FSIS [Docket No. FSIS–2019–0021] Notice of Request To Renew an Approved Information Collection: Specified Risk Materials Singeltary Submission

Food Safety and Inspection Service [Docket No. FSIS–2019–0021] Notice of Request To Renew an Approved Information Collection: Specified Risk Materials

Greetings FSIS et al,

I would kindly like to comment on the following docket;

[Docket No. FSIS–2019–0021] Notice of Request To Renew an Approved Information Collection: Specified Risk Materials

Federal Docket SRM TSE Prion

DEPARTMENT OF AGRICULTURE

Food Safety and Inspection Service [Docket No. FSIS–2019–0021]

Notice of Request To Renew an Approved Information Collection: Specified Risk Materials

AGENCY: Food Safety and Inspection Service, USDA.

ACTION: Notice and request for comments.

https://www.govinfo.gov/content/pkg/FR-2019-09-10/pdf/2019-19443.pdf

This information is critical, and should continue to be collected.

The TSE prion is spreading across the USA in Cervid as in CWD TSE Prion.

The mad cow surveillance, feed ban, testing, and SRM removal there from, has been, and still is, a terrible failure.

WE know that the USA Food and Drug Administration's BSE Feed Regulation (21 CFR 589.2000) of August 1997 was/is a colossal failure, and proven to be so year after year, decade after decade, and this was just admitted by the FDA et al (see below FDA Reports on VFD Compliance Sept. 2019 report).

God, all these decades you hear from all the warning letters on SRM that were released to the public for consumption, that even if they did eat a SRM, the BSE Feed Regulation (21 CFR 589.2000) of August 1997 would save that tissue from that animal from having a TSE Prion, was nothing but lies. what about those children all across the USA that were fed the most high risk cattle for mad cow disease, i.e. dead stock downer cows via the USDA School lunch program, who will watch those kids for the next 50 years for cjd tse prion aka mad cow disease, let alone all the folks consuming SRMs that have been exposed to mad cow type disease in different livestock species, due to the fact the USA colossal failure of the BSE Feed Regulation (21 CFR 589.2000) of August 1997. it's all documented below, see for yourself;

SUNDAY, SEPTEMBER 1, 2019

FDA Reports on VFD Compliance

LET THIS STATEMENT SINK IN!

Before and after the current Veterinary Feed Directive (VFD) rules took full effect in January, 2017, the FDA focused primarily on education and outreach to help feed mills, veterinarians and producers understand and comply with the requirements. Since then, FDA has gradually increased the number of VFD inspections and initiated enforcement actions when necessary.

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

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

http://specifiedriskmaterial.blogspot.com/

WEDNESDAY, JULY 31, 2019

The agent of transmissible mink encephalopathy passaged in sheep is similar to BSE-L

https://transmissible-mink-encephalopathy.blogspot.com/2019/07/the-agent-of-transmissible-mink.html

THURSDAY, AUGUST 08, 2019

Raccoons accumulate PrPSc after intracranial inoculation with the agents of chronic wasting disease (CWD) or transmissible mink encephalopathy (TME) but not atypical scrapie

https://chronic-wasting-disease.blogspot.com/2019/08/raccoons-accumulate-prpsc-after.html

In the USA, USDA et al sometimes serves SRM's up as appetizers or horderves.

Thursday, November 28, 2013

Department of Justice Former Suppliers of Beef to National School Lunch Program Settle Allegations of Improper Practices and Mistreating Cows

http://madcowusda.blogspot.com/2013/11/department-of-justice-former-suppliers.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

try this link ;

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

Sunday, November 13, 2011

*** California BSE mad cow beef recall, QFC, CJD, and dead stock downer livestock

http://transmissiblespongiformencephalopathy.blogspot.com/2011/11/california-bse-mad-cow-beef-recall-qfc.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 (SRM's) 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

USDA BSE TSE PRION SURVEILLANCE, FEED, TESTING, SRM FIREWALLS...LMAO!

THE USDA FDA TRIPLE MAD COW DISEASE FIREWALL, WERE NOTHING MORE THAN INK ON PAPER !

infamous august 4, 1997 BSE TSE prion mad cow feed ban, part of usda fda et al TRIPLE MAD COW FIREWALL, 10 YEARS AFTER ;

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- BYPASS ML W/SMARTA, Recall # V-025-2007 CODE The firm does not utilize a code - only shipping documentation with commodity and weights identified. RECALLING FIRM/MANUFACTURER Rangen, Inc, Buhl, ID, by letters on February 13 and 14, 2007.

