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

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

DEPARTMENT OF AGRICULTURE

Animal and Plant Health Inspection Service

[Docket No. APHIS-2018-0087]

Concurrence With OIE Risk Designation for Bovine Spongiform Encephalopathy

AGENCY: Animal and Plant Health Inspection Service, USDA.

ACTION: Notice.

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

DATES: We will consider all comments that we receive on or before August 16, 2019.

ADDRESSES: You may submit comments by either of the following methods: Federal eRulemaking Portal: Go to http://www.regulations.gov/#!docketDetail;D=APHIS-2018-0087 .

Postal Mail/Commercial Delivery: Send your comment to Docket No. APHIS-2018-0087, Regulatory Analysis and Development, PPD, APHIS, Station 3A-03.8, 4700 River Road, Unit 118, Riverdale, MD 20737- 1238. Supporting documents and any comments we receive on this docket may be viewed at http://www.regulations.gov/#!docketDetail;D=APHIS-2018-0087 or in our reading room, which is located in Room 1141 of the USDA South Building, 14th Street and Independence Avenue SW, Washington, DC. Normal reading room hours are 8 a.m. to 4:30 p.m., Monday through Friday, except holidays. To be sure someone is there to help you, please call (202) 799-7039 before coming.

FOR FURTHER INFORMATION CONTACT: Dr. Kari Coulson, Senior Staff Veterinarian, Strategy and Policy, VS, APHIS, 920 Main Campus Drive, Suite 200, Raleigh, NC 27606; (919) 480-9876; email: kari.f.coulson@usda.gov.

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

https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/animal-and-animal-product-import-information/animal-health-status-of-regions .

The list can also be obtained by writing to APHIS at Regionalization Evaluation Services, 4700 River Road, Unit 38, Riverdale, MD 20737. Under the regulations, APHIS may classify a region for BSE in one of two ways. One way is for regions that have not received a risk classification from

[[Page 28002]]

the World Organization for Animal Health (OIE) to request classification by APHIS. The other way is for APHIS to concur with the classification given to a country or region by the OIE. If the OIE has classified a region as either BSE negligible risk or BSE controlled risk, APHIS will seek information to support concurrence with the OIE classification. This information may be publicly available information, or APHIS may request that regions supply the same information given to the OIE. APHIS will announce in the Federal Register, subject to public comment, its intent to concur with an OIE classification. In accordance with this process, we are giving notice in this document that APHIS intends to concur with the OIE risk classification of Nicaragua as a region of negligible risk for BSE. The OIE recommendation regarding Nicaragua can be viewed at http://www.oie.int/animal-health-in-the-world/official-disease-status/bse/list-of-bse-risk-status/ . The conclusions of the OIE scientific commission for Nicaragua can be viewed at

http://www.oie.int/fileadmin/Home/eng/Internationa_Standard_Setting/docs/pdf/SCAD/A_SCAD_Feb2018.pdf

(page 47). After reviewing any comments we receive, we will announce our final determination regarding the BSE classification of Nicaragua in the Federal Register, along with a discussion of and response to pertinent issues raised by commenters. If APHIS recognizes Nicaragua as negligible risk for BSE, the Agency will include that region on the list of regions of negligible risk for BSE that is available to the public on the Agency's website at

https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/animal-and-animal-product-import-information/animal-health-status-of-regions .

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

Done in Washington, DC, this 11th day of June 2019. Kevin Shea, Administrator, Animal and Plant Health Inspection Service. [FR Doc. 2019-12654 Filed 6-14-19; 8:45 am] BILLING CODE 3410-34-P

https://www.govinfo.gov/content/pkg/FR-2019-06-17/html/2019-12654.htm

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

Greetings APHIS et al,

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

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

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

The FDA PART 589 TSE PRION FEED ban has failed terribly bad, and is still failing, since August 1997. there is tonnage and tonnage of banned potential mad cow feed that went into commerce, and still is, with one decade, 10

YEARS, post August 1997 FDA PART 589 TSE PRION FEED ban, 2007, with 10,000,000 POUNDS, with REASON, Products manufactured from bulk feed containing blood meal that was cross contaminated with prohibited meat and bone meal and the labeling did not bear cautionary BSE statement. you can see all these feed ban warning letters and tonnage of mad cow feed in commerce, year after year, that is not accessible on the internet anymore like it use to be, you can see history of the FDA failure August 1997 FDA PART 589 TSE PRION FEED ban here, but remember this, we have a new outbreak of TSE Prion disease in a new livestock species, the camel, and this too is very worrisome.

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

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

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

Prion Conference 2018

O5 Prion Disease in Dromedary Camels

Babelhadj B (1), Di Bari MA (2), Pirisinu L (2), Chiappini B (2), Gaouar SB (3), Riccardi G (2), Marcon S (2), Agrimi U (2), Nonno R (2), Vaccari G (2) (1) École Normale Supérieure Ouargla. Laboratoire de protection des écosystèmes en zones arides et semi arides University Kasdi Merbah Ouargla, Ouargla, Algeria; (2) Istituto Superiore di Sanità, Department of Food Safety, Nutrition and Veterinary Public Health, Rome, Italy (3) University Abou Bekr Bélkaid, Tlemcen, Algeria.

Prions are responsible for fatal and transmissible neurodegenerative diseases including CreutzfeldtJakob disease in humans, scrapie in small ruminants and bovine spongiform encephalopathy (BSE). Following the BSE epidemic and the demonstration of its zoonotic potential, general concerns have been raised on animal prions.

Here we report the identification of a prion disease in dromedary camels (Camelus dromedarius) in Algeria and designate it as Camel Prion Disease (CPD). In the last years, neurological symptoms have been observed in adult male and female dromedaries presented for slaughter at the Ouargla abattoir. The symptoms include weight loss, behavioral abnormalities and neurological symptoms such as tremors, aggressiveness, hyper-reactivity, typical down and upwards movements of the head, hesitant and uncertain gait, ataxia of the hind limbs, occasional falls and difficult getting up. During 2015 and 2016, symptoms suggestive of prion disease were observed in 3.1% of 2259 dromedaries presented at ante-mortem examination. Laboratory diagnosis was obtained in three symptomatic dromedaries, sampled in 2016 and 2017, by the detection of typical neurodegeneration and disease-specific prion protein (PrPSc) in brain tissues.

Histopathological examination revealed spongiform change, gliosis and neuronal loss preferentially in grey matter of subcortical brain areas. Abundant PrPSc deposition was detected in the same brain areas by immunohistochemistry and PET-blot. Western blot analysis confirmed the presence of PK-resistant PrPSc, whose N-terminal cleaved PK-resistant core was characterized by a mono-glycosylated dominant form and by a distinctive N-terminal cleavage, different from that observed in BSE and scrapie.

PrPSc was also detected, by immunohistochemistry, in all sampled lymph nodes (cervical, prescapular and lumbar aortic) of the only animal from which they were collected.

The PRNP sequence of the two animals for which frozen material was available, showed 100% nucleotide identity with the PRNP sequence already reported for dromedary camel.

Overall, these data demonstrate the presence of a prion disease in dromedary camels whose nature, origin and spread need further investigations. However, our preliminary observations on the rather high prevalence of symptomatic dromedaries and the involvement of lymphoid tissues, are consistent with CPD being an infectious disease. In conclusion, the emergence of a new prion disease in a livestock species of crucial importance for millions of people around the world, makes urgent to assess the risk for humans and to develop policies able to control the spread of the disease in animals and to minimize human exposure.

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

CDC

New Outbreak of TSE Prion in NEW LIVESTOCK SPECIES

Mad Camel Disease

Volume 24, Number 6—June 2018 Research

Prion Disease in Dromedary Camels, Algeria
Abstract

Prions cause fatal and transmissible neurodegenerative diseases, including Creutzfeldt-Jakob disease in humans, scrapie in small ruminants, and bovine spongiform encephalopathy (BSE). After the BSE epidemic, and the associated human infections, began in 1996 in the United Kingdom, general concerns have been raised about animal prions. We detected a prion disease in dromedary camels (Camelus dromedarius) in Algeria. Symptoms suggesting prion disease occurred in 3.1% of dromedaries brought for slaughter to the Ouargla abattoir in 2015–2016. We confirmed diagnosis by detecting pathognomonic neurodegeneration and disease-specific prion protein (PrPSc) in brain tissues from 3 symptomatic animals. Prion detection in lymphoid tissues is suggestive of the infectious nature of the disease. PrPSc biochemical characterization showed differences with BSE and scrapie. Our identification of this prion disease in a geographically widespread livestock species requires urgent enforcement of surveillance and assessment of the potential risks to human and animal health.

SNIP...

The possibility that dromedaries acquired the disease from eating prion-contaminated waste needs to be considered.

Tracing the origin of prion diseases is challenging. In the case of CPD, the traditional extensive and nomadic herding practices of dromedaries represent a formidable factor for accelerating the spread of the disease at long distances, making the path of its diffusion difficult to determine. Finally, the major import flows of live animals to Algeria from Niger, Mali, and Mauritania (27) should be investigated to trace the possible origin of CPD from other countries.

Camels are a vital animal species for millions of persons globally. The world camel population has a yearly growth rate of 2.1% (28). In 2014, the population was estimated at ≈28 million animals, but this number is probably underestimated.. Approximately 88% of camels are found in Africa, especially eastern Africa, and 12% are found in Asia. Official data reported 350,000 dromedaries in Algeria in 2014 (28).

On the basis of phenotypic traits and sociogeographic criteria, several dromedary populations have been suggested to exist in Algeria (29). However, recent genetic studies in Algeria and Egypt point to a weak differentiation of the dromedary population as a consequence of historical use as a cross-continental beast of burden along trans-Saharan caravan routes, coupled with traditional extensive/nomadic herding practices (30).

Such genetic homogeneity also might be reflected in PRNP. Studies on PRNP variability in camels are therefore warranted to explore the existence of genotypes resistant to CPD, which could represent an important tool for CPD management as it was for breeding programs for scrapie eradication in sheep.

In the past 10 years, the camel farming system has changed rapidly, with increasing setup of periurban dairy farms and dairy plants and diversification of camel products and market penetration (13). This evolution requires improved health standards for infectious diseases and, in light of CPD, for prion diseases.

The emergence of another prion disease in an animal species of crucial importance for millions of persons worldwide makes it necessary to assess the risk for humans and develop evidence-based policies to control and limit the spread of the disease in animals and minimize human exposure. The implementation of a surveillance system for prion diseases would be a first step to enable disease control and minimize human and animal exposure. Finally, the diagnostic capacity of prion diseases needs to be improved in all countries in Africa where dromedaries are part of the domestic livestock.

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

> IMPORTS AND EXPORTS <

***SEE MASSIVE AMOUNTS OF BANNED ANIMAL PROTEIN AKA MAD COW FEED IN COMMERCE USA DECADES AFTER POST BAN ***

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

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

"(Cows, sheep and deer can still legally be processed into bone and blood meal feed for pigs, pets and chickens; then they can be rendered and fed back to cows, deer and other ruminants.)" i will get back to this because we now know that cwd and scrapie will transmit to pigs by the oral route, but first;

NOW, LET'S GO BACK EVEN FURTHER, TO THE YEAR 2002, AND WHAT DID THE GREAT STATE OF WISCONSIN HAVE TO SAY ABOUT CHRONIC WASTING DISEASE CWD TSE PRION 6 YEARS BEFORE THIS ARTICLE 'THE KILLER AMONG US', BACK IN 2008, WHAT DID WISCONSIN SAY IN 2002 ABOUT CWD?

