Bibliography for

Transmissible Spongiform Encephalopathies

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Table of Contents

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.

BSE - General

1996

Davies G, Jones S. 1996. Origin of BSE. Veterinary Record 138(1):23.

Earl J. 1996. Origin of BSE. Veterinary Record 138(12):288.

Five candles on NHS-reforms' birthday cake: BSE fears stir the Swiss. 1996. Lancet 347(9007):1035.

Green E. 1996. Anatomy of a panic. The Spectator 276(8754):8.

Scientists, consumer spokespersons and sensationalists in tabloids and broadsheets caused the BSE scare.

Lacey RW. 1996. Creutzfeldt-Jakob disease and bovine spongiform encephalopathy - Bovine spongiform encephalopathy is being maintained by vertical and horizontal transmission. British Medical Journal 312(7024):180-181.

MacIntyre D. 1996. Rare breeds and the BSE situation. Veterinary Record 138(14):338.

Schreuder BEC, Straub OC. 1996. BSE: a European Problem. Veterinary Record 138(23):575.

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.

Small steps forward on BSE. 1996. Veterinary Record 138(16):373.

Transgenic mice give clue to BSE risks. 1996. Veterinary Record 138(1):2.

USDA-APHIS. 1996. USDA-APHIS questions and answers regarding BSE. The Shepherd 41(5):38.

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

1995

BMJ considers risks to humans from BSE. 1995. Veterinary Record 137(24):602.

Eddy RG. 1995. Origin of BSE. Veterinary Record 137(25):648.

Fischler F. 1995. Memorandum on bovine spongiform encephalopathy (BSE). European Food Law Review 6(2):234.240.

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.

Taylor DM. 1995. Origin of BSE. Veterinary Record 137(26):674.

Taylor DM, Woodgate SL, Atkinson MJ. 1995. Inactivation of the bovine spongiform encephalopathy agent by rendering procedures. Veterinary Record 137(24):605-610.

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.

Wells GAH, Sayers AR, Wilesmith JW. 1995. Clinical and epidemiological correlates of the neurohistology of cases of histologically unconfirmed, clinically suspect BSE. Veterinary Record 136(9):211-216.

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.

Wells GAH, Wilesmith JW. 1995. The neuropathology and epidemiology of bovine spongiform encephalopathy. Brain Pathology 5(1):91-103.

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

Bradley R (Editor). 1994. Special issue: bovine spongiform encephalopathy. Livestock Production Science 38(1):1-59.

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

Jeffrey M, Halliday WG. 1994. Numbers of neurons in vacuolated and non-vacuolated neuroanatomical nuclei in BSE-affected brains. Journal of Comparative Pathology 110(3):287-293.

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.

Schreuder BEC. 1994. Animal spongiform encephalopathies - an update. Part 2. Bovine spongiform encephalopathy. Veterinary Quarterly 16(3):182-192.

Wilesmith JW. 1994. Bovine spongiform encephalopathy and related diseases: an epidemiological overview. New Zealand Veterinary Journal 42(1):1-8.

Woodgate SL. 1994. Rendering systems and BSE agent deactivation. Livestock Production Science 38(1):47.

1993

Collee JG. 1993. BSE: stocktaking 1993. Lancet 342(8874):790.

Danner K. 1993. BSE - a risk for man through pharmaceutical products? Position and politics of the German pharmaceutical industry. Developments in Biological Standardization 80:199.

Kimberlin RH. 1993. Bovine spongiform encephalopathy. FAO Animal Production and Health Paper. Rome, Italy. Food and Agriculture Organization, No. 109, pp. 1-68.

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

Lacey RW. 1993. BSE: the gathering crisis. British Food Journal 95(4):17-21.

McGill IS, Wells GAH. 1993. Neuropathological findings in cattle with clinically suspect but histologically unconfirmed BSE. Journal of Comparative Pathology 108(3):241-260.

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

Chansoriya M, Vegad JL. 1992. Bovine spongiform encephalopathy: a review. Journal of Applied Animal Research 1(1):57-63.

European Commission, Committee for Proprietary Medicinal Products. 1992. EEC Regulatory Document. Note for guidance. Guidelines for minimizing the risk of transmitting agents causing

BSE via medicinal products. Biologicals 20(2):155-158.

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.

Jeffrey M, Halliday WG, Goodsir CM. 1992. A morphometric and immunohistochemical study of the vestibular nuclear complex in BSE. Acta Neuropathologica 84(6):651-657.

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.

Kimberlin RH. 1992. Bovine spongiform encephalopathy. Revue Scientifique et Technique - Office International des Epizooties 11(2):347-390.

Weaver AD. 1992. BSE: its clinical features and epidemiology in the UK and significance for the US. Compendium on Continuing Education for the Practicing Veterinarian 14(12):1647-1650.

Wilesmith JW, Hoinville LJ, Ryan JBM, et al. 1992. Bovine spongiform encephalopathy: aspects of the clinical picture and analyses of possible changes 1986-1990. Veterinary Record 130(1):197-201.

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

Bradley R. 1991. Bovine spongiform encephalopathy (BSE): the current situation and research. European Journal of Epidemiology 7(5);532-544.

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

Crawford MA, Budowski P, Drury P, et al. 1991. The nutritional contribution to bovine spongiform encephalopathy. Nutrition and Health 7(2):61-68.

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.

Dealler S, Lacey R. 1991. Beef and bovine spongiform encephalopathy: the risk persists. Nutrition and Health 7(3):117-133.

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.

Wilesmith JW. 1991. Origins of BSE. Veterinary Record 128(13):310.

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.

Wilesmith JW, Wells GAH. 1991. Bovine spongiform encephalopathy. Current Topics in Microbiology and Immunology 172:21-38.

1990

Adams DH. 1990. Bovine spongiform encephalopathy -- a new disease transmissible to humans? Medical Hypotheses 32(4):313-317.

Bolis CL, Gibbs CJ Jr. 1990. Proceedings of an international roundtable on bovine spongiform encephalopathy. Summary report and recommendations. Journal of the American Veterinary Medical Association 196(10):1673-1690.

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.

Bovine spongiform encephalopathy. 1990. Veterinary Record 126(7):170-171.

Policy paper.

Cherfas J. 1990. Mad Cow Disease: Uncertainty rules. Science 249:1492-1493.

Cherfas J. 1990. Virus-like agent blamed for mad cow disease. Science 247(4942):523.

Collee JG. 1990. Bovine spongiform encephalopathy. Lancet (British Edition) 336(8726):1300-1303.

Cooke M. 1990. Guidance for veterinary surgeons handling BSE cases. Veterinary Record 126(9):223.

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

Fear C. 1990. Bovine spongiform encephalopathy. British Medical Journal 300(6727):817.

Hourrigan JL. 1990. Experimentally induced bovine spongiform encephalopathy in cattle in Mission, Tex, and the control of scrapie. Journal of the American Veterinary Medical Association 196(10):1678.

Jericho KWF. 1990. More on the control of bovine spongiform encephalopathy. [Correspondence] Canadian Veterinary Journal 31(4):252.

Liberski PP. 1990. Ultrastructural neuropathologic features of bovine spongiform encephalopathy. Journal of the American Veterinary Medical Association 196(10):1682.

Little PB, Thorsen J. 1990. More on the control of bovine spongiform encephalopathy. [Correspondence] Canadian Veterinary Journal 31(4):252-253.

Matthews WB. 1990. Bovine spongiform encephalopathy. The safety of beef has not yet been tested and may not be testable. [Editorial]. British Medical Journal (Clinical Research Edition) 300(6722):412-413.

Minor. 1990. Bovine spongiform encephalopathy and biological products for human use. Report of an informal meeting held at NIBSC on 16 May 1988. Biologicals 18(1):77-80.

Moolgaard CA, Golbeck AL. 1990. Mad cows and Englishmen: Bovine spongiform encephalopathy. Neuroepidemiology 9(6):285-286.

Office International des Epizooties. 1990. Meeting of the FMD and other Epizootics Commission, Paris, December 1990.

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

Roberts GW. 1990. Bovine spongiform encephalopathy. British Medical Journal 300(6729):943-944.

Scott PR, Aldridge BM, Clarke M, et al. 1990. Cerebrospinal fluid studies in normal cows and cases of bovine spongiform encephalopathy. British Veterinary Journal 146(1):88-90.

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.

USDA:APHIS:VS, Emergency Programs. 1990. Bovine spongiform encephalopathy. USDA:APHIS:VS Emergency Programs Alert, Riverdale, MD.

1989

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

Meldrum KD. 1989. BSE safety precautions. [Correspondence] Veterinary Record 125(3):73-74.

Southwood Sir R (Chairman). 1989. Report of the Working Party on Bovine Spongiform Encephalopathy. Stanmore, Middlesex, UK, Department of Health.

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.

Taylor DM. 1989. Bovine spongiform encephalopathy and human health. Veterinary Record 125(16):413-415.

Wells GAH, Wilesmith JW. 1989. The distribution of neuronal vacuolation in bovine spongiform encephalopathy (BSE) is constant. [Abstract] Neuropathology and Applied Neurobiology 15(6):519.

Wilson S. 1989. BSE: Some unanswered questions. Veterinary Record 125:279-280.

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

1988

Aldridge BM, Scott PR, Holmes LA, et al. 1988. Elevated plasma glucose concentration in a case of bovine spongiform encephalopathy. [Correspondence] Veterinary Record 122(3):71-72.

Andrews. 1988. Bovine spongiform encephalopathy. Veterinary Record 122(23):566-567.

Boothby CB. 1988. Bovine spongiform encephalopathy: Possible toxicity link? [Correspondence]. Veterinary Record 122(4):95.

Cranwell MP. 1988. Bovine spongiform encephalopathy. Veterinary Record 122(8):190.

Gilmour JS, Buxton D, Macleod NSM, et al. 1988. Bovine spongiform encephalopathy. [Correspondence] Veterinary Record 122(6):142-143.

Gracey JF. 1988. Bovine spongiform encephalopathy. [Correspondence] Veterinary Record 123(6):163.

Comments about meat inspection.

Jack EJ. 1988. Bovine spongiform encephalopathy. [Correspondence]. Veterinary Record 122(6):142.

Discusses hexachlorophene toxicity.

Johnson CT, Whitaker CJ. 1988. Bovine spongiform encephalopathy. [Correspondence] Veterinary Record 122(6):142.

Morgan KL. 1988. Bovine spongiform encephalopathy: time to take scrapie seriously. Veterinary Record 122:445-446.

Pulford PN. 1988. Bovine spongiform encephalopathy. [Correspondence] Veterinary Record 122(8):190.

Wells GAH, Wilesmith JW. 1988. Bovine spongiform encephalopathy. [Correspondence]. Veterinary Record 122(6):142.

Discusses hexachlorophene toxicity.

1987

Pain S. 1987. Brain disease drives cows wild. New Scientist 116:30.

Wells GAH, Scott AC, Johnson CT, et al. 1987. A novel progressive spongiform encephalopathy in cattle. Veterinary Record 121(18):419-420.

BSE - United Kingdom

1996

Butler D. 1996. Did UK ‘dump’ contaminated feed after the ban? Nature 381:544-545.

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.

Carter H. 1996. LVIs and BSE control measures. Veterinary Record 138(19):479.

Day CEI, Hale G, Kellagher AL. 1996. The BSE crisis. Veterinary Record 138(16):399.

Love J, de C. Giles MB, Kellagher AL. 1996. The BSE crisis. Veterinary Record 138(17):422.

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.

Stekel DJ, Nowak MA, Southwood TRE. 1996. Prediction of future BSE spread. Nature 381:??

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.

Ward WR. 1996. The BSE crisis. Veterinary Record 138(19):479.

Williams RL, Clarke RA. 1996. The BSE crisis. Veterinary Record 138(18):451.

1995

Ashworth SW, Mainland DD. 1995. The economic impact of BSE on the UK beef industry. Outlook on Agriculture 24(3):151.

Control of BSE: MAFF tightens up on feed production. 1995. Veterinary Record 137(5):107.

Keith NWJ. 1995. BSE compensation. Veterinary Record 137(18):472.

MAFF reports a decline in the incidence of BSE. 1995. Veterinary Record 136(2):26.

Shaw IC. 1995. BSE and farmworkers. Lancet 346(8986):1365.

UK, Institute of Animal Health.1995. Report 94. BBSRC Institute for Animal Health, Newbury, UK.

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

Lacey RW.1994. Mad cow disease: the history of BSE in Britain. Cypsela Publications, St. Helier, UK.