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

VOLUME OF PRODUCT IN COMMERCE 9,997,976 lbs. DISTRIBUTION ID and NV

END OF ENFORCEMENT REPORT FOR MARCH 21, 2007

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

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

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

*** 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

Thursday, July 24, 2014

*** Protocol for further laboratory investigations into the distribution of infectivity of Atypical BSE SCIENTIFIC REPORT OF EFSA New protocol for Atypical BSE investigations

http://bse-atypical.blogspot.com/2014/07/protocol-for-further-laboratory.html

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

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

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

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

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

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

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

cwd scrapie pigs oral routes

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

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

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

***> Conclusions: This study demonstrates that PrPSc accumulates in lymphoid tissues from pigs challenged intracranially or orally with the CWD agent, and can be detected as early as 4 months after challenge. CWD-infected pigs rarely develop clinical disease and if they do, they do so after a long incubation period.

This raises the possibility that CWD-infected pigs could shed prions into their environment long before they develop clinical disease.

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

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

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

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

Friday, December 14, 2012

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

snip.....

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

Animals considered at high risk for CWD include:

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

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

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

The amount of animal PAP that is of deer and/or elk origin imported from the USA to GB can not be determined, however, as it is not specified in TRACES. It may constitute a small percentage of the 8412 kilos of non-fish origin processed animal proteins that were imported from US into GB in 2011.

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

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

snip.....

36% in 2007 (Almberg et al., 2011). In such areas, population declines of deer of up to 30 to 50% have been observed (Almberg et al., 2011). In areas of Colorado, the prevalence can be as high as 30% (EFSA, 2011).

The clinical signs of CWD in affected adults are weight loss and behavioural changes that can span weeks or months (Williams, 2005). In addition, signs might include excessive salivation, behavioural alterations including a fixed stare and changes in interaction with other animals in the herd, and an altered stance (Williams, 2005). These signs are indistinguishable from cervids experimentally infected with bovine spongiform encephalopathy (BSE).

Given this, if CWD was to be introduced into countries with BSE such as GB, for example, infected deer populations would need to be tested to differentiate if they were infected with CWD or BSE to minimise the risk of BSE entering the human food-chain via affected venison.

snip.....

The rate of transmission of CWD has been reported to be as high as 30% and can approach 100% among captive animals in endemic areas (Safar et al., 2008).

snip.....

In summary, in endemic areas, there is a medium probability that the soil and surrounding environment is contaminated with CWD prions and in a bioavailable form. In rural areas where CWD has not been reported and deer are present, there is a greater than negligible risk the soil is contaminated with CWD prion.

snip.....

In summary, given the volume of tourists, hunters and servicemen moving between GB and North America, the probability of at least one person travelling to/from a CWD affected area and, in doing so, contaminating their clothing, footwear and/or equipment prior to arriving in GB is greater than negligible... For deer hunters, specifically, the risk is likely to be greater given the increased contact with deer and their environment. However, there is significant uncertainty associated with these estimates.

snip.....

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

snip.....

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

TUESDAY, APRIL 18, 2017

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

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

MONDAY, JUNE 24, 2019

APHIS, FSIS, USDA, FDA, Transmissible Spongiform Encephalopathy TSE, BSE, CWD, Scrapie, Camel TSE Prion Disease, CJD Humans

https://transmissiblespongiformencephalopathy.blogspot.com/2019/06/aphis-fsis-usda-fda-transmissible.html

WEDNESDAY, AUGUST 15, 2018

The agent of H-type bovine spongiform encephalopathy associated with E211K prion protein polymorphism transmits after oronasal challenge

http://bovineprp.blogspot.com/2018/08/the-agent-of-h-type-bovine-spongiform.html

TUESDAY, JULY 30, 2019

Guidelines for reporting surveillance data on Transmissible Spongiform Encephalopathies (TSE) in the EU within the framework of Regulation (EC) No 999/2001 APPROVED: 9 July 2019

https://animalhealthreportpriontse.blogspot.com/2019/07/guidelines-for-reporting-surveillance.html

PRION 2018 CONFERENCE

P98 The agent of H-type bovine spongiform encephalopathy associated with E211K prion protein polymorphism transmits after oronasal challenge

Greenlee JJ (1), Moore SJ (1), and West Greenlee MH (2) (1) United States Department of Agriculture, Agricultural Research Service, National Animal Disease Center, Virus and Prion Research Unit, Ames, IA, United States (2) Department of Biomedical Sciences, Iowa State University College of Veterinary Medicine, Ames, IA, United States.

reading up on this study from Prion 2018 Conference, very important findings ;

***> This study demonstrates that the H-type BSE agent is transmissible by the oronasal route.

***> These results reinforce the need for ongoing surveillance for classical and atypical BSE to minimize the risk of potentially infectious tissues entering the animal or human food chains.

