Bibliography for
Transmissible Spongiform Encephalopathies

August 1996

Centers for Epidemiology and Animal Health
555 South Howes
Ft. Collins, CO 80521
Table of Contents

BSE - General ............................................................................................................. 1
BSE - United Kingdom ............................................................................................... 11
BSE - Epidemiology ..................................................................................................... 17
TSE’s - General ........................................................................................................... 27
TSE’s - Etiology and Pathogenesis ................................................................................ 35
TSE’s - Diagnosis ........................................................................................................ 55
Human Spongiform Encephalopathies ................................................................. 61
Transmissible Mink Encephalopathy ......................................................................... 69
Scrapie ....................................................................................................................... 73
SE’s in Other Species ................................................................................................ 85
Risk Assessment, Surveillance, and Cases of BSE Outside the United Kingdom ......... 91
Index of First Authors .............................................................................................. 99

Sources: Center for Agriculture and Biosciences International; University of Illinois - Urbana Champaign; Colorado Alliance of Research Libraries; US Dept. of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services, Emergency Programs and Centers for Epidemiology and Animal Health; US Dept. of Health and Human Services, National Library of Medicine.

Articles in languages other than English were included if they had information pertaining to specific countries (i.e., risk analyses or investigations) or were ‘original’; general review articles in languages other than English were not included. In general, we tried to provide a comprehensive bibliography of articles from refereed journals and publications of government, professional associations, and international organizations.
<table>
<thead>
<tr>
<th>Year</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Scientists, consumer spokespersons and sensationalists in tabloids and broadsheets caused the BSE scare.</td>
</tr>
<tr>
<td></td>
<td>The authors report on a quantitative assessment of the risk of BSE in other European countries. They conclude that, based on the numbers of pure bred breeding cattle exported from Great Britain to other European countries, many more cases of BSE should have occurred outside of Great Britain than have been reported.</td>
</tr>
<tr>
<td></td>
<td>Small steps forward on BSE. 1996. Veterinary Record 138(16):373.</td>
</tr>
</tbody>
</table>

WHO gathers international experts to review the situation BSE. 1996. Veterinary Record 138(15):343.

1995


Current knowledge about BSE is considered with regard to the European Commission’s position in relation to scientific and legal issues raised by the necessity to protect humans and animals from BSE.


Bovine brain infected with the BSE agent was used to spike material processed in pilot scale models of 12 rendering processes used in the European Union and 3 which are not. Meat and bone meal (MBM) and tallow were produced from the rendered tissues. Suspensions from all the MBM samples and 2 of the tallow samples were assayed in mice for infectivity. Four (4) of the 15 processes produced MBM with BSE infectivity. Neither of the tallow samples had infectivity.

This study examined the association between 3 categories of neurohistological diagnoses (focal spongiosis of white matter, encephalic listeriosis, no significant lesions) and epidemiological factors in unconfirmed cases of clinically suspect BSE. Confirmed cases of BSE were included for comparison. The authors concluded that, despite their statistical significance, the findings do not have sufficient predictive power to be of value in making clinical decisions.


Aside from a description of the neuropathology of BSE, this review covers the effects of legislation on the epidemic in the UK, changes in incidence (up to April 1994), maternal and lateral transmission, and the risks of BSE epidemics in other countries.

1994


This issue is devoted to papers on BSE including recent research and control, agent hypotheses, molecular genetics, immunodiagnosis, and rendering systems.


The results of this study confirm previous findings, showing that neuronal loss is a significant feature of BSE and may therefore contribute to the development of clinical disease. No association was found between duration of clinical signs and the numbers of remaining neurons or the extent of vacuolation. However, the authors suggest that the absence of such association should be viewed cautiously as there was substantial variation in neuronal populations between individuals.


1993


Topics covered in this monograph include: geographical distribution, economic implications, aetiology, epidemiology, clinical signs, pathology, diagnosis, prevention, control and eradication.


Neuropathological observations were made in 200 clinically suspected cases of BSE in which pathognomonic vacuolar changes were absent. This study identified diseases and lesions which feature in the differential diagnosis of BSE. Their more accurate diagnosis may become particularly important if, as predicted, the BSE epidemic declines.

1992


This note gives guidance on preparation of medicinal products which contain active ingredients and/or excipients derived from bovines, as well as medicinal products for which the production process involves bovine material.


The vestibular nuclei from BSE cattle had an approximately 50% reduction in total numbers of neurons when compared with controls. These results show that previously unsuspected neuronal loss is an important feature of BSE.


A questionnaire was used to record the presence of specific clinical signs reported for 17,154 confirmed cases of BSE. The signs most frequently recorded were apprehension, hyperaesthesia, and ataxia.

1991


The history of the BSE outbreak in the UK and the symptoms and epidemiology of the disease are reviewed.

Use of animal protein and increased nitrogen in cattle feeds would lead to a relative deficiency of essential fatty acids in the cell membranes and hence reduced membrane stability. The possibility that the changes in animal feeds would have depleted cattle tissue membranes and make them susceptible to BSE is discussed.


This paper reviews BSE, the mode of transfer, the possible presence of infective agents in food, the resistance of BSE to cooking, and the likelihood that man may become infected. The origins of BSE are considered, and it is claimed that a substantial danger for man exists.


The author reports that studies into the genetic aspects of BSE are continuing but so far show no evidence to indicate genetic control analogous to that which occurs in sheep scrapie. Preliminary analysis also supports the food-borne hypothesis with meat and bone meal as the primary vehicle of infection. The current increase in cases is reported to be consistent with the recycling of infected cattle tissue through meat and bone meal from 1985-85.


1990


This issue of JAVMA contains the 13 papers presented, a summary and conclusion of a round table discussion, recommendations concerning the risk of BSE in the US, and recommendations
for initiation of research studies on BSE.


Policy paper.


Eddy RG. 1990. Caesarean sections on BSE cow. [Correspondence]. Veterinary Record 126(4):92


This publication consists of a number of papers, including a review on BSE.


Cerebrospinal fluid from 20 cows with BSE and 10 healthy cows was analyzed. There were no significant differences in any of the parameters measured (specific gravity, lymphocytes, neutrophils, histocytes, total protein, total albumin, etc) between the BSE and healthy cows. It is suggested that the absence of any gross immunological response may allow BSE to be differentiated from certain bacterial and viral infections of the CNS.

1989

Madeiros CA. 1989. BSE safety precautions. [Correspondence] Veterinary Record 125(3):73.


This report considers the history of BSE, other SE’s, the epidemiological evidence on the cause of BSE, and the transmission of BSE. It also discusses the future course of the disease, describes the actions already taken to reduce its spread, and makes further recommendations of monitoring offspring, medicinal products, cases of CJD, and occupational groups that could be exposed to the BSE agent.


The author writes from the perspective of the rendering industry in the UK.

1988


Comments about meat inspection.


Discusses hexachlorophene toxicity.


Discusses hexachlorophene toxicity.
1987


BSE - United Kingdom

1996


The author maintains that export statistics show that whereas UK exports of animal feeds had remained almost constant in the years leading up to the 1988 feed ban, they more than doubled the next year. He thus raises the question if the UK ‘dumped’ contaminated animal feed.


Simmons MM. 1996. BSE in Great Britain: consistency of the neurohistopathological findings in two random annual samples of clinically suspect cases. Veterinary Record 138(8):175.


The authors predict that the number of BSE cases will continue to decline from 8,050-11,270 in 1996 to 4,250-7,130 in 1997, 1,970-3,870 in 1998, and 840-1,880 in 1999.

Stevenson RM. 1996. LVIs and BSE control measures. Veterinary Record 138(18):341.


1995


This annual report for 1994 of the Institute contains descriptions of research, including the role of the Institute in research on scrapie, BSE, and CJD.

UK, Ministry of Agriculture, Fisheries and Food. 1995. MAFF sets out the position on BSE. Veterinary Record 137(26):651.

1994

The book is comprised of a diary of events, from the first cases in November 1986 to August 1994, and various other topics including CJD and a description of rendering processes. Throughout the book, the Government, Ministry of Agriculture, and Health Department are accused of minimizing the problem and of being more concerned with reassuring the public that meat is safe rather then trying to control the disease.

---


This report describes the general state of animal health in the UK, including sections on BSE. 1993 was the first year to show a significant decline in the BSE epidemic.

---


Interviews were conducted with 50 dairy farmers who had BSE cases in their herds. Some farmers believed that the April 1994 valuation scheme which lowers the maximum compensation would potentially lead some farmers to find other ways of disposing of their suspect BSE cows, which would no longer be notified to MAFF. Another view was that public relations by MAFF on BSE required considerable improvement.

---


The program of legislative and voluntary action to improve the safety of rendered products is reviewed for the period 1985 to 1994.

---

1993

---


The memorandum reviews research on TSE’s and results of epidemiological studies on BSE and CJD in the UK. It is concluded that the BSE epidemic is on the decline and that policies in the UK are sufficient to minimize the risk of exposure to BSE of all species, including humans.
BSE was the chief concern during 1992, with 29,802 confirmed cases. Progress in research on the disease is outlined.

1992


BSE was not confirmed histologically in 225 of 829 bovine brains submitted for diagnosis. Several previously described disorders of the CNS were observed in these brains as well as disorders not previously recognized in Britain.


Some of the brains submitted for neurohistopathological examination did not show lesions of BSE. They showed, among other findings, neuronal chromatolysis and necrosis of the brainstem. The 25 cows affected came from most parts of Scotland; some cows had no reported access to feed supplements. The cause of the disorder was not determined.

1991


This paper reviews the development of the control program for BSE in Great Britain, including problems posed by the new disease, investigations, and animal and public health measures.


This report covers many animal diseases in Northern Ireland; during the year, 124 cases of BSE were reported.
1990


This report summarizes findings from histopathological examinations made on 313 brains submitted to the Lasswade Laboratory from July 1988 to December 1989.


The article summarizes progress made up to February 1990, both in the field and the laboratory, in controlling BSE, and discusses problems encountered and solutions found.


This report comprises presentations made by the UK and the Republic of Ireland on the epidemiology and prevalence of the disease in their respective countries.


The report consists of seven sections: (1) general introduction; (2) safety of beef; (3) Government response to BSE; (4) Government campaign to reassure public opinion; (5) economic damage to the meat industry; (6) trade in beef within the EC; (7) summary of conclusions and recommendations.
This report gives a brief account of the work of the Institute during 1989, including work on BSE.

Report summarizes activities of the Veterinary Field Services, Veterinary Investigation Service and the Central Veterinary Laboratory. BSE was confirmed in 7,134 cattle, at the rate of 100-200 cases a week.


1989


1988


Report summarizes activities of the Veterinary Field Services, Veterinary Investigation Service and the Central Veterinary Laboratory. BSE was confirmed in 7,134 cattle, at the rate of 100-200 cases a week.

The author discusses the implications of a study showing that macaque monkeys intracerebrally inoculated with BSE brain extracts developed a disease with plaques identical to those of patients with variant-CJD. (See Lasmezas, et al. 1996).


Three macaques intracerebrally inoculated with brain homogenate from BSE-affected cattle developed a disease similar to variant-CJD. Moreover, the neuropathological phenotype was different from that observed in two macaques inoculated with sporadic CJD. The authors conclude that the similarity of the clinical, molecular and neuropathological features observed in the three BSE-infected macaques with the human cases of vCJD is striking, and that this study provides evidence supporting the hypothesis that the BSE agent is responsible for the emergence of the new form of CJD in humans.


The authors report that they have kept a large breeding colony of common marmosets (Callithrix jacchus) for almost 20 years. More than 100 marmosets were born in the colony between 1980 and 1990 that lived for more than 5 years and were exposed to ruminant-derived protein in their daily diet for their entire lives. With the exception of those animals which were injected intracerebrally with infected brain, no animal in the colony has ever developed SE. The authors suggest that this observation serves as a reminder that the oral route is probably an inefficient mode of infection for SE across the species barrier.


1995


Ten cattle were inoculated or challenged orally with scrapie agent from a sheep and a goat. Between 27 and 48 months after inoculation, neurologic disease was observed in 1 of 5 cattle given the sheep brain homogenate and in 2 of 5 cattle given the goat homogenate. In all 3 affected cattle, the disease was expressed clinically as increasing difficulty in rising from recumbency, stilted gait of the pelvic limbs, disorientation, and terminal recumbency during a 6 to 10 week course. Neurohistological changes, though consistent with scrapie, were slight and subtle. It is concluded that clinically and neurohistologically, the experimentally induced disease differed from BSE.


This paper describes a case-control study designed to investigate whether there is any evidence for direct transmission of BSE to cattle born after the introduction of the feed ban. The study found no evidence that maternal transmission occurred. Although there was marginally significant evidence of horizontal transmission (cow to cow or to a calf which is not its own offspring), the authors concluded that this mode of transmission was not capable of maintaining the epidemic.


