TABLED PR SENEL 5/2 BSE INQ. 10.50 AM.

A SUBMISSION TO THE 5th FEBRUARY 2010 SENATE SUB-COMMITTEE HEARING ON THE PROPOSED NEW BSE POLICY. (1) NEW ZEALAND'S TSEs.(2) DAFF's RAPID TESTING FOR TSEs WITH DAFF 'S ACCOUNTABILITY , (3) ANIMAL FOODS SAFETY IN AUSTRALIA. (4) FSANZ AND DAFF ROLES IN THE NEW BSE POLICY (5) EMERGING INFORMATION ON TSEs WITH EMERGING DANGER TO SHEEP INDUSTRIES ,ANIMAL AND HUMAN HEALTH. (6)THE TSEs REVIEW BY PROFESSOR JOHN MATHEWS.(7)THE NEED FOR MORE EXPERT OPINIONS ON THE NEW BSE POLICY(8) SUMMARY AND SUGGESTIONS.

Correction

On page 11 OF TODAY'S SUBMISSION –PREDATORS OF DEAD DEER HAVE NOT YET BEEN FOUND TO BE NATURALLY INFECTED with CWD . AT THIS TIME PREDATORS LIKE RACCOONS AND SCAVENGERS LIKE FIELD MICE,VOLES, ETC ARE ONLY FOUND TO BE INFECTED EXPERIMENTALLY .

INTRODUCTION

(1) **In 19th centuary the UK Govt refused to let Australian colonies import British sheep breeds so we imported Spanish merinos.60 YEARS AGO this was reversed and we imported British bred sheep and we had an outbreak of SCRAPIE WHICH WAS ERADICATED. The Federal Dept of Agriculture,to their eternal credit introduced the SCRAPIE IMPORT ACCREDITATION PROGRAM-SEE ON AUSTVETPLAN WEBSITE.This has kept us free of Scrapie every since and is one of veterinary sciences and the Dept of AGRICULTURES,BOTH FEDERAL AND STATES GREATEST ACTIONS AND RANKS WITH THE ACHIEVEMENTS IN ERADICATION OF PLEUROPNEUMONIA BOVINE BRUCELLOSIS AND bovineTUBERCULOSIS. Of course brucellosis still exists in the wild particularly in hundred of millions of feral pigs and tuberculosis still exists in wild deer with hosts of other zoonoses affecting humans.**

(2) **It is all very well to say that the ruminants introduced since 1990 have been quarantined but are owners required to notify DAFF OF DEATHS AND WHAT ARE THE RESULTS OF RAPID TESTING ON THESE ANIMALS SINCE THE TESTING PROCEDURE AT PM HAS BECOME AVAILABLE?**

(3) **THESE ELIZA BASED IMMUNOHISTOCHEMICAL TESTS WITH MOLECULAR BIOLOGY HAVE REVEALED NEW MISFOLDED PRIONS— ie new strains of TSEs.**

(4) **IN ADDITIONWITH MOLECULAR CHANGES OF PRIONS THEMSELVES ARE NOW IDENTIFIED,**
MUTATIONS of THE PROTEIN PRION GENES OF HOST ANIMALS ARE REPORTED.
ALL OF THESE VARIABLE FACTORS CAUSE VARYING SENSITIVITIES TO TSE AGENTS. For example v CJD is identified in one human genotype only so far,and sheep have a recognised genotype very resistant to the natural scrapie of Britain-in fact this is the basis for the largest genetic selection process ever undertaken .

(5) NEWLY DISCOVERED TSE AGENTS with the new misfolded confirmations are now being recognised with more sensitive testing procedures. for example SERIAL PROTEIN MISFOLDING CYCLIC AMPLIFICATION—SPMCA IN THE ENVIRONMENT AS IN DRINKING WATER say in the wild in forest vernal pools where deer drink. SPMCA HAS IDENTIFIED ROGUE PROTEINS IN ANIMAL TISSUES PREVIOUSLY CONSIDERED FREE OF TSE IN A HOST ANIMAL. SKELETAL MUSCLES, PERIPHERAL NERVES,HEART MUSCLES SPLEEN.
NEW THEORIES HAVE DEVELOPED on pathways to the central nervous system IN ORAL INFECTIONs OF A HOST BY AVENUES SUCH AS PERIPHERAL NERVES INSTEAD OF OR IN ADDITION TO THE LMPHATIC SYSTEMS OF THE BODY.
However serious errors with rapid tests were revealed in 2009 when these tests did not reveal disease in healthy looking deer who were excreting infectious prions in their faeces FOR one year BUT FAILED TO TEST POSITIVELY TO THE RAPID TESTS.

These rapid tests have most importantly of all in Europe revealed 7000 cases of BSE well before they could be recognised by clinical disease-the first symptom is ill thrift and loss of weight later which every European farmer would recognise in his herd. They were not recognised by the farmers or by the vets.

Rogue prions in TSE MAY change themselves AFTER FIRST PASSAGE AND SECOND PASSAGE IN INTER-SPECIES TRANSMISSION EXPERIMENTS .THESE RESULTS ARE VERY REVEALING.ie 10/13 cattle were affected by 1/c delivered CWD IN 5.5 YEARS BUT ON SECOND PASSAGE ALL of CALVES(NOT THEIR CALVES WHICH WOULD HAVE BEEN EVEN MORE REVEALING INDEED) DEVELOPED DISEASE IN 16.5 MONTHS.

TSEs adapt to new hosts so that varying incubation periods in NEW hosts occur.

Size of dose and route of access of misfolded prions affect expression in the host so that disease may not develop in the life span of that host
Thus disease may be concealed by the life span of the host o and AS WELL AS THIS heritable transfer occusr AND DISEASE MAY OCCUR IN LATER GENERATIONS WITHOUT NECESSARILY EXPRESSING IN A PREVIOUS GENERATION.

SCIENCE HAS REVEALED----------

IN SHEEP---IN THE UK PRION PROTEIN GENE RELATED TO RESISTANCE TO NATURAL SCAPIE HAs BEEN IDENTIFIED SO NOW THE world's BIGGEST EVER GENETIC SELECTION PROCESS IS UNDERWAY.Some breeds of sheep viz Cheviot sheep are most sensitive and work is underway to refine this sensitivity in sheep and identify genes related to sensitivity to Scrapie. VARIOUS ALELLES or different molecular types of the prion protein gene exist within breeds and between breeds and mutations of prion protein genes themselves occur in nature

IN CATTLE--IT MAY BE POSSIBLE TO FIND THE GENE MOST RELATED TO RESTSTANCE TO BSE IN CATTLE AND IT MAY BE NECESSARY TO DO THIS IN THE FUTURE .IT HAS NOT BEEN DONE NOR IS THERE much information on alleles of this prion protein which may be associated with THEIR sensitivity.

Sensitivity is related to the degree of and timing in life of the amplification of rogue prions in the affected host animal.

Other factors will alter amplification ie THE AMOUNT OF, the frequence of infective doses and even environmental factors. CALVES HAVE DIFFERING DIGESTION TO ADULT CATTLE SO DIRECTABOMASAL ABORPTION OF ROGUE PROTEINS IS EASY IN THE NEW BORN AND IN THE ARTIFICIALLY FED CALF. PLEASE NOTE THAT SCRAPIE PRIONS ARE INACTIVATED BY ADULT CATTLE RUMINAL DIGESTION AND THIS HAS BEEN A MAJOR LIMIT TO THE SPREAD OF SCRAPIE TO CATTLE IN NATURE.

Infection of Scrapie in cattle COULD occur via thorn injured trauma wounds within the mouth and lips etc but the size of the infective dose may be too small to result in normallife timed disease expression but SPECIES RESILIANCE IS CONSIDERED THE SAVIOUR OF CATTLE FROM SHEEP SCRAPIE.

IN HUMANS v CJD IS REPORTED ONLY IN ONE GROUP OF HUMANS EXPRESSING ONE TYPE OF PRION PROTEIN GENE. IN OTHER WORDS THE MOST SENSTIIVE GROUP TO BSE HAS BEEN IDENTIFIED NOT THE RESISTANCE OF HUMANS WITH THE OTHER ALLELES OR TYPES OF FRION PROTEIN GENES . SO THE MOSTGENETICALLY SENTITIVE GROUP OF HUMANS HAS BEEN IDENTIFIED BUT NOT MUCH OF AN IDEA OF WHAT ARE THE GENETIC RESISTANT GROUPS IN MANKIND.

THE IMPORTANCE OF THE NEW LIVE TSE BLOOD TEST " AMORFIX" CANNOT BE DETERMINED YET IN THE LIGHT OF RECENT MOLECULAR RESEARCH AND THE HERITABLE TRANSMISSION OF TSEs IN ANIMALS AND MAN.

IN HUMANS CJD CAN BE BOTH HERITABLE SPONTANEOUS AND INFECTIOUS.

TISSUE EXPRESSION OF THE PRION GENE IS NOT A PREQUISITE FOR INFECTIOUSNESS AS IN MILK IN BSE GOATS,SHEEP AND CATTLE.

The Dec 2009 OIE register of approved tests is disappointingly meagre and anachronistic. THE EU IS THE PLACE TO FIND INFORMATION NOT THE OIE.

The Hearing needs to know where Australia's present test system--Prionics-Immunoblot stands, (suggestions of excellence have been made) with the resultant significance of DAFF's tests results for BSE, Scrapie and Aypical Scrapie.

Strains of BSE (L –type) are changed and acquire features similar to classical BSE when passaged in mice expressing SHEEP prions.
A carrier role for sheep in this transmutation of BSE is thus noted in variant strains of BSE. NATURALLY occurring inter-species transmissions involve sheep, goats, cattle and humans, as in the latter, vCJD.

(a) Please refer to the statements of Dr.Andy Carroll, DAFF ,the Chief Veterinary Surgeon of Australia,14th December Senate Hearing, RRA&T Pages 93,94,and 99 when he stated on page 93 that **" CWD OF DEER HAS BEEN AROUND FOR AN EXTREMELY LONG TIME."**

This is incorrect.
His incorrect statement could be seen as an attempt to suggest that if CWD has been around for an extremely long time and so why worry about it really?

The Hearing is referred to the journal "Emerging Infectious Diseases, Vol 10, No.6 June 2004 "Chronic Wasting Disease and Potential Transmission to Humans". Abstracts are attached with this article as **ATTACHMENT ONE** including that of the Nobel Laureate, Stanley B.Prusiner, of September 2009.

