Bovine spongiform encephalopathy (BSE) and Creutzfeldt-Jakob disease (CJD) : recent developments minutes of evidence / Agriculture and Health Committees.

HOUSE OF COMMONS SESSION 1995–96

AGRICULTURE AND HEALTH COMMITTEES

MEETING CONCURRENTLY, PURSUANT TO PARAGRAPH (4)(e) OF STANDING ORDER No. 130

(SELECT COMMITTEES RELATED TO GOVERNMENT DEPARTMENTS)

BOVINE SPONGIFORM ENCEPHALOPATHY (BSE) AND CREUTZFELDT-JAKOB DISEASE (CJD): RECENT DEVELOPMENTS

Minutes of Evidence

Ordered by The House of Commons *to be printed*
27 March, 17 and 18 April and 20 May 1996

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LIST OF WITNESSES

Page

Wednesday, 27 March
(Morning Sitting)

DEPARTMENT OF HEALTH

Rt Hon S Dorrell, MP and Sir K Calman, KCB 6

MINISTRY OF AGRICULTURE, FISHERIES AND FOOD

Rt Hon D Hogg, QC, MP, Mr K Meldrum and Mr T Eddy 6

SPONGIFORM ENCEPHALOPATHY ADVISORY COMMITTEE

Professor J Pattison, Dr R Kimberlin and Dr R Will 6

Wednesday 27 March
(Afternoon Sitting)

DEPARTMENT OF HEALTH

Rt Hon S Dorrell, MP and Sir K Calman, KCB 24

MINISTRY OF AGRICULTURE, FISHERIES AND FOOD

Rt Hon D Hogg, QC, MP, Mr K Meldrum and Mr T Eddy 24

SPONGIFORM ENCEPHALOPATHY ADVISORY COMMITTEE

Professor J Pattison, Dr R Kimberlin and Dr R Will 24

Wednesday 17 April

DEPARTMENT OF HEALTH

Sir K Calman KCB 68

MINISTRY OF AGRICULTURE, FISHERIES AND FOOD

Mr K Meldrum CB, Dr S Dealler and Dr H Narang 68

SPONGIFORM ENCEPHALOPATHY ADVISORY COMMITTEE

Professor J Pattison 68

INSTITUTE FOR ANIMAL HEALTH

Professor J Bourne 68

Wednesday 18 April

Professor T Lang 119

MEAT HYGIENE SERVICE

Mr P Soul 119

FEDERATION OF FRESH MEAT WHOLESALERS

Mr A Bowes, Mr J Baker and Mr R Cracknell 119

LIST OF MEMORANDA INCLUDED IN THE MINUTES OF EVIDENCE

No		*Page*
1.	Memorandum by the Ministry of Agriculture, Fisheries and Food (T1/BSE1) ...	1
2.	Joint memorandum by the Ministry of Agriculture, Fisheries and Food, and the Department of Health (T5/BSE11)	44
3.	Memorandum by Dr Stephen Dealler (T7/BSE4)	50
4.	Extract from a memorandum by Dr Harash Narang (T8/BSE5)	65
5.	Extract from a memorandum by Ministry of Agriculture, Fisheries and Food (Letter from Mr R Bradley, Central Veterinary Laboratory (T9/BSE1A)	90
6.	Letter from Dr Stephen Dealler (T10/BSE4A)	90
7.	Supplementary memorandum by Dr Stephen Dealler (T11/BSE4B)	91
8.	Supplementary memorandum by Dr Harash Narang (T12/BSE5A)	99
9.	Further supplementary memorandum by Dr Harash Narang (T13/BSE5B)	101
10.	Extract from supplementary memorandum by the Department of Health (T6/BSE10)	109
11.	Memorandum by the Federation of Fresh Meat Wholesalers (T2/BSE6)	117
12.	Memorandum by the Meat Hygiene Service (T3/BSE8)	135
13.	Memorandum by Professor Tim Lang, Centre for Food Policy, Thames Valley University (T4/BSE9)	139

MINUTES OF EVIDENCE

TAKEN BEFORE THE AGRICULTURE AND HEALTH COMMITTEES
MEETING CONCURRENTLY, PURSUANT TO PARAGRAPH (4)(e)
OF STANDING ORDER NO. 130
(SELECT COMMITTEES RELATED TO GOVERNMENT DEPARTMENTS)

WEDNESDAY 27 MARCH 1996

Members present:

AGRICULTURE COMMITTEE	*HEALTH COMMITTEE*
Mr Richard Alexander	Mr John Austin-Walker
Mr D N Campbell-Savours	Mr Hugh Bayley
Mr Robin Corbett	Mr David Congdon
Mr Ieuan Wyn Jones	Alice Mahon
Mr Martyn Jones	Mr John Marshall
Mr Edward Leigh	Mrs Marion Roe
Sir Roger Moate	Mr Roger Sims
Mr Colin Pickthall	Rev Martin Smyth
Mr William Powell	Mr Richard Spring
Sir Jerry Wiggin	Mr John Whittingdale
Mrs Ann Winterton	Audrey Wise

Sir Jerry Wiggin was called to the Chair.

Memorandum by the Ministry of Agriculture, Fisheries and Food

BOVINE SPONGIFORM ENCEPHALOPATHY (T1/BSE1)

Chronology of events

Date	*Event*
November 1986	Disease identified by Central Veterinary Laboratory following study of affected cow referred to Weybridge for investigation and post mortem. Transmission experiments needed which required fresh material from animals thought to be suffering from the same problem.
April 1987	Initial epidemiological studies started. Objective was to obtain detailed data from a case study of 200 herds.
5 June 1987	CVO informs Ministers about new disease. Transmission experiment then put under way. Not known if disease was transmissible at that stage. Normal time for disease to develop in mice proved to be about 10 months. Results available September 1988 and published October 1988 in Veterinary Record.
15 December 1987	Initial epidemiology studies completed. Concluded ruminant derived meat and bone meal was only viable hypothesis for cause of BSE.
January–March 1988	Double checking of feeding histories of affected animals initiated; request sent to compounders for details of inclusion of meat and bone meal in rations fed.
April–May 1988	Responses from compounders further substantiated hypothesis for cause of BSE.
21 April 1988	Southwood Working Party announced. Government indicated that they would legislate to make BSE notifiable and to ban feeding of rations that contained protein derived from ruminants.
June 1988	Discussions with major compounders on timing of ruminant feed ban.

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Date	*Event*
14 June 1988	The Bovine Spongiform Encephalopathy Order 1988 (SI 1988 No 1039) was made, article 7 came into effect on 18 July and the remainder on 21 June.
20 June 1988	Southwood Working Party held first meeting and decided to issue interim advice immediately.
21 June 1988	Provisions of BSE Order 1988 came into effect with the exception of article 7. Made BSE notifiable and provided for isolation of BSE suspects when calving.
22 June 1988	Interim advice received from Southwood—destroy affected cattle; proposed feed ban welcomed.
7 July 1988	Decision to introduce slaughter policy announced.
18 July 1988	Ruminant feed ban comes into force (included in BSE Order 1988, but implementation delayed until 18 July). Ban to apply until 31 December 1988 while a review of rendering processes was conducted.
8 August 1988	Bovine Spongiform Encephalopathy (Amendment) Order 1988 (SI 1988 No 1345) and the Bovine Spongiform Encephalopathy Compensation Order (SI 1988 No 1346) came into effect. Provided for slaughter policy and compensation to be paid at 50 per cent value for confirmed cases, 100 per cent for negative; both subject to a ceiling.
October 1988	Transmission to mice following intra cerebral inoculation of BSE brain tissue reported in Veterinary Record.
15 November 1988	Further interim advice received from Southwood—extend feed ban and destroy milk from infected cattle.
28 November 1988	Disease made notifiable and slaughter policy introduced in Northern Ireland by the Bovine Spongiform Encephalopathy Order (Northern Ireland) 1988 (SI 1988 No 422) and the Diseases of Animals (Modification) (No 2) Order (Northern Ireland) 1988 (SI 1988 No 421).
30 November 1988	Decision announced to prolong feed ban and prohibit the use of milk from suspect animals for any purpose other than feeding to the cow's own calf.
30 December 1988	The Bovine Spongiform Encephalopathy (No 2) Order 1988 (SI 1988 No 2299) came into force to prolong feed ban and to prohibit use of milk from suspect cattle for any purpose other than feeding to cow's own calf.
11 January 1989	Diseases of Animals (Feeding Stuffs) Order (Northern Ireland) 1989 (SI 1989 No 8) banned the use of animal protein in ruminant feed in Northern Ireland.
9 February 1989	Southwood Report received by Ministers.
27 February 1989	Southwood Report published and Government response announced (all recommendations have or will be introduced).
27 February 1989	Establishment of Tyrrell Committee on research announced (one of Southwood's recommendations).
10 June 1989	Tyrrell Report received by Government.
13 June 1989	Decision to introduce offals ban announced. Ban is Government initiative not a recommendation of Southwood, it was only concerned with baby food.
28 July 1989	EC ban on export of cattle born before 18 July 1988 and offspring of affected or suspect animals. (Decision 89/469/EEC.)
13 November 1989	The Bovine Offal (Prohibition) Regulations 1989 (S.I. 1989 No. 2061) Regulation came into force in England and Wales which banned the use of certain specified bovine offals (SBO) (following consultation—a legal requirement—and consideration by top experts).
9 January 1990	Publication of Tyrrell Report and Government response (all top and medium priority work recommended either under way or would be undertaken). Publication delayed so could ensure finance for R&D was in place. Research itself was *not* delayed.
30 January 1990	The Bovine Offal (Prohibition) (Scotland) Regulations 1990 (S.I. 1990 No. 112) and the Bovine Offal (Prohibition) Regulations (Northern Ireland) 1990 (S.I. 1990 No. 30) introduced the SBO ban in Scotland and Northern Ireland following additional consultation.

Date	*Event*
31 January 1990	Announcement that five antelopes have succumbed to a spongiform encephalopathy (greater kudu, arabian oryx, eland, nyala and gemsbok. The last two were referred to in Southwood report).
3 February 1990	Cattle to cattle transmission following intra-cerebral and intra-venous inoculation of BSE brain tissue and into mice via the oral route reported in Veterinary Record, following press briefing on 2 February).
14 February 1990	The Bovine Spongiform Encephalopathy Compensation Order (S.I. 1990 No. 222) introduced full compensation up to a ceiling. There was no sudden surge of cases indicating that farmers had not been reporting. Pattern of reporting was unaffected.
1 March 1990	EC restricts exports of cattle to those under six months which are slaughtered before that age (Decision 90/59/EEC made 7 February).
30 March 1990	Administrative ban on export of specified offal and certain glands and organs (for uses other than human consumption) to other Member States.
1 April 1990	Disease made notifiable to European Commission (Decision 90/134/EEC made 6 March).
3 April 1990	Announcement about the establishment of permanent advisory group on spongiform encephalopathies under Chairmanship of Dr David Tyrell.
9 April 1990	EC Decision to ban exports of SBO and other tissues (90/200/EEC)—formalises administrative ban imposed on 30 March.
11 April 1990	Humberside County Council withdraw British beef from school meals.
10 May 1990	Announcement about cat with a spongiform encephalopathy.
17 May 1990	Announcement that decisions about breeding from offspring of affected cows should be left to individual farmers and their veterinary advisors.
8 June 1990	Council of Ministers agree arrangements for trade in beef and calves from UK (Decision 90/261/EEC made 8 June).
12 July 1990	Publication of Tyrrell Committee's detailed reasoning on why no need to give official advice on breeding from offspring of BSE cases.
12 July 1990	Report of Agriculture Committee published.
23 July 1990	UK progress report to OIE meeting.
24 September 1990	Announcement on improved record keeping in cattle herds.
24 September 1990	Laboratory transmission of BSE to a pig announced. Tyrrell Committee advise no implications for human health but, as precaution on animal health, ban specified offals in all animal feed (including pet food).
25 September 1990	The Bovine Spongiform Encephalopathy (No. 2) Amendment Order 1990 (S.I. 1990 No. 1930) extended the ban on the use of specified bovine offals to any animal feed. Exports of such feed also effectively banned to other Member States. (Third country exports banned under DTI legislation on 10 July 1991.)
28-29 September 1990	OIE meeting in Paris; recommendations made regarding trade in cattle, beef, dairy and bovine products and co-ordination of research.
2-5 October 1990	OIE Conference in Sofia (Bulgaria); recommendations made regarding trade, prevention, control and surveillance of BSE, the support of research and the need for further consideration on trade in live animals.
15 October 1990	The Bovine Animals (Identification, Marking and Breeding Records) Order 1990 (S. I. 1990 No. 1867) introduced new record keeping arrangements requiring cattle farmers to maintain breeding records. These and movement records to be retained for 10 years.
21 November 1990	Publication of Government response to Agriculture Committee Report.
27 March 1991	First case announced in BSE offspring born after ruminant feed ban.
May 1991	UK Progress Report to the OIE General Assembly.
10 July 1991	The Export of Goods (Control)(Amendment No. 7) Order 1991 came into force controlling export of SBOs to third countries. (Dept. of Trade & Industry legislation.)
16-20 September 1991	Meeting of OIE International Animal Health Code Commission in Paris.

Date	*Event*
28-30 October 1991	OIE Conference in Tehran.
6 November 1991	The Bovine Spongiform Encephalopathy Order 1991 consolidated existing BSE legislation and introduced new provisions to prevent the use of meat and bone meal produced from SBO's as a fertiliser.
4 March 1992	Results of further experiments on the host range of BSE announced. Also that the Tyrrell Committee had considered the latest BSE research and concluded that the measures at present in place provide adequate safeguards for human and animal health.
May 1992	UK Progress Reports to the OIE General Assembly.
May 1992	OIE General Assembly in Paris agree trading conditions for bovine products from countries affected by BSE.
14 May 1992	EC Commission Decision prohibiting intra community trade in bovine embryos derived from BSE suspect or confirmed dams or dams born after 18/7/88 (Decision 92/290/EEC).
30 June 1992	Publication of the "Interim Report on Research" by the Spongiform Encephalopathy Advisory Committee (Tyrrell).
November 1992	UK Progress Report presented to the EC Standing Veterinary Committee.
24 November 1992	Announced by PQ that details of the total number of cases (by county) would be placed regularly in the library of the House of Commons.
15 December 1992	UK Progress Report placed in the library of the House of Commons.
24 May 1993	UK Progress Report presented to the OIE General Assembly.
27 May 1993	UK Progress Report to the OIE placed in the House of Commons Library.
10 June 1993	UK Progress Report presented to the EC Standing Veterinary Committee (same as OIE Progress Report).
14 July 1993	100,000th confirmed case of BSE in Great Britain announced in response to a Parliamentary Question, as an update to the UK Progress Report to the OIE.
25 November 1993	GB Progress Report placed in the library of the House of Commons.
1 April 1994	The Bovine Spongiform Encephalopathy Compensation Order (S.I. 1994 No. 6743) came into force and introduced changes to the BSE compensation arrangements.
26 April 1994	GB Progress Report placed in the library of the House of Commons.
May 1994	UK Progress Report presented to the OIE General Assembly.
27 June 1994	Commission Decision 94/381 on BSE and feeding of mammalian derived protein. Prohibition on the feeding of mammalian protein to ruminants throughout EU other than Denmark.
June 1994	UK Progress Report Updated.
27 June 1994	Commission Decision 94/382 made on the approval of alternative heat treatment systems for processing animal waste. Effective 1 January 1995.
30 June 1994	Interim results of further BSE experiment (pathogenesis) announced. Extension of SBO ban implemented voluntarily by industry.
27 July 1994	Commission Decision 94/474 introduced new measures on beef export as main changes. Required bone-in beef for export to come from cattle certified not to have been on holdings where BSE has been confirmed in previous six years.
2 November 1994	Bovine Offal (Prohibition) (Amendment) Regulations 1994 came into force, extending ban on use of some SBO in human food to calves under six months of age slaughtered for human consumption. The Spongiform Encephalopathy (Miscellaneous Amendments) Order 1994 came into force, extending ban on use of SBOs in animal feed, banning the use of mammalian protein in ruminant feedingstuffs and making notifiable laboratory suspicion of spongiform encephalopathies in species other than cattle, sheep and goats.
16 December 1994	GB progress report placed in the Library of the House of Commons.
14 December 1994	Commission Decision 94/474 amended by Decision 94/794. Beef from cattle born after 1 January 1992 excluded from certification requirement.
February 1995	SEAC report "Transmissible Spongiform Encephalopathies—a summary of present knowledge and research" published.

Date	*Event*
6 March 1995	Commission Decision 95/60 lifts the restriction in Commission Decision 94/381 on the use of milk, gelatin, amino acids, dicalcium phosphate and dried plasma and other blood products from mammalian tissues in feedingstuffs for ruminants. SEAC are being consulted on the Commission Decision and its implications for gelatin.
1 April 1995	Bovine Offal (Prohibition) (Amendment) Regulations 1995 came into force which make it a requirement to stain SBO with a solution of Patent Blue V (E 131, 1971 Colour Index No 42051).
15 May 1995	UK progress report presented to the OIE General Assembly in Paris.
18 May 1995	GB progress report placed in the Library of the House of Commons.
18 July 1995	Commission Decision 94/474 as amended by Commission Decision 94/794 now amended by 95/287 introduced new measures on beef exports. Previous requirements to exempt beef from cattle born after 1 January 1992 from certification requirements replaced with provision to exempt beef from cattle less than two and a half years of age at slaughter. Also introduced requirement for routine monitoring in feed mills.
15 August 1995	The Specified Bovine Offal Order 1995 (SI 1995, No 1928) took effect. The Order consolidated and streamlined the old rules on SBO. The main changes introduced were tighter controls on record keeping; dedicated lines for rendering plants processing SBO; a prohibition on the removal of brains and eyes so that the whole skull must be disposed of as SBO and a prohibition on the removal of the spinal cord from the vertebral column apart from in slaughterhouses.
28 November 1995	Acting on advice from SEAC the Government announced its decision to suspend the use of bovine vertebral column in the manufacture of mechanically recovered meat.
15 December 1995	The Specified Bovine Offal Order (Amendment) Order 1995 and the Export of Goods (Control) (Amendment Number 2) Order 1995 took effect. The SBO (Amendment) Order prohibits the use of the bovine vertebral column in the manufacture of all MRM and also in the production of some other products for human consumption. It prohibits the use of bovine MRM made from the vertebral column in food for humans. It requires all plants producing bovine MRM to register with MAFF. Finally, it prohibits the export of bovine MRM made from the vertebral column of other EC Member States. The other Order prohibits the export of bovine MRM made from the vertebral column to third countries for human consumption.
20 March 1996	Government announces its intention to consult on further control measures following advice from SEAC. They are that carcasses from cattle aged over 30 months must be deboned in specially licensed plants supervised by the Meat Hygiene Service and that the trimmings kept out of the food chain; and that the use of mammalian meat and bonemeal in feed for all farm animals be banned.