PRION 2018 CONFERENCE ABSTRACT

https://prion2018.org/

WEDNESDAY, OCTOBER 24, 2018

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

https://bse-atypical.blogspot.com/2018/10/experimental-infection-of-cattle-with.html

TUESDAY, AUGUST 28, 2018

USDA finds BSE infection in Florida cow 08/28/18 6:43 PM

http://animalhealthreportpriontse..blogspot.com/2018/08/usda-finds-bse-infection-in-florida-cow.html

WEDNESDAY, AUGUST 29, 2018

USDA Announces Atypical Bovine Spongiform Encephalopathy Detection USDA 08/29/2018 10:00 AM EDT

http://bse-atypical.blogspot.com/2018/08/usda-announces-atypical-bovine.html

WEDNESDAY, AUGUST 29, 2018

Transmissible Spongiform Encephalopathy TSE Prion Atypical BSE Confirmed Florida Update USA August 28, 2018

http://transmissiblespongiformencephalopathy.blogspot.com/2018/08/transmissible-spongiform-encephalopathy.html

MONDAY, JANUARY 09, 2017

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

CDC Volume 23, Number 2—February 2017

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

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

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

THURSDAY, JULY 20, 2017

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

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

WEDNESDAY, MARCH 15, 2017

In vitro amplification of H-type atypical bovine spongiform encephalopathy by protein misfolding cyclic amplification

"When considering the atypical L-BSE and H-BSE diseases of cattle, they have been assessed in both non-human primate and transgenic mouse bioassays (with mice transgenic for human PRNP) and both model systems indicate that H-BSE and L-BSE may have increased zoonotic potential compare with C-BSE.

***The detection of all types of BSE is therefore of significant importance."

http://bse-atypical.blogspot.com/2017/03/in-vitro-amplification-of-h-type.html

Saturday, June 25, 2011

Transmissibility of BSE-L and Cattle-Adapted TME Prion Strain to Cynomolgus Macaque

"BSE-L in North America may have existed for decades"

http://transmissiblespongiformencephalopathy.blogspot.com/2011/06/transmissibility-of-bse-l-and-cattle.html

The 2004 enhanced BSE surveillance program was so flawed, that one of the top TSE prion Scientist for the CDC, Dr. Paul Brown stated ; 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.

see ;

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

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

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

PAUL BROWN COMMENT TO ME ON THIS ISSUE

Tuesday, September 12, 2006 11:10 AM

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

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

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

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

IT is of my opinion, that the OIE and the USDA et al, are the soul reason, and responsible parties, for Transmissible Spongiform Encephalopathy TSE prion diseases, including typical and atypical BSE, typical and atypical Scrapie, and all strains of CWD, and human TSE there from, spreading around the globe. I have lost all confidence of this organization as a regulatory authority on animal disease, and consider it nothing more than a National Trading Brokerage for all strains of animal TSE, just to satisfy there commodity. AS i said before, OIE should hang up there jock strap now, since it appears they will buckle every time a country makes some political hay about trade protocol, commodities and futures. IF they are not going to be science based, they should do everyone a favor and dissolve there organization. JUST because of low documented human body count with nvCJD and the long incubation periods, the lack of sound science being replaced by political and corporate science in relations with the fact that science has now linked some sporadic CJD with atypical BSE and atypical scrapie, and the very real threat of CWD being zoonosis, I believed the O.I.E. has failed terribly and again, I call for this organization to be dissolved...

Monday, May 05, 2014

Member Country details for listing OIE CWD 2013 against the criteria of Article 1.2.2., the Code Commission recommends consideration for listing

http://chronic-wasting-disease.blogspot.com/2014/05/member-country-details-for-listing-oie.html

Friday, December 5, 2014

SPECIAL ALERT The OIE recommends strengthening animal disease surveillance worldwide

http://transmissiblespongiformencephalopathy.blogspot.com/2014/12/special-alert-oie-recommends.html