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.

UK, Ministry of Agriculture, Fisheries and Food. 1994. Animal Health 1993. The Report of the Chief Veterinary Officer. HMSO Publications Centre, London, UK.

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.

Weaver, AD. 1994. Bovine spongiform encephalopathy: farmers' attitude to the UK control programme. Proceedings 18th World Buiatrics Congress: 26th Congress of the Italian Association of Buiatrics, Bologna, Italy, August 29 - September 2, 1994 Vol. 1:789-791.

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.

Woodgate SL. 1994. Rendered products: safe products. Proceedings of the International Animal Nutrition Symposium: updating the use of animal by-products in animal feeds. Utrecht, Netherlands, pp 3-9.

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

1993

Bradley R, Wilesmith JW, Matthews D, et al. 1993. Bovine spongiform encephalopathy in the United Kingdom: Memorandum from a WHO meeting. Bulletin of the World Health Organization 71(6):691-694.

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.

UK, Ministry of Agriculture, Fisheries and Food. 1993. Animal health 1992. Report of the Chief Veterinary Officer. HMSO Publications Centre, London, UK.

BSE was the chief concern during 1992, with 29,802 confirmed cases. Progress in research on the disease is outlined.

1992

Jeffrey M. 1992. A neuropathological survey of brains submitted under the bovine spongiform encephalopathy orders in Scotland. Veterinary Record 131(15):332-337.

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.

Jeffrey M, Wilesmith JW. 1992. Idiopathic brainstem neuronal chromatolysis and hippocampal sclerosis: a novel encephalopathy in clinically suspect cases of BSE. Veterinary Record 131(16):359-362.

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

Taylor KC. 1991. The control of bovine spongiform encephalopathy in Great Britain. Veterinary Record 129(24):522-526.

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.

UK, Department of Agriculture for Northern Ireland. Annual report 1990/91. Belfast, UK.

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

1990

Aldhous P. 1990. BSE compensation. Nature 344(6261):5.

Jeffrey M. 1990. Neurohistopathological observations on bovine spongiform encephalopathy submissions in Scotland. State Veterinary Journal 44(125):151-160.

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

Kimberlin RH. 1990. Detection of bovine spongiform encephalopathy in the United Kingdom. Journal of the American Veterinary Medical Association 196(10):1675-1676.

Matthews D. 1990. Bovine spongiform encephalopathy (BSE) - The story so far. The State Veterinary Journal 44(124):3-18.

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.

Office International des Epizooties. 1990. Report of the meeting on BSE. Paris, 23 July 1990. Paris, France. Office International des Epizooties.

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

UK, House of Commons Agriculture Committee. 1990. Fifth report. Bovine spongiform encephalopathy. Report and Proceedings of the Committee, together with minutes of Evidence and Appendices. London, UK, HMSO.

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.

UK, Institute for Animal Health. 1990. Annual Report 1989. Compton, UK, AFRC Institute for Animal Health.

This report gives a brief account of the work of the Institute during 1989, including work on BSE.

UK, Ministry of Agriculture, Fisheries and Food. 1990. Animal health 1989. Report of the Chief Veterinary Officer. London, UK, HMSO.

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.

Wilesmith JW. 1990. Epidemiology and current status of bovine spongiform encephalopathy in the United Kingdom. Journal of the American Veterinary Medical Association 196(10):1675-1676.

1989

Ruminant offal source of BSE in British cattle. 1989. Agriculture international 41(12):417.

Scott PR. 1989. Bovine spongiform encephalopathy in a cow in the United Kingdom. Journal of the American Veterinary Medical Association 195(12):1745-1747.

Winter MH, Aldridge BM, Scott PR, et al. 1989. Occurrence of 14 cases of bovine spongiform encephalopathy in a closed dairy herd. British Veterinary Journal 145(2):191-194.

1988

Scott PR, Aldridge BM, Holmes LA, et al. 1988. Bovine spongiform encephalopathy in an adult British Friesian cow. Veterinary Record 123(16):373-374.

UK, Ministry of Agriculture, Fisheries and Food. 1988. Animal health 1987. Report of the Chief Veterinary Officer. London, UK, HMSO.

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.

BSE - Epidemiology

1996

Aguzzi A. 1996. Between cows and monkeys. Nature 381:734-735.

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).

Fraser JR, Foster JD, Fraser H. 1996. Creutzfeldt-Jakob disease and bovine spongiform encephalopathy - Scrapie can be transmitted to mice by instillation of inoculum into the conjunctiva. British Medical Journal 312(7024):181.

Lasmezas CI, Deslys JP, Demaimay R, et al. 1996. BSE transmission to macaques. Nature 381:743-744.

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.

Purdey M. 1996. The UK epidemic of BSE: Slow virus or chronic pesticide-initiated modification of the prion protein? Part 2: An epidemiological perspective. Medical hypotheses 46(5):445.

Ridley RM, Baker HF, Windle CP. 1996. Failure to transmit bovine spongiform encephalopathy to marmosets with ruminant-derived meal. Lancet 348:56.

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.

Taylor DM. 1996. Absence of detectable infectivity in trachea of BSE-affected cattle. Veterinary Record 138(7):160.

Wickham EA. 1996. Potential transmission of BSE via medicinal products. British Medical Journal 312(7037):988.

1995

Clark WW, Hourrigan JL, Hadlow WJ. 1995. Encephalopathy in cattle experimentally infected with the scrapie agent. American Journal of Veterinary Research 56(5):606-612.

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.

Hoinville LJ, Wilesmith JW, Richards MS. 1995. An investigation of risk factors for cases of BSE born after the introduction of the 'feed ban'. Veterinary Record 136(13):312-318.

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.

Taylor DM, Ferguson CE, Bostock CJ, et al. 1995. Absence of disease in mice receiving milk from cows with BSE. Veterinary Record 136(23):592.

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.

1994

Bruce M, Chree A, McConnell I, et al. 1994. Transmission of BSE and scrapie to mice: strain variation and the species barrier. Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences 343(1306):405-411.

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.

Cutlip RC, Miller JM, Race RE, et al. 1994. Intracerebral transmission of scrapie to cattle. Journal of Infectious Diseases 169(4):814-820.

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.

Dealler SF, Lacey RW. 1994. Suspected vertical transmission of BSE. [Correspondence]. Veterinary Record 134(6):151.

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.

Hoinville LJ. 1994. Decline in the incidence of BSE in cattle born after the introduction of the 'feed ban'. Veterinary Record 134(11):274-275.

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

Purdey M. 1994. Are organophosphate pesticides involved in the causation of BSE? Hypothesis based upon a literature review and limited trials on BSE cattle. Journal of Nutritional Medicine 4(1):43-82.

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.

Wells GAH, Dawson M, Hawkins SAC, et al. 1994. Infectivity in the ileum of cattle challenged orally with BSE. Veterinary Record 135(2):40-41.

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.

Wilesmith JW, Wells GAH, Hoinville LJ, et al. 1994. Suspected vertical transmission of BSE [Correspondence]. Veterinary Record 134(8):198-199.

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

1993

Baker HF, Ridley RM, Wells GAH. 1993. Experimental transmission of BSE and scrapie to the common marmoset. Veterinary Record 132(16):403-406.

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.

Foster JD, Hope J, Fraser H. 1993. Transmission of BSE to sheep and goats. Veterinary Record 133(14):339-341.

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.

Purdey M. 1993. Mad cow disease. [Correspondence] Ecologist 23(1):36-37.

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.

Wilesmith JW. 1993. BSE: epidemiological approaches, trials and tribulations. Preventive Veterinary Medicine 18(1):33.

Wilesmith JW, Ryan JBM. 1993. BSE: observations on the incidence during 1992. Veterinary Record 132(12):300-301.

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

Denny GO, Wilesmith JW, Clements RA, et al. 1992. Bovine spongiform encephalopathy in Northern Ireland: epidemiological observations 1988-1990. Veterinary Record 130(6):113-116.

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.

Fraser H, Bruce ME, Chree A, et al. 1992. Transmission of bovine spongiform encephalopathy and scrapie to mice. Journal of General Virology 73(8):1891-1897.

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.

Wilesmith JW, Ryan JBM, Hueston WD. 1992. Bovine spongiform encephalopathy: case-control studies of calf feeding practices and meat and bone meal inclusion in proprietary concentrates. Research in Veterinary Science 52(3):325-331.

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.

Wilesmith JW, Ryan JBM. 1992. Bovine spongiform encephalopathy: recent observations on the age-specific incidences. Veterinary Record 130(22):491-492.

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.

Wilesmith JW, Ryan JBM, Hueston WD, et al. 1992. Bovine spongiform encephalopathy: epidemiological features 1985 to 1990. Veterinary Record 130(5):90-94.

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.

1991

Barlow RM, Middleton DJ. 1991. Oral transmission studies of BSE to mice. Sub-acute spongiform encephalopathies. Proceedings of a seminar in the CEC Agricultural Research Program, held in Brussels, November 1990 [Edited by Bradley R, Savey M, Marchant B] Dordrecht, Netherlands. Kluwer Academic Publishers, pp.33-39.

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

Dawson M, Wells GAH, Parker BNJ, et al. 1991. Transmission studies of BSE in cattle, hamsters, pigs, and domestic fowl. Sub-acute spongiform encephalopathies. Proceedings of a seminar in the CEC Agricultural Research Program, held in Brussels, November 1990 [Edited by Bradley R, Savey M, Marchant B] Dordrecht, Netherlands. Kluwer Academic Publishers, pp.25-32.

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.

Pattison IH. 1991. Origins of BSE [Correspondence]. Veterinary Record 128(11):262-263.

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.

Wilesmith JW, Ryan JBM, Atkinson MJ. 1991. Bovine spongiform encephalopathy: epidemiology studies on the origin. Veterinary Record 128(9):199-203.

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.

1990

Aldhous P. 1990. BSE. New fears on transmission. Nature (London) 345(6273):280.

Barlow RM, Middleton DJ. 1990. Dietary transmission of bovine spongiform encephalopathy to mice. Veterinary Record 126(5):111-112.

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.

Dawson M, Wells GAH, Parker BNJ. 1990. Preliminary evidence of the experimental transmissibility of bovine spongiform encephalopathy to cattle. Veterinary Record 126(5):112-113.

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.

Gibbs CJ Jr, Safar J, Ceroni M, et al. 1990. Experimental transmission of scrapie to cattle. Lancet (British Edition) 335(8700);1275.

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.

McCracken RM, McIlroy SG, Denny GO, et al. 1990. Epidemiological studies of BSE in Great Britain and Northern Ireland. Society for Veterinary Epidemiology and Preventive Medicine. Proceedings of a meeting held at the Queen's University, Belfast, April 4th-6th, 1990 [edited by Thrusfield MV]. Roslin, Midlothian, UK, Society for Veterinary Epidemiology and Preventive Medicine.

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.

Taylor KC. 1990. Incidence of BSE. [Correspondence]. Veterinary Record 126(20):513.

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.

Wijeratne WVS, Curnow RN. 1990. A study of the inheritance of susceptibility to bovine spongiform encephalopathy. Veterinary Record 126(1):5-8.

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

Fraser H, McConnell I, Wells GAH. 1989. Transmission of bovine spongiform encephalopathy and scrapie to mice. Neuropathology and Applied Neurobiology 15(6):590.

1988

Fraser H, McConnell I, Wells GAH, et al. 1988. Transmission of bovine spongiform encephalopathy to mice. Veterinary Record 123(18):472.

Wilesmith JW, Wells GAH, Cranwell MP, et al. 1988. Bovine spongiform encephalopathy: Epidemiology studies. Veterinary Record 123(25):638-644.

TSE's - General

1996

Holmes S. 1996. Update - Making sense of bovine spongiform encephalopathy. Nursing Times 92(18):38.

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

Ridley RM, Baker HF. 1996. Creutzfeldt-Jakob disease and bovine spongiform encephalopathy - Aetiology of scrapie in certain circumstances is not evidence against another aetiology in different circumstances. British Medical Journal 312(7024):170.

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.

Goldfarb LG, Brown P. 1995. The transmissible spongiform encephalopathies. Annual Review of Medicine 46:57-65.

Prusiner SB. 1995. The prion diseases. Scientific American 272(1):48-57.

SEAC reports on transmissible spongiform encephalopathies. 1995. Veterinary Record 136(11):254-255.

Tateishi J, Kitamoto T. 1995. Inherited prion diseases and transmission to rodents. Brain Pathology 5(1):53-59.