20 March 1996

Examination of Witnesses

RT HON STEPHEN DORRELL, a Member of the House, Secretary of State for Health, RT HON DOUGLAS HOGG, QC, a Member of the House, Minister of Agriculture Fisheries and Food, SIR KENNETH CALMAN, KCB, (Grade 1A), Chief Medical Officer, Department of Health, MR KEITH MELDRUM, CB, (Grade 3), Chief Veterinary Officer, and MR TOM EDDY (Grade 5), Head, Animal Health Disease Control Division, Ministry of Agriculture, Fisheries and Food and Secretary, SEAC, PROFESSOR JOHN PATTISON, Chairman, DR ROBERT WILL, Deputy Chairman, and DR RICHARD KIMBERLIN, Member, Spongiform Encephalopathy Advisory Committee (SEAC), were examined.

Chairman: Good morning. Before we proceed to addressing the witnesses and asking the Ministers to introduce their teams, I am required to ask Members of the Committee to declare their interests. I have a consultancy with British Sugar, who have a company within the same group called ABM who manufacture animal feedstuffs, in which they place animal protein—or have been up until the ban.

Audrey Wise: Until the end of this month, when the scheme ends, I am a sponsored member of the Union of Shop Distributive and Allied Workers. I am also President of that Union.

Mr Corbett: I am also sponsored until the end of the month by USDAW.

Chairman

1. Mr Dorrell, welcome. Would you be kind enough to introduce your team, and then I will ask Mr Hogg to introduce his.

(*Mr Dorrell*) Chairman, thank you very much. I am not sure whether SEAC counts as a joint sponsored body, but perhaps I can introduce the people who sit on my left: Dr Richard Kimberlin, at the end, who is a member of SEAC; Professor John Pattison, who is the Chairman of SEAC; and Sir Kenneth Calman, who is the Chief Medical Officer.

(*Mr Hogg*) Chairman, Tom Eddy is the Secretary of SEAC, and is also responsible within MAFF for animal diseases, most notably for BSE; Dr Robert Will is on SEAC, of which he is Vice-Chairman. He is responsible too for the National CJD Surveillance Unit; Mr Keith Meldrum is the Chief Veterinary Officer within MAFF.

2. Mr Dorrell has indicated to me that he would like to make a short statement before we proceed.

(*Mr Dorrell*) Sir Jerry, thank you very much. I do not intend to detain the two committees by reciting in detail a history that has become extremely familiar, but I would like to begin by setting out the Government's present position on these issues. The ten cases of a new variant of Creutzfeldt-Jakob disease identified in people aged under 42, which have been studied by the CJD Surveillance Unit at Edinburgh University, were considered by SEAC and they concluded that the most likely explanation at present is that these cases are linked to exposure to BSE before the specified offal ban in 1989. SEAC's central conclusion at its meeting last week was that further refinement of the offal ban was necessary but that, against the background of solid enforcement of the offal ban as refined, the risk associated with eating British beef is extremely small. The key further question of age susceptibility to infection, together with a range of secondary questions, was considered by SEAC last weekend. Its conclusion was that, if human infection with the BSE agent does occur, infants and children are not likely to be more susceptible to that infection than are adults. These conclusions were all published promptly. The first statement was approved on Wednesday morning and published on Wednesday afternoon of last week. The statement agreed last Sunday night was published on Monday afternoon of this week. We believe that these decisions reflect that which is scientifically necessary to make British beef acceptably safe for consumers. Yesterday the argument moved on. The issue is no longer a question of the safety of British beef: the best available evidence demonstrates that British beef and beef products can be safely eaten by consumers both here and around the world. The question now is a matter of consumer confidence. It is one thing to have a safe product—it is another to command confidence in the marketplace. When the Prime Minister was asked at Question Time yesterday about the call from Sir David Naish, President of the NFU, for a slaughter policy he said, "There are two reasons for proceeding in the way which Sir David recommends: first, on public health grounds if science recommends it—but science does not recommend it; secondly, if it proves necessary to restore confidence to the market". The Prime Minister has made clear that the issues raised by Sir David will be considered very seriously, and quickly, so that confidence in beef can be restored. Both the Minister of Agriculture and I will be pleased to answer your questions. We do so against the background of the Government having taken the necessary steps to ensure the safety of British beef, and having indicated its willingness to engage in discussions about the steps necessary to restore confidence in a safe product.

3. Thank you very much. May I start by addressing Dr Will and Professor Pattison on the question of the ten cases, which were the bit of news which sparked off this episode. Are you as satisfied as you possibly could be that these were, if associated with BSE, acquired pre-1989?

(*Professor Pattison*) I think the answer to that question, Chairman, is that we again have to make certain assumptions. If it is, as we know it is, a spongiform encephalopathy in man, the most frequent incubation periods for cases of such a disease (and we have to go to a primitive tribe in New Guinea to get the experience—Kuru) is between five and 15 years. It is true that there is one case of Kuru that has a shorter incubation period than five years, but the most probable length of the incubation period is between five and 15. Although we have only identified and described for you this new variant in 1996—we can come back to that in a moment as to

[**Chairman** *Cont*]
why—when you look at the onset of the symptoms of these individuals, the onset was in 1994 in six of them, and in 1995 in four of them. If you trace back for five years, the shortest incubation period, then that would take you back to 1989 and 1990: but the more likely is that this would be a longer incubation period than that, because at the same time, pre the ban, the exposure (if it was due to BSE) would have been much greater, because we know at that time that some central nervous system of cattle, brain and spinal cord, went into the human food chain, and that is always the most dangerous tissue.

4. In a nutshell your answer is, yes?
(*Professor Pattison*) Yes.

5. I do appreciate the problem the scientists have—after all, we have had an inquiry into the subject before. I appreciate that you can never say "never". What we are endeavouring to do this morning is to persuade my colleagues, in simple terms if possible, what are likely to be the basic facts. The basic fact is, if there is this connection (and it seems quite probable there is a connection) it was acquired pre-1989. At that time precautions were taken, on the assumption that there might be some connection between, or it might be passable between cattle and human beings, to preserve the purity of beef by removing specified offal, by slaughtering BSE infected cattle etc. etc. Given that the average life of a beef animal is unlikely to be more than three years, and therefore the vast majority of beef that is eaten probably would not have developed a disease, but that there are beef products in dairy cows that are used, and therefore there is a possibility that an animal incubating BSE could be slaughtered but that the infected material is removed, would you not agree that the odds of any infected material actually appearing on your plate, or even on your butcher's plate, is as near zero as is humanly possible to make it?
(*Professor Pattison*) I believe that that is now the case, yes.

6. You are saying there were imperfections in the process between 1989 and some point at which they were rectified?
(*Professor Pattison*) Yes. It was disturbing last year, on the basis of unannounced inspections of slaughterhouses, that the regulations which had been put in place were not being adhered to on a scale that was uncomfortably high. That was very much tightened up by continuous inspections from the middle to late of last year onwards. We brought in the mechanically recovered meat ban in December, and we have now recommended further refinements simply to ensure that potentially dangerous tissue is rigorously excluded, and passed these on to government.

7. We are talking about items of materials, bits of spinal cord, or bits of material you state could be infected. These are visible to the human eye. Is it likely, even if the processes in the slaughterhouse were incorrectly carried out—and we acknowledge that is the case—that the housewife would get as far as actually ingesting any of this material?
(*Professor Pattison*) We have to say we were convinced that it was possible.

8. There is a great deal of difference between "was possible" and "is likely". If the meat is washed, trimmed and produced for sale and then cooked?
(*Professor Pattison*) Yes, but it was not particularly the prime cuts of beef we were worried about at that time; it was particularly concerning vertebrae of cattle which might have still contained a piece of spinal cord when put into a large machine from which was extruded material that would go into the human food chain; inevitably that spinal cord would be in there.

9. Is cooking likely to destroy the agent?
(*Professor Pattison*) No. They are very resistant. You have to use very high temperatures for a long time, or steam under pressure for a long time.

10. Bearing in mind the odds of an infected beast being sent for slaughter anyhow, the odds of acquiring infected material are very, very small and almost exclusively in manufactured products?
(*Professor Pattison*) That is our belief. I am sure later on this morning we will get round to the problem of the sensitivities of the tests that we have to detect this agent, even in cattle which we have experimentally infected.

Mrs Roe

11. Thank you very much, Chairman. I would like to address my questions to Sir Kenneth Calman and Mr Keith Meldrum, in fact taking the points further which have just been given. Could I put five questions to you. In order to save time I will list the five questions. I would like you to explain the current state of scientific knowledge about, firstly, the likely infective agent, the sensitivity of tests to detect it, and the effects on it, and the effects on it of washing and cooking meat; secondly, the distribution of the infective agent throughout the body tissues; thirdly, health implications of people working with cattle or meat: farmers, abattoir workers etc; fourthly, the progress of the diseases in individual animals and humans; and, fifthly, the prospects for cures or treatments?
(*Sir Kenneth Calman*) Thank you. If I may begin and perhaps pick up some of these questions, but I know that Keith Meldrum, and perhaps Dr Kimberlin, could pick up some of the others. The knowledge of the agent, the prion, is of course growing all the time. It is an agent which is new to us; it is a protein; its exact mode of action is not entirely clear; but it is an infective agent, I think that is the issue which really matters. It is something which does transmit disease from one animal to another. That is the key to it. The sensitivity of tests to detect it vary of course depending on the model. There is not a simple test which can be done on live animals or live humans, although clearly others are working on this, and working on this very hard. The tests, particularly on various tissues, depend on animal models, and the most usual models take a portion of brain, which has been taken from an animal which is clearly infected, and inject it into mice. Then you begin to dilute that so you begin to see when you cannot transmit it. When you do that you can then have a measure of the sensitivity of the test, and against that you can then test a number of other organs—such as muscle, liver,

[**Mrs Roe** *Cont*]

spleen, lymph nodes, bowel etc. That is really the only way in which you can get some sensitivity into the tests. Having done that on tissues like muscle, for example, we have found no evidence of transmission; but that then has to be qualified: what is the sensitivity of that test? Clearly less than the sensitivity of injecting infected brain into a mouse; it gives you only the floor, and does not tell you how far below the floor that goes. In terms of washing, clearly any large particles would be washed off in that process but, as Professor Pattison has said, the cooking of meat itself would not be sufficient to destroy the agent. In terms of the distribution of the agents through the tissues of the cow, perhaps I can pass over to Keith Meldrum to pick that up, and perhaps I will return to some of the others in a moment.

(*Mr Meldrum*) Chairman, on the animal side, infectivity through the mouse as the indicator of presence of infectivity has only been found in clinically affected cattle in the brain and spinal cord, and also in the retina of the eye. In all the other tissues taken from clinically affected cows, we have not found any infectivity using the mouse as the indicator. Experimentally, and exposing calves to infectivity at a very young age, by the oral route we have found infectivity also in the intestine of these calves. I would just point out if I may, all those tissues I have mentioned are covered by the specified bovine offal ban.

(*Dr Kimberlin*) With regard to the nature of the agents, I have picked up very much in recent reportage an underlying uncertainty which goes along the lines of, "How can you be sure of what you are doing and thinking about these agents if you are not quite sure exactly what they are?" Of course, as in many scientific areas of research, there are uncertainties, in this case, about the physical and chemical nature of the transmissible agents. That is true and as Sir Kenneth Calman has said, the prion hypothesis is the favourite one. The point I want to make is that a much firmer body of knowledge was available to us about these agents in terms of the way they behave rather than what they are. Indeed, all the strategies that were devised to protect public health were based far less on the uncertainties about exactly what kind of agents these are, and much more on a firm basis of knowledge of the way they behave biologically. It is a crucial point in my mind. With regard to the sensitivities of detection, you know what I mean by the term "bio-assay", this is the usual way of doing the quantitative measurements of infectivity. Potentially bio-assays are very sensitive, but there are limitations. The two I would draw attention to are: one, if you use injection of a tissue into a susceptible mouse by the intra-cerebral route you are maximising the sensitivities, but you are always limited physically in the amount of material you can inject. We are accumulating a large body of data which says no detectable infectivity in most tissues. What that really means is that we have got to the limit of the threshold of detection. Much of the bio-assay work has been done, of necessity, in mice, that is, we have been looking at tissue infectivities from cows bio-assayed in mice. That means crossing a species barrier which reduces sensitivity. The way around that, of course, is to do some sensitivity tests in cattle when there is no longer a species barrier. It would have been phenomenally expensive to do all the testing that has been done in cattle versus mice. What do we end up with? We end up with a two-stage process. One is where you get a good feel for how much infectivity is in different tissues by using mice as the subject of the test; and the other is that you do comparative studies in cattle to measure the insensitivity of the mouse assay. If you put those two together then you begin to get potentially very sensitive indicators of infectivity. But there will always be a limit of detectability, and we have to live with that.

(*Sir Kenneth Calman*) May I just pick up the other issues about the health indicators of people coming into contact with cattle or meat. The assumption we have to make is that potential for infection is there. That does not say that there is infection, but the potential. It may occur through two routes: the oral route, or through things like cuts and grazes on the skin, or perhaps through an aerosol. As far as meat is concerned, what we eat, I think the statement by SEAC is the one with which I agree—that the risk of contracting any human disease from cattle who are BSE infected is extremely small. We have looked at that in great detail, and I agree entirely with SEAC's statement on that. The second issue is, if you like, occupational exposure to those working in abattoirs etc. The CJD Unit in Edinburgh has looked at this over the last five years to see if there are any particular occupations which are at risk. So far there is nothing which has come through from that. The Health and Safety Executive have of course set out guidelines for working in such areas, including laboratories, and they will be looking at that again. In conclusion, the risk, if there is a risk at all, is extremely low, and certainly by the oral route extremely low indeed.

12. And the prospects of cures or treatment and the progress of the disease in individual animals and humans?

(*Sir Kenneth Calman*) As far as the progression of disease in individual animals and humans is concerned Dr Will, as a neurologist, may well be able to say more about that. As you know, it is a rapidly progressive disease with a variety of neurological problems. At the present time there is no prospect of cure or effective treatment available.

(*Mr Meldrum*) May I come back to the question of the progression of disease: so far as animals are concerned I mentioned earlier the pathogenesis study in which we found infectivity in calves after experimental exposure by mouth. That particular experiment is designed to determine, if we can, the route by which the agent of BSE would get from the mouth to the brain. So far we have only found the agent in that particular batch of calves in the intestine. We are now up to 18 months of age after exposure and we have not found the agent outside the intestine. In due course I hope that experiment will give us a clear indication as to how it does move, and the route by which it moves, from the intestine to the spinal cord.

Alice Mahon

13. Could I ask if somebody would comment, you have already said it would have been phenomenally expensive to do the tests in cows. Can you tell us how much that would have cost if that route had been followed? Secondly, would somebody like to comment on the work that Dr Harash Narang was doing on tests, which I understood MAFF looked at and said worked?

(*Dr Kimberlin*) I cannot really put a figure on "phenomenally expensive", so I would invite you to use your imagination.

14. If somebody makes the statement "phenomenally expensive" surely the Committee is entitled for somebody to have a guess?

(*Dr Kimberlin*) Millions. We are talking about bio-assays that have been done in mice, using thousands of mice and replacing those with thousands of cattle. That really was not the point I wanted to make. The point I want to make is that actually bio-assays are potentially very susceptible. What you do is you use a two tier system. You use a susceptible, reasonably sensitive mouse strain to give you a rough ranking of what infectivities you have got in different tissues. For example, you can measure quite accurately how much infectivity there is in a bovine brain using mice. The problem is we have a lot of tissues which are negative when bio-assayed in mice. That does not necessarily mean there is nothing there. The reason is because the bio-assays are less sensitive in mice than they would be in cattle. The way you get round that problem is to take some of the important tissues which you are concerned about which are negative when you test them in mice, and then test them in cattle. That way you increase the sensitivity of the test. You also do it in a much more cost-effective way. My comment about "phenomenally expensive" was not a judgment about whether it should or should not have been done; it just was not a cost-effective way of doing it.

(*Mr Dorrell*) Can I suggest, Chairman, if the Committee is concerned about the cost of that experiment we will send them information both on the relative costs but, much more importantly, on the scientific justification for the correlation between the mouse—

(*Dr Will*) You wanted to discuss Dr Narang's test. I was very fortunate to have the opportunity to visit Dr Narang fairly recently in order to discuss this issue because, as far as I am concerned, in relation to the surveillance of CJD it is very important to consider any mechanism to improve diagnostic accuracy. My understanding is that Dr Narang has a test which has only been tried out on a very small number of patients. I think we do not have enough evidence in relation to whether there are false positives or false negatives etc. in order to use this test systematically. However, what we have agreed to do is to try and see whether, by providing some specimens for Dr Narang from the CJD Surveillance Unit, we can determine whether or not the test really was useful or not. Just one other thing I should mention is that there are other developments in relation to tests. We would hope that a specific test on cerebro-spinal fluid might be a valid test to help with the diagnosis; and there is currently evidence in the United States that this may be a very useful test. We hope to be collaborating within the next week or two in trying to see whether this test works in the United Kingdom.

15. May I ask why Dr Narang's research was withdrawn and why, with hindsight, notice was not taken of the test he was developing?

(*Mr Eddy*) Dr Narang, as I understand it, has in fact developed a number of tests over the years. Back in about 1990 he developed a test which was used on the dead animal. We already have tests on the dead animal. We did some work with him to validate that test and make sure it worked. The conclusion at that time was that it was a test on a dead animal; we already had that kind of test; and in comparison with the existing tests it did not give such a good performance. It was not taken up at that time. I think that might be the test you have in mind when you say MAFF looked at one of his tests and it worked—that is true. There were other tests which had a better performance, and these were all tests for dead animals. In terms of a live test, I understand the newspapers have reported he has developed a test; I believe it is probably similar to the human test which Dr Will has just mentioned. We have tried to find out from him how the test works, but I understand from his replies that he has it under commercial development and does not want to give us any details at this stage. Clearly that is his personal choice.