The results from this study showed that no neurological disease occurred in any of the 275 mice which survived for more than 300 days after drinking, or injection of, milk from cases of BSE.

Results from various transmission experiments are reviewed. It is concluded that the BSE agent has retained its identity when passaged through a range of species and the ‘donor’ species has little specific influence on disease characteristics in mice, adding to evidence for an agent-specific informational molecule.


To determine if sheep scrapie agent in the US would induce a disease in cattle resembling BSE, 18 newborn calves were inoculated intracerebrally with scrapie agent. All calves kept longer than 1 year became severely lethargic and demonstrated clinical signs of motor neuron dysfunction, ending in permanent recumbency. Brain lesions were subtle, but a disease-specific isoform of the prion protein was present in the brain of all calves. Neither signs nor lesions were characteristic of those for BSE.


The history of cow with BSE born after the ruminant feed ban and whose dam had also had BSE is suggestive of vertical transmission of BSE.


The results of further analyses of data on the progress of the BSE epidemic are reported.


The literature is reviewed and circumstantial evidence is presented to support the hypothesis that the BSE epidemic in the UK was initiated as a result of a combination of factors. These factors, it
is suggested, were genetic, nutritional, and chronic exposure to mutagenic organophosphate pesticides which disrupt the genetic pathway of prion protein synthesis.


Forty 4-month-old calves from farms free of BSE were dosed orally with BSE agent. Starting at 6 months old and thereafter at 4-month intervals, calves were killed and tissues assayed for infectivity. Evidence of oral experimental transmission of BSE to the cattle was obtained from the mouse assay of the distal ileum from calves killed 6 and 10 months after challenge.


The research which has been undertaken into potential sources of infection and means of transmission is briefly described.

1993


Two young marmosets were injected intracerebrally and intraperitoneally with brain homogenate from a cow with BSE, and two other marmosets were similarly injected with brain homogenate from a sheep with natural scrapie. All four marmosets developed neurologic signs 38-47 months after injection, and post-mortem examination showed SE.


SE has been confirmed in both sheep and goats after intracerebral inoculation or oral dosing with brain homogenate derived from cattle with BSE. This is the first report of the experimental transmission of BSE to sheep and goats.

Middleton DJ, Barlow RM. 1993. Failure to transmit BSE to mice by feeding them with extraneural tissues of affected cattle. Veterinary Record 132(22):545-547.
Tissue samples from brain and 5 extraneural tissues were prepared from 4 cases of BSE and fed to mice. The disease was transmitted only to mice fed brain. Spleen and spinal cord homogenate from these affected mice were then intracerebrally inoculated into another group of mice, which also got the disease. Similar passages from all other groups of mice failed to produce evidence of infection.


The author, who is a dairy farmer, describes more case reports to support his hypothesis, put forward in a previous article (Ecologist 22(2), 1992), that exposure to organophosphorus compounds triggers the onset of BSE.


The age-specific incidences of BSE in herds in which cases occurred in 1989, 1990, 1991, and January to June 1992 are presented. It is concluded that the analyses indicate that preventing the exposure of cattle to infected meat and bone meal has had the effect expected.

1992


A total of 164 cases were confirmed as BSE in Northern Ireland between July 1988 and December 1990. They were very similar to those observed in Great Britain except that the annual incidence in 1990 in Northern Ireland, 2.3 confirmed cases per 10,000 adult cows, was approximately one 10th of that in Great Britain. The findings were also consistent with the current hypothesis that affected cattle had been exposed to scrapie-like agent via feedstuffs containing ruminant-derived protein.

Transmission from 4 cases of BSE to mice resulted in neurological disease in 100% of the recipients. The results from the 4 cases were very similar to one another; however, there were major differences in the incubation period between the 4 inbred strains of mice tested. The distribution of vacuolar degeneration in the brains of mice infected with scrapie differed from those infected with the BSE isolates.


Proprietary calf feedstuffs and whether or not they included meat and bone meal were compared for animals born between July 1983 and June 1984 in 1,042 herds. Feeding of proprietary concentrates containing meat and bone meal was a statistically significant risk factor for the occurrence of BSE. It was concluded that BSE occurred as a result of exposure to a scrapie-like agent via meat and bone meal.


The age-specific incidences of BSE were analyzed in herds in which cases occurred in homebred animals where the exact date of birth was available for every case and for which age distribution of the adult herd was known. The incidence within the 2-year-old age class was lower in 1991 (0.01%) than in 1989 (0.04%) and 1990 (0.05%) while incidence in animals 3 years of age or older was greater in 1991 than in 1989 and 1990.


This paper provides an updated account of the epidemiological features of BSE. The number of cases up to December 1989 represents an annual incidence of 3.9 confirmed cases per 1,000 adult animals in Great Britain. Many more dairy herds were affected than beef herds, a difference attributable to the difference in feeding practices. The geographical variation in incidence previously described has persisted with the highest incidence in the south and east of England. The low within-herd incidence also remained unaltered. The results support the hypothesis that the outbreak of BSE was due to sudden exposure of the cattle population to a scrapie-like agent in 1981/82.

This report apparently describes the same studies as the paper published by the authors in 1993 (Veterinary Record 132(22), 1993).


Primary transmission of natural BSE was attempted to cattle, hamsters, pigs, and domestic fowl by parenteral inoculation of brain homogenate; to pigs and domestic fowl by oral exposure, and to cattle by oronasal exposure to fetal membranes. All cattle parenterally challenged developed BSE. Transmission was also confirmed in one of 10 parenterally challenged pigs. Transmission had not been demonstrated in the remaining studies, most of which were still incomplete.


Reasons for suggesting that BSE in Britain may not have been caused by processed protein feed containing offal from scrapie-affected sheep are given. These include (1) scrapie in sheep is relatively uncommon; (2) many scrapie cases are disposed of on the farm; (3) histopathologic indications are that BSE is a naturally occurring disease; (4) scrapie in sheep can appear do novo following selective breeding and this may have occurred in cattle.


The results of further epidemiological studies of BSE in the UK support the previous findings that the onset of exposure of the cattle population to a scrapie-like agent occurred in 1981/82. The onset of this exposure was related to the cessation, in all but 2 rendering plants, of the hydrocarbon solvent extraction of fat from meat and bone meal. A further possible explanation, related to the geographical variation in the reprocessing of greaves to produce meat and bone meal, was identified for the geographical variation in the incidence of BSE.


Mice were fed minced brain tissue and cerebrospinal fluid collected from 4 clinical cases of BSE. After 15-18 months of feeding, histological examination showed signs typical of TSE. These features were absent in control mice killed at the same time.


Twenty-four calves from herds in which no cases of BSE had been recorded were allocated into challenge and control groups. Challenge groups were inoculated intracerebrally and intravenously with homogenized brain stem from a case of BSE; control groups were inoculated with saline. Two of the challenged animals developed clinical signs. Histopathologic examination showed vacuolar lesions and fibrils typical of BSE.

Dawson M, Wells GAH, Parker BNJ, et al. 1990. Primary parenteral transmission of bovine spongiform encephalopathy to the pig. [Correspondence]. Veterinary Record 127(13):338.

Ten one- to two-week old piglets from a specific pathogen free breeding herd were inoculated by simultaneous injections intracerebrally, intravenously, and intraperitoneally with an inoculum from 4 natural BSE cases. Control piglets were similarly inoculated with saline. One challenged pig developed clinical signs after 69 weeks. Histopathological examination of the brain revealed spongiosis of the grey matter, and characteristic fibrils associated with TSE were detected by electron microscopy.


In experiments in 1979 (unpublished), 10 cattle were injected with scrapie-infected brain homogenate from sheep or goats, as well as being dosed orally. Three cattle developed neurological signs 27-48 months after inoculation, consisting of progressive difficulty rising, stiff-
legged stilted gait, incoordination, disorientation and terminal recumbency. From onset to terminal signs the disease lasted 1-2.5 months. PM examination revealed insufficient changes to confirm a clinical diagnosis of scrapie. A follow-up examination of the brains has revealed the protease-resistant protein also found in BSE-affected cattle. It is suggested that BSE may have occurred in the US under the clinical picture of the downer cow syndrome.


Epidemiologic data from 33 cases of BSE in Northern Ireland from November 1988 to December 1989 are analyzed and the findings compared with those from 9,000 cases in Great Britain between November 1986 and December 1989. The 2-year difference in the first confirmed cases and the low incidence in Northern Ireland are thought to be due to the low incidence of scrapie in the sheep population. Also, a proportion of the BSE cases in Northern Ireland may have resulted from a known importation of meat and bone meal from Great Britain in 1983.


Approximately 5.1% of all cattle herds in Great Britain have been affected by BSE. Among affected herds, 63% in England, 80.9% in Wales, and 88.1% in Scotland have had only one case.


A genetic study of 75 cases of BSE revealed that 73% of the cases had first or second degree relatives also affected. The number of common ancestors and the degree of relatedness of the affected animals in a multiple-case herd was no more than would be expected from the breeding structure of the herd. The data show that the disease itself is not simply inherited. However, there remains a real possibility that the susceptibility of individual animals to BSE is inherited.

1989

1988


TSE’s - General

1996


The author explains what nurses need to know about BSE and CJD.


1995

Darcel C. 1995. Reflections on scrapie and related disorders, with consideration of the possibility of a viral aetiology. Veterinary Research Communications 19(3):231-252

This discussion paper addresses the possibility of a viral aetiology of scrapie and related diseases, and emphasizes the need for broader understanding of the state of the immune system in animals with encephalopathy.


This is the third report produced by the Committee which was established in 1990. The objective is to summarize work done in the UK and the rest of the world, and to make this information available to as wide an audience as possible.

1994


Data on embryo transfer and the control of SE in sheep and cattle is being investigated in the US and the UK. In regard to sheep there are conflicting results. Washed embryos from experimentally infected sheep in the US have not transmitted scrapie to recipients. In contrast, in the UK and using unwashed embryos, scrapie did occur in the offspring. It is concluded that the question as to whether ET can be used to control natural scrapie is unresolved. Experiments with ET and cattle are expected to be completed in 2001.


Various methods of decontamination were tested for ability to inactivate BSE and scrapie. Only treatment with sodium hypochlorite proved effective in inactivating the BSE agent.
Austin AR, Simmons MM. 1993. Reduced rumination in BSE and scrapie. Veterinary Record

The ruminating behavior of BSE and scrapie suspects was compared with control cattle and
sheep. Ruminating time of the affected animals varied substantially from normality. It is
suggested that diminished rumination reduced food intake rather than the reverse.

DeArmond SJ. 1993. Overview of the transmissible spongiform encephalopathies: prion protein

Esmond TFG, Will RG. 1993. Transmissible spongiform encephalopathies and human

Hunter GD. 1993. Scrapie and mad cow disease: the smallest and most lethal living thing. New
York, USA. Vantage Press.

This book traces the history of scrapie, BSE, and the human SE’s, and describes the unusual
properties and nature of the scrapie agent.


Recent research on this topic is reviewed.

Kretzschmar HA. 1993. Human prion diseases (spongiform encephalopathies). Archives of
Virology Supplement 7:261-293.

Liberski PP. 1993. Subacute spongiform encephalopathies - the transmissible brain amyloidoses: a
comparison with the non-transmissible brain amyloidoses of Alzheimer type. Journal of
Comparative Pathology 109(2):103-127.

Verlag.
A comprehensive review of the pathogenesis and neuropathology of CJD, kuru, scrapie, and other spongiform viral encephalopathies, with mention of the bovine variety.


1992


This publication claims to bring together all the information available on the TSE’s of animals, including BSE, scrapie, TME, and SE’s in Cervidae.


1991


This symposium comprises 23 papers on SE’s, presented in 5 sections; 3 sections on CJD and GSS, 1 section on scrapie and BSE, and 1 section on nature of the agent. There are some papers on the epidemiology of scrapie in Slovakia, and scrapie and visna in the (former) USSR.

This book provides a comprehensive summary of the current status on the agents of CJD, BSE and related diseases.


1990


This seminar was attended by representatives from 18 countries, as well as the WHO, OIE, and the European Commission. Topics include BSE, human disease, disease agents, and research, among others.


1989


1987


Autoclaving scrapie infected mouse brain to 134-138°C for 18 minutes, recommended for decontamination of CJD infected brain, caused some tissue damage, but not sufficient to prevent useful qualitative and quantitative histopathological examination.

1986


1980

The paper describes the oral transmission of kuru, CJD, and scrapie to squirrel monkeys that had been allowed to eat brain, kidney, and spleen tissues from animals that had died with these diseases. This report provides the first evidence that virus in raw tissues from infected animals can induce subacute SE in squirrel monkeys.