Elizabeth S.William's,group at,Dept.of Veterinary Sciences,University of Wyoming, and Dr.Prusiner's group, University of California are the world's leading prion and CWD researchers.

CWD was in fact first identified as a disease in the late 1960s in research facilities, in 1978 was recognised as a TSE BUT ONLY in 1981 was it first detected in the wild .

CWD is a TSE epidemic and highly contagious disease-90%affected in one farmed herd.It is transmissible orally to red deer which are farmed and more seriously wild in Australia in great numbers. CWD IS EXPERIMENTALLY TRANSMISSIBE TO PRIMATES BY I/C ROUTE 7/8 MONKEYS IN 3-4 YEARS BUT MUCH MORE SIGNIFICANTLY BY AN ORAL ROUTE IN 2 MONKEYS

(b) on Page 94, Dr. Carroll continues to re-present a role for the OIE which it does not have, in a less obvious way to the previous Australian Representative at the OIE and Chief Veterinary Officer of Australia, Dr.Gardner Murray .You are referred to his evidence at the Hearing February 2005 .

Dr.Carrol conveniently deflects DAFF responsibilities in the future, under the new policy framework,even in animal health, to the FSANZ categorisation scheme. FRANZ does not disclose the scientific basis on which these animal health issues are based, other than by Professor Matthews Review of TSEs.

The General Manager FSANZ and the recommended Dept of Health and Ageing contact are on holidays or interstate -27th January 2010 and are unavailable.

I SPOKE ON THE TELEPHONE TO ANDREW BARTOLOMEDEUS,GENERAL MANAGER RISK MANAGEMENT FSANZ. HIS STATEMENTS ABOUT THE PRESENCE or absence OF TSEs in AUSTRALIA SUGGEST THAT HE HAS MADE UP HIS MIND ALREADY OR THAT HE IS PARTY TO INFORMATION NOT MADE AVAILABE.There is no legal obligation for a country to report a disease outbreak or the presence of a disease.They may or may not.

WHEN ASKED TO PROVIDE THE SCIENTIFIC BASIS FOR HE NEW POLICY THE ONLY REFERENCE SUPPLIED WAS PROFESSOR MATHEWS REVIEW

The FSANZ questionnaires for countries which wish to import beef into Australia "have not been drafted yet but ring back at the end of February 2010." This may suggest a lack of accountability to come from FSANZ in the manner revealed by DAFF and particularly Bio-Security Animals (BA) in past years.

BA were the policy determiners for the Brazilian Beef imports in 2004, for the 2007 equine influenza policies and for the 2007 New Caledonian cattle tick fever scandal which is now costing Australia million

You are referred to the attached letter to the Hon. Tony Burke M.P. of the 22nd January2010 and to the attached EIA Inquiry's Counsel Assisting ,Outline of Submissions as **"ATTACHMENT TWO",** in which he is asked to explain Bio-Security Australia's science on vaccines in it's relationship to the other measures which were supposedly supporting the vaccine policies .

He has been asked to explain why the Chief Executive of Bio-Security Australia Mr. John Cahill, , was asked to withdraw from the Inquiry on the 21st February 2007,Pages 4014-4017—**"ATTACHMENT THREE"**.

This attachment is directly related to unresolved attempts by BA to justify it's policies on vaccines by additional measures at quarantine stations which are not revealed.

It is hoped that the Hon.Tony Burke will address the questions asked of him.

The expected response is complete silence. This would confirm DAFF culture of lack of accountability but it is at least better than the dishonesty shown by some DAFF executives at the Senate Hearings in 2005.

This EIA negligence by BA's policies cost Australia over 1billion dollars. AQIS as usual had to accept the blame for BA.

The Hearing should appreciate the limited Callinan Inquiry's terms of reference. The Beale examination has not revealed it's findings on this issue.

(c) Mr Morris's statement at 14th December 2009 Senate Hearing, page 97, **"IF AUSTRALIA HAD A SINGLE CASE OF BSE ANYWHERE THEN INTERNATIONAL CONSISTENCY AND NATIONAL TREATMENT UNDER THE WTO WOULD REQUIRE US TO REMOVE BEEF FROM THE SHELVES"** It would be appreciated if Mr. Morris could give a detailed explanation of what he means in layman's terms this afternoon.

This statement requires legal opinion from ANU's expert in international law Professor Rothwell to examine Australia's position in law for this Senate Hearing.

(d) on page 99, Dr Carroll states in discussing BSE that "this is not a disease that passes from animal to animal…"and we would be importing from controlled risk countries only **MUSCLE (tissue) MEAT** and when…".and in a later statement, " implementation of the current policy (he means the old policy} would make the negotiation process easier." if Australia has a single case of BSE."
This last statement needs explanation.
The first statement is scientifically wrong, the second is irrelevant to any scientifically based risk strategy to avoid introducing TSEs into Australia.
TSEs in muscle tissues and fat tissues are now recognised in animals.
BSE in cattle, sheep and goats has been shown to be transferable from animal to animal in milk to suckling offspring and to others via drinking milk IN CLINICALLY NORMAL CATTLE SHEEP AND GOATS.
Humans MAY BE infected by contact, with BSE prion containing products, such as cosmetics and leather goods BUT THIS FINDING IS UNDER REVIEW.
It will be surprising if direct animal infection is not further confirmed in the future.
Farmers are aware of the intimate contacts by licking etc, in herds .Tonsillar lymphoid crypts are favoured areas for BSE prions (PrP bse) so salivary secretions may be the first to be found to contain PrPbse.

Animal food safety in Australia is a national disgrace. The Hearing is referred to the EU standards, in place for many years, as an example for Australia's animal food safety in the future.
DAFF and the States Agriculture Departments have been unable to address these issues mainly because of DIRECT industry pressures on combined Government-Industry Committee decisions taken since 1990. REFERENCE APPENDIX 3
DAFF is only responsible in export areas for Australian animal food safety. Australia is the world's largest exporter of pet meats.

It has been revealed to DAFF that some exported pet meats have contained preservatives at levels that are life threatening to pet animals.
DAFF has refused to acknowledge that some frozen pet foods even contain these preservatives when notified of this.
Even the long awaited, 2004, "**Australian Standard for the Hygienic Production of Pet Food**" does not limit the amount of preservatives added but only states that labelling of preservative contained, must occur.
The veterinary profession has been calling for help to address these serious life-threatening animal food safety issues since 1989 without any help.

There IS NOT EVEN A VOLUNTARY GOVERNMENTAL BAN ON FEEDING KNACKERY OR FALLEN CATTLE OR CATTLE MBMs FROM ANY SOURCE, IN PET, POULTRY OR PIG FOODS IN AUSTRALIA. LABELS DO STATE THAT IT IS NOT PERMITED TO FEED THESE MBMs DIRECTLY TO RUMINANTS

The new policy for BSE starts on the 1st March 2010 but the Government assures that imported beef and beef products will not be fed to ruminants.
Does this exclude or include bans on it for pig, pet and poultry processed foods?
Feline TSEs are recognised worldwide.
The States have been held hostage by the pet food industries. Consumers have forfeited animal food safety to their agendas in exactly the same way as is occurring now by the beef processing and importing industries leaning on the Government to approve the new BSE policy.

Will the Government again fail the public in the human health issue, in the same way it has failed the Australian public in the animal safe food issue since 1989? The Governments, Federal, States and Territories must resist the direct pressures from powerful market entrepreneurs in both areas of food safety. Please refer to APPENDIX 3 to examine unsafe animal food in practice for 30 years.

(4) FSANZ 's Chief Executive, Mr. Steve McCutcheon has advised the Hearing, 14TH December 2009 that he envisages a "dedicated team of about 4 people" with one person from DAFF will undertake the risk assessments and in-country inspections "if they are necessary".

FSANZ's categorical risk assessments of importing countries will be supported by independent experts, input from the QIE (this is really internet reading of OIE recommendations.THE OIE'S BSE CHAPTER OF OIE TERRESTRIAL ANIMAL HEALTH CODE FOR RED MUSCLE MEAT. OIE guides by recommendation) and by "THE **DEVELOPMENT OF THE ACTUAL METHODOLOGY**." for these risk assessments.

DAFF has not even undertaken ANY risk assessment analysis on this new Australian BSE as of mid January according to information received from within DAFF.

The Hearing should be aware of concerns about the FSANZ framework, it's relationship to DAFF and about the most vital need to have people of the highest quality involved in these category risk assessment processes.
I express thanks to Professor Jim Bishop, for his letter of 20th January 2010, **ATTACHMENT FOUR** in response to my letter to him asking for his advice.
He has inspired my wife and myself for many years by his TV appearances, missed over last few years since he left for Melbourne. He inspires confidence.
With great respect, his Department of Health and Ageing is advised that tissues, such as TSE uncontaminated muscle meats and fats should no longer to be regarded as tissues free of TSE prions, however scrupulous the slaughter processes may be.
They can no longer be regarded as Category C (tissues of no detectable infectivity).
TSEs from the highly infectious and rapidly spreading TSE, CWD, have been found in both these tissues.

It must be now incorrect to state that these tissues may not contain rogue prions,PrP TSEs, and that they " do not inherently contain infectivity" as per his letter.

(5) **TSEs have now been detected in skeletal muscle tissues and in fat in animals, in deer with chronic wasting disease (CWD) ,the rapidly spreading TSE of USA and Canada and in PRIMATE SKELETAL MUSCLE AND FATTY TISSUES AND IN MUSCLES AND FATTY TISSUES OF MICE.**

The Australian Government SHOULD no longer suggest that there is no danger from the importation into Australia from skeletal muscle tissues and associated fatty tissues such as " meat" and " meat products" from TSEs rogue prions, from the 32 countries described as "conditional risk assessment" by the OIE for any TSEs,such as B.S.E..

This detection of rogue prions of CWD in skeletal meats and fats in sub-clinically affected CWD deer, is an important first ever detection in these tissues for TSEs.

Furthermore, faecal excretion of CWD rogue prions has been identified from sub-clinically affected CWD deer and this finding has further explained the incredible infectivity of this TSE and it's rapid spread within the North American Continent.

Bovine Spongiform Encephalopathy (B.S.E.) multiple strains have now been identified to have all 3 ecological properties of other TSEs --- ie to have spontaneous, heritable and infective transmissions.

These modes of transmission are varied between the variant and multiple BSE strains recently identified.