16. Could you comment on why he did not get the resources at the time?

(*Mr Dorrell*) The position as far as research funding is concerned is that I made clear to the House last week we are providing extra money for research. One of the objectives of research clearly, as far as the human health angle is concerned, is to ensure that we have, as soon as we can, an accurate diagnosis of CJD, and with this particular new strain of CJD; that is an objective we all share. The steer from the politicians has been to ensure that we move as quickly as possible to develop the test that will be useful in the accurate diagnosis of these conditions.

(*Mr Hogg*) Particularly a live test.

(*Mr Meldrum*) Chairman, may I just come back on one point. Dr Will mentioned the encouraging work that has been carried out in the USA using CSF as a source material. We have already supplied material to that same team from known BSE affected cattle and from control material, so we also are working with that particular team. We will work with any team if we believe there is a reasonable chance that we can from that work develop a diagnostic test, either for use for screening purposes or for differential diagnostic purposes.

Mr Marshall

17. Sir Kenneth did say "the risk was extremely small". Would the Secretary of State not agree that that demonstrates the sheer irresponsibility of some of the headlines put out by the popular press last week, and the hysteria that has developed from it? One can remember the headline last week which said it could be worse than AIDS; or the headline in another paper which implied that half a million people might be dying of BSE. Has that irresponsibility not done a great deal of harm to a great British industry?

[**Mr Marshall** *Cont*]

(*Mr Dorrell*) What we have certainly seen over the last week is a series of speculations about what could or could not happen against a background of clear advice from those who have examined the evidence that the risks are extremely low. If we had the same degree of coverage and the same amount of speculation on the assessment of what is likely to happen rather than what on some of the wildest speculation could conceivably happen, then I believe that the reaction of people would have been markedly different.

Mr Corbett

18. Dr Kimberlin, can I take you back to this comment you made about tests on cattle would be phenomenally expensive. Was the decision not to conduct those tests taken on scientific or financial grounds?

(*Dr Kimberlin*) It was not a question of deciding one against the other—at least not in my mind. It was a question of getting as much information as quickly and as effectively as possible. The mouse assay, despite the fact it has a relative insensitivity compared to cattle—because you are measuring across a species barrier—is actually quicker. It is not just a question of sensitivity, but a question of time as well.

19. Which is the most scientifically justified?

(*Dr Kimberlin*) They both are. The trick, as I say, is to make use of the virtues of both tests.

(*Mr Dorrell*) I have already offered, Chairman, to write to the Committee setting out the basis on which that decision was taken. If I may suggest that would allow the Committee to examine how the decision was taken about the precise structure of that test.

Chairman: Thank you very much. We accept your offer.

Mr Bayley

20. I have three short but related questions. Professor Pattison, you made reference to the unannounced visits at the end of last year to abattoirs in which, according to Mr Hogg's parliamentary answer, 48 per cent of the abattoirs were found to be failing in the handling of specified bovine offal, and 65 per cent of knackeries and hunt kennels were failing to comply with the rules for handling selected bovine offal. You mentioned to us that the rigour of the inspection regime has been improved. Have you gone back with unannounced visits to test what proportion are now complying?

(*Professor Pattison*) Yes, we have. The Advisory Committee is continually updated about the results of those visits. If you would like the details I am sure Tom Eddy can provide them for you. It is most encouraging, the change which has taken place.

(*Mr Meldrum*) I anticipated this question—in fact, I will take about an hour to go through all the various procedures we put in place as from 1988-89——

21. It is not the procedures; I want to know how many inspections there have been.

(*Mr Meldrum*)——the procedures are in fact to check compliance with the regulations laid down by Parliament—and I went through this. More recently, of course, we have continued our system of unannounced visits to check upon, first of all, the responsibility that lies on the slaughterhouse owner to comply with the rules; secondly, the meat hygiene service is there on a day-to-day basis to ensure enforcement; and the State Veterinary Service comes in later to do an audit of the system. Our visits will continue, have continued; we make all that information available to the Advisory Committee.

22. With respect, I must go back to that question. In the parliamentary answer we were told there were 193 visits to slaughterhouses, and failings in the handling of bovine offal were found in 92. Since the changes were made how many unannounced visits have there been, and in how many of those cases have parts of bovine offal, which should have been removed, been found?

(*Mr Hogg*) That is probably the subject of a written response to you setting out that information. It might be helpful just to add, however, firstly, I called in the industry on at least two occasions in the latter part of last year, (meetings which indeed attracted a certain amount of publicity at the time), to make it plain how important it was to achieve the full implementation of the regulations. You will also bear in mind that I then introduced a ban on mechanically recovered meat; that was done in December. My colleague, Angela Browning, has also called in representatives from knacker houses to make the same point I made to the slaughterhouse operators. We attach a very high importance to implementation of the regulations.

Chairman

23. It strikes me that if these precautions were taken as long ago as four or five months, the chances of any of this meat remaining in the food chain, which could conceivably have been affected, are pretty minuscule?

(*Mr Hogg*) We would agree with that, Chairman, because that really is what underpins the assertion, along with other facts, that eating British beef is extremely safe; or, if you want to put it differently, the risk is very low.

(*Mr Meldrum*) Could I just answer Mr Bayley's question in part. I think one has to separate when we have identified and reported upon non-compliance with the rules on the SBOs those that might be an animal health risk and those that might be a public health risk. The public health issue is to ensure that these SBOs are removed from the human food chain. I can say to you that so far in 1996 there have been only four occasions when we have found pieces of spinal cord in the carcass at the auditing visits we have done. That is the extent of the non-compliance in the public health arena.

24. We must make progress. Is it like any other poison, that its effect is proportional to the quantity? Therefore, if you ingest a tiny, minute amount the chances of being infected are very small; you do have to have a reasonable quantity before there is any likelihood of infection?

(*Professor Pattison*) Yes, yes.

Mr Pickthall

25. Mr Meldrum raised the question of infectivity in intestines of cows. Common sense would suggest, moving on from that, that it would be possible for the agent to be in animal faeces, and animal faeces—slurry—is spread on grassland, the grass, of course, grows better and is eaten by the next set of cattle along. Can you explain to me, because I have not found it anywhere, whether my fears that that process could also be part of the infective chain are groundless or otherwise?

(*Mr Meldrum*) My answer very quickly would be that there is no such chance, but would you be kind enough to allow Dr Kimberlin to answer this question because he is a particular expert in this area?

(*Dr Kimberlin*) I am just nervous about breaking into another long seminar which I would like to avoid.

Chairman

26. Please do not do that!

(*Dr Kimberlin*) There are two quite separate scenarios here. One is, of course, that in the experiments that the CVO has described on deliberately infecting cattle by the oral route in order to study where the agent goes, and when and so on and so forth, one of the crucial essentials was to be absolutely sure that all the animals that you were infecting did indeed become infected—otherwise the experiment becomes extremely wasteful. That was achieved, in fact, by using a phenomenal (I keep using this word but I can quantify this "phenomenal") exposure—100 grams of BSE infected brain. We did it that way simply because we did not know how much lower the exposure could be. The consequence of it was that it certainly told us the first site of infection. But, if you like, it inflated the result quantitatively. It is what I call the "Heineken effect" because by putting in such a huge exposure to achieve infection, which is what you want, you introduce literally, an enormous amount of material to the first place where it is going to be taken up in the gut. Can we cut there and move on to scenario two? Scenario two is that when you look at the field epidemic of BSE you get a very different impression. In fact, we use the phrase "low-dose exposure" because it is remarkable the extent to which that epidemic has involved just one animal of this herd, two in that herd, and so on and so forth. The explanation for this is simply because the average exposure to cattle in the field has been at an extremely low level. It is this discrepancy, I think, which leads me to conclude that the predictions you would make about contamination of pasture will be very different if the pathogenesis experiment was, in fact, a description of the field exposure. Field exposure at low doses means that very little infection is going in, but because cows eat a lot, sooner or later there will be enough for them to become infected. But what goes out will also be at a low level of infection. So the pathogenesis experiment does not inform on the risks from the environment, and I think you have to separate the two. I can see some very puzzled expressions—I have not explained that well, so I will shut up!

27. It is a fascinating answer but it still does not put my mind at rest on this particular issue. Is the land on which BSE infected beasts have been discovered tested? Has it been tested over a period of time and nothing has been found?

(*Professor Pattison*) No, it has not been tested.

Audrey Wise

28. Reverting to the question of abattoirs, can I ask, in view of the high level of non-compliance which has been identified by our witnesses, how many prosecutions resulted from these examples of non-compliance? Clearly, non-compliance in such a matter should be regarded very seriously, and compliance is more likely if adverse results for the non-compliers are seen to be clearly demonstrated in prosecutions, if you want those. Furthermore, can we be told how many inspectors are employed now for how many abattoirs and how many, say, a year ago?

(*Mr Hogg*) Chairman, on the question of prosecutions, primarily they are a matter for the Meat Hygiene Service. They are looking at a number of cases to see whether there is sufficient evidence to justify bringing criminal cases, because they are criminal prosecutions. On the actual number of inspectors, I do not know the exact numbers but we can furnish them. It may be, Tom, you have some figures.

(*Mr Eddy*) Unfortunately I do not. I think one of the problems is that a year ago there was a completely different arrangement; it was all done by local authorities and now we have the Meat Hygiene Service. So the figures are going to be difficult to compare. I am sure they can be put together, but they will not necessarily be very easy to compare because you are not comparing one organisation at two times but one organisation with a completely different set-up.

(*Mr Hogg*) It is perhaps important to remember, Sir Jerry, that the Meat Hygiene Service took over responsibility on 1 April 1995 for this particular function. Prior to that it was the local authorities.

29. Are we saying, then, that there was no governmental responsibility at all in this matter? It does not seem to me that a change in the detailed organisation should justify a lack of information about levels of criminal responsibility. Am I to understand that our witnesses do not know, as they sit here, how many inspectors are inspecting how many abattoirs?

(*Mr Hogg*) Chairman, the position is this: that before 1 April 1995 the local authorities were responsible for supervising the operation of the abattoirs. There was an overarching responsibility on the part of the State Veterinary Service, which it still exercises. On 1 April 1995 the Meat Hygiene Service took over responsibility for the work previously done by the local authority. There is an over-arching responsibility over them which is exercised by the State Veterinary Service. There are a very large number of inspectors operating in slaughter houses, and if you go there you will see very many inspectors from the Meat Hygiene Service. I do

[**Audrey Wise** *Cont*]
not have to hand in front of me the exact number, but we can certainly provide that without any difficulty at all.

Mr Jones

30. Just a brief question. A lot of bovine tissues were tested, I understand, for infectivity, which is why we have got the information we have been given up to now. Was bovine faeces tested? I think this is important in terms of Mr Pickthall's question.

(*Professor Pattison*) Others will look that up. There is a limited amount of testing in some species that I can remember without looking it up, and faeces are negative. I should perhaps say also that the fact that the intestine is positive is, we believe, because the local lymphoid tissue in the intestines is the repository of this agent. That is very different from saying that the lining of the intestine is positive, in which case you might well expect there to be an exchange with the contents of the intestine itself and it would come out the other end. It is not an infectious agent, whatever its nature is, that behaves in the same way as bacteria or viruses do, and that is they build up in the cell and are discharged out of the cell. This is really an abnormal cellular protein that accumulates, and so we do not believe that in the pathogenesis there is a great outpouring of the transmissable agent into—in this case—the contents of the intestine.

Mr Campbell-Savours

31. Mr Hogg, can I ask a question on Mr Dorrell's statement? It has been reported this morning that you are set to reveal the extent of a selective slaughtering plan when you appear before us today. Is that true or not true?

(*Mr Hogg*) Untrue. I simply, on this matter, endorse what Stephen Dorrell has said to this Committee. I have nothing to add on that point to what he has already said.

Mr Powell

32. Sir Kenneth, I want to take you away, for a moment or two, from the rather calm, clinical atmosphere of the science laboratory in various experimentations—important though they are—to consider some of the matters which I think are of interest to my own constituents and, I suspect, the constituents of my colleagues in this Committee. I represent the consumers and the farmers—who are also consumers—in the north east Northampton area. They are anxious in a way which I have never known before—and I suspect I speak for everybody else in this room when I say that—and a doctor of very great experience said to me on Friday that I was 200,000 times more likely to get cancer from smoking than I was to get CJD. Would you find that an inappropriate remark from an experienced doctor?

(*Sir Kenneth Calman*) It is a remark which I think is very easy to make. I think the issue, and Professor Pattison may well wish to develop this in a moment, is the issue of what is the risk? I think one of the most interesting issues for this Committee, because it is an issue that I have raised before in the Health Committee, is the issue of risk. I think the comment made by your medical friend puts the risk into perspective; the risk of smoking cigarettes is very much higher than we think the risk is in relation to eating meat.

33. The actual figure he gave was 200,000 times more likely, which is a striking one and worth bearing in mind. You will recall the Leader of the Opposition asked the Prime Minister yesterday specifically about the likelihood of risk. When one quantifies it in that kind of form then one can begin to talk in a language which our constituents understand rather than against the rather clinical talk—important though it is—that we have had for the last hour and a quarter. Lord Wyatt, in an article in *The Times* yesterday, suggested that women were 50,000 times more likely to get cancer of the breast than they were to get CJD. Was Lord Wyatt wrong when he made that remark?

(*Sir Kenneth Calman*) I am sure Lord Wyatt has all the reasons that you know of to make that kind of remark and be able to justify that scientifically. The issue for us in the Committee is that if we look at the evidence across the board, the evidence presented to SEAC, to which SEAC has responded, does suggest that the risk is extremely low. If you compare that to cigarette smoking, crossing the street or getting breast cancer, then you can make a whole series of assumptions about that. No matter how you do it, and I would not wish to be drawn into individual figures because it may be several orders of magnitude more than you have suggested—in fact, there may be no risk at all—we have to keep returning to this: the evidence that there is a risk between BSE and CJD and human disease has not yet been proven. Let me return to that. So these risks are theoretical risks. Again, if I can call on Professor Pattison just to go through some of the discussions that they have had in SEAC about trying to put that risk in perspective.

34. I understand you may want to do that, but this Committee cannot sit for 24 hours upon 24 hours upon 24 hours. I want to make a few brief points, because I know there are many in this Committee who want to follow up their own remarks and mine as well. Mr Dorrell, many of my constituents are extremely worried because they fear that as a result of this extraordinary panic which has taken place about the quality of our beef inferior beef is going to be imported and sold in this country from various continental countries. You will be aware of the number of reports which have appeared in the newspapers in recent days. May I draw your attention to the front page of *The Times* today, dealing with meat in Italy and deaths from CJD? May I particularly draw your attention to the Peterborough column in the *Daily Telegraph* last Saturday dealing with beef in Belgium, where we are told that Belgian beef has been injected with illegal hormones, including clembuturol, cortisone and angel dust—which is, apparently, cement powder! There is a real danger, apparently, that meat of this sort, which must be far inferior to anything which is available in this country, is now going to be sold as hamburgers and other joints as though it was superior to British beef which has been the subject of such criticism. What action is going to be taken to ensure that such meat is not made available in this country?

[Mr Powell *Cont*]

(*Mr Dorrell*) Chairman, I think that the case Mr Powell puts is one that will have occurred to very, very many people who have read reports and looked at the background to this case. They will, for example, have observed that this country has conducted, for many years, certainly through the 1980s, a trade both in live animals and in feedstuffs with other continental countries, and they will wonder how it can be that science tells us that that produces one result in Britain and, apparently, a quite different result on the continent. Those are questions that many people, who have examined the background to this—and certainly my farming constituents—will be asking themselves: "Why is it that science apparently, on the evidence that is published, produces one result on one side of the Channel and another result on the other side of the Channel?"

35. Can I ask you, finally at this stage, how the CJD Surveillance Unit set about eliminating other possible causes of the ten cases which have come to light? Might there be other explanations? What other explanations are considered for the fact that this has occurred?

(*Mr Dorrell*) I do not propose to speculate on what is a scientific question. Mr Powell is taking us back into clinical questions and I refer them back to Dr Will.

(*Dr Will*) If I could try and answer that question, because it is a very important question. We felt, earlier this year when we started to identify these cases, that, of course, these cases should be made public and that SEAC should be made aware of them etc. We have spent a great deal of time trying to think of alternative explanations for why these cases might have occurred. For example, could it simply be that these cases have occurred as a matter of bias in identification of cases because of publicity? This is certainly one thing we have seriously considered. We had to also consider whether there was a possibility that these cases were not a new phenomena and may have occurred before but may not have been identified. We also had to consider whether the features of these cases are really as unusual as we believe, and we have done a great deal of research into that as well. It would seem that other risk factors in these cases have been excluded, including genetic factors or risks in relation to medical treatment (the tragic occurrence as a result of human growth hormone treatment is one example). I think we have concluded that we can find no other explanation for these cases, and it does appear to be a new phenomenon. However, it is very important to consider whether other information may be available in the future from other countries that may suggest that it is not a new phenomenon. We do not believe there is such evidence, but we do have to be cautious. The other thing I have to say is that it is not a good scientific argument to say because there is no other explanation this must be related to BSE. However, this is a very odd occurrence; this cluster of cases is completely unexpected and unlike anything we have seen before in the CJD Surveillance Unit. Therefore, in my opinion it raises the possibility of a link with BSE, but we cannot confirm that on the basis of current evidence.

36. When do you expect to be able to give us further information about this?

(*Dr Will*) We are continuing to try to obtain information from other countries on, for example, this relationship to frequency in this age-group of CJD. The evidence we have suggests this is an exceptional phenomenon and apparently has only occurred in the United Kingdom. We also hope to carry out further studies to see if we can determine whether this is truly a new variant of CJD.

(*Professor Pattison*) Specifically on your last point, which is a crucial one, there are laboratory tests available, which have been set up, and they involve either normal strains of mice or transgenic mice. Assuming we are trying to exclude the possibility, that will take two years—to see whether the agent that you find in the brain in these cases is BSE. Of course, if it turns out that it is due to it, we might get the answer more quickly, but it is of that sort of timescale.

37. You mentioned that you would be seeking evidence from abroad. One of the matters which I think concerns my constituents is the quality and integrity of information about BSE and CJD which is available from abroad, and whether or not there is, in fact, a great cover-up which is taking place elsewhere, (*The Times* rather strongly suggested that this was the case in Italy in its article this morning, to which I have already drawn your attention) or whether we should really rely upon people from elsewhere who are trying to pretend that they do not have a problem when everybody knows they do have a problem.

