From: Leo Millan
To: FWP Wildlife
Subject: [EXTERNAL] CWD and urine based scents
Date: Friday, April 30, 2021 8:51:12 PM

Hello, it is my belief if you allow ANY scents other than fully synthetic scents, there WILL be some real contraband scents that will undoubtedly leak into the market to bypass laws etc. When money is involved people WILL cheat whatever system is in place. Therefore no ifs ands or buts, outlaw ALL real scents and allow only synthetics and STOP this vector of CWD
.
My 2c...
Sincerely,
Leo A Millan

From: d pier
To: FWP Wildlife
Subject: [EXTERNAL] deer scent, B Bear carcass utilization
Date: Tuesday, May 11, 2021 12:06:47 AM

I wish to go on record as supporting a ban on deer scents which do not align with CWD safety measures. It is far too risky to take a chance on introducing further CWD exposure to wildlife via deer urine/scents which do not follow safeguards against disease transmission.
I also want to support the requirement to require utilization of the entire carcass of any legally taken black bear. It is not remotely in keeping with fair chase and ethical standards to shoot a bear merely for the hide.

D. Pier
Columbia Falls, MT

From: Sam Burgeson
To: FWP Wildlife
Subject: [EXTERNAL] Montana Hunting Regulation comments
Date: Thursday, May 13, 2021 12:54:37 PM

RE: Proposed adoption pertaining to the use of deer or elk urine to mask human odor.

We want to formally thank the commissioners and FWP staff that have been working on the language of the proposed rule regarding the use of urine-based scents in Montana. We have reviewed the language and support it. It is in compliance with the Montana Code 87-6-221, and maintains Montana's leadership role in implementing practical safeguards that Montana hunters and retailers will be able to implement.

The Responsible Hunting Scent Association looks forward to working with the Commission and agency in the future, and have encouraged our members and customers to support this rule.

Respectfully,

Sam Burgeson
President – Wildlife Research Center
President – Responsible Hunting Scent Association

From: William Roberts
To: FWP Wildlife
Subject: [EXTERNAL] Urine Based Scents for Hunting
Date: Thursday, May 13, 2021 8:50:59 AM

Hello,

While I don't use urine lures, I support their use. However, I think the use should require tested products over origin location. Tested products are the only way to ensure they truly are CWD free, not origin.

Bill Roberts
Non resident hunter
4411 McGregor Ln, Dripping Springs, TX 78620

585-455-1883

From: ccohan@bresnan.net
To: FWP Wildlife
Subject: [EXTERNAL] Urine cover smell.
Date: Tuesday, May 11, 2021 2:48:33 PM

Please vote against urine cover smells. We don't want to risk more CWD in our state.

Cindy Cohan

From: Terry Singeltary
To: FWP Wildlife
Subject: [EXTERNAL] COMMISSION TO HOLD PUBLIC HEARING ON RULES REGARDING THE USE OF ELK AND DEER URINE AND GLANDULAR SCENTS SINGELTARY COMMENT SUBMISSION
Date: Wednesday, April 21, 2021 3:26:31 PM

COMMISSION TO HOLD PUBLIC HEARING ON RULES REGARDING THE USE OF ELK AND DEER URINE AND GLANDULAR SCENTS SINGELTARY COMMENT SUBMISSION

COMMISSION TO HOLD PUBLIC HEARING ON RULES REGARDING THE USE OF ELK AND DEER URINE AND GLANDULAR SCENTS

Apr 16, 2021 11:45 AM

HELENA – The Fish and Wildlife Commission will hold a public hearing on May 13, at 9 a.m., to consider the proposed adoption and amendments of three new rules pertaining to the use of deer or elk urine to mask human odor. The purpose of the new rules is to prevent the spread of chronic wasting disease, which can be present in the urine and glandular scents of infected deer and elk.

The hearing will be conducted via Zoom. Due to COVID-19, there will be no in-person hearing. For information on participating in the hearing, visit the Fish and Wildlife Commission page on fwp.mt.gov.

The first new rule identifies states and provinces with documented occurrences of chronic wasting disease. These include the following: Arkansas, Colorado, Illinois, Iowa, Kansas, Maryland, Minnesota, Mississippi, Missouri, Montana, Nebraska, New Mexico, New York, North Dakota, Ohio, Oklahoma, Pennsylvania, South Dakota, Tennessee, Texas, Utah, Virginia, West Virginia, Wisconsin and Wyoming; and the following Canadian provinces: Alberta, Quebec and Saskatchewan.

The second new rule defines the process for identifying facilities in the U.S and Canada that produce urine allowed for the purpose of masking human odor. Urine from any facility approved by the Archery Trade Association and Responsible Hunting Scent Association is allowed, so long as the ATA/RHSA requirements continue to meet the standards identified in Montana statute. Products meeting requirements must display a mark indicating ATA/RHSA approval on the product packaging for identification by the consumer.

The third new rule identifies the requirements used by the commission for approval of urine-based and natural glandular scents for the purposes of attracting game animals and game birds. The scents must:

originate from a state or province not listed in the first rule, or originate from a facility that is approved by the commission under the second rule and display the required marks on the product packaging. According to the Montana Administrative Register, artificial scents and RHSA-approved natural glandular scents may be used by hunters to attract game animals, except black bears, by spraying or pouring the scent on the ground or other objects.

Public comment will be accepted during the hearing. Written comment can be submitted to: Wildlife Division, Montana Fish, Wildlife & Parks, P.O. Box 200701, Helena, Montana, 59620-0701; or emailed to fwpwld@mt.gov. Written comment must be received no later than May 14.

https://fwp.mt.gov/homepage/news/2021/april/0416-commission-to-hold-public-hearing-on-rules-regarding-the-use-of-elk-and-deer-urine-and-glandular-scents

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

Title: Successful transmission of the chronic wasting disease (CWD) agent to white-tailed deer by intravenous blood transfusion

Author item MAMMADOVA, NAJIBA - Orise Fellow item CASSMAN, ERIC - Orise Fellow item Greenlee, Justin Submitted to: Research in Veterinary Science Publication Type: Peer Reviewed Journal Publication Acceptance Date: 10/14/2020 Publication Date: 12/20/2020 Citation: Mammadova, N., Cassman, E., Greenlee, J.J. 2020. Successful transmission of the chronic wasting disease (CWD) agent to white-tailed deer by intravenous blood transfusion. Research in Veterinary Science. 133:304-306. https://doi.org/10.1016/j.rvsc.2020.10.009 [doi.org]. DOI: https://doi.org/10.1016/j.rvsc.2020.10.009 [doi.org]

Interpretive Summary: Chronic wasting disease (CWD) is a fatal disease of cervids that causes damaging changes in the brain. The infectious agent is an abnormal protein called a prion that has misfolded from its normal state. Chronic wasting disease may be transmitted from ingestion of prions shed in bodily fluids (e.g. feces, urine, saliva, placenta tissue) of infected animals. Few studies have also reported detection of infectious prions in blood. To determine if CWD-infected blood can transmit prion disease, recipient deer were inoculated intravenously (IV) with blood derived from a CWD-infected white-tailed deer. We found that two out of three animals developed disease. This study complements and supports an earlier finding that CWD can be transmitted to deer by intravenous blood transfusion from white-tailed deer with CWD. This information is useful to wildlife and agricultural officials that are involved in efforts to control the spread of chronic wasting disease.

Technical Abstract: Chronic wasting disease (CWD) is a transmissible spongiform encephalopathy (TSEs) that affects free-ranging and captive cervid species. The infectious agent of CWD may be transmitted from ingestion of prions shed in bodily fluids (e.g. feces, urine, saliva, placenta tissue) of infected animals, contaminated pastures, and/or decomposing carcasses from dead animals. Studies have also demonstrated prion infectivity in whole blood or blood fractions of CWD infected animals. To determine if CWD-infected blood contained sufficient levels of prion infectivity to cause disease, recipient deer were inoculated intravenously (IV) with blood derived from a CWD-infected white-tailed deer. We found that the CWD agent can be successfully transmitted to white-tailed deer by a single intravenous blood transfusion with a mean incubation period of approximately 35 months and an attack rate of 100%. This study complements and supports an earlier finding that CWD can be transmitted to deer by intravenous blood transfusion from white-tailed deer with CWD.

https://www.ars.usda.gov/research/publications/publication/?seqNo115=373622 [ars.usda.gov]

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

Published: 16 January 2019

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

Zerui Wang, Matteo Manca, Aaron Foutz, Manuel V. Camacho, Gregory J. Raymond, Brent Race, Christina D. Orru, Jue Yuan, Pingping Shen, Baiya Li, Yue Lang, Johnny Dang, Alise Adornato, Katie Williams, Nicholas R. Maurer, Pierluigi Gambetti, Bin Xu, Witold Surewicz, Robert B. Petersen, Xiaoping Dong, Brian S. Appleby, Byron Caughey, Li Cui, Qingzhong Kong & Wen-Quan Zou Nature Communicationsvolume 10, Article number: 247 (2019) |

A Publisher Correction to this article was published on 04 February 2019 This article has been updated

Abstract

A definitive pre-mortem diagnosis of prion disease depends on brain biopsy for prion detection currently and no validated alternative preclinical diagnostic tests have been reported to date. To determine the feasibility of using skin for preclinical diagnosis, here we report ultrasensitive serial protein misfolding cyclic amplification (sPMCA) and real-time quaking-induced conversion (RT-QuIC) assays of skin samples from hamsters and humanized transgenic mice (Tg40h) at different time points after intracerebral inoculation with 263K and sCJDMM1 prions, respectively. sPMCA detects skin PrPSc as early as 2 weeks post inoculation (wpi) in hamsters and 4 wpi in Tg40h mice; RT-QuIC assay reveals earliest skin prion-seeding activity at 3 wpi in hamsters and 20 wpi in Tg40h mice. Unlike 263K-inoculated animals, mock-inoculated animals show detectable skin/brain PrPSc only after long cohabitation periods with scrapie-infected animals. Our study provides the proof-of-concept evidence that skin prions could be a biomarker for preclinical diagnosis of prion disease.

snip...

Discussion Several lines of evidence have recently suggested that skin is the place where misfolded proteins often stay, which may play a role in the pathogenesis and early detection of neurodegenerative diseases25. While it has been known for a long time that sheep and goats with scrapie often have skin lesions26, prions had not been detected in skin until prion infectivity was first found in skin of prion-infected greater kudu using an animal-based bioassay27. Subsequently, skin PrPSc was detected directly by western blotting after the enrichment of PrPSc in experimentally or naturally scrapie-infected hamsters and sheep, as well as in a single cadaver with vCJD28,29. We recently observed both prion-seeding activity and prion infectivity in the skin of patients with sCJD and vCJD at the terminal stage of the diseases using RT-QuIC assay and a bioassay humanized Tg mouse-based, respectively13. In the current study, we further demonstrated that skin PrPSc is preclinically detectable not only by RT-QuIC, but also by sPMCA before brain damage occurs in two animal models of prion diseases, 263K-inoculated hamsters and sCJDMM1-inoculated humanized Tg40h mice, with parallel RT-QuIC findings in an independent set of scrapie-infected hamsters done in an independent laboratory.

In terms of the earliest time point at which skin PrPSc becomes detectable in animals infected by the intracerebral inoculation of prions, sPMCA showed detection at 2 wpi for hamsters and 4 wpi for Tg40h mice, while RT-QuIC detection was at 3 wpi for hamsters and 20 wpi for Tg40h (Fig. 8a). These findings indicate that skin PrPSc is detectable at least 5 weeks earlier in scrapie-infected animals before brain pathology is observed. Of the five body areas examined, the ear pinna and back skin were the areas that showed earliest prion-seeding activity (3 wpi), while the thigh skin was the latest (9 wpi). The latter was also confirmed by two sets of hamsters examined in two-independent laboratories in this study. In contrast to the prion-seeding activity found in the skin of infected hamsters at the early stage of infection, the earliest time point showing skin prion-seeding activity was at 20 wpi in humanized Tg mice by RT-QuIC (Fig. 8b). This time point was similar whether recombinant hamster or human PrP substrate was used despite our expectation that human PrP might provide a more sensitive RT-QuIC for human PrPSc based on better sequence homology between the seeds and substrate.

Although the reasons for early and widespread presence of PrPSc in the skin remain unclear, possibilities include the spread of the prion inoculum itself, or endogenously replicating prions, from the brain through the peripheral nerves to the skin within the 2–3 weeks required for the first detection by our ultrasensitive sPMCA and RT-QuIC assays. PrP seeding activity has been detected in the blood in the prion-infected hamsters and deer immediately after peripheral inoculation including oral, nasal, or blood route30. However, no reports have shown that PrPSc is consistently detectable in the blood of prion-infected hamsters within 2 weeks post intracerebral inoculation. Thus, the early spread of PrPSc from the brain-to-the skin in the intracerebrally 263K-inoculated hamsters is likely either not through the blood or, if initially from the blood, requires time-dependent concentration or replication in the skin to become detectable.