Within the cattle genome, molecular differences IN THE PROTEIN PRION GENE ARE BEING IDENTIFIED AS THERE ARE ALLELES (DIFFERENT MOLECULAR VARIATIONS) OF THIS GENE. THESE ARE NOW BEING EXAMINED to explain genetic sensitivities to BSE in cattle.

This technology is more advanced in sheep where the largest genetic selection ever undertaken in the world is using PrP genetics to find those sheep which have the greatest resistance to Scrapie.

It is possible that the same approach for BSE in all ruminants will be necessary.

BSE infection occurs naturally in cattle, sheep and goats.

New variant strains of BSE in cattle have been identified in most European countries, the United States and Japan in the last 3 years.

Some of these BSE strains are more virulent with varying incubation periods, both in natural infections and when induced experimentally in other animals.

Natural BSE infection occurs through ingestion of milk in BSE cattle and in other animals with BSE such as sheep and goats.

Similarly, Scrapie is found in milk of sub-clinical diseased sheep and goats with Scrapie.

Milk products from sub-clinical and normal looking BSE infected cattle,sheep and goats must therefore present an unquantified public health risk.

Direct BSE infection by direct contact, as per licking each other has not been identified as yet.

Sub-clinical BSE disease in apparently normal BSE cattle has been identified by the TSE "rapid" diagnostic testing in MOST European countries.

These "rapid" TSE diagnostic tests are approved (by the European Commission,the Panel on Biological Hazards), only for post-mortem testing and are for BSE ,Scrapie and Atypical Scrapie strains and CWD but not for TSEs such as in mink.

Rapid tests take a minimum of 24 hours for completion.

There are no approved "rapid" tests for live animals.

7000 new cases of BSE in cattle were detected by these"rapid"test after the screening of 50 million cattle in Europe.

These new 7000 cases of BSE in cattle had not been anticipated nor detected by clinical examination, either by Governmental veterinary surgeons or by owners of these diseased BSE cattle in Europe.

This suggests the very minor role of importance for clinical surveillance in BSE, in any FSANZ risk assessment process in cattle prior to slaughter.

PLEASE REFER TO THE OIE BSE CHAPTER OF THE OIE TERRESTIALANIMAL CODE FOR RED MEAT show THAT ANIMALS FROM 30 MONTHS OR LESS , with SLAUGHTER HVGIENE MEASURES TO AVOID SPECIFIED RISK MATERIALS (SRMs) BUT there is NO BSE TESTING AT ALL OF ANIMALS BRAINS(where the highest concentration of rogue proteins are found) OF CATTLE FROM WHICH THE MEAT IS COMING!!! --- EXCEPT ----------

EXCEPT IF THE ANIMALS COME FROM A BSE AFFECTED COHORT IN THE COUNTRY.----------

WITHOUT IDENTIFICATION OF ANIMALS FROM BIRTH AND WITHOUT FULL COUNTRY OF ORIGIN LEGISLATION AND WITHOUT THE NEW TECKNOLOGY USING SPMCA AND POSSIBLY LOOK ALIKE THECKNOLOGY OF THE FRENCH "AMORFIX" IN THE FUTURE.

THIS LACK OF TESTING REQUIREMENT IS UNBELIEVABLE RISK FOR AUSTRALIA WHICH HAS NO REPORTED TSEs TO DATE.

In December 2009, the Community Reference Laboratory of the European Commission released a vital first report--- "Scientific Opinion on Analytical Sensitivity of Approved TSEs Rapid Tests".

As a result of this first time ever comparative analysis, some approved rapid tests "cannot be recommended for the monitoring of BSE in cattle and the TSE in small ruminants in the EU".

Thus there is a possible under reporting, from the above finding, and that the number of sub-clinical apparently normal cattle with BSE may be unreported in the EU because of faulty testing. It may be insignificant!!!

BSE cases in cattle with the BSE variant strains have prolonged incubation periods before clinical expression occurs and this is important in dogmatic time scheduling of BSE forecasting.

OF MAJOR CONCERNS TO AUSTRALIAN AGRICULTURE ARE THE FOLLOWING FACTS.

Atypical cattle isolates of BSE— for example the BSE (L-type), in transmission experiments were changed and acquired strain features similar to the classical BSE agent when propagated in mice expressing SHEEP prions.

Furthermore, in 2009, chronic wasting disease (CWD) prions in elk and deer of Northern America and Canada have been found to transmit directly to SHEEP in transgenic mice. Ref. J.Gen.Virol. 2009 Ap 90(Pt4) :1035-47.
THIS IS ABSENT FROM TABLE I, PAGE 27 of Professor Mathews Review, under the heading –"Experimental Transmission to"

This is considered unusual as this finding is of great epidemiological significance and very significant for animal and human health.

Furthermore prions from BSE cattle did not transmit directly in transgenic mice expressing the elk (Tg Elk PrP) gene but DID IN FACT transmit after being passaged through SHEEP.

Professor Mathews DOES MENTION, PAGE22, the following:-
Prions of BSE in cattle, PrPbse, did transmit directly in mice expressing pig PrP rogue prions but were MORE SUSCEPTIBLE(shorter incubation period) after passage through SHEEP.

He mentions that there is NO Australian voluntary or enforced ban on feeding knackery or ruminant MBM to pigs but assumes remote risk to human health with the new policy!
LABELLING OF PIG FOODS IN AUSTRALIA DISCLOSE THAT THEY CONTAIN PROTEINS DERIVED FROM RUMINANTS AND THAT THEY SHOULD NOT BE FED TO RUMINANTS.
HOST ENCODED PRION PROTEINS FROM CATTLE,SHEEP AND GOAT AND OTHER ANIMALS SUCH AS HORSE CAMEL KANGAROO MEAT MEALS DO NOW ENTER THE HUMAN FOOD CHAIN AFTER PASSAGE THROUGH PIGS AND POULTRY IN THIS WAY.
UNDER THE NEW POLICY IT IS VERY POSSIBLE MISFOLDED PRION PROTEINS IE ROGUE PROTEINS WILL BE ADDED TO THE HUMAN FOOD CHAIN VIA THIS ROUTE UNDER THE NEW POLICY
THE REASON-- IS THAT MOST COUNTRIES HAVE NOT RAPID TESTED ALL THEIR HERDS OF CATTLE AND THAT THE SPMCA TECHNOLOGY FOR DETECTION OF MINUTE AMOUNTS OF PrP SC IS NOT USED.
SPMCA TECHNOLOGY CAN PICK UP MINUTE AMOUNTS OF ROGUE PROTEIN IN TISSUES THAT WERE PREVIOUSLY CONSIDERED TO BE FREE AND IN THE ENVIRONMENT

LOOK AT THE NUMBER OF BSE cases 7000 in Europe detected by rapid testing and refer to the faults disclosed in rapid testing in deer with CWD that we infecting pastures with CWD FOR A YEAR BEFORE TEST RESULTS WERE POSITIVE,
FOR PROFESSOR MATHEWS TO SAY THAT THIS RISK OF FEEDING RUMINANT MBM TO SHEEP GOATS AND CATTLE AFTER PASSAGE THROUGH PIGS AND POULTRY TO HUMANS IS UNUSUAL FOR A SCIENTIST AS THE PEL PRINCIPLES OF SCIENCE SEEM TO BE UNDER ATTACK.
HE ASSUMES REMOTE RISK TO HEALTH UNDER THE OLD POLICY AND UNDER THE NEW POLICY!!!!! He seems not differentiate between them.
PIGS ARE MORE SENSITIVE TO BSE AFTER PASSAGE THROUGH SHEEP.
IT IS FELT THAT PROFESSOR MATTHEWS REVIEW DOES NOT CONSIDER THE EXPERIMENTAL INTER-SPECIES TRANSMISSIONS SATISFACTORILY INVOLVING PRIMATES
SHEEP,CATTLE,DEER ,GOATS AND OTHER ANIMALS IN NATURE.

Surely these facts are warning signs of the possible dangers at least to our sheep industries in the future 50 years from inter –species transmissions!!

Australian sheep industries are TSEs free ,the only country now in the world. Australia is blessed and this blessing is due to the great work by DAFF in the past.

The conversion of human prion protein by CWD associated rogue prions has been demonstrated in in-vitro cell free experiment.

The conversion of human PrPC by Scapie and BSE PrP-res have been shown to be similar to the conversion of human prion protein by Cervid PrPcwd associated prions.

These are incredibly important findings in themselves and bring these 3 TSEs together as very real increasing dangers to human health in the future.

These are very important findings in TSE inter-species transmission experiments and demonstrate the serious dangers to Australia from trading with countries affected by TSEs such as Scrapie, CWD and BSE with all their newly discovered variant forms, which Professor Mathews fails to detail in his Review.

Attempts to eradicate CWD have failed and it is spreading rapidly in 14 States of the USA and 2 provinces of Canada despite expensive and exhaustive programs.
13 CATTLE were infected with CWD, following intra-cerebral inoculation with 10/13 affected after 5.5 YEARS ON FIRST PASSAGE,but in another experiment 100% INFECTION occurred AFTERa 2ND PASSAGE.

In England there are an estimated 5000-10000 new cases of Scrapie each year despite the huge amount of money spent each year by the British Government.
Scrapie like CWD is highly infectious with almost eternal survival in the environment.

For example Scrapie containing material when rubbed on broken skin of mice, has the same efficiency in inoculation as if it had been injected by intravenous or subcutaneous injections.

Please note that there is an available rapid tests of the TSE, CWD, for beef and beef products coming from the USA or Canada.

Many of the 32 countries have NOT undertaken any "rapid"testing of any clinically normal but sub-clinical diseased BSE cases in bovines from either the classical strain BSE cases or the newly discovered BSE variant strains.

(6) **Please refer to Professor John Mathew's submission for the Australian Government .**

"Review of Scientific Evidence to Inform Australian Policy on Transmissible Spongiform Encephalopathies".

This review is the scientific basis for the new policy to allow importation of meat and meat products(?) from the 32 countries classified by OIE as "controlled risk assessment".

The review has been examined and approved of by the National Medical and Research Council of Australia and it's TSE Council, the Department of Health and Ageing, the Therapeutic Goods Administration, FSANZ ,DAFF and the BSE Advisory Council.

This document is disturbing and inaccurate in details of great importance, Many of the conclusions presented need to examined for their validity in the light of present knowledge and with better considerations of emerging knowledge. Disparate facts on inter-species transmissions experiments are coming each month from primary researchers on TSE diseases. Reference Sept 2009 Dr.S. Prusiner.