(*Mr Dorrell*) I have already suggested, Chairman, that I think the questions Mr Powell poses are indeed questions that will have occurred to many hundreds of thousands—probably millions—of people, if I may put a figure on it, as they have read the coverage of this story over the last week.

Mr Jones

38. Can I go on to what we are discussing this morning, which is the origins of the present problem and the reason for your statement the other day. In the 1980s there was an 18-month delay before BSE was made notifiable. There was a 20-month delay before the Government brought in a compulsory slaughter scheme. There was another 18-month delay before we got full compensation paid for the slaughter of cattle. In retrospect, was it not a crucial mistake that from August 1988 to February 1990 farmers were only offered 50 per cent compensation for infected cattle and that from 1994 the level has been reduced yet again? Has this not tempted farmers to sell their suspect BSE cattle, thus exposing consumers to BSE in the food chain for several years?

(*Mr Hogg*) Chairman, you, I think, know that there was a Select Committee Report into these very issues in 1990. Paragraphs 37 to 44 really contain the points to which Mr Jones is referring. They were responded to by Government in its formal response to the Committee's report, and those are contained in paragraphs 41 to 54 of the response. These are the contemporary documents, there was an exchange with us in written form back in 1990. I would suggest to this Committee that the best way is go to those

[Mr Jones *Cont*]

documents and see what was said at the time, some six years ago. On the question of compensation, which was a particular point made by Mr Jones, he will find that paragraph 52 of the Department's response deals in turn with that, and paragraph 54 concludes that "... as the Committee knows, increasing compensation does not appear to have had any effect on the numbers being reported". He will find that in the response. He will also, I think, wish to look with some care at Sir Richard Southwood's report which he published in February 1989, and in particular to page 21 of that report, which I happen to have with me. Paragraph 9.2 is, perhaps, the most interesting. "From present evidence it is likely that cattle will prove to be a dead-end host for the disease agent, and most unlikely that BSE will have any implication for human health. Nevertheless, if our assessments of these likelihoods have been incorrect the implication will be extremely serious. Thus we greatly welcome the speed with which the Ministry of Agriculture, Fisheries and Food have brought forward regulations based on the veterinary evidence and our recommendations ... " and so on. That is to be found at paragraph 9.2 of Sir Richard Southwood's report in February 1990.

39. I asked: in retrospect, would the Minister say that what was done at the time was not enough? We are talking about the Government's response. The Government's response, in terms of four years' delay in getting to the situation where we are now, must have meant, regardless of what the apparent evidence was at the time, the fact is many farmers could possibly have put cattle into the food chain at that time and exposed consumers to a greater risk than was necessary. In retrospect, does the Minister not think that is true?

(*Mr Hogg*) Chairman, I have nothing to add to what was said in 1990 when your Select Committee examined this very question expressly and the Department responded to it expressly. That was the subject of your report and of our response. I have no reason to depart from the response that we gave six years ago.

40. Why, if it was the case that the Ministry at the time thought that compensation was adequate at 50 per cent, did they then increase it?

(*Mr Hogg*) Because there was a certain amount of equity involved in this, but the essential question is whether it led to any under-reporting. The Agriculture Committee did not think that, and the Department agreed with the view expressed by your Committee.

Mr Alexander

41. Further to that question, there has, of course, been a good deal of advice to Government this week and last, based on the benefits of hindsight. Further to the line of questioning which Mr Jones put to you, are there any things which, in retrospect, you feel the Government should have done over the past 15 years?

(*Mr Hogg*) It is a very difficult question to answer. Some points are, in fact, picked up in the response, because, as I say, your Committee did make some criticism, to which we responded, and we did identify one or two areas where things might have been done differently. I do not believe they were in any way substantive in their impact. Looking at it broadly and over the whole period, I think we have responded fully and promptly to all the substantive recommendations made. That, as I say, was certainly the view of Sir Richard Southwood when he concluded at the end of his report that he welcomed the expedition with which the Department had acted.

42. I noticed, in fact, in *The Times* today a letter from the past Chairman of the NFU, where he indicates that if the Government had taken his advice at the time things may have been different today. I am going to ask you, Minister, for your comment on that, but would it help you to know, and you have seen our report, that in his evidence to the Agriculture Committee on 20 June 1990, Sir Simon Gourlay told the Chairman this: "As far as having faith in the Ministry of Agriculture is concerned, the farming community fully supports, and has complete confidence in, the measures that the Ministry of Agriculture, in conjunction with the Department of Health, have taken to safeguard the consumer so far as eating beef is concerned and as far as the safety of the meat is concerned." Bearing in mind the evidence given on that occasion, what comment have you on the criticism made by Sir Simon in this morning's *The Times*?

(*Mr Hogg*) I think, in a sense, Mr Alexander, you have answered the question that you have posed. I do not want in any way to be unduly critical of people who have changed their position—it is a perfectly fair thing to do with the benefit of hindsight—but you have laid the foundation for what I am asserting, that we acted in accordance with the best judgement at the time. I come back to the point that Sir Richard Southwood made in his conclusions of paragraph 9.2: "From present evidence it is likely that cattle will prove to be a dead-end host for the disease agent, and most unlikely that BSE will have any implications for human health." That was the best advice that we had at the time, and I think to have embarked on a policy which assumed a different assessment of the likelihood would have been very difficult to justify.

(*Mr Dorrell*) Can I add something to that, Chairman? One of the lines that has been pursued by some commentators in the last few days is that the development of the BSE condition, traced as it is back to foodstuffs, can be attributed to deregulatory moves in the early 1980s. I do think it is very important to answer that charge directly. The position is that there was no deregulation of the foodstuffs industry that affects this case in the early 1980s. Miss Harman, in her contribution on this subject on the floor of the House, alleged that the out-going Labour Party had put in place, or planned to put in place, regulatory steps that would have prevented the development of this condition in animal foodstuffs. That is not true. We have been back through the relevant papers. The position is that a proposal was indeed under discussion at the time of the 1979 General Election. It was not directed at the control of BSE or any similar condition, because that was not known at the time to be a threat; it was actually directed at the control of salmonella. So it was not directed at this problem, and nor would it have had the by-product of dealing with this

[**Mr Alexander** *Cont*]

problem because the terms of that regulation—had it been imposed and it was not imposed—would not have had the by-product of regulating the industry in such a way as to prevent the development of this disease. What happened during the 1970s and 1980s was a change in the processes of the rendering industry, driven by changing practices within the industry and, ironically, also driven by a desire to improve the safety standards of people working in the industry. It took place against a background of no known threat of this type of condition emerging.

(*Mr Hogg*) Perhaps I might add, Chairman, that this particular issue, the causes of BSE, were analysed by Sir Richard Southwood in his report in February 1989. You will find the relevant parts on page 11, paragraph 4.2.8. I quote from part of that paragraph. "The introduction of continuous rendering processes during the 1970s and the 1980s, which may have resulted in the rendering of animal material at a lower temperature and/or for less time than previously, and the decline in the practice of using hydrocarbon salves for fat extraction since the mid-1970s ... ". There was nothing here about deregulation. Indeed, the first regulation that was made relevant to protein was made in 1981. The Labour Party, which was the government of the day, was concerned about salmonella, which was a different proposition entirely.

Chairman: I recognise that this Committee is not familiar with our former inquiry. We have done our best to obtain copies of that report, but time has not entirely permitted. There are one or two Members here who were on the previous Committee at that time.

Mr Pickthall

43. Referring, Chairman, to that report, which the Minister has prayed-in-aid, one of its recommendations was for the random testing for BSE in routinely slaughtered cattle—which was also the recommendation of Tyrrell. In retrospect, what does the Minister think of that decision at the time?

(*Mr Hogg*) It is, in fact, in the Tyrrell Report. There was a recommendation in that Report on page 10. The recommendation is that although there is no firm evidence for evasion of compulsory notification and slaughter of BSE suspect cattle, the formal study of cattle presented as acceptable for human consumption would provide reassurance and might even reveal spongiform changes in animals with atypical, sub-clinical infection that has not been recognised. That was then addressed by your Committee in 1990, paragraph 69, which made the point that there was no proven diagnostic test for BSE in live animals; the disease could only be detected in three ways, and I am paraphrasing here, including the post mortem microscopic examination of the brain tissues. They then went to comment on the Tyrrell Report recommendation to which I have just referred. "This form of research was not accorded high priority by the Tyrrell Committee because of its heavy demand on technical resources, although the Committee did recommend it having some value. We believe the Minister is correct to adopt the research priorities recommended by the Tyrrell Committee, but trust that he will give due consideration to this proposal when improved diagnostic tests become available". We commented on that, in paragraph 29 of our response, where we make the point, at that time, that random testing in abattoirs would make no difference to public health safeguards since the offal ban removed from the human food chain those tissues which may contain significant amounts of the agent in infected animals. We had, at that stage, put in the offal ban. Moreover, in respect of the tests on brains, because the changes in the brain only take place very close to the period when the clinical condition is apparent, it is not in itself an effective way of determining the number of sub-clinical animals that might be going through these slaughter houses. That is a summary.

44. Do you not think that a common-sense precaution in a situation where the disease, its providence, its extent and so forth was not certain, and it still is uncertain now, would have been a system of random testing of animals at slaughter houses? It would seem to the ordinary man and woman in the street the most common-sense and straightforward thing the Government should have insisted upon.

(*Professor Pattison*) It is impossible for me, really, to go back and have the same experience then as the Chairman now has, and I have only been Chairman since 1 November last year. There are two issues that, of course, one might also draw to your attention. The first is that some research was commissioned at the Central Veterinary Laboratory (and it is still on-going), and we had a report about that at a seminar in Warwick about ten days ago. The raw material was urine, which, of course, would give a very convenient sample, but it has only got to the stage where it is as accurate as the ability of a vet to look at a slightly sick animal and decide whether or not it has got BSE. There is still a long way to go to get an operational test. The other problem now is that the rarer the event you are looking for the greater the sample you have to take in order to find what the answer is. So we are now faced with that problem. We need a very sensitive and specific test, otherwise, potentially, sampling and testing may do more harm than good.

45. How long do we have to wait before we get this sensitive test?

(*Professor Pattison*) Dr Will referred to one, potentially, for cattle and for humans. There is, at least, an interesting development that workers in America have, and, as the Chief Veterinary Officer said, cattle samples have been supplied to them, but I do not believe we have any results back yet.

Mr Jones

46. Can I go back to our report—and I was a Member of the Committee at the time and I disagreed with the recommendation over Tyrrell, but there we go, it was an all-party report. Bearing in mind that the Government said all along they were taking scientific advice, not taking the advice of Tyrrell of sampling in slaughter houses cattle infected by BSE to get some idea, I think, was a mistake. Never mind. In paragraph 13 of our report it says: "It is therefore likely that sub-clinical animals are being sent for slaughter", which is referring directly to the

[**Mr Jones** *Cont*]
period that I am talking about. My initial question was about the four years when compensation and the arrangements for notification and so on were not in place. I think beef is safe now, but I still think that the consumer in the country for four years was exposed unnecessarily because of Government delay.

(*Mr Hogg*) I think the question is, why did we take so long in introducing an offal ban. If I might say that perhaps the most convenient way of looking at that is to look at the very extensive reply we gave in paragraphs 43 to 48 of our response. Clearly I can read it all out, but I do not suppose the Committee wants me to do that because we have already set out our reasons for that in detail.

(*Mr Meldrum*) Can I add two points? One is I do not think we should forget that in August 1988 we were, at that time, removing from the human food chain any clinically affected cattle. They were destroyed by incineration. That was the first step that was taken as a result of the Southwood Committee recommendations. The second point I would make is about the sub-clinically affected cattle. We said, and the Ministry said, in June 1989 that we anticipated that some sub-clinically affected cattle might enter the human food chain, and that was one reason why the Government at the time brought in the Specified Bovine Offal Ban that, in many respects, went further than the Southwood Committee recommended.

Mr Congdon

47. Can I go back, Chairman, to something both Secretaries of State said, which was, I think, of importance to the general public: that the problem probably occurred because of a change in the processing of meat. Can the Secretaries of State confirm when animal feed was first fed to cattle. I looked back in the earlier Select Committee report, and a previous Minister of State says it goes back to the Romans! I am not bothered whether it goes back as long ago as that, but does it go at least back to the First World War? Could the Minister confirm the point that it was the rendering process that was the significant change and that that was introduced ironically for health and safety reasons for the workers concerned? Could that be confirmed?

(*Mr Meldrum*) So far as I am aware, meat and bonemeal derived from rendered waste from the abattoir industry has been a constituent of animal feed for a very long period of time and certainly was when I was a student at university. It may well go back as long as the First World War, but the earlier systems were quite, quite different to the systems that were introduced in the 1970s. The earlier systems were on a batch processing system with a known time and temperature to which the material was exposed. For reasons of quality, the introduction took place in the early 1970s of so-called continuous rendering systems. Some of these systems that were introduced in the early 1970s, we now know on the basis of information from the inactivation studies that have been carried out, were ineffective in destroying the agent of BSE. That is why certain further amendments were made last year to the processing standards that we require throughout the whole of the Union. I might add of course that we took—we, the ministry—action in July 1988 to ban the feeding of ruminant protein to ruminants on the assumption that, first of all, the cause was in the feed and, secondly, we were uncertain about the effect of current systems on the inactivation of both BSE and scrapie.

Mr Campbell-Savours

48. Can I ask you, Mr Hogg, to appoint a civil servant to answer my question? When you answered a colleague before on compensation, you referred to the government's reply to the 1990 Select Committee report. I do not want to ask about compensation that arose before that date. I want to ask about compensation which arose in 1994 and changes to compensation. Dr Latto, the Chairman of the Board of Governors of the British Safety Council said about those changes in compensation which led to reduced payments to farmers: "There is no doubt that the change of policy has contributed to a much higher incidence of BSE than would otherwise be the case." That is arising out of the reduced levels of compensation payable in 1994. Can I have a civil servant, and not a Minister, confirm that that is the case?

(*Mr Eddy*) It is certainly true that the arrangements were changed in 1994. The original compensation was based on the market value of a prime dairy cow. As a result of the success of the ruminant feed ban which was introduced in 1988, a growing proportion of the cases were older cattle which, at the end of their milking life, as I am sure you appreciate, have a much lower value because they can only be salvaged for meat. The arrangements were therefore over-compensated because they were paying the prime dairy cow price for a population which consisted of a significant number of older cattle.

49. I am not querying whether they were over-compensated. What I want to know is: did the change in compensation regime, for whatever reason, which led to reduced payments for farmers, lead to a higher incidence of BSE which has been asserted by Dr Latto? The answer to that is either yes or no because, if it is no in your view, then we will follow it up in later evidence.

(*Mr Hogg*) With respect, Mr Eddy must be allowed to answer the question as fully and as openly as he can. Yes or no answers are not always appropriate.

Chairman

50. I think there may have been some confusion in the question. Mr Eddy, will you continue?

(*Mr Eddy*) The short answer is that the compensation price now reflects the value of the kind of animal on which compensation is being paid. Those animals include a high proportion of lower value cull cows and that is reflected in the compensation price. On that basis, I see no reason why one would think that Dr Latto's comments were correct. That is a civil servant's way of saying, "No".

Alice Mahon

51. I would like to challenge the statement by the Minister when he said that ironically changes in the rendering techniques were due to improved health and safety. In the debate in May 1990, Dr David Clark MP, who was then Shadow Minister for Agriculture, brought out just this very thing, referring to the deregulation of the industry when the Conservative Government was elected. He quoted the chairman of the United Kingdom Renderers who said, "The original proposals"—that is the Labour Government proposals—"were very expensive but there was a distinct change of heart when the Conservatives came into office. They were happy to drop the idea of a code and settle for random testing." An executive member, Mr Field, went a stage further when referring to the different technology permitted when the weakened regulations came in and he said, "This was partly as a result of changes in animal feed techniques but the basic motive was profit", so I put it to you that the deregulation was to allow the industry to maximise profits and had absolutely nothing to do with improved health and safety.

(*Mr Hogg*) With respect, this is simply wrong and I am, in a moment with your permission Chairman, going to ask the Chief Veterinary Officer, who was in the Department at the time associated with these matters, to go into somewhat greater detail. In the first place, there was no deregulation. There was not a regulation in place when the government was in office. What there were at the time were draft proposals directed to the question of salmonella, a wholly different condition.

52. We did not know about BSE then, did we?

(*Mr Hogg*) I am endeavouring to answer the question that has been asked of me. Moreover, what they contemplated in the draft regulations would have had no impact whatsoever upon the agent that we now know may have existed, so the broad answer to the question, Chairman, is that the lady is wrong. However, the more detailed answer will come from the Chief Veterinary Officer.

(*Mr Meldrum*) I was involved in policy advice at that time, so I was involved in those discussions. I do know that proposals were in fact put out for consultation to produce protein of a standard to ensure, so far as possible, that it was free of salmonella. One proposal would have laid down specific time and temperature combinations for each particular plant to ensure that the material was free of salmonella. No regulations were made. I can confirm that. I can also confirm, from memory, that the time and temperatures that we were then talking about that might have been incorporated into the protocol for an individual plant would not have been sufficient to destroy the agent of scrapie or BSE, partly because these are continuous production plants. They actually are producing protein of a very high quality from the point of view of amino acids, although of course we now know that they can also contain the agent of scrapie or indeed BSE.

Mr Marshall

53. My constituents tell me that the United Kingdom is not the only country to employ intensive farming methods. Therefore, they are surprised that the United Kingdom is the only country which apparently seems to suffer from BSE on a significant scale. Is this because we are the only country to have used animal protein in cattle feed? Is it, as is suggested by those who are normally far removed from the food production industry, because our industry is uniquely deregulated or is it because, in other countries, perhaps they suffer from BSE but they are not very good at reporting it? There is a suggestion in *The Telegraph*, last Saturday, as my colleague Mr Powell said, that in Belgium they actually suffer from contaminated meat but do not tell us about it and it is suggested in *The Times* today that the meat in Italy has been well and truly contaminated by the Mafia and when the Mafia contaminate things they do it on a good scale and people suffer. Are we not in danger of the British industry being sold down the river by individuals who try and pretend there is a problem in this country and no problem anywhere else in Europe or in the world?

(*Mr Hogg*) I think maybe, because it is a somewhat technical question, it is best addressed in the first instance by the Chief Veterinary Officer and then maybe Professor Pattison might wish to amplify the point.