1979


The papers in this book cover four diseases: kuru and CJD of man; scrapie of sheep and goats; and TME.
Accumulation of the prion protein PrPSc, a pathological and protease-resistant form of the normal host protein PrPC, is a feature of prion disease such as scrapie. To investigate the mechanism of PrPSc neurotoxicity, neural tissue overexpressing PrPC (normal protein) was grafted into the brain of Prp-deficient mice. These mice were then intracerebrally inoculated with scrapie prions. Only the graft tissue subsequently developed histopathologic changes characteristic of scrapie and accumulated higher level of PrPSc. At 16 months post-inoculation, the host Prp-deficient tissue showed no pathologic changes and was not damaged by the presence of the graft tissue PrPSc, even though substantial amounts of graft-derived PrPSc migrated into the host brain.


Safar J. The folding intermediate concept of prion protein formation and conformational links to infectivity. Current Topics in Microbiology and Immunology 207:69-76.


1995


The results from this study indicate that only a limited proportion of astrocytes proliferate in the experimental model of subacute spongiform encephalopathies and that microglia are probably postmitotic cells.


The pathology of spongiform encephalopathies is reviewed. It is concluded that a prion protein is released from the surface of neurons, diffuses through the extracellular space around infected cells where it accumulates and becomes aggregated as amyloid fibrils. It is likely that the accumulation of prion protein within the extracellular space is instrumental in causing nerve cell dysfunction and neurological disease.


In spite of the growing acceptance of the prion hypothesis, there is no explanation for the supposed ‘autocatalytic’ activity of this protein molecule. The authors’ molecular tumor hypothesis proposes that the prion protein is a genotoxin which interacts specifically with its homologous cellular gene introducing mutations which lead to aberrant processing and accumulation of the protein.


The results from this study support studies in man which show that specific amino acid residue changes within PrP can affect disease pathogenesis and transmission of TSE’s across species barriers.


This symposium comprises 7 reviews related to scrapie, kuru, CJD, fatal familial insomnia, GSS disease, and BSE.


This review describes prions as infectious pathogens that cause fatal neurodegeneration in man and animals and are composed largely, or entirely, of an aberrant form of the host-encoded prion protein (PrP). The conservation of the PrP primary structure among mammals provides the opportunity for prions to ‘jump’ between certain species. Prions may be able to emerge de novo either by overexpression of the PrP-encoding genes or by mutation of their coding sequences. It is therefore concluded that continual vigilance is required to preempt further epidemics of prion-induced disease.


1994


This journal issue contains the papers presented at a special meeting aimed to bring together the leading investigators in the field of prion diseases for extensive discussion and to provide an overview of the novel concepts inherent in prion diseases.


The species specific nature of an antigenic determinant previously discovered in the scrapie form of the prion protein (PrP) from cattle, sheep, and mice, was further investigated in normal PrP from these and other species. It is concluded that the region close to the actual or putative proteinase K cleavage sites of PrP seems to exhibit high structural variability among mammalian species.


There are 2 known polymorphisms of the coding region of the bovine PrP gene. An analysis of 370 cattle in Scotland detected no difference between the frequencies of these PrP genotypes in healthy cattle and cattle with BSE.


This study investigated the association between genotype and BSE in 56 BSE-affected animals and 177 unaffected animals. The data suggested that BSE-affected animals and their relatives were more likely to have the AA SSCP genotype than unrelated animals of the same breed or animals of different breeds.


A review.


1993


Based on the data presented, the authors conclude that BSE PrP and ovine and murine scrapie PrP can be distinguished from each other, and that these differences might help elucidate the species barrier effect.


The sequence of the coding regions of the PrP genes of the Arabian oryx and greater kudu were compared with the related sheep and bovine gene sequences. The oryx gene sequence was very closely related to that of the sheep; the greater kudu gene sequence was more closely related to the bovine. The effect that the gene sequences have on the transmission of SE to these antelope species is discussed.


Six brain regions from 11 cattle were examined for the presence of the abnormal isoform of the prion protein (PrPBSE). The highest concentrations of PrPBSE were found in the brain stem, where the greatest degree of spongiform change was observed. Since the transmission of prions across species seems to be restricted by differences in PrP sequence, the high degree of homology between sheep and bovine PrP (98%) correlates with the proposed cause of BSE.


This paper reviews current hypotheses on the pathogenesis of TSE’s in animals and man, and the authors’ preliminary studies to test the ‘protein only’ hypothesis. This hypothesis predicts that, in the absence of prion protein C, mice should be resistant to scrapie. The authors generated mutant mice, and briefly describe some preliminary studies on these mice.


1992


The author suggests that experimental evidence supporting the hypothesis of a membrane fragment as agent has not been taken into account, and proposes that a modified form of the membrane hypothesis could account for immunological as well as genetic aspects of these diseases.


The authors present arguments to support the hypothesis that spiroplasmas may be the agents responsible for spongiform encephalopathies such as scrapie, BSE, kuru, and CJD, or that they may be cofactors of such prion-associated disorders.


The ultrastructural neuropathology of mice experimentally inoculated with brain tissue of nyala or kudu affected with SE was compared with that of mice inoculated with brain tissue from cows with BSE. The nature and distribution of the pathological changes were similar irrespective of the source of inoculum or whether the inoculum was from fresh or fixed tissue. These changes are described.


The causation, structural origin, and mechanism of formation of spongiform lesions in transmissible encephalopathies are unknown. It is suggested that spongiform change is brought about by cytoskeletal disruption in neuronal processes caused by liberation of hydrolytic enzymes from lysosomes overloaded with the abnormal isoform of PrP and that the lysosomal system is probably acting as the bioreactor for processing of normal PrP to the abnormal isoform.


The formation of autophagic vacuoles in rodents with experimental scrapie and CJD may contribute to neuronal degeneration and ultimately to neuronal loss.

It is concluded that axonal and myelin pathology is a widespread phenomenon and the differences between panencephalopathic CJD and polioencephalopathic BSE and scrapie are only quantitative.


The ultrastructural neuropathology of BSE was compared to that of experimental scrapie and CJD; except for intraneuronal inclusions, all of the ultrastructural features of BSE resembled those found in scrapie and CJD.


Examination of sections from the cerebral cortex of scrapie-infected hamster brains detected characteristic circular tubulofilamentous particles, identical to those previously described in both experimentally induced scrapie in mice and hamsters and natural scrapie of sheep, BSE, and CJD and mice and chimpanzees infected with CJD.


Tubulofilamentous particles and scrapie-associated fibrils (SAF) are ultrastructural markers, while protease-resistant protein (PrP) is a molecular biological marker for all SE’s. Review of all published work has suggested that PrP molecules aggregate to form a 3-dimensional SAF. Further reports have suggested that a single-stranded DNA wraps round SAF and acquires an outer protein coat to form tubulofilamentous particles.


The term prion was introduced in 1982 to distinguish the proteinaceous infectious particles that cause scrapie and other SE’s from both viroids and viruses. This book brings together papers that cover many aspects of prions, including terminology, history, scrapie, kuru, and transgenics and animal models.


1991


A review of the pathogenesis of SE’s which hypothesizes how the infectious agent causes a genetic disease.

Dealler S. 1991. Transmissible spongiform encephalopathy agents as crystalline forms of the prion protein (PrP) that multiply by allowing normal metabolic forms of PrP to join the crystal. Medical Hypothesis 36(2):131-134.

The prion protein (PrP), found only in the brain of animals infected with TSE, is a modified form of a normal protein (PrPn) produced from the genome of the animal. The finding of fibre-like structures and the rapid turnover of PrPn may mean that normal biochemical pathway PrPn forms can join a crystal seed of PrP to produce these fibrils. This hypothesis, that the modification of PrP is physical rather than chemical, avoids the major problems with theories of PrP as the infective agent.


The sequence of different forms of the bovine PrP gene is reported. Eight of 12 cattle were homozygous for genes with six copies of the Gly-rich peptide, while four where heterozygous. Two confirmed cases of BSE occurred in homozygous animals.


Results showed that scrapie-associated fibrils from scrapie-affected hamsters can be ultrastructurally distinguished from those of CJD-affected mice, an observation that presumably reflects differences in their respective host-encoded amyloid protein subunits.


The results of this study show the proteolytic resistance of the membrane-bound infectious isoform and also indicate the presence of a different, apparently disease-induced mechanism of membrane interaction in the scrapie- and CJD-infected microsomal and synaptosomal membranes.
1990


This paper reviews the genetic control of neurodegenerative diseases that affect animals (scrapie, BSE, TME, and CWD) and humans.


Tubulovesicular structures appeared in mice inoculated intracerebrally or intraocularly with CJD agent 5 weeks before the onset of clinical signs, in hamsters infected with scrapie agent before the appearance of other neuropathological changes.


The investigation used immunocytochemical techniques to identify and localize heparan sulfate proteoglycan in human cases of GSS and CJD, as well as in experimental scrapie of hamsters. The specific accumulation in the amyloid deposits of both the prion diseases and Alzheimers disease suggests that a common mechanism may occur in the pathogenesis of amyloidosis in each of these diseases.


A letter followed by 5 others on the same subject, one of which, by SC Arya, suggests that the cases of CJD in India may have resulted from scrapie in rabies vaccine made from sheep’s brain.

1989


1988


Antiserum to SAF protein was reacted with brain sections from scrapie-infected mice, two familial cases of transmissible dementia, and three cases of Alzheimer’s disease (AD). Specific
immunostaining of cerebral amyloid plaques occurred in the scrapie-infected mice and in the two familial cases of transmissible dementia. No immunoreactivity was detected in the three cases of AD. The results suggest that SAF, the causative pathogenic agent, and extracellular deposits of amyloid in the brain are closely related.


There is evidence that prions contain protease-resistant proteins, designated PrPSc, encoded by a cellular gene. Clonal cell lines which synthesize PrPSc molecules possessed scrapie prion infectivity as measured by bioassay; clones without PrPSc showed no infectivity. Detection of PrPSc molecules in scrapie-infected cells supports the contention that PrPSc is a component of the infectious scrapie particle.


1987


Golgi impregnation studies showed that neurons in the scrapie-infected brains of hamsters contained varicose swellings and diminished numbers of dendritic spines. In this investigation, less than 2% of the control cells showed these varicosities, while greater than 80% of the scrapie
dendrites showed varicosities. These changes in scrapie are similar to those reported in CJD and Alzheimer’s disease in man.

---


This book consists of 21 chapters which include the following topics: the prion hypothesis; terminology; prion transmission and replication; purification of scrapie prions and their ultrastructure; properties of scrapie protein, immunology; molecular biology of prions; and the pathology of various diseases.

---

1985


Antisera prepared in rabbits and, for the first time in mice, against SAF protein from hamster brain were used for a detailed analysis of SAF proteins from hamsters, mice, and patients who had died from CJD. In control material from healthy brain SAF protein was absent.

---


---


---


The lesion profiles of spongiform change and gliosis in the hamster occurring after intracerebral inoculation of scrapie virus are calculated and compared to the lesion profile of spongiform change of scrapie in mice and of scrapie and CJD in the squirrel monkey. The profile of scrapie in hamsters differs considerably from that of a closely related strain of scrapie in mice, and both differ from scrapie and CJD in the squirrel monkey.


Scrapie-associated fibrils (SAF) are reported in the brain of scrapie-infected hamster and squirrel monkeys, human cases of CJD, and a kuru-infected squirrel monkey. These fibrils were not found in a series of control brains from man and animals. SAF were expected but not found in one case of naturally occurring scrapie in sheep, two cases of kuru and one case of CJD in squirrel monkeys.


Scrapie-associated fibrils (SAF) that have been described in the brain of scrapie affected mice and hamsters were found in the brain of a human case of CJD and the brain of a case of GSS; SAF were not present in three control brains. SAF were also found in the brains of clinically affected guinea pigs, hamsters and mice in which CJD tissue was passaged, and also the spleens of scrapie affected mice and a CJD affected hamster.


Because the novel properties of the scrapie agent distinguish it form viruses, plasmids, and
viroids, a new term ‘prion’ is proposed to denote a small proteinaceous infectious particle which is resistant to inactivation by most procedures that modify nucleic acids.

1973


1963

TSE’s - Diagnosis

1996


The authors detected prion protein in tonsils of sheep in the preclinical stage of scrapie, long before the onset of clinical signs. They suggest that this approach might be a diagnostic method for other TSE’s.