It is unclear why, according to RT-QuIC, the back skin more consistently accumulates PrPSc than the other skin areas tested. It may depend on the dermatomes of nerves and their distance from the CNS. Between the back and thigh areas examined, the back dermatome is more proximate to the CNS. Similarly, we found prion-seeding activity much earlier in the ear area than the thigh (3 wpi vs 9 wpi). Analogously, misfolded α-synuclein deposition in Parkinson's disease patients is more frequently detected in proximate (100% in the cervical C7 site) compared to distal (35% in the thoracic T12 region) skin areas by immunofluorescence microscopy31,32,33. In future studies, it would be interesting to determine whether PrPSc in the skin of sCJD has a similar distribution, and whether factors besides dermatome distance from the brain are involved.

Both sPMCA and RT-QuIC assays detected skin PrPSc early in scrapie-infected hamsters. However, sPMCA amplified PrPSc in skin samples from CJD-infected Tg40 mice at 4 wpi, while RT-QuIC assay detected prion-seeding activity only at 20 wpi (Fig. 8). The reason for the difference in Tg40 mice is not clear, but may be due in part to differences between the assays and the prion strains involved. sPMCA is performed in brain homogenates, which provide naturally post-translationally modified (glycosylated and GPI-anchored) PrPC as the substrate, and other potential brain-derived co-factors. RT-QuIC reactions include only unmodified recombinant PrPC as substrate, and no natural co-factors. sPMCA reactions are accelerated by sonication, whereas RT-QuIC reactions are shaken. Also, in successive rounds of sPMCA, the substrate and other brain components are refreshed, but our RT-QuIC reactions were performed in one round, with no refreshment. To exclude the effect of mismatch between seeds and substrates on the sensitivity of RT-QuIC reactions, we tested two recombinant PrP molecules as substrates from two different species including hamster and human and they all showed the similar sensitivity with the same earliest time point at 20 wpi. Finally, 263K scrapie and MM1 sCJD prions undoubtedly differ in conformation, and therefore, perhaps, their interactions with co-factors, various PrPC substrates, and/or skin-derived inhibitors of RT-QuIC reactions. These factors might differentially affect the sensitivity of detection of MM1 sCJD in the skin of Tg40 mice by sPMCA and RT-QuIC. It is also possible that the RT-QuIC assay may become as sensitive as sPMCA for skin prion detection in the Tg40h mice after further optimization of RT-QuIC's experimental conditions.

Our early detection of PrPSc in the skin of sCJD- and scrapie-infected rodents suggests that it may be possible to do the same with the skin of humans who carry PrP mutations associated with genetic prion diseases such as familial CJD, Gerstmann–Sträussler–Scheinker syndrome, or fatal familial insomnia because it is expected that their mutant PrPC spontaneously converts into PrPSc and accumulates later in life. Skin-based RT-QuIC may reveal early prion-seeding activity in PrP mutation-carriers, or people with suspected exposures to prion infections, while they are still asymptomatic. Even for suspected sCJD cases, who are only identified in the symptomatic phase, skin-based RT-QuIC might be useful for monitoring disease progression, defining severity and diversity, and evaluating the treatment efficacy when potential drugs become available.

Although neither clinical signs nor brain PrPSc were observed in control animals cohabitating with 263K-inoculated hamsters within 12 weeks, the mock-inoculated animals that were housed with scrapie-infected animals had amplifiable PrPSc in the brain and skin via amplification techniques. Moreover, in contrast to the skin PrPSc amplified from the 263K-inoculated hamsters as early as 2 wpi, the control animals that co-habitated with infected hamsters were found to have amplified skin PrPSc after cohabitation for 11 weeks. This finding implies that the skin PrPSc detected early in the scrapie-infected hamsters is not the result of environmental contamination; otherwise, the control animals would exhibit skin PrPSc at 2 wpi as well. The finding of skin PrPSc in the cohabitating control animals may be relevant to the environmental transmission of prions observed in natural animal prion diseases, such as scrapie and CWD. Interestingly, prion transmission has been observed in hamsters by contact with prion-contaminated surfaces through rubbing and bedding34, in which cases skin is expected to be involved. The role that skin may plays in the environmental transmission of prions warrants future investigation.

***> Skin PrPSc may derive from urine or fecal prion contamination in addition to possible skin shedding due to scratching or biting each other. Indeed, scrapie infectivity was reported in the urine of prion-infected mice coincident with lymphocytic nephritis during their preclinical and clinical stages of prion infection35,36. It was also observed in their urine in intracerebrally inoculated hamsters even without any apparent inflammation21. In addition, deer with clinical CWD and mild to moderate nephritis were found to have sPMCA-detectable PrPSc and CWD-infectivity in urine22. Using sPMCA, PrPSc was detected in urine of ~80% of the hamsters intraperitoneally inoculated with 263K prions at the symptomatic stage23. Notably, PrPSc was detected in urine, but only at the terminal stage of disease in intracerebrally inoculated hamsters, except for a few days immediately after oral administration24. Similar to the observations by Gonzalez-Romero et al.23, Murayama et al. also found that not all infected hamsters had detectable urine PrPSc even at the terminal stage24. The skin PrPSc detected early in the intracerebrally infected hamsters, but not in the co-habitated-negative controls, at 2 wpi suggests that skin prions may not result from urine at the early stage of infection.

Unlike the situation with urine, it has not been very clear whether PrPSc is present in feces of intracerebrally inoculated hamsters at the early stage of prion infection. High titers of prion infectivity were detected in feces throughout the disease incubation in orally inoculated hamsters while low levels of infectivity were occasionally observed in intracerebrally- or intraperitoneally-inoculated animals18. For instance, no prion infectivity was detected in feces of hamsters within 3 wpi, including at 1, 2, and 22 days post inoculation (dpi), except for 8 dpi when 17% transmission rate was detected in feces18. However, fecal PrPSc was only detected during the clinical stage of disease by sPMCA in hamsters with lower doses of oral inoculum19. Western blotting of fecal extracts showed shedding of PrPSc in the excrement at 24–72 h post inoculation, but not at 0–24 h post inoculation, or at later preclinical or clinical time points19. Consistent with this observation, prion infectivity was not detected in feces of mule deer after oral challenge with CWD prions within the first 12–16 wpi, but feces contained infectivity after 36 wpi through to clinical disease stages at 64–80 wpi20. It is likely that PrPSc is present in feces of infected animals at the late stage of prion infection, which may contaminate the skin of cohabitating control animals.

Although prion contamination of skin by excrement may not be a major concern in human prion diseases, it is an important issue for prion transmission in animals, such as cattle, sheep, goats, and cervids. It is worth noting that the high incidence of scrapie in sheep and goats as well as CWD in cervids is believed to be attributable to contamination of the environment due to high prion shedding. The detection of PrPSc in excretions including saliva, urine, and feces clearly indicates this shedding. Oral ingestion due to the coprophagic behavior of animals has been believed to cause wide horizontal transmission of scrapie and CWD. However, our current finding of skin PrPSc in cohabitating prion-inoculated and PBS-inoculated control animals, as well as the occurrence of brain PrPSc in the PBS-inoculated animals at the late stage suggests that prion contamination of skin may be a potential route of transmission of prion diseases.

In conclusion, our study indicates that skin PrPSc may be a useful biomarker not only for the preclinical diagnosis of prion diseases, but also for monitoring disease progression following infection and treatment. Since the chance that PrPSc can be consistently detected in blood and urine of sCJD patients by sPMCA and RT-QuIC assays has been virtually very low10,11,37, it is possible that detection of PrPSc in the skin, a highly accessible tissue, could be developed for evaluating therapeutic efficiency and drug screening. As mentioned earlier, RT-QuIC analysis of CSF and nasal brushing specimens to date has been used for diagnosis of human prion diseases only at the clinical stage. Moreover, it is much less practical in live animals to collect CSF and nasal brushing specimens. In cervids, at least, there has been more focus on RT-QuIC analyses of RAMALT biopsies and various excreta38. Although these analyses are currently the most accurate tests available for chronic wasting disease in live cervids, they do not yet provide 100% diagnostic sensitivity and specificity38. Thus, it may be helpful to have additional or alternative diagnostic specimens, such as skin or ear pinna punches, for RT-QuIC and sPMCA testing.

https://www.nature.com/articles/s41467-018-08130-9 [nature.com]

179. PrPCWD detection in CWD-infected TgElk mice model using RT-QUIC

Hyun Joo Sohn, Kyung Je Park, In Soon Roh, Hyo Jin Kim, Hae Eun Kang

Foreign animal disease division, Animal and Plant Quarantine Agency, Gimcheon, Gyeongsangbuk-do, Korea

ABSTRACT

Introduction: Chronic wasting disease (CWD) is the only prion disease affecting free-ranging animals, reported in North America, South Korea, and Norway. Unlike in most other prion disease CWD agents are shed in blood, urine, and faeces which most likely contribute to the horizontal transmission between cervid species. The developments of amplification-based seeding assays have been instrumental in the detection of low levels of prions in clinical samples. Using real-time quaking-induced conversion (RT-QUIC), we established an ultrasensitive detection method for PrPCWD in the urine from CWD-infected sequentially sampled transgenic mice overexpressing elk prion protein (TgElk mice). In addtion, RT-QUIC was performed in the kidney and brain of theses mice model to trace abnormal prion.

Materials and Methods: 44 brain and kidney, urine samples from sequentially collected from CWD-infected TgElk mice (TgElk CWD) were stored at −80°C. In brain and kidney, 10%

(w/v) homogenate was prepared in 0.9% sterilized saline. In urine 100 µL of each sample was mixed with 10 µL 2.8% sodium phosphotungustic acid (NaPTA) and incubated for 1hr at 37°C with shaking at 1,350 rpm. Samples were centrifuged for 30 min at 16,100 g. The pellet was resuspended in 10 µL of 0.1% SDS/PBS for 30 min at 55°C. RT-QUIC reactions were set up in 96-well clear bottom optic plates and consisted of 98 µL RT-QUIC buffer [final concentrations of 1XPBS, 1 mM EDTA, 10 µM Thioflavin, 300 mM NaCl buffer and 0.1 mg/ml recombinant Syrian hamster recombinant protein (23–231), and 2 µL of sample. The RT-QUIC assay was performed on a FLUOstar Omega fluorescence plate reader that was preheated to 42°C for 60 h with 90 s shaking at 700 rpm followed by 1 min incubation.

Results: Five randomly selected mice were sequentially culled on every 15 days from 30dpi to 120dpi during CWD infected TgElk mice reached terminal stage. Rough hair coats among clinical signs were showed from 90 dpi. PrPCWD in the brain in TgElk CWD was detectable persistently from early stages (30dpi), and in the kidney PrPCWD was also detectable in clinical and terminal stages (90 dpi and 120dpi). PrPCWD in the urine in TgElk CWD reached the highest levels at 120dpi. NaPTA/RT-QUIC was applied to measure PrPCWD in urine samples collected on every 15 days from 30dpi to 120dpi when CWD infected TgElk mice reached terminal stage. PrPCWD in the urine in TgElk CWD reached the highest levels at 90dpi. PrPCWD was also detectable in late and terminal stages (120dpi).

Conclusions: We demonstrate that CWD prions can be detected by RT-QUIC or NaPTA/RT-QUIC in the brain, kidney and urine of TgElk mice at the early and terminal stages of disease. Based on these data, we suggest that PrPCWD is excreted into only urine until 90 dpi and then slowly accumulated in kidney. Our results can be used in designing future study of CWD pathogenesis in TgElk mice.

References Henderson DM et al., Rapid antemortem detection of CWD prions in deer saliva. PLOS one. 2013:e74377 1–12 [Google Scholar] Nagooka K, Yoshika M, Shimozaki N. et al., Sensitive detection of scrapie prion protein in soil. Biochem Biophys Res Commun. 2010;397:626–630. [Google Scholar]

199. Chronic wasting disease transmission studies to non-human primates and transgenic mice

Brent Race, Katie Williams, Christina D. Orrù, Andrew G. Hughson, Lori Lubke and Bruce Chesebro

National Institute of Allergy and Infectious Diseases, Laboratory of Persistent Viral Diseases, Rocky Mountain Laboratories, Hamilton, MT, USA

CONTACT Brent Race raceb@niaid.nih.gov

ABSTRACT

Introduction: Chronic wasting disease (CWD) is the only prion disease affecting free-ranging animals, reported in North America, South Korea and Norway. Unlike in most other prion disease CWD agents are shed in blood, urine and feces which most likely contribute to the horizontal transmission between cervid species. The developments of amplification-based seeding assays have been instrumental in the detection of low levels of prions in clinical samples. Using real-time quaking-induced conversion (RT-QUIC), we established an ultrasensitive detection method for PrPCWD in the urine from CWD-infected sequentially sampled transgenic mice overexpressing elk prion protein (TgElk mice). In addtion, RT-QUIC was performed in the kidney and brain of theses mice model to trace abnormal prion.

