ATTACHMENT ONE

Source:
Jennifer O'Brien

415-476-2557

September 9, 2009

Prions found in feces of deer asymptomatic for chronic wasting disease

Scientists have discovered that deer asymptomatic for a fatal brain condition known as chronic wasting disease excrete the infectious prions that cause the disease in their feces. The finding, they say, suggests a plausible explanation for transmission of the disease among deer and, possibly, elk and moose in the environment.

The study is reported as an advance online paper on September 9, 2009 in the journal "Nature."

While the study reveals that prions are shed in feces of symptomatic deer as well, the discovery that the infected deer shed prions (PREE-ons) in their feces many months before they show clinical symptoms has particularly provocative implications, according to the research team, at University of California, San Francisco and the Colorado Division of Wildlife's Wildlife Research Center.

Deer, elk and moose inadvertently consume feces and soil in the course of their daily grazing. Given this, the team set out to determine whether the animals could develop chronic wasting disease through long-term consumption of contaminated feces. They did so by measuring the amount of prions contained in the feces of orally infected deer up until the time they became symptomatic and then calculated whether prolonged exposure to the concentrations of prions in these feces would be enough to cause the disease.

"Prion levels in feces samples of asymptomatic deer were very low compared to those in the brains of the same deer at the time of death," says the lead author of the study, Erdem Tamguney, PhD, an assistant professor at the Institute for Neurodegenerative Diseases, based at UCSF. "However, the total number of prions excreted over time was sufficiently high enough to cause disease in other deer."

The susceptibility of animals to infection, he says, might be increased by the simultaneous ingestion of clay soil, which is thought to enhance the infectivity of prions, possibly by slowing their clearance from the gastrointestinal tract.

"Our findings suggest that prolonged fecal prion excretion by infected deer provides a plausible explanation for the high level of transmission of chronic wasting disease within deer herds, as well as prion transmission among deer and other cervid species. Our work may also explain transmission of scrapie prions among sheep and goats," says senior author and Nobel laureate Stanley B. Prusiner, MD, UCSF professor of neurology and director of the UCSF Institute for Neurodegenerative Diseases.

The study did not examine whether chronic wasting disease could be transmitted to humans via exposure to deer feces. To date, transgenic mouse studies have indicated that

chronic wasting disease does not transmit to humans, but scientists remain open to the possibility that it could.

"We can only say that prions of chronic wasting disease have not transmitted to mice genetically engineered to carry the normal, healthy form of human prion protein in earlier studies," says Prusiner. "That said, we do not know for sure that deer or elk prions cannot be transmitted to humans."

The prion is an infectious form of the normal prion protein, which has been found in all mammals examined, including humans. The lethal, infectious form induces the normal protein to twist into a malconformation, initiating a disease process that ravages the brain. Prion diseases, seen in cervids, sheep, cows and humans, are also referred to as spongiform encephalopathies. Prusiner won the Nobel Prize in Physiology or Medicine in 1997 for the discovery of prions as a new biological principle of infection.

The new study sheds light on a question that has puzzled scientists for a number of years: how chronic wasting disease spreads so widely through herds and species in contrast to bovine spongiform encephalopathy in cattle, in which horizontal transmission between animals is rare.

First detected in a research facility in Colorado in 1967, chronic wasting disease has since been detected in fourteen states in the U.S. and in two Canadian provinces, predominantly in the West, both in the wild and on commercial farms. In wild herds, it can sometimes be found in up to 30 percent of animals; in captivity nearly entire herds can be affected.

Studies have shown that the disease can be transmitted orally – deer experimentally fed infected brain tissue become sick – but the animals are not carnivores, nor cannibalistic. And while prions have been reported in the saliva, blood, muscle, urine and antler velvet of symptomatic animals with late-stage disease, there is little evidence that these sources are responsible for high incidence of the disease within herds.

Epidemiological findings argue that chronic wasting disease spreads efficiently across populations. When healthy deer grazed on pastureland previously used by deer with chronic wasting disease, the healthy deer eventually develop the disease. But there has been no clear mechanism of transmission of prions in this way.

The UCSF scientists teamed up with co-investigators led by Michael Miller, DVM, PhD, of the Colorado Division of Wildlife's Wildlife Research Center, to investigate the issue. Five deer were orally infected with one gram of brain tissue from a deer that had died of chronic wasting disease. Then fecal samples from the animals were collected at five time points – once before they were infected (as a control group), and then post infection at 3-4 months, 9-10 months, 13-14 months and 16-20 months (when animals show symptoms of the disease). Symptoms include inability to hold the head erect, excessive salivation, unsteady gait and poor grooming skills.

The UCSF scientists irradiated the deer feces to kill all bacteria and viruses, and inoculated the fecal material into the brains of mice genetically engineered to over-express the normal, healthy version of the elk prion protein.

An analysis showed that feces collected before infection and at 3-4 months post infection did not transmit the disease to the mice. At all subsequent time points, however, many fecal samples did transmit the disease. Prion infectivity was found in 14 of the 15 samples collected from the five deer as early as 7-11 months before neurological symptoms developed.

To measure the prions in feces, the team conducted a separate experiment in which they correlated the time required for mice to become ill from various prion concentrations in infected elk brain tissue (brain samples were diluted with varying amounts of water). Then they compared this data to the incubation times of the mice inoculated with contaminated feces, determining the amount of infectivity of the samples at different time points. While the number of prions in individual samples was low, the amount that accumulated over a 10-month period was similar to that in brain at the end-stage of the disease.

The findings offer strong evidence, says Prusiner, that prion contamination of forest, shrub-steppe and grassland habitats may be largely responsible for the high incidence of the disease both within and between cervid species and account for geographic spread as deer move between seasonal ranges.