IN A NUT SHELL ; (Adopted by the International Committee of the OIE on 23 May 2006) 11. Information published by the OIE is derived from appropriate declarations made by the official Veterinary Services of Member Countries. The OIE is not responsible for inaccurate publication of country disease status based on inaccurate information or changes in epidemiological status or other significant events that were not promptly reported to the Central Bureau,

http://www.oie.int/eng/Session2007/RF2006.pdf

MONDAY, JANUARY 21, 2019

Bovine Spongiform Encephalopathy BSE TSE Prion Surveillance FDA USDA APHIS FSIS UPDATE 2019

https://bovineprp.blogspot.com/2019/01/bovine-spongiform-encephalopathy-bse.html

Saturday, December 15, 2018

***> ADRD Summit RFI Singeltary COMMENT SUBMISSION BSE, SCRAPIE, CWD, AND HUMAN TSE PRION DISEASE December 14, 2018

https://prionprp.blogspot.com/2018/12/adrd-summit-rfi-singeltary-comment.html

SATURDAY, JANUARY 5, 2019

Low levels of classical BSE infectivity in rendered fat tissue

https://bovineprp.blogspot.com/2019/01/low-levels-of-classical-bse-infectivity.html

***> FRIDAY, DECEMBER 14, 2018 MAD COW USA FLASHBACK Texas Style

FRIDAY DECEMBER 14, 2018

https://madcowusda.blogspot.com/2018/12/mad-cow-usa-flashback-friday-december.html

THURSDAY, JANUARY 3, 2019

MAD COW USDA DISEASE BSE TSE Prion

https://madcowusda.blogspot.com/2019/01/mad-cow-usda-disease-bse-tse-prion.html

THURSDAY, OCTOBER 22, 2015

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

HOW TO COVER UP MAD COW DISEASE IN TEXAS

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

http://madcowusda.blogspot.com/2012/06/johanns-introduces-legislation-banning.html

http://madcowusda.blogspot.com/2012_06_01_archive.html

WEDNESDAY, AUGUST 29, 2018

OIE Bovine spongiform encephalopathy, United States of America Information received on 29/08/2018 from Dr John Clifford, Official Delegate, Chief Trade Advisor, APHIS USDA

"The event is resolved. No more reports will be submitted."

well, so much for those herd mates exposed to this atypical BSE cow, and all those trace in and trace outs.

The OIE, USDA, and the BSE MRR policy is a joke, a sad, very sad joke...

http://bovineprp.blogspot.com/2018/08/oie-bovine-spongiform-encephalopathy.html

Saturday, July 23, 2016

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

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

Tuesday, July 26, 2016

Atypical Bovine Spongiform Encephalopathy BSE TSE Prion UPDATE JULY 2016

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

Monday, June 20, 2016

Specified Risk Materials SRMs BSE TSE Prion Program

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

WEDNESDAY, APRIL 24, 2019

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

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

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

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

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

http://journals.plos.org/plosone/article/comment?id=info:doi/10.1371/annotation/09676b86-bbc2-4c69-9032-c319f13a7ad0

*** 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: 12/30/2014ID: 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

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

APHIS-2006-0118-0096 CWD

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

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 EMC 1 Terry S. Singeltary Sr. Vol #: 1

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

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

PLEASE SEE FULL TEXT SUBMISSION ;

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

2001 Terry S. Singeltary Sr. comment submission

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

ZOONOSIS OF SCRAPIE TSE PRION

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

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

Prion diseases (PD) are the unique neurodegenerative proteinopathies reputed to be transmissible under field conditions since decades. The transmission of Bovine Spongiform Encephalopathy (BSE) to humans evidenced that an animal PD might be zoonotic under appropriate conditions. Contrarily, in the absence of obvious (epidemiological or experimental)

elements supporting a transmission or genetic predispositions, PD, like the other proteinopathies, are reputed to occur spontaneously (atpical animal prion strains, sporadic CJD summing 80% of human prion cases).

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

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

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

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

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

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

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thus questioning the origin of human sporadic cases

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

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

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

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

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

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

PRION 2016 TOKYO

Saturday, April 23, 2016

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

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

Taylor & Francis

Prion 2016 Animal Prion Disease Workshop Abstracts

WS-01: Prion diseases in animals and zoonotic potential

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

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

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

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

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

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

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

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

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

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

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

5. A positive result from a chimpanzee challenged severly would likely create alarm in some circles even if the result could not be interpreted for man.

***> I have a view that all these agents could be transmitted provided a large enough dose by appropriate routes was given and the animals kept long enough.

***> Until the mechanisms of the species barrier are more clearly understood it might be best to retain that hypothesis.

snip...