Materials and Methods: 44 brain and kidney, urine samples from sequentially collected from CWD-infected TgElk mice (TgElk CWD) were stored at -80℃. In brain and kidney, 10% (w/v) homogenate was prepared in 0.9% sterilized saline. In urine 100uL of each sample was mixed with 10uL 2.8% sodium phosphotungustic acid (NaPTA) and incubated for 1hr at 37℃ with shaking at 1,350 rpm. Samples were centrifuged for 30min at 16,100g. The pellet was resuspended in 10uL of 0.1% SDS/PBS for 30min at 55℃. RT-QUIC reactions were set up in 96-well clear bottom optic plates and consisted of 98uL RT-QUIC buffer [final concentrations of 1XPBS, 1mM EDTA, 10uM Thioflavin, 300mM NaCl buffer and 0.1mg/ml recombinant Syrian hamster recombinant protein (23-231) and 2uL of sample. The RT-QUIC assay was performed on a FLUOstar Omega fluorescence plate reader that was preheated to 42℃ for 60hr with 90sec shaking at 700rpm followed by 1min incubation.

Results: Five randomly selected mice were sequentially culled on every 15 days from 30dpi to 120dpi during CWD infected TgElk mice reached terminal stage. Rough hair coats among clinical signs were showed from 90 dpi. PrPCWD in the brain in TgElk CWD was detectable persistently from early stages (30dpi), and in the kidney PrPCWD was also detectable in clinical and terminal stages (90 dpi and 120dpi). PrPCWD in the urine in TgElk CWD reached the highest levels at 120dpi. NaPTA/RT-QUIC was applied to measure PrPCWD in urine samples collected on every 15 days from 30dpi to 120dpi when CWD infected TgElk mice reached terminal stage. PrPCWD in the urine in TgElk CWD reached the highest levels at 90dpi. PrPCWD was also detectable in late and terminal stages (120dpi).

Conclusions: We demonstrate that CWD prions can be detected by RT-QUIC or NaPTA/RT-QUIC in the brain, kidney and urine of TgElk mice at the early and terminal stages of disease. Based on these data, we suggest that PrPCWD is excreted into only urine until 90 dpi and then slowly accumulated in kidney. Our results can be used in designing future study of CWD pathogenesis in TgElk mice.

KEYWORDS: Cynomolgus macaques; squirrel monkeys; non-human primate; transgenic mice; CWD; RT-QuIC; prion; cross-species transmission; barrier; chronic wasting disease

References

Henderson DM et al Rapid antemortem detection of CWD prions in deer saliva PLOS one 2013, 397, 626-630. [Google Scholar] Nagooka K, Yoshika M, Shimozaki N. et al. Sensitive detection of scrapie prion protein in soil. Biochem Biophys Res Commun. 2010 e74377 1-12. [Google Scholar]

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

JOURNAL OF VIROLOGY, July 2011, p. 6309–6318 Vol. 85, No. 13 0022-538X/11/$12.00 doi:10.1128/JVI.00425-11

Detection of Chronic Wasting Disease Prions in Salivary, Urinary, and Intestinal Tissues of Deer: Potential Mechanisms of Prion Shedding and Transmission

Nicholas J. Haley,1 Candace K. Mathiason,1 Scott Carver,1 Mark Zabel,1 Glenn C. Telling,2 and Edward A. Hoover1 * Department of Microbiology, Immunology, and Pathology, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, Colorado,1 and Department of Molecular Biology and Genetics, University of Kentucky, Lexington, Kentucky2

Received 2 March 2011/Accepted 12 April 2011

Efficient horizontal transmission is a signature trait of chronic wasting disease (CWD) in cervids. Infectious prions shed into excreta appear to play a key role in this facile transmission, as has been demonstrated by bioassays of cervid and transgenic species and serial protein misfolding cyclic amplification (sPMCA). However, the source(s) of infectious prions in these body fluids has yet to be identified. In the present study, we analyzed tissues proximate to saliva, urine, and fecal production by sPMCA in an attempt to elucidate this unique aspect of CWD pathogenesis. Oropharyngeal, urogenital, and gastrointestinal tissues along with blood and obex from CWD-exposed cervids (comprising 27 animals and >350 individual samples) were analyzed and scored based on the apparent relative CWD burden. PrPCWDgenerating activity was detected in a range of tissues and was highest in the salivary gland, urinary bladder, and distal intestinal tract. In the same assays, blood from the same animals and unseeded normal brain homogenate controls (n 116 of 117) remained negative. The PrP-converting activity in peripheral tissues varied from 1011- to 100 -fold of that found in brain of the same animal. Deer with highest levels of PrPCWD amplification in the brain had higher and more widely disseminated prion amplification in excretory tissues. Interestingly, PrPCWD was not demonstrable in these excretory tissues by conventional Western blotting, suggesting a low prion burden or the presence of protease-sensitive infectious prions destroyed by harsh proteolytic treatments. These findings offer unique insights into the transmission of CWD in particular and prion infection and trafficking overall.

SNIP...

In summary, the present study demonstrates for the first time prion-amplifying activity in organs and tissues associated with prion shedding. The ultimate source and mechanism of release into bodily fluids remain unknown. The elevated and consistent activity found in salivary gland and urinary bladder may suggest an active role in prion excretion. These findings minimally warrant additional, more detailed, and longitudinal studies of the nature and kinetics of excreted prions.

https://jvi.asm.org/content/jvi/85/13/6309.full.pdf [jvi.asm.org]

In animals incubating CWD, abnormal PrP and/or infectivity has been demonstrated in placenta, saliva, faeces and urine which are all likely to contribute to inter-individual transmission but also to the general contamination of the environment (Mathiason et al., 2006; Tamguney et al., 2009a; Haley and Hoover, 2015; Plummer et al., 2017).

Deer urine as a commercial lure used by hunters/photographers could be sourced fromcaptive populations with preclinical cases of infection and be transported via commercial

platforms (Defra, 2016a,b)

https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2019.5863 [efsa.onlinelibrary.wiley.com]

J Gen Virol

. 2017 Jul;98(7):1932-1942. doi: 10.1099/jgv.0.000845. Epub 2017 Jul 15.

Temporal patterns of chronic wasting disease prion excretion in three cervid species

Ian H Plummer 1, Scott D Wright 2 3, Chad J Johnson 4, Joel A Pedersen 4, Michael D Samuel 5

PMID: 28708047 DOI: 10.1099/jgv.0.000845

Abstract

Chronic wasting disease (CWD) is the only naturally occurring transmissible spongiform encephalopathy affecting free-ranging wildlife populations. Transmission of CWD occurs by direct contact or through contaminated environments; however, little is known about the temporal patterns of CWD prion excretion and shedding in wild cervids. We tested the urine and faeces of three species of captive cervids (elk, mule and white-tailed deer) at 6, 12, 18 and 24 months after oral inoculation to evaluate the temporal, species- and genotype-specific factors affecting the excretion of CWD prions. Although none of the animals exhibited clinical signs of CWD during the study, we determined that all three cervid species were excreting CWD prions by 6 months post-inoculation. Faecal samples were consistently positive for CWD prions for all three cervid species (88 %), and were more likely to be positive than urine samples (28 %). Cervids with genotypes encoding for the prion protein (PRNP) that were considered to be more susceptible to CWD were more likely to excrete CWD prions (94 %) than cervids with genotypes considered to be less susceptible (64 %). All cervids with CWD prions in their urine also had positive faeces (n=5), but the converse was not true. Our study is the first to demonstrate CWD prion excretion in urine by asymptomatic elk and mule deer. Our results indicate that the excretion of CWD prions in faeces and, to a lesser extent, urine may provide an important avenue for depositing prions in the environment.

https://pubmed.ncbi.nlm.nih.gov/28708047/ [pubmed.ncbi.nlm.nih.gov]

Longitudinal Detection of Prion Shedding in Saliva and Urine by

Chronic Wasting Disease-Infected Deer by Real-Time QuakingInduced Conversion

Davin M. Henderson, Nathaniel D. Denkers, Clare E. Hoover, Nina Garbino, Candace K. Mathiason, Edward A. Hoover

Prion Research Center, Department of Microbiology, Immunology, and Pathology, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, Colorado, USA

ABSTRACT

Chronic wasting disease (CWD) is an emergent, rapidly spreading prion disease of cervids. Shedding of infectious prions in saliva and urine is thought to be an important factor in CWD transmission. To help to elucidate this issue, we applied an in vitro amplification assay to determine the onset, duration, and magnitude of prion shedding in longitudinally collected saliva and urine samples from CWD-exposed white-tailed deer. We detected prion shedding as early as 3 months after CWD exposure and sustained shedding throughout the disease course. We estimated that the 50% lethal dose (LD50) for cervidized transgenic mice would be contained in 1 ml of infected deer saliva or 10 ml of urine. Given the average course of infection and daily production of these body fluids, an infected deer would shed thousands of prion infectious doses over the course of CWD infection. The direct and indirect environmental impacts of this magnitude of prion shedding on cervid and noncervid species are surely significant.

IMPORTANCE

Chronic wasting disease (CWD) is an emerging and uniformly fatal prion disease affecting free-ranging deer and elk and is now recognized in 22 U.S. states and 2 Canadian provinces. It is unique among prion diseases in that it is transmitted naturally through wild populations. A major hypothesis to explain CWD's florid spread is that prions are shed in excreta and transmitted via direct or indirect environmental contact. Here we use a rapid in vitro assay to show that infectious doses of CWD prions are in fact shed throughout the multiyear disease course in deer. This finding is an important advance in assessing the risks posed by shed CWD prions to animals as well as humans.

https://jvi.asm.org/content/jvi/89/18/9338.full.pdf [jvi.asm.org]

Short Communication

Early detection of chronic wasting disease prions in urine of pre-symptomatic deer by real-time quaking-induced conversion assay

Theodore R. John, Hermann M. Schätzl &Sabine Gilch

Pages 253-258 | Received 07 Feb 2013, Accepted 24 Mar 2013, Published online: 10 Apr 2013

Abstract

Chronic wasting disease (CWD) is a prion disease of captive and free-ranging deer (Odocoileus spp), elk (Cervus elaphus nelsonii) and moose (Alces alces shirasi). Unlike in most other prion diseases, in CWD prions are shed in urine and feces, which most likely contributes to the horizontal transmission within and between cervid species. To date, CWD ante-mortem diagnosis is only possible by immunohistochemical detection of protease resistant prion protein (PrPSc) in tonsil or recto-anal mucosa-associated lymphoid tissue (RAMALT) biopsies, which requires anesthesia of animals. We report on detection of CWD prions in urine collected from pre-symptomatic deer and in fecal extracts by using real time quaking-induced conversion (RT-QuIC). This assay can be useful for non-invasive pre-symptomatic diagnosis and surveillance of CWD.

In summary, we demonstrate that CWD prions can be detected by RT-QuIC in urine of orally infected white-tailed deer and mule deer at a pre-symptomatic stage of the disease.

https://www.tandfonline.com/doi/full/10.4161/pri.24430 [tandfonline.com]

Published: 09 September 2009

Asymptomatic deer excrete infectious prions in faeces

Gültekin Tamgüney, Michael W. Miller, Lisa L. Wolfe, Tracey M. Sirochman, David V. Glidden, Christina Palmer, Azucena Lemus, Stephen J. DeArmond & Stanley B. Prusiner

Abstract

Infectious prion diseases1—scrapie of sheep2 and chronic wasting disease (CWD) of several species in the deer family3,4—are transmitted naturally within affected host populations. Although several possible sources of contagion have been identified in excretions and secretions from symptomatic animals5,6,7,8, the biological importance of these sources in sustaining epidemics remains unclear. Here we show that asymptomatic CWD-infected mule deer (Odocoileus hemionus) excrete CWD prions in their faeces long before they develop clinical signs of prion disease. Intracerebral inoculation of irradiated deer faeces into transgenic mice overexpressing cervid prion protein (PrP) revealed infectivity in 14 of 15 faecal samples collected from five deer at 7–11 months before the onset of neurological disease. Although prion concentrations in deer faeces were considerably lower than in brain tissue from the same deer collected at the end of the disease, the estimated total infectious dose excreted in faeces by an infected deer over the disease course may approximate the total contained in a brain. Prolonged faecal prion excretion by infected deer provides a plausible natural mechanism that might explain the high incidence and efficient horizontal transmission of CWD within deer herds3,4,9, as well as prion transmission among other susceptible cervids.

SNIP...

https://www.nature.com/articles/nature08289 [nature.com]

Urine

Pooled urine from five terminally CWD infected white-tailed deer was inoculated into nine tg mice. Two of the nine mice developed disease consistent with a TSE at 370 and 376 days post inoculation suggesting infectious prions are present in the urine of infected cervids but at a lower infectivity than other bodily fluids such as saliva.