Surely new confirmed findings with their suspicions must be included in considerations and be anticipated but acknowledged as possible facts-to-be.

Please find attached as ATTACHMENT FIVE, an extract from Lisa Waddell's seminal article.

The Methodological Soundness of Literature Reviews Addressing Three Zoonotic Public Health Issues.

Please note that most reviews of TSEs failed to validate the conclusions drawn by the reviewer of TSE scientific literature.

"a" The "active surveillance" group in Professor Mathews Review Page 34, Fig.1, chart 85 is presented in red, and graphs those cases detected in normal animals with BSE ,only after rapid TSE testing in the EU from mid 1999 onwards to only mid 2006.

This is surely an indication for Australian Authorities, by analogy, of the real extent and level of BSE infection in those countries which have (1) never tested for BSE and TSEs or (2) have just started testing or (3)have not competed testing of their herds .

Thus there is the implicit suspicion that the number of BSE cases notified by these 32 countries to the OIE may not in any way represent the number of BSE cases actually present in these countries, which do not test or have just commenced testing.

The number of unrecognised BSE cases in these other countries which have not progressed as far as Europe in their testing would be grossly unreported and may be comparable (ie they may be proportionately comparable, relative to their number of clinically recognised cases of Mad Cow Disease), RELATIVE to the number disclosed by these tests in the EU in Chart 85.
Please note that there is no recommendation to report the number of BSE CASES TO THE OIE AT ALL.

Please refer to Fig 3, "Bovine Infective Units(ID50) Entering the UK Chain by Year" where it is disclosed that infective BSE material coming into the food chain from 1997 to 2005 from animals under 30 months follows exactly the total unit line, because only cattle <30 months were allowed to enter the food chain.
Please advise Professor Mathews's source of reference for that proportion of units added for this <30 months group from 1983 to 1997. IS THIS A BACK CALCULATING MODELLING,THE R(O) APPPROACH AND AGE PERIOD-COHORT MODELS –OR IS A REAL CALCULATION BUT .IF IT WAS A BACK CALCULATING MODELLING WHY NOT ADVISE THE READER OF THE REVIEW?
What value will an > 30 months slaughter ban policy have in any of the 32 countries who are planning to import WITH THE HERITABLE NATURE OF BSE NOW REVEALED AND WITH THE NEW STRAINS OF BSE DISCOVERED WHICH MAY HAVE DIFFERENT ORIGINS THAN CATTLE?
Surely this should be explained country by country, after FSANZ has supplied the details on levels BSE BY rapid testing, the numbers of tests completed with the total herd numbers in these OIE 32 controlled risk countries.

What is the position in France particularly, re Bovine ID50 units entering their food? THEIR LEVELS OF v CJD HAS NOT FALLEN IN THE LAST 3 YEARS MENTIONED.

15,141 U.K. cases of bovine BSE were recorded in the U.K.from 1997 until 2009 with 7000 odd in Europe.

The Hearing should be aware that there is not even a voluntary ban in Australia, let alone a mandatory one, on feeding ruminant MBM to other animals (pets) including domestic animals(poultry and poultry),the latter DO ENTER the human food chain in poultry and pig tissues. Imagine the industry pressure resistance and how Government acceded to industry pressures in animal food safety in the past!

Professor Mathews's Review Page22 describes as " hypothetical" the nature of risk of BSE amplification cycles in pigs(they are more BSE sensitive after passage through SHEEP!!!) . Mice expressing Pig PrP are resistant to classical Scrapie but may be sensitive to Atypical Scrapie .There is no mention of BSE L type transmutation to classical BSE in mice expressing SHEEP prions.

Evolutionary molecular biologists depend on precedents but in prions diseases there are few. Exquisite prion sensitive animal models are necessary- as in mice.

Science depends on inter-species transmission experimentation and recognition of animal husbandry practices.

Please note after oral infection by PrP BSE prions in vCJD., the average incubation period is 16-17 years ,so it is only after 2014 that the main danger period will pass for vCJD in the UK if no further Bovine infective units had entered the food chain after 1997.

With heritable transmission recognised in BSE, additional units will be added,

However infective units are stated in Fig 3 continue to be added to the food chain until at least 2004, so the main danger period for v CJD will pass later than 2020.

Heritable transmission occurs in CJD and delayed expression in humans with varied alleles of the prion protein gene are expected.

(b) Many facts of science which imply great risks to Australia, particularly in the area of risks to animal health are not mentioned at all, let alone quantified by Professor Mathews..

For example, it is beyond belief that this Review wrongly states on, page 27 Table I under the heading

"**SUPPORTING INFORMATION & RISK ASSESSMENT**" which does suggests matters of importance.

Table1 " **IMPORTANT TSEs and THEIR CHARACTERISTICS**".

Scrapie –usual transmission— ""Spontaneous" possibly by milk"

CWD (deer and elk)---usual transmission "Spontaneous".

This indicates to a reader that Professor Mathews believes that in Scrapie and CWD, the usual mode of transmission is spontaneous.

THIS IS INCORRECT.

Please also refer to Page 9,**BACKGROUND AND HISTORY** ,section 28, in which he states that " Scrapie has been transmitted to mice and other experimental animals in the laboratory."

Professor Mathews omits Scrapie experimental infection to cattle, a domestic animal which has carried BSE to cause v CJD .

ALL 9 CATTLE SUCCUMBED EASILY TO SCRAPIE BUT ON SECOND PASSAGE MORE QUICKLY STILL IN 14-18 MONTHS.

Scrapie and CWD are highly infectious TSEs with almost eternal survival properties in the environment outside the host animals.

These TSEs, and BSE itself, have all three transmission pathways ie spontaneous, heritable and infective transmission.

Spontaneous or sporadic cases of Scrapie and CWD are of unknown origin but may be expected particularly in the genetically susceptible .

Inherited cases of BSE, CWD and Scrapie do occur.

Scrapie and CWD transmission is incredibly infective.

Pastures remain infective for Scrapie and CWD for many years to grazing sheep and deer species.

Direct animal to animal transmission occurs in Scrapie and CWD via body fluids such as saliva, milk, AND URINE IN NORMAL ANIMALS FOR ONE YEAR PRIOR TO BEING CONFIRMED TO BE DISEASED AND WHICH HAVE TESTED NEGATIVELY TO RAPID TESTING. AT POSTMORTEM

In Table I BSE's "Experiment transmission to"heading, are listed "mice ,sheep and goats" but Professor Mathews makes no mention in the Review of the following important "back -cross" information from sheep to BSE in cattle.

This information is about the changes that occur when Atypical cattle isolates of BSE acquire strain features similar to the classical BSE agent when propagated in mice expressing SHEEP prions.

Professor Mathews does not mention The **EXPERIMENTAL TRANSMISSION OF SCRAPIE TO CATTLE** in Table I under "Experimental transmission to".

It is not mentioned at all and not mentioned even in the Review.

All of a group of nine cattle infected intra-cerebrally with Scrapie prions succumbed easily to Scrapie. WHY DOES PROFESSOR MATHEWS NOT CONSIDER WHAT HAPPENS AFTER MULTIPLE PASSAGES AS AFTER JUST 2 PASSAGES ALL CATTLE DIED FROM SCRAPIE AT 14-18MONTHS OF AGE WHICH IS FASTER THAN BSE KILLS IN CATTLE.!!!!!!

Surely this is evidence of great importance for Australian animal and human health and **IS MISSING UNDER THE APPROPRIATE HEADING** in Table I and in the Review.

There is no mention in the Review that cattle BSE prions did not transmit directly in transgenic mice expressing the Elk gene (Tg Elk PrP) but did transmit after being passaged through SHEEP.

Why was this vital epidemiological information missing on this sheep carrier role in BSE with CWD programmed mice?

Table I for CWD, under heading "Experimental transmission to" the CWD TRANSMISSION TO CATTLE IS MISSING FROM THE APPROPRIATE HEADING AND IS MISSING FROM THE REVIEW, with the implications for animal –human health again missing from the Review itself.

ON FIRST PASSAGE 38% WERE INFECTED ON FIRST PASSAGE IN 5.5YEARS BUT ON SECOND PASSAGE 100 %DEVELOPED WEIGHT LOSS AN D CLINICAL DISEASE IN 16.5 MONTHS.

Table I for CWD again, under heading "Experimental Transmissions to" lists only ferrets ,monkeys and goats.

In fact predators COULD BE found to be infected NATURALLY BUT EXPERIMENTAL INFECTION HAS BEEN CONFIRMED IN RACCOONS FIELD MICE AND VOLES and many scavengers let alone predators which abound in the wild. Raccoons are being used to experimentally differentiate TSEs by the varied incubation periods in this animal.

There is no mention at all of the vital finding that Prp BSE ie Bovine Spongiform Encephalopathy itself, did not transmit directly in transgenic mice expressing the elk gene (Tg Elk Prp) but **DID IN FACT TRANSMIT AFTER BEING PASSAGED THROUGH SHEEP.**
REFERENCE , 'Sakaguchi et al (2009)" PAGE 6 DISCUSSING SECONDARY TRANSMISSION WITH THE SENTENCE ENDING "COULD ONLY EMERGE AFTER A VERY MUCH LONGER INCUBATION PERIOD ",IF AT ALL". AS THIS AUTHOR IS A FAMOUS PRIMARY RESEARCHER AND MAY NOT HAVE USED ANY TERMS IN ANY WAY SIMILAR TO THIS "IF AT ALL". Inter-species transmissions, both experimental and naturally occurring are important to human –animal health and may hopefully anticipate and try to explain past, present and future evolutionary molecular changes in nature with the past and future animal husbandry practices of man.

. Professor Mathews fails to mention the enormous significance to human health of the experimental ORAL CWD transmission to primates-monkeys in his Review but it is at least it is stated in Table I as "Experimental transmission to" .