Truyen U, Parrish CR, Harder TC, et al. 1995. There is nothing permanent except change. The emergence of new virus diseases. Laboratory Animals 29(1):103-122.

UK, Spongiform Encephalopathy Advisory Committee. 1995. Transmissible spongiform encephalopathies: a summary of present knowledge and research. September 1994. HMSO Publications Centre, London, UK.

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

Bradley R. 1994. Embryo transfer and its potential role in control of scrapie and BSE. Livestock Production Science 38(1):51-59.

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.

Liberski PP. 1994. Transmissible spongiform encephalopathies or prion disorders -- current views. Folia Neurology 32(2):65-73.

Schreuder BEC. 1994. Animal spongiform encephalopathies - an update. Part 1. Scrapie and lesser known animal spongiform encephalopathies. Veterinary Quarterly 16(3):174-181.

Taylor DM, Fraser H, McConnell I, et al. 1994. Decontamination studies with the agents of BSE and scrapie. Archives of Virology 139(3):313-326.

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.

1993

Austin AR, Simmons MM. 1993. Reduced rumination in BSE and scrapie. Veterinary Record 132(13):324-325.

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 disorders. British Medical Bulletin 49(4):725-737.

Esmond TFG, Will RG. 1993. Transmissible spongiform encephalopathies and human neurodegenerative disease. British Journal of Hospital Medicine 49(6):400-404.

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.

Hunter N. 1993. Genetic control of scrapie incidence in sheep and its relevance for BSE in cattle. Reviews in Medical Virology 3(4):195-200.

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.

Liberski PP. 1993. The enigma of slow viruses. Facts and artefacts. Wien, Austria. Springer Verlag.

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

Prusiner SB. 1993. Genetic and infectious prion diseases. Archives of Neurology 50(11):1129-1153.

Prusiner SB. 1993. Prion encephalopathies of animals and humans. Developmental Biology Standards 80:31-44.

Schreuder BEC. 1993. General aspects of transmissible spongiform encephalopathies and hypotheses about the agents. Veterinary Quarterly 15(4):167-174.

1992

Bradley R, Matthews D [Editors]. 1992. Transmissible spongiform encephalopathies of animals. Revue Scientifique et Technique - Office International des Epizooties 11(2):333-634.

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.

Lantos PL. 1992. From slow virus to prion: a review of transmissible spongiform encephalopathies. Histopathology 20(1):1-11.

1991

Brown P, Mitrova E (Editors). 1991. Symposium on human and zoonotic spongiform encephalopathies. European Journal of Epidemiology 7(5):439-578.

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.

Chesebro BW (Editor). 1991. Transmissible spongiform encephalopathies: scrapie, BSE and related human disorders. Current Topics in Microbiology and Immunology 172.

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

Taylor DM. 1991. Inactivation of the unconventional agents of scrapie, BSE, and CJD. Journal of Hospital Infection 18(A):141-146.

1990

Bradley R, Savey M, Marchant B (Editors). 1990. Sub-acute spongiform encephalopathies. Proceedings of a Seminar in the CEC Agricultural Research Program, held in Brussels, 12-14 November 1990. Dordrecht, Netherlands, Kluwer Academic Publishers.

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.

Dealler SF, Lacey RW. 1990. Transmissible spongiform encephalopathies: the threat of BSE to man. Food Microbiology 7(4):253-279.

Gibson PH. 1990. Transmission of encephalopathies. [Correspondence] Veterinary Record 126(10):248.

Grant HC. 1990. Transmission of encephalopathies. Veterinary Record 126(10):248.

Lewis PK. 1990. Scrapie and human neurodegenerative diseases. [Correspondence] Canadian Medical Association Journal 142(9):928.

McKerrell RE, Farmer H. 1990. Spongiform encephalopathies: Implications of recent developments. Report of a joint meeting of Sections of Comparative Medicine, Epidemiology, Community Medicine, and Pathology. Journal of the Royal Society of Medicine 83(5):334-336.

Westaway D, Prusiner SB. 1990. Link between scrapie and BSE. [Correspondence] Nature 346(6280):113.

1989

Blakemore WF. 1989. Bovine spongiform encephalopathy and scrapie: potential human hazards. Outlook Agriculture 18(4):165-168.

Burns KN. 1989. Current animal events concerning human health. Journal of the Royal Agricultural Society of England 150:90-100.

Taylor DM. 1989. Scrapie agent decontamination: Implications for bovine spongiform encephalopathy. Veterinary Record 124(12):291-292.

1987

Taylor DM, McBride PA. 1987. Autoclaved, formol-fixed scrapie mouse brain is suitable for histopathological examination, but may still be infective. Acta Neuropathologica 74(2):194-196.

Autoclaving scrapie infected mouse brain to 134-138C 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

Brown P, Rohwer RG, Gajdusek DC. 1986. Newer data on the inactivation of scrapie virus or Creutzfeldt-Jakob disease virus in brain tissue. Journal of Infectious Diseases 153(6):1145-1148.

Manuelidis L, Manuelidis EE. 1986. Recent developments in scrapie and Creutzfeldt-Jakob disease. Progress in Medical Virology 33:78-98.

1980

Gibbs CJ Jr, Amyx, HL, Bacote A, et al. 1980. Oral transmission of kuru, Creutzfeldt-Jakob disease, and scrapie to nonhuman primates. Journal of Infectious Diseases 142(2):205-208.

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

Latarjet R. 1979. Inactivation of the agents of scrapie, Creutzfeldt-Jakob disease, and kuru by radiations. Slow transmissible diseases of the nervous system. Volume 2. [edited by Prusiner SB, Hadlow WJ]. New York, USA, Academic Press, pp. 387-407.

Prusiner SB, Hadlow WJ [Editors]. 1979. Slow transmissible diseases of the nervous system. Volume 1. Clinical, epidemiological, genetic, and pathological aspects of the spongiform encephalopathies. New York, USA, Academic Press.

The papers in this book cover four diseases: kuru and CJD of man; scrapie of sheep and goats; and TME.

TSE's - Etiology and Pathogenesis

1996

Brandner S, Isenmann S, Raeber A, et al. 1996. Normal host prion protein necessary for scrapie-induced neurotoxicity. [Correspondence] Nature (London) 379(6563):339-343.

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.

Carlson GA. 1996. Prion strains. Current Topics in Microbiology and Immunology 207:35-47.

DeArmond SJ, Prusiner SB. 1996. Transgenetics and neuropathology of prion diseases. Current Topics in Microbiology and Immunology 207:25-46.

Gabison R, Telling G, Meiner Z, et al. 1996. Insoluble wild-type and protease-resistant mutant prion protein in brains of patients with inherited prion disease. Nature Medicine 2(1):59-64.

Huang Z, Prusiner SB, Cohen FE. 1996. Structures of prion proteins and conformational models for prion diseases. Current Topics in Microbiology and Immunology 207:49-67.

Lehmann S, Harris DA. 1996. Mutant and infectious prion proteins display common biochemical properties in cultured cells. Journal of Biological Chemistry 271(3):1633-1637.

Prusiner SB. 1996. Human prion diseases and neurodegeneration. Current Topics in Microbiology and Immunology 207:1-17.

Purdey M. 1996. The UK epidemic of BSE: Slow virus or chronic pesticide-initiated modification

of the prion protein? Part1: Mechanisms for a chemically induced pathogenesis/transmissibility. Medical hypotheses 46(5):429.

Saeki K, Matsumoto Y, Matsumoto Y, et al. 1996. Identification of a promoter region in the rat prion protein gene. Biochemical and Biophysical Research Communications 219(1):47-52.

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

Scott MR, Telling GC, Prusiner SB. 1996. Transgenetics and gene targeting in studies of prion diseases. Current Topics in Microbiology and Immunology 207:95-123.

Somerville RA, Dunn AJ. 1996. The association between PrP and infectivity in scrapie and BSE infected mouse brain. Archives of Virology 141(2):275-289

Sommer SS, Rocca WA. 1996. Prion analogues and twin studies in Parkinson's disease. Neurology 46(1):273-275.

Sutherland K, Goodbrand IA, Bell JE, et al. 1996. Objective quantification of prion protein in spinal cords of cases of Creutzfeldt-Jakob disease. Analytical Cellular Pathology 10(1):25-35.

Weiss S, Rieger R, Edenhofer F, et al. 1996. Recombinant prion protein rPrP27-30 from Syrian golden hamster reveals proteinase K sensitivity. Biochemical and Biophysical Research Communications 219(1):173-179.

1995

Bessen RA, Kocisko DA, Raymond GJ, et al. 1995. Non-genetic propagation of strain-specific properties of scrapie prion protein. Nature 375(6533):698-700.

Biernat W, Liberski PP, Guiroy DC, et al. 1995. Proliferating cell nuclear antigen immunohistochemistry in astrocytes in experimental Creutzfeldt-Jakob disease and in human kuru,

Creutzfeldt-Jakob disease and Gerstmann-Straussler-Scheinker syndrome. Neurodegeneration 4(2):195-201.

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.

Caughey B, Kocisko DA, Raymond GJ, et al. 1995. Aggregates of scrapie-associated prion protein induce the cell-free conversion of protease-sensitive prion protein to the protease-resistant state. Chemistry & Biology 2(12):807-817.

Chen SG, Teplow DB, Parchi P, et al. 1995. Truncated forms of the human prion protein in normal brain and in prion diseases. Journal of Biological Chemistry 270(32):19173-19180.

Collinge J, Palmer MS, Sidle KCL. 1995. Unaltered susceptibility to BSE in transgenic mice expressing human prion protein. Nature (London) 378(6559):779-783.

DeArmond SJ, Prusiner SB. 1995. Prion protein transgenes and the neuropathology in prion diseases. Brain Pathology 5(1):77-89.

DeArmond SJ, Prusiner SB. 1995. Etiology and pathogenesis of prion diseases. American Journal of Pathology 146(4):785-811.

Hauw JJ, Lazarini F, Seilhean D, et al. 1995. Unconventional infectious agents or prions. Annales de Pathologie 15(6):409-414.

Jeffrey M, Goodbrand IA, Goodsir CM. 1995. Pathology of the TSE's with special emphasis on ultrastructure. Micron 26(3):277-298.

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.

Kannenberg K, Groschup MH, Sigel E. 1995. Cellular prion protein and GABA(A) receptors: No physical association? Neuroreport 7(1):77-80.

Lee JT. 1995. Prions as a cause of infectious, genetic, and sporadic neurodegenerative diseases. Clinical Microbiology Newsletter 17(5):33-38.

Ossa JE, Machado G, Giraldo MA. 1995. Prion plaques: molecular tumors. A hypothesis on the etiopathogenesis of prion diseases. Medical Hypotheses 44(2):124-126.

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.

Parchi P, Castellani R, Cortelli P, et al. 1995. Regional distribution of protease-resistant prion protein in fatal familial insomnia. Annals of Neurology 38(1):21-29.

Priola SA, Chesebro B. 1995. A single hamster PrP amino acid blocks conversion to protease-resistant PrP in scrapie-infected mouse neuroblastoma cells. Journal of Virology 69(12):7754-7758.

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.

Prusiner SB, DeArmond SJ (Editors) 1995. Neuropathology of prion diseases. [Symposium] Brain Pathology 5(1):25-103.

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

Smith C, Collinge J. 1995. Molecular pathology of prion diseases. Essays in Biochemistry 29:157-174.

Telling GC, Scott M, Mastrianni J, et al. 1995. Prion propagation in mice expressing human and chimeric PrP transgenes implicates the interaction of cellular PrP with another protein. Cell 83(1):79-90.

Weissmann C. 1995. Prion diseases. Yielding under the strain. [Comment] Nature 375(6533):628-629.

Westaway D, Carlson GA, Prusiner SB. 1995. On safari with PrP: prion diseases of animals. Trends in Microbiology 3(4):141-147.

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.

Whittington MA, Sidle KC, Gowland I, et al. 1995. Rescue of neurophysiological phenotype seen in PrP null mice by transgene encoding human prion protein [published erratum appears in Nature Genetics 9(4):451]. Nature Genetics 9(2):197-201.

1994

Berg LJ. 1994. Insights into the role of the immune system in prion diseases. Proceedings of the National Academy of Sciences, USA 91(2):429-432.

Carlson GA, DeArmond SJ, Torchia M, et al. 1994. Genetics of prion diseases and prion diversity in mice. Biological Sciences 343(1306):363-369.