(*Mr Meldrum*) It is true to say that there are a number of countries in Europe that have been reporting cases of BSE, and have been for some time, outside the British Isles. The extent to which there is under-reporting is hard to determine, if there is any at all, but I think what it is fair to say is that the problem in the United Kingdom is, by whatever measure you use, significantly greater than in any other country. In particular, the other two countries with a significant number of cases are the Republic of Ireland and Switzerland. Switzerland have had, so far, 206 confirmed cases, so far as I am aware, and the Republic of Ireland about 120. All Member States are now aware of the encephalopathies but there is bound to be an ascertainment issue. The more aware you are of the encephalopathies, the harder the authorities or veterinarians look and therefore the more reports you are likely to find. I do believe that the few cases of encephalopathy in cats are simply and solely due to awareness by the veterinary profession here in the UK of the encephalopathies and they look very carefully, when they are carrying out a differential diagnosis, to see whether that particular cat might be affected by FSE.

(*Professor Pattison*) I do not think there is anything really to add to that, except to say that Dr Kimberlin points out to me that there are four essential issues. It is not just meat and bonemeal. It is likely that you need sheep. That is one. You need sheep with scrapie. That is another one. You need the amount that is fed to cattle. That is the third, and you need the problems of the rendering process, which is the fourth. All of those things have to come together to create the sort of epidemic in cattle that we have had in the UK.

[**Mr Marshall** *Cont*]

54. Can I go back to part of my question which was: is the UK food industry uniquely deregulated? My experience is of going round food processing plants where one senses that the degree of regulation and control is very great indeed and the standard of hygiene is very high. Can the Minister tell us whether our industry is uniquely deregulated, as is normally suggested by individuals who know relatively little about it?

(*Mr Hogg*) My impression is that the industry would say that it was very heavily regulated. My impression, going round, for example, slaughter houses, is of the weight of supervision and control in place. I would not accept that it was uniquely deregulated. It is in fact heavily regulated.

Mrs Winterton

55. May I ask whether the epidemic in cattle has responded as the scientists have predicted and, if it has, whether you have seen that being reported in the media at all?

(*Mr Hogg*) The numbers of BSE confirmed cases have been steadily falling but they have not been falling as fast as we would have predicted eight or nine years ago. It is certainly not as fast as we would have wished, but the numbers are coming down substantially. They are at a weekly rate of around 250 to 300 as contrasted with about 1,000 at the high point. Those are confirmed cases.

56. It would appear to me that the damage to confidence in British beef has been predominantly caused by the hype and the hysteria of reporting in the media. One of the claims being pursued in print by a professor with a scientific qualification from Leeds is that land itself is contaminated and that is how the transmission of BSE takes place. Could I have a definitive response from the scientists on this point?

(*Professor Pattison*) The short answer is there is no evidence of that whatsoever.

57. Does that not actually bear out what I have just said, that credibility is being given to people putting forward pet theories with no scientific basis to back them up which is actually causing the hysteria etc., and the problems with British beef? Has the media not a major part to play in putting the right story forward before the British people?

(*Professor Pattison*) I find it extraordinarily difficult to answer that question.

Chairman: I think we probably all do.

Alice Mahon

58. I want to just speak about critics of government policy. Whilst we are here as politicians, I have to say, speaking as a mother and grandmother, my emotions have ranged over the past week through all kinds of things and I have ended up feeling extremely angry. I think I reflect many of my constituents in that, because I think that maybe my family has been put at risk. Can I ask what the government's attitude is now to theories related to BSE and the criticisms of government policy and research priorities put forward by such scientists as Professor Lacey, who was referred to, who advocated, I believe, wholesale slaughter policies and warned of dire human health consequences in the past from BSE? He was ridiculed a great deal. There was Dr Stephen Dealler, who has argued that the dangers of beef eating have been consistently understated. Could I come back to Dr Harash Narang, who claims that the funding for research on a live test on cows which he was developing was unjustifiably denied? Indeed, he was made redundant. We know that research funding was held from him. Even worse, I understand he has been subjected to a reign of terror in his own home. I would like comments from the scientists please, when it comes to the other scientists; they are probably better equipped to give detail than politicians.

(*Mr Dorrell*) The first question was directed to the government which is made up of politicians, so perhaps I can answer that and then pass it back to Dr Will on the subject of Dr Narang. As far as the government's overall position on the various scientific theories that have been around, I and my colleagues at the Ministry of Agriculture have at no stage in the arguments over the last week sought to rubbish any person putting forward a scientific argument. The consistent position of the government has been that anybody who has any scientific argument to make about the causes, the diagnosis or the treatment either of BSE or of human conditions, should put their evidence to those who are in a position to advise the government on the proper, political response to it. That is why I have said to Dr Dealler and I said to Professor Lacey on a television programme that it is for them to put their evidence in support of their theories to the advisory committee that is chaired by Professor Pattison. I have no doubt whatever that is the right way to compare competing scientific explanations with what we can all agree is an extremely worrying condition. In the case of Dr Narang, as Ms Mahon has referred to, his employment with the PHLS has been brought to an end. The reason why his employment with the PHLS has been brought to an end has been stated many times in the media. [1]That is because he was conducting a private research effort and not conducting himself as an employee of the PHLS in accordance with his contract of employment. He has, however, made it clear that he thinks he has some tests that would be of value to those responsible for diagnosing both BSE and CJD. My private secretary wrote to Dr Narang on 29 January inviting him to put forward the tests which he is developing and we are still waiting for that evidence to come forward. I do not know if Dr Will has anything to add. Dr Will has actually had contact with Dr Narang, as he referred to earlier on. What I hope we can develop with Dr Narang is a dialogue based on the science and assessment of the test rather than exchanges in the newspapers.

(*Dr Will*) One thing that may be often forgotten is that the issue of whether or not BSE was likely to be a risk to man has been discussed by many scientists throughout the world in many different committees,

[1]Note by witness: In fact, contrary to reports in the media, Dr Narang was made redundant. In addition he was conducting a private research effort and not conducting himself as an employee of the PHLS in accordance with his contract of employment.

[**Alice Mahon** *Cont*]

meetings etc., including the World Health Organisation on three occasions, when all these issues have been gone into in great detail. On each occasion, the conclusion of the WHO meeting has been that the measures that are being taken are adequate to protect human health. One final thing I might say is that, if these ten cases are related to BSE in a causal way, that does not necessarily indicate any connection with eating beef. By far the most likely explanation in relation to the incubation period of these diseases is that the cases, if they were related to BSE, were related to some exposure in the 1980s at a stage when there was no specified bovine offals ban. Therefore, to me, it seemed the most likely explanation was that the SBO ban has been incredibly important in protecting human health since 1989 and also currently.

(*Sir Kenneth Calman*) You specifically referred to one or two scientists who have been commenting. First of all, for those of us who have worked outside the Civil Service, it is not at all unusual, in whatever field you work, to have colleagues who are stimulating and interesting and put forward a wide range of theories, but as Dr Will has said, this has been the subject of intensive discussion across the world. It would be a pity if all the comments referred to two or three people. We have had wide ranging discussions with experts everywhere and that really is very important to say. If people do have information, new ideas and advice, then I am delighted to get them. I got a letter this morning from somebody who gave me, I thought, quite an interesting bit of information, but we will regularly take evidence, advice and information from any scientist out there because you, I and everybody in this room want to get to the bottom of this matter as rapidly as possible.

59. What research into live tests has been conducted over the last ten years for the disease in cattle or humans and what are the prospects for developing such a test?

(*Professor Pattison*) I think that we have substantially answered that, unless you want the particular detail which I think would be best in the form of a written answer. There has been MAFF sponsored research at the Central Veterinary Laboratory into a urine based test and that was the one I referred to earlier. The most limiting aspect that we have had in terms of the sort of tests that you would normally use in man or in animals is the lack of a highly specific monoclonal antibody to the abnormal protein. We have to distinguish the abnormal from the normal protein.

Chairman

60. Perhaps we had better have a written answer.

(*Professor Pattison*) May I just say one thing about Professor Lacey and Drs Dealler and Narang, because I think it is very important? I can assure you that, in my time on the committee—and I am sure it was the case beforehand—nobody's opinion has been dismissed or ignored. Professor Lacey I have known for a long time and much of what he says is within the bounds of possibility. Frankly, there are things that he says that are wrong. He has never written to me whilst I have been chairman of the committee and I am not aware that he wrote before that. Dr Stephen Dealler has done a lot of work and published extensively. He and I are in correspondence and he writes to me approximately once every two weeks. I will have to apologise for not having replied to his recent letters, but I simply have not had time. We do actually take that information very seriously indeed. The same with Dr Narang. If he has got a test that is worth looking at and applying, we would dearly love to know what it is, but he has to tell us.

Mr Marshall

61. It is very interesting that Professor Lacey is too busy forecasting Armageddon on television to write to the Committee with his evidence. Can I ask the Minister, in respect of the suggested wholesale slaughter ban put forward by Professor Lacey, would he confirm that that would lead to the decimation of the British dairy herd, which would mean that we would have to import milk to sell in supermarkets? Many British customers would feel that that was not a bonus for British health or British hygiene. It would also mean that we would be importing a large quantity of dairy products that are currently made in the United Kingdom with a consequential impact both on the balance of payments and employment in certain rural communities.

(*Mr Hogg*) A wholesale slaughter policy would indeed have all the consequences that Mr Marshall has identified. Moreover, there is no scientific justification for a wholesale slaughter policy.

Mr Bayley

62. Government ministers in the last week have been at pains to point out that your policy decisions are based on sound science. Because of a lack of confidence amongst the public as a whole on this issue, there is a widespread fear—and this is no reflection on the members of SEAC who are all extremely eminent scientists—that the government is listening to the science that it wants to listen to and not listening to other opinion and the fact that some of the Southwood and Tyrrell recommendations were not implemented lends credibility to that. Over the weekend, I had an opportunity to get advice from Sir Bernard Tomlinson, who, as you know, has a number of reservations about whether the government's policies go far enough in terms of administrative decisions, following on from the clinical evidence. Would it not help to restore public confidence if some people who have taken a more cautious line than the government has taken—a more precautionary line, perhaps I should say, than the government has taken—were added to the membership of SEAC to reassure people that the widest range of sound and well informed scientific opinion is being listened to? If some of those people are not specialists in the field of these diseases, nevertheless, I could see some sense in that because science has a tradition of peer review and having some people with experience in connected fields might be a useful scientific test bed for the ideas to be tested out against.

[**Mr Bayley** *Cont*]

(*Mr Dorrell*) Mr Bayley is disarmingly mild in the way that he puts his case, but the truth is that, while he started out by saying that he did not challenge the efficacy of the independence and value of SEAC advice, that is precisely what he has just done, because you cannot establish the membership of a committee on the basis that you go on enlarging the committee every time a point of view is put forward which is not endorsed by the committee. That is the only real way in which it would be possible to respond to Mr Bayley's approach. Sir Bernard Tomlinson is, of course, an extremely eminent man and he has expressed concerns, primarily I believe, around the possibility that infectivity can be harboured within the liver. That is a proposition which SEAC examined, as I understand it, specifically and on which I will, if I may, ask Professor Pattison to respond.

(*Professor Pattison*) In reality of course the committee was doubled in size last November and I have to say, as chairman, it is still controllable, but it is getting close to being difficult.

Chairman

63. I have some sympathy for you!

(*Professor Pattison*) The increase was very important and it does I think broaden out the number of scientists that are there and the perspective from which they approach this problem. Secondly, of course, those scientists do not simply stop thinking about this issue and start thinking about it at the end and the beginning of a meeting. I know for a fact that myself, Dr Will, Dr Kimberlin, certainly Professor Collinge and certainly Professor Almond talk about the issues with many of their colleagues, so they bring to the committee quite a broad spread of opinion. I think that, on the issue of Sir Bernard Tomlinson, Mrs Browning invited him to write to me at the end of last year. I had met Sir Bernard over other issues around London health and medical schools. I was delighted to hear from him because he is an eminent person and a very wise man. We exchanged long letters before Christmas and the last one is from myself to Sir Bernard. It does come down essentially to his anxiety about liver. When we met at the weekend, we discussed all the non-banned offals including liver and again reached the conclusion that it was not necessary to further extend the offals in the ban.

Mr Bayley

64. Given that you have a wide range of expertise on the committee, are there ever occasions when, either on the clinical science before you, or on the administrative policy that needs to follow from that in terms of regulation of abattoirs, bits of animals that are not to be released, there are disagreements amongst members? Do you end up with, in effect, a majority decision or some members with reservations? Are those individuals who have reservations or who have reached different conclusions on either matter, clinical or administrative action, permitted to voice those opinions and publish those opinions, even though they might contradict with the collective responsibility of SEAC as a whole?

(*Professor Pattison*) There is quite a long answer to that and it is such an important question I wonder if I may take the time to answer it. I think there is a difference between the use of the word "disagreement" and the very necessary process that must take place in the committee of exploring every aspect of every question. There are times when individuals, and perhaps the chairman himself, will deliberately actually say, "Why do we not consider so and so?" or "Why do we not do such and such?" simply to draw that into the debate so that we consider it and then come to a conclusion about it. That is the essence of debate and the way committees work. What we end up with is not a compromise; it is the view of the committee members present that that is the conclusion that they come to, having widely discussed all the issues.

(*Sir Kenneth Calman*) As the Government's Chief Medical Officer, one of my functions of course is to bring to Government and Ministers a very wide range of advice, advice that may well go beyond SEAC. Indeed, I would be failing in my duty if I did not do so. At the last SEAC meeting, indeed, we did bring in some experts outwith the committee to deal with very specific issues. That must be the function. We must use any expert in this country or beyond who will give additional information on this or indeed on any other matter.

Mr Campbell-Savours

65. Can I ask you, Sir Kenneth and also Dr Will, whether you regard the work of Dr Harash Narang as relevant?

(*Sir Kenneth Calman*) First of all, if I can respond to a question which was asked a little earlier, I have never had a letter from Professor Lacey, for example.

66. I am asking you about Dr Narang.

(*Sir Kenneth Calman*) I know precisely what you are asking me about. I have never had a letter from Professor Lacey. I have had a letter from Stephen Dealler and we have corresponded. I do not think I have ever had a letter from Dr Narang. I have tried, through Dr Will and indeed others, to find out more about the test. I would be delighted to have some information from Dr Narang to actually answer your question.

(*Dr Will*) I think we should listen to any scientist or anything that someone suggests might be relevant. However, I have to say that an assessment of the possible relevance depends upon an assessment of the scientific basis of a thesis or test or whatever. People have been looking for a conventional virus in these diseases for 25 years without finding anything. If it is now suggested that a conventional virus is visible in urine, it is certainly something that we should explore, but, in my view, it is exceedingly unlikely it would lead to a diagnostic test.

67. Do you see this box I have here? Half the papers in it—and it is full of papers—are correspondence between Dr Narang and the Department and ministers over the last three years on his activities. I find it quite remarkable that Sir

[Mr Campbell-Savours *Cont*]
Kenneth does not even appear to know the detail of the areas which he is working in. I want to ask a very simple question: why is it that Ken Bell International, a company in Newcastle upon Tyne, has had to fund the activities of this man who is working in an area of critical importance, an area that would certainly be supported by a majority of the general public? I want to read to you a letter dated 1 September 1994 signed by Ken Bell International to David Clark MP, a Labour Shadow Secretary. He says: "You remember five years ago, at your instigation, I"—that is to say, Ken Bell International—"began to fund Dr Narang's research into BSE. For the last four years his employers at PHLS have banned any further research in this country or his acceptance of my funding, having been suspended all this time pending a disciplinary tribunal to face unspecified charges. Fortunately, at considerable inconvenience and expense working during vacation at Stoney Brook, New York, he has slowly been able to produce a post mortem test on the basis of the live test." He then goes on basically to complain about the treatment of this man. I want to know, my constituents want to know and I think the public are entitled to know why this man was not funded by taxpayers' money when he was working in an area which is now critical in the debate that is currently taking place. Indeed, he is not even being funded by the taxpayer now.

(*Mr Dorrell*) The simple answer on the question of Narang is contained in two sentences which, if I may, I will read from the letter written to him by my private secretary on 29 January of this year. It says: "The Government fully appreciates the seriousness of CJD. It is a rare disease and were a validated test to become available it would overcome current disgnostic difficulties. It was for this reason that SEAC wrote to you on 4 December seeking details of your urine test. The Committee still wishes to give the Government an independent assessment of its clinical robustness and would welcome receiving details necessary for this." That, as I understand it, remains the position.

68. Can I put it to you finally that what has actually happened is that the government have relied on what they believe to be declining statistics of the incidence of BSE and they have set out to deliberately obstruct work in this vitally important area because they did not want people at any stage in the future walking round abattoirs doing live tests on animals, upsetting public opinion and undermining the market for beef. There has been effectively within the Department a conspiracy to undermine work that is being done which was of critical importance because it did not suit the ministers to see that work being done.

(*Mr Dorrell*) It is a grotesque misrepresentation of the government's position to suggest that of the Department of Health and I am sure exactly the same thing is true of the Ministry of Agriculture, Fisheries and Food. Mr Hogg will reply on their behalf. It is grotesque to suggest that the Department of Health, responsible as it is for public health policy in Britain, should not be interested—

69. MAFF.

(*Mr Dorrell*)—in pursuing any course that is going to deliver better understanding of a threat to human health.

70. MAFF.

(*Mr Dorrell*) That is simply not true.

(*Mr Hogg*) I entirely agree with what Stephen Dorrell has just said. Obviously, I have a duty to British agriculture. Everybody knows that, but I have an over-arching, paramount duty which is the safety of human folk. That is my paramount duty and there is no way that I or any of my officials would act in the way that Mr Campbell-Savours has just suggested.

(*Sir Kenneth Calman*) If I may respond as the Chief Medical Officer for the Government, I find the statement made by Mr Campbell-Savours disturbing, there is no evidence whatsoever for that statement and I have stated very clearly that, if Dr Narang has information which he wishes to give—I have not seen all of these letters which you report there—if there is further information which he wishes to give on the test, we would be delighted to receive that information.