FOR PROFESSOR MATHEWS TO SAY THAT THE RISKS TO HUMAN HEALTH FROM FEEDING RUMINANT MBM AFTER PASSAGE THROUGH PIG AND POULTRY TO HUMANS IS REMOTE IS BEYOND BELIEF AND DEFIES ALL PEL PRINCIPLES IN SCIENCE.
HE SHOULD KNOW OR HAVE BEEN ADVISED OF THE OIE RECOMMENDATIONS FOR THE 32 "CONTROLLED RISK ASSESSMENT" COUNTRIES DO NOT CONSIDER ANY RAPID TESTING OF SLAUGHTERED CATTLE OR ANY SOPHISTICATED DETECTION FOR DETECTING MISFOLDED PRIONS SUCH AS PER SPMCA.
UNDER THE NEW POLICY FOR BSE THIS IS EVEN MORE THAN NECESSARY.
PLEASE LOOK AT THE QIE TERRESTRIAL CODE FOR THEIR PALLID RECOMMENDATIONS WHICH ALMOST ASSUMES THAT BSE AND TSES WILL NOT BE PRESENT IN MEATS. ONLY IN COHORTS FROMBSE CATTLE IS RAPID TESTING RECOMMENDED BY THE OIE!!!!
This may be ok when both importing and exporting counties have had BSE but we have not and have no TSEs at all.
"BSE CHAPTER OF THE OIE TERRESTRIAL ANIMAL HEALTH CODE

1.1 RED MEAT—APART FROM HYGIENE AND SLAUGHTER REQUIREMENTS TO AVOID SPECIFIED RISK MATERIALS (SRMs), AM LIVE ANIMAL AND PM INSPECTIONS,ANY COUNTRY SHOULD BE ALLOWED TO TRADE RED MEATAS LONG AS IT CAN MEET THESE pallid CONDITIONS REGARDLESS OF ITS BSE STATUS .

1.2 REGARDLESS OF THE BSE STATUS OF A COUNTRY-IT IS SPECIFICALLY STATED AS SUCH, SO THIS MEANS THAT IT DOES NOT MATTER HOW MUCH ROGUE PROTEIN IS INTRODUCED INTO AUSTRALIA.

1.3 WE WILL NOT BE FREE OF TSEs FOR MUCH LONGER.

It is felt that Professor Mathews Review should be referred. Sections of interest to a reader include(76) where oral BSE infection by the monogastric suckling calf via maternal colostrum, then via milk and then in artifical calf pen feeding with milk or milk derived supplements it is WRONG to say that BSE "CAN BE OCCASIONALLY TRANSMITTED".IT IS NO LONGER "EQUIVOCAL" BUT FACT. BSE PRIONS ARE REPORTED TO BE FOUND IN MILK OF CLINICALLY NORMAL CATTLE AND GOATS WITH NO SYMPTOMS OF ANY DISEASE AT ALL.
SECTIONS SUCH AS 77,78,80,AND THE lack of appreciation of SIGNIFICANCE OF SECTON102 ARE OF INTEREST AS MUCH AS THE ERRORS AND lack of appreciation of the SIGNIFICANCE OF THESE ERRORS IN THE INTER-SPECIESEXPERIMENTAL TRANSMISSIONS of TABLE I.The lack of appreciation of the significance of RISK TO AUSTRALIA BY the red or active surveillance group in Fig chart 85,(in those countries which have not advanced as far as the EU in revealing the true extent of BSE IN THEIR HERDS BY RAPID TESTING).THERE IS NO STATEMENT IN THE REVIEW THAT SOME OF THESE TESTING SYSTEMS HAVE BEEN FOUND TO BE FAULTY.

It is suggested that reference be sought from the 1997 Nobel Laureate (for his pioneering work on prions), Dr. Stanley B .Prusiner,MD, or from his associates at the Prusiner Lab.The Institute of Neurodegenerative Diseases The University of California San Francisco CA 94 143, USA tel---(415) 476 -9000.

Dr. Prusiser is actually working on CWD now and the Hearing is referred to Jennifer O'Brien who was the internet source to him. jobrien@pubaff.ucef.edu 415 476 2557.
An Australian world expert on TSE diseases is Professor Simon Hawke at the Brain and Mind Research Institute, Prince Alfred Hospital, Sydney 02 93510730.

SUMMARY:

(8) Any errors in Australia's decision on the new policy on BSE may not be noticed for many years (10-70years!), quite unlike the immediacy of F&M disease outbreak.

Every month, new information becomes available which seems to increase the risks to animal health in Australia ,from this new policy
This new policy ignores the advances in science and is not supported by the Review which needs expert appraisal to examine it's validity and the validity of it's conclusions.

The new policy will cost Australia it's unique position of freedom from all TSEs and increases our risks of introducing Foot and Mouth Disease.
Trading with the 32 countries which are contiguous to, are not free in every way from this disease, particularly from Foot and Mouth vaccinating countries, or have OIE zonings purporting to be enforced even with supposed strict border control for domestic and wild animals, should not be considered by DAFF and later by FSANZ.

Skeletal muscle tissues and fat tissues from domestic animals should no longer be regarded as inherently TSE safe tissues- Category C tissues.

Skeletal muscle tissues and fat tissues from sheep from New Zealand should no longer be classified as Category C tissues following the Atypical Scrapie detection in New Zealand.

Skeletal muscle tissues and fat tissues from cattle from any country with an OIE classification of "controlled risk assessment"(they have declared BSE in the past) should no longer be classified as Category C tissues.

Australia needs legal advice from an expert in international law, such as Professor Rothwell at the ANU, to examine statements made by FSANZ which suggest penalty and exclusion under WHO trade accords.

Please refer to APPENDIX 1---WHERE CHALLENGE BY MEXICO/CANADA is reported to the COUNTRY OF ORIGIN US LEGISLATION. PLEASE ALSO NOTE THAT THE US HAS NO ANIMAL IDENTIFICATION AND NOTE THE CHANGE IN OPINION OF THE USDA TO ORIGIN LABELLING ON MEAT.

It should make us very proud to be world leaders by our, from birth N.L.I.S.

Australia should only depend on it's own staff, stationed permanently at overseas abattoirs to supervise ante-mortem **IDENTIFICATIONS BY N.L.I.S. FROM BIRTH**, their clinical examinations, the slaughtering and processing to avoid TSE contamination of meat and finally it's quarantining to avoid substitution.

AUSTRALIAN AUTHORITIES MUST REQUIRE ALL CATTLE WHOSE MEAT IS FOR IMPORTATION INTO AUSTRALIA, TO BE "RAPID" TESTED FOR TSEs, BSE AND SMALL RUMINANT TSEs AT THE TIME OF SLAUGHTER AND BEFORE THEIR MEAT IS IMPORTED INTO AUSTRALIA USING ONLY AN APPROVED RAPID TEST WHICH MEETS THE FUTURE LEGISLATIVE 2010 EU CRITERIONS FOR SENSITIVITY FOR DETECTION OF BSE AND SMALL RUMINANT TSEs . THESE TESTS SHOULD BE PERFORMED BY AUSTRALIAN AUTHORITIES ON SITE THEMSELVES. ANIMALS SHOULD BE LESS THAN 30 MONTHS OF AGE. PLEASE REFER TO APPENDIX 2 –ARTICLE 11.6.2. OIE TERRESTRIAL ANIMAL CODE.

HAS BIO-SECURITY AUSTRALIA CONDUCTED THE ANNUAL RISK ASSESSMENT UNDER THE OLD POLICY as advised in this OIE article? UNDER a Release assessment-HAS BA ADVISED THAT THE RISKS OF INTRODUCING BSE TO THE HUMAN FOOD CHAIN WILL INCREASE AS A RESULT OF UNTESTED(IMMUNOHISTOCHEMISTRY –RAPID TESTS) SKELETAL AND FAT TISSUES WHICH SHOULD NO LONGER BE CLASSIFED AT CATEGORY C tissues by the OIE FROM COUNTRIES HAVING UNKNOWN OR UNDECLARED LEVELS OF PrpSC OF BOVINE SPONGIFORM ENCEPHALOPATHY IN THESE IMPORTED TISSUES.

Under b Exposure assessment of the OIE ARTICLE ,HAS BA NOTIFIED THE OIE THAT RUMINANT MBM ENTERS THE HUMAN FOOD CHAIN VIA PASSAGE FIRSTLY THROUGH PIG FOODS WHERE PIG MEATS ARE LATER SOLD TO HUMANS.

Pig rogue prions are in fact amplified by passage through sheep.

THESE RUMINANT MBMs ARE FOUND IN PET AND POULTRY FOODS DIRECTLY AND WITH THE NEW POLICY ON BSE MAY REPRESENT A RISK TO ANIMALS SUCH AS CATS DIRECTLY.

The serious immediate threat to Australian sheep industries with the more distant implications for human health, as described here-in, is revealed by the disclosure of Atypical Scrapie in N.Z.

Atypical Scrapie is more infectious than the conventional Scrapie and thus much more difficult to control.

Due to the vigilance of DAFF and State Departments of Agriculture, over an extremely long time, Australia is now unique in the world, being free of all animal TSEs.

This new policy threatens this freedom from animal prion diseases in the future.

Aypical Scrapie immediately threatens our sheep and cattle industries in 2010.

Dr. Stanley Prusiner,9th Sept.2009, describing the significance of his work on CWD:-

"OUR WORK(on CWD) MAY ALSO EXPLAIN THE TRANSMISSION OF SCRAPIE PRIONS AMOUNG SHEEP AND GOATS"—Attachment one.

Using evolutionary molecular biology on CWD and it's new strains,he is working back to the beginning of prion disease –Scrapie.

Robert Steel

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

SUPPORTING INFORMATION & RISK ASSESSMENTS

TABLE 1. Important TSEs and their characteristics

TSE (host)	Clinical features	Usual transmission	Mean incubation period	Experimental transmission to
Scrapie (sheep)	Behavioural disorder & ataxia	"Spontaneous" – possibly by milk	Usually more than 6 months	Goats, mice and other species
BSE	Behavioural disorder & ataxia	Bovine meat and bone meal to calves	5 years (range 2-10)	Mice, sheep, goats
CWD[67] **(deer & elk)**	Behavioural disorder & ataxia	"Spontaneous"	Several years	Ferrets, monkeys, goats
HUMAN				
Kuru	Ataxia & terminal dementia in Fore	Oral (cannibalism)	10-12 yrs (range 4-40)	Primates & others
vCJD ('human BSE")	Behavioural disorder & dementia in younger persons	Oral (BSE contaminated food)	16-17 years (range 4-30?)	Humanised mice and other species[68]
Sporadic CJD	Dementia in an older person	"Spontaneous"	?	Primates and other species
Iatrogenic CJD	Progressive dementia (usually in adults)	Grafts of cornea or dura mater or hormones or instruments contaminated with CJD material	Range 1.5 - 30 years	Primates and other species
Familial CJD	Progressive dementia	Associated with inherited mutations in PrP genes[69]	?Lifetime	Primates and other species

[68] Inadvertent secondary transmission to humans by blood transfusion, with a mean incubation period of perhaps 7-8 years.