1995


Using an improved extraction method and Western blot detection, TSE-specific amyloid was found in various parts of the CNS of hamsters orally infected with scrapie, squirrel monkeys orally infected with kuru, sporadic CJD and scrapie, of human patients with sporadic CJD, of sheep with natural scrapie, and of a cow with BSE. The results show the potential of the method for the routine diagnosis of TSE.


Brain sections were analyzed from confirmed BSE cases and 4 cattle that were suspect but histologically unconfirmed. There was a BSE-specific staining pattern. It is concluded that
immunohistochemistry for the prion protein is an elegant and time saving alternative to scrapie associated fibril isolation and electron microscopy.


This report describes the expression of a PrP subunit protein and its application in the production of diagnostically useful antisera.


Tissue samples from the brains of 50 sheep with natural scrapie and 20 sheep without histopathological signs of scrapie were treated with formic acid and hydrated autoclaving. A scrapie-associated cellular prion protein (PrPSC) was detected using an antipeptide antisera. PrPSC was located in the brains of all sheep with scrapie; no immunostaining occurred in sheep without scrapie.


1994


The efficacy of 3 pretreatment techniques for the detection of prion protein in BSE-affected brain tissue was compared. Hydrated autoclaving of section before PrP immunolabelling detects widespread sites of abnormal PrP deposition in the brain, allowing detailed study of the form and distribution of the protein in routinely fixed CNS tissue affected with BSE.

To improve the diagnosis of TSE, a protocol was developed which allows several samples to be tested for TSE within 24 hours, starting with only 10-100 mg of brain tissue from different species.


It was found that lumbosacral cerebrospinal fluid (CSF) can safely be collected from cattle in sternal recumbency or standing animals restrained in cattle stocks. There were no changes in CSF composition in BSE, which permitted elimination of those diseases which provoke an inflammatory response from the differential diagnosis.


A statistical comparison was made between the results of the statutory neurohistopathological method for post-mortem diagnosis of BSE and the detection of abnormal brain fibrils. It is concluded that, despite the potentially greater specificity of fibril detection, a reliance on fibril detection alone may result in some false negative diagnoses, probably owing to inadequate sampling of the tissue.

1993


Extracts of brain and peripheral tissues were tested for the presence of disease-specific PrP (PrPD) fraction. 14 brains from suspected BSE cases were examined, 12 were subsequently confirmed to have BSE. Readily detectable amounts of the PrPD were found in brain extracts from all 12 BSE cases but not in the other 2 brains. PrPD was also detected in brain extracts of 3 naturally and 3 experimentally scrapie-infected sheep. No PrPD was found in cattle and sheep controls.


Brain tissue from 3 cases of metastatic septic encephalitis by fungi, 6 cases of CJD, and 1 case of GSS were examined. A positive reaction to the prion protein antiserum was seen in the case of GSS, 1 of the CJD cases, and in the fungi. Reasons for the unspecific positive reactions with this antiserum are suggested.


The effect of autolysis on the electron microscopic detection of the characteristic abnormal fibrils, originally called ‘scrapie-associated fibrils’, was investigated. The authors conclude that fibril detection is of diagnostic value in BSE when post-mortem autolysis renders CNS material unsuitable for histopathology.


This review outlines the diagnostic criteria for the case definition of TSE’s, with reference to BSE and to the similar diseases in exotic birds and domestic cats.

The repetitive capillary micro-electrolysis technique, used in Alzheimer’s disease, revealed significant differences in the urinary peak between sheep with scrapie and healthy sheep. The responsible substance is considered to be the same as that detected in Alzheimer’s disease.


The problems encountered in diagnosing BSE and its differential diagnoses are reviewed. It is emphasized that although a positive diagnosis of BSE is only possible following histological examination, several epidemiological and clinical symptoms may suggest the disease.


The author reports on the potential use of electroencephalographic (EEG) pattern as an antemortem diagnostic test.

1990


Fibril detection was compared with histopathological diagnoses in the brains of 167 cattle. The study confirms the specificity of fibril detection for BSE, shows that the ease of fibril detection depends on anatomic region sampled and suggests an association between PrP accumulation and vacuolar changes in certain neuroanatomic areas.


Human Spongiform Encephalopathies

1996


This paper describes the clinical and histopathological features of a case of a new CJD variant in France. The patient was a mechanic and had no particular contacts with cattle. The authors suggest that the case questions the possible causal relationship between BSE and the new CJD variant.


The authors report that comprehensive prion protein gene sequencing was done on 8 of the 10 cases of variant CJD, and that no mutations, known or new, were detected. Since all known families with one of the familial forms of SE have been shown to have coding mutations of the prion protein gene, the authors conclude that these new variant cases can be excluded as being inherited forms of prion disease with previously unrecognized coding mutations.


1995


The results of this study support the hypothesis that impairment of slow axoplasmic transport is a common pathogenic mechanism for CJD and many other neurodegenerative conditions.


The authors report two cases of CJD in a 48 year old woman and a 60 year old man. A review of the medical literature suggests that these constitute the fourth and fifth cases reported in Venezuela.


The authors report the successful transmission of the disease to experimental animals, placing FFI within the group of infectious cerebral amyloidoses.


1994


Several French teams including clinicians and researchers have created a group within the European network for the study of CJD and other humans SE’s. The main objectives are to monitor the incidence of the disease and to search for possible risk factors with a case-control study.


Transgenic mice were constructed in which a segment of mouse prion protein was replaced with the corresponding human sequence. All of the transgenic mice developed neurologic disease approximately 200 days after inoculation with brain homogenates from CJD patients.
1993


A report of a case of CJD in a previously healthy 54-year old dairy farmer in the UK who was directly exposed BSE cases (3) on his farm.


1991


1990


A comparison was made of the effects of experimental intracerebral inoculation into marmosets of brain homogenates from a case of CJD and from a case of GSS syndrome. There were only minor and inconsistent differences between the disease transmitted from CJD compared with GSS.


A discussion of the high rate of CJD in Slovakia.


1989


1988


Evidence for and against the hypothesis that spiroplasmas as opposed to subviral prions (e.g. the scrapie agent) may be involved in the aetiology of CJD is discussed.


Scrapie infectivity of this hamster adapted strain is reduced 5-6 logs after filtration through 100,000 MW cut-off filter and overnight treatment of 6M urea. These steps, applied to purified human growth hormone (hGH), increase the margin of safety of hGH.

1987


1986


1985


1984


Four patients with CJD are described, who had a history of eating the brains of wild goat or squirrel. Those patients indicate the possible acquisition of CJD by ingestion of the agent from a presumptive reservoir in the CNS of wild animals
1983


1981


1980


1979


1977


1973

Transmissible Mink Encephalopathy

1995


Cattle are susceptible to experimental infection with the Stetsonville isolate of the TME agent. Susceptibility to other TME isolates, as well as to cattle-passaged Stetsonville agent and cattle-passaged scrapie agent was investigated. Clinical signs of neurological disease appeared in each steer of every group between 15 and 25 months after inoculation. The neurohistological changes in the steers inoculated with cattle-passaged scrapie agent were slight and subtle.

1994


The black ferret and the closely-related mink showed differences in susceptibility to challenge with the Stetsonville TME agent: the incubation period was longer in ferrets (28-38 months) than in mink (4 months). Comparison of amino acid sequences in the PrP gene identified 6 silent base changes and 2 amino acid changes between mink and ferret. These changes may indicate the region of PrP that is responsible for the species barrier effect.


Inoculation of the Stetsonville TME agent into mice has identified 2 strains of the TME agent having distinct biological properties and producing disease-specific proteins with different physicochemical properties. Although several strains of the sheep scrapie agent have been identified in Great Britain, this is the first indication that agents producing TSE’s in the US also are capable of producing distinct strains.

Standard dark mink were intracerebrally inoculated or fed with brain homogenate from 2 British cows affected with BSE. Neurological signs were seen in the mink an average of 12 months after inoculation and 15 months after feeding; signs were unlike the classical clinical picture of TME. These results extend the host range of the BSE agent and show for the first time the experimental oral transmission of mink with TSE agent from a naturally infected ruminant species.

1992


Experimental transmission of the Stetsonville strain of TME to hamsters resulted in 2 distinct syndromes that diverge by the third hamster passage. The syndromes differed with respect to clinical signs, incubation period, brain titre, brain lesion profile, and pathogenicity in mink. Hamster TME passaged back into mink showed that only one of the strains retained mink pathogenicity.


Mink PrP gene was investigated; it shows similarity of 84 to 90% with the sequences of the PrPs of other mammalian species. It remains to be determined whether these differences in the primary structures of PrP will explain the peculiar host range of TME (the experimental host range of TME includes sheep, cattle, monkeys, and hamster, but not mice).


1991


The authors conclude that it is doubtful that cattle are the primary source for TME, and that little evidence exists to support an increasing risk of TME in the US.
Epidemiological investigation of a new incident of TME in Stetsonville, Wisconsin, USA, in 1985 showed that the mink rancher had never fed sheep products to his mink but had fed them large amounts of products from fallen or sick dairy cattle. To investigate the possibility that this occurrence of TME may have resulted from exposure to infected cattle, two bull calves were injected intracerebrally with mink brain from the Stetsonville ranch. Each bull developed a fatal SE, and both bovine brains passaged back into mink were highly pathogenic by either intracerebral or oral inoculation.

1987


1970


1968


A disease of ranch mink that occurred in Ontario in 1963 is described - it was diagnosed in retrospect as transmissible encephalopathy.

Mink injected with brain suspensions from natural cases of TME developed the disease 5 months after intramuscular inoculation. Following alimentary infection a lengthened incubation period of about 8 months was observed. Although encephalopathy is poorly contagious among mink, field evidence suggests that on occasion the disease can be acquired by cannibalistic ingestion of flesh from diseased animals.


A description of several outbreaks of the disease in Wisconsin. The first case occurred in 1947; a few comparatively mild outbreaks occurred in 1961; and there were 2 more severe cases in 1963.
Scrapie

1996


The investigation studied the maternal transmission of scrapie in sheep by using embryo transfer to examine the viability of highly susceptible offspring derived from scrapie-affected and uninfected donors. The study also examined the effect of washing the embryos. Scrapie occurred in both washed and unwashed embryo-derived progeny from both groups of donor ewes.


The authors present data that suggest that the scrapie agent replicated in mites and that mites may represent a self-sustaining reservoir for scrapie-like agents.

1994


The study’s objective was to investigate whether polymorphisms in the PrP gene are directly correlated with survival time. Sheep of different PrP genotypes were challenged with scrapie or BSE and survival time and incidence of disease were monitored. Genotype analysis showed that dimorphisms of the ovine PrP gene correlated with control of disease and modulation of incubation time.


The results of this study show that astrocytes are a target for the scrapie agent even in the early temporal evolution of the disease. The changes they undergo clearly implicate astrocytes in the pathogenesis of scrapie.


The authors conclude that it is likely that sheep-to-sheep transmission has taken place, and that a new type of scrapie disease has been spreading in Japan.

1993


1992


A detailed review is presented of the history, geographical distribution, cause, epidemiology, clinical features, pathogenesis, pathology, diagnosis, prevention, control and economic effects of scrapie in sheep. National efforts to control scrapie in various countries are outlined.


A description of the neuropathology of the brains of 20 goats affected with natural scrapie received at the Central Veterinary Laboratory in the UK.


1991


The author recommends a mandatory histological screen for scrapie in a proportion of sheep (10-20%) in all flocks used to produce sheep-brain rabies vaccine to prevent the spread of viruses which can cause SE’s in India.


Fluid from a scrapie-infected hamster brain homogenate was mixed with soil, packed into perforated petri dishes that were then embedded within soil-containing pots and buried in a garden in the Washington, DC area for 3 years. Between 2 and 3 log units of the input infectivity of nearly 5 log units survived this exposure, with little leaching of virus into deeper soil layers.


This paper reviews studies in the UK in which sheep were divergently selected for scrapie resistance, and explains how these led to the identification of the gene, Sip, which determines susceptibility to the disease.

Three goats which came from a flock in which scrapie had previously been suspected showed histologic signs of SE patterns. Electron microscopy revealed clusters of scrapie-associated fibrils.


Extracts from the cervical spinal cord and from the medulla, thalamus, cerebellum and cerebral cortex of the brains of 10 scrapie-confirmed sheep were examined for the presence of scrapie-associated fibrils. Characteristic fibrils were observed in all the extracts except for that from the thalamus of one sheep; no fibrils were found in any extracts from 3 control sheep.

1990


In order to confirm the clinical and histological diagnosis of scrapie and to determine the infectivity titre of the scrapie agent in the brain of a naturally infected Suffolk sheep, 123 white Swiss mice were inoculated intracerebrally with dilutions of sheep medulla oblongata suspensions.


This very comprehensive book describes viral diseases, including a chapter on scrapie.