Mr Powell

71. There is no evidence whatsoever, you say, for the allegations made?

(*Mr Meldrum*) Can I be more specific on this issue, since the allegation has been made against MAFF? There are three specific areas concerned with Dr Narang's work. The first was he developed, he believed, a touch test. This was on touching a cut surface of the brain, post mortem. We worked with him on that. We provided samples for him, for his tests. That information is now in the public domain. Secondly and later on, he developed what he calls a diagnostic test on the nemavirus theory that he had put forward. We worked with Dr Harash Narang on that particular project. We put money into a second experiment to replicate in detail exactly what Dr Narang had in fact put forward. It was done by a totally independent group, independently audited outside MAFF and I believe the results of that particular work will be published very shortly. Thirdly, there is the recent urine test put forward by Dr Harash Narang about which we have no details. I can make it absolutely clear, as my Minister has done, that if we could find a diagnostic test that we could use in the living animal I would be absolutely delighted.

Audrey Wise

72. There was an answer given to my colleague, Hugh Bayley, about the position of agreement and disagreement within SEAC. Since we are still on this question of possible dissidents, I want to say that I found that answer less than complete. Professor Pattison outlined the kind of discussions which go on on all committees, trying to arrive at consensus, testing out different ideas, but what he did not say and what I would like him to say is what happens if there is not a consensus, or is he telling us that there has always been a consensus on every proposal, clinical and administrative? If there is not always a consensus, what is the position of those who disagree? Can they state their disagreement publicly

[**Audrey Wise** *Cont*]
or would they have to resign from the committee and would they in any case be bound by any secrecy provisions?

(*Professor Pattison*) I can say quite simply we have never been close to the latter scenario at all, but I emphasise in my time on the committee and my chairmanship from 1 November last year. I will be quite open with you: last weekend when we met, we knew that we had to produce advice for government. The committee broke up on Sunday evening and I was to take that advice to government the next morning. I asked the committee, because I did not know what government would do with that advice, to wait until they heard what the government said and after that they were perfectly at liberty to talk as individuals to any member of the press or the media. There has never been any attempt whatsoever to limit anybody's ability to talk to anybody else.

Mr Leigh

73. You have given me permission to jump ahead a little because this is a vital question. Because this is a vital question, I think we should deal with it before lunch. We can always return to it after lunch if we do not get a satisfactory answer. Mr Marshall has already referred to it. It is the question of slaughter policy. We have spent now two hours and we have had a very interesting discussion on clinical matters, but we are faced with a crisis of confidence in a great British industry. We have to restore public confidence and we have to investigate how we are going to do it. Therefore, I want to ask Mr Hogg about slaughter policy. After all, Mr Dorrell raised this question right at the very beginning, in his opening statement, and we have not returned to it. He seemed to suggest that the government maybe was in the process of changing its policy on slaughter. Mr Marshall really only asked half a question and he got half an answer. Mr Marshall asked whether we were going to slaughter the entire national herd and of course Mr Hogg very easily replied that there is no question of doing that because there is no possible scientific justification for doing so. The question I want to ask him is, what policy options the Government is considering to restore public confidence? What is the cost? What is the purpose of such policies? For instance, we have had various proposals which have been put forward, not least—which might be a very sensible one—that dairy cows coming to the end of their natural life could be slaughtered, bought in by the Government, so they could not enter the food chain. However, we are told that that might cost up to £500 million a year. I think we now need, before lunch and so that we can return to it after lunch, an answer from Mr Hogg on these vital questions.

(*Mr Hogg*) Chairman, there is no suggestion or recommendation from SEAC for any kind of policy which involves slaughter, and in particular because the phrase "slaughter policy" is attached to what has been described by some as "the NFU policy", though it is not strictly a slaughter policy. SEAC has not recommended to us to take out of the human food chain the older cow. The core of the NFU proposal, which has found echoes elsewhere, is that the older cow—the 30-month-old and above cow—should not enter the food chain. In the case, for example, of the dairy cow it should continue being used, for instance, as a milking cow, and when it has ended its milking career, instead of going into the human food chain it should be otherwise disposed of—slaughtered—at that stage. That is the core of the NFU proposal. SEAC has indeed considered the question of the older cow. What they have recommended—though Professor Pattison may wish to emphasise that exactly for the assistance of this Committee—is that in respect of the meat from the older cow it can be sold into the human food chain, but only in a de-boned state, the de-boning taking place in licensed premises and under the supervision of the Meat Hygiene Service. Therefore, SEAC has tried to address the same kind of problem and has come to a different conclusion. That is where the scientific evidence and recommendations rest. We also, of course, do have to consider consumer confidence. That is absolutely right, because if we could restore consumer confidence and tranquillity in the market that would address many of the problems that need to be addressed. To do that one has to consider what steps can be taken which would be reassuring to consumers, have the support of the market and buyers and also address the concerns that are obviously concerning the European Union, hence the decision which was made by the Standing Veterinary Committee. So we are now thinking whether there are any further steps, over and above that which we have been advised on the scientific evidence to adopt, which we should contemplate bringing forward. In the course of Government—indeed, as Mr Leigh knows, as he has been in Government—there are a range of options which are contemplated and identified, but I think it must be right for us to conclude what our own view is, before going public with our view. All I can say is that there is a problem of consumer confidence which I think may have to be addressed. That may involve doing things which the scientific evidence has not recommended, which the Scientific Advisory Committee has not recommended us to do, but which may become necessary in order to restore the consumers' confidence.

74. Yes, I understand that, Mr Hogg, but obviously if you do announce such a policy you would have to announce it on the floor of the House first, and you could not therefore share it with the Committee. I understand that. Also the point which you are making—and I am sure you could confirm whether I have got it right—is that SEAC may come to a view on how this meat is used, but they are not currently advising you to undertake any kind of slaughter policy. That is a very important point to get on the record that that is the scientific advice. But you seem to be telling us, and you are hinting very strongly—indeed, I think it is of vital importance in this debate—that although there is no scientific case for any kind of slaughter policy, there may well be a case in terms of restoring confidence in this industry, and it seems to me that really you cannot go any further than that. Or is that an unfair summary of what you are saying?

(*Mr Hogg*) What I would like to do, Chairman, is to confirm the position, then because I think the Committee might like to hear what Professor

[**Mr Leigh** *Cont*]
Pattison will say on the recommendations of the Scientific Committee, might I just ask him, when I have said what I have to say, either to agree or to disagree. We have not received any recommendation from SEAC to slaughter any cattle. In respect of the 30-month beast and above, the recommendation is a de-boning recommendation. So looking strictly at the recommendations from the Scientific Advisory Committee, we have no basis in a recommendation form for any kind of policy which results in slaughter. But Mr Leigh is right in saying that there are wider questions of consumer confidence, and of course there are wider questions which relate to the ban on our exports. Therefore, as the Prime Minister indicated yesterday, it is right that we should look at options to see whether there is anything further that we should do for the objective that I have just identified. If we conclude that there is, then we will be carrying forward that in the usual way, which I am sure would involve a statement to this House at some stage.

75. Before you ask your official to comment, though, it is very important

(*Mr Hogg*) Professor Pattison is not my official, he is the Chairman of SEAC.

76. Before you ask him to comment, I want to make this point to you. You obviously cannot tell us now what the outcome of those discussions is going to be, but can you not tell us—because you must have this information available to you—what are the costs of the various options and what are the various options that are being considered? You are not announcing a policy, but this Committee is concerned with the detail of this. Is it not possible for you to share this information with us?

(*Mr Hogg*) There are a range of policies that you could adopt. At the most extreme—I forget, Chairman, which Member raised the question, though I think it was Mr Marshall—at one end of the spectrum you could advocate wholesale slaughter. That has never been a policy recommended to us by anybody, but that is at one end of the spectrum. At the other end of the spectrum there would be a very much more focused policy between the two points in the spectrum. There are a number of points on which you could rest, each of which would have cost implications, implications in terms of numbers and consequences. With respect, Chairman, I do think that it is necessary for Government to go through the process of formulating any change that it wants to make and then come forward to justify it, and I do not think it would be helpful for me to embark on a lengthy explanation of what might be possible within the spectrum to which I have just been referring. I would like to ask Professor Pattison, if he might, to deal with the question of the Scientific Committee.

Chairman

77. I have Members pressing me for supplementaries, I have the Minister asking for other witnesses to contribute, and I am now running well past the time I said we would conclude this morning's session, so I am going to stop now. We shall be back in closed session at 2.45, and I hope that all the witnesses will be able to return at 3 o'clock.

(*Mr Hogg*) Chairman, there is one thing, if I may, which does concern me. Mr Leigh did ask whether we had received from the Scientific Committee any recommendations on slaughter policy. I have stated my position, but it may be that you would like now to hear from Professor Pattison on that point.

(*Professor Pattison*) It is very brief, Chairman. That is, the position is as the Minister has stated. We have given our advice. It is about de-boning, but our advice actually includes the phrase "provided all the measures that we have recommended now and in the past are fully implemented and sustainable". So that is one proviso. The second proviso is about public confidence, and really SEAC is not constituted to discuss issues of public confidence.

Chairman: Thank you. I am absolutely certain we shall be returning to the subject, indeed probably immediately after lunch. Thank you all for your answers.

WEDNESDAY 27 MARCH 1996

Members present:

AGRICULTURE COMMITTEE

Mr Richard Alexander
Mr D N Campbell-Savours
Mr Robin Corbett
Mr Ieuan Wyn Jones
Mr Martyn Jones
Mr Edward Leigh
Sir Roger Moate
Mr Colin Pickthall
Mr William Powell
Sir Jerry Wiggin
Mrs Ann Winterton

HEALTH COMMITTEE

Mr John Austin-Walker
Mr Hugh Bayley
Mr David Congdon
Alice Mahon
Mr John Marshall
Mrs Marion Roe
Mr Roger Sims
Rev Martin Smyth
Mr Richard Spring
Mr John Whittingdale
Audrey Wise

Sir Jerry Wiggin was called to the Chair

Examination of Witnesses

RT HON STEPHEN DORRELL, a Member of the House, Secretary of State for Health, RT HON DOUGLAS HOGG, QC, a Member of the House, Minister of Agriculture Fisheries and Food, SIR KENNETH CALMAN, KCB, (Grade 1A), Chief Medical Officer, Department of Health, MR KEITH MELDRUM, CB, (Grade 3), Chief Veterinary Officer, and MR TOM EDDY (GRADE 5), Head, Animal Health Disease Control Division, Ministry of Agriculture, Fisheries and Food and Secretary, SEAC, PROFESSOR JOHN PATTISON, Chairman, DR ROBERT WILL, Deputy Chairman, and DR RICHARD KIMBERLIN, Member, Spongiform Encephalopathy Advisory Committee (SEAC), were further examined.

Chairman: We are very grateful to the witnesses for returning, in particular to the Ministers who I know had to alter their diaries. It is most helpful, and we are most grateful. We would like to return to the subject we were on before lunch, which was the slaughter policy. I shall ask Mr Edward Leigh to start.

Mr Leigh

78. Mr Hogg, if I may return to the line of questioning I was putting to you just before lunch—and I hope you have had an extremely good lunch, and that you feel restored and happy to answer our questions—it seems to me that you will not tell us, understandably, what changes in the slaughter policy you are planning. I understand that, but what I find less easy to understand is that you will not even quantify the different options or describe what is going through your mind, although you must know perfectly well what are the various costs and options involved. In answer to Mr Powell's question about the risks of this disease, your scientists are totally unwilling or unable to quantify any kind of risk—1 in 1, 1 in 50, 1 in 1,000, 1 in 250,000, 1 in 10 million. Therefore, it seems to me they are giving few answers here in order to restore public confidence. I want to put this question to you again, because I think it is a vitally important one. You have—or rather your colleague Mr Dorrell has—dropped enormous hints today that the Government is thinking of changing its policy in order to restore confidence. You have made quite clear to us that there is not the remotest scientific justification for having any kind of slaughter policy. What I fear is that because these questions are not being answered today, what this Committee will be faced with is first of all an inability to do its job properly, secondly there will be a statement made to Parliament, presumably on some kind of slaughter policy, then up will jump Professor Lacey or one of our other friends who will say, "This is far too little too late," and therefore they will start undermining public confidence again and we will be back where we were. We will also end up with an open-ended policy which is costing this country billions of pounds. Therefore, I think we are entitled to some answers on these vitally important questions. What are the various options that the Government is considering on slaughter policy, the numbers of animals involved, the purpose of slaughter policy and the likely cost? Would you now like to answer those questions?

(*Mr Hogg*) Chairman, first of all, what I said before lunch was that there was no scientific

[Mr Leigh *Cont]*
recommendation from the Advisory Committee for any kind of slaughter policy or, for that matter, for any policy beyond that which the Secretary of State and I have outlined in our two statements—we each have made two statements, a total of four statements—on two occasions on which we have spoken to Parliament. What we are essentially addressing is a question of market confidence, and also we are concerned to get the ban imposed by the European Union lifted. The question that arises then is whether there are steps that we can and should take which would reassure the market and achieve the lifting of the ban. We are reflecting as to whether there are any such steps. You know as well as I do that there are a range of possibilities, but it seems to me that the proper thing for a Government to do, if it decides to come forward with any such proposals or such objectives, is to decide internally what it wishes to propose and then come forward with the proposal, and in the course of the debate which will then most certainly follow it will have to justify those decisions.

79. I am not asking you, Mr Hogg, to say what your final policy will be, I am just asking you to share with the Committee what are the various options that might be considered. It seems to me that by dropping these broad hints you are putting more and more pressure on, public debate will build up to fever pitch in favour of some kind of slaughter policy, whereas maybe one should be directing the public's attention to restoring confidence by discussing are the Commission justified in taking the action that they are taking? But we have had these hints, and you have not actually given any direct answers at all.

(*Mr Hogg*) I am not dropping broad hints. I am responding to your questions. I am not in the business of dropping broad hints. You have asked me certain questions to which I have tried to give an honest answer. We are reflecting on whether there are steps we would wish to bring forward with the object of achieving the two objectives which we have discussed. It is obvious that there are a range of options which one could adopt in general terms, and ultimately, if we decide to adopt any particular policy in addition to that which we have so far announced, we will have to announce it and justify it. That is the proper time, I would suggest, to do that.

Mr Wyn Jones

80. Can I ask you, Mr Hogg, this very basic question? You and your colleague have informed both the House and the media that you are pursuing and you have put forward certain proposals on the basis of the scientific advice given to you?

(*Mr Hogg*) That is correct, yes.

81. There is a suggestion in your reply today that you are prepared, in order to restore public confidence, to go beyond what might be advised to you by your scientific officers, is that right?

(*Mr Hogg*) That is what the Prime Minister suggested yesterday, and I am accepting that in order to restore public confidence it might be necessary to pursue a policy additional to that already announced, yes. That is the answer I have given.

82. My colleague Mr Leigh has put to you that the Government might be considering a range of options. The only realistic option which has been put forward hitherto, which has not been criticised profoundly, is the proposal put to you by Sir David Naish on behalf of the NFU. Will you tell us whether or not that policy is now seriously being considered by the Government?

(*Mr Hogg*) Chairman, that is a delicate and, of course, a very proper way of asking the question that Mr Leigh has asked in an even more challenging way, and I simply reply in the way that I have already replied to Mr Leigh. There are inevitably a range of policies. I know what Sir David Naish and the NFU have said, and we do reflect on all the possibilities, and go on reflecting until we have come to a conclusion which we will expect to have to defend to the House of Commons and elsewhere.

83. Can I put this to you, then? I understand your reluctance to be definitive, but I need to put this to you: that the advantage of the proposal put to you by Sir David Naish is that it commands not only the confidence of the farming community, but it also has the confidence of the retailers and the meat processors. Would you accept that?

(*Mr Hogg*) It was put to me on that basis. I had the pleasure, Chairman, of seeing Sir David Naish and indeed Mr Tom Vyner who is the Chief Executive of Sainsburys who came to see me, I think it was, on Monday night, to tell me of where they stood. I was extremely grateful to them for doing so. So I am aware of the support for that particular proposal.

84. Can I put this final point to you on this issue? There was a statement made on 20 March with the aim of seeking to persuade the consumer that eating beef was safe. There was a further statement made on 25 March with that aim again in mind, and subsequently I think it is all common ground that consumer confidence still needs to be reassured, we need to reassure the consumer about beef. The point I want to put to you, following on from Mr Leigh's question, is that every day that now goes on without the Government making an announcement sinks public confidence even lower. How long will we have to wait until the Government makes an announcement?

(*Mr Hogg*) I think you can accept, Chairman, that the Government is indeed very concerned about the state of the market (I am now talking about only the beef market), and the difficulties of the beef market flow from the lack of confidence on the part of the consumer. Therefore, it is, of course, true that with all possible speed the consumer should be reassured, and one has to see if there are measures that we can take properly to do that. Now Government, if they want to make proposals, must come to a conclusion and then advance them as soon as they reasonably can. You can be sure that Government is aware of the importance of the point that you are making.

[Mr Wyn Jones *Cont*]

85. Can you tell me whether you will expect an announcement in days rather than weeks?

(*Mr Hogg*) We are going to move as speedily as we can.

Mr Spring

86. Since we joined the Common Market I think our net contribution has been somewhere in the region of £40-50 million. Of course, the bulk of it, by definition, has gone into the Common Agricultural Policy. In the event of slaughter taking place, or in the event of any emergency affecting cattle, or chickens, or any kind of agricultural activity, is there a contingency reserve which is available within Europe, within the European Commission, actually to meet, at least in part, the costs of any expensive action that a member nation may have to take?

(*Mr Hogg*) I would put it slightly differently. There is a disease eradication fund, which is not a contingency fund, it is a specific fund. I think at the moment that is spoken for for the entirety of 1996. But if I can approach the problem from a different perspective, the European Commission and individual Commissioners have indicated that there are a range of measures that might attract EU support. Now that clearly follows anyway from the fact that intervention, private storage, assistance for the slaughter of male dairy calves, etcetera, are measures that are contemplated within the framework of the Common Agricultural Policy, and I certainly would expect the European Union, through those existing mechanisms, to make contributions to the cost of supporting the market in the way I have been describing, if that were necessary from our point of view and their point of view. So I do look to the European Union for financial assistance on the question of beef, if I can.

Mr Marshall

87. We have heard from the scientists that eating British beef is perfectly safe, and that the beef that is over 30 months old is safe provided it is de-boned. Those of us who are taxpayers would therefore find it strange if what is called healthy beef is then destroyed. There must be a danger that if this beef is destroyed, then the sceptics out there who sought to destroy confidence in the industry will say, "Well, we were right all along." Can I ask the Ministers—one or both of you, it does not matter which—whether you in fact intend to have some sort of advertising or publicity campaign to emphasise the points that have been made very eloquently by the scientists this morning, that in fact British beef is very safe indeed and that we can all eat it with complete certainty this coming weekend?