R. BRADLEY

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

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

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

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

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

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

> 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. <

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

Emmanuel E. Comoy, Jacqueline Mikol, Sophie Luccantoni-Freire, Evelyne Correia, Nathalie Lescoutra-Etchegaray, Valérie Durand, Capucine Dehen, Olivier Andreoletti, Cristina Casalone, Juergen A. Richt, Justin J. Greenlee, Thierry Baron, Sylvie L. Benestad, Paul Brown & Jean-Philippe Deslys Scientific Reports volume 5, Article number: 11573 (2015) | Download Citation

Abstract

Classical bovine spongiform encephalopathy (c-BSE) is the only animal prion disease reputed to be zoonotic, causing variant Creutzfeldt-Jakob disease (vCJD) in humans and having guided protective measures for animal and human health against animal prion diseases. Recently, partial transmissions to humanized mice showed that the zoonotic potential of scrapie might be similar to c-BSE. We here report the direct transmission of a natural classical scrapie isolate to cynomolgus macaque, a highly relevant model for human prion diseases, after a 10-year silent incubation period, with features similar to those reported for human cases of sporadic CJD. Scrapie is thus actually transmissible to primates with incubation periods compatible with their life expectancy, although fourfold longer than BSE. Long-term experimental transmission studies are necessary to better assess the zoonotic potential of other prion diseases with high prevalence, notably Chronic Wasting Disease of deer and elk and atypical/Nor98 scrapie.

SNIP...

Discussion 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.

The second possibility is a laboratory cross-contamination. Three facts make this possibility equally unlikely. First, handling of specimens in our laboratory is performed with fastidious attention to the avoidance of any such cross-contamination. Second, no laboratory cross-contamination has ever been documented in other primate laboratories, including the NIH, even between infected and uninfected animals housed in the same or adjacent cages with daily intimate contact (P. Brown, personal communication). Third, the cerebral lesion profile is different from all the other prion diseases we have studied in this model19, with a correlation between cerebellar lesions (massive spongiform change of Purkinje cells, intense PrPres staining and reactive gliosis26) and ataxia. The iron deposits present in the globus pallidus are a non specific finding that have been reported previously in neurodegenerative diseases and aging27. Conversely, the thalamic lesion was reminiscent of a metabolic disease due to thiamine deficiency28 but blood thiamine levels were within normal limits (data not shown). The preferential distribution of spongiform change in cortex associated with a limited distribution in the brainstem is reminiscent of the lesion profile in MM2c and VV1 sCJD patients29, but interspecies comparison of lesion profiles should be interpreted with caution. It is of note that the same classical scrapie isolate induced TSE in C57Bl/6 mice with similar incubation periods and lesional profiles as a sample derived from a MM1 sCJD patient30.

We are therefore confident that the illness in this cynomolgus macaque represents a true transmission of a sheep c-scrapie isolate directly to an old-world monkey, which taxonomically resides in the primate subdivision (parvorder of catarrhini) that includes humans. With an homology of its PrP protein with humans of 96.4%31, cynomolgus macaque constitutes a highly relevant model for assessing zoonotic risk of prion diseases. Since our initial aim was to show the absence of transmission of scrapie to macaques in the worst-case scenario, we obtained materials from a flock of naturally-infected sheep, affecting animals with different genotypes32. This c-scrapie isolate exhibited complete transmission in ARQ/ARQ sheep (332 ± 56 days) and Tg338 transgenic mice expressing ovine VRQ/VRQ prion protein (220 ± 5 days) (O. Andreoletti, personal communication). From the standpoint of zoonotic risk, it is important to note that sheep with c-scrapie (including the isolate used in our study) have demonstrable infectivity throughout their lymphoreticular system early in the incubation period of the disease (3 months-old for all the lymphoid organs, and as early as 2 months-old in gut-associated lymph nodes)33. In

addition, scrapie infectivity has been identified in blood34, milk35 and skeletal muscle36 from asymptomatic but scrapie infected small ruminants which implies a potential dietary exposure for consumers.

Two earlier studies have reported the occurrence of clinical TSE in cynomolgus macaques after exposures to scrapie isolates. In the first study, the “Compton” scrapie isolate (derived from an English sheep) and serially propagated for 9 passages in goats did not transmit TSE in cynomolgus macaque, rhesus macaque or chimpanzee within 7 years following intracerebral challenge1; conversely, after 8 supplementary passages in conventional mice, this “Compton” isolate induced TSE in a cynomolgus macaque 5 years after intracerebral challenge, but rhesus macaques and chimpanzee remained asymptomatic 8.5 years post-exposure8. However, multiple successive passages that are classically used to select laboratory-adapted prion strains can significantly modify the initial properties of a scrapie isolate, thus questioning the relevance of zoonotic potential for the initial sheep-derived isolate. The same isolate had also induced disease into squirrel monkeys (new-world monkey)9. A second historical observation reported that a cynomolgus macaque developed TSE 6 years post-inoculation with brain homogenate from a scrapie-infected Suffolk ewe (derived from USA), whereas a rhesus macaque and a chimpanzee exposed to the same inoculum remained healthy 9 years post-exposure1. This inoculum also induced TSE in squirrel monkeys after 4 passages in mice. Other scrapie transmission attempts in macaque failed but had more shorter periods of observation in comparison to the current study. Further, it is possible that there are differences in the zoonotic potential of different scrapie strains.

