary levels of transgene expression and provide a more precise means of initiating the disease process, advances that may translate into more predictive pre-clinical models.

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P188 Transmission of amyloid pathology by peripheral administration of misfolded A β

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Misfolding and aggregation of Amyloid- β (A β) is one of the primary events involved in the pathogenesis of Alzheimer's disease (AD). Recently, it has been proposed that A β aggregates can transmit and spread the pathology following a prion-like mechanism. Prions can be exogenously transmitted by many different routes of administration. In the case of A β , previous studies showed that intraperitoneal (i.p.) injection of seeds can accelerate cerebral amyloidosis in mouse models. However, other potential routes have not yet been studied. The goal of this work was to assess whether A β amyloidosis can be seeded in the brain of a transgenic mouse model of AD by peripheral administration of misfolded particles.

Young tg2576 animals (50 days old) were inoculated with a pool of brain extract coming from old Tg2576 animals (10%w/v) by different routes: i.p. (100 μ L), eye drops (5 μ L each eye, 3 times), intramuscular (i.m., 50 μ L), and per os (p.o., 1000 μ L). Animals were sacrificed at 300 days old, and brain samples were analyzed for amyloid pathology by IHC and ELISA.

The i.p., i.m., and eye drops administration of A β seeds significantly accelerated pathological features in tg2576. Regardless of the higher volume administered, p.o. treated animals did not show any pathological changes when compared to untreated controls. Differences in the proportion of diffuse, core and vascular deposition was observed within experimental groups. Our data show that peripheral administration of A β seeds could accelerate pathological changes in the brain and suggest that an orchestrated cross-talk between the brain and peripheral tissues occurs in AD.

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

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

PLEASE REMEMBER, IN 55 YEARS AND OLDER, THE RATE OF DOCUMENTED CJD JUMPS TO ONE IN 9,000. but officials don't tell you that either. carry on...

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>

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

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

vCJD in the USA * BSE in U.S.

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

The Lancet Infectious Diseases, Volume 3, Issue 8, Page 463,
August 2003 doi:10.1016/S1473-3099(03)00715-1 Cite or Link
Using DOI

Tracking spongiform encephalopathies in North America

Original

Xavier Bosch

"My name is Terry S Singeltary Sr, and I live in Bacliff, Texas. I lost my mom to hvCJD (Heidenhain variant CJD) and have been searching for answers ever since. What I have found is that we have not been told the truth. CWD in deer and elk is a small portion of a much bigger problem." 49-year-old Singeltary is one of a number of people who have remained largely unsatisfied after being told that a close relative died from a rapidly progressive dementia compatible with spontaneous Creutzfeldt-Jakob ...

[http://www.thelancet.com/journals/laninf/article/PIIS1473-3099\(03\)00715-1/abstract](http://www.thelancet.com/journals/laninf/article/PIIS1473-3099(03)00715-1/abstract)

Suspect symptoms

What if you can catch old-fashioned CJD by eating meat from a sheep infected with scrapie?

28 Mar 01

Most doctors believe that sCJD is caused by a prion protein deforming by chance into a killer. But Singeltary thinks otherwise. He is one of a number of campaigners who say that some sCJD, like the variant CJD related to BSE, is caused by eating meat from infected animals. Their suspicions have focused on sheep carrying scrapie, a BSE-like disease that is widespread in flocks across Europe and North America. Now scientists in France have stumbled across new evidence that adds weight to the campaigners' fears. To their complete surprise, the researchers found that one strain of scrapie causes the same brain damage in mice as sCJD.

"This means we cannot rule out that at least some sCJD may be caused by some strains of scrapie," says team member Jean-Philippe Deslys of the French Atomic Energy Commission's medical research laboratory in Fontenay-aux-Roses, south-west of Paris. Hans Kretschmar of the University of Göttingen, who coordinates CJD surveillance in Germany, is so concerned by the findings that he now wants to trawl back through past sCJD cases to see if any might have been caused by eating infected mutton or lamb...

<http://www.newscientist.com/article/mg16922840.300-like-lambs-to-the-slaughter.html>

Terry S. Singeltary Sr.

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>

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