[69] Other mutations in PrP genes cause other familial neurological disorders such as GSS or FFI.

Fig. 1. BSE surveillance in UK and rest of EU by year

Chart B4: Evolution of BSE cases detected by passive surveillance and active monitoring in the UK

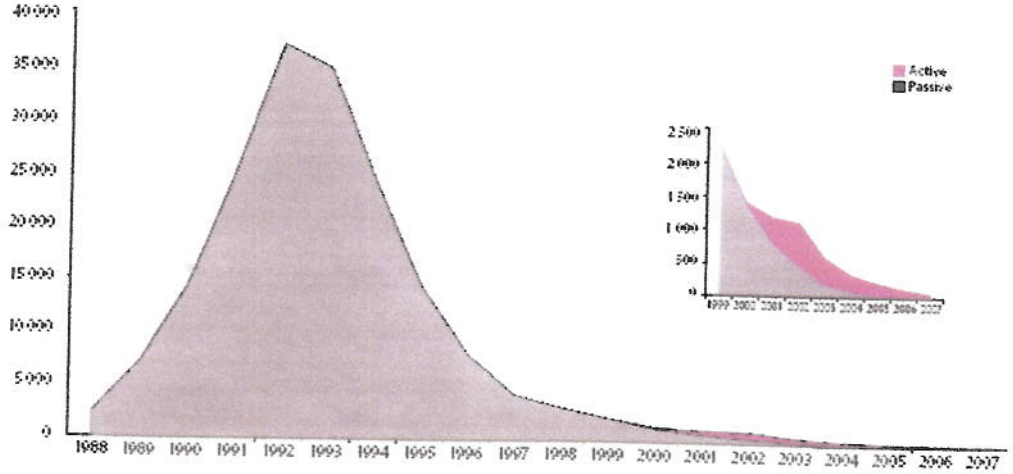
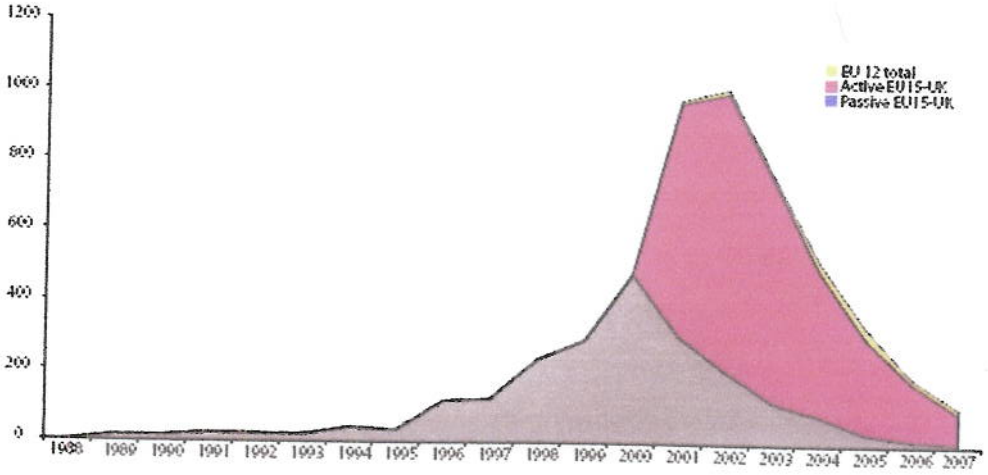
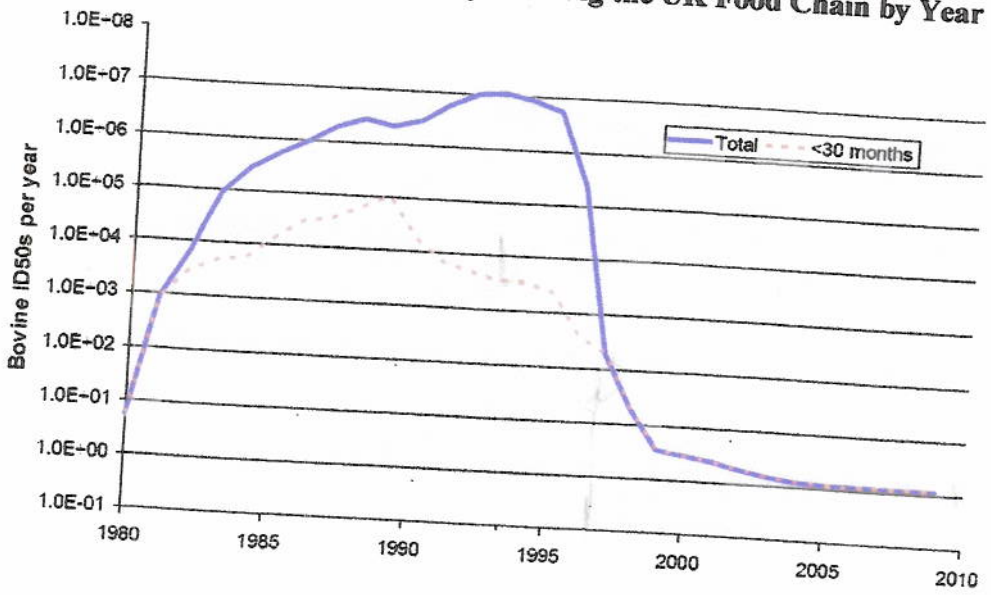


Chart B5: Evolution of BSE cases detected by passive surveillance and active monitoring in the rest of the EU



From the *"Report on the monitoring and testing of ruminants for the presence of TSE in the EU in 2007"*. See http://ec.europa.eu

Fig. 3. Bovine Infective Units (ID50) Entering the UK Food Chain by Year



Note the logarithmic scale of doses, so that the amount of infective material entering the food chain in 1993 is about 10 million times greater than in 2006. The dotted red line shows the contribution from younger animals (less than 30 months at slaughter), and the gap between that line and the top blue line shows the contribution from older animals. As older animals were removed from the food chain by 1997, the lines converge in later years. From Comer and Huntly (2003).

ATTACHMENT SIX

Australian Government
Department of Agriculture, Fisheries and Forestry

PLEASE NOTE RUMINANT MEALS DO ENTER THE HUMAN FOOD CHAIN AFTER PASSAGE THROUGH PIGS & POULTRY VIA RUMINANT MEAT MEALS FED TO THESE 2 MONOGASTRIC ANIMALS

20 January 2010

Dr Bob Steel BVSc MRCVS
8 Martin Rd
CENTENNIAL PARK NSW 2021

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

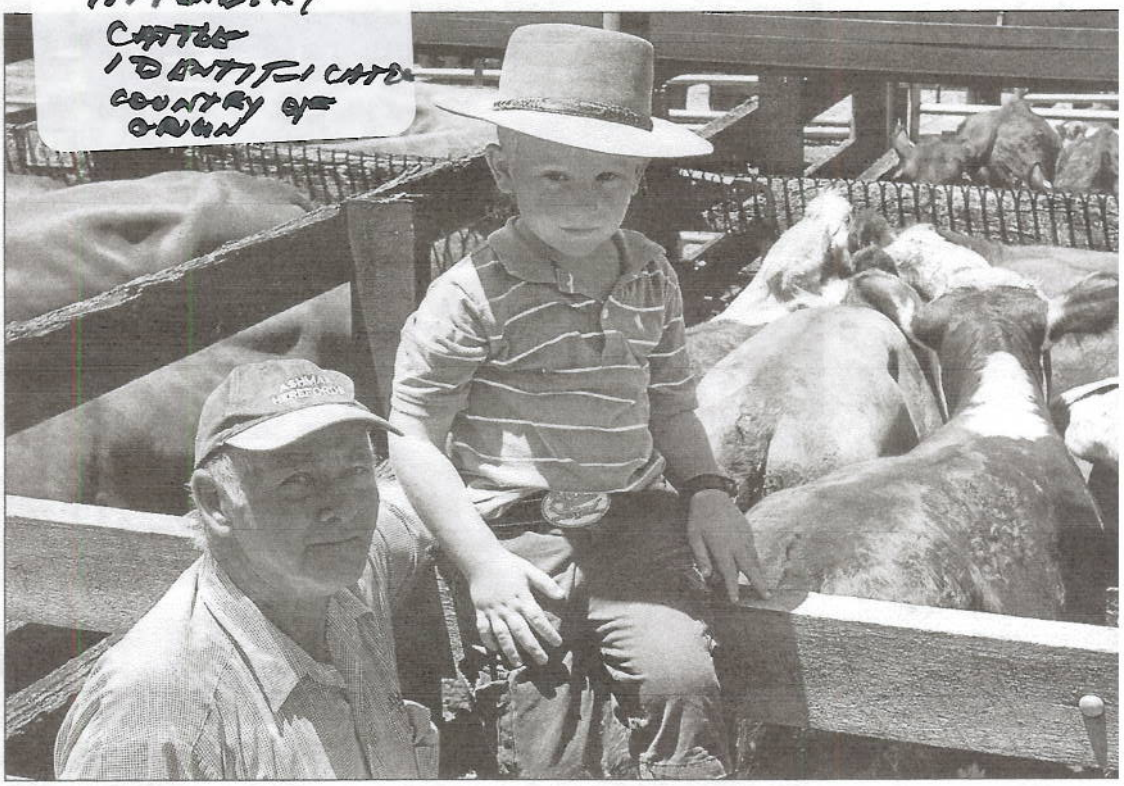
18 Marcus Clarke Street Canberra City ACT GPO Box 858 Canberra ACT 2601 ph +61 2 6272 3933 fax +61 6272 3008 www.daff.gov.au ABN 24 113 085 695

US faces hurdles over introduction of cattle identification

US MEAT MATTERS

Steve Kay

APPENDIX I
CATTLE IDENTIFICATION COUNTRY OF ORIGIN



Ian Hollingsworth, "Ashmar", Gloucester, with his grandson, Julius, sold two pens of European Union-accredited Hereford steers for $635 at last Thursday's store cattle sale at Gloucester. Full report, p98.

THE US cattle industry is still debating the merits of a national cattle identification system and country of origin labelling (commonly known as COOL) for meat and food products.