The most striking observation in our study is the extended incubation period of scrapie in the macaque model, which has several implications. Firstly, our observations constitute experimental evidence in favor of the zoonotic potential of c-scrapie, at least for this isolate that has been extensively studied32,33,34,35,36. The cross-species zoonotic ability of this isolate should be confirmed by performing duplicate intracerebral exposures and assessing the transmissibility by the oral route (a successful transmission of prion strains through the intracerebral route may not necessarily indicate the potential for oral transmission37). However, such confirmatory experiments may require more than one decade, which is hardly compatible with current general management and support of scientific projects; thus this study should be rather considered as a case report.

Secondly, transmission of c-BSE to primates occurred within 8 years post exposure for the lowest doses able to transmit the disease (the survival period after inoculation is inversely proportional to the initial amount of infectious inoculum). The occurrence of scrapie 10 years after exposure to a high dose (25 mg) of scrapie-infected sheep brain suggests that the macaque has a higher species barrier for sheep c-scrapie than c-BSE, although it is notable that previous studies based on in vitro conversion of PrP suggested that BSE and scrapie prions would have a similar conversion potential for human PrP38.

Thirdly, prion diseases typically have longer incubation periods after oral exposure than after intracerebral inoculations: since humans can develop Kuru 47 years after oral exposure39, an incubation time of several decades after oral exposure to scrapie would therefore be expected, leading the disease to occur in older adults, i.e. the peak age for cases considered to be sporadic disease, and making a distinction between scrapie-associated and truly sporadic disease extremely difficult to appreciate.

Fourthly, epidemiologic evidence is necessary to confirm the zoonotic potential of an animal disease suggested by experimental studies. A relatively short incubation period and a peculiar epidemiological situation (e.g., all the first vCJD cases occurring in the country with the most important ongoing c-BSE epizootic) led to a high degree of suspicion that c-BSE was the cause of vCJD. Sporadic CJD are considered spontaneous diseases with an almost stable and constant worldwide prevalence (0.5–2 cases per million inhabitants per year), and previous epidemiological studies were unable to draw a link between sCJD and classical scrapie6,7,40,41, even though external causes were hypothesized to explain the occurrence of some sCJD clusters42,43,44. However, extended incubation periods exceeding several decades would impair the predictive values of epidemiological surveillance for prion diseases, already weakened by a limited prevalence of prion diseases and the multiplicity of isolates gathered under the phenotypes of “scrapie” and “sporadic CJD”.

Fifthly, considering this 10 year-long incubation period, together with both laboratory and epidemiological evidence of decade or longer intervals between infection and clinical onset of disease, no premature conclusions should be drawn from negative transmission studies in cynomolgus macaques with less than a decade of observation, as in the aforementioned historical transmission studies of scrapie to primates1,8,9. Our observations and those of others45,46 to date are unable to provide definitive evidence regarding the zoonotic potential of CWD, atypical/Nor98 scrapie or H-type BSE. The extended incubation period of the scrapie-affected macaque in the current study also underscores the limitations of rodent models expressing human PrP for assessing the zoonotic potential of some prion diseases since their lifespan remains limited to approximately two years21,47,48. This point is illustrated by the fact that the recently reported transmission of scrapie to humanized mice was not associated with clinical signs for up to 750 days and occurred in an extreme minority of mice with only a marginal increase in attack rate upon second passage13. The low attack rate in these studies is certainly linked to the limited lifespan of mice compared to the very long periods of observation necessary to demonstrate the development of scrapie. Alternatively, one could estimate that a successful second passage is the result of strain adaptation to the species barrier, thus poorly relevant of the real zoonotic potential of the original scrapie isolate of sheep origin49. The development of scrapie in this primate after an incubation period compatible with its lifespan complements the study conducted in transgenic (humanized) mice; taken together these studies suggest that some isolates of sheep scrapie can promote misfolding of the human prion protein and that scrapie can develop within the lifespan of some primate species.