An anonymous, self-administered questionnaire was used in two independent surveys to try to determine the prevalence of scrapie in the national sheep flock. The disease was recorded in 35 counties in England and Wales. About a third (26.5 and 37.3%) of respondents owning 100 or more sheep indicated that they had seen sheep with scrapie in their flocks. The incidences of clinical cases recorded in affected flocks in the two surveys were 0.5 and 1.1 cases/100 ewes/year.


The authors studied ten years of the records of a sheep flock in which scrapie was endemic. Each year, scrapie had appeared in 20% of the sheep retained on the farm. The aim of the study was to
discover if eating meat from scrapie sheep produced CJD in humans, but the work shed no light on this.


1988


Foster JD, Dickinson AG. 1988. The unusual properties of CH1641, a sheep-passaged isolate of scrapie. Veterinary Record 123:5-8.


The author recounts his personal involvement with scrapie in the UK from 1939 to 1988.

1987


1986


The findings of this study reduce the possibility that scrapie is a DNA virus.


1985


Hamsters developed scrapie 100-160 days after eating either scrapie-infected hamsters or infected brain. The clinical signs and neuropathology of scrapie transmitted by cannibalism were identical to those observed after intracerebral or intraperitoneal inoculation of the agent. Oral transmission of scrapie appears to be extremely inefficient. Cannibalism requires a dose of the scrapie agent about 109 times greater than that needed to produce the disease by intracerebral injection for comparable periods of incubation.
1983


1982


1981


1980

1979


1977


1976


1975


1964


1963


1962


1961


SE’s in Other Species

1996


This letter reports a case of SE in a rhesus monkey in a zoo in Montpelier, France, in 1992. The animal was born in a zoo in the UK in 1982 and was acquired by the French zoo in 1986. The animal had been fed standard monkey feed which contains meat products declared fit for human consumption. This is the first reported case of spontaneously developed SE in a monkey, and the authors suggest that the feeding of this monkey with animal protein raises the possibility of cross-species transmission of the disease through contaminated feedstuff.

1995


This appears to be the first case of FSE in a domestic cat outside of the UK. The cat had been fed several imported commercial dry cat food products. There was no genetic link or any form of contact with FSE cases in the UK.

1994


Mice inoculated with brain material from 3 cats with FSE showed progressive neurological signs similar to those in mice inoculated with scrapie or BSE. The lesion profile in the brains of mice inoculated with FSE was similar to that observed in mice inoculated with BSE, rather than scrapie. It is suggested that because of similarities of results in mice inoculated with FSE and BSE, they probably arose from a common source.

Since 1986, SE has been diagnosed in 19 captive wild animals of 8 species at or from 8 zoological collections in the British Isles. The affected animals have comprised members of the family Bovidae and members of the family Felidae. In addition, 3 cases of SE of unknown aetiology have been reported in ostriches from 2 zoos in Germany. Three features suggest that some of these cases may have been caused by the BSE agent: they were temporally and geographically coincident, it is possible that they were exposed to feeds containing ruminant-derived protein, and results of mouse assays were similar to results of inoculating mice with BSE brain tissue.


A 39-month old female greater kudu, which had been transferred to Regent’s Park Zoo 27 months earlier, was destroyed in November 1992 after showing progressive neurological signs consistent with SE for 8 weeks. SE was confirmed histologically. The kudu had apparently not eaten feeds containing ruminant-derived protein, although her dam had been exposed to such feeds. The kudu had contact with 2 other kudu in which the disease had been diagnosed. The possible modes of transmission are discussed.

1993


Three further cases of SE in greater kudu at the Zoological Society of London, none of which was thought to have access to feed containing ruminant-derived protein, are reported.


The results of this study support the clinical and pathological diagnosis of the disease and provide further evidence that CWD belongs to the subacute SE’s.


Ultrastructural neuropathological findings of CWD are described. Similar findings have been previously observed in scrapie-infected hamsters and CJD-infected mice, BSE, and CJD.
indicating the CWD in captive mule deer belongs to the subacute SE’s.


A small herd of greater kudu has been maintained at the Zoological Society of London since 1970. SE has been diagnosed in 5 out of the 8 animals born in this herd since 1987. With the possible exception of the first case, none of these is thought to have been exposed to feeds containing ruminant-derived protein. The pattern of incidence suggests that greater kudu are very susceptible to the disease and that natural lateral transmission may have occurred among them.


A novel scrapie-like SE was first seen in a domestic cat in 1990 and since then 23 further cases have been recorded in the UK. This review covers clinical signs, pathology, the presence of fibrils and prion proteins, and transmission.


The pathology of the CNS of 9 mule deer and 6 elk with CWD is described. Lesions of CWD were qualitatively comparable to those of scrapie, BSE, TME, and the human SE’s. Topographical distribution and lesion severity of CWD were most similar to those of scrapie and BSE.


Between April 1990 and February 1992, a total of 24 cases of FSE was reported in the UK. Most affected cats were between 4 and 9 years of age. There were more males than females. Most cats were non-pedigree and came from a wide range of geographical locations throughout the UK. The first signs to be noted were often changes in behavior. All affected cats had been fed a variety of foods ranging from proprietary cat foods to table scraps. So far no cases of FSE have been reported in domestic cats outside the UK.

A 19-month-old greater kudu, whose dam had died 15 months earlier with SE, was diagnosed with SE. The animal was born 9 months after the statutory ban on the inclusion of ruminant-derived protein in ruminant feeds and, as no other possible sources of the disease were apparent, it appears likely that the infection was acquired from the dam.


A 5.5 year-old cheetah, which was born in England in 1986 and was imported to Australia in 1989, was diagnosed with SE in early 1992. The cheetah probably ingested the infective agent while still in England. This is the first diagnosis of SE in a cheetah and of SE in a zoo animal outside the UK.


The brains from 18 cats were examined for the presence of the fibrils and modified PrP protein which are markers for scrapie-like diseases. Fibrils and modified PrP protein were found in the brains of the 5 cats with FSE and in one of the cats with neurological signs but no histopathologic changes in the CNS. Fibrils were present in the absence of modified PrP in the brains of 2 cats, one with neurologic signs and confirmed meningioma, and one with no neurologic signs and a normal brain.


The report describes chronic wasting disease of mule deer and Rocky Mountain elk in Colorado and Wyoming and includes sections on the geographical distribution, economic implications, aetiology, epidemiology, clinical signs, pathology, diagnosis, and prevention and control.

In 1991 a captive adult puma developed clinical signs suggestive of SE; histopathologic examination demonstrated SE. Her diet had consisted of chicken and rabbit carcasses and part of cattle carcasses. She had no known access to sheep or goat meat. This is the first confirmed cases of a scrapie-like SE described in a non-domestic cat in the UK.

1991


The topographic distribution of amyloid plaques reactive to antibodies prepared against scrapie amyloid in CWD-affected captive mule deer is described. The results confirm that CWD in captive mule deer belongs to the subacute virus SE’s.


The diagnosis of 7 cases of naturally occurring scrapie-like encephalopathy in the domestic cat are reported. The cases were diagnosed on histopathological examination of the brain; modified host protein was also demonstrated in the brain of each case both by immunoblotting and by detection of fibrils.


Clinical signs and post mortem examination including histopathology of an adult female ostrich destroyed due to central nervous and locomotion disorders are described. Signs of SE are found; aetiology remained unknown. The disease is compared to the similar findings in mammals with BSE/scrapie.


This paper describes the clinical and pathological findings in 5 cats with SE.


An eland (Taurotragus oryx) born at a zoo in England in 1987 and kept on the zoo premises developed clinical signs consistent with SE in late 1989; histopathological examination of the brain revealed a SE. The animal may have been exposed to the causal agent of BSE or scrapie through processed animal protein fed in the diet. This appears to be the first report of a SE in an eland.


Clinical, pathological and epidemiological details of scrapie-like encephalopathies are described in an Arabian oryx and a greater kudu in a zoo in London, UK. Scrapie-like SE’s have now been described in five species of exotic artiodactyls in Britain indicating a hitherto inapparent wider range of ruminant species as natural hosts for these diseases.


In 1990 a 7.5 year-old cat was confirmed to have a SE. The cat had been fed throughout life on a proprietary canned diet, supplemented with dry cat food and fresh cooked chicken.


This is the first report of a naturally occurring disease in a domestic cat with microscopic changes consistent with the TSE’s due to unconventional viruses.
Risk Assessment, Surveillance, and Cases of BSE Outside the United Kingdom

1996


US officials are taking aggressive measures to ensure that BSE never has the chance to cause a problem in the US.


The affected cow was born in August 1986 and raised on its farm of origin in the UK. She was 1 of 8 Salers cattle imported into Canada from the UK in January 1987.


The United States has already addressed much within the seven international recommendations to minimize the potential transmission of BSE.


This report provides a follow-up to the 1993 report.

1995

Schlachtrindern zum Vorkommen von BSE in Italien unter Beachtung unspezifischer neuronal Vakuolen. Schweizer Archiv fuer Tierheilkunde 137(3):101-103. (In German)

378 brains of normally slaughtered cattle 2-10 years of age from slaughterhouses all over Italy were examined histologically. None showed the typical lesions of BSE.

1994


The policies implemented by the US rendering industry to reduce the risk of disease transmission from sheep offal to other animals are described.


After the occurrence of BSE in Switzerland in 1990, extensive epidemiological investigations and risk factor analyses were carried out. Statistical data on meat and bone meal traded from 1985 to 1989 were analyzed, including the export of meat and bone meal from the UK. It is suggested that imported material was the cause of BSE in Switzerland.


This article reports on the first German case of BSE diagnosed in a Scottish Highland cow. The affected cow was imported into Germany before the import ban for cattle from the UK was implemented.


A surveillance program was implemented in the field and in abattoirs. A total of 1,019 brains from high risk animals (dairy cows >5 years of age) were examined; all cases were negative for BSE.

Refers to the US.


Genetic resistance to BSE may be determined in part by alleles of the prion gene. The frequencies of prion allelic structural variants were determined in 210 Holstein and 46 Hereford bulls in the US. The frequency of a variant of the prion gene that has been found in BSE-infected bulls in the UK was .97 in the Holstein and .99 in the Hereford bulls. Near uniformity of the US cattle population for this allele may constitute a risk factor if an association of pion genotype with BSE susceptibility is established in the future.


The pathological findings in the brains of 36 bovine cases with neurological symptoms are described. No lesions of vacuolation or any other degenerative change compatible with BSE were observed.


BSE has been diagnosed in a beef cow in Alberta, Canada. The animal was of British origin and is the first to be diagnosed with the disease in North America. The Canadian government has ordered 300 cows to be slaughtered, 270 of which were from one ranch in Alberta, and the remainder from 9 farms in Ontario.


Clinical signs similar to SE were observed in two heifers. Histological changes were present in the liver, kidney, and brain. Severe, symmetrical, spongiform changes and gliosis of white matter
were observed at all levels of the brain. It is concluded that the SE is related to the hepatic vasculopathy.


Brain tissue from 19 cattle with clinical symptoms similar to SE were examined histologically as part of the BSE screening program started in New Zealand in 1991. None was found to have BSE, but 2 were diagnosed with hepatic encephalopathy. In cattle in New Zealand the main cause is Senecio jacobaea poisoning.


Scientists guard against BSE in the US through research and surveillance.


This symposium was held to inform producers and agricultural business on the present BSE outbreak in the UK and new experiments in the US testing the susceptibility of cattle to inoculation with American sources of the sheep scrapie agent.


This report comprises 4 articles on: an update on the disease in the UK; an update of risk factors for BSE in the US; a review of BSE surveillance in the US; and an assessment of the possible role of nonambulatory cattle in TSE in the USA.

1992


In the program for the surveillance of SE’s in Belgium, between May 1990 and February 1992
examination of 223 cattle, 20 wild ruminants, 47 dogs and 66 cats gave negative results to histological examination of the brain.


The PrP gene from 65 cattle representing 14 breeds was analyzed; results are presented.


A special project was set up in May 1990 to survey cattle in the US for lesions of BSE. Specimens from 117 cattle representing 20 states and Puerto Rico were examined and none contained lesions typical of BSE.

1991


Because of the widespread use of Semple-type rabies vaccine produced from sheep brains there is a possibility of the number of SE cases in man increasing due to transmission of the scrapie agent. The author suggests a number of measures that should be adopted to monitor prevalence of CJD in those vaccinated with the Semple-type vaccine.


The first case of the disease reported in Switzerland was a 6-year-old cow with extreme anxiety and slight ataxia. Spongiform lesions were present in the brain. The origin of the disease was untraceable.