CHRONIC WASTING DISEASE CONGRESS Serial No. 107-117 May 16, 2002

CHRONIC WASTING DISEASE

JOINT OVERSIGHT HEARING BEFORE THE SUBCOMMITTEE ON FORESTS AND FOREST HEALTH JOINT WITH THE SUBCOMMITTEE ON FISHERIES CONSERVATION, WILDLIFE AND OCEANS OF THE

COMMITTEE ON RESOURCES U.S. HOUSE OF REPRESENTATIVES ONE HUNDRED SEVENTH CONGRESS SECOND SESSION

May 16, 2002

Serial No. 107-117

snip...

Mr. MCINNIS. Today, this joint Subcommittee hearing will explore an issue of immeasurable importance to the growing number of communities in wide-ranging parts of this country, the growing incidence of Chronic Wasting Disease in North America's wild and captive deer and elk populations. In a matter of just a few months, this once parochial concern has grown into something much larger and much more insidious than anyone could have imagined or predicted.

As each day passes, this problem grows in its size, scope, and consequence. One thing becomes clear. Chronic Wasting Disease is not a Colorado problem. It is a Wisconsin problem or a Nebraska or Wyoming problem. It is a national problem and anything short of a fully integrated, systematic national assault on this simply will not do, which is precisely why we brought our group together here today.

snip...

So this is a disease that is spreading throughout the continent and it is going to require a national response as well as the efforts that are currently taking place in States like Wisconsin, Colorado, Nebraska, Wyoming, the interest they now have down in Texas and some of the neighboring States that have large white-tailed deer population and also elk.

This is a huge issue for us, Mr. Chairman, in the State of Wisconsin. I want to commend Governor McCallum and your staff and the various agencies for the rapid response that you have shown, given the early detection of CWD after the last deer hunting season. The problem that we have, though, is just a lack of information, good science in regards to what is the best response, how dangerous is this disease. We cannot close the door, quite frankly, with the paucity of scientific research that is out there right now in regards to how the disease spreads, the exposure of other livestock herds—given the importance of our dairy industry in the State, that is a big issue—and also the human health effects.

https://www.govinfo.gov/content/pkg/CHRG-107hhrg79658/pdf/CHRG-107hhrg79658.pdf?fbclid=IwAR1-dMPpYLher4m8SyMICwoGXNyQcVjcinPvAw8CvIys1IEG7hxgzWplJlk

SUNDAY, JUNE 02, 2019

WISCONSIN THE KILLER AMONG US CWD TSE PRION JANUARY 31, 2008 revisited May 2019

Greetings TSE Prion world, Milwaukee Magazine, and Mary Van de Kamp Nohl.

i thought i would post again, after over a decade, an article that was printed in the Milwaukee Journal Magazine some 11 years ago about chronic wasting disease cwd tse prion in deer and elk. this was a brilliantly written article about aka mad deer disease. i wanted to post this again in full, and then update the article with the latest science up to 2019, and what kind of dire straights we are in right now with the cwd tse prion, and how, by letting corporate America regulated itself, will not work, and in some cases, it could kill you, and in other cases, release a plague on us all, which in this case, is exactly what happened. wake up America, here's your sign...

THE KILLER AMONG US

https://chronic-wasting-disease.blogspot.com/2019/06/wisconsin-killer-among-us-cwd-tse-prion.html

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

Let us review this BSE MRR TSE Prion policy shall we, but before doing so, let's clear up something, i.e. atypical BSE TSE Prion.

PLEASE BE ADVISED THERE IS NO SCIENTIFIC PROOF THAT ANY ATYPICAL BSE TSE PRION IS OF A SPONTANEOUS OLD AGE DISEASE, NOT CAUSED BY FEED, THIS IS FALSE AND UNPROVEN, IN FACT, ATYPICAL BSE OF THE L AND H TYPE ARE TRANSMISSIBLE BY ORAL ROUTE. THIS STATEMENT THAT ATYPICAL BSE IS A SPONTANEOUS EVENT CAUSED BY OLD AGE, CAUSED BY NOTHING, IS ABSOLUTELY A LIE, AND THE GOVERNMENT OF BRAZIL, AND OTHER GOVERNMENTS (Poland and France) THAT PRODUCE SUCH STATEMENTS, KNOWS THIS IS AN UNPROVEN STATEMENT...TERRY SINGELTARY SR.

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/

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.

In 2006, a case of H-type bovine spongiform encephalopathy (BSE) was reported in a cow with a previously unreported prion protein polymorphism (E211K).

The E211K polymorphism is heritable and homologous to the E200K mutation in humans that is the most frequent PRNP mutation associated with familial Creutzfeldt-Jakob disease.

Although the prevalence of the E211K polymorphism is low, cattle carrying the K211 allele develop H-type BSE with a rapid onset after experimental inoculation by the intracranial route.

The purpose of this study was to investigate whether the agents of H-type BSE or H-type BSE associated with the E211K polymorphism transmit to wild type cattle or cattle with the K211 allele after oronasal exposure.

Wild type (EE211) or heterozygous (EK211) cattle were oronasally inoculated with either H-type BSE from the 2004 US Htype BSE case (n=3) or from the 2006 US H-type case associated with the E211K polymorphism (n=4) using 10% w/v brain homogenates.

Cattle were observed daily throughout the course of the experiment for the development of clinical signs.

At approximately 50 months post-inoculation, one steer (EK211 inoculated with E211K associated H-BSE) developed clinical signs including inattentiveness, loss of body condition, weakness, ataxia, and muscle fasciculations and was euthanized.

Enzyme immunoassay confirmed that abundant misfolded protein was present in the brainstem, and immunohistochemistry demonstrated PrPSc throughout the brain.

Western blot analysis of brain tissue from the clinically affected steer was consistent with the E211K H-type BSE inoculum.

With the experiment currently at 55 months post-inoculation, no other cattle in this study have developed clinical signs suggestive of prion disease. 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, 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

***> 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.

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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

SATURDAY, JUNE 01, 2019

Brazil reports another cases of mad cow disease atypical BSE TSE Prion

PLEASE BE ADVISED THERE IS NO SCIENTIFIC PROOF THAT ANY ATYPICAL BSE TSE PRION IS OF A SPONTANEOUS OLD AGE DISEASE, NOT CAUSED BY FEED, THIS IS FALSE AND UNPROVEN, IN FACT,

ATYPICAL BSE OF THE L AND H TYPE ARE TRANSMISSIBLE BY ORAL ROUTE. THIS STATEMENT THAT ATYPICAL BSE IS A SPONTANEOUS EVENT CAUSED BY OLD AGE, CAUSED BY NOTHING, IS ABSOLUTELY A LIE, AND THE GOVERNMENT OF BRAZIL, AND OTHER GOVERNMENTS THAT PRODUCE SUCH STATEMENTS, KNOWS THIS IS AN UNPROVEN STATEMENT...TERRY SINGELTARY SR.

https://bse-atypical.blogspot.com/2019/06/brazil-reports-another-cases-of-mad-cow.html

PLEASE BE ADVISED, THE USA FDA PART 589 TSE PRION FEED BAN OF AUGUST 1997, IS/WAS/HAS BEEN, EXTREMELY FLAWED AND A TOTAL FAILURE, PLEASE SEE;

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

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

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

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

FDA BSE/Ruminant Feed Inspections Firms Inventory

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

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

NAI = NO ACTION INDICATED

OAI = OFFICIAL ACTION INDICATED

VAI = VOLUNTARY ACTION INDICATED

RTS = REFERRED TO STATE

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

SEE MASSIVE TONNAGE OF BANNED FDA PART 589 -- SUBSTANCES PROHIBITED FROM USE IN ANIMAL FOOD OR FEED VIOLATIONS PRODUCT IN COMMERCE SINCE AUGUST 1997. THE MAD COW FEED BAN WAS AND IS STILL NOTHING BUT INK ON PAPER...TERRY

TUESDAY, APRIL 18, 2017

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

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

TUESDAY, JANUARY 17, 2017

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

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

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

***> Wednesday, January 23, 2019

> CFIA SFCR Guidance on Specified risk material (SRM) came into force on January 15, 2019 <

https://specifiedriskmaterial.blogspot.com/2019/01/cfia-sfcr-guidance-on-specified-risk.html

PLOS ONE Journal

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

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

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

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

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

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

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

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

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

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

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

***> P.108: Successful oral challenge of adult cattle with classical BSE

Sandor Dudas1,*, Kristina Santiago-Mateo1, Tammy Pickles1, Catherine Graham2, and Stefanie Czub1
1Canadian Food Inspection Agency; NCAD Lethbridge; Lethbridge, Alberta, Canada; 2Nova Scotia Department of Agriculture; Pathology Laboratory; Truro, Nova Scotia, Canada

Classical Bovine spongiform encephalopathy (C-type BSE) is a feed- and food-borne fatal neurological disease which can be orally transmitted to cattle and humans. Due to the presence of contaminated milk replacer, it is generally assumed that cattle become infected early in life as calves and then succumb to disease as adults. Here we challenged three 14 months old cattle per-orally with 100 grams of C-type BSE brain to investigate age-related susceptibility or resistance. During incubation, the animals were sampled monthly for blood and feces and subjected to standardized testing to identify changes related to neurological disease. At 53 months post exposure, progressive signs of central nervous system disease were observed in these 3 animals, and they were euthanized. Two of the C-BSE animals tested strongly positive using standard BSE rapid tests, 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.

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

We are further examining explanations for the unusual disease presentation in the third challenged animal.

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

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

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

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

National Institute of Animal Health; Tsukuba, Japan

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

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

from all tested prion strains. On the other hand, human PrPC was converted by PrPSc from typical and H-type BSE in this PMCA condition.

Although these results were not compatible with the previous reports describing the lack of transmissibility of H-type BSE to ovine and human transgenic mice, our findings suggest that possible transmission risk of H-type BSE to sheep and human. Bioassay will be required to determine whether the PMCA products are infectious to these animals.

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

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

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

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

Keywords: Atypical BSE, oral transmission, RT-QuIC

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

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

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

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

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

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

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

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

Ivett Ackermann1 , Anne Balkema-Buschmann1 , Reiner Ulrich2 , Kerstin Tauscher2 , James C. Shawulu1 , Markus Keller1 , Olanrewaju I. Fatola1 , Paul Brown3 and Martin H. Groschup1*

Abstract

In classical bovine spongiform encephalopathy (C-BSE), an orally acquired prion disease of cattle, the ileal Peyer's patch (IPP) represents the main entry port for the BSE agent. In earlier C-BSE pathogenesis studies, cattle at 4–6 months of age were orally challenged, while there are strong indications that the risk of infection is highest in young animals. In the present study, unweaned calves aged 4–6 weeks were orally challenged to determine the earli- est time point at which newly formed PrPBSE and BSE infectivity are detectable in the IPP. For this purpose, calves were culled 1 week as well as 2, 4, 6 and 8 months post-infection (mpi) and IPPs were examined for BSE infectivity using a bovine PrP transgenic mouse bioassay, and for PrPBSE by immunohistochemistry (IHC) and protein misfolding cyclic amplifcation (PMCA) assays. For the frst time, BSE prions were detected in the IPP as early as 2 mpi by transgenic mouse bioassay and PMCA and 4 mpi by IHC in the follicular dendritic cells (FDCs) of the IPP follicles. These data indi- cate that BSE prions propagate in the IPP of unweaned calves within 2 months of oral uptake of the agent.