Cohen FE, Pan KM, Huang Z, et al. 1994. Structural clues to prion replication. Science 264(5158):530-531.

Collinge J, Weissmann C (Editors). 1994. Molecular biology of prion diseases. Proceedings of a Royal Society discussion meeting held on 22 and 23 September 1993. Philosophical Transactions

of the Royal Society of London. Series B, Biological Sciences 343(1306):357-463.

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.

Collinge J, Whittington MA, Sidle KCL, et al. 1994. Prion protein is necessary for normal synaptic function. [Correspondence] Nature (London) 370(6487):295-297.

Fatal familial insomnia and familial Creutzfeldt-Jakob disease: different prion proteins determined by a DNA polymorphism. Proceedings of the National Academy of Sciences, USA, 91(7):2839-2842.

Fink JK, Peacock ML, Waren JT, et al. 1994. Detecting prion protein gene mutations by denaturing gradient gel electrophoresis. Human Mutations 4(1):42-50.

Gomi H, Ikeda T, Kunieda T, et al. 1994. Prion protein (PrP) is not involved in the pathogenesis of spongiform encephalopathy in zitter rats. Neuroscience Letters 166(2):171-174.

Groschup MH, Langeveld J, Pfaff E. 1994. The major species specific epitope in prion proteins of ruminants. Archives of Virology 136(3/4):423-431.

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.

Hope J, Chong A. 1994. Scrapie, CJD, and BSE: the key role of nerve membrane protein (PrP). Biochemical Society Transactions 22(1):159-163.

Hsiao KK. 1994. The genetics and transgenetics of human prion disease. Annals of the New York Academy of Sciences 724:241-245.

Hunter N, Goldmann W, Smith G, et al. 1994. Frequencies of PrP gene variants in healthy cattle and cattle with BSE in Scotland. Veterinary Record 135(17):400-403.

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.

Kuramoto T, Mori M, Yamada J, et al. 1994. Tremor and zitter, causative mutant genes for epilepsy with spongiform encephalopathy in spontaneously epileptic rat, are tightly linked to synaptobrevin-2 and prion protein genes, respectively. Biochemical and Biophysical Research Communications 200(2):1161-1168.

Laplanche JL, Delasnerie-Laupretre N, Brandel JP, et al. 1994. Molecular genetics of prion diseases in France. French Research Group on Epidemiology of Human Spongiform Encephalopathies. Neurology 44(12):2347-2351.

Neibergs HL, Ryan AM, Womack JE, et al. 1994. Polymerase analysis of the prion gene in BSE-affected and unaffected cattle. Animal Genetics 25(5):313-317.

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.

Petersen RB, Goldfarb LG, Tabaton M, et al. 1994. A novel mechanism of phenotypic heterogeneity demonstrated by the effect of a polymorphism on a pathogenic mutation in the prion protein gene. Molecular Neurobiology 8(2/3):99-103.

Prusiner SB. 1994. Biology and genetics of prion diseases. Annual Review of Microbiology 48:655-686

Prusiner SB. 1994. Molecular biology and genetics of prion diseases. Philosophical Transaction of the Royal Society of London. Series B, Biological Sciences 343(1306):447-463.

A review.

Prusiner SB. 1994. Inherited prion diseases. Proceedings of the National Academy of Sciences, USA 91(11):4611-4614.

Ridley RM. 1994. Perceptions of prion diseases. Journal of Clinical Pathology 47(10):876-879.

Westaway D, DeArmond SJ, Cayetano-Canlas J, et al. 1994. Degeneration of skeletal muscle, peripheral nerves, and the central nervous system in transgenic mice overexpressing wild-type prion proteins. Cell 76(1):117-129.

Wisniewski HM, Wegiel J, Kozielski R. 1994. Amyloidosis in prion diseases and cells involved in PrP fibrillogenesis. Annals of the New York Academy of Sciences 724-191-201.

1993

Carr K. 1993. Pion diseases. A question of conformation. Nature 365(6445):386.

Come JH, Fraser PE, Lansbury PT. 1993. A kinetic model for amyloid formation in the prion diseases: importance of seeding. Proceedings of the National Academy of Sciences, USA 90(13):5959-5963.

DeArmond SJ, Prusiner SB. 1993. The neurochemistry of prion diseases. Journal of Neurochemistry 61(5):1589-1601.

Groschup MH, Pfaff E. 1993. Studies on a species-specific epitope in murine, ovine, and bovine prion protein. Journal of General Virology 74(7):1451-1456.

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.

Jarret JT, Lansbury PT, JR. 1993. Seeding ‘one-dimensional crystallization’ of amyloid: a pathogenic mechanism in Alzheimer’s disease and scrapie? Cell (Cambridge) 73(6):1055-1058.

Medori R, Tritschler HJ. 1993. Prion protein gene analysis in three kindreds with fatal familial insomnia (FFI): codon 178 mutation and codon 129 polymorphism. American Journal of Human Genetics 53(4):822-827.

Narang HK. 1993. Evidence that tubulofilamentous particles are the common agent of scrapie, CJD, and BSE. [Abstract]. Neuropathology and Applied Neurobiology 19(5):447.

Narang HK. 1993. Evidence that scrapie-associated tubulofilamentous particles contain a single-stranded DNA. Intervirology 36(1):1-10.

Palmer MS, Collinge J. 1993. Mutations and polymorphisms in the prion protein gene. Human Mutations 2(3):168-173.

Poidinger M, Kirkwood J, Almond W. 1993. Sequence analysis of the PrP protein from two species of antelope susceptible to TSE. Archives of Virology 131(1/2):193-199.

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.

Pollanen MS, Bergeron C. Wyer L. 1993. Absence of protease-resistant prion protein in dementia characterized by neuronal loss and status spongiosus. Acta Neuropathologica 86(5):515-517.

Prusiner SB. 1993. Transgenetics and cell biology of prion diseases: investigation of PrPSc synthesis and diversity. British Medical Bulletin 49(4):873-912.

Prusiner SB. 1993. Transgenetic investigations of prion diseases of humans and animals. Philosophical Transactions of the Royal Society of London, Series B, Biological Sciences 339(1288):239-254.

Prusiner SB, Fuzi M, Scott M, et al. 1993. Immunologic and molecular biologic studies of prion proteins in BSE. Journal of Infectious Diseases. 167(3):602-613.

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.

Watanabe R, Duchen LW. 1993. Cerebral amyloid in human prion disease. Neuropathology and Applied Neurobiology 19(3):253-260.

Weissmann C, Bueler H, Fischer M, et al. 1993. The Prp-less mouse: a tool for prion research. Transgenic animals as model systems for human diseases [edited by Wagner EF, Theuring F]. Berlin, Germany. Springer Verlag, pp. 39-56.

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.

Weissmann C, Beuler H, Fischer M, et al. 1993. Role of the PrP gene in transmissible spongiform encephalopathies. Intervirology 35(1/4):164-175.

Weissmann C, Bueler H, Sailer A, et al. 1993. Role of PrP in prion diseases. British Medical Bulletin 49(4):995-1011.

1992

Alper T. 1992. The infectivity of spongiform encephalopathies: does a modified membrane hypothesis account for lack of immune response? FEMS Microbiology Immunology 89(5):235-242.

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.

Bennett AD, Birkett CR, Bostock CJ. 1992. Molecular biology of scrapie-like agents. Revue Scientifique et Technique - Office International des Epizooties 11(2):569-603.

Humphery-Smith I, Chastel C, Goff FL. 1992. Spiroplasmas and spongiform encephalopathies. [Correspondence] Medical Journal of Australia 156(2):142.

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.

Jeffrey M, Scott JR, Williams A, et al. 1992. Ultrastructural features of spongiform encephalopathy transmitted to mice from three species of bovidae. Acta Neuropathologica 84(5):559-569.

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.

Kitamoto T, Shin RW, Dob Ura K, et al. 1992. Abnormal isoform of prion proteins accumulates in the synaptic structures of the central nervous system in patients with Creutzfeldt-Jakob disease. American Journal of Pathology 140:1285-1294.

Laszlo L, Lowe J, Self T, et al. 1992. Lysosomes as key organelles in the pathogenesis of prion encephalopathies. Journal of Pathology 166(4):333-341.

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.

Liberski PP, Yanagihara R, Gibbs CJ Jr., et al. 1992. Neuronal autophagic vacuoles in experimental scrapie and CJD. Acta Neuropathologica 83(2):134-139.

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

Liberski PP, Yanagihara R, Wells GAH, et al. 1992. Ultrastructural pathology of axons and myelin in experimental scrapie in hamsters and BSE in cattle and a comparison with the panencephalopathic type of CJD. Journal of Comparative Pathology 106(4):383-398.

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.

Liberski PP, Yanagihara R. Wells GAH, et al. 1992. Comparative ultrastructural neuropathology of naturally occurring bovine spongiform encephalopathy and experimentally induced scrapie and CJD. Journal of Comparative Pathology 106(4):361-381.

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.

Narang HK. 1992. Scrapie-associated tubulofilamentous particles in scrapie hamsters. Intervirology 34(2):105-111.

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.

Narang HK. 1992. Relationship of protease-resistant protein, scrapie-associated fibrils and tubulofilamentous particles to the agent of SE's. Research in Virology 143(6):381-386.

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.

Prusiner SB. 1992. Prion biology. Prion diseases of humans and animals [Edited by Prusiner SB, Collinge J, Powell J, et al.]. Chichester, West Sussex, UK. Ellis Horwood Limited.

Prusiner SB, Collinge J, Powell J, et al. [Editors] 1992. Prion diseases of humans and animals. Chicheser, West Sussex, UK. Ellis Horwood Limited.

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.

Roberts GW, Clinton J. 1992. Prion disease: the spectrum of pathology and diagnostic considerations. Prion diseases of humans and animals [Edited by Prusiner SB, Collinge J, Powell J, et al.]. Chichester, West Sussex, UK. Ellis Horwood Limited.

Yoshimoto J, Iinuma T, Ishiguro N, et al. 1992. Comparative sequence analysis and expression of bovine PrP gene in mouse L-929 cells. Virus Genes 6(4):343-356.

1991

Brown P, Goldfarb LG, Gajdusek DC. 1991. The new biology of spongiform encephalopathy: infectious amyloidoses with a genetic twist. Lancet (British edition) 337(8748):1019-1022.

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.

Diringer H. 1991. Transmissible spongiform encephalopathies virus-induced amyloidoses of the central nervous system. European Journal of Epidemiology 7(5):562-566.

Gajdusek DC. 1991. The transmissible amyloidoses: genetical control of spontaneous generation of infectious amyloid proteins by nucleation of configurational change in host precursors: kuru-CJD-GSS-scrapie-BSE. European Journal of Epidemiology 7(5):567-577.

Goldmann W, Hunter N, Martin T, et al. 1991. Different forms of the bovine PrP gene have five or six copies of a short G-C-rich element within the protein-coding exon. Journal of General Virology 72(1):201-204.

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.

Liberski PP, Brown P, Xiao SY, et al. 1991. The ultrastructural diversity of scrapie-associated fibrils isolated from experimental scrapie and CJD. Journal of Comparative Pathology 105(4):377-386.

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.

Prusiner SB. 1991. Molecular Biology of Prion Diseases. Science 252:1515-1522.

Prusiner SB, Torchia M, Westaway D. 1991. Molecular biology and genetics of prions - implications for sheep scrapie, “mad cows” and the BSE epidemic [Editorial]. Cornell Veterinarian 81(2):85-101.

Prusiner SB, Westaway D. 1991. Infectious and genetic manifestations of prion diseases. Molecular Plant-Microbe Interactions 4(3):226-233.

Safar J, Ceroni M, Gajdusek DC, et al. 1991. Differences in the membrane interaction of scrapie amyloid precursor proteins in normal and scrapie- or CJD-infected brains. Journal of Infectious Diseases 163(3):488-494.

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

Chesebro B. 1990. Spongiform encephalopathies: the transmissible agents. Fields Virology. Volume 2 [edited by Fields BN, Knipe DM]. New York, USA, Raven Press, pp. 2335-2336.

Gajdusek DC. 1990. Subacute spongiform encephalopathies: transmissible cerebral amyloidoses caused by unconventional viruses. Fields Virology. Volume 2 [edited by Fields BN, Knipe DM]. New York, USA, Raven Press, pp. 2289-2324..