This report summarizes an analysis of the different epidemiological factors believed conducive to the introduction of BSE in the UK and applies them to the US (Walker KD, Hueston WD, Hurd HS). The analysts concluded that the potential risk of BSE at the aggregate level is substantially less in the US than in the UK.


The authors conclude that US dairy cattle, which have the greatest risk of exposure to the scrapie agent in the US, appear to be at much lower risk for development of BSE than British dairy cattle, even under the worst-case scenario.

1990


BSE is reported in two cows, both of which were born in 1983 on the same farm in England and exported to Oman in 1985. Clinical signs of the disease appeared in early 1989. These cases were the first to be reported outside the UK and Ireland. The history of the animals indicates that they were exposed in the UK to feedstuffs contaminated with a scrapie-like agent.

Reports on the number of cases of BSE in the Republic of Ireland (the first case was confirmed in January 1989) and measures taken to combat BSE.


Import of live cattle and zoo ruminants from the UK into the US was banned in July 1989. No meat or bone meal was being imported into the US. About 500 cattle had been imported into the US from the UK since 1981, and those still alive were under surveillance.

1989


In October 1988, a retrospective study of all histological brain material available in New Zealand was conducted to look for evidence of BSE. No brains with lesions consistent with BSE were detected. The paper also describes the on-going surveillance for BSE in New Zealand.