(*Mr Hogg*) We have said that on very many occasions, and I do not think that we at this moment have an advertising campaign in mind. I can imagine circumstances in which supermarkets and others will be very anxious to make that message.

Mr Bayley

88. Just a moment ago, Mr Hogg, you said that you do look to the European Union for financial assistance, did you not?

(*Mr Hogg*) That is correct.

89. Has the British Government made an application to the EU for financial assistance?

(*Mr Hogg*) We obviously have not at this moment put specific packages of measures, but we have been in discussion with the Commissioner and the Commission officials. For example, I talked to Commissioner Fischler yesterday, and officials from MAFF are in Brussels at the moment discussing possible ways forward which would involve Community money. For the avoidance of doubt, perhaps I might just re-emphasise that there are existing mechanisms within the Common Agricultural Policy for the support of the market, and it is in that context largely that the officials in Brussels are currently talking.

90. Are you therefore saying that the Government does not intend to make a special application for additional CAP support?

(*Mr Hogg*) No, that does not follow at all. I was just clarifying it so that you did not draw any false conclusions.

91. When might such an application for additional assistance be made?

(*Mr Hogg*) It depends on what measures may or may not be adopted. They are all part of one group of considerations.

Mr Whittingdale

92. Up until now the actions taken by the Government have clearly been based upon the scientific evidence given to the Government. If additional measures are taken, the kind of slaughter policy that we have been discussing, and this is not carried out on the basis of scientific evidence but in order to restore public confidence, is there not a danger that that will make it more difficult for us to criticise the measures taken by the European Commission for not having been based on scientific evidence, when they might also claim that their measures are necessary to restore the confidence of consumers across Europe?

(*Mr Hogg*) I see we are going to have a very interesting debate with a lot of people on those sorts of subjects!

Reverend Smyth

93. When we are speaking about European intervention and such like, what steps has the Government taken to emphasise the concept of a tracing system that is present in Northern Ireland where they can trace the herds as to where they are going, that the beef is remarkably safe, that the purchasers want to purchase it but, because the Government has made a decision, they cannot? Are we in a position to press for that type of

[Reverend Smyth *Cont*]
understanding which will go a long way towards restoring confidence in a sphere of the market where there is confidence that the beef is all right?

(*Mr Hogg*) In fact, Professor Pattison, Sir Kenneth and indeed the Chief Veterinary Officer have been in Brussels earlier this week and last week as well. It might be more appropriate for them to respond to that.

(*Mr Meldrum*) Perhaps I should kick off, Chairman, because I did attend the meetings of the Standing Veterinary Committee this week, on both Monday and Tuesday, and on both those occasions I was accompanied by Mr Ronnie Martin who is the Chief Veterinary Officer in Northern Ireland. So yes, of course we are very much aware of your situation in Northern Ireland, the number of cases, the fact that you do have a tracing system, and undoubtedly that will be borne in mind when we get into the next round of discussions with the Commission, bearing in mind their request in the Commission decision that was finally adopted yesterday afternoon when Article 4 says that the United Kingdom is invited to present further proposals to control BSE in the United Kingdom. That, of course, would involve proposals for Northern Ireland.

Chairman: Mr Spring, in the light of that, would you like to ask the questions you have been allocated earlier, in order to get the subject-matter out of the way.

Mr Spring

94. My constituents do not understand the role of the European Commission in all of this, very simply put. I would like to understand on what basis has the European Commission the right not only to ban the export of British beef within Europe, but actually to attempt to exercise a worldwide ban, when one considers the horrific consequences to the beef-producing sector of agriculture in this country? On what basis is this permissible?

(*Mr Hogg*) There are, I think, rather two questions there. One relates to the prohibition of exports beyond the European Union, and the other relates to prohibitions on exports within the European Union. We take the view that there is a legal challenge that could be mounted to both. I would certainly argue that it is very difficult indeed to see any compelling legal justification for a ban in regard to exports outside the European Union. Also within the European Union we think that there is a legal challenge to be mounted, but there are Directives on which the European Union and the Commission rest their defence, and it would be for the Court ultimately to decide whose interpretation of the law is to be preferred. Chairman, we get into extremely complicated territory here. If Mr Spring is asking for all the Directives and the language of the Directives which provide the basis for the legal argumentation it can, of course, be provided, but I could not conveniently give it to this Committee now.

95. I think we all draw comfort from the fact that you would consider a legal challenge, because it does seem to me to be extraordinary that it is possible for this particular body actually to ban our exports to any part of the world.

(*Mr Meldrum*) Could I comment on that one point? Just for the avoidance of doubt may I say that during the Standing Veterinary Committee on Monday afternoon the point was challenged that this Commission decision could legally cover prohibition on exports of beef to third countries. The Commission responded that they believed they did have the power, but I believe there are a number of experts who might take a different view.

96. Mr Meldrum, the whole issue is so critical to our economy and to our employment, in your discussions—and you, after all, were sitting with fellow professionals on that Standing Veterinary Committee and were giving advice to the European Commission—what, in essence, do you think, in your judgement, in talking with them, they require for either ban (a) or ban (b) to be lifted? Is it possible to give a succinct answer to that?

(*Mr Meldrum*) It is very difficult indeed, Chairman. The discussion first of all revolved around the discussions of the Scientific Veterinary Committee of the Commission that met last Friday, their recommendations to the Standing Veterinary Committee first of all, and then we moved on very quickly thereafter to a round-table discussion and a view from Member States on whether or not the bans that had been applied unilaterally should be applied and confirmed on a Community-wide basis. There was very little discussion positively about where we went from here, except that the Commission and the Member States were saying to the United Kingdom, "We will be very happy for you to come forward with further proposals to control BSE, and when you do we will convene a further meeting of the Standing Veterinary Committee to consider your proposals."

97. May I put one final question to Mr Hogg? On this question of a possible legal challenge, the wheels of the law grind slowly. If there were to be a legal challenge, in what sort of timeframe could this be mounted?

(*Mr Hogg*) To be realistic, Chairman, I think it would take a number of months. It is not an immediate solution to this problem, if I might answer Mr Spring's question in a slightly different way.

Mr Campbell-Savours

98. Mr Hogg, can I ask you two questions? I do not intend to press you on the options, but could you put a precise ticket on the NFU option? I am not asking you whether you wish to accept it or reject it or whatever, but could you give us an idea of how much that option would cost? That is my first question.

(*Mr Hogg*) If what we are talking about is the possibility of taking out of the human food chain all cattle over the age of 30 months, by which I mean not that they are slaughtered immediately but that they continue their ordinary working life—that is to say, dairy cows continue to be milked, beef cows continue to produce calves and bulls do their stuff—if you

[Mr Campbell-Savours *Cont*]

mean that, and they then have to be slaughtered at the end of their working career, the initial costs (and one would immediately think there might be others as well) would certainly be of the order of £550 million, but I would not be in the least surprised that there were considerable add-ons, and I am focusing just on that, not on other questions of market support or compensation to this or that person or whatever.

99. Would that be in the first year?

(*Mr Hogg*) That would be in the first year.

100. Then in subsequent years there would be costs of equivalent amount?

(*Mr Hogg*) Broadly, I should say.

101. Possibly?

(*Mr Hogg*) Broadly, yes.

102. My second question is the heretical proposition. Have you thought through the consequences of instituting yourself, as Minister, a ban on all imports and exports of beef into and from the United Kingdom, and what the consequences of that would be for the British market and in particular bullock production? Do you think it is possible that behind such a wall of an import and export ban instituted by yourself, it might be easier for the British beef industry to restructure?

(*Mr Hogg*) I have not reflected in detail—

103. Would you consider it?

(*Mr Hogg*) May I answer the question? I have not reflected in detail on that proposal, but my immediate and quite confident response is that it could be attacked on a number of fronts as being illegal.

104. I understand you obviously have to say that. I understand why.

(*Mr Hogg*) It is not just that I have to say it; it is because I believe that the answer I have given is true.

Chairman

105. Could I go back to the NFU's proposals and the cost? The proposal, as I understand it, is that any animal born before 1989 would, at the moment of cull, be sent for incineration rather than for meat, and there would be a compensation payment?

(*Mr Hogg*) My recollection of what Sir David—and he was quite clear on this point—was saying is that his proposal was that beasts over the age of 30 months (the threshold is taken largely for reasons of identification, because by dentition examination you can actually determine the age of such beasts) should not go into the human food chain, but they could remain as milking cows or as beef cows until that period of their career had come to an end, and that they then be slaughtered.

106. As to the calf slaughter premium, presumably the cost of that would depend on what, if there were to be a calf slaughter premium, the premium per calf were to be?

(*Mr Hogg*) Yes. That is a different policy not forming part of the NFU package, but there clearly has been a suggestion—you have seen it yourself—that there should be a policy designed to slaughter the very young male calves who cannot at this moment be slaughtered, and there is clearly a figure attached to that. I believe the figure is £100 at the moment, but I may be wrong.

107. It may not be part of the package, but it says here "At the same time the NFU is vigorously demanding the introduction", etcetera.

(*Mr Hogg*) Yes, but I was trying to answer Mr Campbell-Savours' narrow point, and I hope I was responding to that specific question.

Mr Powell

108. Mr Hogg, I do not think it will be a great surprise to you to learn from me that most of my constituents would find that spending £550 million a year to destroy beef which is otherwise perfectly edible is not the best use for £550 million. If you were to start engaging in a slaughter policy which has no scientific justification for it, you would find yourself open to very substantial criticism. I want to build on the questions which were asked by Mr Spring and Mr Campbell-Savours in relation to the role of the European Union. I do not think it will be a surprise to you to know also that in Northamptonshire there is very considerable doubt as to whether the action which has been taken by our partners in the European Community could possibly be described as within the law; there is a feeling that the other countries in the Community are using this as an opportunity in order to introduce protectionist measures against the best meat which is actually available within the Community. Therefore, there is increasing demand that we should be considering retaliatory measures against those who are acting unlawfully against our own products. I am wondering what sort of consideration you have been giving within the Government to banning the entering into this country of meat which has been injected with artificial substances, hormones, etcetera, which would otherwise be completely unacceptable to our consumers?

(*Mr Hogg*) If you are referring to hormones coming from the European Community, there already is a total ban in respect of those products, so that particular possibility does not arise. What I would say to this Committee is what I have said already: that there are a number of legal measures that can be taken to challenge what has been decided, but they do not solve the problem in the short term; that if we tried to pursue the kind of policy of retaliation that Mr Campbell-Savours has suggested and you have reinforced—

Mr Campbell-Savours

109. Not retaliation.

(*Mr Hogg*) You have expressed it slightly differently, but I am not sure it did not come to the same thing. You said "fortress Britain" rather than "retaliation". Yes, it is a perfectly fair qualification. I am quite sure we would have very, very serious problems. Our basic strategy must be to try to restore

[Mr Campbell-Savours *Cont*]
consumer confidence, market confidence and get the ban lifted. Now we have to consider the best way forward.

Mr Congdon

110. Can I return to the important subject of the level of risk to human health? This was touched on earlier today. I must say, we did not really manage to get much further forward, and I understand the reason for caution. Can I ask you, Sir Kenneth, in terms of the situation we are in, what calculations have been made into the numbers of people who should have been infected in the same way as the ten unfortunate people who contracted, or who are believed to have contracted, CJD via BSE? Secondly, what estimates have you made of the number of animals which may have been infected and slaughtered for human consumption before the SBO ban?

(*Sir Kenneth Calman*) I think my colleagues will be able to expand on what I will say very briefly, but the first point to make—and I will make it again—is that there is no evidence of a link between BSE and these ten cases. That is the first point. Secondly, with ten cases—and I agree with you too about the sadness about these ten cases—it is, I think, impossible to make further predictions as to what they might then mean. I think that any predictions would actually be misleading, and it would be unhelpful to take that further. Perhaps I can ask Professor Pattison and Dr Will to comment further.

(*Professor Pattison*) Taking those ten cases, at the moment the features of those ten cases do not allow us to define narrowly anything which might indicate susceptibility or exposure, if indeed there is an exposure, to BSE. The best that we can do at the moment, I think—and Dr Will will help me with this—is that there is a particular feature of one of the genetic tests, the so-called methionine homozygosity, which appears to be common to all these cases, but that unfortunately is present in 38 per cent of the population. So it is beginning to narrow down possibly a little, but not usefully.

(*Dr Will*) I have very little to add to that. I think it really is impossible to make any predictions on the basis of current evidence, but I think it is very important for us to continue to try to do our very best to ascertain these cases and to get a very clear idea if there is any change in numbers with time. But I quite agree with what has been said before, making a prediction is really impossible at present.

111. Can I come back on that? I understand the difficulties, but here we are in a situation where these statements have all been made before in public, and terms have been used like "there is an extremely low risk". With all due respect, I think the problem the public have with the term "extremely low risk" is: what does it mean? An attempt was made earlier this morning by my colleague Mr Powell, when he quoted a doctor friend. Was his order of magnitude quoted earlier a sensible view, or not? Cannot anyone make some sort of assessment as to what "extremely low" is? Is extremely low 1:1 million, 1:10 million, whatever? The trouble is the terms being used, with all due respect, are unhelpful. No-one is prepared to give any indication to the public, as far as I understand, of what "extremely low" means.

(*Professor Pattison*) We did actually put that in our advice to the Government as one of the introductory paragraphs. We tried very hard at the weekend, because we know that everybody in the land would be grateful if we could put a figure of 1:100 million on it. We do have a number of factors which we can specify accurately. For example, if instead of injecting something into the brain you take it orally, we know that reduces the risk (even in animal experiments where you are in the same species and there is no species barrier) by at least 100,000-fold. We know, for example, if you are dealing with muscle as opposed to brain you are reducing the amounts even in clinically sick animals 1 million-fold, and actually probably much more than that. The sensitivity of our assays leaves it at a million-fold. These are very big numbers. Unfortunately, there are 50 million people in the country, and if you have a small risk it can actually multiply up to a large number of cases. There are three critical things we do not know: we do not know what the species barrier is between cattle and man. It may be very high, but we could not actually advance to you any information that there is not one at all; in other words, man is as susceptible as cattle. In fact, one or two people are suggesting we have no evidence that man is not more susceptible than cattle. Frankly, that seems to us to be inconceivable, but if pressed we would say that we cannot prove it is not. One of the other things we do not have is an accurate figure for the infectious dose for man. Finally, as Sir Kenneth put it rather graphically this morning, the sensitivity of our tests give us a floor, but we do not know how far below the floor the result is with muscle. If you put all that together, we are dealing with a risk, in our estimation, that may well lead to no cases per year if you eat the average of 20 kilogrammes of beef, which is what the British diet contains. We could not justify that for you accurately. It is not a zero; it is just that if everybody in the country ate the average amount of beef it may be so low you get zero cases; but if somebody said to me, "Can you prove you would not get one?", I cannot.

Chairman

112. We would settle for one—that is the problem.
(*Professor Pattison*) It is.

(*Sir Kenneth Calman*) If I may just add a brief word to that. I think you have been trying to get us to give you a figure, and I think we have been trying to say that is extraordinarily difficult, and that is about assessing the risk. The issue which is much more important in public terms is the perception of that risk. We already have ways in which we perceive the risk of crossing the road; we know that cigarette smoking is not a good thing to do, yet 27 per cent of the population in this country continue to smoke cigarettes. There is no question that there is a risk there; that risk is very clear, and yet the perception is, "It won't happen to me". I think the issue which you

[**Chairman** *Cont*]
have raised, and the public response, has been the risk has been perceived to be much greater than I think the science says. Indeed, I return to the fact that it is extremely low. I cannot put a figure on that, but it is extremely low.

Mr Congdon

113. I understand that, but even making the statement "extremely low" means that you have some feel in the way that was just described for the level of risk. We have already heard that the chances of getting CJD now from meat are far less than they were five or six years ago. We have already heard further measures that are being taken now will make that even less likely. We have now been told that it is possible, only possible, that no-one else will get it. We do not know whether that is true or whether it is not, but there are many things where it is quite difficult to assess the chances, but people do actually make some sort of guesstimate based on all of that—probability, theory whatever—as to what that level of risk is. In saying "extremely low" what I would be intrigued to know, and I am sure the Committee and the public would be intrigued to know, are you thinking of 1:100,000, 1:1 million, 1:10 million or whatever?

(*Professor Pattison*) No, we are thinking of a risk that would multiply up when you multiplied it by the population of this country and the consumption of that risk, around muscle, which may actually have a zero risk but we are not sure about it, which would lead probably to no or a single figure number of cases, if it exists at all. That is the sort of number which I believe we are talking about.

Chairman

114. It sounds as if there is a much greater chance of winning the lottery than we have of being struck by this disease.

(*Professor Pattison*) People do win the lottery!

Mr Powell: They do not win the lottery four weeks in a row, and you are suggesting odds which are as remote as that.

Sir Roger Moate

115. Where medical science does seem to be more positive is that these ten have been identified as having a separate distinctive and aggressive strain of CJD, which might or might not be attributable to BSE. CJD has been with us, sadly, for a long time, and been present in other countries where there is no great instance of BSE. We know that. In the past the link has never been made and CJD has never been attributable to BSE. The fear is that in future all cases of CJD will be attributed to BSE; scare stories will run and run here and abroad. I do not think I could understand a medical lecture, but are we sure that this new distinctive strain is clearly distinctive and definable? Are we reasonably confident that our own medical profession in the United Kingdom, but also, significantly, practitioners abroad, will be able positively to identify this separate new strain, so that we can clearly isolate the BSE possible linkage and the other continuing number of CJD cases from the other causes, whatever those other causes might be and might have been in the past? How distinctive and clear-cut can we be on this?

(*Dr Will*) These ten tragic cases have a particular neuropathological appearance which we believe has not been seen previously in CJD. We have done a great deal of work. My colleague, James Ironside, a neuropathologist, has been trying his very hardest to review every case we can see in the literature, to see if there are any cases like these from the past, and we have been unable to find any. We have been in contact with many colleagues around the world to ask them the question of whether they have seen this neuropathological appearance before, and the answer so far has been "no". The clinical features in these cases are also somewhat unusual for CJD. The age of the patients is unusual for CJD. Fitting it altogether it looks as though this may be a new variant. It is extraordinarily difficult to prove that on current evidence because it is quite possible, for example, that in a month's time someone will say, "We had a case like this in another country 30 years ago". However, on current evidence, we believe that this is a new variant of Creutzfeldt-Jakob disease. It is, of course, a matter of concern if there is a new variant, and there have been a number of cases over a short time period. This is the reason for concern. In relation to other countries, we are involved with collaborations with a number of other countries in Europe in the surveillance of Creutzfeldt-Jakob disease, and have been able to obtain information from other countries about their experience in recent years of this condition. Again, on current evidence we do believe that this is something that appears to be distinctive and seems to have occurred in the United Kingdom. As I say, the appearance, which we have described, we hope will be published shortly in medical journals; and in that way we will be able to obtain information from other countries in more detail as to whether they have ever seen anything like this before.