Haley et al., (2009)

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 [web.archive.org]

Detection of CWD Prions in Urine and Saliva of Deer by Transgenic Mouse Bioassay

Nicholas J. Haley,Davis M. Seelig,Mark D. Zabel,Glenn C. Telling,Edward A. Hoover

Published: March 18, 2009 https://doi.org/10.1371/journal.pone.0004848 [doi.org]

Abstract

Chronic wasting disease (CWD) is a prion disease affecting captive and free-ranging cervids (e.g. deer, elk, and moose). The mechanisms of CWD transmission are poorly understood, though bodily fluids are thought to play an important role. Here we report the presence of infectious prions in the urine and saliva of deer with chronic wasting disease (CWD). Prion infectivity was detected by bioassay of concentrated, dialyzed urine and saliva in transgenic mice expressing the cervid PrP gene (Tg[CerPrP] mice). In addition, PrPCWD was detected in pooled and concentrated urine by protein misfolding cyclic amplification (PMCA). The concentration of abnormal prion protein in bodily fluids was very low, as indicated by: undetectable PrPCWD levels by traditional assays (western blot, ELISA) and prolonged incubation periods and incomplete TSE attack rates in inoculated Tg(CerPrP) mice (373±3days in 2 of 9 urine-inoculated mice and 342±109 days in 8 of 9 saliva-inoculated mice). These findings help extend our understanding of CWD prion shedding and transmission and portend the detection of infectious prions in body fluids in other prion infections.

SNIP...

As CWD transmission may model communicability of other TSE's, the transmissible nature of prion diseases may serve as a model for other protein-misfolding diseases. For example, feces, but not urine, from both mice and cheetahs affected with systemic amyloidosis A (SAA) was recently shown to induce SAA in a mouse model, although negative controls were not available in those studies [36]. In light of the prionuria detected in CWD and in models of scrapie, further investigations of infectivity in body fluids in other protein folding diseases may be warranted in the event that prion diseases are not the only infectious proteinopathies.

In summary, we confirm prionsialia in CWD-affected deer by bioassay in cervidized mice and demonstrate for the first time infectious prions in the urine of these cervids by both bioassay and sPMCA. We are currently evaluating urine and saliva from individual animals in hopes of identifying predisposing factors, such as genotypic background and underlying pathology, which may contribute to prionuria and prionsialia. Concurrently, we have begun to explore the tissue origins and protease sensitivity of the infectious prions as well as the onset and duration of shedding in these bodily fluids.

https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0004848 [journals.plos.org]

Texas CWD Symposium: Transmission by Saliva, Feces, Urine & Blood

the other part, these tissues and things in the body then shed or secrete prions which then are the route to other animals into the environment, so in particular, the things, the secretions that are infectious are salvia, feces, blood and urine. so pretty much anything that comes out of a deer is going to be infectious and potential for transmitting disease.

https://www.youtube.com/watch?v=bItnEElzuKo&index=6&list=PL7ZG8MkruQh3wI96XQ8_EymytO828rGxj [youtube.com]

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

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

you cannot cook the TSE prion disease out of meat.

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

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

the TSE prion agent also survives Simulated Wastewater Treatment Processes.

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

you can bury it and it will not go away.

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

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

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

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

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

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

Laboratory of Central Nervous System Studies, National Institute of

Neurological Disorders and Stroke, National Institutes of Health,

Bethesda, MD 20892.

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

PMID: 8006664 [PubMed - indexed for MEDLINE]

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

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 [pnas.org]

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

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

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 [ncbi...nlm.nih.gov]

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 [onlinelibrary.wiley.com]

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

https://bmcvetres.biomedcentral.com/track/pdf/10.1186/1746-6148-9-134.pdf [bmcvetres.biomedcentral.com]

PPo4-4:

Survival and Limited Spread of TSE Infectivity after Burial

PPo4-4:

Survival and Limited Spread of TSE Infectivity after Burial

Karen Fernie, Allister Smith and Robert A. Somerville The Roslin Institute and R(D)SVS; University of Edinburgh; Roslin, Scotland UK

Scrapie and chronic wasting disease probably spread via environmental routes, and there are also concerns about BSE infection remaining in the environment after carcass burial or waste 3disposal. In two demonstration experiments we are determining survival and migration of TSE infectivity when buried for up to five years, as an uncontained point source or within bovine heads. Firstly boluses of TSE infected mouse brain were buried in lysimeters containing either sandy or clay soil. Migration from the boluses is being assessed from soil cores taken over time. With the exception of a very small amount of infectivity found 25 cm from the bolus in sandy soil after 12 months, no other infectivity has been detected up to three years. Secondly, ten bovine heads were spiked with TSE infected mouse brain and buried in the two soil types. Pairs of heads have been exhumed annually and assessed for infectivity within and around them. After one year and after two years, infectivity was detected in most intracranial samples and in some of the soil samples taken from immediately surrounding the heads. The infectivity assays for the samples in and around the heads exhumed at years three and four are underway. These data show that TSE infectivity can survive burial for long periods but migrates slowly. Risk assessments should take into account the likely long survival rate when infected material has been buried.

The authors gratefully acknowledge funding from DEFRA.

PRION CONFERENCE 2010 ABSTRACT REFERENCE

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 [dx.doi.org]

Kevin Christopher Gough, BSc (Hons), PhD1, Claire Alison Baker, BSc (Hons)2, Steve Hawkins, MIBiol3, Hugh Simmons, BVSc, MRCVS, MBA, MA3, Timm Konold, DrMedVet, PhD, MRCVS3 and Ben Charles Maddison, BSc (Hons), PhD2

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.

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Competing interests None declared.

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

https://bvajournals.onlinelibrary.wiley.com/doi/abs/10.1136/vr.105054 [bvajournals.onlinelibrary.wiley.com]

https://insights.ovid.com/veterinary-record/tvre/2019/01/190/rapid-recontamination-farm-building-occurs/19/00008049 [insights.ovid.com]

https://search.proquest.com/openview/4544d5837142a98dd1bc8c1e32e79984/1?pq-origsite=gscholar&cbl=2041027 [search.proquest.com]

https://pubmed.ncbi.nlm.nih.gov/30602491/ [pubmed.ncbi.nlm.nih.gov]

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 [prionprp.blogspot.com]

The effectiveness of on-farm decontamination methods for scrapie - SE1865

Description

Scrapie infectivity persists on farms where infected animals have been removed1. Recently we have demonstrated that it is possible to detect environmental scrapie contamination biochemically using serial Protein Misfolding Cyclic Amplification (sPMCA)2, allowing the monitoring of scrapie infectivity on farm premises. Ongoing Defra study SE1863 has compared pen decontamination regimes on a scrapie-infected farm by both sheep bioassay and sPMCA. For bioassay, scrapie-free genetically susceptible lambs were introduced into pens decontaminated using distinct methodologies, all pens contained scrapie-positive lambs within 1 year. Remarkably this included lambs housed within a pen which had been jet washed/chloros treated, followed by regalvanisation/ replacement of all metalwork and painting of all other surfaces.

We have recently demonstrated using sPMCA, that material collected on swabs from vertical surfaces at heights inaccessible to sheep within a barn on the same scrapie affected farm contained scrapie prions (unpublished observations). We hypothesise that scrapie prions are most likely to have been deposited in these areas by bioaerosol movement. We propose that this bioaerosol movement contributes to scrapie transmission within the barn, and could account for the sheep that became positive within the pen containing re-galvanised/new metalwork and repainted surfaces (project SE1863). It is proposed that a thorough decontamination that would minimise prion-contaminated dust, both within the building and its immediate vicinity, is likely to increase the effectiveness of current methods for decontaminating farm buildings following outbreaks of scrapie. The proposed study builds on our previous data and will thoroughly investigate the potential for farm building scrapie-contamination via the bioaerosol route, a previously unrecognised route for dissemination of scrapie infectivity. This route could lead to the direct infection of healthy animals and/or indirect transmission of disease via contamination of surfaces within animal pens. The proposed study would analyse material collected using air samplers set up within “scrapie-infected” barns and their immediate vicinity, to confirm that prion containing material can be airborne within a scrapie infected farm environment. The study would incorporate a biochemical assessment of different surface decontamination methods, in order to demonstrate the best methodology and then the analysis of air and surface samples after a complete building decontamination to remove sources of dust and surface bound prions from both the building and its immediate vicinity. Analysis of such surface and air samples collected before and after treatment would measure the reduction in levels of infectivity. It is envisaged that the biochemical demonstration of airborne prions and the effective reduction in such prion dissemination would lead to a sheep bioassay experiment that would be conducted after a full farm decontamination. This would fully assess the effectiveness of an optimised scrapie decontamination strategy.

This study will contribute directly to Defra policy on best practice for on-farm decontamination after outbreaks of scrapie; a situation particularly relevant to decontamination after scrapie cases on goat farms where no genetic resistance to scrapie has currently been identified, and where complete decontamination is essential in order to stop recurrence of scrapie after restocking.

Objective

Phase 1

• Determine the presence and relative levels of airborne prions on a scrapie infected farm.

• Evaluate different pen surface decontamination procedures.

Phase 2

• Determine the presence of any airborne prions in a barn after a full decontamination.

Phase 3

• Further assess the efficacy of the decontamination procedure investigated in phase 2 by sheep bioassay.

Time-Scale and Cost

From: 2012

To: 2016

Cost: £326,784

Contractor / Funded Organisations

A D A S UK Ltd (ADAS)

Keywords Animals Fields of Study Animal Health

http://randd.defra.gov.uk/Default.aspx?FromSearch=Y&Location=None&Menu=Menu&Module=More&Paging=10&ProjectID=18479&Publisher=1&SearchText=SE1865&SortOrder=Asc&SortString=ProjectCode#Description [randd.defra.gov.uk]

The Effectiveness of on-Farm Decontamination Methods for Scrapie

Institutions ADAS

Start date 2012

End date 2016

Objective Phase 1

Determine the presence and relative levels of airborne prions on a scrapie infected farm. Evaluate different pen surface decontamination procedures.

Phase 2

Determine the presence of any airborne prions in a barn after a full decontamination.

Phase 3

Further assess the efficacy of the decontamination procedure investigated in phase 2 by sheep bioassay.

More information

Scrapie infectivity persists on farms where infected animals have been removed1. Recently we have demonstrated that it is possible to detect environmental scrapie contamination biochemically using serial Protein Misfolding Cyclic Amplification (sPMCA)2, allowing the monitoring of scrapie infectivity on farm premises. Ongoing Defra study SE1863 has compared pen decontamination regimes on a scrapie-infected farm by both sheep bioassay and sPMCA. For bioassay, scrapie-free genetically susceptible lambs were introduced into pens decontaminated using distinct methodologies, all pens contained scrapie-positive lambs within 1 year. Remarkably this included lambs housed within a pen which had been jet washed/chloros treated, followed by regalvanisation/replacement of all metalwork and painting of all other surfaces.

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building and its immediate vicinity, is likely to increase the effectiveness of current methods for decontaminating farm buildings following outbreaks of scrapie. The proposed study builds on our previous data and will thoroughly investigate the potential for farm building scrapie contamination via the bioaerosol route, a previously unrecognised route for dissemination of scrapie infectivity. This route could lead to the direct infection of healthy animals and/or indirect transmission of disease via contamination of surfaces within animal pens. The proposed study would analyse material collected using air samplers set up within “scrapie-infected” barns and their immediate vicinity, to confirm that prion containing material can be airborne within a scrapie infected farm environment. The study would incorporate a biochemical assessment of different surface decontamination methods, in order to demonstrate the best methodology and then the analysis of air and surface samples after a complete building decontamination to remove sources of dust and surface bound prions from both the building and its immediate vicinity. Analysis of such surface and air samples collected before and after treatment would measure the reduction in levels of infectivity. It is envisaged that the biochemical demonstration of airborne prions and the effective reduction in such prion dissemination would lead to a sheep bioassay experiment that would be conducted after a full farm decontamination. This would fully assess the effectiveness of an optimised scrapie decontamination strategy.

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Funding Source

Department for Environment, Food and Rural Affairs

Project source

View this project

Project number

SE1865

Categories

Foodborne Disease

Policy and Planning

https://fsrio.nal.usda.gov/fsrio/research-projects/effectiveness-farm-decontamination-methods-scrapie [fsrio.nal.usda.gov]

Circulation of prions within dust on a scrapie affected farm

Kevin C Gough1 , Claire A Baker2 , Hugh A Simmons3 , Steve A Hawkins3 and Ben C Maddison2*

Abstract

Prion diseases are fatal neurological disorders that affect humans and animals. Scrapie of sheep/goats and Chronic Wasting Disease (CWD) of deer/elk are contagious prion diseases where environmental reservoirs have a direct link to the transmission of disease. Using protein misfolding cyclic amplification we demonstrate that scrapie PrPSc can be detected within circulating dusts that are present on a farm that is naturally contaminated with sheep scrapie. The presence of infectious scrapie within airborne dusts may represent a possible route of infection and illustrates the difficulties that may be associated with the effective decontamination of such scrapie affected premises.

snip...