Other co-authors of the study are Lisa L. Wolfe, MS, DVM, and Tracey M. Sirochman, BA, of the Colorado Division of Wildlife, Wildlife Research Center; David V. Glidden, PhD, professor of epidemiology and biostatistics, Christina Palmer, MS, Azucena Lemus, BS, of pathology, and Stephen J. DeArmond, MD, PhD, professor of pathology, all of UCSF.

The study was funded by the Larry L. Hillblom Foundation, the Colorado Division of Wildlife, grants from the U.S. Department of Defense National Prion Research Program and the National Institutes of Health.

UCSF is a leading university dedicated to promoting health worldwide through advanced biomedical research, graduate-level education in the life sciences and health professions, and excellence in patient care.

Related links:

Link to map of CWD
http://www.cwd-info.org/images/CWDmap.gif

Chronic Wasting Disease Alliance
http://www.cwd-info.org/

Prusiner lab - Institute for Neurodegenerative Diseases
http://ind.universityofcalifornia.edu/

Video

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Past Issue
Vol. 10, No. 6
June 2004

Perspective

Chronic Wasting Disease and Potential Transmission to Humans

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Chronic wasting disease (CWD) of deer and elk is endemic in a tri-corner area of Colorado, Wyoming, and Nebraska, and new foci of CWD have been detected in other parts of the United States. Although detection in some areas may be related to increased surveillance, introduction of CWD due to translocation or natural migration of animals may account for some new foci of infection. Increasing spread of CWD has raised concerns about the potential for increasing human exposure to the CWD agent. The foodborne transmission of bovine spongiform encephalopathy to humans indicates that the species barrier may not completely protect humans from animal prion diseases. Conversion of human prion protein by CWD-associated prions has been demonstrated in an in vitro cell-free experiment, but limited investigations have not identified strong evidence for CWD transmission to humans. More epidemiologic and laboratory studies are needed to monitor the possibility of such transmissions.

Chronic wasting disease (CWD) is classified as a transmissible spongiform encephalopathy (TSE), or prion disease, along with other animal diseases, such as scrapie and bovine spongiform encephalopathy. The only known natural hosts for CWD are deer (*Odocoileus* species) and Rocky Mountain elk (*Cervus elaphus nelsoni*) (1,2). CWD and other TSEs are believed to be caused by a pathogenic effect on neurons of an abnormal isoform of a host-encoded glycoprotein, the prion protein. The pathogenic form of this protein appears to be devoid of nucleic acids and supports its own amplification in the host. TSEs in animals primarily occur by transmitting the etiologic agent within a species, either naturally or through domestic husbandry practices. In contrast, most such encephalopathies in humans occur as a sporadic disease with no identifiable source of infection or as a familial disease linked with mutations of the prion protein gene (3). A notable exception among the human TSEs is the variant form of Creutzfeldt-Jakob disease (vCJD), which is believed to have resulted from the foodborne transmission of bovine spongiform encephalopathy (BSE) to humans (4,5).

CWD was first identified as a fatal wasting syndrome of captive mule deer in the late 1960s in research facilities in Colorado and was recognized as a TSE in 1978 (6,7). Subsequently, this wasting disease was identified in mule deer in a research facility in Wyoming and in captive elk in both the Colorado and Wyoming facilities (6–8). The disease was first recognized in the wild in 1981, when it was diagnosed in a free-ranging elk in Colorado (1,9). By the mid-1990s, CWD had been diagnosed among free-ranging deer and elk in a contiguous area in northeastern Colorado and southeastern Wyoming, where subsequent surveillance studies confirmed it to be endemic (10). Epidemic modeling suggested that this wasting disease might have been present among free-ranging animals in some portions of the disease-endemic area several decades before it was initially recognized (10). On the basis of hunter-harvested animal surveillance, the overall prevalence of the disease in this area from 1996 through 1999 was estimated at approximately 5% in mule deer, 2% in white-tailed deer, and <1% in elk (10). In 2000, surveillance data indicated that the disease-endemic focus extended eastward into adjacent areas of Nebraska (1,11), and ongoing surveillance continues to redefine the limits of this focus.

Clinical manifestations of CWD include weight loss over weeks or months, behavioral changes, excessive salivation, difficulty swallowing, polydipsia, and polyuria (1,6–8). In some animals, ataxia and head tremors may occur. Most animals with the disease die within several months of illness onset, sometimes from aspiration pneumonia. In rare cases, illness may last for ≥1 year. In captive cervids, most cases occur in animals 2–7 years of age; however, the disease has been reported in cervids as young as 17 months and as old as >15 years of age (1). This disease can be highly transmissible within captive deer and elk populations. A prevalence of >90% was reported among mule deer in facilities where the disease has been endemic for >2 years (2,6,7,12). The mode of transmission among deer and elk is not fully understood; however, evidence supports lateral transmission through direct animal-to-animal contact or as a result of indirect exposure to the causative agent in the environment, including contaminated feed and water sources (12).

Geographic Distribution of Chronic Wasting Disease

The geographic extent of CWD has changed dramatically since 1996 (2). Two largely independent and simultaneous epidemics, one in free-ranging deer and elk and another in the captive elk and deer industry, appear to represent the main framework for explaining the disease's current distribution (2). More extensive and coordinated surveillance has provided a clearer picture of its distribution over the last few years. Since 2000, the disease in free-ranging cervids has been increasingly identified outside of the original CWD-endemic areas of Colorado and Wyoming (Figure). The observed distribution seems to be related in part to natural movement of deer and elk and to commercial movement of infected animals to areas far from the disease-endemic zone. Considerable attention has been given to recent increases in the geographic spread of the disease, which in some areas is likely a result of increased surveillance rather than evidence of explosive geographic spread.

Figure

Click to view enlarged image

Figure. Chronic wasting disease among free-ranging deer and elk by county, United States.