In summary, our study demonstrates for the frst time PrPBSE (by PMCA) and prion infectivity (by mouse bioassay) in the ileal Peyer's patch (IPP) of young calves as early as 2 months after infection. From 4 mpi nearly all calves showed PrPBSE positive IPP follicles (by IHC), even with PrPBSE accumulation detectable in FDCs in some animals. Finally, our results confrm the IPP as the early port of entry for the BSE agent and a site of initial propagation of PrPBSE and infectivity during the early pathogenesis of the disease. Terefore, our study supports the recommendation to remove the last four metres of the small intestine (distal ileum) at slaughter, as designated by current legal requirements for countries with a controlled BSE risk status, as an essential measure for consumer and public health protection.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5738053/pdf/13567_2017_Article_495.pdf

A study comparing preclinical cattle infected naturally with BSE to clinically affected cattle either naturally or experimentally infected with BSE by the oral route found the most abundant PrPSc in the brainstem area (39), which is consistent with ascension to the brain from the gut by sympathetic and parasympathetic projections (40). In our experiment, abundant prions were observed in the brainstem of cattle with clinical signs of BSE, which is similar to the amount in their thalamus or midbrain regions. Interestingly, prions in the brainstem of cattle with clinical evidence of BSE seeded the RT-QuIC reactions faster than any other brain region despite the brainstem area having lower EIA OD values (Table 2) in comparison to other brain regions. This suggests that higher concentrations of prions do not necessarily seed the reaction faster. Perhaps prions of the brainstem exist in a preferred conformation for better conversion despite being present in lower concentrations.

snip...

https://www.frontiersin.org/articles/10.3389/fvets.2017.00242/full

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

https://www.reginfo.gov/public/do/DownloadDocument?objectID=54003900

http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.230.8886&rep=rep1&type=pdf

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

THURSDAY, JUNE 22, 2017

World Organisation for Animal Health (OIE) to establish liaison office in College Station, Texas OIE to establish liaison office in College Station, Texas

http://animalhealthreportpriontse.blogspot.com/2017/06/world-organisation-for-animal-health.html

MAYBE since the OIE will be closer now, right here in Texas, they will do their job, because the past will tell us, the OIE failed terribly with Transmissible Spongiform Encephalopathy, and to this day, they still fail in terms of the mad cow type disease. we know now that sporadic CJD is linked to typical and atypical BSE, typical and atypical Scrapie, and to Chronic Wasting Disease CWD TSE Prion, and to this day the OIE has denied any of this. the dead of every family member can lay that blame directly on the OIE and the USDA et al, imo, and these body bags are mounting as we speak...shame on you!

https://www.nature.com/nature/journal/v485/n7398/full/485279b.html#/comments

MONDAY, JANUARY 4, 2016

Long live the OIE, or time to close the doors on a failed entity?

http://transmissiblespongiformencephalopathy.blogspot.com/2016/01/long-live-oie-or-time-to-close-doors-on.html

the OIE BSE TSE Prion policy now, the BSE MRR, legalized the free trading of the TSE Prion disease, humans and animals have now become expendable. ...

‘’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.’’

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

snip...see ;

Sunday, October 18, 2015

World Organisation for Animal Health (OIE) and the Institut Pasteur Cooperating on animal disease and zoonosis research

http://bovineprp.blogspot.com/2015/10/world-organisation-for-animal-health.html

Thursday, December 17, 2015

Annual report of the Scientific Network on BSE-TSE 2015 EFSA-Q-2015-00738 10 December 2015

http://efsaopinionbseanimalprotein.blogspot.com/2015/12/annual-report-of-scientific-network-on.html

Saturday, December 12, 2015

*** BOVINE SPONGIFORM ENCEPHALOPATHY BSE TSE PRION REPORT DECEMBER 14, 2015

http://bovineprp.blogspot.com/2015/12/bovine-spongiform-encephalopathy-bse.html

TUESDAY, AUGUST 9, 2016

Concurrence with OIE Risk Designations for Bovine Spongiform Encephalopathy [Docket No. APHIS-2015-0055]

http://bovineprp.blogspot.com/2016/08/concurrence-with-oie-risk-designations.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

Saturday, July 16, 2016

Importation of Sheep, Goats, and Certain Other Ruminants [Docket No. APHIS-2009-0095]RIN 0579-AD10

WITH great disgust and concern, I report to you that the OIE, USDA, APHIS, are working to further legalize the trading of Transmissible Spongiform Encephalopathy TSE Pion disease around the globe.

THIS is absolutely insane. it's USDA INC.

http://scrapie-usa.blogspot.com/2016/07/importation-of-sheep-goats-and-certain.html

WEDNESDAY, JUNE 21, 2017

Docket No. FDA– 2009–N–0505 Agency Information Collection Activities; Proposed Collection; Recordkeeping and Reporting Requirements for Human Food and Cosmetics Manufactured From, Processed With, or Otherwise Containing Material From Cattle

http://animalhealthreportpriontse.blogspot.com/2017/06/docket-no-fda-2009n0505-agency.html

Wednesday, March 11, 2015

OIE and Centers for Disease Control and Prevention Reinforce Collaboration

http://madcowusda.blogspot.com/2015/03/oie-and-centers-for-disease-control-and.html

Friday, April 4, 2014

China, Australia, Argentina, Brazil, Uruguay, Morocco, Israel, South Africa and Saudi Arabia still retain BSE-related closures

http://usdameatexport.blogspot.com/2014/04/china-australia-argentina-brazil.html

Thursday, May 30, 2013

World Organization for Animal Health (OIE) has upgraded the United States' risk classification for mad cow disease to "negligible" from "controlled", and risk further exposing the globe to the TSE prion mad cow type disease

U.S. gets top mad-cow rating from international group and risk further exposing the globe to the TSE prion mad cow type disease

http://madcowusda.blogspot.com/2013/05/world-organization-for-animal-health.html

http://transmissiblespongiformencephalopathy.blogspot.com/2013/05/statement-from-agriculture-secretary.html

The OIE is nothing more than a trading brokerage for the Transmissible Spongiform Encephalopathy TSE prion disease aka mad cow type disease. Frances is still in the midst of a mad cow disease outbreak with atypical BSE cases still growing. mad cow disease is so bad in France, as with the USA, they stopped testing for mad cow disease (France altogether and the USA to figures so low, you would only detect a case of mad cow disease, only by chance).

from the inside looking out ;

Quote: Maybe familirise yourself with the OIE. The primary concern is animal health of the world they are the animal version of the WHO. It is a long way down from that ivory tower but here we go, until pressured by the USA representatives a country could not export animals for 6 years after finding a BSE/BASE positive animal so under the old rules the US would not be able to export anywhere in the world for another 4 1/2 years. Who got the risk levels system put in to allow some trade - your US representatives. You guys want to change rules - OK , but you do not get special rules that only apply to the US. As i have told you before Sand h I market all my own slaughter animals and you know that, so don't do the whole holier than thow act.

With all due respect, it is obvious that you know little about the OIE and how it actually works. Having been to their offices in Paris and talked personally with the Head of the Animal Test Section, you would choke if you knew how many lobby groups attend that office daily. There is a steady stream of paid lobby groups that have one goal in life and that is to sway the Section Heads of each department within the OIE to suit the needs of different jurisdictions around the world, which curiously enough, also includes the USA and Canada. Anyone can go there and chat with them - providing they can provide valid cause to be let in. To say that the only goal of the OIE is animal health is actually only part of their function. They are more than that and my discussions with Dr. Diaz there has showed me that. But to blindly make a statement regarding what they do when you have no idea what they actually do is like eating the skin of the orange and not knowing what is actually under.

Interestingly you state that the US Government applied pressure (to the OIE) I assume and that is a great example of the lobby groups doing their job. So, at the end of the day, one can safely assume that it is the pressure applied by certain influential lobby groups that will determine a likely outcome to an apparent OIE directive. Man alive, isn't it great to live in a democracy wherein the people get to make the choices and not just some "other" interested party or group - say like.....Cargill or Tyson for example?

So, one last question, question?

Who wags the tail of that dog?? And for what reason other than one that is purely associated with trade and international agreements and greed?

And you think it is so simply explainable.

end...tss

Subject: UPDATED WHO Guidelines include tissues from Cervidae affected with Chronic Wasting Disease (CWD)

Distribution of infectivity in animal tissue and body fluids

Introduction

A.2.1 The following table (Table A2) presents a guide to the possible presence of TSE infectivity in various tissues and body fluids of cattle (exposed naturally or experimentally and orally to first passage BSE agent), sheep and goats (exposed naturally to scrapie agents and potentially to the BSE agent) irrespective of the stage of incubation. Table A2 has been updated in June 2010 taking account of the updated WHO Guidelines published in 2010 (WHO, 2010).

*** A.2.2 This is the first time that the WHO Guidelines include tissues from Cervidae affected with Chronic Wasting Disease (CWD). CWD has not been reported in Europe despite some surveillance for it and there are no specific regulations in force. Should information on TSE infectivity in CWD be required, for example in regard to Cervidae in zoos or in animals in transit through our ports,*** please see updated 2010 WHO Guidelines.

The inclusion of infectivity data in CWD in these Tables was considered important for three reasons: 1) CWD is continuing its spread to new regions of North America, 2) infectivity has been convincingly demonstrated in several bodily secretions and excretions of infected deer and 3) CWD is the only form of animal Transmissible Spongiform Encephalopathies (TSE) that exists in the wild and, although not presently considered to be an important concern for human, could pose serious problems of control in the future, especially as a potential source of infection in other animal species.

http://www.who.int/bloodproducts/tablestissueinfectivity.pdf

https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/209760/Annex_A2_-_Distribution_of_infectivity_in_animal_tissue.pdf

http://www.who.int/bloodproducts/TSEPUBLISHEDREPORT.pdf

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

O.4.3

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

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

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

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

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

Results: In clinically diseased macaques (5 years p.i. + 6 mo.), PrPres deposits were widely spread in neuronal tissues (including the peripheral sympathetic and parasympathetic nervous system) and in lymphoid tissues including tonsils. In asymptomatic disease carriers, PrPres deposits could be detected in intestinal lymph nodes as early as 1 year p.i., but CNS tissues were negative until 3 – 4 years p.i. Lumbal/sacral segments of the spinal cord and medulla oblongata were PrPres positive as early as 4.1 years p.i., whereas sympathetic trunk and all thoracic/cervical segments of the spinal cord were still negative for PrPres. However, tonsil samples were negative in all asymptomatic cases.

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

P.4.23

Transmission of atypical BSE in humanized mouse models

Liuting Qing1, Wenquan Zou1, Cristina Casalone2, Martin Groschup3, Miroslaw Polak4, Maria Caramelli2, Pierluigi Gambetti1, Juergen Richt5, Qingzhong Kong1 1Case Western Reserve University, USA; 2Instituto Zooprofilattico Sperimentale, Italy; 3Friedrich-Loeffler-Institut, Germany; 4National Veterinary Research Institute, Poland; 5Kansas State University (Previously at USDA National Animal Disease Center), USA

Background: Classical BSE is a world-wide prion disease in cattle, and the classical BSE strain (BSE-C) has led to over 200 cases of clinical human infection (variant CJD). Atypical BSE cases have been discovered in three continents since 2004; they include the L-type (also named BASE), the H-type, and the first reported case of naturally occurring BSE with mutated bovine PRNP (termed BSE-M). The public health risks posed by atypical BSE were largely undefined.

Objectives: To investigate these atypical BSE types in terms of their transmissibility and phenotypes in humanized mice. Methods: Transgenic mice expressing human PrP were inoculated with several classical (C-type) and atypical (L-, H-, or Mtype) BSE isolates, and the transmission rate, incubation time, characteristics and distribution of PrPSc, symptoms, and histopathology were or will be examined and compared.

Results: Sixty percent of BASE-inoculated humanized mice became infected with minimal spongiosis and an average incubation time of 20-22 months, whereas only one of the C-type BSE-inoculated mice developed prion disease after more than 2 years. Protease-resistant PrPSc in BASE-infected humanized Tg mouse brains was biochemically different from bovine BASE or sCJD. PrPSc was also detected in the spleen of 22% of BASE-infected humanized mice, but not in those infected with sCJD. Secondary transmission of BASE in the humanized mice led

to a small reduction in incubation time. The atypical BSE-H strain is also transmissible with distinct phenotypes in the humanized mice, but no BSE-M transmission has been observed so far.