Discussion We present biochemical data illustrating the airborne movement of scrapie containing material within a contaminated farm environment. We were able to detect scrapie PrPSc within extracts from dusts collected over a 70 day period, in the absence of any sheep activity. We were also able to detect scrapie PrPSc within dusts collected within pasture at 30 m but not at 60 m distance away from the scrapie contaminated buildings, suggesting that the chance of contamination of pasture by scrapie contaminated dusts decreases with distance from contaminated farm buildings. PrPSc amplification by sPMCA has been shown to correlate with infectivity and amplified products have been shown to be infectious [14,15]. These experiments illustrate the potential for low dose scrapie infectivity to be present within such samples. We estimate low ng levels of scrapie positive brain equivalent were deposited per m2 over 70 days, in a barn previously occupied by sheep affected with scrapie. This movement of dusts and the accumulation of low levels of scrapie infectivity within this environment may in part explain previous observations where despite stringent pen decontamination regimens healthy lambs still became scrapie infected after apparent exposure from their environment alone [16]. The presence of sPMCA seeding activity and by inference, infectious prions within dusts, and their potential for airborne dissemination is highly novel and may have implications for the spread of scrapie within infected premises. The low level circulation and accumulation of scrapie prion containing dust material within the farm environment will likely impede the efficient decontamination of such scrapie contaminated buildings unless all possible reservoirs of dust are removed. Scrapie containing dusts could possibly infect animals during feeding and drinking, and respiratory and conjunctival routes may also be involved. It has been demonstrated that scrapie can be efficiently transmitted via the nasal route in sheep [17], as is also the case for CWD in both murine models and in white tailed deer [18-20].

The sources of dust borne prions are unknown but it seems reasonable to assume that faecal, urine, skin, parturient material and saliva-derived prions may contribute to this mobile environmental reservoir of infectivity. This work highlights a possible transmission route for scrapie within the farm environment, and this is likely to be paralleled in CWD which shows strong similarities with scrapie in terms of prion dissemination and disease transmission. The data indicate that the presence of scrapie prions in dust is likely to make the control of these diseases a considerable challenge.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4397813/ [ncbi.nlm.nih.gov]

Research Project: TRANSMISSION, DIFFERENTIATION, AND PATHOBIOLOGY OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES Location: Virus and Prion Research

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

Author

item Greenlee, Justin item Moore, S - Orise Fellow item Smith, Jodi - Iowa State University item Kunkle, Robert item West Greenlee, M - Iowa State University Submitted to: American College of Veterinary Pathologists Meeting Publication Type: Abstract Only Publication Acceptance Date: 8/12/2015 Publication Date: N/A Citation: N/A

Interpretive Summary:

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

https://www.ars.usda.gov/research/publications/publication/?seqNo115=317901 [ars.usda.gov]

THURSDAY, FEBRUARY 28, 2019

BSE infectivity survives burial for five years with only limited spread

https://link.springer.com/content/pdf/10.1007%2Fs00705-019-04154-8.pdf [link.springer.com]

***> 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.

SOURCE REFERENCE 2018 PRION CONFERENCE ABSTRACT

Research Project: TRANSMISSION, DIFFERENTIATION, AND PATHOBIOLOGY OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES Location: Virus and Prion Research

Title: Horizontal transmission of chronic wasting disease in reindeer

Author

item MOORE, SARAH - ORISE FELLOW item KUNKLE, ROBERT item WEST GREENLEE, MARY - IOWA STATE UNIVERSITY item Nicholson, Eric item RICHT, JUERGEN item HAMIR, AMIRALI item WATERS, WADE item Greenlee, Justin

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Interpretive Summary: Chronic wasting disease (CWD) is a fatal neurodegenerative disease that occurs in farmed and wild cervids (deer and elk) of North America and was recently diagnosed in a single free-ranging reindeer (Rangifer tarandus tarandus) in Norway. CWD is a transmissible spongiform encephalopathy (TSE) that is caused by infectious proteins called prions that are resistant to various methods of decontamination and environmental degradation. Little is known about the susceptibility of or potential for transmission amongst reindeer. In this experiment, we tested the susceptibility of reindeer to CWD from various sources (elk, mule deer, or white-tailed deer) after intracranial inoculation and tested the potential for infected reindeer to transmit to non-inoculated animals by co-housing or housing in adjacent pens. Reindeer were susceptible to CWD from elk, mule deer, or white-tailed deer sources after experimental inoculation. Most importantly, non-inoculated reindeer that were co-housed with infected reindeer or housed in pens adjacent to infected reindeer but without the potential for nose-to-nose contact also developed evidence of CWD infection. This is a major new finding that may have a great impact on the recently diagnosed case of CWD in the only remaining free-ranging reindeer population in Europe as our findings imply that horizontal transmission to other reindeer within that herd has already occurred. Further, this information will help regulatory and wildlife officials developing plans to reduce or eliminate CWD and cervid farmers that want to ensure that their herd remains CWD-free, but were previously unsure of the potential for reindeer to transmit CWD.

Technical Abstract: Chronic wasting disease (CWD) is a naturally-occurring, fatal prion disease of cervids. Reindeer (Rangifer tarandus tarandus) are susceptible to CWD following oral challenge, and CWD was recently reported in a free-ranging reindeer of Norway. Potential contact between CWD-affected cervids and Rangifer species that are free-ranging or co-housed on farms presents a potential risk of CWD transmission. The aims of this study were to 1) investigate the transmission of CWD from white-tailed deer (Odocoileus virginianus; CWDwtd), mule deer (Odocoileus hemionus; CWDmd), or elk (Cervus elaphus nelsoni; CWDelk) to reindeer via the intracranial route, and 2) to assess for direct and indirect horizontal transmission to non-inoculated sentinels. Three groups of 5 reindeer fawns were challenged intracranially with CWDwtd, CWDmd, or CWDelk. Two years after challenge of inoculated reindeer, non-inoculated negative control reindeer were introduced into the same pen as the CWDwtd inoculated reindeer (direct contact; n=4) or into a pen adjacent to the CWDmd inoculated reindeer (indirect contact; n=2). Experimentally inoculated reindeer were allowed to develop clinical disease. At death/euthanasia a complete necropsy examination was performed, including immunohistochemical testing of tissues for disease-associated CWD prion protein (PrPcwd). Intracranially challenged reindeer developed clinical disease from 21 months post-inoculation (months PI). PrPcwd was detected in 5 out of 6 sentinel reindeer although only 2 out of 6 developed clinical disease during the study period (< 57 months PI). We have shown that reindeer are susceptible to CWD from various cervid sources and can transmit CWD to naïve reindeer both directly and indirectly.

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

***> 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.

JOURNAL OF GENERAL VIROLOGY Volume 87, Issue 12

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

Gudmundur Georgsson1, Sigurdur Sigurdarson2, Paul Brown3

First Published: 01 December 2006 https://doi.org/10.1099/vir.0.82011-0 [doi.org] ABSTRACT 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 sheep-house 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.

Gudmundur Georgsson,1 Sigurdur Sigurdarson2 and Paul Brown3

Correspondence

Gudmundur Georgsson ggeorgs@hi.is

1 Institute for Experimental Pathology, University of Iceland, Keldur v/vesturlandsveg, IS-112 Reykjavı´k, Iceland

2 Laboratory of the Chief Veterinary Officer, Keldur, Iceland

3 Bethesda, Maryland, USA

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

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

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

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

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

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 [scrapie-usa.blogspot.com]

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 [europa.eu.int]

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 [web.archive.org]

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.

source reference Prion Conference 2015 abstract book

Grass Plants Bind, Retain, Uptake, and Transport Infectious Prions

Sandra Pritzkow,1 Rodrigo Morales,1 Fabio Moda,1,3 Uffaf Khan,1 Glenn C. Telling,2 Edward Hoover,2 and Claudio Soto1, * 1Mitchell Center for Alzheimer’s Disease and Related Brain Disorders, Department of Neurology, University of Texas Medical School at Houston, 6431 Fannin Street, Houston, TX 77030, USA

2Prion Research Center, Department of Microbiology, Immunology, and Pathology, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, CO 80523, USA

3Present address: IRCCS Foundation Carlo Besta Neurological Institute, 20133 Milan, Italy *Correspondence: claudio.soto@uth.tmc.edu http://dx.doi.org/10.1016/j.celrep.2015.04.036 [dx.doi.org]

SUMMARY

Prions are the protein-based infectious agents responsible for prion diseases. Environmental prion contamination has been implicated in disease transmission. Here, we analyzed the binding and retention of infectious prion protein (PrPSc) to plants. Small quantities of PrPSc contained in diluted brain homogenate or in excretory materials (urine and feces) can bind to wheat grass roots and

leaves. Wild-type hamsters were efficiently infected by ingestion of prion-contaminated plants. The prion-plant interaction occurs with prions from diverse origins, including chronic wasting disease. Furthermore, leaves contaminated by spraying with a prion-containing preparation retained PrPSc for several weeks in the living plant. Finally, plants can uptake prions from contaminated soil and transport them to aerial parts of the plant (stem and leaves). These findings demonstrate that plants can efficiently bind infectious prions and act as carriers of infectivity, suggesting a possible role of environmental prion contamination in the horizontal transmission of the disease.

INTRODUCTION

snip...

DISCUSSION

This study shows that plants can efficiently bind prions contained in brain extracts from diverse prion infected animals, including CWD-affected cervids. PrPSc attached to leaves and roots from wheat grass plants remains capable of seeding prion replication in vitro. Surprisingly, the small quantity of PrPSc naturally excreted in urine and feces from sick hamster or cervids was enough to efficiently contaminate plant tissue. Indeed, our results suggest that the majority of excreted PrPSc is efficiently captured by plants' leaves and roots. Moreover, leaves can be contaminated by spraying them with a prion-containing extract, and PrPSc remains detectable in living plants for as long as the study was performed (several weeks). Remarkably, prion contaminated plants transmit prion disease to animals upon ingestion, producing a 100% attack rate and incubation periods not substantially longer than direct oral administration of sick brain homogenates.

Finally, an unexpected but exciting result was that plants were able to uptake prions from contaminated soil and transport them to aerial parts of the plant tissue. Although it may seem farfetched that plants can uptake proteins from the soil and transport it to the parts above the ground, there are already published reports of this phenomenon (McLaren et al., 1960; Jensen and McLaren, 1960;Paungfoo-Lonhienne et al., 2008). The high resistance of prions to degradation and their ability to efficiently cross biological barriers may play a role in this process. The mechanism by which plants bind, retain, uptake, and transport prions is unknown. We are currently studying the way in which prions interact with plants using purified, radioactively labeled PrPSc to determine specificity of the interaction, association constant, reversibility, saturation, movement, etc.

Epidemiological studies have shown numerous instances of scrapie or CWD recurrence upon reintroduction of animals on pastures previously exposed to prion-infected animals. Indeed, reappearance of scrapie has been documented following fallow periods of up to 16 years (Georgsson et al., 2006), and pastures were shown to retain infectious CWD prions for at least 2 years after exposure (Miller et al., 2004). It is likely that the environmentally mediated transmission of prion diseases depends upon the interaction of prions with diverse elements, including soil, water, environmental surfaces, various invertebrate animals, and plants.

However, since plants are such an important component of the environment and also a major source of food for many animal species, including humans, our results may have far-reaching implications for animal and human health. Currently, the perception of the riskfor animal-to-human prion transmission has beenmostly limited to consumption or exposure to contaminated meat; our results indicate that plants might also be an important vector of transmission that needs to be considered in risk assessment.

https://www.cell.com/cell-reports/pdf/S2211-1247%2815%2900437-4.pdf [cell.com]

RIGINAL RESEARCH ARTICLE

Front. Vet. Sci., 14 September 2015 | https://doi.org/10.3389/fvets.2015.00032 [doi.org]

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

imageTimm Konold1*, imageStephen A. C. Hawkins2, imageLisa C. Thurston3, imageBen C. Maddison4, imageKevin C. Gough5, imageAnthony Duarte1 and imageHugh A. Simmons1

1Animal Sciences Unit, Animal and Plant Health Agency Weybridge, Addlestone, UK

2Pathology Department, Animal and Plant Health Agency Weybridge, Addlestone, UK

3Surveillance and Laboratory Services, Animal and Plant Health Agency Penrith, Penrith, UK

4ADAS UK, School of Veterinary Medicine and Science, University of Nottingham, Sutton Bonington, UK

5School of Veterinary Medicine and Science, University of Nottingham, Sutton Bonington, UK

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

snip...