No single original event or source links all wasting disease foci documented to date. Given the disease's insidious nature and the apparent duration (at least several decades) of epidemics among captive and free-ranging cervids, gaps in knowledge about its spread and distribution are not surprising, particularly within the captive deer and elk industry. However, our current knowledge cannot explain some of the distinct foci of CWD among free-ranging animals (e.g., in New Mexico and Utah). Thus, unidentified risk factors may be contributing to the occurrence of CWD among free-ranging and captive cervid populations in some areas.

Chronic Wasting Disease in Free-ranging Deer and Elk

In 2000, surveillance of hunter-harvested deer first detected the occurrence of CWD in counties in southwestern Nebraska, adjacent to the previously recognized areas of Colorado and Wyoming that are endemic for this disease (Figure) (1,11). It was reported

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Prion Infectivity in Fat of Deer with Chronic Wasting Disease▽

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Chronic wasting disease (CWD) is a neurodegenerative prion disease of cervids. Some animal prion diseases, such as bovine spongiform encephalopathy, can infect humans; however, human susceptibility to CWD is unknown. In ruminants, prion infectivity is found in central nervous system and lymphoid tissues, with smaller amounts in intestine and muscle. 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.

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CHRONIC WASTING DISEASE

MONDAY, JULY 06, 2009

Prion infectivity in fat of deer with Chronic Wasting Disease

Prion infectivity in fat of deer with Chronic Wasting Disease Brent Race, Kimberly Meade-White, Richard Race, and Bruce Chesebro*
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Abstract

Chronic wasting disease (CWD) is a neurodegenerative prion disease of cervids. Some animal prion diseases, such as bovine spongiform encephalopathy, can infect humans; however, human susceptibility to CWD is unknown. In ruminants prion infectivity is found in CNS and lymphoid tissues, with lower amounts in intestine and muscle. In mice, prion infectivity was recently detected in fat. Since ruminant fat is consumed by humans and fed to animals, we titered infectivity 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/cgi/content/abstract/JVI.01127-09v1

ABOUT ME

TERRY SINGELTARY
BACLIFF, TEXAS, UNITED STATES

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

VIEW MY COMPLETE PROFILE

PREVIOUS POSTS

Infectious Prions in Pre-Clinical Deer and Transmi...

Seven Deer Test Positive for Chronic Wasting Disea...

Chronic Wasting Disease Herd

J Gen Virol. 2009 Apr;90(Pt 4):1035-47. Epub 2009 Mar 4.

Transmission of scrapie and sheep-passaged bovine spongiform encephalopathy prions to transgenic mice expressing elk prion protein.

Tamguney G, Miller MW, Giles K, Lemus A, Glidden DV, DeArmond SJ, Prusiner SB.

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Chronic wasting disease (CWD) is a transmissible, fatal prion disease of cervids and is largely confined to North America. The origin of CWD continues to pose a conundrum: does the disease arise spontaneously or result from some other naturally occurring reservoir? To address whether prions from sheep might be able to cause disease in cervids, we inoculated mice expressing the elk prion protein (PrP) transgene [Tg(ElkPrP) mice] with two scrapie prion isolates. The SSBP/1 scrapie isolate transmitted disease to Tg(ElkPrP) mice with a median incubation time of 270 days, but a second isolate failed to produce neurological dysfunction in these mice. Although prions from cattle with bovine spongiform encephalopathy (BSE) did not transmit to the Tg(ElkPrP) mice, they did transmit after being passaged through sheep. In Tg (ElkPrP) mice, the sheep-passaged BSE prions exhibited an incubation time of approximately 300 days. SSBP/1 prions produced abundant deposits of the disease-causing PrP isoform, denoted PrP(Sc), in the cerebellum and pons of Tg (ElkPrP) mice, whereas PrP(Sc) accumulation in Tg mice inoculated with sheep-passaged BSE prions was confined to the deep cerebellar nuclei, habenula and the brainstem. The susceptibility of 'cervidized' mice to 'ovinized' prions raises the question about why CWD has not been reported in other parts of the world where cervids and scrapie-infected sheep coexist.

PMID: 19264659 [PubMed - indexed for MEDLINE]

ATTACHMENT
TWO A

22/1/2010

The Hon.Tony Burke M.P.
Federal Minister for Agriculture, Fisheries and Forestry
Parliament House
Canberra
Dear Minister,

In relation to your letter to the rural community in "The Land",page25, November 5 ,2009, advice has been received that this letter was NOT written by you at all but by a staff member of DAFF.
The letter is grossly misleading to the rural community.
Please advise why this letter was allowed to be published without being checked for contained false and misleading information.
We, in the rural world depend on the integrity and professional competence of DAFF staff.
This letter is also a disgrace for most of DAFF staff members.
These members must accept this letter with the resignation that was necessary for them, following the Brazilian Beef Scandal of 2005, and the EIA escape in 2007.
Misleading information has been noted since 2000 when false and misleading incorrect information provided by Bio-Security Australia, about the Eastern Creek Quarantine Station's(ECQS) hygiene and level of care there, was exposed in the Australian Veterinary Journal in 2000 and which DAFF did NOT even consider necessary to reply to, at that time, or later .
Some colleagues consider this indefensible in the light of the escape of the EI A virus from this DAFF quarantine facility.
These colleagues are referred to the Callinan Inquiry,--- The Equine Influenza Inquiry, Outline of Submissions ,Counsel Assisting, SUBS.INQ 001.00036—Sections 6.30 and 6.31 where it is stated that Bio-Security Australia took the view that it did not
A-- "recommend or require by any import conditions or otherwise that vaccines containing out of date strains not be used," " or"
B-- " that vaccines which were regarded as less efficacious than others which were available not be used".
Two reasons were proffered by Bio-Security Australia, as to why neither of these courses were taken.
The first was " if the currently available vaccines did contain the most up to date strains, Bio-Security Australia may have looked at specifying a particular vaccine or vaccines".
This statement is unusual in that does not state that Bio-Security Australia would have specified the use of the best vaccine available.
It begs the question "Was the vaccine in use at the time of the introduction of the EIA virus into Australia considered to be the best available by the world's leading experts or is this an attempt to conceal ?
The second reason given by Bio-Security Australia was that " vaccination was only one of a number of measures taken to minimise the risk of horses with equine influenza being introduced into the general Australian horse population."