Both subjects were first mooted in the mid-1990s and have been the subject of controversy ever since.

Mandatory COOL was finally introduced on September 30, 2008, but a comprehensive national ID system is still a long way from being developed.

The irony is some US cattlemen vociferously supported mandatory COOL but equally strongly opposed mandatory ID.

Nothing in my years of writing about the livestock industry worldwide has flummoxed me more than this contradiction.

Even worse, COOL has so far only added cost to the beef production system and provided no benefits.

Conversely, a national cattle ID system would provide the industry with a vital safety net, protecting both domestic and international beef sales, and the national herd, in the event of a serious disease outbreak.

A mandatory ID system was always going to run into opposition.

But the federal government, as its architect, also made a series of blunders.

It tried to introduce the concept for all commercial livestock species, instead of just cattle and hogs.

So the hobby farmers came out of the woodwork and protested.

> The irony, though, is in the years of discussion about labelling, USDA (US Department of Agriculture) repeatedly said COOL (country of origin labelling) might violate international trade rules. Moreover, its economic analysis could find no likely benefits

The US Department of Agriculture (USDA) also blundered in not tackling the issues of data confidentiality and producer relief from liability.

With a mandatory ID system now all but dead, a group of eight cattle organisations have put forward a statement of 12 principles they hope will guide the beef industry towards a more comprehensive ID system.

There is though no mention of left wondering how you can have an effective ID system to be used in traceback and trace forward of animals if a lot of producers don't participate.

Nonetheless, the statement of principles is notable because it brought together organisations that at times have been bitter rivals and compete for producers' support.

It's the first time in years these groups have agreed on anything.

The groups agreed development of a national cattle ID system should be based on minimal added costs to the beef and dairy industry.

ID information must be kept confidential and kept under the control of state animal health officials.

There should be renewed emphasis on preventing the introduction of foreign animal diseases.

The groups developed the principles at a meeting organised by the Livestock Marketing Association (LMA), which represents livestock auctions.

Discussion of a national ID plan had moved away from those most affected by it, cattle producers and marketers, said the LMA.

So the groups worked toward a cattle ID and traceability systems for animal disease surveillance and control.

The groups agreed an ID plan for the cattle industry should be species-specific because of the diverse way cattle were raised, marketed and processed, said the LMA.

The organisations have now presented their ideas to USDA and to members of Congress.

Let's hope USDA grabs the ideas with both hands as quickly as possible.

Meanwhile, challenges to mandatory COOL are now with the World Trade Organisation.

On one side are Canada, Mexico and the US meatpacking industry, all of whom strongly opposed COOL.

On the other side is the US Government, notably USDA and the Office of the US Trade Representative (USTR).

The government is duty-bound to defend COOL because it is a federal law.

The irony, though, is in the years of discussion about labelling, USDA repeatedly said COOL might violate international trade rules.

Moreover, its economic analysis could find no likely benefits.

Canada and Mexico in late 2009 filed a case against the US with the WTO.

USTR then filed a federal notice about its intent to defend COOL at the WTO and asked for comments.

Needless to say, industry

As far as the packing industry was concerned, COOL violated the US's international trade obligations for many reasons and the US needed to honour these obligations.

COOL discriminated against imported meat and live animals and did not meet an international standard regarding country origin.

That's what the American Meat Institute wrote in its comments to USTR.

These were the same arguments used by Canada and Mexico in its WTO case.

I daresay Australia would agree with these arguments, even though COOL does not appear to have negatively impacted its meat and other food exports to the US.

A different argument against COOL is that it only applies to 25 per cent of US beef production.

Yet it cost the beef industry more than $1.5 billion to implement just in the first year.

COOL continues to disrupt Canadian and Mexican cattle exports to the US at a time when US feedlots and packers need those cattle to stay in business.

WTO complaints can take years to be decided, especially as most rulings are appealed.

So origin labelling on meat and other food will remain a cost to the industry and an irritant to the two countries that are the biggest export markets for US beef and pork.

What a way to treat the neighbours.

■ Steve Kay is the editor of US

Appendix 2

Article 11.6.2 OIE Terrestrial Animal Code

The BSE risk status of the cattle population of a country, zone or compartment

The BSE risk status of the cattle population of a country, zone or compartment should be determined on the basis of the following criteria:

1. the outcome of a risk assessment, based on the provisions of the Terrestrial Code, identifying all potential factors for BSE occurrence and their historic perspective. Members should review the risk assessment annually to determine whether the situation has changed.

   a. Release assessment

   Release assessment consists of assessing, through consideration of the following, the likelihood that the BSE agent has either been introduced into the country, zone or compartment via commodities potentially contaminated with it, or is already present in the country, zone or compartment:

   i. the presence or absence of the BSE agent in the indigenous ruminant population of the country, zone or compartment and, if present, evidence regarding its prevalence;
   ii. production of meat-and-bone meal or greaves from the indigenous ruminant population;
   iii. imported meat-and-bone meal or greaves;
   iv imported cattle, sheep and goats;
   v. imported animal feed and feed ingredients;
   vi. imported products of ruminant origin for human consumption, which may have contained tissues listed in Article 11.6.14. and may have been fed to cattle;
   vii. imported products of ruminant origin intended for in vivo use in cattle.

   The results of surveillance and other epidemiological investigations into the disposition of the commodities identified above should be taken into account in carrying out the assessment.

   b. Exposure assessment

   If the release assessment identifies a risk factor, an exposure assessment should be conducted, consisting of assessing the likelihood of cattle being exposed to the BSE agent, through a consideration of the following:

   i. recycling and amplification of the BSE agent through consumption by cattle of meat-and-bone meal or greaves of ruminant origin, or other feed or feed ingredients contaminated with these;
   ii. the use of ruminant carcasses (including from fallen stock), by-products and slaughterhouse waste, the parameters of the rendering processes and the methods of animal feed manufacture;

iii. the feeding or not of ruminants with meat-and-bone meal and greaves derived from ruminants, including measures to prevent cross-contamination of animal feed;

iv.. the level of surveillance for BSE conducted on the cattle population up to that time and the results of that surveillance;

2. on-going awareness programme for veterinarians, farmers, and workers involved in transportation, marketing and slaughter of cattle to encourage reporting of all cases showing clinical signs consistent with BSE in target sub-populations as defined in Articles 11.6.20. to 11.6.22.;
3. the compulsory notification and investigation of all cattle showing clinical signs consistent with BSE;
4. the examination carried out in accordance with the Terrestrial Manual in a laboratory of brain or other tissues collected within the framework of the aforementioned surveillance and monitoring system.

When the risk assessment demonstrates negligible risk, the Member should conduct Type B surveillance in accordance with Articles 11.6.20. to 11.6.22.

When the risk assessment fails to demonstrate negligible risk, the Member should conduct Type A surveillance in accordance with Articles 11.6.20. to 11.6.22.

APPENDIX 3

ANIMAL FOOD SAFETY IN AUSTRALIA FROM ARMCAN

Thiamine deficiency in a cat associated with the preservation of 'pet meat' with sulphur dioxide

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A cat with allergic dermatitis was fed a diet of fresh meat and a multi-vitamin supplement for 38 days to exclude food allergy as a cause of its dermatopathy. The cat died as a result of acute thiamine deficiency, which was caused by inactivation of thiamine by the preservative, sulphur dioxide. The continuing undeclared usage of sulphites in the Australian pet food industry is discussed.

Aust Vet J 1997;75:719-721
Key words: Thiamine deficiency, sulphur dioxide, sodium metabisulphite, cat.

po	Orally
im	Intramuscularly

Case report

An 11-year-old, Feline immunodeficiency virus-negative, spayed domestic short-haired cat (weight 4 kg) with recurrent, non-seasonal pruritic dermatitis was presented in November 1995 after a symptom-free period of 15 months. Skin biopsies indicated a chronic active allergic dermatitis with secondary pyoderma.

The diet was changed to exclude fish. The cat was treated with methylprednisolone acetate (20 mg im), doxycycline (2.5 mg/kg once daily po) and acetylpromazine (2.5 mg po) as necessary for pruritus.

Marked deterioration in the cat's dermatopathy occurred over the next 3 weeks. Areas of erythema developed on the ventral trunk and legs, associated with constant licking. The neck was contused from scratching and generalised hyperkeratosis became obvious elsewhere. Further biopsies confirmed that type 1 hypersensitivity dermatitis persisted and indicated the development of epidermal and follicular infundibular mucinosis. Treatment with prednisolone (1 mg/kg po twice daily) was commenced together with enrofloxacin (25 mg once daily po), chloropheniramine (4 mg twice daily po) and phenobarbitone for sedation.

The cat's diet was changed to an exclusive diet of fresh kangaroo meat in an attempt to eliminate the possibility of food allergy or intolerance. The diet fed was raw vacuum-packed kangaroo mince with supplementary calcium carbonate powder (two teaspoonfuls per kg of meat).

The cat became increasingly difficult to medicate and was hospitalised 10 days later. The administration of a multi-vitamin supplement initially given po each day, was stopped temporarily.

The skin condition improved gradually over the next 3 weeks. However, the cat's appetite was noted to decrease during the last week. Occasional vomiting and diarrhoea were observed. Treatment with antibiotics and sedative drugs was discontinued and the prednisolone dosage reduced to 0.5 mg/kg twice daily.

Four days later (38 days after starting the kangaroo meat), the cat developed an acute central vestibular syndrome with nystagmus, the direction of which changed in relation to head positioning. Tetraparesis with mental depression were also noted. Nystagmus was less pronounced the following day, but the cat had dilated, poorly light-responsive pupils, although it was not blind. Next morning the cat was stronger and could walk unsteadily, but had marked dyspnoea. Its heart rate was within normal limits and regular. However she died suddenly and quietly 3 h later.

Histopathological findings

At necropsy, the only abnormality noted was asymmetry of the tectum. On transverse sectioning there was bilateral petechiation of the caudal colliculi. Histopathological examination revealed bilaterally symmetrical haemorrhagic polioencephalomalacia involving structures in the vicinity of the periaqueductal region. These various findings were consistent with a diagnosis of thiamine deficiency. Death was attributed to destruction of vital centres of the brain stem due to the thiamine deficiency, or to a secondary catecholamine-mediated 'brain-heart syndrome' causing myocardial necrosis, or to myocardial necrosis directly referable to thiamine deficiency.