Discussion

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

WEDNESDAY, MARCH 13, 2019

CWD, TSE, PRION, MATERNAL mother to offspring, testes, epididymis, seminal fluid, and blood

Subject: Prion 2019 Conference

See full Prion 2019 Conference Abstracts

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

Transmissible Spongiform Encephalopathies in exotic species

In exotic species, the last one was in 2007.

SPECIES No. DATES AFFECTED

Ankole cow 2 1991, 95

Bison 1 1996

Cheetah 5 1992 – 98

Eland 6 1989 – 95

Gemsbok 1 1987

Kudu 6 1989 – 92

Asian Leopard Cat1 1 2005

Lion 5 1998 - 2007

Nyala 1 1986

Ocelot 3 1994 – 99

Oryx 2 1989, 92

Puma 3 1992 – 95

Tiger 3 1995 – 99

Data valid to 30 September 2019

1Felis (Prionailurus) bengalensis.

https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/840096/pub-tse-stats-exotic.pdf [assets.publishing.service.gov.uk]

ZOO ANIMALS AND TSE PRION DISEASE

The 82 zoo animals with BSE:

Id TSE Genus Species Subsp Birth Origin Death Place of Death

654 x Microcebus murinus - 1997 U.Montpellier 1998 U.Montpellier

656 x Microcebus murinus - 1997 U.Montpellier 1998 U.Montpellier

481 + Eulemur fulvus mayottensis 1974 Madagascar 1992 Montpellier zoo

474 + Eulemur fulvus mayottensis 1974 Madagascar 1990 Montpellier zoo

584 - Eulemur fulvus mayottensis 1984 Montpellier 1991 Montpellier zoo

455 + Eulemur fulvus mayottensis 1983 Montpellier 1989 Montpellier zoo

- + Eulemur fulvus mayottensis 1988 Montpellier 1992 Montpellier zoo

- + Eulemur fulvus mayottensis 1995 Montpellier 1996 Montpellier zoo

- + Eulemur fulvus albifrons 1988 Paris 1992 Montpellier zoo

- + Eulemur fulvus albifrons 1988 Paris 1990 Montpellier zoo

- + Eulemur fulvus albifrons 1988 Paris 1992 Montpellier zoo

456 + Eulemur fulvus albifrons 1988 Paris 1990 Montpellier zoo

586 + Eulemur mongoz - 1979 Madagascar 1998 Montpellier zoo

- p Eulemur mongoz - 1989 Mulhouse 1991 Montpellier zoo

- p Eulemur mongoz - 1989 Mulhouse 1990 Montpellier zoo

- p Eulemur macaco - 1986 Montpellier 1996 Montpellier zoo

- p Lemur catta - 1976 Montpellier 1994 Montpellier zoo

- p Varecia variegata variegata 1985 Mulhouse 1990 Montpellier zoo

- p Varecia variegata variegata 1993 xxx 1994 Montpellier zoo

455 + Macaca mulatta - 1986 Ravensden UK 1992 Montpellier zoo

- p Macaca mulatta - 1986 Ravensden UK 1993 Montpellier zoo

- p Macaca mulatta - 1988 Ravensden UK 1991 Montpellier zoo

- p Saimiri sciureus - 1987 Frejus France 1990 Frejus zoo

700 pc eulemur hybrid - - Besancon zoo 1998 Besancon zoo

701 pc eulemur hybrid - - Besancon zoo 1998 Besancon zoo

702 pc eulemur hybrid - - Besancon zoo 1998 Besancon zoo

703 pc eulemur hybrid - - Besancon zoo 1998 Besancon zoo

704 pc eulemur hybrid - - Besancon zoo 1998 Besancon zoo

705 pc eulemur hybrid - - Besancon zoo 1998 Besancon zoo

706 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

707 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

708 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

709 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

710 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

711 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

712 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

713 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

714 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

715 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

716 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

717 pc eulemur hybrid - - Strasbourg zoo 1998 Strasbourg zoo

x p genus species - - Lille zoo 1996 Lille zoo

y p genus species - - Lille zoo 1996 Lille zoo

z p genus species - - Lille zoo 1996 Lille zoo

1 + Actinonyx jubatus cheetah 1986 Marwell zoo 1991 Pearle Coast AU

Duke + Actinonyx jubatus cheetah 1984 Marwell zoo 1992 Colchester zoo? UK

Saki + Actinonyx jubatus cheetah 1986 Marwell zoo 1993 unknown UK

Mich + Actinonyx jubatus cheetah 1986 Whipsnade 1993 Whipsnade UK

Fr1 + Actinonyx jubatus cheetah 1987 Whipsnade 1997 Safari de Peaugres FR

Fr2 + Actinonyx jubatus cheetah 1991 Marwell zoo 1997 Safari de Peaugres Fr

xx + Actinonyx jubatus cheetah 19xx xxx zoo 199x Fota zoo IR

yy + Actinonyx jubatus cheetah 19xx yyy zoo 1996+ yyyy zoo UK

zz + Actinonyx jubatus cheetah 19xx zzz zoo 1996+ yyyy zoo UK

aaa + Felis concolor puma 1986 Chester zoo 1991 Chester zoo UK

yy + Felis concolor puma 1980 yyy zoo 1995 yyyy zoo UK

zz + Felis concolor puma 1978 zzz zoo 1995 zzzz zoo UK

xxx + Felis pardalis ocelot 1987 xxx 1994 Chester zoo UK

zzz + Felis pardalis ocelot 1980 zzz 1995 zzzz zoo UK

85 + Felis catus cat 1990+ various 1999+ various UK LI NO

19 + Canis familia. dog 1992+ various 1999+ various UK

Fota + Panthera tigris tiger 1981 xxx zoo 1995 xxxx zoo UK

yy + Panthera tigris tiger 1983 yyy zoo 1998 yyyy zoo UK

Lump + Panthera leo lion 1986 Woburn SP 1998 Edinburgh zoo UK [since 1994]

1 + Taurotragus oryx eland 1987 Port Lympne 1989 Port Lympne zoo UK

Moll + Taurotragus oryx eland 1989 xx UK 1991 not Port Lympne UK

Nedd + Taurotragus oryx eland 1989 xx UK 1991 not Port Lympne UK

Elec + Taurotragus oryx eland 1990 xx UK 1992 not Port Lympne Uk

Daph p Taurotragus oryx eland 1988 xx UK 1990 not Port Lympne UK

zzz + Taurotragus oryx eland 1991 zz UK 1994 zzz UK

yyy + Taurotragus oryx eland 1993 yy UK 1995 yyy UK

Fran p Tragelaphus strepsi. kudu 1985 London zoo 1987 London zoo UK

Lind + Tragelaphus strepsi. kudu 1987 London zoo 1989 London zoo UK

Karl + Tragelaphus strepsi. kudu 1988 London zoo 1990 London zoo UK

Kaz + Tragelaphus strepsi. kudu 1988 London zoo 1991 London zoo UK

Bamb pc Tragelaphus strepsi. kudu 1988 London zoo 1991 London zoo UK

Step - Tragelaphus strepsi. kudu 1984 London zoo 1991 London zoo UK

346 pc Tragelaphus strepsi. kudu 1990 London zoo 1992 London zoo UK

324 + Tragelaphus strepsi. kudu 1989 Marwell zoo 1992 London zoo UK

xxx + Tragelaphus angasi nyala 1983 Marwell zoo 1986 Marwell zoo UK

yy + Oryx gazella gemsbok 1983 Marwell zoo 1986 Marwell zoo UK

zz + Oryx gazella gemsbok 1994+ zzz zoo 1996+ zzzz zoo UK

xx + Oryx dammah scim oryx 1990 xxxx zoo 1993 Chester zoo UK

yy + Oryx leucoryx arab oryx 1986 Zurich zoo 1991 London zoo UK

yy + Bos taurus ankole cow 1987 yyy zoo 1995 yyyy zoo UK

zz + Bos taurus ankole cow 1986 zzz zoo 1991 zzzz zoo UK

xx + Bison bison Eu bison 1989 xxx zoo 1996 xxxx zoo UK

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

http://www.mad-cow.org/99feb_cwd_special.html#fff [mad-cow.org]

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

http://www.mad-cow.org/00/aug00_last_news.html#fff [mad-cow.org]

http://www.pnas.org/content/96/7/4046.full [pnas.org];

THURSDAY, DECEMBER 19, 2019

TSE surveillance statistics exotic species and domestic cats Update December 2019

https://transmissiblespongiformencephalopathy.blogspot.com/2019/12/tse-surveillance-statistics-exotic.html [transmissiblespongiformencephalopathy.blogspot.com]

172. Establishment of PrP_{CWD} extraction and detection methods in the farm soil

Kyung Je Park, Hoo Chang Park, In Soon Roh, Hyo Jin Kim, Hae-Eun Kang and Hyun Joo Sohn

Foreign Animal Disease Division, Animal and Plant Quarantine Agency, Gimcheon, Gyeongsangbuk-do, Korea

ABSTRACT

Introduction: Transmissible spongiform encephalopathy (TSE) is a fatal neurodegenerative disorder, which is so-called as prion diseases due to the causative agents (PrP_{Sc}). TSEs are believed to be due to the template-directed accumulation of disease-associated prion protein, generally designated PrP_{Sc}. Chronic wasting disease (CWD) is the prion disease that is known spread horizontally. CWD has confirmed last in Republic of Korea in 2016 since first outbreak of CWD in 2001. The environmental reservoirs mediate the transmission of this disease. The significant levels of infectivity have been detected in the saliva, urine, and faeces of TSE-infected animals. Soil can serve as a stable reservoir for infectious prion proteins. We found that PrP_{CWD} can be extracted and detected in CWD contaminated soil which has kept at room temperature until 4 years after 0.001 ~ 1% CWD exposure and natural CWD-affected farm soil through PBS washing and sPMCAb.

Materials and Methods: Procedure of serial PMCAb. CWD contaminated soil which has kept at room temperature (RT) for 1 ~ 4 year after 0.001%~1% CWD brain homogenates exposure for 4 months collected 0.14 g. The soil was collected by the same method once of year until 4 year after stop CWD exposure. We had conducted the two steps. There are two kinds of 10 times washing step and one amplification step. The washing step was detached PrP_{Sc} from contaminated soil by strong vortex with maximum rpm. We harvest supernatant every time by 10 times. As the other washing step, the Washed soil was made by washing 10 times soil using slow rotator and then harvest resuspended PBS for removing large impurity material. Last step was prion amplification step for detection of PrP_{CWD} in soil supernatant and the washed soil by sPMCAb. Normal brain homogenate (NBH) was prepared by homogenization of brains with glass dounce in 9 volumes of cold PBS with TritonX-100, 5 mM EDTA, 150 mM NaCl and 0.05% Digitonin (sigma) plus Complete mini protease inhibitors (Roche) to a final concentration of 5%(w/v) NBHs were centrifuged at 2000 g for 1 min, and supernatant removed and frozen at −70 C for use. CWD consisted of brain from natural case in Korea and was prepared as 10%(w/v) homogenate. Positive sample was diluted to a final dilution 1:1000 in NBH, with serial 3:7 dilutions in NBH. Sonication was performed with a Misonix 4000 sonicator with amplitude set to level 70, generating an average output of 160W with two teflon beads during each cycle. One round consisted of 56 cycles of 30 s of sonication followed 9 min 30 s of 37°C incubation. **Western Blotting (WB) for PrP_{Sc} detection**. The samples (20 µL) after each round of amplification were mixed with proteinase K (2 mg/ml) and incubated 37°C for 1 h. Samples were separated by SDS-PAGE and transferred onto PVDF membrane. After blocking, the membrane was incubated for 1 h with 1st antibody S1 anti rabbit serum (APQA, 1:3000) and developed with enhanced chemiluminescence detection system.

Results: We excluded from first to third supernatant in view of sample contamination. It was confirmed abnormal PrP amplification in all soil supernatants from fourth to tenth. From 0.01% to 1% contaminated washed soils were identified as abnormal prions. 0.001% contaminated washed soil did not show PrP specific band (Fig 1). The soil was collected by the same method once of year until 4 year after stop CWD exposure. After sPMCAb, there were no PrP_{CWD} band in from second to fourth year 0.001% washed soil. but It was confirmed that the abnormal prion was amplified in the washing supernatant which was not amplified in the washed soil. we have decided to use soil supernatant for soil testing (Fig. 2). After third rounds of amplification, PrP_{Sc} signals observed in three out of four sites from CWD positive farm playground. No signals were observed in all soil samples from four CWD negative farm (Fig. 3).

Conclusions: Our studies showed that PrP_{CWD} persist in 0.001% CWD contaminated soil for at least 4 year and natural CWD-affected farm soil. When cervid reintroduced into CWD outbreak farm, the strict decontamination procedures of the infectious agent should be performed in the environment of CWD-affected cervid habitat.