Discussion: Our results demonstrate that BASE is more virulent than classical BSE, has a lymphotropic phenotype, and displays a modest transmission barrier in our humanized mice.

BSE-H is also transmissible in our humanized Tg mice.

The possibility of more than two atypical BSE strains will be discussed.

Supported by NINDS NS052319, NIA AG14359, and NIH AI 77774.

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

P03.137

Transmission of BSE to Cynomolgus Macaque, a Non-human Primate; Development of Clinical Symptoms and Tissue Distribution of PrPSC

Yamakawa, Y1; Ono, F2; Tase, N3; Terao, K3; Tannno, J3; Wada, N4; Tobiume, M5; Sato, Y5; Okemoto-Nakamura, Y1; Hagiwara, K1; Sata, T5 1National Institure of Infectious diseases, Cell biology and Biochemistry, Japan; 2Corporation for Production and Research Laboratory Primates., Japan; 3National Institure of Biomedical Innovation, Tsukuba Primate Reserch Center, Japan; 4Yamauchi Univ., Veterinary Medicine, Japan; 5National Institure of Infectious diseases, Pathology, Japan

Two of three cynomolgus monkeys developed abnormal neuronal behavioral signs at 30-(#7) and 28-(#10) months after intracerebral inoculation of 200ul of 10% brain homogenates of BSE affected cattle (BSE/JP6). Around 30 months post inoculation (mpi), they developed sporadic anorexia and hyperekplexia with squeal against environmental stimulations such as light and sound. Tremor, myoclonic jerk and paralysis became conspicuous during 32 to 33-mpi, and symptoms become worsened according to the disease progression. Finally, one monkey (#7) fell into total paralysis at 36-mpi. This monkey was sacrificed at 10 days after intensive veterinary care including infusion and per oral supply of liquid food. The other monkey (#10) had to grasp the cage bars to keep an upright posture caused by the sever ataxia. This monkey was sacrificed at 35-mpi. EEG of both monkeys showed diffuse slowing. PSD characteristic for sporadic CJD was not observed in both monkeys. The result of forearm movement test showed the hypofunction that was observed at onset of clinical symptoms. Their cognitive function determined by finger maze test was maintained at the early stage of sideration. However, it was rapidly impaired followed by the disease progression. Their autopsied tissues were immunochemically investigated for the tissue distribution of PrPSc. Severe spongiform change in the brain together with heavy accumulation of PrPSc having the type 2B/4 glycoform profile confirmed successful transmission of BSE to Cynomolgus macaques. Granular and linear deposition of PrPSC was detected by IHC in the CNS of both monkeys. At cerebral cortex, PrPSC was prominently accumulated in the large plaques. Sparse accumulation of PrPSc was detected in several peripheral nerves of #7 but not in #10 monkey, upon the WB analysis. Neither #7 nor #10 monkey accumulated detectable amounts of PrPSc in their lymphatic organs such as tonsil, spleen, adrenal grands and thymus although PrPSc was barely detected in the submandibular lymph node of #7 monkey. Such confined tissue distribution of PrPSc after intracerebral infection with BSE agent is not compatible to that reported on the Cynomolgus macaques infected with BSE by oral or intra-venous (intra-peritoneal) routs, in which PrPSc was accumulated at not only CNS but also widely distributed lymphatic tissues.

P04.27

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

Holznagel, E1; Yutzy, B1; Deslys, J-P2; Lasmézas, C2; Pocchiari, M3; Ingrosso, L3; Bierke, P4; Schulz-Schaeffer, W5; Motzkus, D6; Hunsmann, G6; Löwer, J1 1Paul-Ehrlich-Institut, Germany; 2Commissariat à l´Energie Atomique, France; 3Instituto Superiore di Sanità, Italy; 4Swedish Institute for Infectious Disease control, Sweden; 5Georg August University, Germany; 6German Primate Center, Germany

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

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

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

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

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

The work referenced was performed in partial fulfilment of the study “BSE in primates“ supported by the EU (QLK1-2002-01096).http://www.neuroprion.org/resources/pdf_docs/conferences/prion2007/abstract_book.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

WE know now, and we knew decades ago, that 5.5 grams of suspect feed in TEXAS was enough to kill 100 cows.

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

Risk of oral infection with bovine spongiform encephalopathy agent in primates

Corinne Ida Lasmézas, Emmanuel Comoy, Stephen Hawkins, Christian Herzog, Franck Mouthon, Timm Konold, Frédéric Auvré, Evelyne Correia, Nathalie Lescoutra-Etchegaray, Nicole Salès, Gerald Wells, Paul Brown, Jean-Philippe Deslys Summary The uncertain extent of human exposure to bovine spongiform encephalopathy (BSE)--which can lead to variant Creutzfeldt-Jakob disease (vCJD)--is compounded by incomplete knowledge about the efficiency of oral infection and the magnitude of any bovine-to-human biological barrier to transmission. We therefore investigated oral transmission of BSE to non-human primates. We gave two macaques a 5 g oral dose of brain homogenate from a BSE-infected cow. One macaque developed vCJD-like neurological disease 60 months after exposure, whereas the other remained free of disease at 76 months. On the basis of these findings and data from other studies, we made a preliminary estimate of the food exposure risk for man, which provides additional assurance that existing public health measures can prevent transmission of BSE to man.

snip...

BSE bovine brain inoculum

100 g 10 g 5 g 1 g 100 mg 10 mg 1 mg 0·1 mg 0·01 mg

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

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

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

PrPres biochemical detection

The comparison is made on the basis of calibration of the bovine inoculum used in our study with primates against a bovine brain inoculum with a similar PrPres concentration that was inoculated into mice and cattle.8 *Data are number of animals positive/number of animals surviving at the time of clinical onset of disease in the first positive animal (%). The accuracy of bioassays is generally judged to be about plus or minus 1 log. ic ip=intracerebral and intraperitoneal.

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

Published online January 27, 2005

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

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

http://web.archive.org/web/20040523230128/www.bseinquiry.gov.uk/files/ws/s145d.pdf

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http://web.archive.org/web/20030526212610/http://www.bseinquiry.gov.uk/files/ws/s147f.pdf

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

I ask Professor Kong ;

Thursday, December 04, 2008 3:37 PM

Subject: RE: re--Chronic Wating Disease (CWD) and Bovine Spongiform Encephalopathies (BSE): Public Health Risk Assessment

IS the h-BSE more virulent than typical BSE as well, or the same as cBSE, or less virulent than cBSE? just curious.....

Professor Kong reply ;

.....snip

As to the H-BSE, we do not have sufficient data to say one way or another, but we have found that H-BSE can infect humans. I hope we could publish these data once the study is complete. Thanks for your interest.

Best regards, Qingzhong Kong, PhD Associate Professor Department of Pathology Case Western Reserve University Cleveland, OH 44106 USA

P.4.23 Transmission of atypical BSE in humanized mouse models

Liuting Qing1, Wenquan Zou1, Cristina Casalone2, Martin Groschup3, Miroslaw Polak4, Maria Caramelli2, Pierluigi Gambetti1, Juergen Richt5, Qingzhong Kong1 1Case Western Reserve University, USA; 2Instituto Zooprofilattico Sperimentale, Italy; 3Friedrich-Loeffler-Institut, Germany; 4National Veterinary Research Institute, Poland; 5Kansas State University (Previously at USDA National Animal Disease Center), USA

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continents since 2004; they include the L-type (also named BASE), the H-type, and the first reported case of naturally occurring BSE with mutated bovine PRNP (termed BSE-M). The public health risks posed by atypical BSE were argely undefined.

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Supported by NINDS NS052319, NIA AG14359, and NIH AI 77774.

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

see full text ;

http://bse-atypical.blogspot.com/2009/10/atypical-bse-bse-and-other-human-and.html

>>> It is distinct from atypical BSE, which may develop spontaneously, according to information from the U.S. Centers for Disease Control and Prevention.

THIS IS A MYTH $$$

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

Thursday, March 24, 2016

FRANCE CONFIRMS BOVINE SPONGIFORM ENCEPHALOPATHY BSE MAD COW (ESB) chez une vache dans les Ardennes

http://bovineprp.blogspot.com/2016/03/france-confirms-bovine-spongiform.html

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 $$$

http://bovineprp.blogspot.com/2016/05/france-confirms-case-of-classical-mad.html

we have seen the spontaneous BSE epidemic in France, what about the other HIGH INCIDENCE ATYPICAL BSE COUNTRY OF POLAND, another atypical spontaneous event of high incidence. how can this be blamed on a happenstance of nothing, i.e. old age? goes against all junk science to date on the spontaneous atypical BSE i.e.

> In 2015, the OIE determined that atypical BSE occurred spontaneously at a low rate in all cattle populations and would be excluded for BSE risk. ...

>Atypical BSE occurs in older cattle, usually 8 years of age or greater, and does not appear to be associated with contaminated feed. Like classic or sporadic CJD in humans, it seems to arise rarely and spontaneously.

POLAND ATYPICAL BSE AND SPORADIC CJD

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

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POLAND ATYPICAL BSE AND SPORADIC CJD

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

Atypical status of bovine spongiform encephalopathy in Poland: a molecular typing study

Summary

The aim of this study was to analyze molecular features of protease-resistant prion protein (PrPres) in Western blots of BSE cases diagnosed in Poland with respect to a possible atypical status. Confirmed cases were analyzed by Western blotting with several monoclonal antibodies directed at N-terminal and core epitopes of prion protein (PrP). Most cases showed the classical glycoprofile characterized by the dominance of the di- over the monoglycosylated PrPres band, yielding di-/mono- ratios well above 2 and by reactivity with antibodies having their epitopes in bovine PrP region 110–242 (C-type cases). Surprisingly, seven cases of BSE were atypical. Six were classified as L-type based on a slightly lower molecular mass (Mr) of the non- glycosylated band with respect to C-types and a conspicuously low di-/mono- ratio of glycosylated PrPres bands approaching unity. One case was classified as H-type because of a higher Mr of PrPres bands on the blot when compared with C-type cases. A characteristic epitope of H-type PrPres occurred in the 101–110 region of PrP for which only antibody 12B2 had a sufficient affinity. The occurrence of atypical cases only in animals 9 years of age and older raises questions about the mechanisms of prion diseases and the origin of BSE.

https://link.springer.com/article/10.1007%2Fs00705-007-1062-6

Atypical status of bovine spongiform encephalopathy in Poland: a molecular typing study

M. P. Polak1, J. F. Zmudzinski1, J. G. Jacobs2, J. P. M. Langeveld2

1 National Veterinary Research Institute, Pulawy, Poland

2 Central Institute for Animal Disease Control (CIDC-Lelystad), Lelystad, The Netherlands Received 24 April 2007; Accepted 27 August 2007; Published online 26 September 2007 # Springer-Verlag 2007

snip...