The latter two possibilities could not be confirmed as the heart had not been submitted for histopathological examination. Marked dyspnoea, noted on the final morning, was clinically suggestive of failing cardiac function and/or an altered respiratory pattern secondary to central nervous disease.

Analysis of foods

The remaining meat and two unopened containers, which had been purchased for future use were analysed by the New South Wales Health Department. Sulphur dioxide concentrations of 275 mg/kg, 355 mg/kg and 325 mg/kg, respectively, were fo

The thiamine content of these samples was not assayed but would be expected to be greatly reduced.[1] Each container displayed the name and address of the producer and packager, the species of origin: 'fresh-minced roo', use-by-date, advice on storage and country of origin (Australia). The label on the container advised that the product was vacuum packed for the pet's protection. There was no reference to the presence of preservatives.

Further samples of raw pet food were purchased from various sources and seven of eight specimens tested were found to contain sulphur dioxide (average 360 mg/kg) by the New South Wales Health Department. Further sampling and in-house testing for the presence of sulphites with a solution of malachite green (200 mg/L), a qualitative test, and/or with the commercially available, Merckoquant Sulphite Test strips (detection threshold stated as 15 ppm free sulphite but not found to be satisfactory until 270 ppm, with suitable specimen preparation) was undertaken.[2]

These tests revealed that of 63 meats (beef, lamb, rabbit, poultry, pig, venison, turkey, fish and kangaroo) and composite preparations of these meats, variously mixed together with non-animal carbohydrates (sausage type prepared pet food or 'food rolls') and sold for pet consumption in New South Wales, 54 samples contained sulphites. Samples from four States representing all major packagers and processors were included in this survey. Sulphites were detected in 39 of 42 fresh or frozen meats, and 15 of 21 composite preparations tested in-house. Five of these composites analysed by the New South Wales Health Department were found to have similar concentrations, (average 380 mg SO2/kg) to those found in the 13 fresh or frozen meats tested. Beef specimens tested in-house contained more preservative than any other species. 'Food rolls' labelled as being free of preservatives were found to be free of sulphites. In New South Wales, kangaroo meat packaged and sold for human consumption was found to be free of sulphites.

Discussion

In this case, the cat was fed for 38 days an exclusive diet of meat preserved with sulphur dioxide. Destruction of thiamine by this preservative in both the meat and in the multi-vitamin supplement fed concurrently is thought to have caused the development of thiamine deficiency. Progressive inappetence, occasional vomiting and diarrhoea were considered to be non-specific signs of evolving deficiency in this cat. Inactivity and mild weakness may have been masked by cage confinement. Inappetence was marginal until the 34th day, but vomiting increased in frequency to once every 2 to 3 days over the last 10-day period, while diarrhoea was less frequent. Clinical signs of thiamine deficiency may also appear to be of a very acute nature and run a short course, although experimental evidence has shown that there is a long prodromal period (Studdert VP personal communication).[1,3,4]

Descriptions of feline thiamine deficiency refer to inappetence with weight loss, vomiting and diarrhoea, general weakness and mild ataxia, as early signs.[3,4] Postural abnormalities then develop and may include a spastic gait, active ventroflexion of head and neck (reported as a 'classical' sign), and progressively severe vestibular signs such as worsening ataxia, curling up when lifted or a head tilt. Seizures or abnormal behaviour such as stupor or hyperaesthesia may develop.[3,4] Dilated unresponsive pupils are a common finding. Opisthotonus and tetraparesis are reported. Dogs develop upper motor neuron parapareis that progress to tetrapareis and convulsions.[1]

Uncharacteristically ventroflexion did not occur in this patient.

Treatment with thiamine hydrochloride (100 to 250 mg twice daily, initially by injection) has been recommended for acute presentations in cats and dogs.[1,3,5] In less severe or suspected cases of thiamine deficiency, parenteral dosages of 20 to 50 mg twice daily are suggested initially. Follow-up oral treatment with 25 to 50 mg once daily is an option only when enteric function is normal and if the food provided is free of sulphites, or if this supplement is given at least 12 h after feeding sulphite-containing food to cats. The average time for complete emptying of the stomach of normal cats after feeding ranges from 7 to 17 h,[6] thus antidotal thiamine supplementation could conceivably be inactivated by sulphite containing food in the stomach and small intestine for up to 17 h after a meal.

Thiamine is a heat-labile, water-soluble vitamin which is not stored in the body to any extent, and therefore must be continually ingested in food.[7,8] As thiamine is essential for energy metabolism, its requirements may be expressed as mg/1000 kcal, (minimum amounts for cats 1.0 mg/1000 kcal; dogs 0.3 mg/1000 kcal). Expressed as mg/kg of food (dry matter), cats require 5 mg/kg and dogs 1 mg/kg. These vary with changing demands for energy as in growth, systemic disease states and by the nature of the diet.[7-9]

Inappetence or anorexia in a sick cat, particularly if associated with polydipsia and polyuria or fluid diuresis with increased losses of this vitamin, may precipitate sub-clinical deficiency to complicate the original cause of ill-health. Deficiency results in abnormal glucose metabolism with reduced appetite, loss of weight, weakness, vomiting and diarrhoea, with neurological symptoms referable to the development of encephalopathy and haemorrhage into periaqueductal brain stem nuclei.[1,3]

Sufficient quantities of thiamine to meet the requirements of cats and dogs are usually found in fresh meat. Cooking and processing may cause the loss of 40 to 50%.[7]

Mixing unpreserved fresh meat with fish or fish products containing active thiaminases results in the destruction of thiamine in that meat. Canned pet food manufacturers adjust for this loss by supplementation with exogenous thiamine and by improved processing procedures.[7-9]

Commercial canned and dry products in Australia contain negligible quantities of sulphite preservatives. The feeding of fresh beef, lamb, horse and kangaroo meats and 'food rolls' or 'chubbs' preserved with sulphites causing thiamine deficiency in cats and dogs was reported recently.[1,10]

Studdert and Labuc concluded that fresh pet meat and composites in Australia must be assumed to contain sulphur dioxide because its use is so wide spread. Their findings were confirmed by the analyses of samples in this study where 64 of 74 pet meats and food rolls were shown to contain

quantities of sulphur dioxide likely to inactivate a considerable portion of the endogenous thiamine present in these raw and processed meat products.[1] The only exception were 'food rolls' labelled as free of preservatives.

Sulphites react irreversibly to cleave the thiamine molecule rendering it inactive. The extent of destruction of thiamine depends on the sulphur dioxide concentration so that 400 mg and 1000 mg SO_2/kg in meat depletes the thiamine content by 55% and 95% respectively.[1] Supplementary thiamine, such as brewer's yeast, added at the preparatory or packaging stage, is similarly inactivated. Likewise, food containing adequate quantities of thiamine or added vitamin, mixed as a pre-mix with the sulphite meat or fed at the same time, is also inactivated.

The effect of sulphites and sulphur dioxide on thiamine in stored food is recognised to be of nutritional significance in humans, despite their perceived greater freedom of choice and variety of food. Sulphites are permitted as food additives for human consumption in some Australian foods such as processed and manufactured meats, but are prohibited in others by the Australian Food Standards Code under the direction of the Australia New Zealand National Food Authority. Sulphites and SO_2, are recognised also as an allergen in some people. Maximum permitted concentrations of sulphites are defined by this Code for all foods. They are measured as sulphur dioxide concentration and, if over 25 mg/kg, their presence must be declared on food packages. This labelling identification is either by name or by identification number (220, 221, 223, 224, 225, 228 all identify sulphite salts). Unpacked foods such as restaurant and take-away meals are exempted from carrying these ingredient labels but are required to comply with additive permissions specified in the Code. Sulphites are not permitted to be used in fresh meat for human consumption, with the exception of sausages.

No standards are in place for meats used in the pet industry, despite the important findings of Studdert and Labuc, and Sames.[1,10] There is no requirement to identify those meats which contain preservatives. Furthermore there is no limit to the amount of preservative, which may be added to these meats. Sulphites, particularly sodium metabisulphite, are used freely in the industry for their biostatic and biocidal effects. The growth of salmonellas is particularly inhibited.[1] The addition of sulphites is a cost-effective method of extending storage life of meat by slightly delaying and masking spoilage, without preventing putrefaction.[1] As colouring agents sulphite enhance the appeal of the meat to the purchaser. Their presence not only maintains the redness of fresh meat for longer periods than normal but they will restore redness to discoloured spoiled meat.[1] These preservatives are important to an industry where standards of hygiene are as yet undefined.

The inclusion of sulphites in Australian pet meat is being examined by the Agricultural and Resource Management Council of Australian and New Zealand. A Working Group has commenced the first development of a safe food system by developing an Australian Standard for the Hygienic Production of Pet Foods (fresh, frozen and packaged composite-type meat products).

This case study confirms that the problem of unsafe, uncontrolled and undeclared use of sulphites in the fresh, frozen and packaged pet meat industry in Australia, identified earlier,[1] has not been addressed.

Any exclusion diet for cats or dogs, particularly involving a unique protein source such as kangaroo, can be checked for sulphites. The bleaching of malachite green solution (200 mg/L most easily obtained from aquarium suppliers), is the basis of precise colorimetric determinations.[2] It can also be used as a spot test on a white background using meat for human consumption as a control (1/2 teaspoonful of sample mixed well with 0.5 mL reagent for 2 min). Alternatively, use of kangaroo meat marketed for human consumption should not cause thiamine deficiency if given to cats as an exclusion diet.

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

Tamgüney 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.

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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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APPENDIX 4

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LANDES BIOSCIENCE

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Prion. 2009 Oct 23;3(4). [Epub ahead of print]

In vitro amplification of prions from milk in the detection of subclinical infections.

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Prions can be amplified by serial protein misfolding cyclic amplification (sPMCA) from the milk of a high proportion of apparently healthy, scrapie exposed sheep with PRNP genotypes not previously associated with high disease penetrance. (1) These data strongly suggest the widespread presence of subclinical scrapie infections within scrapie-exposed flocks containing sheep with a range of susceptible PRNP genotypes. These data also lead to the hypothesis that similar subclinical disease states may be common for other animal and human prion diseases. Furthermore, the application of sPMCA to milk provides a method to detect such subclinical disease. Here, we describe the high level amplification of bovine spongiform encephalopathy (BSE) prions from both ovine and bovine origin, a methodology that will facilitate the detection of any prions secreted within bovine and ovine milk during subclinical and clinical BSE disease.

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