Kimberlin RH. 1990. Unconventional ‘slow’ viruses. Topley & Wilson’s Principles of bacteriology, virology and immunity. Volume 4. Virology [edited by Collier LH, Timbury MC]. London, UK, Edward Arnold (Publisher) Ltd.

Kingsbury DT. 1990. Genetics of response to slow virus (prion) infection. Annual Review of Genetics 24:115-132.

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

Liberski PP, Yanagihara R, Gibbs CJ Jr, et al. 1990. Appearance of tubulovesicular structures in experimental Creutzfeldt-Jakob disease and scrapie precedes the onset of clinical disease. Acta Neuropathologica 79(4):349-354.

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.

Prusiner SB. 1990. Novel structure and genetics of prions causing neurodegeneration in humans and animals. Biologicals 18:24-262.

Prusiner SB, De Armond S. 1990. Prion diseases of the central nervous system. Monographs in Pathology 1990 pp 86-122.

Safar J, Ceroni M, Piccardo P, et al. 1990. Scrapie-associated precursor proteins: antigenic relationship between species and immunocytochemical localization in normal, scrapie, and Creutzfeldt-Jakob disease brains. Neurology 49(3):513-517.

Snow AD, Wight TN, Nochlin D, et al. 1990. Immunolocalization of heparan sulfate proteoglycans to the prion protein amyloid plaques of Gerstmann-Straussler, Creutzfeldt-Jakob disease and scrapie. Laboratory Investigation 63(5):601-611.

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.

Will RG. 1990. Prion disease [Correspondence]. Lancet (British Edition) 336(8711):369-370.

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

Fraser H, Bruce ME, McBride PA, et al. 1989. The molecular pathology of scrapie and the biological basis of lesion targeting. In: Alzheimer's Disease and Related Disorders. Alan R. Liss, Inc, pp 637-644.

Hunter N, Foster JD, Dickinson AG, et al. 1989. Linkage of the gene for the scrapie-associated fibril protein (PrP) to the Sip gene in Cheviot sheep. Veterinary Record 124:364-366.

1988

Baron H, Baron-Van Evercooren A, Brucher JM. 1988. Antiserum to scrapie-associated fibril protein reacts with amyloid plaques in familial transmission dementia. Journal of Neuropathology and Experimental Neurology 47(2):158-165.

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.

Bockman JM, Kingsbury DT. 1988. Immunological analysis of host and agent effects on Creutzfeldt-Jakob disease and scrapie prion proteins. Journal of Virology 62(9):3120-3127.

Butler DA, Scott MRD, Bockman JM, et al. 1988. Scrapie-infected murine neuroblastoma cells produce protease-resistant prion proteins. Journal of Virology 62(5):1558-1564.

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.

Hope J, Reekie LJD, Hunter N, et al. 1988. Fibrils from brains of cows with new cattle disease contain scrapie-associated protein. Nature 336(6197):390-392.

Liberski PP, Yanagihara R, Gibbs CJ Jr, et al. 1988. Tubulovesicular structures in experimental Creutzfeldt-Jakob disease and scrapie. Intervirology 29(2):115-119.

Millot P, Chatelain J, Dautheville C, et al. 1988. Sheep major histocompatibility (OLA) complex: linkage between a scrapie susceptibility/resistance locus and the OLA comlex in Ile-de-France sheep progenies. Immunogenetics 27:1-11.

1987

Hogan RN, Baringer JR, Prusiner SB. 1987. Scrapie infection diminishes spines and increases varicosities of dendrites in hamsters: a quantitative Golgi analysis. Journal of Neuropathology and Experimental Neurology 46(4):461-473.

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.

Prusiner SB. 1987. An introduction to scrapie and Creutzfeldt-Jakob disease research. Prions. Novel infectious pathogens causing scrapie and Creutzfeldt-Jakob disease. [Edited by Prusiner SB, McKinley MP]. London, UK, Academic Press Inc.

Prusiner SB, McKinley MP. [Editors]. 1987. Prions. Novel infectious pathogens causing scrapie and Creutzfeldt-Jakob disease. London, UK, Academic Press Inc.

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

Bode L, Pocchiara M, Gelderblom H, et al. 1985. Characterization of antisera against scrapie-associated fibrils (SAF) from affected hamster and cross-reactivity with SAF from scrapie-affected mice and from patients with Creutzfeldt-Jakob disease. Journal of General Virology 66(11):2471-2478.

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.

Gilmour JS, Bruce ME, Mackellar A. Cerebrovascular amyloidosis in scrapie-affected sheep. Neuropathology and Applied Neurobiology 11:173-183.

Millot P, Chatelain J, Cathala F. Sheep major histocompatibility complex OLA: gene frquencies in two French breeds with scrapie. Immunogenetics 21:117-123.

Ridley RM, Baker HF, Crow TJ. 1985. Virogenes in scrapie and Creutzfeldt-Jakob disease. [Correspondence]. Journal of the Royal Society of Medicine 78(11):970.

1984

Masters CL, Rohwer RG, Franko MC, et al. 1984. The sequential development of spongiform change and gliosis of scrapie in the golden Syrian hamster. Journal of Neuropathology and Experimental Neurology 43(3):242-252.

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.

Merz PA, Rohwer RG, Kascsak R, et al. 1984. Infection-specific particle from the unconventional slow virus diseases. Science 225(4660):437-440.

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.

1983

Merz PA, Somerville RA, Wisniewski HM, et al. 1983. Scrapie-associated fibrils in Creutzfeldt-Jakob disease. Nature, UK 306(5942):474-476.

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.

1982

Prusiner SB. 1982. Novel proteinaceous infectious particles cause scrapie. Science 216:136-144.

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

Marx JL. 1973. Slow viruses: Role in persistent disease. Science 181:1351-1354.

Marx JL. 1973. Slow viruses (II): The unconventional agents. Science 181:44-45.

1963

Draper GJ. 1963. ‘Epidemics’ caused by a late-manifesting gene; application to scrapie. Heredity 18:165-171.

TSE's - Diagnosis

1996

Featherstone T. 1996. Creutzfeldt-Jakob disease and bovine spongiform encephalopathy - Magnetic resonance imaging may have a role in diagnosing Creutzfeldt-Jakob disease. British Medical Journal 312(7074):180.

Hainfellner JA, Jellinger K, Budka H. 1996. Testing for prion protein does not confirm previously reported conjugal CJD. Lancet 347(9001):616-617.

Schreuder BEC, van Keulen LJM, Vromens MEW, et al. 1996. Preclinical test for prion diseases. Nature 381:563.

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

Beekes M, Baldauf E, Cassens S, et al. 1995. Western blot mapping of disease-specific amyloid in various animal species and humans with TSE's using high-yield purification method. Journal of General Virology 76(10):2567-2576.

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.

Graber HU, Meyer RK, Fatzer R, et al. 1995. In situ hybridization and immunohistochemistry for prion protein (PrP) in bovine spongiform encephalopathy. Journal of Veterinary Medicine. Series A, 42(7):453-459.

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.

Katz JB, Shafer AM, Miller JM. 1995. Production of antiserum for the diagnosis of scrapie and BSE using a baculovirus-expressed prion protein antigen. Journal of Veterinary Diagnostic Investigation 7(2):245-247.

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

Keulen LJM van, Schreuder BEC, Meloen RH, et al. 1995. Immunohistochemical detection and localization of prion protein in brain tissue of sheep with natural scrapie. Veterinary Pathology 32(3):299-308.

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.

Short N, Otte J. 1995. Diagnosis of BSE. Veterinary Record 136(20):523.

Short N, Otte J. 1995. Diagnosis of BSE. Veterinary Record 136(11):274.

Taylor KC, Wilesmith JW. 1995. Diagnosis of BSE. Veterinary Record 136(13):335.

1994

Haritani M, Spencer YI, Wells GAH. 1994. Hydrated autoclave pretreatment enhancement of prion protein immunoreactivity in formalin-fixed BSE-affected brain. Acta Neuropathologica 87(1):86-90.

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.

Oberdieck U, Xi YG, Pocchiari M, et al. 1994. Characterization of antisera raised against species-specific peptide sequences from scrapie-associated fibril protein and their application for post-mortem immunodiagnosis of spongiform encephalopathies. Archives of Virology 136(1/2):99-110.

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.

Pocchiari M, Xi YG, Ingrosso L, et al. 1994. Immunodiagnosis of bovine spongiform encephalopathy. Livestock Production Science 38(1):41-46.

Scott PR. 1994. Cerebrospinal fluid analysis in the diagnosis of bovine neurological disease. Proceedings 18th World Buiatrics Congress: 26th Congress of the Italian Association of Buuiatrics, Bologna, Italy, August 29 - September 2, 1994 Vol. 1:365-368.

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.

Wells GAH, Scott AC, Wilesmith JW, et al. 1994. Correlation between the results of a histopathologic examination and the detection of abnormal brain fibrils in the diagnosis of BSE. Research in Veterinary Science 56(3):346-351.

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

Tateishi J, Kitamoto T. 1993. Developments in diagnosis for prion diseases. British Medical Journal 49(4):971-979.

1992

Mohri S, Farwuhar CF, Somerville RA, et al. 1992. Immunodetection of disease specific Prp fraction in scrapie-affected sheep and BSE-affected cattle. Veterinary Record 131(23):537-539.

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.

Peiffer J, Doerr-Schott J, Tateishi J. 1992. Immunohistochemistry with anti-prion protein 27-30 gives reactions with fungi. [Correspondence] Acta Neuropathologica 84(3):346-347.

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.

Scott AC, Wells GAH, Chaplin MJ, et al. 1992. Bovine spongiform encephalopathy: detection of fibrils in the central nervous system is not affected by autolysis. Research in Veterinary Science 52(3):332-336.

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.

Wells GAH, McGill IS. 1992. Recently described scrapie-like encephalopathies of animals: case definitions. Research in Veterinary Science 53(1):1-10.

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.

1991

Brugere H, Banissi C, Brugere-Picoux J, et al. 1991. Contribution of urinary examination to the diagnosis of spongiform encephalopathy of sheep. Contribution d'un examen urinaire au diagnostic de l'encephalopathie spongiforme du mouton. Bulletin Mensuel de la Society Veterinaire de France 75(5):277-279. (In French)

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.

Davis AJ, Jenny AL, Miller LD. 1991. Diagnostic characteristics of bovine spongiform encephalopathy. Journal of Veterinary Diagnostic Investigation 3(3):266-271.

Ghergariu S. 1991. Some problems of diagnosis of the spongiform encephalopathies in ruminants. European Journal of Epidemiology 7(5):28-531.

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.

Strain GM. 1991. Antemortem diagnosis of scrapie and bovine spongiform encephalopathy. Journal of the American Veterinary Medical Association 198(3):360.

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

1990

Scott AC, Wells GAH, Stack MJ, et al. 1990. Bovine spongiform encephalopathy: detection and quantitation of fibrils, fibril protein (PrP) and vacuolation in brain. Veterinary Microbiology 23(1-4):295-304.

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.

Serban D, Taraboulos A, DeArmond SJ, et al. 1990. Rapid detection of CJD and scrapie prion proteins. Neurology 40(1):110-117.

1989

Farquhar CF, Somerville RA, Ritchie LA. 1989. Post-mortem immunodiagnosis of scrapie and bovine spongiform encephalopathy. Journal of Virological Methods 24:215-222.

Wells GAH, Hancock RD, Cooley WA, et al. 1989. Bovine spongiform encephalopathy: Diagnostic significance of vacuolar changes in selected nuclei of the medulla oblongata. Veterinary Record 125(21):521-524.

1988

Hopkins AR. 1988. BSE: Not a self evident diagnosis. [Correspondence] Veterinary Record 122(26):639.

Human Spongiform Encephalopathies

1996

Chazot G, Broussolle E, Lapras CI, et al. 1996. New variant of Creutzfeldt-Jakob disease in a 26-year-old French man. Lancet 347(9009):1181.

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.

Collinge J, Beck J, Campbell T, et al. 1996. Prion protein gene analysis in new variant cases of Creutzfeldt-Jakob disease. Lancet 348:56.

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.

Manuelidis L, Fritch W. 1996. Infectivity and host responses in Creutzfeldt-Jakob disease. Virology 216(1):46-59.

Will RG, Ironside JW, Zeidler M, et al. 1996. A new variant of Creutzfeldt-Jakob disease in the UK. Lancet 347:921-925.

1995

Delasnerie-Laupretre N, et al. 1995. CJD in Europe. The Lancet 346:898.

Diringer H. 1995. Proposed link between transmissible spongiform encephalopathies of man and animals. Lancet 346:1208-1210.