Clarification of whether these atypical cases represent genuine strains of BSE would be accomplished by transmission studies in mice. Such studies have already been performed in France, Germany and Italy [3, 5, 14, 15]. For H-type cases in France, successful transmission was achieved in both wild-type, and transgenic mice expressing bovine and ovine PrPC. In Germany, successful transmission of both an L-type and an H-type case to transgenic mice overexpressing bovine PrPC has been described. PrPres from those mice was identical to the inoculum used in the study, proving the existence of distinct strains of BSE. All atypical features of those isolates were maintained in the inoculated mice, indicating the existence of several prion strains in cattle, or alternatively a possible evolution to a single BSE strain, as suggested from data obtained by Capobianco et al. with wild-type inbred mice [15]. This second hypothesis could fit with data from the United Kingdom, where over 180,000 cases of BSE were diagnosed by passive surveillance. British and European experience based on tissue analysis from clinically affected animals showed consistent characteristics of BSE agent not only on histological sections from cattle brains but also when inoculating mice, pointing to the existence of one uniform strain of BSE. Therefore, it is possible that a sporadic form of BSE present in the cattle population at a very low rate in the past could have spread to naive animals via contaminated meatand-bone meals. Spontaneous BSE, if it occurs, must be a very rare phenomenon. However, data for Poland, where 14% of all cases comprised an atypical form of BSE, seems to be in contradiction to this hypothesis. But when the average age of all positive cases in Poland is taken into account, BSE is generally found in older animals (mean age of 7.7). Analysis of the age structure of cattle in Poland in the period of 2002–2006 shows that 56–60% of all animals were 7 years old and above. A much larger number of cattle should be tested to get better insight into the real prevalence of atypical BSE. However, current tendencies based on economic analysis point to a decrease in the number of tests performed rather than expanding this scheme any further. It would be sensible to maintain a certain level of testing focused on the older age group to distinguish between a stable, thus sporadic-based, situation of BSE, or alternatively a fade-out, thus epidemic-based, situation. Exploring the subject of spontaneous BSE in the cattle population may be ceased for economic reasons, and it may never be known while this answer is in our reach thanks to great financial efforts in recent years.

https://core.ac.uk/download/pdf/29259038.pdf

Poland is Proof atypical BSE is NOT an old cow spontaneous disease...tss

Number of reported cases of bovine spongiform encephalopathy (BSE) in farmed cattle worldwide* (excluding the United Kingdom) Country/Year

Poland

89 90 91 92 93 94 95 96 97 98 99 00 01 02 03 04 05 06 07 08 09 10 11 12 13 14 15 16

0 0 0 0 0 0 0 0 0 0 0 0 0 4f 5 11 19 10 9 5 4 2 1m 3 1 0 0 0

http://www.oie.int/animal-health-in-the-world/bse-situation-in-the-world-and-annual-incidence-rate/number-of-reported-cases-worldwide-excluding-the-united-kingdom/

Source: USDA, APHIS, VS

What is the level of passenger traffic arriving in the United States from Poland?

A total of 188,946 passengers arrived at US airports on direct flights from Poland in fiscal year 2000.

An undetermined number of passengers arrived in the US from Poland via indirect flights.

Under APHIS-PPQ's agricultural quarantine inspection monitoring, 451 air passengers from Poland were sampled for items of agricultural interest in fiscal year 2000.

Thirteen (13) of these passengers, or 2.9 percent, carried a total of 26.2 kg of meat items that could potentially harbor the pathogen(s) that cause BSE.

None of these passengers from whom meat items were confiscated reported plans to visit or work on a ranch or farm during their visit to the US.

Source: US Department of Transportation, and APHIS-PPQ Agricultural Quarantine Inspection data base CEI's plans for follow up: CEI has no plans to provide additional information on this situation. If you need more information or wish to comment, you may contact Judy Akkina at (970) 490-7852 or Carol Tuszynski at (970) 490-7893.

http://www.aphis.usda.gov/vs/ceah/cei/IW_2002_files/bse_poland0502.htm

What measures has USDA-APHIS taken to prevent the introduction of BSE? To prevent BSE from entering the United States, APHIS has restricted the importation of live ruminants and certain ruminant products from countries where BSE is known to exist.

Greetings FDA and public,

if you go to the below site, and search all BSE known countries and check out their air traffic illegal meat they have confiscated, and check out the low number checked, compared to actual passenger traffic, would not take too much for some nut to bring in FMD/TSEs into the USA as a 'suitcase bomb'.

[[Under APHIS-PPQ's agricultural quarantine inspection monitoring, 284 air passengers from Israel were sampled for items of agricultural interest in fiscal year 2001. Seven of these passengers, or 2 percent, carried a total of 11 kg of meat items that could potentially harbor the pathogen that causes BSE. None of these passengers from whom meat items were confiscated reported plans to visit or work on a ranch or farm during their visit to the U.S.]]

if they were to have questioned the terrorist that bombed the Twin Towers with jets, if they were to have questioned them at flight school in the USA, i am sure that they would have said they did not intend to visit the Twin Towers as a flying bomb either. what am i thinking, they probably did ask this? stupid me.

[[In 1999 a small amount of non-species specific meat and offal was imported and a small amount of fetal bovine serum (FBS) was also imported. FBS is considered to have a relatively low risk of transmitting BSE.]] more of

the USA infamous 'non-species coding system', wonder how many of these species are capable of carrying a TSE?

snip...

A total of 524,401 passengers arrived on direct flights to the U.S. from Israel in fiscal year 2000. This number does not include passengers who arrived in the U.S. from Israel via indirect flights. Under APHIS-PPQ's agricultural quarantine inspection monitoring, 284 air passengers from Israel were sampled for items of agricultural interest in fiscal year 2001. Seven of these passengers, or 2 percent, carried a total of 11 kg of meat items that could potentially harbor the pathogen that causes BSE. None of these passengers from whom meat items were confiscated reported plans to visit or work on a ranch or farm during their visit to the U.S.

http://www.aphis.usda.gov/vs/ceah/cei/bse_israel0602.htm

http://madcowusda.blogspot.com/2015/05/us-federal-government-is-unprepared-for.html

MONDAY, FEBRUARY 04, 2019

POLAND DETECTS BOVINE SPONGIFORM ENCEPHALOPATHY BSE TSE PRION

Poland is Proof atypical BSE is NOT an old cow spontaneous disease...tss

https://bse-atypical.blogspot.com/2019/02/poland-detects-bovine-spongiform.html

FRIDAY, FEBRUARY 01, 2019

Poland Exported 5,500 Pounds of Meat From Sick Cows to EU, what about mad cow disease?

Poland is Proof atypical BSE is NOT an old cow spontaneous disease...tss

https://bse-atypical.blogspot.com/2019/02/poland-exported-5500-pounds-of-meat.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

SATURDAY, JUNE 01, 2019

Brazil reports another cases of mad cow disease atypical BSE TSE Prion

PLEASE BE ADVISED THERE IS NO SCIENTIFIC PROOF THAT ANY ATYPICAL BSE TSE PRION IS OF A SPONTANEOUS OLD AGE DISEASE, NOT CAUSED BY FEED, THIS IS FALSE AND UNPROVEN, IN FACT, ATYPICAL BSE OF THE L AND H TYPE ARE TRANSMISSIBLE BY ORAL ROUTE. THIS STATEMENT THAT ATYPICAL BSE IS A SPONTANEOUS EVENT CAUSED BY OLD AGE, CAUSED BY NOTHING, IS ABSOLUTELY A LIE, AND THE GOVERNMENT OF BRAZIL, AND OTHER GOVERNMENTS THAT PRODUCE SUCH STATEMENTS, KNOWS THIS IS AN UNPROVEN STATEMENT...TERRY SINGELTARY SR.

https://bse-atypical.blogspot.com/2019/06/brazil-reports-another-cases-of-mad-cow.html

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

Subject: Prion 2019 Conference

See full Prion 2019 Conference Abstracts

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

see scientific program and follow the cwd studies here;

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

FRIDAY, MAY 24, 2019

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

https://chronic-wasting-disease.blogspot.com/2019/05/assessing-chronic-wasting-disease.html

MONDAY, MAY 20, 2019

APHIS, USDA, Announces the Finalized Chronic Wasting Disease Herd Certification Program Standards Singeltary Submissions

https://chronic-wasting-disease.blogspot.com/2019/05/aphis-usda-announces-finalized-chronic.html

WEDNESDAY, MAY 29, 2019

The European Union Summary Report On Surveillance For The Presence Of Transmissible Spongiform Encephalopathies (TSE): The Situation In 2017

https://transmissiblespongiformencephalopathy.blogspot.com/2019/05/the-european-union-summary-report-on.html

ORIGIN OF CHRONIC WASTING DISEASE TSE PRION?

COLORADO THE ORIGIN OF CHRONIC WASTING DISEASE CWD TSE PRION?

*** Spraker suggested an interesting explanation for the occurrence of CWD. The deer pens at the Foot Hills Campus were built some 30-40 years ago by a Dr. Bob Davis. At or abut that time, allegedly, some scrapie work was conducted at this site. When deer were introduced to the pens they occupied ground that had previously been occupied by sheep.

IN CONFIDENCE, REPORT OF AN UNCONVENTIONAL SLOW VIRUS DISEASE IN ANIMALS IN THE USA 1989

http://webarchive.nationalarchives.gov.uk/20080102193705/http://www.bseinquiry.gov.uk/files/mb/m11b/tab01.pdf

ALSO, one of the most, if not the most top TSE Prion God in Science today is Professor Adriano Aguzzi, and he recently commented on just this, on a cwd post on my facebook page August 20 at 1:44pm, quote;

"it pains me to no end to even contemplate the possibility, but it seems entirely plausible that CWD originated from scientist-made spread of scrapie from sheep to deer in the colorado research facility. If true, a terrible burden for those involved." August 20 at 1:44pm ...end

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

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

snip...

Colorado Chronic Wasting Disease Response Plan December 2018 EMERGENCY!

I. Executive Summary Mule deer, white-tailed deer, elk and moose are highly valued species in North America. Some of Colorado's herds of these species are increasingly becoming infected with chronic wasting disease (CWD). As of July 2018, at least 31 of Colorado's 54 deer herds (57%), 16 of 43 elk herds (37%), and 2 of 9 moose herds (22%) are known to be infected with CWD. Four of Colorado's 5 largest deer herds and 2 of the state's 5 largest elk herds are infected. Deer herds tend to be more heavily infected than elk and moose herds living in the same geographic area. Not only are the number of infected herds increasing, the past 15 years of disease trends generally show an increase in the proportion of infected animals within herds as well. Of most concern, greater than a 10-fold increase in CWD prevalence has been estimated in some mule deer herds since the early 2000s; CWD is now adversely affecting the performance of these herds.

snip...

IMPORTANT PUBLIC HEALTH MESSAGE

Disease in humans resulting from CWD exposure has not been reported to date. However, public health officials cannot determine there is no risk from eating meat from infected animals. Consequently, officials recommend that people avoid exposure to CWD-infected animals. Please see the Colorado Department of Public Health and Environment website (http://www.colorado.gov/pacific/cdphe/priondiseases) for the most current recommendations on carcass testing and other preventive measures.

To minimize exposure to CWD and other diseases of potential concern, Colorado Parks and Wildlife (CPW) and state public health officials advise hunters not to shoot, handle or consume any deer, elk or moose that is acting abnormally or appears to be sick. When fielddressing game, wear rubber gloves and minimize the use of a bone saw to cut through the brain or spinal cord (backbone). Minimize contact with brain or spinal cord tissues, eyes, spleen or lymph nodes. Always wash hands and utensils thoroughly after dressing and processing game meat.

(the map on page 71, cwd marked in red, is shocking...tss)

https://cpw.state.co.us/Documents/RulesRegs/Brochure/BigGame/biggame.pdf

snip...see full report and more updated science on cwd tse prion here;

THURSDAY, OCTOBER 04, 2018

Colorado Parks and Wildlife seeks input on chronic wasting disease plan

http://chronic-wasting-disease.blogspot.com/2018/10/colorado-parks-and-wildlife-seeks-input.html

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

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

2018 - 2019

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

Rapid recontamination of a farm building occurs after attempted prion removal

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

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Abstract

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

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

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

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

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

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

The data illustrates the difficulty in decontaminating farm buildings from scrapie, and demonstrates the likely contribution of farm dust to the recontamination of these environments to levels that are capable of causing disease.

snip...

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

This study clearly demonstrates the difficulty in removing scrapie infectivity from the farm environment. Practical and effective prion decontamination methods are still urgently required for decontamination of scrapie infectivity from farms that have had cases of scrapie and this is particularly relevant for scrapiepositive goatherds, which currently have limited genetic resistance to scrapie within commercial breeds.24 This is very likely to have parallels with control efforts for CWD in cervids.