Please explain what these number of measures were.
Please include in your explanation ,Mr. John Cahill ,the most senior Bio-Security Australia executive's actions and evidence , of the 21st February 2008,pages 4015 - 4017, and why he was asked to withdraw from the Inquiry room.
On his return to the Inquiry Hearing, he gave a completely different answer to the question asked of him previously before his expulsion.
Please advise why he changed his evidence.
He said this time, that "no pre-entry quarantine premises had ever been examined for the purpose of undertaking a risk analysis so as to draft conditions."
What were those "number of measures" referred to by Bio-security Australia in 6.31 in the Outline of Submissions at the Callinan Inquiry?
Were these measures associated with the assessment and health recordings of quarantined horses stabled at ECQS and/or with the assessment of hygiene and bio-security practised at the ECQS facility in November 2007?
Please examine the evidence of DAFF executives at the 3 Senate Sub-Committee Hearings in 2005.
For example, please examine the evidences of the late Chief Scientist Bio-Security Australia, Dr Banks (RRA&T on Page 48 and PAGES 62-64)and the Chief Veterinary Surgeon of Australia, Dr. G. Murray (RRA&T PAGE 73) and advise why incorrect and misleading information was supplied to the Hearing on the 15th February 2005?
Please answer the questions asked of you as they are important to the rural world which you represent.
Bob Steel B.V.SC. M.R.C.V.S
Honorary Veterinary Surgeon N.S.W.

ATTACHMENT TWO B

Equine Influenza Inquiry

Outline of Submissions

Counsel Assisting

1 BACKGROUND TO THE INQUIRY

The equine influenza outbreak in Australia

1.1 Equine influenza is a virus which causes an acute respiratory disease in horses, donkeys, mules and zebras. Before August 2007 Australia was free of equine influenza, as was New Zealand. The importation of horses from New Zealand is of minor relevance here as that country prides itself upon its strict standards of biosecurity in the same way as Australia rightly could do so until August 2007.

1.2 Equine influenza is endemic in Europe (except Iceland) and in North and South America. Although sporadic outbreaks occur in these areas they are usually minor because of the high level of vaccination. However epidemics do occur. In the past 20 years serious epidemics have occurred in South Africa (1986 and 2003), India (1987), Hong Kong (1992), Dubai (1995) and the Philippines (1997). Most of these epidemics have been associated with the import of sub-clinically infected horses by air and inadequate post-arrival quarantine procedures. Each of these epidemics was widely discussed both publicly and in scientific circles.

1.3 Australia has a long history of importing horses. The first recorded imports were with the First Fleet in 1788. Horses were first imported by air in 1973. Since that time the number of horses imported by air has increased markedly. In the last 10 years the number imported from countries other than New Zealand has averaged in excess of 500 per year. In 2006 the number was 897. Those horses included thoroughbred and standard bred stallions imported for the Australian breeding season which commences on 1 September each year. At the end of that season most of those stallions are exported to northern hemisphere countries to participate in their breeding seasons. Those stallions are commonly referred to as "shuttle" stallions.

1.4 With the exception of horses from New Zealand, horses imported into Australia for release into the general horse population are required, by conditions imposed by their import permits, to satisfy a number of conditions before and after entry. They include vaccination against equine influenza, the undertaking of a period of Pre-Export Quarantine (PEQ) and of a further period of Post-

6.30 With one exception in 1995 (which required that the inactivated vaccine incorporate the Suffolk/89 antigen)[133] neither Biosecurity Australia nor any of its predecessors has specified that the vaccine contain any particular strain or representative strain.[134] During that same period Biosecurity Australia has been aware that some vaccines may not provide adequate protection or are less effective than others. For example, in 2005 in response to comments from Mr Barry Smyth who was then President of the Australian Horse Industry Council Inc that currently available vaccines did not contain "epidemiologically relevant strains", Biosecurity Australia noted that it was "aware that many currently available vaccines, including Duvaxyn IE Plus, may not provide adequate protection".[135] Notwithstanding that this was apparently the view within Biosecurity Australia, it did not recommend or require by any import conditions or otherwise that vaccines containing out of date strains not be used[136] or that vaccines which were regarded as less efficacious than others which were available not be used.[137]

6.31 Two reasons were proffered by Dr Martin as to why neither of these courses were taken. The first was that if the currently available vaccines did contain the most up to date strains Biosecurity Australia may have looked at specifying a particular vaccine or vaccines.[138] The second was that vaccination was only one of a number of measures taken to minimise the risk of horses with equine influenza being introduced into the general Australian horse population.[139]

PAQ

6.32 The primary course of a vaccination comprises at least two doses. Once a primary course has been administered a horse may receive annual vaccinations or boosters to that primary course. The conditions current as at August 2007 permitted either vaccination once as a booster to a certified primary course or twice at an interval of four to six weeks. They did not specify that the vaccinations have to be in accordance with manufacturers recommendations which would, presumably, require that the same vaccine be used either as the booster to the certified primary course or where there are to be two vaccinations at an interval of four to six weeks.[140] Whilst it is generally accepted that sequential vaccinations with different vaccines is "sub-optimal" some investigations conducted by the Animal Health Trust have suggested that the mixing of vaccines does not appear to have a significant affect on levels of immunity.[141] However, the benefits of using the same vaccines depend upon the product chosen and whether it contains more recently circulating strains. If it does the use of that product as a booster, as distinct from requiring it to be

[133] DAFF.0001.564.0017.
[134] T2903.
[135] DAFF.0001.091.0347.
[136] T2903.
[137] T2904 – T2910.
[138] T2902.
[139] T2904.
[140] T4221 – T4222.
[141] T4185 – T4186.