Jibiki I, Fukushima T, Kobayashi K, et al. 1995. Antagonizing correlation between periodic

synchronous discharges and photically induced giant evoked responses in Creutzfeldt-Jakob disease (Heidenhain type): A case study. Psychiatry and Clinical Neurosciences 49(1):87-90.

Liberski PP, Budka H, Yanagihara R, et al. 1995. Neuroaxonal dystrophy in experimental CJD: electron microscopical and immunohistochemical demonstration of neurofilament accumulations within affected neurites. Journal of Comparative Pathology 112(3):243-255.

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.

Mendez-Martinez O, Luzardo-Small G, Molina-Viloria O, et al.1995. Creutzfeldt-Jakob disease. Report of 2 cases. Enfermedad de Creutzfeldt-Jakob. Reporte de dos casos. Investigaciones Clinicas 36(1):23-30. (In Spanish)

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.

Muhleisen H, Gehrmann J, Meyermann R. 1995. Reactive microglia in Creutzfeldt-Jakob disease. Neuropathology and Applied Neurobiology. 21(6):505-517.

Nicholl D, Windl O, de Silva R, et al. 1995. Inherited Creutzfeldt-Jakob disease in a British family associated with a novel 144 base pair insertion of the prion protein gene. Journal of Neurology, Neurosurgery, and Psychiatry 58(1):65-69.

Oken RJ, McGeer PL. 1995. Human prion diseases: possible new directions in prophylaxis and therapy. Medical Hypotheses 44(3):167-168.

Parchi P, Gambetti P. 1995. Human prion diseases. Current Opinions in Neurology 8(4):286-293.

Smith PEM, et al. 1995. CJD in a dairy farmer. The Lancet 346:898.

Tateishi J, Brown P, Kitamoto T, et al. 1995. First experimental transmission of fatal familial insomnia. Nature 1995 376(6539):434-435.

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

Terzano MG, Parrino L, Pietrini V, et al. 1995. Precocious loss of physiological sleep in a case of Creutzfeldt-Jakob disease: A serial polygraphic study. Sleep 18(10):849-858.

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van Gool WA, Hensels GW, Hoogerwaard EM, et al. 1995. Hypokinesia and presenile dementia in a Dutch family with a novel insertion in the prion protein gene. Brain 118(6):1565-1571.

Worthington JM, Stone SM. 1995. Epidemiology of Jakob-Creutzfeldt disease in Australia. Australian and New Zealand Journal of Medicine 25(3):243-244.

1994

Brandel JP, Delasnerie-Laupretre N, Laplanche JL, et al. 1994. A study network of human spongiform encephalopathies: 1st results. Reseau d'etudes des encephalopathies spongiformes humaines: premiers resultats. Revue Neurologique 150(10):684-688. (In French)

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.

Collinge J, Palmer MS. 1994. Human prion diseases. Clinical Neurology 3(2):241-247.

Prusiner SB, Hsiao KK. 1994. Human prion diseases. Annals of Neurology 35(4):385-395.

Telling GC, Scott M, Hsiao KK, et al. 1994. Transmission of CJD from humans to transgenic mice expressing chimeric human-mouse prion protein. Proceedings of the National Academy of Sciences of the USA 91(21):9936-9940.

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

Brown P, Kaur P, Sulima MP, et al. 1993. Real and imagined clinicopathological limits of prion dementia. Lancet 341(127-129.

Davies PTG, Jahfar S, Ferguson IT, et al. 1993. Creutzfeldt-Jakob disease in individual occupationally exposed to BSE. Lancet 342(8872):680.

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.

Duchen LW, Poulter M, Harding AE. 1993. Dementia associated with a 216 base pair insertion in the prion protein gene. Clinical and neuropathological features. Brain 116(3):555-567.

Price DL, Borchelt DR, Sisodia SS. 1993. Alzheimer disease and the prion disorders amyloid beta-protein and prion protein amyloidoses [published erratum appears in the Proceedings of the National Academy of Sciences, USA 90(19):9233]. Proceedings of the National Academy of Sciences, USA 90(14):6381-6384.

Ridley RM, Baker HF. 1993. Genetics of human prion disease. Developmental Biology Standards 80:15-23.

1991

Baker HF, Ridley RM. 1991. Human spongiform encephalopathies. Chemistry and Industry (London) 5:166-168.

Brown P. 1991. The clinical epidemiology of CJD in the context of BSE. In: Sub-acute spongiform encephalopathies. Proceedings of a seminar in the CEC Agricultural Research Program, held in Brussels, November 1990 [Edited by Bradley R, Savey M, Marchant B] Dordrecht, Netherlands. Kluwer Academic Publishers, pp. 195-202.

Hsiao K, Meiner Z, Kahana E, et al. 1991. Mutation of the prion protein in Libyan jews with Creutzfeldt-Jakob disease. New England Journal of Medicine 324(16):1091-1097.

Will RG. 1991. The epidemiological surveillance of Creutzfeldt-Jakob disease in the United Kingdom. European Journal of Epidemiology 7:460-465.

1990

Baker HF, Duchen LW, Jacobs JM, et al. 1990. Spongiform encephalopathy transmitted experimentally from Creutzfeldt-Jakob and familial Gerstmann-Straussler-Scheinker diseases. Brain 113:1891-1909.

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.

Beardsley T. 1990. Oravske Kuru. A human dementia raises the stakes in mad cow disease. Scientific American 263(2):24-26.

A discussion of the high rate of CJD in Slovakia.

Human spongiform encephalopathy. 1990. State Veterinary Journal 44(124):19-30.

Kitamoto T. Tateishi J, Sawa H, et al. 1989. Positive transmission of Creutzfeldt-Jakob disease verified by murine kuru plaques. Laboratory Investigations 60(4):507-512.

Narang HK, Perry RH. 1990. High incidence of Creutzfeldt-Jakob disease in Northern England. [Abstract] Neuropathology and Applied Neurobiology 16(3):259.

1989

Kim JH, Manuelidis EE. 1989. Neuronal alterations in experimental Creutzfeldt-Jakob disease: a Golgi study. Journal of Neurological Sciences 89(1):93-101.

Kitamoto T, Mohri S, Tateishi J. 1989. Organ distribution of proteinase-resistant prion protein in humans and mice with Creutzfeldt-Jakob disease. Journal of General Virology 70(12):3371-3379.

Mohri S, Hamada C, Kumanishi T, et al. 1989. A Creutzfeldt-Jakob disease agent (Echigo-1 strain) recovered from brain tissue showing the 'panencephalopathic type' disease. Neurology 39(10):1337-1342.

1988

Humphery-Smith I, Chastel C. 1988. Creutzfeldt-Jakob disease, spiroplasmas, and crystalline artifacts [Correspondence]. Lancet (British Edition) 2(8621):1199.

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.

Kim JH, Lach B, Manuelidis EE. 1988. Creutzfeldt-Jakob disease with intranuclear vacuolar inclusions: a biopsy case of negative light microscopic findings and successful animal transmission. Acta Neuropathology 76(4):422-426.

Pocchiari M, Macchi G, Peano S, et al. 1988. Can potential hazard of Creutzfeldt-Jakob disease infectivity be reduced in the production of human Growth Hormone? Inactivation experiments with the 263K strain of scrapie. Archives of Virology 98(1/2):131-135.

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

Brown P, Cathala F, Raubertas RF, et al. 1987. The epidemiology of Creutzfeldt-Jakob disease: conclusion of a 15-year investigation in France and review of world literature. Neurology 37(6):895-904.

Gourmelon P, Amyx HL, Baron H, et al. 1987. Sleep abnormalities with REM disorder in experimental Creutzfeldt-Jakob disease in cats: a new pathological feature. Brain Research 411(2):391-396.

Masters CL. 1987. Epidemiology of Creutzfeldt-Jakob disease: studies on the natural mechanisms of transmission. In: Fields Virology [Edited by Fields BN, Knipe DM] New York, Raven Press, pp 511-534.

1986

Davanipour Z, Alter M, Sobel E. 1986. Creutzfeldt-Jakob disease. Neurologic Clinics 4:415-426.

Will RG, Matthews WB, Smith PG, et al. 1986. A retrospective study of Creutzfeldt-Jakob disease in England and Wales 1970-1979 II: epidemiology. Journal of Neurosurgery and Psychiatry 49:749-755.

1985

Davanipour Z, Alter M, Sobel E, et al. 1985. A case-control study of Creutzfeldt-Jakob disease: dietary risk factors. American Journal of Epidemiology 122:443-451.

1984

Davanipour Z. 1984. A case control study of Creutzfeldt-Jakob disease and evaluation of a zoonotic hypothesis. Dissertation Abstracts International, B 45(5):1437-1438.

Kamin M, Patten BM. 1984. Creutzfeldt-Jakob disease. Possible transmission to humans by consumption of wild animal brains. American Journal of Medicine 76(1):142-145.

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

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1983

Singhal BS, Dastur DK. 1983. Creutzfeldt-Jakob disease in Western India. Neuroepidemiology 93(2):100.

1981

Matthews WB, Will RG. 1981. Creutzfeldt-Jakob disease in a lifelong vegetarian. Lancet 2:937.

1980

Brown P. 1980. An epidemiologic critique of Creutzfeldt-Jakob disease. Epidemiologic Reviews 2:113-135.

1979

Masters CL, Harris JO, Gajdusek DC, et al. 1979. Creutzfeldt-Jakob disease: patterns of worldwide occurrence and the significance of familial and sporadic clustering. Annals of Neurology 5:177-188.

1977

Gajdusek DC. 1977. Unconventional viruses and the origin and disappearance of kuru. Science 197:943-960.

1973

Bobowick AR, Brody JA, Matthews MR, et al. 1973. Creutzfeldt-Jakob disease: A case-control study. American Journal of Epidemiology 98:381-394.

Transmissible Mink Encephalopathy

1995

Robinson MM, Hadlow WJ, Knowles DP. et al. 1995. Experimental infection of cattle with the agents of transmissible mink encephalopathy and scrapie. Journal of Comparative Pathology 113(3):241-251.

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

Bartz JC, McKenzie DI, Bessen RA, et al. 1994. Transmissible mink encephalopathy species barrier effect between ferret and mink: PrP gene and protein analysis. Journal of General Virology 75(11):2947-2953.

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.

Marsh RF, Bessen RA. 1994. Physicochemical and biological characterizations of distinct strains of the TME agent.

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.

Robinson MM, Hadlow WJ, Huff TP, et al. 1994. Experimental infection of mink with bovine spongiform encephalopathy. Journal of General Virology 75(9):2151-2155.

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

Bessen RA, Marsh RF. 1992. Identification of two biologically distinct strains of TME in hamsters. Journal of General Virology 73(2):329-334.

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.

Kretzschmar HA, Neumann M, Reithmuller G, et al. 1992. Molecular cloning of a mink prion protein gene. Journal of General Virology 73(1):2757-2761.

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).

Marsh RF, Hadlow WJ. 1992. Transmissible mink encephalopathy. Revue Scientifique et Technique - Office International des Epizooties 11(2):539-550.

1991

Bridges V, Bleem A, Walker K. 1991. Risk of transmissible mink encephalopathy in the US. USDA:APHIS, Centers for Epidemiology and Animal Health, Ft. Collins, CO.

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.

Marsh RF, Bessen RA, Lehmann S, et al. 1991. Epidemiological and experimental studies on a new incident of transmissible mink encephalopathy. Journal of General Virology 72(3):590-594.

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

Hadlow WJ, Race RE, Kennedy RC. 1987. Temporal distribution of transmissible mink encephalopathy virus in mink inoculated subcutaneously. Journal of Virology 61:3235-3240.

Hadlow WJ, Race RE, Kennedy RC. 1987. Experimental infection of sheep and goats with transmissible mink encephalopathy virus. Canadian Journal of Veterinary Research 51:135-144.

1970

Barlow RM, Rennie JC. 1970. Transmission experiments with a scrapie-like encephalopathy of mink. Journal of Comparative Pathology 80:75-79.

1968

Hadlow WJ, Karstad L. 1968. Transmissible encephalopathy of mink in Ontario. Canadian Veterinary Journal 99(8):193-196.

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

1965

Burger D, Hartsough GR. 1965. Encephalopathy of mink. II. Experimental and natural transmission. Journal of infectious diseases 115:393-399.

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.

Hartsough GR, Burger D. 1965. Encephalopathy of mink. I. Epizootiologic and clinical observations. Journal of Infectious Diseases 115:387-392.