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

Funding This study was funded by DEFRA within project SE1865.

Competing interests None declared.

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

Saturday, January 5, 2019

Rapid recontamination of a farm building occurs after attempted prion removal

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

THURSDAY, FEBRUARY 28, 2019

BSE infectivity survives burial for five years with only limited spread

https://bovineprp.blogspot.com/2019/02/bse-infectivity-survives-burial-for.html

***> CONGRESSIONAL ABSTRACTS PRION CONFERENCE 2018

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

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

(1) National & OIE Reference Laboratory for Scrapie and CWD, Canadian Food Inspection Agency, Ottawa, Ontario, Canada.

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

Two white-tailed deer were orally inoculated with a brain homogenate prepared from a farmed Canadian white-tailed deer previously diagnosed with CWD. Two reindeer, with no history of exposure to CWD, were housed in the same enclosure as the white-tailed deer, 3.5 months after the deer were orally inoculated. The white-tailed deer developed clinical signs consistent with CWD beginning at 15.2 and 21 months post-inoculation (mpi), and were euthanized at 18.7 and 23.1 mpi, respectively. Confirmatory testing by immunohistochemistry (IHC) and western blot demonstrated widespread aggregates of pathological prion protein (PrPCWD) in the central nervous system and lymphoid tissues of both inoculated white-tailed deer. Both reindeer were subjected to recto-anal mucosal associated lymphoid tissue (RAMALT) biopsy at 20 months post-exposure (mpe) to the white-tailed deer. The biopsy from one reindeer contained PrPCWD confirmed by IHC. This reindeer displayed only subtle clinical evidence of disease prior to a rapid decline in condition requiring euthanasia at 22.5 mpe. Analysis of tissues from this reindeer by IHC revealed widespread PrPCWD deposition, predominantly

in central nervous system and lymphoreticular tissues. Western blot molecular profiles were similar between both orally inoculated white-tailed deer and the CWD positive reindeer. Despite sharing the same enclosure, the other reindeer was RAMALT negative at 20 mpe, and PrPCWD was not detected in brainstem and lymphoid tissues following necropsy at 35 mpe. Sequencing of the prion protein gene from both reindeer revealed differences at several codons, which may have influenced susceptibility to infection.

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

https://prion2018.org/

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

***> Nine of these recurrences occurred 14–21 years after culling, apparently as the result of environmental contamination, but outside entry could not always be absolutely excluded.

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Received 7 March 2006 Accepted 6 August 2006

In 1978, a rigorous programme was implemented to stop the spread of, and subsequently eradicate, sheep scrapie in Iceland. Affected flocks were culled, premises were disinfected and, after 2–3 years, restocked with lambs from scrapie-free areas. Between 1978 and 2004, scrapie recurred on 33 farms. Nine of these recurrences occurred 14–21 years after culling, apparently as the result of environmental contamination, but outside entry could not always be absolutely excluded. Of special interest was one farm with a small, completely self-contained flock where scrapie recurred 18 years after culling, 2 years after some lambs had been housed in an old sheephouse that had never been disinfected. Epidemiological investigation established with near certitude that the disease had not been introduced from the outside and it is concluded that the agent may have persisted in the old sheep-house for at least 16 years.

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

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

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

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

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

SEE;

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

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

Confidential!!!!

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

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

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

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

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

Dr. Paul Brown Scrapie Soil Test BSE Inquiry Document

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

PPo4-4:

Survival and Limited Spread of TSE Infectivity after Burial

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

led to infection in sheep, this method may not always be reliable in predicting the risk of scrapie infection through environmental contamination.

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

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

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

Wednesday, December 16, 2015

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

http://scrapie-usa.blogspot.com/2015/12/objects-in-contact-with-classical.html

Scientific Advisors and Consultants Staff 2001 Advisory Committee TSE PRION Singeltary Submission Freas Monday, January 08,2001 3:03 PM FDA Singeltary submission 2001

Greetings again Dr. Freas and Committee Members,

I wish to submit the following information to the Scientific Advisors and Consultants Staff 2001 Advisory Committee (short version). I understand the reason of having to shorten my submission, but only hope that you add it to a copy of the long version, for members to take and read at their pleasure, (if cost is problem, bill me, address below). So when they realize some time in the near future of the 'real' risks i speak of from human/animal TSEs and blood/surgical products. I cannot explain the 'real' risk of this in 5 or 10 minutes at some meeting, or on 2 or 3 pages, but will attempt here:

fda link is dead in the water;

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

snip...see full text

https://bseusa.blogspot.com/2019/03/scientific-advisors-and-consultants.html

the British disease...NOT, the UKBSEnvCJD only theory was/is bogus $$$

https://histodb11.usz.ch/Images/videos/video-009/video-009.html

*** USA sporadic CJD MAD COW DISEASE HAS HUGE PROBLEM Video

*** sporadic CJD linked to mad cow disease

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

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

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

BSE INQUIRY EVIDENCE

Why did the appearance of new TSEs in animals matter so much? It has always been known that TSEs will transfer across species boundaries. The reason for this was never known until the genetic nature of the prion gene was fully investigated and found to be involved. The gene is found to have well preserved sites and as such there is a similar gene throughout the animal kingdom...and indeed a similar gene is found in insects! It is NOT clear that the precise close nature of the PrP gene structure is essention for low species barriers. Indeed it is probably easier to infect cats with BSE than it is to infect sheep. As such it is not clear that simply because it is possible to infect BSE from cattle into certain monkeys then other apes will necessarily be infectable with the disease. One factor has stood out, however, and that is that BSE, when inoculated into mice would retain its apparent nature of disease strain, and hence when it was inoculated back into cattle, then the same disease was produced. Similarly if the TSE from kudu was inoculated into mice then a similar distribution of disease in the brain of the mouse is seen as if BSE had been inoculated into the mouse. This phenomenon was not true with scrapie, in which the transmission across a species barrier was known to lose many of the scrapie strain phenomena in terms of incubation period or disease histopathology. This also suggested that BSE was not derived from scrapie originally but we probably will never know.

TSE in wild UK deer? The first case of BSE (as we now realise) was in a nyala in London zoo and the further zoo cases in ungulates were simply thought of as being interesting transmissions of scrapie initially. The big problem started to appear with animals in 1993-5 when it became clear that there was an increase in the CJD cases in people that had eaten deer although the statistics involved must have been questionable. The reason for this was that the CJD Surveillance was well funded to look into the diet of people dying of CJD. This effect is not clear with vCJD...if only because the numbers involved are much smaller and hence it is difficult to gain enough statistics. They found that many other foods did not appear to have much association at all but that deer certainly did and as years went by the association actually became clearer. The appearance of vCJD in 1996 made all this much more difficult in that it was suddenly clearer that the cases of sporadic CJD that they had been checking up until then probably had nothing to do with beef...and the study decreased. During the period there was an increasing worry that deer were involved with CJD..

see references:

DEER BRAIN SURVEY

https://web.archive.org/web/20090506025229/http://www.bseinquiry.gov.uk/files/yb/1991/11/20004001.pdf

https://www.facebook.com/groups/1557515941145821/permalink/2299600233604051/?hc_location=ufi

i have not updated my blogspot url with all this data archived, but i will work on it...but until then, i have updated this on the above links with live urls to the actual BSE Inquiry documents...

Subject: Re: DEER SPONGIFORM ENCEPHALOPATHY SURVEY & HOUND STUDY

Date: Fri, 18 Oct 2002 23:12:22 +0100

From: Steve Dealler

Reply-To: Bovine Spongiform Encephalopathy Organization: Netscape Online member

To: BSE-L@ References: <3daf5023 .4080804="" wt.net="">

Dear Terry,

An excellent piece of review as this literature is desparately difficult to get back from Government sites.

What happened with the deer was that an association between deer meat eating and sporadic CJD was found in about 1993. The evidence was not great but did not disappear after several years of asking CJD cases what they had eaten. I think that the work into deer disease largely stopped because it was not helpful to the UK industry...and no specific cases were reported. Well, if you dont look adequately like they are in USA currenly then you wont find any!

Steve Dealler ==========

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

Stephen Dealler is a consultant medical microbiologist deal@airtime.co.uk

BSE Inquiry Steve Dealler

Management In Confidence

BSE: Private Submission of Bovine Brain Dealler

https://web.archive.org/web/20090506043910/http://www.bseinquiry.gov.uk/files/yb/1993/12/08003001.pdf

reports of sheep and calf carcasses dumped...

https://web.archive.org/web/20090505232801/http://www.bseinquiry.gov.uk/files/yb/1993/12/07002001.pdf

re-scrapie to cattle GAH Wells BSE Inquiry

https://web.archive.org/web/20090506043931/http://www.bseinquiry.gov.uk/files/yb/1993/12/09001001.pdf

Dr. Dealler goes rogue to confirm BSE

https://web.archive.org/web/20090506043930/http://www.bseinquiry.gov.uk/files/yb/1993/12/14003001.pdf

https://web.archive.org/web/20090506043930/http://www.bseinquiry.gov.uk/files/yb/1993/12/14003001.pdf

https://web.archive.org/web/20090505231533/http://www.bseinquiry.gov.uk/files/yb/1993/12/17003001.pdf

Confirmation BSE Dealler's mad cow

https://web.archive.org/web/20090505231342/http://www.bseinquiry.gov.uk/files/yb/1993/12/16006001.pdf

BSE vertical transmission

https://web.archive.org/web/20090506043834/http://www.bseinquiry.gov.uk/files/yb/1993/12/13003001.pdf

1993 cjd report finds relationship with eat venison and cjd increases 9 fold, let the cover up begin...tss

https://web.archive.org/web/20090506050244/http://www.bseinquiry.gov.uk/files/yb/1994/07/00001001.pdf

FINDINGS

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

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

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

snip...

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

snip...

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

snip...

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

snip...see full report ;

https://web.archive.org/web/20170126073306/http://collections..europarchive..org/tna/20090505194948/http://bseinquiry.gov.uk/files/yb/1994/08/00004001.pdf

GAME FARM INDUSTRY WANTS TO COVER UP FINDINGS OF INCREASE RISK TO CJD FROM CERVID

BSE INQUIRY

CJD9/10022

October 1994

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

BerksWell Coventry CV7 7BZ

Dear Mr Elmhirst,

CREUTZFELDT-JAKOB DISEASE (CJD) SURVEILLANCE UNIT REPORT

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

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

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

The statistical results reqarding the consumption of venison was put into perspective in the body of the report and was not mentioned at all in the press release. Media attention regarding this report was low key but gave a realistic presentation of the statistical findings of the Unit. This approach to publication was successful in that consumption of venison was highlighted only once by the media ie. in the News at one television proqramme.

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

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

The BSE Inquiry / Statement No 324

Dr James Kirkwood (not scheduled to give oral evidence)

Statement to the BSE Inquiry

James K Kirkwood BVSc PhD FIBiol MRCVS

[This witness has not been asked to give oral evidence in Phase 1 of the Inquiry]

1. I became involved in the field of TSEs through my work as Head of the Veterinary Science Group at the Zoological Society of London's Institute of Zoology. I held this post from November 1984 until June 1996, when I took up my present post at UFAW. During this time, concurrent with the BSE epidemic, cases of scrapie-like spongiform encephalopathies occurred in animals at the Zoological Society of London's collections at Regent's Park and Whipsnade and in other zoos. It was appropriate to investigate the epidemiology of these cases in order to try to determine the possible impact on zoo animals and breeding programmes, and to consider how the disease in zoo animals might be controlled.

2. Throughout the period from 1985 to March 1996, I worked at the Institute of Zoology (IoZ). I was Head of the Veterinary Science Group of the IoZ and Senior Veterinary Officer of the Zoological Society of London (ZSL). I was responsible for the provision of the veterinary service for the ZSL collections.