ATTACHMENT THREE

<EXAMINATION BY MR MEAGHER:

MR MEAGHER: Q. I need to understand what you mean by that last answer, Mr Cahill. You say that the question of assessing risks associated specifically with pre-embarkation quarantine facilities is more to do with operational procedures, the on-the-ground arrangements, the risk management measures that are on the ground. What are you referring to there?
A. Well, certainly when we look at the attachments to the horse policy documents at the present time, you will see in those documents descriptions of the kinds of things that the PEQ facilities are expected to have and the kinds of practices that we would expect to be undertaken within those facilities.

Q. That's what you are referring to as the matters which Biosecurity Australia should address?
A. That's correct.

Q. You mentioned earlier that you thought that Dr Robyn Martin and Dr Clegg had at some stage visited overseas quarantine facilities?
A. Yes, that's correct.

Q. But you know that neither of them has visited those facilities with a view of undertaking some risk analysis for the purpose of being satisfied that the conditions being suggested are satisfactory?
A. No, that's not correct.

Q. You say that's not correct?
A. That's right, yes.

Q. Is it your understanding that one or both of them have been to pre-entry quarantine premises for the purpose of undertaking a risk analysis to assess risk for the purpose of preparing conditions?
A. I'm aware that Dr Clegg --

Q. Please address my question.
A. Yes.

Q. What is your understanding as to what they did?
A. Prior to the 2000 Olympics, Dr Clegg travelled overseas, and one of the specific purposes of those visits was to look at PEQ facilities.

Q. They were the PEQ facilities in which country?
A. In the countries that were exporting horses to Australia for the Olympics.

Q. Do you know which countries?
A. I think there were four or five: UK, Germany - I'm not sure of the other ones. The US I think was in there as well.

Q. What is your understanding in relation to Dr Martin?
A. Dr Martin, I believe, has visited PEQ facilities at least as incidental parts of other visits that she has undertaken overseas.

Q. But not for the purpose of undertaking some risk analysis so as to draft conditions?
A. Yes, it contributes to our knowledge and understanding of those.

Q. I suggest to you that that answer is false, Mr Cahill. You know that Dr Martin hasn't gone to those premises specifically for the purpose of undertaking a risk analysis so as to draft conditions, don't you?

MR ANDRONOS: Commissioner, I object to this question. The witness has answered the previous question, "Yes, it contributes to our knowledge and understanding of those."

THE COMMISSIONER: There is no need for to you explain the answer in the presence of the witness. If you are going to go into this in detail, in view of what Mr Meagher has put, I will ask the witness to leave the room.

Would you mind leaving the room, please, Mr Cahill. It is conventional to do this sometimes; it is no affront to you.

(The witness leaves the hearing room)

MR ANDRONOS: My objection is to the express suggestion made to this witness that he has knowingly given false evidence to this Commission, when, fairly read, the previous question and answer don't support any kind of submission to that effect.

THE COMMISSIONER: I don't agree with you, Mr Andronos.

I think it is eminently proper for Mr Meagher to put the matter directly, as he has. He didn't get a responsive answer; he had to struggle to get any sort of response at all, and, when he did, he formed an advocate's view of it which was proper. I disallow the objection.

Would somebody ask Mr Cahill on come back, please. You may have to repeat the answer, Mr Meagher, and your question, to make it clear to the witness.

(The witness returns to the hearing room)

MR MEAGHER: Q. Mr Cahill, I want to be completely fair to you about this. My question was that, to your knowledge, Dr Martin has not gone overseas to a PEQ premises for the purpose of assessing risks in the premises so as to draft import conditions? That was my question. To your knowledge, she has never done that, has she?
A. That's correct.

Q. Similarly with Dr Clegg - to your knowledge, she has never gone overseas to visit premises for that specific purpose?
A. That's not correct.

Q. You say that's not correct? In saying that it is not correct, you are relying on the evidence that you have given earlier as to what you understood she did?
A. That's correct.

Q. What conditions do you understand she was specifically addressing by making those visits or which were the subject of the purpose for making those visits?
A. We're talking about a period at the end of the 1990s prior to the 2000 Olympics, and Dr Clegg visited pre-embarkation quarantine facilities in order to determine first-hand whether those facilities would meet Australia's import conditions and, if not, what modifications needed to be made to ensure that they did.

Q. Is it your recollection that those premises included premises in the United Kingdom?
A. I believe so, yes.

Q. Ireland?
A. I believe so, yes.

Q. Japan?
A. I can't be certain about Japan.

Q. The United States?
A. I believe so, yes.

Q. Germany?
A. Yes.

MR MEAGHER: I have no further questions, Commissioner.

THE COMMISSIONER: Thank you, Mr Cahill, you are excused.

<THE WITNESS WITHDREW

MR MEAGHER: Before Dr Britton is called, Commissioner, could I raise one matter. We have been in the course of putting on courtbook a number of statements from grooms, principally grooms from Darley. Those statements are identified by the prefixes WIT.DLYA or WIT.JANAH. I think there are a couple of statements that we are still waiting for from Mr Dick, but we hope to get those in the next couple of days.

Our position, as counsel assisting, is that we were not going to require that those people be available for cross-examination. Most of them would be available for cross-examination by video. We accept that other parties may wish to urge that that should occur, and we simply put the other parties on notice of that and perhaps give them some fair opportunity to respond by some time on Monday - if possible tomorrow, but certainly by Monday - so that we can make those arrangements next week.