In addition to previous studies on scrapie transmission to primate1,8,9 and the recently published study on transgenic humanized mice13, our results constitute new evidence for recommending that the potential risk of scrapie for human health should not be dismissed. Indeed, human PrP transgenic mice and primates are the most relevant models for investigating the human transmission barrier. To what extent such models are informative for measuring the zoonotic potential of an animal TSE under field exposure conditions is unknown. During the past decades, many protective measures have been successfully implemented to protect cattle from the spread of c-BSE, and some of these measures have been extended to sheep and goats to protect from scrapie according to the principle of precaution. Since cases of c-BSE have greatly reduced in number, those protective measures are currently being challenged and relaxed in the absence of other known zoonotic animal prion disease. We recommend that risk managers should be aware of the long term potential risk to human health of at least certain scrapie isolates, notably for lymphotropic strains like the classical scrapie strain used in the current study. Relatively high amounts of infectivity in peripheral lymphoid organs in animals infected with these strains could lead to contamination of food products produced for human consumption. Efforts should also be maintained to further assess the zoonotic potential of other animal prion strains in long-term studies, notably lymphotropic strains with high prevalence like CWD, which is spreading across North America, and atypical/Nor98 scrapie (Nor98)50 that was first detected in the past two decades and now represents approximately half of all reported cases of prion diseases in small ruminants worldwide, including territories previously considered as scrapie free... Even if the prevailing view is that sporadic CJD is due to the spontaneous formation of CJD prions, it remains possible that its apparent sporadic nature may, at least in part, result from our limited capacity to identify an environmental origin.

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

Chronic Wasting Disease CWD TSE Prion

Cervid to human prion transmission

Kong, Qingzhong Case Western Reserve University, Cleveland, OH, United States

We hypothesize that:

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

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

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

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

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

ZOONOTIC CHRONIC WASTING DISEASE CWD TSE PRION UPDATE

here is the latest;

PRION 2018 CONFERENCE

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

Hermann M. Schatzl, Samia Hannaoui, Yo-Ching Cheng, Sabine Gilch (Calgary Prion Research Unit,

University of Calgary, Calgary, Canada) Michael Beekes (RKI Berlin), Walter Schulz-Schaeffer (University of Homburg/Saar, Germany), Christiane Stahl-Hennig (German Primate Center) & Stefanie Czub (CFIA Lethbridge).

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

After 5-7 years of incubation time some animals showed clinical symptoms indicative of prion disease, and prion neuropathology and PrPSc deposition were detected in spinal cord and brain of some euthanized animals. PrPSc in immunoblot was weakly detected in some spinal cord materials and various tissues tested positive in RT-QuIC, including lymph node and spleen homogenates. To prove prion infectivity in the macaque tissues, we have intracerebrally inoculated 2 lines of transgenic mice, expressing either elk or human PrP. At least 3 TgElk mice, receiving tissues from 2 different macaques, showed clinical signs of a progressive prion disease and brains were positive in immunoblot and RT-QuIC. Tissues (brain, spinal cord and spleen) from these and pre-clinical mice are currently tested using various read-outs and by second passage in mice. Transgenic mice expressing human PrP were so far negative for clear clinical prion disease (some mice >300 days p.i.). In parallel, the same macaque materials are inoculated into bank voles.

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

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

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

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

https://prion2018.org/

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

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

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

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

AND ANOTHER STUDY;

P172 Peripheral Neuropathy in Patients with Prion Disease

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

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

AND

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

AND

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

snip...

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

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

https://prion2018.org/

THURSDAY, OCTOBER 04, 2018

Cervid to human prion transmission 5R01NS088604-04 Update

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

http://chronic-wasting-disease.blogspot.com/2018/10/cervid-to-human-prion-transmission.html

snip...full text;

SATURDAY, FEBRUARY 09, 2019

Experts: Yes, chronic wasting disease in deer is a public health issue — for people

https://chronic-wasting-disease.blogspot.com/2019/02/experts-yes-chronic-wasting-disease-in.html

FRIDAY, JULY 26, 2019

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

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

SUNDAY, SEPTEMBER 08, 2019

***> Wisconsin Laboratory Testing Options for Prion Diseases, Wisconsin Neurologists, Clinical Laboratory Directors, and Infection Preventionists, Please Distribute Widely

Preparing for the Storm

https://creutzfeldt-jakob-disease.blogspot.com/2019/09/wisconsin-laboratory-testing-options.html

friendly fire, pass it forward, they call it iatrogenic cjd, or what i call 'tse prion poker', are you all in $$$

SATURDAY, MARCH 16, 2019

Medical Devices Containing Materials Derived from Animal Sources (Except for In Vitro Diagnostic Devices) Guidance for Industry and Food and Drug Administration Staff Document issued on March 15, 2019 Singeltary Submission

https://bovineprp.blogspot.com/2019/03/medical-devices-containing-materials.html

TUESDAY, APRIL 09, 2019

Horizon Health Network Moncton Hospital notified more than 700 patients after two cases of CJD were diagnosed both patients had undergone cataracts surgery before being diagnosed

https://creutzfeldt-jakob-disease.blogspot.com/2019/04/horizon-health-network-moncton-hospital.html