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

Foster JD, Hunter N, Williams A, et al. 1996. Observations on the transmission of scrapie in experiments using embryo transfer. Veterinary Record 138:559-562.

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.

Lasmezas CI, Cesbron JY, Deslys JP, et al. 1996. Immune system-dependent and -independent replication of the scrapie agent. Journal of Virology 70(2):1292-1295.

Wisniewski HM, Sigurdarson S, Rubenstein R, et al. 1996. Mites as vectors for scrapie. The Lancet 347:1114.

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

Goldmann W, Hunter N, Smith G, et al. 1994. PrP genotype and agent effects in scrapie: change in allelic interaction with different isolates of agent in sheep, a natural host of scrapie. Journal of General Virology 75(5):989-995.

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.

Harpster DE. 1994. Update of the voluntary scrapie flock certification program. Proceedings of the United States Animal Health Association 98:484-485.

Lafrancois T, Fages C, Brugere-Picoux J, et al. 1994. Astroglial reactivity in natural scrapie of sheep. Microbial Pathogenesis 17(5):283-289.

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.

Onodera T, Hayashi T. 1994. Diversity of clinical signs in natural scrapie cases occurring in Japan. Japan Agricultural Research Quarterly 28(1):59-61.

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

Bendheim PE. 1993. Natural scrapie in sheep - a review. Israel Journal of Veterinary Medicine 48(3):96-109.

1992

Andrews AH, Lavern R, Matthews JG. 1992. Clinical observations on four cases of scrapie in goats. Veterinary Record 130:101.

Detwiler LA. 1992. Scrapie. Revue Scientifique et Technique - Office International des Epizooties 11(2):491-537.

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.

Foster JD, McKelvey WAC, Mylne MJA, et al. 1992. Studies on maternal transmission of scrapie in sheep by embryo transfer. Veterinary Record 130:341-343.

Wood JLN, Done SH. 1992. Natural scrapie in goats: neuropathology. Veterinary Record 131(5):93-96.

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

Wood JLN, Lund LJ, Done SH. 1992. The natural occurrence of scrapie in moufflon. Veterinary Record 130:25-27.

Wooldridge MJA, Hoinville LJ, Wilesmith JW. 1992. A scrapie survey by postal questionnaire: aims, problems and results. In: Proceedings of the Society of Veterinary Epidemiology and Preventive Medicine [Edited by Thrusfield MV]. Edinburgh.

1991

Arya SC. 1991. Spread of 'unconventional viruses' through sheep-brain rabies vaccines. Vaccine 9(1):70.

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.

Brown P, Gajdusek DC. 1991. Survival of scrapie virus after 3 years interment. Lancet (British Edition) 337(8736):169-270.

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.

Hunter N, Hope J. 1991. The genetics of scrapie susceptibility in sheep (and its implications for BSE). Breeding for disease resistance in farm animals [edited by Owen JB, Axford RFE]. Wallingford, UK, Cab International, pp. 329-344.

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.

Perrin GG, Perrin GJ, Benoit C. 1991. Detection of scrapie-associated fibrils in goats. Veterinary Record 129(19):432.

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.

Sigurdarson S. 1991. Epidemiology of scrapie in Iceland and experience with control measures. In: Sub-acute spongiform encephalopathies. Proceedings of a seminar in the CEC Agricultural Research Program, held in Brussels, November 1990 [Edited by Bradley R, Savey M, Marchant B] Dordrecht, Netherlands. Kluwer Academic Publishers, pp. 233-242.

Stack MJ, Scott AC, Done SH, et al. 1991. Natural scrapie: detection of fibrils in extracts from the central nervous system of sheep. Veterinary Record 128(23):539-540.

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

Bundza A. 1990. Experimental scrapie in white Swiss mice. Journal of Veterinary Medicine, Series B 37(10):777-780.

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.

Cathala F. 1990. Scrapie in France. Journal of the American Veterinary Medical Association 196(10):1680.

Dinter Z, Morein B (Editors). 1990. Virus infections of ruminants. Amsterdam, Netherlands, Elsevier Science Publishers.

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

Hadlow WJ. 1990. An overview of scrapie in the United States. Journal of the American Veterinary Medical Association 196(10):1676-1677.

Kimberlin RH. 1990. Unconventional 'slow' viruses. In: Topley and Wilson's Principles of Bacteriology, Virology and Immunity. [Edited by Parker MT,Collier LH]. Edward Arnold, Publisher, London, 4:671-693.

Martin WB. 1990. Prevalence of scrapie in British flocks. Veterinary Record 127:409.

Morgan KL, Nicholas K, Glover MJ, et al. 1990. A questionnaire survey of the prevalence of scrapie in sheep in Britain. Veterinary Record 127(15):373-376.

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.

Pattison, I. 1990. Scrapie agent in muscle. [Correspondence] Veterinary Record 126(3):68.

Pocchiari M. 1990. Methods for inactivating experimentally induced scrapie in hamster tissues. Journal of the American Veterinary Medical Association 196(10):1683-1684.

1989

Carp RI, Yong SK, Kascsak RJ, et al. 1989. Classic genetics of scapie. In: Alzheimer's Disease and Related Disorders. Alan R. Liss, Inc., pp 567-582.

Chatelain J, Dautheville-Guibal C. 1989. Ovine scrapie: follow-up of sheep belonging to an endemic scrapie-infected flock. European Journal of Epidemiology 5(1):113-116.

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.

Foster JD, Dickinson AG. 1989. Age at death from natural scrapie in a flock of Suffolk sheep. Veterinary Record 125:415-417.

Kimberlin RH. 1989. Introduction to scrapie and perspectives on current scrapie research. In: Alzheimer's Disease and Related Disorders. Alan R. Liss, Inc, pp 559-566.

1988

Dickinson AG, Taylor DM. 1988. Options for the control of scrapie in sheep and its counterpart in cattle. Proceedings, 3rd World Congress on Sheep and Beef Cattle Breeding, Paris, France 1:553-564.

Foote WC, Pitcher JR. 1988. Approaches to controlling scrapie in the United States. Proceedings of the 92nd Annual Meeting of the United States Animal Health Association, pp 402-412.

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

Pattison IH. 1988. Fifty years with scrapie: A personal reminiscence. Veterinary Record 123:661-666.

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

1987

Bruce ME, Dickinson AG. 1987. Biological evidence that scrapie agent has an independent genome. Journal of General Virology 68:79-89.

Gibson PH, Somerville RA, Fraser H, et al. 1987. Scrapie associated fibrils in the diagnosis of scrapie in sheep. Veterinary Record 120:125-127.

Kimberlin RH, Cole S, Walker CA. 1987. Temporary and permanent modifications to a single strain of mouse scrapie on transmission to rats and hamsters. Journal of General Virology 68:1875-1881.

Pocchiari M, Schmittinger S, Masullo C. 1987. Amphotericin B delays the incubation period of scrapie in intracerebrally inoculated hamsters. Journal of General Virology 68:219-223.

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Borras T, Gibbs CJ Jr. 1986. Molecular hybridization studies with scrapie brain nucleic acids. I. Search for specific DNA sequences. Archives of Virology 88(1):67-78.

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

Chatelain J, Baron H, Baille V, et al. 1986. Study of endemic scrapie in a flock of 'Ile-de-France' sheep. European Journal of Epidemiology 2:31-35.

Foote WC, Call JW, Bunch TD, et al. 1986. Embryo transfer in the control of transmission of scrapie in sheep and goats. Proceedings of the US Animal Health Association 90:413-416.

1985

Davies DC, Kimberlin RH. 1985. Selection of Swaledale sheep of reduced susceptibility to experimental scrapie. Veterinary Record 116:211-214.

Prusiner SB, Cochran SP, Alpers MP. 1985. Transmission of scrapie in hamsters. Journal of Infectious Diseases 152(5):971-978.

Hamsters developed scrapie 100-160 days after eating either scrapie-infected hamsters ir 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

Chatelain J, Delasnerie-Laupretre N, Cathala F. 1983. Scrapie in France: some possible predisposing factors in the naturally-acquired disease of sheep. Veterinary Microbiology 8:511-515.

Fraser H. 1983. A survey of primary transmission of Icelandic scrapie (Rida) in mice. In: Virus non-conventionnels et affections du systeme nerveux central [Edited by Court LA, Cathala F]. Masson, Paris, pp 34-36.

Parry HB. 1983. Scrapie Disease in Sheep. [edited by Oppenheimer DR]. Academic Press, New York, pp 31-51.

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Carp RI. 1982. Transmission of scrapie by oral route: effect of gingival scarification. Lancet 1:170-171.

Hadlow WJ, Kennedy RC, Race RE. 1982. Natural infection of Suffolk sheep with scrapie virus. Journal of Infectious Diseases 146:657-664.

1981

Kimberlin RH. 1981. Scrapie. British Veterinary Journal 137:1-5-112.

1980

Hadlow WJ, Kennedy RC, Race RE, et al. 1980. Virologic and neurohistologic findings in dairy goats affected with natural scrapie. Veterinary Pathology 17:187-199.

1979

Dickinson AG, Fraser H. 1979. An assessment of the genetics of scrapie in sheep and mice. In: Slow Transmissible Diseases of the Nervous System, Vol I. [edited by Prusiner SB, Hadlow WJ]. Academic Press, New York, pp 367-385.

Hourrigan JL, Klingsporn A, Clark WW, et al. 1979. Epidemiology of scrapie in the United States. In: Slow Transmissible Diseases of the Nervous System, Vol I. [edited by Prusiner SB, Hadlow WJ]. Academic Press, New York, pp 331-356.

Kimberlin RH. 1979. An assessment of genetical methods in the control of scrapie. Livestock Production Science 6:233-242.

1977

Kimberlin RH, Walker CA. 1977. Characteristics of a short incubation model of scrapie in the golden hamster. Journal of General Virology 34:295-304.

1976

Dickinson AG. 1976. Scrapie in sheep and goats. In: Slow Virus Diseases of Animals and Man. [Edited by Kimberlin RH]. North-Holland, Amsterdam, pp 209-241.

Fraser H. 1976. The pathology of natural and experimental scrapie. In: Slow Virus Diseases of Animals and Man. [Edited by Kimberlin RH]. North-Holland, Amsterdam, pp 267-305.

1975

Nussbaum RE, Henderson WM, Pattison IH, et al. 1975. The establishment of sheep flocks of predictable susceptibility to experimental scrapie. Research in Veterinary Science 18:49-58.

1974

Dickinson AG, Stamp JT, Renwick CC. 1974. Maternal and lateral transmission of scrapie in sheep. Journal of Comparative Pathology 84:19-25.

Pattison IH, Hoare MN, Jebbett JN, et al. 1974. Further observations on the production of scrapie in sheep by oral dosing with foetal membranes from scrapie-affected sheep. British Veterinary Journal 130:65-67.

1972

Pattison IH, Hoare MN, Jebbett JN, et al. 1972. Spread of scrapie to sheep and goats by oral dosing with foetal membranes from scrapie-affected sheep. Veterinary Record 90:465-468.

1969

Dickinson AG, Stamp JT. 1969. Experimental scrapie in Cheviot and Suffolk sheep. Journal of Comparative Pathology 79:23-26.

1968

Brotherston JG, Renwick CC, Stamp JT, et al. 1968. Spread of scrapie by contact to goats and sheep. Journal of Comparative Pathology 78:9-17.

1965

Dickinson AG, Young GB, Stamp JT, et al. 1965. An analysis of natural scrapie in Suffolk sheep. Heredity 20:485-503.

Pattison IH. 1965. Scrapie in the welsh mountain breed of sheep and its experimental transmission to goats. Veterinary Record 77:1388-1390.

1964

Dickinson AG, Young GB, Renwick CC. 1964. Scrapie: experiments involving maternal transmision in sheep. In: Report of Scrapie Seminar, 27-30 January, 1964. (ARS 91-53). Washington, DC, pp 244-247.

Parry, HB. 1964. Natural scrapie in sheep. I. Clinical manifestation and general incidence, treatment, and related syndromes. In: Report of Scrapie Seminar, 27-30 January, 1964. (ARS 91-53). Washington, DC, pp 95-97.

Young GB, Stamp JT, Renwick CC, et al. 1964. Field observations on scrapie incidence. In: Report of Scrapie Seminar, 27-30 January, 1964. (ARS 91-53). Washington, DC, pp 199-206.

1963

Chandler RL. 1963. Experimental scrapie in the mouse. Research in Veterinary Science 4:276.