THE COMMISSIONER: You may let us know your position. If anybody wishes to examine those witnesses - you don't want to, Mr Andronos? I am not suggesting you should. I just thought you were about to rise.

MR ANDRONOS: No, not at all.

THE COMMISSIONER: Mr Dick, you are the one who, I think, may have expressed some interest in it. Will you let us know your position?

MR DICK: They are my witnesses, Commissioner.

ATTACHMENT FOUR

Australian Government

Department of Health and Ageing

CHIEF MEDICAL OFFICER

Dr Robert Steel

Dear Dr Steel

Thank you for your letter of 28 December 2009. It was good to meet with you at the Senate Inquiry Hearing into the Government's decision to allow beef imports from Bovine Spongiform Encephalopathy (BSE) affected countries.

I have read your submission with interest but my response to you will necessarily be restricted to my area of expertise, human health. In response to your query about the possible risk to human health in light of up to date information, the review of the scientific evidence commissioned by the Department of Health and Ageing concluded that the risk to human health from imported beef remains extremely low, provided the appropriate risk mitigation strategies are put in place. This review was subject to a peer review and its claims were supported by expert scientists from the National Health and Medical Research Council (NHMRC) and I will provide a copy of your submission to the NHMRC for their information. Please find enclosed a copy of Professor Mathew's Review with my compliments.

As you know Dr Mathew's review has stated that the risk to human health is very small, described as "negligible risk" and calculated at "40 million times less than the risk from road accidents". Dr Mathews has calculated that the risk to Australians is greater from prior exposure in the UK in the last century rather than from the proposed importation change. In this case even this risk is extremely small.

In response to your request for additional knowledge of inter-species transmission of TSEs, I suggest the Department of Agriculture, Fisheries and Forestry (DAFF) is better placed to respond to this because it is an animal health issue. I have copied my response to Dr Andy Carroll Chief Veterinary Officer at DAFF.

Countries that wish to apply for access to Australia's beef market will be required to undergo a rigorous risk assessment to ensure that all beef and beef products entering Australia are derived from animals free from BSE. The new import conditions will require exporting countries to prove they have acceptable controls in place, regardless of whether or not the country has reported BSE, and demonstrate that those controls are monitored.

The risk assessment will include a close examination of the history of importation of meat and bone meal, potentially infected live cattle and potentially infected bovine products into the country making an application to Australia. It will also investigate cattle feeding practices and the processing methods for bovine carcasses, by-products and abattoir waste. This measure will be administered by the Biosecurity Services Group in DAFF and informed by advice from Food Standards Australia New Zealand (FSANZ).

MDP 84 GPO Box 9848 Canberra ACT 2601
Telephone: (02) 6289 8408 Facsimile: (02) 6285 1994

In regards to your concern about contamination of beef skeletal muscle by known TSE containing tissues, evidence since 2001 confirms the list of tissues with significant infectivity ("specified risk materials" or SRM) and confirms that tissues, such as muscle, do not inherently contain infectivity. The risk of exposure via the human food chain can be minimised if SRM are excluded and measures are implemented to reduce cross-contamination of tissues during slaughter and carcass processing. In this respect, Australia is more conservative than the OIE standard because muscle meat and blood products are not exempt from the requirements and the assessment methodology will examine the risks associated with cross- contamination of beef and beef products with infectious material.

FSANZ advises that although the SRM ban is the most effective method of reducing infectious tissues entering the food chain, it should be noted that effective control of BSE is unlikely to be achieved through a single measure. It is also necessary to ensure that animals are not fed meat and bone meal, animals that are slaughtered for the human food chain are healthy, that appropriate methods of slaughter are used and strict hygiene rules are enforced during slaughter and carcass processing to reduce the risk of cross-contamination of non-infectious tissues with SRM.

I trust that the above information is of assistance.

Yours sincerely

Professor Jim Bishop AO
MD MMed MBBS FRACP FRCPA

20 January 2010

Encl. Professor Mathew's Review – Review of Scientific Evidence to Inform Australian Policy on Transmissible Spongiform Encephalopathies (TSEs).

Cc: Dr Andy Carroll, Chief Veterinary Officer, DAFF

ATTACHMENT 5

Table 3. Scope of 132 review articles on three potential zoonotic public health issues published between January 2000 and August 2006

Issue scope	Scope of the zoonotic content		Total reviews
	Main focus	Subsection	
Total review articles	59	73	132
Association between MAP and Crohn's disease	2	15	17
The association and exposure to MAP through milk	3	3	6
The association and other exposure routes	6	1	7
Total MAP articles	11	19	30
Risk associated with antimicrobial use in animals for developing AMR in humans	20	9	29
Risk associated with antimicrobial use in a specific animal group or specific pathogen for AMR in humans	3	4	7
Total AMR articles	23	13	36
Zoonotic potential of TSEs	11	19	30
Association between bovine spongiform encephalopathy and variant Creutzfeldt–Jakob disease in humans	14	19	33
Zoonotic potential of Chronic Wasting Disease in deer	0	1	1
Zoonotic potential of Scrapie in sheep	0	2	2
Total TSE articles	25	41	66

MAP, *Mycobacterium avium* ssp. *paratuberculosis* as a potential cause of Crohn's disease; AMR, antimicrobial use in animals as a risk factor for development of antimicrobial resistance in human pathogens; TSE, potential zoonotic risk of transmissible spongiform encephalopathies.

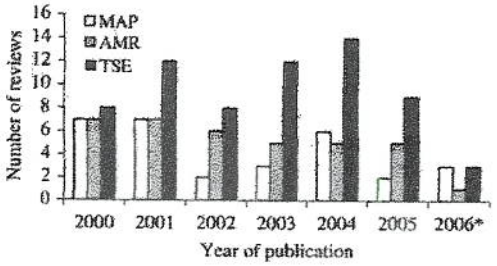

Fig. 1. Frequency of review publication between January 2000 and August 2006* on three potential zoonotic public health issues. MAP, *Mycobacterium avium* ssp. *paratuberculosis* as a potential cause of Crohn's disease (n = 30); AMR, antimicrobial use in animals as a risk factor for development of antimicrobial resistance in human pathogens (n = 36); TSE, potential zoonotic risk of transmissible spongiform encephalopathies (n = 66).