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186. Serial detection of hematogenous prions in CWD-infected deer

Amy V. Nalls, Erin E. McNulty, Nathaniel D. Denkers, Edward A. Hoover and Candace K. Mathiason

Department of Microbiology, Immunology, and Pathology, Colorado State University, Fort Collins, CO, USA

CONTACT Amy V. Nalls amy.nalls@colostate.edu

ABSTRACT

Blood contains the infectious agent associated with prion disease affecting several mammalian species, including humans, cervids, sheep, and cattle. It has been confirmed that sufficient prion agent is present in the blood of both symptomatic and asymptomatic carriers to initiate the amyloid templating and accumulation process that results in this fatal neurodegenerative disease. Yet, to date, the ability to detect blood-borne prions by *in vitro* methods remains difficult.

We have capitalized on blood samples collected from longitudinal chronic wasting disease (CWD) studies in the native white-tailed deer host to examine hematogenous prion load in blood collected minutes, days, weeks and months post exposure. Our work has focused on refinement of the amplification methods RT-QuIC and PMCA. We demonstrate enhanced *in vitro* detection of amyloid seeding activity (prions) in blood cell fractions harvested from deer orally-exposed to 300 ng CWD positive brain or saliva.

These findings permit assessment of the role hematogenous prions play in the pathogenesis of CWD and provide tools to assess the same for prion diseases of other mammalian species.

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

Considering the oral secretion of prions, saliva from CWD-infected deer was shown to transmit disease to other susceptible naïve deer when harvested from the animals in both the prions in the saliva and blood of deer with chronic wasting disease.

and preclinical stages69 [tandfonline.com]

of infection, albeit within relatively large volumes of saliva (50 ml). In sheep with preclinical, natural scrapie infections, sPMCA facilitated the detection of PrP_{Sc} within buccal swabs throughout most of the incubation period of the disease with an apparent peak in prion secretion around the mid-term of disease progression.70 [tandfonline.com]

The amounts of prion present in saliva are likely to be low as indicated by CWD-infected saliva producing prolonged incubation periods and incomplete attack rates within the transgenic mouse bioassay.41 [tandfonline.com]

snip...

Indeed, it has also been shown that the scrapie and CWD prions are excreted in urine, feces and saliva and are likely to be excreted from skin. While levels of prion within these excreta/secreta are very low, they are produced throughout long periods of preclinical disease as well as clinical disease. Furthermore, the levels of prion in such materials are likely to be increased by concurrent inflammatory conditions affecting the relevant secretory organ or site. Such dissemination of prion into the environment is very likely to facilitate the repeat exposure of flockmates to low levels of the disease agent, possibly over years.

snip...

Given the results with scrapie-contaminated milk and CWD-contaminated saliva, it seems very likely that these low levels of prion in different secreta/excreta are capable of transmitting disease upon prolonged exposure, either through direct animal-to-animal contact or through environmental reservoirs of infectivity.

https://www.tandfonline.com/doi/full/10.4161/pri.4.4.13678 [tandfonline.com]

the other part, these tissues and things in the body then shed or secrete prions which then are the route to other animals into the environment, so in particular, the things, the secretions that are infectious are salvia, feces, blood and urine. so pretty much anything that comes out of a deer is going to be infectious and potential for transmitting disease.

https://www.youtube.com/watch?v=bItnEEIzuKo&index=6&list=PL7ZG8MkruQh3wI96XQ8_EymytO828rGxj [youtube.com]

HUNTERS, CWD TSE PRION, THIS SHOULD A WAKE UP CALL TO ALL OF YOU GUTTING AND BONING OUT YOUR KILL IN THE FIELD, AND YOUR TOOLS YOU USE...

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

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

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

Laboratory of Central Nervous System Studies, National Institute of

Neurological Disorders and Stroke, National Institutes of Health,

Bethesda, MD 20892.

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

PMID: 8006664 [PubMed - indexed for MEDLINE]

http://jnnp.bmj.com/content/57/6/757.long [jnnp.bmj.com]

Wednesday, September 11, 2019

Is the re-use of sterilized implant abutments safe enough? (Implant abutment safety) iatrogenic TSE Prion

https://prionprp.blogspot.com/2019/09/is-re-use-of-sterilized-implant.html [prionprp.blogspot.com]

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 [bovineprp.blogspot.com]

Monday, November 30, 2020

CAMEL PRION DISEASE OR MAD CAMEL DISASE

***>Tunisia has become the second country after Algeria to detect a case of CPD within a year

https://camelusprp.blogspot.com/2020/11/tunisia-has-become-second-country-after.html [camelusprp.blogspot.com]

TUESDAY, NOVEMBER 17, 2020

The European Union summary report on surveillance for the presence of transmissible spongiform encephalopathies (TSE) in 2019 First published 17 November 2020

https://efsaopinionbseanimalprotein.blogspot.com/2020/11/the-european-union-summary-report-on.html [efsaopinionbseanimalprotein.blogspot.com]

WEDNESDAY, OCTOBER 28, 2020

EFSA Annual report of the Scientific Network on BSE-TSE 2020 Singeltary Submission

https://efsaopinionbseanimalprotein.blogspot.com/2020/10/efsa-annual-report-of-scientific.html [efsaopinionbseanimalprotein.blogspot.com]

WEDNESDAY, OCTOBER 28, 2020

EFSA Scientific Opinion Potential BSE risk posed by the use of ruminant collagen and gelatine in feed for non-ruminant farmed animals

https://efsaopinionbseanimalprotein.blogspot.com/2020/10/efsa-scientific-opinion-potential-bse.html [efsaopinionbseanimalprotein.blogspot.com]

WEDNESDAY, DECEMBER 2, 2020

EFSA Evaluation of public and animal health risks in case of a delayed post-mortem inspection in ungulates EFSA Panel on Biological Hazards (BIOHAZ) ADOPTED: 21 October 2020

i wonder if a 7 month delay on a suspect BSE case in Texas is too long, on a 48 hour turnaround, asking for a friend???

https://efsaopinionbseanimalprotein.blogspot.com/2020/12/efsa-evaluation-of-public-and-animal.html [efsaopinionbseanimalprotein.blogspot.com]

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

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

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

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

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

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

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

Chronic Wasting Disease CWD TSE Prion aka mad deer disease zoonosis

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 [grantome.com]

ZOONOTIC CHRONIC WASTING DISEASE CWD TSE PRION UPDATE

Prion 2017 Conference

First evidence of intracranial and peroral transmission of Chronic Wasting Disease (CWD) into Cynomolgus macaques: a work in progress Stefanie Czub1, Walter Schulz-Schaeffer2, Christiane Stahl-Hennig3, Michael Beekes4, Hermann Schaetzl5 and Dirk Motzkus6 1

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

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

Here we present first data on a group of animals either challenged ic with steel wires or per orally and sacrificed with incubation times ranging from 4.5 to 6.9 years at postmortem. Three animals displayed signs of mild clinical disease, including anxiety, apathy, ataxia and/or tremor. In four animals wasting was observed, two of those had confirmed diabetes. All animals have variable signs of prion neuropathology in spinal cords and brains and by supersensitive IHC, reaction was detected in spinal cord segments of all animals. Protein misfolding cyclic amplification (PMCA), real-time quaking-induced conversion (RT-QuiC) and PET-blot assays to further substantiate these findings are on the way, as well as bioassays in bank voles and transgenic mice.

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

PRION 2017 DECIPHERING NEURODEGENERATIVE DISORDERS

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/ [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 [prion2018.org]

https://prion2018.org/ [prion2018.org]

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

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

International Conference on Emerging Diseases, Outbreaks & Case Studies & 16th Annual Meeting on Influenza March 28-29, 2018 | Orlando, USA

Qingzhong Kong

Case Western Reserve University School of Medicine, USA

Zoonotic potential of chronic wasting disease prions from cervids

Chronic wasting disease (CWD) is the prion disease in cervids (mule deer, white-tailed deer, American elk, moose, and reindeer). It has become an epidemic in North America, and it has been detected in the Europe (Norway) since 2016. The widespread CWD and popular hunting and consumption of cervid meat and other products raise serious public health concerns, but questions remain on human susceptibility to CWD prions, especially on the potential difference in zoonotic potential among the various CWD prion strains. We have been working to address this critical question for well over a decade. We used CWD samples from various cervid species to inoculate transgenic mice expressing human or elk prion protein (PrP). We found infectious prions in the spleen or brain in a small fraction of CWD-inoculated transgenic mice expressing human PrP, indicating that humans are not completely resistant to CWD prions; this finding has significant ramifications on the public health impact of CWD prions. The influence of cervid PrP polymorphisms, the prion strain dependence of CWD-to-human transmission barrier, and the characterization of experimental human CWD prions will be discussed.

Speaker Biography Qingzhong Kong has completed his PhD from the University of Massachusetts at Amherst and Post-doctoral studies at Yale University. He is currently an Associate Professor of Pathology, Neurology and Regenerative Medicine. He has published over 50 original research papers in reputable journals (including Science Translational Medicine, JCI, PNAS and Cell Reports) and has been serving as an Editorial Board Member on seven scientific journals. He has multiple research interests, including public health risks of animal prions (CWD of cervids and atypical BSE of cattle), animal modeling of human prion diseases, mechanisms of prion replication and pathogenesis, etiology of sporadic Creutzfeldt-Jacob disease (CJD) in humans, normal cellular PrP in the biology and pathology of multiple brain and peripheral diseases, proteins responsible for the α-cleavage of

cellular PrP, as well as gene therapy and DNA vaccination.

qxk2@case.edu

https://www.alliedacademies.org/conference-abstracts-files/zoonotic-potential-of-chronic-wasting-disease-prions-from.pdf [alliedacademies.org]

https://prionconference.blogspot.com/2018/02/prion-round-table-conference-2018-may.html [prionconference.blogspot.com]

http://prionconference.blogspot.com/2018/02/prion-round-table-conference-2018-may.html [prionconference.blogspot.com]

http://prionconference.blogspot.com/ [prionconference.blogspot.com]

SATURDAY, FEBRUARY 23, 2019

Chronic Wasting Disease CWD TSE Prion and THE FEAST 2003 CDC an updated review of the science 2019

https://chronic-wasting-disease.blogspot.com/2019/02/chronic-wasting-disease-cwd-tse-prion.html [chronic-wasting-disease.blogspot.com]

TUESDAY, NOVEMBER 04, 2014

Six-year follow-up of a point-source exposure to CWD contaminated venison in an Upstate New York community: risk behaviours and health outcomes 2005–2011

Authors, though, acknowledged the study was limited in geography and sample size and so it couldn't draw a conclusion about the risk to humans. They recommended more study. Dr. Ermias Belay was the report's principal author but he said New York and Oneida County officials are following the proper course by not launching a study. "There's really nothing to monitor presently. No one's sick," Belay said, noting the disease's incubation period in deer and elk is measured in years. "

http://chronic-wasting-disease.blogspot.com/2014/11/six-year-follow-up-of-point-source.html [chronic-wasting-disease.blogspot.com]

Transmission Studies

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

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

snip....

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

Prion Infectivity in Fat of Deer with Chronic Wasting Disease

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

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

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

Prions in Skeletal Muscles of Deer with Chronic Wasting Disease

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

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

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

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

From: TSS

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

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

From: "Belay, Ermias"

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

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

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

Dear Sir/Madam,

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

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

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

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

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

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

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

Thursday, April 03, 2008

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

snip...

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

snip... full text ;

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

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

sporadic, spontaneous CJD, 85%+ of all human TSE, just not just happen. never in scientific literature has this been proven.

if one looks up the word sporadic or spontaneous at pubmed, you will get a laundry list of disease that are classified in such a way;

sporadic = 54,983 hits https://www.ncbi.nlm.nih.gov/pubmed/?term=sporadic [ncbi.nlm.nih.gov]

spontaneous = 325,650 hits https://www.ncbi.nlm.nih.gov/pubmed/?term=spontaneous [ncbi.nlm.nih.gov]

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

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

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

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

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

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

*** IF CWD is not a risk factor for humans, then I guess the FDA et al recalled all this CWD tainted elk tenderloin (2009 Exotic Meats USA of San Antonio, TX) for the welfare and safety of the dead elk. ...tss

Exotic Meats USA Announces Urgent Statewide Recall of Elk Tenderloin Because It May Contain Meat Derived From An Elk Confirmed To Have Chronic Wasting Disease

Contact: Exotic Meats USA **1-800-680-4375**

FOR IMMEDIATE RELEASE -- February 9, 2009 -- Exotic Meats USA of San Antonio, TX is initiating a voluntary recall of Elk Tenderloin because it may contain meat derived from an elk confirmed to have Chronic Wasting Disease (CWD). The meat with production dates of December 29, 30 and 31, 2008 was purchased from Sierra Meat Company in Reno, NV. The infected elk came from Elk Farm LLC in Pine Island, MN and was among animals slaughtered and processed at USDA facility Noah's Ark Processors LLC.