# Index of First Authors

<table>
<thead>
<tr>
<th>Name</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adams DH</td>
<td>6</td>
</tr>
<tr>
<td>Aguzzi A</td>
<td>17</td>
</tr>
<tr>
<td>Aldhous P</td>
<td>14, 24, 90</td>
</tr>
<tr>
<td>Aldridge BM</td>
<td>9</td>
</tr>
<tr>
<td>Alper T</td>
<td>44</td>
</tr>
<tr>
<td>Andrews</td>
<td>9</td>
</tr>
<tr>
<td>Andrews AH</td>
<td>74</td>
</tr>
<tr>
<td>Arya SC</td>
<td>75, 95</td>
</tr>
<tr>
<td>Asher DM</td>
<td>96</td>
</tr>
<tr>
<td>Ashworth SW</td>
<td>12</td>
</tr>
<tr>
<td>Austin AR</td>
<td>29</td>
</tr>
<tr>
<td>Baker HF</td>
<td>20, 64, 65</td>
</tr>
<tr>
<td>Barlow RM</td>
<td>23, 24, 71</td>
</tr>
<tr>
<td>Baron H</td>
<td>50</td>
</tr>
<tr>
<td>Bartz JC</td>
<td>69</td>
</tr>
<tr>
<td>Basset H</td>
<td>97</td>
</tr>
<tr>
<td>Beardsley T</td>
<td>65</td>
</tr>
<tr>
<td>Beekes M</td>
<td>55</td>
</tr>
<tr>
<td>Bendheim PE</td>
<td>74</td>
</tr>
<tr>
<td>Bennett AD</td>
<td>44</td>
</tr>
<tr>
<td>Berg LJ</td>
<td>39</td>
</tr>
<tr>
<td>Bessen RA</td>
<td>36, 70</td>
</tr>
<tr>
<td>Biernat W</td>
<td>36</td>
</tr>
<tr>
<td>Blakemore WF</td>
<td>32</td>
</tr>
<tr>
<td>Bobowick AR</td>
<td>68</td>
</tr>
<tr>
<td>Bockman JM</td>
<td>50</td>
</tr>
<tr>
<td>Bode L</td>
<td>52</td>
</tr>
<tr>
<td>Bolis CL</td>
<td>6</td>
</tr>
<tr>
<td>Bons N</td>
<td>85</td>
</tr>
<tr>
<td>Boothby CB</td>
<td>9</td>
</tr>
<tr>
<td>Borras T</td>
<td>79</td>
</tr>
<tr>
<td>Bradley R</td>
<td>3, 5, 13, 28, 30, 31</td>
</tr>
<tr>
<td>Brandel JP</td>
<td>63</td>
</tr>
<tr>
<td>Brandner S</td>
<td>35</td>
</tr>
<tr>
<td>Bratberg B</td>
<td>85</td>
</tr>
<tr>
<td>Bridges V</td>
<td>70</td>
</tr>
<tr>
<td>Brochier B</td>
<td>94</td>
</tr>
<tr>
<td>Brotherstone JG</td>
<td>82</td>
</tr>
<tr>
<td>Brown DR</td>
<td>93</td>
</tr>
<tr>
<td>Brown P</td>
<td>30, 32, 47, 64, 68, 75, 93</td>
</tr>
<tr>
<td>Bruce M</td>
<td>19, 78</td>
</tr>
<tr>
<td>Name</td>
<td>Page(s)</td>
</tr>
<tr>
<td>-----------------</td>
<td>---------</td>
</tr>
<tr>
<td>Brugere H</td>
<td>59</td>
</tr>
<tr>
<td>Bundza A</td>
<td>76</td>
</tr>
<tr>
<td>Burger D</td>
<td>72</td>
</tr>
<tr>
<td>Burns KN</td>
<td>32</td>
</tr>
<tr>
<td>Butler D</td>
<td>11</td>
</tr>
<tr>
<td>Butler DA</td>
<td>51</td>
</tr>
<tr>
<td>Cachin M</td>
<td>95</td>
</tr>
<tr>
<td>Carlson GA</td>
<td>35, 39</td>
</tr>
<tr>
<td>Carolan DJP</td>
<td>96</td>
</tr>
<tr>
<td>Carp RI</td>
<td>77, 80</td>
</tr>
<tr>
<td>Carr K</td>
<td>42</td>
</tr>
<tr>
<td>Carrillo BJ</td>
<td>93</td>
</tr>
<tr>
<td>Carter H</td>
<td>11</td>
</tr>
<tr>
<td>Cathala F</td>
<td>76</td>
</tr>
<tr>
<td>Caughey B</td>
<td>37</td>
</tr>
<tr>
<td>Chandler RL</td>
<td>83</td>
</tr>
<tr>
<td>Chansoriya M</td>
<td>4</td>
</tr>
<tr>
<td>Chatelain J</td>
<td>77, 79, 80</td>
</tr>
<tr>
<td>Chazot G</td>
<td>61</td>
</tr>
<tr>
<td>Chen SG</td>
<td>37</td>
</tr>
<tr>
<td>Chen SS</td>
<td>91</td>
</tr>
<tr>
<td>Cherfas J</td>
<td>7</td>
</tr>
<tr>
<td>Chesebro BW</td>
<td>30, 48</td>
</tr>
<tr>
<td>Clark WW</td>
<td>18</td>
</tr>
<tr>
<td>Cohen FE</td>
<td>39</td>
</tr>
<tr>
<td>Collee JG</td>
<td>4, 7</td>
</tr>
<tr>
<td>Collinge J</td>
<td>37, 39, 40, 61, 63</td>
</tr>
<tr>
<td>Come JH</td>
<td>42</td>
</tr>
<tr>
<td>Cooke M</td>
<td>7</td>
</tr>
<tr>
<td>Cranwell</td>
<td>9</td>
</tr>
<tr>
<td>Crawford MA</td>
<td>5</td>
</tr>
<tr>
<td>Cullen RG</td>
<td>97</td>
</tr>
<tr>
<td>Cunningham AA</td>
<td>86</td>
</tr>
<tr>
<td>Cutlip RC</td>
<td>19</td>
</tr>
<tr>
<td>Danner K</td>
<td>4</td>
</tr>
<tr>
<td>Darcel C</td>
<td>27</td>
</tr>
<tr>
<td>Davanipour Z</td>
<td>67</td>
</tr>
<tr>
<td>Davies DC</td>
<td>79</td>
</tr>
<tr>
<td>Davies G</td>
<td>1</td>
</tr>
<tr>
<td>Davies PTG</td>
<td>64</td>
</tr>
<tr>
<td>Davis AJ</td>
<td>59</td>
</tr>
<tr>
<td>Dawson M</td>
<td>23, 24</td>
</tr>
<tr>
<td>Day CEI</td>
<td>11</td>
</tr>
<tr>
<td>Name</td>
<td>Pages</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>-------</td>
</tr>
<tr>
<td>Dealler S</td>
<td>6, 19, 31, 47</td>
</tr>
<tr>
<td>DeArmond SJ</td>
<td>29, 35, 37, 42</td>
</tr>
<tr>
<td>Delasnerie-Laupretre N</td>
<td>61</td>
</tr>
<tr>
<td>Denny GO</td>
<td>21</td>
</tr>
<tr>
<td>Department of Agriculture for Northern Ireland</td>
<td>14</td>
</tr>
<tr>
<td>Detwiler LA</td>
<td>74</td>
</tr>
<tr>
<td>Dickinson AG</td>
<td>78, 81-83</td>
</tr>
<tr>
<td>Dinter Z</td>
<td>76</td>
</tr>
<tr>
<td>Diringer H</td>
<td>47, 61</td>
</tr>
<tr>
<td>Draper GJ</td>
<td>54</td>
</tr>
<tr>
<td>Duchen LW</td>
<td>64</td>
</tr>
<tr>
<td>Earl J</td>
<td>1</td>
</tr>
<tr>
<td>Eddy RG</td>
<td>2, 7</td>
</tr>
<tr>
<td>Esmond TFG</td>
<td>29</td>
</tr>
<tr>
<td>European Commission</td>
<td>4</td>
</tr>
<tr>
<td>Farquhar CF</td>
<td>60</td>
</tr>
<tr>
<td>Fear C</td>
<td>7</td>
</tr>
<tr>
<td>Featherstone T</td>
<td>55</td>
</tr>
<tr>
<td>Fink JK</td>
<td>40</td>
</tr>
<tr>
<td>Fischler F</td>
<td>2</td>
</tr>
<tr>
<td>Fleetwood AJ</td>
<td>90</td>
</tr>
<tr>
<td>Foote WC</td>
<td>78, 79</td>
</tr>
<tr>
<td>Foster JD</td>
<td>20, 73, 74, 78</td>
</tr>
<tr>
<td>Franco DA</td>
<td>92</td>
</tr>
<tr>
<td>Fraser H</td>
<td>21, 25, 26, 50, 80, 81, 85</td>
</tr>
<tr>
<td>Fraser JR</td>
<td>17</td>
</tr>
<tr>
<td>Gabison R</td>
<td>35</td>
</tr>
<tr>
<td>Gajdusek DC</td>
<td>47, 49, 68</td>
</tr>
<tr>
<td>Ghergariu S</td>
<td>59</td>
</tr>
<tr>
<td>Gibbs CJ</td>
<td>24, 32</td>
</tr>
<tr>
<td>Gibson PH</td>
<td>31, 78</td>
</tr>
<tr>
<td>Gill PA</td>
<td>93</td>
</tr>
<tr>
<td>Gilmour JS</td>
<td>10, 52</td>
</tr>
<tr>
<td>Goldfarb LG</td>
<td>27</td>
</tr>
<tr>
<td>Goldmann W</td>
<td>47, 73</td>
</tr>
<tr>
<td>Gomi H</td>
<td>40</td>
</tr>
<tr>
<td>Gourmelon P</td>
<td>67</td>
</tr>
<tr>
<td>Graber HU</td>
<td>55</td>
</tr>
<tr>
<td>Gracey JF</td>
<td>10</td>
</tr>
<tr>
<td>Grant HC</td>
<td>31</td>
</tr>
<tr>
<td>Green E.</td>
<td>1</td>
</tr>
<tr>
<td>Groschup MH</td>
<td>40, 42</td>
</tr>
<tr>
<td>Guarda F</td>
<td>91</td>
</tr>
</tbody>
</table>
Guiroy DC ............................................................. 86, 89  
Hadlow WJ ............................................................. 71, 77, 80  
Hainfellner JA ............................................................. 55  
Haritani M ............................................................... 56  
Harpster DE .............................................................. 73  
Hartsough GR ............................................................. 72  
Hauw JJ .................................................................. 37  
Hogan RN ................................................................. 51  
Hoinville LJ ............................................................. 18-20, 22, 23  
Holmes S .................................................................. 27  
Hope J .................................................................. 40, 51  
Hopkins AR .................................................................. 60  
Hornlimann B .......................................................... 92, 95  
Hourrigan JL ............................................................ 7, 81  
House of Commons Agriculture Committee ....................................... 15  
Hsiao KK ............................................................. 40, 64  
Huang Z ................................................................. 35  
Humphery-Smith I ............................................................. 44, 66  
Hunter GD ............................................................... 29  
Hunter N ........................................................ 29, 40, 50, 75  
Institute for Animal Health .............................................................. 15  
Jack EJ .................................................................. 10  
Jarret JT ................................................................. 42  
Jeffrey M ................................................................. 3, 5, 14, 37, 45  
Jericho KWF ............................................................... 7  
Jibiki I ................................................................. 61  
Johnson CT .............................................................. 10  
Johnstone AC ........................................................... 94, 97  
Julian AF ................................................................. 97  
Kamin M ................................................................. 67  
Kannenberg K ............................................................. 37  
Katz JB ................................................................. 56  
Keith NWJ ............................................................... 12  
Keulen LJM van ........................................................... 56  
Kim JH ................................................................. 65, 66  
Kimberlin RH ........................................................... 4, 5, 15, 49, 77, 78, 80, 81  
King LJ ................................................................. 97  
Kingsbury DT ............................................................. 49  
Kirkwood JK ............................................................. 85-88, 90  
Kitamoto T ................................................................. 45, 65, 66  
Kraaden OR ............................................................... 92  
Kretzschmar HA ........................................................... 29, 70  
Kuramato T ............................................................... 41
Lacey RW ........................................................................... 1, 4, 12
Lantos PL .............................................................................. 30
Laplanche JL ........................................................................... 41
Lasmezas CI ........................................................................... 17, 73
Laszlo L .................................................................................. 45
Latarjet R ................................................................................. 33
Lee JT ....................................................................................... 38
Leggett MM ............................................................................ 90
Lehmann S ............................................................................. 35
Lewis PK ................................................................................... 31
Liberski PP .............................................................................. 7, 28, 29, 45, 46, 48, 49, 51, 62
Little PB .................................................................................... 7
Love J ......................................................................................... 11
MacIntyre D ............................................................................. 1
Madeiros CA ............................................................................. 8
Manuelidis L ............................................................................ 32, 61
Marsh RF .................................................................................. 69-71, 94
Martin WB ................................................................................ 77
Marx JL ...................................................................................... 53, 54
Masters CL .............................................................................. 52, 67, 68
Matthews D .............................................................................. 15
Matthews WB ........................................................................... 8, 68
McCracken RM ......................................................................... 25
McGill IS ................................................................................... 4
McKenzie DI ............................................................................. 95
McKerrell RE ............................................................................ 31
Medori R ..................................................................................... 42
Meldrum KD .............................................................................. 9
Mendez-Martinez O .................................................................. 62
Merz PA ..................................................................................... 53
Middleton DJ ............................................................................ 20
Miller LD .................................................................................... 95
Millot P ....................................................................................... 51, 52
Ministry of Agriculture, Fisheries and Food .................................. 12, 13, 15, 16
Minor ......................................................................................... 8
Mohri S ....................................................................................... 58, 66
Moolgaard CA .......................................................................... 8
Morgan KL ................................................................................ 10, 77
Muhleisen H ............................................................................. 62
Muirhead S ............................................................................... 96
Narang HK ................................................................................. 43, 46, 65
Neborgs HL ............................................................................... 41
Nicholl D ..................................................................................... 62
<table>
<thead>
<tr>
<th>Name</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nussbaum RE</td>
<td>81</td>
</tr>
<tr>
<td>Oberdieck U</td>
<td>57</td>
</tr>
<tr>
<td>Office International des Epizooties</td>
<td>8, 15</td>
</tr>
<tr>
<td>Oken RJ</td>
<td>62</td>
</tr>
<tr>
<td>Onodera T</td>
<td>74</td>
</tr>
<tr>
<td>Ossa JE</td>
<td>38</td>
</tr>
<tr>
<td>Pain S</td>
<td>10</td>
</tr>
<tr>