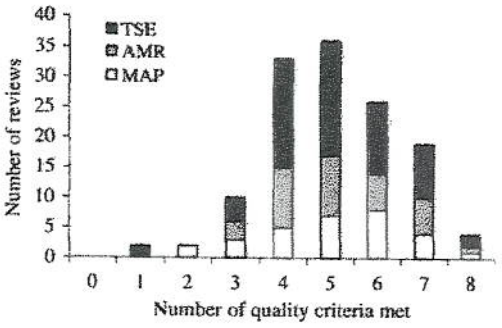

Fig. 2. Histogram of the number of quality criteria out of 13 met by 132 reviews on three zoonotic public health issues. MAP, *Mycobacterium avium* ssp. *paratuberculosis* as a potential cause of Crohn's disease (n = 30); AMR, antimicrobial use in animals as a risk factor for development of antimicrobial resistance in human pathogens (n = 36); TSE, potential zoonotic risk of transmissible spongiform encephalopathies (n = 66).

The authors stated a position that the evidence for a zoonotic association was sufficient in 30%, 83% and 74% of MAP, AMR and TSE literature reviews respectively. While authors of 57%, 14% and 20% of reviews stated the evidence for a zoonotic association was inconclusive (Table 2). In addition, there were six (4.5%) reviews where the author's position was that insufficient evidence was available to support a zoonotic association and in an equal number of reviews this question was not addressed. The authors stated a position that there was evidence to support a zoonotic risk to public health in 20% of MAP, 75% of AMR and 83% of TSE reviews, while other authors' position was that the evidence for a zoonotic risk to public health was inconclusive (60% of MAP, 14% of AMR and 11% of TSE reviews). The position of authors in seven (5.3%) reviews was the zoonotic risk to public health is not supported by the evidence and in an equal number of reviews this question was not addressed (Table 2). In only two reviews (Bosque, 2002; Caramelli et al., 2006), the authors made an attempt to provide a quantitative synthesis of research evidence and in only four reviews (Bosque, 2002; Will, 2002; Smith, 2003; Molbak, 2004) the authors summarized the magnitude of the zoonotic risk to humans through specific measures of effect (Bosque, 2002; Will, 2002).

ATTACHMENT SIX A

Australian Government

Department of Agriculture, Fisheries and Forestry

20 January 2010

Dr Bob Steel BVSc MRCVS

Dear Dr Steel,

Thank you for your letter of 28 December 2009 addressed to the Hon. Tony Burke MP, Minister for Agriculture, Fisheries and Forestry, Dr Nunn and myself, requesting that specialist staff in the Department of Agriculture, Fisheries and Forestry (DAFF) advise of errors or flaws in your submission to the Senate inquiry into "the possible impacts and consequences for public health, trade and agriculture of the Government's decision to relax import restrictions on beef". Minister Burke and Dr Nunn have asked me to reply on their behalf.

An assessment of the submission that was attached to your letter has been conducted as you requested and I can advise as follows. In regard to an alleged increase in the risks of introduction of foot-and-mouth disease to Australia, there will be no change to Australia's existing quarantine measures for this disease. In regard to BSE and the safety of imported beef, the risks are adequately addressed in the *Review of Scientific Evidence to Inform Australian Policy on Transmissible Spongiform Encephalopathies (TSEs) 2009 Addendum* by Professor Mathews. This document is available on the Department of Health and Ageing's website http://www.health.gov.au/internet/main/publishing.nsf/Content/ohp-bse-review-2009-addendum).

In regard to chronic wasting disease (CWD) of deer and scrapie of sheep and goats, there is no evidence that these diseases can naturally infect cattle grazing on the same pastures as infected deer, sheep or goats. There is also no evidence that people can be infected by CWD or scrapie by consumption of meat from, respectively, infected deer or sheep and goats. DAFF veterinarians working on transmissible spongiform encephalopathy issues are aware of the CWD research conducted by Dr. Elizabeth Williams that you highlight in your submission.

In your letter you also raise concerns with the processing of cattle in Victorian knackeries and the possibility that this may lead to BSE infection of Australian cattle or pets. I can allay your concerns because national and international risk assessments have confirmed that Australia meets the requirements of a negligible BSE risk country and the feeding of meat and bone meal to cattle has been prohibited in all Australian states and territories since 1997.

As your letter refers to related correspondence you have entered into with Professor Jim Bishop, Chief Medical Officer, I have copied this response to him. Thank you again for your letter.

Yours sincerely

Dr Andy Carroll
Chief Veterinary Officer (Australia)
Delegate to the OIE (Australia)

cc Professor Jim Bishop, Chief Medical Officer

18 Marcus Clarke Street Canberra City ACT GPO Box 858 Canberra ACT 2601 ph +61 2 6272 3933 fax +61 6272 3008 www.daff.gov.au ABN 24 113 085 695

ATTACHMENT
SIX
B

29th December 2009

The Hon Tony Burke M.P.
The Minister for Agriculture ,Fisheries and Forestry
Parliament House
Canberra ACT 2600