Chronic Wasting Disease (CWD) is a fatal brain and nervous system disease found in elk and deer. The disease is caused by an abnormally shaped protein called a prion, which can damage the brain and nerves of animals in the deer family. Currently, it is believed that the prion responsible for causing CWD in deer and elk is not capable of infecting humans who eat deer or elk contaminated with the prion, but the observation of animal-to-human transmission of other prion-mediated diseases, such as bovine spongiform encephalopathy (BSE), has raised a theoretical concern regarding the transmission of CWD from deer or elk to humans. At the present time, FDA believes the risk of becoming ill from eating CWD-positive elk or deer meat is remote. However, FDA strongly advises consumers to return the product to the place of purchase, rather than disposing of it themselves, due to environmental concerns.

Exotic Meats USA purchased 1 case of Elk Tenderloins weighing 16.9 lbs. The Elk Tenderloin was sold from January 16 – 27, 2009. The Elk Tenderloins was packaged in individual vacuum packs weighing approximately 3 pounds each. A total of six packs of the Elk Tenderloins were sold to the public at the Exotic Meats USA retail store. Consumers who still have the Elk Tenderloins should return the product to Exotic Meats USA at 1003 NE Loop 410, San Antonio, TX 78209. Customers with concerns or questions about the Voluntary Elk Recall can call **1-800-680-4375**. The safety of our customer has always been and always will be our number one priority.

Exotic Meats USA requests that for those customers who have products with the production dates in question, do not consume or sell them and return them to the point of purchase. Customers should return the product to the vendor. The vendor should return it to the distributor and the distributor should work with the state to decide upon how best to dispose. If the consumer is disposing of the product he/she should consult with the local state EPA office.

#

RSS Feed for FDA Recalls Information11 [what's this?12]

http://www.fda.gov/Safety/Recalls/ArchiveRecalls/2009/ucm128543.htm [fda.gov]

USGS Outstanding in the Field podcast, Episode 3: Chronic Wasting Disease - Oh, Deer (Credit: USGS)

https://prd-wret.s3.us-west-2.amazonaws.com/assets/palladium/production/s3fs-public/atoms/audio/20190628_3_oh_deerpodcast.mp3 [prd-wret.s3.us-west-2.amazonaws.com]

https://www.usgs.gov/news/chronic-wasting-disease-can-science-save-our-dear-deer?qt-news_science_products=1#qt-news_science_products [usgs.gov]

TUESDAY, DECEMBER 29, 2020

Chronic Wasting Disease: Can Science Save Our Dear Deer?

https://chronic-wasting-disease.blogspot.com/2020/12/chronic-wasting-disease-can-science.html [chronic-wasting-disease.blogspot.com]

FRIDAY, JULY 26, 2019

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

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

TUESDAY, JANUARY 21, 2020

***> 2004 European Commission Chronic wasting disease AND TISSUES THAT MIGHT CARRY A RISK FOR HUMAN FOOD AND ANIMAL FEED CHAINS REPORT UPDATED 2020

https://chronic-wasting-disease.blogspot.com/2020/01/2004-european-commission-chronic.html [chronic-wasting-disease.blogspot.com]

CWD TSE PRION AND ZOONOTIC, ZOONOSIS, POTENTIAL

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 [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 [caninespongiformencephalopathy.blogspot.com]

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

snip...see full text;

"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)."

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

Consumption of venison and veal was much less widespread among both cases and controls. For both of these meats there was evidence of a trend with increasing frequency of consumption being associated with increasing risk of CJD. (not nvCJD, but sporadic CJD...tss) These associations were largely unchanged when attention was restricted to pairs with data obtained from relatives. ...

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

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

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

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

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

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

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

snip...

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

snip...

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

snip...

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

snip...see full report ;

http://web.archive.org/web/20090506050043/http://www.bseinquiry.gov.uk/files/yb/1994/08/00004001.pdf [web.archive.org]

http://web.archive.org/web/20090506050007/http://www.bseinquiry.gov.uk/files/yb/1994/10/00003001.pdf [web.archive.org]

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

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 [bseinquiry.blogspot.com]

> In conclusion, sensory symptoms and loss of reflexes in Gerstmann-Sträussler-Scheinker syndrome can be explained by neuropathological changes in the spinal cord. We conclude that the sensory symptoms and loss of lower limb reflexes in Gerstmann-Sträussler-Scheinker syndrome is due to pathology in the caudal spinal cord. <

> The clinical and pathological presentation in macaques was mostly atypical, with a strong emphasis on spinal cord pathology.<

> 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. <

> All animals have variable signs of prion neuropathology in spinal cords and brains and by supersensitive IHC, reaction was detected in spinal cord segments of all animals.<

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

https://familialcjdtseprion.blogspot.com/2019/02/cwd-gss-tse-prion-spinal-cord-confucius.html [familialcjdtseprion.blogspot.com]

TUESDAY, NOVEMBER 17, 2020

The European Union summary report on surveillance for the presence of transmissible spongiform encephalopathies (TSE) in 2019 First published 17 November 2020

https://efsaopinionbseanimalprotein.blogspot.com/2020/11/the-european-union-summary-report-on.html [efsaopinionbseanimalprotein.blogspot.com]

FRIDAY, OCTOBER 30, 2020

Efficient transmission of US scrapie agent by intralingual route to genetically susceptible sheep with a low dose inoculum

https://scrapie-usa.blogspot.com/2020/10/efficient-transmission-of-us-scrapie.html [scrapie-usa.blogspot.com]

TUESDAY, JANUARY 12, 2021

Annual Scrapie Report Available for Fiscal Year 2020 USA October 1, 2019 to September 30, 2020

https://scrapie-usa.blogspot.com/2021/01/annual-scrapie-report-available-for.html [scrapie-usa.blogspot.com]

THURSDAY, JANUARY 7, 2021

Atypical Nor-98 Scrapie TSE Prion USA State by State Update January 2021

https://nor-98.blogspot.com/2021/01/atypical-nor-98-scrapie-tse-prion-usa.html [nor-98.blogspot.com]

FRIDAY, FEBRUARY 12, 2021

Transmission of the atypical/Nor98 scrapie agent to Suffolk sheep with VRQ/ARQ, ARQ/ARQ, and ARQ/ARR genotypes

https://transmissiblespongiformencephalopathy.blogspot.com/2021/02/transmission-of-atypicalnor98-scrapie.html [transmissiblespongiformencephalopathy.blogspot.com]

WEDNESDAY, FEBRUARY 10, 2021

SENATORS URGE BIDEN TO WITHDRAW SHEEP IMPORT RULE DUE TO SCRAPIE TSE Prion CONCERNS

https://scrapie-usa.blogspot.com/2021/02/senators-urge-biden-to-withdraw-sheep.html [scrapie-usa.blogspot.com]

WEDNESDAY, FEBRUARY 03, 2021

Scrapie TSE Prion United States of America a Review February 2021 Singeltary et al

https://scrapie-usa.blogspot.com/2021/02/scrapie-tse-prion-united-states-of.html [scrapie-usa.blogspot.com]

TUESDAY, JANUARY 5, 2021

Exploration of genetic factors resulting in abnormal disease in cattle experimentally challenged with bovine spongiform encephalopathy

https://bovineprp.blogspot.com/2021/01/exploration-of-genetic-factors.html [bovineprp.blogspot.com]

2.3.2. New evidence on the zoonotic potential of atypical BSE and atypical scrapie prion strains

PLEASE NOTE;

2.3.2. New evidence on the zoonotic potential of atypical BSE and atypical scrapie prion strainsNo

Olivier Andreoletti, INRA Research Director, Institut National de la Recherche Agronomique (INRA) – École Nationale Vétérinaire de Toulouse (ENVT), invited speaker, presented the results of two recently published scientific articles of interest, of which he is co-author: 'Radical Change in Zoonotic Abilities of Atypical BSE Prion Strains as Evidenced by Crossing of Sheep Species Barrier in Transgenic Mice' (MarinMoreno et al., 2020) and 'The emergence of classical BSE from atypical/Nor98 scrapie' (Huor et al., 2019).

In the first experimental study, H-type and L-type BSE were inoculated into transgenic mice expressing all three genotypes of the human PRNP at codon 129 and into adapted into ARQ and VRQ transgenic sheep mice. The results showed the alterations of the capacities to cross the human barrier species (mouse model) and emergence of sporadic CJD agents in Hu PrP expressing mice: type 2 sCJD in homozygous TgVal129 VRQ-passaged L-BSE, and type 1 sCJD in homozygous TgVal 129 and TgMet129 VRQ-passaged H-BSE.

https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/sp.efsa.2020.EN-1946 [efsa.onlinelibrary.wiley.com]

WEDNESDAY, OCTOBER 28, 2020

***> EFSA Annual report of the Scientific Network on BSE-TSE 2020 Singeltary Submission

https://efsaopinionbseanimalprotein.blogspot.com/2020/10/efsa-annual-report-of-scientific.html [efsaopinionbseanimalprotein.blogspot.com]

SUNDAY, OCTOBER 11, 2020

Bovine adapted transmissible mink encephalopathy is similar to L-BSE after passage through sheep with the VRQ/VRQ genotype but not VRQ/ARQ

https://transmissible-mink-encephalopathy.blogspot.com/2020/10/bovine-adapted-transmissible-mink.html [transmissible-mink-encephalopathy.blogspot.com]

THURSDAY, SEPTEMBER 24, 2020

The emergence of classical BSE from atypical/ Nor98 scrapie

https://nor-98.blogspot.com/2020/09/the-emergence-of-classical-bse-from.html [nor-98.blogspot.com]

FRIDAY, OCTOBER 23, 2020

Scrapie TSE Prion Zoonosis Zoonotic, what if?

https://transmissiblespongiformencephalopathy.blogspot.com/2020/10/scrapie-tse-prion-zoonosis-zoonotic.html [transmissiblespongiformencephalopathy.blogspot.com]

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.

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.

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

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.

==========

thus questioning the origin of human sporadic cases

==========

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

==========

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

***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.

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

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

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.

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

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.

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

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

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

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

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

snip...

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

PMID: 6997404

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

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

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

snip...

76/10.12/4.6

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

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

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

Gibbs CJ Jr, Gajdusek DC.

Nature 236, 73 - 74 (10 March 1972); doi:10.1038/236073a0

Transmission of Scrapie to the Cynomolgus Monkey (Macaca fascicularis)

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

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

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

http://www.nature.com/nature/journal/v236/n5341/abs/236073a0.html [nature.com]

http://scrapie-usa.blogspot.com/2010/04/scrapie-and-atypical-scrapie.html [scrapie-usa.blogspot.com]

Wednesday, February 16, 2011

IN CONFIDENCE

SCRAPIE TRANSMISSION TO CHIMPANZEES

IN CONFIDENCE

http://scrapie-usa.blogspot.com/2011/02/in-confidence-scrapie-transmission-to.html [scrapie-usa.blogspot.com]

MONDAY, DECEMBER 16, 2019

Chronic Wasting Disease CWD TSE Prion aka mad cow type disease in cervid Zoonosis Update

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

What if?

https://chronic-wasting-disease.blogspot.com/2019/12/chronic-wasting-disease-cwd-tse-prion.html [chronic-wasting-disease.blogspot.com]

DECEMBER 2020 TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHY TSE BSE, SCRAPIE, CWD, CPD, PPD, CJD END OF YEAR REPORTS

MONDAY, DECEMBER 14, 2020

Experimental oral transmission of chronic wasting disease to sika deer (Cervus nippon)

https://chronic-wasting-disease.blogspot.com/2020/12/experimental-oral-transmission-of.html [chronic-wasting-disease.blogspot.com]

FRIDAY, FEBRUARY 05, 2021

USA 50 STATE CWD TSE Prion UPDATE FEBRUARY 2021

https://chronic-wasting-disease.blogspot.com/2021/02/usa-50-state-cwd-tse-prion-update.html [chronic-wasting-disease.blogspot.com]

MONDAY, NOVEMBER 23, 2020

Chronic Wasting Disease CWD TSE Prion Cervid State by State and Global Update November 2020

https://chronic-wasting-disease.blogspot.com/2020/11/chronic-wasting-disease-cwd-tse-prion.html [chronic-wasting-disease.blogspot.com]

WEDNESDAY, APRIL 21, 2021

A Texas Rancher Cloned Deer For Years. Some Lawmakers Want To Legalize It (what about cwd tse prion)?

https://chronic-wasting-disease.blogspot.com/2021/04/a-texas-rancher-cloned-deer-for-years.html [chronic-wasting-disease.blogspot.com]

THURSDAY, FEBRUARY 4, 2021

Guidance for reporting 2021 surveillance data on Transmissible Spongiform Encephalopathies (TSE)

APPROVED: 1 February 2021

https://efsaopinionbseanimalprotein.blogspot.com/2021/02/guidance-for-reporting-2021.html [efsaopinionbseanimalprotein.blogspot.com]

Terry S. Singeltary Sr.