<td>Palmer MS</td>
<td>43</td>
</tr>
<tr>
<td>Parchi P</td>
<td>38, 62</td>
</tr>
<tr>
<td>Parry HB</td>
<td>80, 83</td>
</tr>
<tr>
<td>Pattison IH</td>
<td>23, 78, 82, 83</td>
</tr>
<tr>
<td>Pearson GR</td>
<td>87-89</td>
</tr>
<tr>
<td>Peet RL</td>
<td>88</td>
</tr>
<tr>
<td>Peiffer J</td>
<td>58</td>
</tr>
<tr>
<td>Perrin GG</td>
<td>75</td>
</tr>
<tr>
<td>Petersen RB</td>
<td>41</td>
</tr>
<tr>
<td>Pocchiari M</td>
<td>57, 66, 77, 79</td>
</tr>
<tr>
<td>Poidinger M</td>
<td>43</td>
</tr>
<tr>
<td>Pollanen MS</td>
<td>43</td>
</tr>
<tr>
<td>Price DL</td>
<td>64</td>
</tr>
<tr>
<td>Priola SA</td>
<td>38</td>
</tr>
<tr>
<td>Prusiner SB</td>
<td>27, 30, 33, 35, 38, 41, 43, 46, 48, 49, 51-53, 63, 79</td>
</tr>
<tr>
<td>Pulford PN</td>
<td>10</td>
</tr>
<tr>
<td>Purdey M</td>
<td>17, 19, 21, 35</td>
</tr>
<tr>
<td>Ridley RM</td>
<td>17, 27, 41, 52, 64</td>
</tr>
<tr>
<td>Roberts GW</td>
<td>8, 47</td>
</tr>
<tr>
<td>Robinson MM</td>
<td>69</td>
</tr>
<tr>
<td>Saeki K</td>
<td>36</td>
</tr>
<tr>
<td>Safar J</td>
<td>36, 48, 49</td>
</tr>
<tr>
<td>Schoon HA</td>
<td>89</td>
</tr>
<tr>
<td>Schreuder BEC</td>
<td>1, 3, 28, 30, 55</td>
</tr>
<tr>
<td>Schudel AA</td>
<td>91, 92</td>
</tr>
<tr>
<td>Scott AC</td>
<td>58, 59</td>
</tr>
<tr>
<td>Scott MR</td>
<td>36</td>
</tr>
<tr>
<td>Scott PR</td>
<td>8, 16, 57</td>
</tr>
<tr>
<td>Serban D</td>
<td>59</td>
</tr>
<tr>
<td>Shaw IC</td>
<td>12</td>
</tr>
<tr>
<td>Short N</td>
<td>56</td>
</tr>
<tr>
<td>Sigurdarson S</td>
<td>76</td>
</tr>
<tr>
<td>Simmons MM</td>
<td>11</td>
</tr>
<tr>
<td>Singhal BS</td>
<td>68</td>
</tr>
<tr>
<td>Smith C</td>
<td>38</td>
</tr>
<tr>
<td>Smith PEM</td>
<td>62</td>
</tr>
<tr>
<td>Name</td>
<td>Pages</td>
</tr>
<tr>
<td>-----------------------</td>
<td>----------------------------------------------------------------------</td>
</tr>
<tr>
<td>Adams DH</td>
<td>8</td>
</tr>
<tr>
<td>Aguzzi A</td>
<td>19</td>
</tr>
<tr>
<td>Aldhous P</td>
<td>16, 25, 79</td>
</tr>
<tr>
<td>Aldridge BM</td>
<td>11</td>
</tr>
<tr>
<td>Alper T</td>
<td>44</td>
</tr>
<tr>
<td>Andrews</td>
<td>11</td>
</tr>
<tr>
<td>Arya SC</td>
<td>71, 85</td>
</tr>
<tr>
<td>Asher DM</td>
<td>86</td>
</tr>
<tr>
<td>Ashworth SW</td>
<td>13</td>
</tr>
<tr>
<td>Austin AR</td>
<td>30</td>
</tr>
<tr>
<td>Baker HF</td>
<td>22, 63</td>
</tr>
<tr>
<td>Barlow RM</td>
<td>24, 25</td>
</tr>
<tr>
<td>Baron H</td>
<td>50</td>
</tr>
<tr>
<td>Bartz JC</td>
<td>67</td>
</tr>
<tr>
<td>Basset H</td>
<td>86</td>
</tr>
<tr>
<td>Beardsley T</td>
<td>63</td>
</tr>
<tr>
<td>Beekees M</td>
<td>54</td>
</tr>
<tr>
<td>Bendheim PE</td>
<td>71</td>
</tr>
<tr>
<td>Bennett AD</td>
<td>44</td>
</tr>
<tr>
<td>Berg LJ</td>
<td>39</td>
</tr>
<tr>
<td>Bessen RA</td>
<td>36, 68</td>
</tr>
<tr>
<td>Biernat W</td>
<td>36</td>
</tr>
<tr>
<td>Blakemore WF</td>
<td>33</td>
</tr>
<tr>
<td>Bobowick AR</td>
<td>66</td>
</tr>
<tr>
<td>Bockman JM</td>
<td>50</td>
</tr>
<tr>
<td>Bode L</td>
<td>51</td>
</tr>
<tr>
<td>Bolis CL</td>
<td>8</td>
</tr>
<tr>
<td>Boothby CB</td>
<td>11</td>
</tr>
<tr>
<td>Borras T</td>
<td>74</td>
</tr>
<tr>
<td>Bradley R</td>
<td>5, 7, 15, 29, 31, 32</td>
</tr>
<tr>
<td>Brandel JP</td>
<td>62</td>
</tr>
<tr>
<td>Brandner S</td>
<td>35</td>
</tr>
<tr>
<td>Bratberg B</td>
<td>75</td>
</tr>
<tr>
<td>Bridges V</td>
<td>68</td>
</tr>
<tr>
<td>Brochier B</td>
<td>84</td>
</tr>
<tr>
<td>Brown DR</td>
<td>82</td>
</tr>
<tr>
<td>Brown P</td>
<td>31, 33, 47, 62, 63, 65, 66, 71, 82</td>
</tr>
<tr>
<td>Bruce M</td>
<td>20</td>
</tr>
<tr>
<td>Brugere H</td>
<td>57</td>
</tr>
<tr>
<td>Bundza A</td>
<td>72</td>
</tr>
<tr>
<td>Burger D</td>
<td>69</td>
</tr>
<tr>
<td>Burns KN</td>
<td>33</td>
</tr>
<tr>
<td>Butler D</td>
<td>13</td>
</tr>
<tr>
<td>Name</td>
<td>Pages</td>
</tr>
<tr>
<td>----------------------</td>
<td>-------</td>
</tr>
<tr>
<td>Butler DA</td>
<td>50</td>
</tr>
<tr>
<td>Cachin M</td>
<td>85</td>
</tr>
<tr>
<td>Carlson GA</td>
<td>35, 39</td>
</tr>
<tr>
<td>Carolan DJP</td>
<td>86</td>
</tr>
<tr>
<td>Carr K</td>
<td>42</td>
</tr>
<tr>
<td>Carrillo BJ</td>
<td>83</td>
</tr>
<tr>
<td>Carter H</td>
<td>13</td>
</tr>
<tr>
<td>Cathala F</td>
<td>72</td>
</tr>
<tr>
<td>Chansoriya M</td>
<td>6</td>
</tr>
<tr>
<td>Chatelain J</td>
<td>73</td>
</tr>
<tr>
<td>Chazot G</td>
<td>60</td>
</tr>
<tr>
<td>Chen SG</td>
<td>37</td>
</tr>
<tr>
<td>Cherfas J</td>
<td>8</td>
</tr>
<tr>
<td>Chesebro BW</td>
<td>31, 48</td>
</tr>
<tr>
<td>Clark WW</td>
<td>19</td>
</tr>
<tr>
<td>Cohen FE</td>
<td>39</td>
</tr>
<tr>
<td>Collee JG</td>
<td>5, 9</td>
</tr>
<tr>
<td>Collinge J</td>
<td>37, 39, 40, 62</td>
</tr>
<tr>
<td>Come JH</td>
<td>42</td>
</tr>
<tr>
<td>Cooke</td>
<td>9</td>
</tr>
<tr>
<td>Cranwell</td>
<td>11</td>
</tr>
<tr>
<td>Crawford MA</td>
<td>7</td>
</tr>
<tr>
<td>Cullen</td>
<td>86</td>
</tr>
<tr>
<td>Cunningham AA</td>
<td>76</td>
</tr>
<tr>
<td>Cutlip RC</td>
<td>20</td>
</tr>
<tr>
<td>Danner K</td>
<td>5</td>
</tr>
<tr>
<td>Darcel C</td>
<td>28</td>
</tr>
<tr>
<td>Davanipour Z</td>
<td>65</td>
</tr>
<tr>
<td>Davies G</td>
<td>3</td>
</tr>
<tr>
<td>Davies PTG</td>
<td>62</td>
</tr>
<tr>
<td>Davis AJ</td>
<td>58</td>
</tr>
<tr>
<td>Dawson M</td>
<td>24-26</td>
</tr>
<tr>
<td>Day CEI</td>
<td>13</td>
</tr>
<tr>
<td>Dealler S</td>
<td>7, 21, 32, 47</td>
</tr>
<tr>
<td>DeArmond SJ</td>
<td>30, 35, 37, 42</td>
</tr>
<tr>
<td>Delasnerie-Laupretre N</td>
<td>60</td>
</tr>
<tr>
<td>Denny GO</td>
<td>23</td>
</tr>
<tr>
<td>Department of Agriculture for Northern Ireland</td>
<td>16</td>
</tr>
<tr>
<td>Detwiler LA</td>
<td>71</td>
</tr>
<tr>
<td>Dickinson AG</td>
<td>73</td>
</tr>
<tr>
<td>Dinter Z</td>
<td>72</td>
</tr>
<tr>
<td>Diringer H</td>
<td>47</td>
</tr>
<tr>
<td>Duchen LW</td>
<td>62</td>
</tr>
<tr>
<td>Name</td>
<td>Pages</td>
</tr>
<tr>
<td>------------------</td>
<td>-------</td>
</tr>
<tr>
<td>Earl J.</td>
<td>3</td>
</tr>
<tr>
<td>Eddy RG</td>
<td>4, 9</td>
</tr>
<tr>
<td>Esmond TFG</td>
<td>30</td>
</tr>
<tr>
<td>European Commission</td>
<td>6</td>
</tr>
<tr>
<td>Farquhar CF</td>
<td>58</td>
</tr>
<tr>
<td>Fear</td>
<td>9</td>
</tr>
<tr>
<td>Featherstone T</td>
<td>54</td>
</tr>
<tr>
<td>Fink JK</td>
<td>40</td>
</tr>
<tr>
<td>Fischler F</td>
<td>4</td>
</tr>
<tr>
<td>Fleetwood AJ</td>
<td>79</td>
</tr>
<tr>
<td>Foote</td>
<td>73</td>
</tr>
<tr>
<td>Foster JD</td>
<td>22</td>
</tr>
<tr>
<td>Franco DA</td>
<td>81</td>
</tr>
<tr>
<td>Fraser H</td>
<td>23, 27, 75</td>
</tr>
<tr>
<td>Fraser JR</td>
<td>19</td>
</tr>
<tr>
<td>Gabison R</td>
<td>35</td>
</tr>
<tr>
<td>Gajdusek DC</td>
<td>47, 49, 66</td>
</tr>
<tr>
<td>Ghergariu S</td>
<td>58</td>
</tr>
<tr>
<td>Gibbs CJ</td>
<td>26, 33</td>
</tr>
<tr>
<td>Gibson PH</td>
<td>32</td>
</tr>
<tr>
<td>Gill PA</td>
<td>83</td>
</tr>
<tr>
<td>Gilmour JS</td>
<td>11</td>
</tr>
<tr>
<td>Goldfarb LG</td>
<td>28</td>
</tr>
<tr>
<td>Goldman W</td>
<td>47</td>
</tr>
<tr>
<td>Goldmann W</td>
<td>47, 70</td>
</tr>
<tr>
<td>Gomi H</td>
<td>40</td>
</tr>
<tr>
<td>Gourmelon P</td>
<td>65</td>
</tr>
<tr>
<td>Graber HU</td>
<td>54</td>
</tr>
<tr>
<td>Gracey JF</td>
<td>11</td>
</tr>
<tr>
<td>Grant</td>
<td>32</td>
</tr>
<tr>
<td>Green E.</td>
<td>3</td>
</tr>
<tr>
<td>Groschup MH</td>
<td>40, 42</td>
</tr>
<tr>
<td>Guarda F</td>
<td>81</td>
</tr>
<tr>
<td>Guiroy DC</td>
<td>76, 78</td>
</tr>
<tr>
<td>Hadlow WJ</td>
<td>69, 73</td>
</tr>
<tr>
<td>Hainfellner JA</td>
<td>54</td>
</tr>
<tr>
<td>Haritani M</td>
<td>55</td>
</tr>
<tr>
<td>Harpster DE</td>
<td>70</td>
</tr>
<tr>
<td>Hauw JJ</td>
<td>37</td>
</tr>
<tr>
<td>Hogan RN</td>
<td>51</td>
</tr>
<tr>
<td>Hoinville LJ</td>
<td>20, 21</td>
</tr>
<tr>
<td>Holmes S</td>
<td>28</td>
</tr>
<tr>
<td>Hope J</td>
<td>40, 50</td>
</tr>
<tr>
<td>Name</td>
<td>Pages</td>
</tr>
<tr>
<td>---------------------------</td>
<td>----------------------------</td>
</tr>
<tr>
<td>Hopkins AR</td>
<td>59</td>
</tr>
<tr>
<td>Hornlimann B</td>
<td>82, 85</td>
</tr>
<tr>
<td>Hourrigan</td>
<td>9</td>
</tr>
<tr>
<td>House of Commons Agriculture Committee</td>
<td>17</td>
</tr>
<tr>
<td>Hsiao KK</td>
<td>40, 63</td>
</tr>
<tr>
<td>Huang Z</td>
<td>35</td>
</tr>
<tr>
<td>Humphery-Smith I</td>
<td>44, 64</td>
</tr>
<tr>
<td>Hunter GD</td>
<td>30</td>
</tr>
<tr>
<td>Hunter N</td>
<td>30, 40, 72</td>
</tr>
<tr>
<td>Institute for Animal Health</td>
<td>14, 17</td>
</tr>
<tr>
<td>Jack EJ</td>
<td>11</td>
</tr>
<tr>
<td>Jarret JT</td>
<td>42</td>
</tr>
<tr>
<td>Jeffrey M</td>
<td>5, 6, 16, 37, 45</td>
</tr>
<tr>
<td>Jericho KWF</td>
<td>9</td>
</tr>
<tr>
<td>Jibiki I</td>
<td>60</td>
</tr>
<tr>
<td>Johnson CT</td>
<td>12</td>
</tr>
<tr>
<td>Johnstone AC</td>
<td>83, 86</td>
</tr>
<tr>
<td>Julian AF</td>
<td>87</td>
</tr>
<tr>
<td>Kamin M</td>
<td>65</td>
</tr>
<tr>
<td>Kannenberg K</td>
<td>37</td>
</tr>
<tr>
<td>Katz JB</td>
<td>55</td>
</tr>
<tr>
<td>Keith NWJ</td>
<td>14</td>
</tr>
<tr>
<td>Keulen LJM van</td>
<td>55</td>
</tr>
<tr>
<td>Kim JH</td>
<td>64</td>
</tr>
<tr>
<td>Kimberlin RH</td>
<td>6, 7, 17, 49</td>
</tr>
<tr>
<td>King LJ</td>
<td>86</td>
</tr>
<tr>
<td>Kingsbury DT</td>
<td>49</td>
</tr>
<tr>
<td>Kirkwood JK</td>
<td>75-77, 79</td>
</tr>
<tr>
<td>Kitamoto T</td>
<td>45, 64</td>
</tr>
<tr>
<td>Kraaden OR</td>
<td>82</td>
</tr>
<tr>
<td>Kretzschmar HA</td>
<td>30, 68</td>
</tr>
<tr>
<td>Kuramoto T</td>
<td>41</td>
</tr>
<tr>
<td>Lacey RW</td>
<td>3, 6, 14</td>
</tr>
<tr>
<td>Lantos PL</td>
<td>31</td>
</tr>
<tr>
<td>Laplanche JL</td>
<td>41</td>
</tr>
<tr>
<td>Lasmezas CI</td>
<td>19, 70</td>
</tr>
<tr>
<td>Laszlo L</td>
<td>45</td>
</tr>
<tr>
<td>Latarjet R</td>
<td>34</td>
</tr>
<tr>
<td>Lee JT</td>
<td>38</td>
</tr>
<tr>
<td>Leggett MM</td>
<td>79</td>
</tr>
<tr>
<td>Lehman S</td>
<td>35</td>
</tr>
<tr>
<td>Lehmann S</td>
<td>35</td>
</tr>
<tr>
<td>Lewis PK</td>
<td>32</td>
</tr>
<tr>
<td>Name</td>
<td>Pages</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>-------</td>
</tr>
<tr>
<td>Perrin GG</td>
<td>72</td>
</tr>
<tr>
<td>Petersen RB</td>
<td>41</td>
</tr>
<tr>
<td>Pocchiari M</td>
<td>56, 64, 73</td>
</tr>
<tr>
<td>Poidinger M</td>
<td>43</td>
</tr>
<tr>
<td>Pollanen MS</td>
<td>43</td>
</tr>
<tr>
<td>Price DL</td>
<td>63</td>
</tr>
<tr>
<td>Priola SA</td>
<td>38</td>
</tr>
<tr>
<td>Prusiner SB</td>
<td>28, 31, 34, 35, 38, 41, 43, 46, 48, 49, 51, 52, 62, 74</td>
</tr>
<tr>
<td>Pulford PN</td>
<td>12</td>
</tr>
<tr>
<td>Purdey M</td>
<td>19, 21, 22, 35</td>
</tr>
<tr>
<td>Ridley RM</td>
<td>28, 42, 52, 63</td>
</tr>
<tr>
<td>Roberts GW</td>
<td>10, 47</td>
</tr>
<tr>
<td>Robinson MM</td>
<td>67</td>
</tr>
<tr>
<td>Saeki K</td>
<td>36</td>
</tr>
<tr>
<td>Safar J</td>
<td>36, 48, 49</td>
</tr>
<tr>
<td>Schoon HA</td>
<td>78</td>
</tr>
<tr>
<td>Schreuder BEC</td>
<td>5, 29, 31, 54</td>
</tr>
<tr>
<td>Schudel AA</td>
<td>81, 82</td>
</tr>
<tr>
<td>Scott AC</td>
<td>57, 58</td>
</tr>
<tr>
<td>Scott MR</td>
<td>36</td>
</tr>
<tr>
<td>Scott PR</td>
<td>10, 18, 56</td>
</tr>
<tr>
<td>Serban D</td>
<td>58</td>
</tr>
<tr>
<td>Shaw IC</td>
<td>14</td>
</tr>
<tr>
<td>Short N</td>
<td>55</td>
</tr>
<tr>
<td>Simmons MM</td>
<td>13</td>
</tr>
<tr>
<td>Smith C</td>
<td>38</td>
</tr>
<tr>
<td>Smith PEM</td>
<td>61</td>
</tr>
<tr>
<td>Snow AD</td>
<td>49</td>
</tr>
<tr>
<td>Somerville RA</td>
<td>36</td>
</tr>
<tr>
<td>Sommer SS</td>
<td>36</td>
</tr>
<tr>
<td>Southwood R</td>
<td>10</td>
</tr>
<tr>
<td>Spongiform Encephalopathy Advisory Committee</td>
<td>28</td>
</tr>
<tr>
<td>Stack MJ</td>
<td>72</td>
</tr>
<tr>
<td>Stekel DJ</td>
<td>13</td>
</tr>
<tr>
<td>Stevenson RM</td>
<td>13</td>
</tr>
<tr>
<td>Strain GM</td>
<td>58</td>
</tr>
<tr>
<td>Sutherland K</td>
<td>36</td>
</tr>
<tr>
<td>Tateishi J</td>
<td>28, 56, 61</td>
</tr>
<tr>
<td>Taylor DM</td>
<td>4, 11, 19, 20, 29, 32, 33</td>
</tr>
<tr>
<td>Taylor KC</td>
<td>16, 27, 55</td>
</tr>
<tr>
<td>Telling GC</td>
<td>38, 62</td>
</tr>
<tr>
<td>Terzano MG</td>
<td>61</td>
</tr>
<tr>
<td>Truyen U</td>
<td>29</td>
</tr>
</tbody>
</table>
USDA-APHIS-VS .................................................... 10, 84, 85
USDA-APHIS ..............................................................3
van Gool WA .............................................................6 1
Walker KD ...............................................................8 6
Ward WR ................................................................. 13
Watanabe R ...............................................................4 4
Weaver AD .............................................................7, 15
Weiss S ................................................................. 36
Weissmann C ........................................................... 39, 44
Wells GAH .............................................................. 4, 11, 12, 21, 56, 57, 59
Westaway D ........................................................ 32, 39, 42
Whittington MA ...........................................................3 9
Wickham EA ..............................................................1 9
Wijeratne WVS ............................................................2 7
Wilesmith JW ......................................................... 5, 7, 8, 18, 21-23, 27
Will RG ............................................................ 50, 60, 65
Williams ES .............................................................. 77
Williams RL ............................................................ 13
Willoughby K .............................................................7 8
Wilson S ................................................................. 11
Winter MH ............................................................. 18
Wisniewski HM ....................................................... 42, 70
Wood JLN ................................................................. 71
Woodgate SL ........................................................... 5, 15
Worthington JM ...........................................................6 1
Wyatt JM .............................................................. 77, 79, 80
Yoshimoto J .............................................................. 47