SUNDAY, MARCH 10, 2019

National Prion Disease Pathology Surveillance Center Cases Examined[1] Updated Feb 1, 2019 Variably protease-sensitive prionopathy VPSPr

https://prionunitusaupdate.blogspot.com/2019/03/national-prion-disease-pathology.html

MONDAY, AUGUST 26, 2019

Creutzfeldt Jakob Disease CJD, TSE, Prion, Surveillance Update August 2019

https://creutzfeldt-jakob-disease.blogspot.com/2019/08/creutzfeldt-jakob-disease-cjd-tse-prion.html

TSE PRION

https://www.facebook.com/groups/1557515941145821/?ref=bookmarks

Subject: Prion 2019 Conference

Thursday, May 23, 2019

Prion 2019 Emerging Concepts CWD, BSE, SCRAPIE, CJD, SCIENTIFIC PROGRAM Schedule and Abstracts

https://prionconference.blogspot.com/2019/05/prion-2019-emerging-concepts-cwd-bse.html

see full Prion 2019 Conference Abstracts

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

SATURDAY, JUNE 1, 2019

Traceability of animal protein byproducts in ruminants by multivariate analysis of isotope ratio mass spectrometry to prevent transmission of prion diseases

https://bovineprp.blogspot.com/2019/06/traceability-of-animal-protein.html

P132 Aged cattle brain displays Alzheimer's-like pathology that can be propagated in a prionlike manner

Ines Moreno-Gonzalez (1), George Edwards III (1), Rodrigo Morales (1), Claudia Duran-Aniotz (1), Mercedes Marquez (2), Marti Pumarola (2), Claudio Soto (1)

snip...

These results may contribute to uncover a previously unsuspected etiology surrounding some cases of sporadic AD. However, the early and controversial stage of the field of prion-like transmission in non-prion diseases added to the artificial nature of the animal models utilized for these studies, indicate that extrapolation of the results to humans should not be done without further experiments.

P75 Determining transmissibility and proteome changes associated with abnormal bovine prionopathy

Dudas S (1,2), Seuberlich T (3), Czub S (1,2)

In prion diseases, it is believed that altered protein conformation encodes for different pathogenic strains. Currently 3 different strains of bovine spongiform encephalopathy (BSE) are confirmed. Diagnostic tests for BSE are able to identify animals infected with all 3 strains, however, several diagnostic laboratories have reported samples with inconclusive results which are challenging to classify. It was suggested that these may be novel strains of BSE; to determine transmissibility, brain material from index cases were inoculated into cattle.

In the first passage, cattle were intra-cranially challenged with brain homogenate from 2 Swiss animals with abnormal prionopathy. The challenged cattle incubated for 3 years and were euthanized with no clinical signs of neurologic disease. Animals were negative when tested on validated diagnostic tests but several research methods demonstrated changes in the prion conformation in these cattle, including density gradient centrifugation and immunohistochemistry. Currently, samples from the P1 animals are being tested for changes in protein levels using 2-D Fluorescence Difference Gel Electrophoresis (2D DIGE) and mass spectrometry. It is anticipated that, if a prionopathy is present, this approach should identify pathways and targets to decipher the source of altered protein conformation. In addition, a second set of cattle have been challenged with brain material from the first passage. Ideally, these cattle will be given a sufficient incubation period to provide a definitive answer to the question of transmissibility.

=====prion 2018===

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

https://prion2018.org/

Singeltary PloS

IBNC BSE TSE Prion mad cow disease

***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

SUNDAY, MAY 26, 2019

Arguments for Alzheimer's and Parkinson's diseases caused by prions Stanley B. Prusiner

"From a large array of bioassays, we conclude that AD, PD, MSA, and the frontotemporal dementias, including PSP and CBD, are all prion diseases"

https://betaamyloidcjd.blogspot.com/2019/05/arguments-for-alzheimers-and-parkinsons.html

MONDAY, FEBRUARY 25, 2019

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

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

wasted days and wasted nights...Freddy Fender

Terry S. Singeltary Sr.

Tuesday, September 10, 2019

FSIS [Docket No. FSIS–2019–0021] Notice of Request To Renew an Approved Information Collection: Specified Risk Materials Singeltary Submission

https://specifiedriskmaterial.blogspot.com/2019/09/fsis-docket-no-fsis20190021-notice-of.html