Dear Minister,
In reference to your letter to the rural community in the "Land" ,page 25, 5th November 2009, advice has been received that this letter was not written by you but by a staff member of DAFF.
Please advise why this letter was allowed to be published, without being checked for contained false and misleading information.
We, in the rural community and the veterinary profession depend on the integrity and professional competence of DAFF staff.
Please explain what the writer means by "We have so far avoided an economic wipe-out of our red meat industries-but it has been a gamble.", with the following " if there was a case of BSE in Tasmania ,then all beef would be banned from the shelves in Australia." and in the 4th last paragraph " all-off the shelves rule"
These statements are incorrect and misleading and can only be seen as misinformation to frighten the rural red meat producing community.
The writer is obviously not aware of our N.L.I.S, FROM BIRTH identifications of live cattle, of the States' abattoir approvals and classifications and control systems in place at saleyards in Australia. This would reduce the sale restrictions to a minimum ,far less even than in the U.K. during the storm of the Mad Cow Disease.
The writer is obviously unaware that, not even the U.K. Governments, has ever acted to ban beef sales in such a way. Considered controls were put in place there, but one's own relatives, who were lucky to have never had any cases of B.S.E., were able to sell their cattle for slaughter, within these Governmental restrictions and specific guidelines without any loss or hardship.
Those farms which had reported a case of B.S.E. had necessary total bans applied.
The writer suggest that " Today researcher have made major progress in understanding B.S.E." but fails to mention the evolving T.S.Es ,one of which has now spread to 14 States of America and 2 provinces of Canada.
Another in the U.K has 5000-10000 new cases detected each year.
All these countries are spending millions on unsuccessful but necessary control programs. Eradication is most often impossible.
The writer is correct when he infers that B.S.E. inter-species spread (bovine-man) has been controlled without the anticipated epidemic. It is a great scientific success.

Some TSEs are highly infectious within an affected species, some have eternal resistance in any environment but most importantly, inter-species transmission research is in it's infancy as many years may pass, before clinical expression or detection of rogue prions occur post-mortem, either following natural infection or in inter-species transmission experiments.

The writer is dissembling if he infers that the real reason all other beef importing nations have changed their policy is because they have NOT all the advantages of Australian beef.

Australia and New Zealand are the only 2 countries in the world which are free of Scrapie ,without ANY known TSE at all. This is wonderful but will be risked under the new policy

These other nations have had (B.S.E cases) or are having or may have had, known and spreading TSEs .

The writer adds to his misleading information when he states in this letter

"Our policy no longer matches the science and WE ARE FACING THE LIKEHOOD OF INTERNATIONAL LEGAL ACTION OR RETALIATORY BANS"

You are referred to the Senate Sub-Committee Hearing,15th February 2005, (RRA&T Page 53)when the then Chief Scientist ,Bio-Security Australia was forced to admit that he had mislead the Hearing when he stated " we would be expecting and indeed demanding that our trading partners accept OIE definitions of zones within Australia"

This senior executive had to admit that there was no legal obligation for our trading partners to accept any OIE definitions at all and we could not force or demand anything.

The context of this exchange referred to Foot and Mouth Disease and zones free of this, in the event of it's Australian introduction .

Nothing has changed with time or with B.S.E..

With B.S.E ,neither Australia or any of the 32 of the OIE's "controlled risk assessment" for B.S.E, can force or demand anything in law.

There has never been an expectation that this could or did exist, except in the flawed evidences provided to Senate Sub-Committee Hearings in 2005.

What is true is that the international trade in beef is highly competitive and anything goes, as it did with the wheat trade and AWB.

The writer is also unaware of the irony of his statement "If necessary we'll send officials over to help thoroughly assess other countries' controls"

Please note the evidence at the 15th February 2005 Senate Sub-Committee Hearing, from the Chief Scientist Bio-Security Australia when he stated that NO DAFF staff had ever checked the beef to be imported(from Brazil) would be coming from a FMD free zones.

The only inspections occurred on the 20-23 December 2004, after the cartons of meat arrived on the 29th November 2004.

His follow up statement is so precious ,it should be included for the writer "It was a preliminary visit to do an initial investigation into the zoning arrangements and also the public health situation in the meatworks in Brazil. It was only an initial investigation" He is referring to the 20-23rd December visit as above..

AQIS issued 9 permits for unlimited Brazilian beef importations for 2 years on the policy decisions of Bio-Security Australia in 2003-2004 .Their degree of professional

responsibility is questioned and the evidence of the Chief Veterinary Surgeon of Australia at that time is also questioned.
The writer must explain how the new policy could possibly protect consumers and farmers at the same time.
It would possibly reduce beef prices for the consumers but that depends on the importers. At the same time, farmers incomes would be reduced but processors and importers would enjoy gains.
This marketing equilibrium is one thing but the statement which goes with it, is untrue -- "with exposing our own industry to the devastation of the all-meat-off- the-shelves". This would be a world first ,even for the U.K.
The writer should be aware that "science and the rest of the world had moved on" is true in that science has discovered, since the late 1990's, far more about the dangers from inter-species transmissions of T.S.E.s ,since our old policy on BSE was originally formulated.
Please confirm that no risk analysis assessment has been undertaken on this new policy for BSE by Bio-Security Australia.
Please advise how DAFF executives could state at the Senate Hearing of the 14th December 2009, that there was no expectation that beef imports would increase significantly as a result of the new policy?.
They were already aware that 31 applications to import beef into Australia, had already been received by AQIS.
Unlimited amounts of Brazilian Beef were permitted to be imported by AQIS for 2 years in 2003-2004 with 9 permits.
What does the writer mean when "you simply don't gamble with such a vital agricultural industry".
The new policy does gamble with emerging TSEs and threatens us more from Foot and Mouth Disease risks by trading with countries where Foot and Mouth Disease occurs or has recently occurred.
The letter is not an intra-departmental advice but one which has sought to reveal the reasons for the new policy on BSE to the whole rural community of Australia!
The letter is seriously flawed and is nothing else. One has to be so concerned about the misleading information supplied in the writer's letter, that an urgent reply would be greatly appreciated.
Yours Sincerely

Robert Steel B.V.Sc M.R.C.V.S
Honorary Veterinary Surgeon N.S.W.