3. During the period from 1985 to March 1996, scrapie-like spongiform encephalopathies were diagnosed in the following animals which died, or were euthanased, at London Zoo and Whipsnade:

Animal Sex Date of Death Age (mos)

Arabian Oryx Oryx leucoryx F 24.3.89 38

Greater kudu Tragelaphus strepsiceros (Linda) F 18.8.89 30

Greater kudu (Karla) F 13.11.90 19 Greater kudu (Kaz) M 6.6.91 37

Greater kudu (Bambi) M 24.10.91 36

Greater kudu (346/90) M 26.2.92 18

Greater kudu (324/90) F 22.11.92 38

Cheetah Acinonyx jubatus (Michelle) F 22.12.93 91

All these cases were described in papers published in the scientific literature (as cited below).

https://web.archive.org/web/20090506004016/http://www.bseinquiry.gov.uk/files/ws/s324.pdf

EYES, RETINA, SHOULD NOT BE USED IN SCHOOLS, BAB, SOB, MRM,

https://web.archive.org/web/20090506065716/http://www.bseinquiry.gov.uk/files/yb/1995/06/21005001.pdf

BSE, PET FOOD, CRUSHED HEADS

https://web.archive.org/web/20090505225022/http://www.bseinquiry.gov.uk/files/yb/1989/03/17004001.pdf

IN PARTICULAR CRUSHED HEADS

https://web.archive.org/web/20090505233039/http://www.bseinquiry.gov.uk/files/yb/1989/04/14001001.pdf

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

https://web.archive.org/web/20090505233039/http://www.bseinquiry.gov.uk/files/yb/1989/04/14001001.pdf

https://web.archive.org/web/20090506054223/http://www.bseinquiry.gov.uk/files/yb/1989/04/14005001.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.

https://web.archive.org/web/20090505233046/http://www.bseinquiry.gov.uk/files/yb/1989/04/24003001.pdf

BSE IN PETFOOD

1. The Secretary asked on 19 April whether I was content with the advice in para 3 of the record of the meeting on 17 March with the Parliamentary Secretary (Mr Thompson). The simple answer is "not entirely".

2. On occasions, material obtained from slaughterhouses will be derived from sheep affected with scrapie or cattle that may be incubating BSE for use in petfood manufacture. Some of this material must be classified as high risk since it contains brain, spinal cord, spleen or lymphatic glands.

https://web.archive.org/web/20090505233052/http://www.bseinquiry.gov.uk/files/yb/1989/05/03007001.pdf

Meldrum's notes on pet foods and materials used

https://web.archive.org/web/20090505233041/http://www.bseinquiry.gov.uk/files/yb/1989/05/16001001.pdf

https://web.archive.org/web/20090506015917/http://www.bseinquiry.gov.uk/files/yb/1989/05/16002001.pdf

IN CONFIDENCE CJD TO CATS...

It should be noted that under experimental conditions cats succumb to an encephalopathy after intracerebral inoculation of material derived from patients affected with Creutzfeldt-Jakob Disease.

https://web.archive.org/web/20090506055329/http://www.bseinquiry.gov.uk/files/yb/1989/05/18002001.pdf

Confidential BSE and _____

3. I have thought very hard about whether the Branch should carry out a similar exercise with meat and meat products for human foods. On balance I do NOT think we should undertake it, but a final decision has not been taken and you may wish to discuss this further. ...

https://web.archive.org/web/20090505220118/http://www.bseinquiry.gov.uk/files/yb/1989/05/22012001.pdf

1st case natural FSE

NATURAL SPONGIFORM ENCEPHALOPATHY IN A DOMESTIC CAT

1. We have heard from MAFF that a domestic Siamese cat from the Bristol area has had spongiform encephalopathy confirmed. Although there are previous instances of experimental infection in cats, there have been no previous natural infections reported. The assumption must be the cat became infected by scrapie/BSE agent in it's food. ...

https://web.archive.org/web/20090506015733/http://www.bseinquiry.gov.uk/files/yb/1990/05/09002001.pdf

FSE and pharmaceuticals

1. An analysis by MCA Professional staff of the results to the questionnaire sent out to industry to obtain additional data about the use of animal materials of any origin in the manufacture of pharmaceutical products for human use, reveals that material of feline or canine origin is used in only two licensed products. In both instances the material is sourced from outside the U.K. and from areas currently believed to be free from B.S.E.

https://web.archive.org/web/20090506041332/http://www.bseinquiry.gov.uk/files/yb/1990/05/10005001.pdf

CONFIDENTIAL

Confidential cats/dogs and unsatisfactory posture MAFFs failure to assure key research

3. First, I am very uneasy about the relative lack of urgency and interest that MAFF appear to hold for getting the necessary research programme on BSE and related encephalopathies started, and getting it going fast. FOR EXAMPLE, MR BRADLEY of CVL said that there were difficulties in organizing transmission experiments from the brain of the cat which died of an encephalopathy in Bristol. There were arguments going on about who should pay for this work. Should it be MAFF, the Bristol Veterinary School or someone else? Dr. Tyrrell was clearly exasperated.

snip...

11. The Committee were even LESS FORTHCOMING on what their reaction might be if an encephalopathy is found in another species, perhaps in DOGS. Their first reaction was that, as with the cats, the first step could be to investigate whether this was really a new disease, or simply one that had not previously been recognized and to see whether it has any links to BSE, scrapie or other transmissible encephalopathies. Indeed, some members of the Committee seem to regard the whole question of another species as a hypothetical question to be addressed only when it happened. A rather UNSATISFACTORY POSTURE.

12. In advance of your meeting with Dr Tyrrell on Monday morning, I have not voiced my ANXIETIES about the support the Committee is receiving from MAFF to anyone OTHER THAN DR PICKLES. ...

https://web.archive.org/web/20090506040545/http://www.bseinquiry.gov.uk/files/yb/1990/06/14006001.pdf

SPONGIFORM ENCEPHALOPATHY IN A CAPTIVE PUMA

an article in yesterday's Times (attached) which suggested that the puma concerned had never "eaten any part of a cow or sheep which, in the opinion of Government Scientists, could transmit the species to a different species".

3. You explained to me that this was INCORRECT. The position was as set out in the briefing for Prime Minister's questions attached to Mr Taylor's note. The puma had probably been fed low quality beef meat in the form of split carcasses. ...

https://web.archive.org/web/20090506032628/http://www.bseinquiry.gov.uk/files/yb/1992/11/13001001.pdf

Subject: DEER SPONGIFORM ENCEPHALOPATHY SURVEY & HOUND STUDY Date: Thu, 17 Oct 2002 17:04:51 -0700 From: "Terry S. Singeltary Sr." Reply-To: Bovine Spongiform Encephalopathy To: BSE-L

Greetings BSE-L,

is there any other CWD surveys/testing in the UK on their deer? what sort of testing has been done to date on UK/EU deer? any input would be helpful... thank you

DEER SPONGIFORM ENCEPHALOPATHY SURVEY

3. This will be a low key study with no publicity to avoid unnecessary media interest. It will be carried out in two stages ;

(I) A small scale examination of around 30 deer brains to establish the normal histology of the healthy brain; and

(II) A larger scale random examination of 300 or more adult deer brains drawn from both deer farms and parks to establish whether there is any evidence of a cervine spongiform encephalopathy. ...

https://web.archive.org/web/20090506025229/http://www.bseinquiry.gov.uk/files/yb/1991/11/20004001.pdf

Ministry of Agriculture Fisheries and Food Veterinary Investigation Centre West House. Station Road. Thirsk Y07 IPZ Telephone: 0845·522065 Fax: 0845·525224

Your reference

Our reference RJH/ASB

Date 4 November 1992

DEER SPONGIFORM ENCEPHALOPATHY SURVEY

Dear Paul

I have now found time to review the 10 deer- brains collected from Mr Walker farm··via Winchester Via Winchester VIC. In answer to your specific question was there sufficient difference in preservation of brain tissue to warrant the extra effort involved in rapid brain removal on the farm, the answer is definitely "Yes." The original five brains (Winchester ref M487/11) showed varying degrees of autolytic vacuolation affecting both white and grey matter throughout the brain. vacuolation and separation of Purkinje cells and marked perivascular spaces. These artifacts made interpretation of subtle, specific pathological vacuolation more difficult. By contrast the second submission (Winchester reference N736/2) showed excellent preservation of white and grey matter. Any vacuolar Change present could be confidently interpreted as pathological albeit of unknown pathogenesis.

I can only reiterate the comments made by Gerald Wells and myself at the preliminary discussion at Weybridge in Autumn 1991. If the survey's purpose is an accurate histopathological interpretation of brain tissue. the material must be collected in a pristine state. This is particularly valid when looking for ar unrecognised and undefined spongiform encephalopathy in a new species. Deer brains are very large structures which take a lot of fixation and therefore must be handled sympathetically from the start. We have already seen the problems encountered in comparatively smaller hound brains where delayed fixation was a major limitation on interpretation of true pathological change.

The bottom line must be that if a pathologist's expertise is to be used, it is critical to collect artefact free brain material. If the politics or economics do not allow this, then I would suggest that an electron microscopy survey involving detection of scrapie associated fibrils would be much more appropriate.

Best wishes Yours sincerely

R J HIGGINS VIO 92/11.4/2.1

https://web.archive.org/web/20090506032702/http://www.bseinquiry.gov.uk/files/yb/1992/11/04002001.pdf

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

https://web.archive.org/web/20090506031345/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.

https://web.archive.org/web/20090506043913/http://www.bseinquiry.gov.uk/files/yb/1993/12/06001001.pdf

HOUND SURVEY PATHOLOGICAL REPORT (see positive results) and MAD DOGS AND ENGLISHMAN...

ya'll thought i was making this stuff up didn't ya...i don't make this stuff up!

https://web.archive.org/web/20090506035936/http://www.bseinquiry.gov.uk/files/yb/1990/11/28001001.pdf

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

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

second supplementary

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

Why did the appearance of new TSEs in animals matter so much? It has always been known that TSEs will transfer across species boundaries. The reason for this was never known until the genetic nature of the prion gene was fully investigated and found to be involved. The gene is found to have well preserved sites and as such there is a similar gene throughout the animal kingdom...and indeed a similar gene is found in insects! It is NOT clear that the precise close nature of the PrP gene structure is essention for low species barriers. Indeed it is probably easier to infect cats with BSE than it is to infect sheep. As such it is not clear that simply because it is possible to infect BSE from cattle into certain monkeys then other apes will necessarily be infectable with the disease. One factor has stood out, however, and that is that BSE, when inoculated into mice would retain its apparent nature of disease strain, and hence when it was inoculated back into cattle, then the same disease was produced. Similarly if the TSE from kudu was inoculated into mice then a similar distribution of disease in the brain of the mouse is seen as if BSE had been inoculated into the mouse. This phenomenon was not true with scrapie, in which the transmission across a species barrier was known to lose many of the scrapie strain phenomena in terms of incubation period or disease histopathology. This also suggested that BSE was not derived from scrapie originally but we probably will never know.