1962

Parry HB. 1962. Scrapie: a transmissible and hereditary disease of sheep. Heredity 17:75-105.

1961

Chandler RL. 1961. Encephalopathy in mice produced by inoculation with scrapie brain material. Lancet 1:1378-1379.

Pattison IH, Millson GC. 1961. Scrapie produced experimentally in goats with special reference to the clinical syndrome. Journal of Comparative Pathology 71:101-108.

1959

Pattison IH, Gordon WS, Millson GC. 1959. Experimental production of scrapie in goats. Journal of Comparative Pathology 69:300-314.

SE's in Other Species

1996

Bons N, Mestre-Frances N, Charnay Y, et al. 1996. Spontaneous spongiform encephalopathy in a young adult rhesus monkey. Lancet 348:55.

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

Bratberg B, Ueland K, Wells GAH. 1995. Feline spongiform encephalopathy in a cat in Norway. Veterinary Record 136(17):444.

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

Fraser H, Pearson GR, McConnell I, et al. 1994. Transmission of feline spongiform encephalopathy to mice. Veterinary Record 134(7):449.

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.

Kirkwood JK, Cunningham AA. 1994. Epidemiological observations on spongiform encephalopathies in captive wild animals in the British Isles. Veterinary Record 135(13):296-303.

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.

Kirkwood JK, Cunningham AA, Austin AR, et al. 1994. Spongiform encephalopathy in a greater kudu (Tragelaphus strepsiceros) introduced into an affected group. Veterinary Record 134(7):167-168.

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

Cunningham AA, Wells, GAH, Scott, et al. 1993. Transmissible spongiform encephalopathy in greater kudu (Tragelaphus strepsiceros). Veterinary Record 132(3):68.

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.

Guiroy DC, Williams ES, Song KJ, et al. 1993. Fibrils in brains of Rocky Mountain Elk with chronic wasting disease contain scrapie amyloid. Acta Neuropathologica 86(1):77-80.

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.

Guiroy DC, Williams ES, Liberski PP, et al. 1993. Ultrastructural neuropathology of chronic wasting disease in captive mule deer. Acta Neuropathologica 85(4):437-444.

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.

Kirkwood JK, Cunningham AA, Wells GAH, et al. 1993. Spongiform encephalopathy in a herd of greater kudu (Tragelaphus strepsiceros): epidemiological observations. Veterinary Record 133(15):360-364.

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.

Pearson GR, Wyatt JM, Henderson JP, et al. 1993. Feline spongiform encephalopathy: a review. Veterinary Annual 33:1-10.

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.

Williams ES, Young S. 1993. Neuropathology of CWD of mule deer (Odocoileus hemionus) and elk (Cervus elaphus nelsoni). Veterinary Pathology 30(1):36-45.

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.

Wyatt JM, Pearson GR, Gruffydd-Jones TJ. 1993. Feline spongiform encephalopathy. Feline Practice 21(3):7-9.

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.

1992

Kirkwood JK, Wells GAH, Cunningham AA, et al. 1992. Scrapie-like encephalopathy in a greater kudu (Tragelaphus strepsiceros) which had not been fed ruminant-derived protein. Veterinary Record 130(17):365-367.

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.

Peet RL, Curran JM. 1992. Spongiform encephalopathy in an imported cheetah. Australian Veterinary Journal 69(7):171.

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.

Pearson GR, Wyatt JM, Gruffydd-Jones TJ, et al. 1992. Feline spongiform encephalopathy: fibril and PrP studies. Veterinary Record 131(14):307-310.

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.

Williams ES, Young S. 1992. Spongiform encephalopathies in Cervidae. Revue Scientifique et Technique - Office International des Epizooties 11:551-567.

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.

Willoughby K, Kelly DF, Lyon DG, et al. 1992. Spongiform encephalopathy in a captive puma (Felis concolor). Veterinary Record 131(19):431-434.

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

Guiroy DC, Williams ES, Yanagihara R, et al. 1991. Topographic distribution of scrapie amyloid-immunoreactive plaques in chronic wasting disease in captive mule deer (Odocoileus hemionus). Acta Neuropathologica 81(5):475-478.

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.

Pearson GR, Gruffydd-Jones TJ, Wyatt JM, et al. 1991. Feline spongiform encephalopathy [Correspondence]. Veterinary Record 128(22):532.

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.

Schoon HA, Brunckhorst D, Pohlenz J. 1991. Spongiform encephalopathy in an ostrich (Struthio camelus). A case report. Spongiforme Enzephalopathie beim Rothalsstrauss (Struthio camelus). Ein kasuistischer Beitrag. Tierarztliche Praxis 19(3):263-265. In German with English abstract.

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.

Wyatt JM, Pearson GR, Smerdon TN, et al. 1991. Naturally occurring scrapie-like spongiform encephalopathy in five domestic cats. Veterinary Record 129(11):233-236.

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

1990

Aldhous P. 1990. Antelopes die of “mad cow” disease. Nature 344(6263):183.

Aldhous P. 1990. Spongiform encephalopathy found in a cat. Nature 345(6261):194.

Fleetwood AJ, Furley CW. 1990. Spongiform encephalopathy in an eland. [Correspondence]. Veterinary Record 126(16):408-409.

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.

Kirkwood JK, Wells GAH, Wilesmith JW, et al. 1990. Spongiform encephalopathy in an Arabian oryx (Oryx leucoryx) and a greater kudu (Tragelaphus strepsiceros). Veterinary Record 127(17):418-420.

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.

Leggett MM, Dukes J, Pirie HM. 1990. A spongiform encephalopathy in a cat. Veterinary Record 127(24):586-588.

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.

Wyatt JM, Pearson GR, Smerdon T, et al. 1990. Spongiform encephalopathy in a cat. [Correspondence]. Veterinary Record 126(20):513.

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

BSE emergency meeting inspires confidence over US safeguards. Journal of the American Veterinary Medical Association 208(9):1362.

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

Chen SS, Charlton KM, Balachandran AV, et al. 1996. Bovine spongiform encephalopathy identified in a cow imported to Canada from the United Kingdom - a case report. Canadian Veterinary Journal 37:38-40.

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.

Schudel AA, Carrillo BJ, Cane BG. 1996. Risk assessment and surveillance for bovine spongiform encephalopathy (BSE) in Argentina. Preventive Veterinary Medicine 25(3/4):271.

The WHO recommendations on BSE: what they mean to the United States. 1996. Journal of the American Veterinary Medical Association 208(11):1771.

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

USDA:APHIS:VS, Centers for Epidemiology and Animal Health. 1996. Bovine spongiform encephalopathy: implications for the United States - A follow-up. Ft. Collins, CO

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

1995

Guarda F, Fatzer R. 1995. Examinations of brains of slaughter cattle for the occurrence of BSE in Italy with particular reference to nonspecific neuronal vacuoles. Untersuchungen an Gehirnen von

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

Franco DA. 1994. International animal nutrition symposium: updating the use of animal by-products in animal feeds. Proceedings of the International Animal Nutrition Symposium, Utrecht, Netherlands, pp 10-16.

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

Hornlimann B, Guidon D, Griot C. 1994. Evaluation of the risk of the introduction of BSE. Risikoeinschaetzung fuer die Einschleppung von BSE. Deutsche Tierartztliche Wochenschrift 101(7):295-298. (In German)

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.

Kraaden OR, Truyen U, Groschup MH, et al. 1994. Bovine spongiform encephalopathy in Germany. Journal of Veterinary Medicine, Series B 41(4):294-304.

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.

Schudel AA, Carrillo BJ, Gimeno EJ, et al. 1994. Bovine spongiform encephalopathy surveillance in Argentina. Revue Scientifique et Technique - Office International des Epizooties 13(3):801-836.

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.

1993

Bovine spongiform encephalopathy (BSE): A plan for prevention. 1993. The shepherd 38(6):37.

Refers to the US.

Brown DR, Zhang HM, DeNise SK, et al. 1993. Bovine prion gene allele frequencies determined by AMFLP and RFLP analysis. Animal Biotechnology 4(1):47-51.

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.

Carrillo BJ, Bardon JC, Combesies G, et al. 1993. Diagnosis of bovine neurological diseases in the pampeana region in Argentina: observations on aetiology. Diagnostico de neuropathologias en bovinas del area pampeana: observaciones de casuistica. Revista de Medicina Veterinaria (Buenes Aires) 74(5):282-287. (In Spanish)

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.

Canadian beef cow diagnosed with BSE. 1993. Agra Europe (British edition) 1575:N4.

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.

Gill PA, Townsend WL. 1993. Hepatic vasculopathy and encephalopathy in Brahman-type calves. Australian Veterinary Journal 70(2):69.

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.

Johnstone AC. 1993. Hepatic encephalopathy and BSE. Surveillance (Wellington) 20(2):28.

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.

Keeping vigil over US cattle for BSE. 1993. Journal of the American Veterinary Medical Association 203(9):1231.

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

Marsh RF. 1993. Symposium on risk assessment of the possible occurrence of bovine spongiform encephalopathy in the US. Journal of the American Veterinary Medical Association 204(1):70-73.

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.

USDA:APHIS:VS, Centers for Epidemiology and Animal Health. 1993. Bovine spongiform encephalopathy: implications for the United States. Ft. Collins, CO.

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

Brochier B, Vanopdenbosch E, Coppens P, et al. 1992. System for epidemiological surveillance of spongiform encephalopathies in Belgium: first results. Reseau d'epidemiosurveillance des encephalopathies spongiformes en Belgique: premiers resultats. Annales de Medecine Veterinaire 136(4):245-247. (In French)

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.

McKenzie DI, Cowan CM, Marsh RF, et al. 1992. PrP gene variability in the US cattle population. Animal Biotechnology 3(2):309-315.

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

Miller LD, Davis AJ, Jenny AL. 1992. Surveillance for lesions of BSE in US cattle. Journal of Veterinary Diagnostic Investigation 4(3):338-339.

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

Arya SC. 1991. Acquisition of SE's in India through sheep-brain rabies vaccination [Correspondence] National Medical Journal of India 4(6):311-312.

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.

Cachin M, Vandevelde M, Zurbriggen A. 1991. Spongiform encephalopathy in a cow in Switzerland. Ein Fall von spongiformer Enzephalopathie ("Rinderwahnsinn") bei einer Kuh in der Schweiz. Schweizer Archiv fur Tierheilkunde 133(2):53-57. In German with English abstract.

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.

Hornlimann B. 1991. Bovine spongiform encephalopathy (Mad cow disease): Present knowledge and control measures in Switzerland. Disease Control Service, Swiss Federal Veterinary Service, Liebefeld-Bern, Switzerland.

Muirhead S. 1991. Qualitative assessment finds BSE less likely to occur in the US than in the UK. Feedstuffs 63(13):10.

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.

USDA:APHIS:VS, Centers for Epidemiology and Animal Health. 1991. Quantitative risk assessment of BSE in the United States. Fort Collins, CO.

USDA:APHIS:VS, Centers for Epidemiology and Animal Health. 1991. Qualitative analysis of BSE risk factors in United States. Fort Collins, CO.

Walker KD, Hueston WD, Hurd HS, et al. 1991. Comparison of bovine spongiform encephalopathy risk factors in the United States and Great Britain. Journal of the American Veterinary Medical Association 199(11):1554-1561.

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

Asher DM. 1990. Recommendations concerning the risk of bovine spongiform encephalopathy in the United States. Journal of the American Veterinary Medical Association 196(10):1687.

Carolan DJP, Wells GAH, Wilesmith JW. 1990. BSE in Oman. [Correspondence]. Veterinary Record 126(4):92.

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.

Cullen RG. 1990. Bovine spongiform encephalopathy (BSE) in Ireland. Bulletin of the Office International des Epizooties 102(4):225-226.

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.

King LJ. 1990. US Department of Agriculture's response to the bovine spongiform encephalopathy situation. Proceedings - Annual Meeting of the United States Animal Health Association 94:252-255.

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

Basset H, Sheridan C. 1989. Case of BSE in the Irish Republic. Veterinary Record 124(6):151.

Johnstone AC, McKenzie JS. 1989. Bovine spongiform encephalopathy: A new scrapie-like disease of cattle. Surveillance (Wellington) 16(3):25-26.

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.

Julian AF, Moore DJ. 1989. Suspected case of bovine spongiform encephalopathy (final diagnosis: Hepatic encephalopathy). Surveillance (Wellington) 16(4):6.

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