TSE in wild UK deer? The first case of BSE (as we now realise) was in a nyala in London zoo and the further zoo cases in ungulates were simply thought of as being interesting transmissions of scrapie initially. The big problem started to appear with animals in 1993-5 when it became clear that there was an increase in the CJD cases in people that had eaten deer although the statistics involved must have been questionable. The reason for this was that the CJD Surveillance was well funded to look into the diet of people dying of CJD. This effect is not clear with

vCJD...if only because the numbers involved are much smaller and hence it is difficult to gain enough statistics. They found that many other foods did not appear to have much association at all but that deer certainly did and as years went by the association actually became clearer. The appearance of vCJD in 1996 made all this much more difficult in that it was suddenly clearer that the cases of sporadic CJD that they had been checking up until then probably had nothing to do with beef...and the study decreased. During the period there was an increasing worry that deer were involved with CJD..

see references:

DEER BRAIN SURVEY

https://web.archive.org/web/20090506025229/http://www.bseinquiry.gov.uk/files/yb/1991/11/20004001.pdf

1993 cjd report finds relationship with eat venison and cjd increases 9 fold, let the cover up begin...tss

https://webarchive.nationalarchives.gov.uk/.../00001001.pdf

FINDINGS

*** 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 ;

https://web.archive.org/web/20170126073306/http://collections..europarchive..org/tna/20090505194948/http://bseinquiry.gov.uk/files/yb/1994/08/00004001.pdf

GAME FARM INDUSTRY WANTS TO COVER UP FINDINGS OF INCREASE RISK TO CJD FROM CERVID

BSE INQUIRY

CJD9/10022

October 1994

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

BerksWell Coventry CV7 7BZ

Dear Mr Elmhirst,

CREUTZFELDT-JAKOB DISEASE (CJD) SURVEILLANCE UNIT REPORT

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

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

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

The statistical results reqarding the consumption of venison was put into perspective in the body of the report and was not mentioned at all in the press release. Media attention regarding this report was low key but gave a realistic presentation of the statistical findings of the Unit. This approach to publication was successful in that consumption of venison was highlighted only once by the media ie. in the News at one television proqramme.

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

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

Management In Confidence

BSE: Private Submission of Bovine Brain Dealler

https://web.archive.org/web/20090506043910/http://www.bseinquiry.gov.uk/files/yb/1993/12/08003001.pdf

Subject: Re: DEER SPONGIFORM ENCEPHALOPATHY SURVEY & HOUND STUDY

Date: Fri, 18 Oct 2002 23:12:22 +0100

From: Steve Dealler

Reply-To: Bovine Spongiform Encephalopathy Organization: Netscape Online member

To: BSE-L@ References: <3daf5023 .4080804="" wt.net="">

Dear Terry,

An excellent piece of review as this literature is desparately difficult to get back from Government sites.

What happened with the deer was that an association between deer meat eating and sporadic CJD was found in about 1993. The evidence was not great but did not disappear after several years of asking CJD cases what they had eaten. I think that the work into deer disease largely stopped because it was not helpful to the UK industry...and no specific cases were reported. Well, if you dont look adequately like they are in USA currenly then you wont find any!

Steve Dealler

Stephen Dealler is a consultant medical microbiologist deal@airtime.co.uk

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

*** DEFRA TO SINGELTARY ON HOUND STUDY AND BSE 2001 ***

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 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

new url;

https://web.archive.org/web/20040513023227/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 su

https://caninespongiformencephalopathy.blogspot.com/

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

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

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

From: "Terry S. Singeltary Sr."

Reply-To: Bovine Spongiform Encephalopathy

To: BSE-L@uni-karlsruhe.de

snip...

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

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

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

[host Richard] could you repeat the question?

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

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

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

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

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

[not sure whom speaking] NO

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

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

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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

SATURDAY, MARCH 2, 2019

MAD COW TSE PRION DISEASE AND THE PEER REVIEW PROCESS OF BSe Science $$$

https://bovineprp.blogspot.com/2019/03/mad-cow-tse-prion-disease-and-peer.html

WEDNESDAY, MAY 29, 2019

The European Union Summary Report On Surveillance For The Presence Of Transmissible Spongiform Encephalopathies (TSE): The Situation In 2017

https://transmissiblespongiformencephalopathy.blogspot.com/2019/05/the-european-union-summary-report-on.html

the British disease...NOT, the UKBSEnvCJD only theory was/is bogus $$$

https://histodb11.usz.ch/Images/videos/video-009/video-009.html

*** USA sporadic CJD MAD COW DISEASE HAS HUGE PROBLEM Video

*** sporadic CJD linked to mad cow disease

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

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

Scientific Advisors and Consultants Staff 2001 Advisory Committee TSE PRION Singeltary Submission Freas Monday, January 08,2001 3:03 PM FDA Singeltary submission 2001

Greetings again Dr. Freas and Committee Members,

I wish to submit the following information to the Scientific Advisors and Consultants Staff 2001 Advisory Committee (short version). I understand the reason of having to shorten my submission, but only hope that you add it to a copy of the long version, for members to take and read at their pleasure, (if cost is problem, bill me, address below). So when they realize some time in the near future of the 'real' risks i speak of from human/animal TSEs and blood/surgical products. I cannot explain the 'real' risk of this in 5 or 10 minutes at some meeting, or on 2 or 3 pages, but will attempt here:

fda link is dead in the water;

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

snip...see full text

https://bseusa.blogspot.com/2019/03/scientific-advisors-and-consultants.html

http://creutzfeldt-jakob-disease.blogspot.com/

Harvard BSE Risk Assessment Singeltary et al

Owens, Julie

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

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

To: FSIS RegulationsComments

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

Page 1 of 98

8/3/2006

Greetings FSIS,

I would kindly like to comment on the following ;

[Federal Register: July 12, 2006 (Volume 71, Number 133)] [Notices] [Page 39282-39283] From the Federal Register Online via GPO Access [wais.access.gpo.gov] [DOCID:fr12jy06-35]

http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.230.8886&rep=rep1&type=pdf

Response to Singeltary et al

https://www.fsis.usda.gov/wps/wcm/connect/a4b69cec-0233-49bb-b5fa-d198876bad90/BSE_Risk_Assess_Response_Public_Comments.pdf?MOD=AJPERES

SUNDAY, FEBRUARY 14, 2010 [Docket No. FSIS-2006-0011] FSIS Harvard Risk Assessment of Bovine Spongiform Encephalopathy (BSE) Suppressed peer review of Harvard study October 31, 2002.

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

http://www.fsis.usda.gov/oa/topics/BSE_Peer_Review.pdf

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

*** U.S.A. 50 STATE BSE MAD COW CONFERENCE CALL Jan. 9, 2001 Singeltary et al

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

UESDAY, NOVEMBER 20, 2018

CDC Eyes of CJD patients show evidence of prions concerns for iatrogenic transmission

https://creutzfeldt-jakob-disease.blogspot.com/2018/11/cdc-eyes-of-cjd-patients-show-evidence.html

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, MARCH 12, 2019

Early preclinical detection of prions in the skin of prion-infected animals

https://creutzfeldt-jakob-disease.blogspot.com/2019/03/early-preclinical-detection-of-prions.html

TUESDAY, JANUARY 1, 2019

***> CHILDHOOD EXPOSURE TO CADAVERIC DURA Singeltary et al

https://betaamyloidcjd.blogspot.com/2019/01/childhood-exposure-to-cadaveric-dura.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

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

WEDNESDAY, JUNE 05, 2019

Alberta mom who died shortly after giving birth from CJD TSE Prion donates brain, placenta to research

https://creutzfeldt-jakob-disease.blogspot.com/2019/06/alberta-mom-who-died-shortly-after.html

MONDAY, JUNE 10, 2019

22 Year Old Adena High School graduate Eli Kunkel passes away after battling Creutzfeldt-Jakob Disease

https://creutzfeldt-jakob-disease.blogspot.com/2019/06/22-year-old-adena-high-school-graduate.html

FRIDAY, JANUARY 10, 2014

vpspr, sgss, sffi, TSE, an iatrogenic by-product of gss, ffi, familial type prion disease, what it ???

Greetings Friends, Neighbors, and Colleagues,

vpspr, sgss, sffi, TSE, an iatrogenic by-product of gss, ffi, familial type prion disease, what it ???

Confucius is confused again.

I was just sitting and thinking about why there is no genetic link to some of these TSE prion sGSS, sFFi, and it's really been working on my brain, and then it hit me today.

what if, vpspr, sgss, sffi, TSE prion disease, was a by-product from iatrogenic gss, ffi, familial type prion disease ???

it could explain the cases of no genetic link to the gss, ffi, familial type prion disease, to the family.

sporadic and familial is a red herring, in my opinion, and underestimation is spot on, due to the crude prehistoric diagnostic procedures and criteria and definition of a prion disease.

I say again, what if, iatrogenic, what if, with all these neurological disorders, with a common denominator that is increasingly showing up in the picture, called the prion.

I urge all scientist to come together here, with this as the utmost of importance about all these neurological disease that are increasingly showing up as a prion mechanism, to put on the front burners, the IATROGENIC aspect and the potential of transmission there from, with diseases/disease??? in question.

by definition, could they be a Transmissible Spongiform Encephalopathy TSE prion type disease, and if so, what are the iatrogenic chances of transmission?

this is very important, and should be at the forefront of research, and if proven, could be a monumental breakthrough in science and battle against the spreading of these disease/diseases.

the US National Library of Medicine National Institutes of Health pub-med site, a quick search of the word SPORADIC will give you a hit of 40,747. of those, there are a plethora of disease listed under sporadic. sporadic simply means (UNKNOWN).

http://www.ncbi.nlm.nih.gov/pubmed/?term=sporadic

the US National Library of Medicine National Institutes of Health pub-med site, a quick search of the word FAMILIAL will give you a hit of 921,815. of those, there are a plethora of disease listed under familial.

http://www.ncbi.nlm.nih.gov/pubmed/?term=familial

again, sporadic and familial is a red herring, in my opinion.

also, in my opinion, when you start have disease such as sporadic Fatal Familial Insomnia, (and or sporadic GSS, or the VPSPr type prion disease), and there is NO familial genetic linkage to the family of the diseased, I have serious questions there as to a familial type disease, and thus, being defined as such.

snip...see full text;

Friday, January 10, 2014

vpspr, sgss, sffi, TSE, an iatrogenic by-product of gss, ffi, familial type prion disease, what it ???

http://transmissiblespongiformencephalopathy.blogspot.com/2014/01/vpspr-sgss-sffi-tse-iatrogenic-by.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/

***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

P.9.21

Molecular characterization of BSE in Canada

Jianmin Yang 1 , Sandor Dudas 2 , Catherine Graham 2 , Markus Czub 3 , Tim McAllister 1 , Stefanie Czub 1 1 Agriculture and Agri-Food Canada Research Centre, Canada; 2 National and OIE BSE Reference Laboratory, Canada; 3 University of Calgary, Canada

Background: Three BSE types (classical and two atypical) have been identified on the basis of molecular characteristics of the misfolded protein associated with the disease. To date, each of these three types have been detected in Canadian cattle. Objectives: This study was conducted to further characterize the 16 Canadian BSE cases based on the biochemical properties of there associated PrPres.

Methods: Immuno-reactivity, molecular weight, glycoform profiles and relative proteinase K sensitivity of the PrPres from each of the 16 confirmed Canadian BSE cases was determined using modified Western blot analysis.

Results: Fourteen of the 16 Canadian BSE cases were C type, 1 was H type and 1 was L type. The Canadian H and L-type BSE cases exhibited size shifts and changes in glycosylation similar to other atypical BSE cases. PK digestion under mild and stringent conditions revealed a reduced protease resistance of the atypical cases compared to the C-type cases. N terminal-specific antibodies bound to PrPres from H type but not from C or L type. The C-terminal-specific antibodies resulted in a shift in the glycoform profile and detected a fourth band in the Canadian H-type BSE.

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.

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.neuroprion.org/resources/pdf_docs/conferences/prion2009/prion2009_bookofabstracts.pdf

https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0010638

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

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

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

http://bovineprp.blogspot.com/2018/02/

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

2001

Mad cow disease: Could it be here?

Man's stubborn crusade attracts experts' notice

By Carol Christian, Chron.com / Houston Chronicle Published 5:30 am CDT, Sunday, August 5, 2001

https://www.chron.com/news/houston-texas/article/Mad-cow-disease-Could-it-be-here-2042860.php

https://www.youtube.com/watch?v=zf3lfz9NrT4

Terry S. Singeltary Sr.