(*Sir Kenneth Calman*) Could I add to that, I have of course written to all doctors informing them of the background to this and giving them some information about CJD and the new variant. This will almost certainly lead to people thinking hard about it, and it may well be that over the next few months other cases will be identified on suspicious grounds, and of course that is where Dr Will's Unit will be able to give advice to look and see whether that is the diagnosis. I do not think you should be surprised if, over the next few months, individual cases are identified one way or another. The second part is, if there is a link, and I suggest *if* there is a link, between BSE and this new variant of CJD, this means the other cases of CJD are likely not to be. This is a new entity we are seeing. The last point I would wish to make is that the World Health Organisation next week will be holding an international seminar which people from Dr Will's Unit will be at to discuss this further and begin to take the comparative work which you have described beyond the UK into the international arena.

Mr Martyn Jones

116. I understand that there is a strain-typing procedure for BSE—bio-assay in mice. Has anybody thought about using pathological material from these ten unusual cases in that same way?

(*Dr Will*) Yes, that is quite correct. One of the ways of obtaining further evidence as to whether or not these cases are in any way linked with BSE might be obtained from carrying out such transmission studies, and these studies are already funded and planned.

117. When will we have the results?

(*Dr Will*) Unfortunately, by the very nature of these studies, it will take two years at least to get a definitive result.

Alice Mahon

118. Are you trying to isolate the ten cases and say they are related to eating beef, or been in contact with beef, but the others are not?

(*Professor Pattison*) Yes, absolutely. The situation, due really to Dr Will's more active surveillance during the early 1990s, was that we progressively saw the number of cases of CJD going up and that, first of all, was the cause for concern. Actually they came down last year. Overall the numbers are not going up. Then you look within the numbers to see if anything else is changing. If you recall, we first concentrated on CJD in dairy farmers, and that built up to four cases of CJD in dairy farmers. Those dairy farmers are not in this series of ten cases at all. Indeed, there was good European evidence to show that for some reason you got an over-representation of dairy farmers in classical CJD. Then we focused on age, with the first teenager in this country to get it and the second teenager, and then Dr Will and colleagues expanded that to thinking about the under 30s, and now of course we have an age range at death of 18-41 years of age. The age focused us in the first place, but then it is becoming much broader now. It is a distinct cluster within the overall numbers of CJD.

119. It could be an under-estimate, could it not? Mrs Oxley, the mother of Professor Judith Oxley, died of CJD in 1992 aged 73 and CJD was only diagnosed because Professor Oxley insisted on having her mother's brain examined. If it had not been for that examination she would not have been diagnosed as having CJD. That under-estimate could occur in the ten you are now talking about.

(*Professor Pattison*) Before handing over to Dr Will, I think it crucially important to say overall how good we are at diagnosing CJD in this country—probably better than anywhere else in the world. I am sure Dr Will will want to follow that up.

(*Dr Will*) I think what I would say is that we can never guarantee the 100 per cent ascertainment of cases, however hard we try. The way we identify cases is primarily through direct notification from neurologists, neuropathologists and others. Indeed, it is only through an extraordinary level of co-operation within the neurological community that we are able to do the study at all. We believe we have very good ascertainment. We also obtain death certificates which mention CJD as a safety net, so that in retrospect we can try and identify cases that we missed through the direct notification. Inevitably, however, there will be occasional cases in which the diagnosis may not be recognised. I think this may be particularly liable to happen in the elderly population. However, as Professor Pattison said, the numbers of cases have risen in the 1990s overall. The major reason for this is an increased number of cases identified in the elderly population. I think we have better ascertainment in the elderly population than we have before. Of course, it is possible that there are some cases that we miss, but I personally believe—and I will be happy to discuss the evidence—that these cases are not very frequent at all.

(*Sir Kenneth Calman*) If I might add to that, Chairman, if we assume that the figure in the population is around one per million (and it is actually less than that) then the General Practitioner with a list size of about 1,500 patients is unlikely to see a case in his or her professional lifetime. The difficulties of recognising them are quite, quite great, which is why I put now to all doctors, to make them aware of it. I would not be surprised if we see a number of cases being recognised which were not recognised before (and I hope this Committee will recognise that is a positive step, not a negative step) so that we know more about the disease and how to control it rather than trying to hide behind the figures. We may well see an increase, but it will be as a result of better medical care and better identification of the problem.

Mr Bayley

120. May I put a point to Dr Will? Since there is reasonable evidence that an infective agent is responsible for BSE, how can scientists possibly justify the statement that immune-compromised patients or children whose immune systems are not fully developed are under no increased risk of infection? Would it not therefore be wise to advise caution, given your conclusion that with these ten cases—although not proven—the likely explanation is that there has been some species jumping?

(*Professor Pattison*) I wonder if Mr Bayley would mind if I answered this, because it was the subject of considerable discussion at SEAC at the weekend. These agents and these diseases do rather contradict many of the classical aspects of bacterial and viral infections. One of the interesting aspects is that in animal models—if you use, for example, scrapie—you find that if you use immune-compromised animals they tend to be more protected than fully immune-competent animals. So we concluded that this is one of those areas, unlike any other infection that we know of already, where it is conceivable that immune-suppression might be protective, but it was simply left as "at no more risk".

121. You say it is conceivable there might be more protection, but it seems to me that there is a huge area of doubt. Could I put another question? If you were a mother weaning a baby for the first time on to food (which would mean that that baby would never have

[**Mr Bayley** *Cont*]
ingested a beef product to date), would it make sense, until one is clear about the degree of probability with these ten cases—and I can see Dr Will nodding his head—to advise that mother, until there is greater clarity about whether there is a risk of species jumping, to play safe and not to expose that child to beef products?

(*Professor Pattison*) I wonder if I might finish answering the last question and then I will answer that one as well. The other thing about the immune response is that in these diseases there is no detectable immune response. In most other infections we are talking about, what you suppress when you immune-suppress the individual is their acquired defence mechanism which they have acquired because they have had the infection before. That does not apply to this group of diseases. In relation to the question about the infant, I think we have approached it from the point of view of saying "What do we need to do to ensure that if anybody, on their own behalf or on behalf of their child or on behalf of their infant, actually wants to eat British beef then it is safe?" Of course we are not in the business of telling anybody what to eat or what not to eat, but if you want to achieve zero risk in anything in life the only way to do that is to avoid it completely. I can tell you from a personal point of view that that thinking also applies to my thinking about my two grandchildren.

Mr Sims

122. Just picking up the point of a few minutes ago in relation to the difficulty of diagnosing with elderly people. I heard it said that the symptoms are rather similar between CJD and Alzheimer's Disease. Would you care to comment on that? Is it possible that both in this country and, indeed, abroad the deaths of people suffering from CJD may not be accurate?

(*Dr Will*) This is a subject that has been raised before and, indeed, was raised in 1990. In actual fact, I wrote a memorandum about this particular topic, which just happens to be the last page of the Agriculture Select Committee Report, and it details what I said at that stage. There is a possibility of confusion with Alzheimer's Disease in terms of diagnosis. This only applied to a very small number of all patients with Creutzfeldt-Jakob Disease. The great majority of patients have a very short duration of illness, measured in months, in contrast to the duration of Alzheimer's Disease and other forms of dementia, which usually go on for some years. So I think we are confident that in the great majority of cases of CJD we are able to distinguish them from Alzheimer's.

123. Reverting to the original cause of the problem, as I understand it it was cattle feed which was from sheep which had been infected with scrapie. Scrapie, as far as we know, has been around for 200 years or more but it was only when it came into the cattle chain, as distinct from sheep, that this possible difficulty arose. Can you confirm that there is no possible danger to human health from scrapie in sheep?

(*Professor Pattison*) The best person to answer that is Dr Kimberlin.

(*Dr Kimberlin*) I think this is a very good question because it really brings to mind one of the key starting points in beginning to think about potential risks from BSE. Can I tell you a short story? In 1968, when we first knew about the transmissibility of Creutzfeldt-Jakob Disease, at that time we also knew a little bit about the mink disease TME, which we had reason to believe was probably due to rare infections of the prion in mink from scrapie. That led to the awful thought that maybe Creutzfeldt-Jakob was also rare transmissions of scrapie to man. In those days it was possible to panic quietly and privately and just worry in terms of doing the necessary surveillance to establish whether or not there was a link at all. That research did not formally establish all the answers about the cause of CJD. What it did make perfectly clear is that there was no evidence whatsoever that scrapie was linked to Creutzfeldt-Jakob Disease. At the same time, we knew that logically it was not because people had never been exposed—and that is the point of your question—because historically there has been potentially an enormous exposure (I was going to use the word "phenomenal", but I will not push my luck!) over the decades and centuries. It was that kind of experience, based on 20 or more years of epidemiological surveillance in many countries with and without scrapie which led to the conclusion that with these diseases you can certainly expect there to be a very substantial species barrier, otherwise we would have had a problem with scrapie years ago. You might then say "Why did we ever worry about BSE at all?" The only answer to that, really, is we just could not assume that if the origin of BSE was scrapie getting into cattle, the very fact it was in cattle did not necessarily make it identical to scrapie. It was only that uncertainty in my mind which started the processes of thought which said "We have to do something. We have to make some pre-emptive strike as early as possible to reduce exposure". In a way, that was the whole basis of taking any actions at all, and the key message from all the historic experience and research of these diseases is because they have a long incubation period, the important thing is to take action as soon as possible, even at a stage when, in fact, we had very few cases of BSE indeed. If you can remember back to the 1988/89 period, we had no predictions about what kind of epidemic BSE would grow into. Nevertheless, having figured out that we could not rest on our laurels of past historical assurances, something had to be done and done reasonably quickly. As far as I am concerned, that was done reasonably quickly and reasonably well.

124. However, it appears, from what you have said, that BSE almost certainly came to be entering the food chain from scrapie-infected cattle feed. Do we know where the original batches of cattle feed that were so infected came from? Were they home produced or from elsewhere?

(*Mr Meldrum*) Can I just introduce another thought to you, if I may? Richard Kimberlin has talked about the origins from scrapie, but there is an

[**Mr Sims** *Cont*]

alternative hypothesis, and it is only a thought. Maybe BSE has been in cattle for many years and is a cattle strain. I do not think we know and we shall not know for a very long period of time. To answer your specific question, going back to 1985, when we carried out the back-tracing on the cases that had been revealed in 1986, we went to the farmers and asked them when they had first seen the disease, and it appeared to be about April 1985. Looking around the country, it was, in fact, covering already, at that time, most regions of the United Kingdom. So it was, apparently, very widely distributed and did not appear to be related at that time to any particular feed or feed mill or compounder.

Mr Austin-Walker

125. Going back to the question that Mr Sims asked, he was referring to confusion over symptoms, and I think Sir Kenneth was saying that doctors are now proving their diagnoses. I understand that there will be a long period of time after someone is infected where they will be asymptomatic. Answers earlier indicated we do not know how many people will be affected; it could be anything from 0 to half a million or so. We have also been told the transmission of the disease can be done more effectively and efficiently other than by oral means and ingestion. In our hospitals, when a hospital is treating someone with CJD or any prion disease, there are special surgical and clinical procedures. Is it possible that in hospitals and other such places we could be treating people who are prion infected but are asymptomatic, and therefore it is not known. Should not, therefore, surgical procedures be changed as a preventative measure? As I understand it, the prion protein is particularly resilient at the sort of temperatures that bacteria and viruses are. Is it possible that some of the infection within the species may be by transmission through cross-infection?

(*Sir Kenneth Calman*) I know that Dr Will and Professor Pattison will wish to comment but that is a very important issue. Of course, you have to go back to the biology of the disease—CJD in man we are now talking about, which is essentially a brain disease—and that is the area where, if there was any risk, that would be the risk. We know that can happen in terms of transplanting matter. Neurosurgical procedures or anything that goes near the brain are actually covered already. In terms of repairing hernias, for example, or whatever, that is just negligible because there is no evidence that that would occur at all. Part of the information collected on the ten cases has looked at: have they had any surgical procedures of one sort or another and was there or could there be any connection to that. That is a very interesting question and I will let Dr Will answer it.

(*Dr Will*) It is a very important question. All I would say in addition is that studies of Creutzfeldt-Jakob-Disease really do not provide any good evidence of any risk in relation to surgery other than that which involves brain surgery. There have been very rare instances in which that has been a risk and these were mainly many, many years ago. The problem with all this is that people ask a question: why do people get Creutzfeldt-Jakob-Disease and if you cannot give an answer people immediately assume that it must be scrapie or BSE. If I could just be allowed to make one remark about that, Creutzfeldt-Jakob-Disease occurs all round the world at about the same incidence in countries that are free of scrapie and free of BSE. Within an individual country, they appear to occur completely at random. This has led to the proposition that Creutzfeldt-Jakob-Disease is not due to any environmental contamination at all but is due to a spontaneous change in the protein in the brain itself, occurring as a random event. I think it is important to understand that, although we do not know the cause of Creutzfeldt-Jakob-Disease, there is a lot of evidence that is very important in considering what is not likely to be the cause of Creutzfeldt-Jakob-Disease. I think I would finally say that, in relation to the question you ask, we have no good evidence that Creutzfeldt-Jakob-Disease is caused in any way by cross-contamination other than by very special circumstances.

Mr Corbett

126. Sir Kenneth, can I ask you, please, to remind the Committee on what date officials first alerted ministers to the possibility of a link between CJD and BSE? Dr Will, perhaps I might ask you: what would your reaction be if there were six new CJD cases in the younger age group in the next six months?

(*Sir Kenneth Calman*) Before I respond to that, I assume you are meaning the ten cases which we were talking about just now? Is that correct?[1]

127. No; first.

(*Mr Dorrell*) What do you mean when you say "first"? Do you mean the first reporting of CJD?

128. The first time officials alerted ministers to the possible link between CJD and BSE.

(*Mr Dorrell*) The only way that question can be answered in terms is the first advice ministers were given about what were then the nine cases.

(*Sir Kenneth Calman*) No, I do not think that is what we are talking about.

129. You answered the question. If that is the answer, that is the answer, but just remind me what the date was, please.

[1] *Note by witness:* In May 1988, the Working Party on Bovine Spongiform Encephalopathy was established under the chairmanship of Professor Sir Richard Southwood, to examine the implications of Bovine Spongiform Encephalopathy (BSE), a newly identified neurological disorder of cattle, in relation to both animal health and any possible human health hazards and to advise the Government on any necessary measures. In its Report presented to Ministers on 3 February 1989, the Working Party concluded on p. 13 para 5.3.1 that "Kuru and Creutzfeldt-Jakob Disease demonstrate that humans are susceptible to spongiform encephalopathies. The potential routes of transmission of BSE from cattle to humans have been examined closely. With very long incubation period of spongiform encephalopathies in humans, it may be a decade or more before complete reassurance can be given".

[Mr Corbett *Cont*]

(*Sir Kenneth Calman*) I first heard about the ten cases that we were talking about on 8 March this year.

130. Ministers would have been told about that time?

(*Sir Kenneth Calman*) Yes. This is quite an important issue. Dr Will can respond to this but clearly over the few weeks before that there were some discussions, which I was not part of and did not know about, to the effect of, "Gosh, here are ten cases that look different". I was first notified about this on 8 March.

(*Mr Dorrell*) Can I just take the story forward so that the Committee is completely aware of the way that developed? I was alerted and I think other ministers were alerted to the fact that these cases were emerging that were going to need to be considered. I was told that SEAC were convening to analyse the evidence and to draw up its conclusions and that was the first meeting of SEAC, roughly ten days ago. That led to the recommendations and the conclusion which were reported to the House on Wednesday of last week.

(*Dr Will*) I am not quite sure what you would like me to answer.

131. What would your reaction be if there were six new cases of CJD over the next six months which you suspected, and others suspected, had a link to BSE? Would you be startled or are they the kind of numbers that are possible without getting alarmed?

(*Dr Will*) I think any case of CJD is a tragic occurrence and six more would be indeed tragic. However, I think it would be very difficult if the implication of your question is to predict what will happen. I think we really have to carry out very careful surveillance. If we do have new cases, they will have to be discussed with the experts, including the epidemiologists and the SEAC committee in order to decide—

132. I am not asking you to forecast what is likely to happen. I am just saying, if it turns out in the next six months that there are six new cases with a suspected link in that younger age group, what would be your reaction, as a scientist? Would you be totally alarmed or would you say, "Well, that is okay; we can live with it", or what?

(*Dr Will*) What I am trying to say to you is that any case of Creutzfeldt-Jakob-Disease is a tragic occurrence and I think that I would be obviously very upset if more should happen but all I can say to your question is that I would discuss it with my colleagues before deciding what the implications would be. I am not quite sure what you are asking me to do: to predict what will happen on the basis of these cases?

Chairman: May we leave it there for the moment? There will be further questions on this.

Mr Alexander

133. I just wanted to ask Dr Will whether it is possible—and I think he has perhaps answered it already—for CJD to come to human beings through ingesting other than BSE infected products. In other words, you can acquire CJD through perhaps eating sheep, through perhaps eating BSE infected products or perhaps through random facts of nature, as you said a moment ago. Is that correct?

(*Dr Will*) I am not quite sure of your question. I am slightly confused about exactly what you want me to answer.

(*Sir Kenneth Calman*) If I can correct the comment on sheep, we have no evidence of that at all. I would like that to be corrected immediately. I think the issue is that there may be other factors and indeed I think that is Dr Will's initial response. There may be other factors which are unknown to us which cause CJD and we do not know what they are. They may be something other than eating meat.

Audrey Wise

134. We have been told that scrapie has been around a long time and that they more or less kept themselves to themselves, as it were. Is scrapie increasing among sheep? The characteristic with BSE seems to be that there was such a huge explosion in it, not just that it happened, which is worrying, but that it happened on a big scale. What is the situation with sheep?

(*Mr Meldrum*) I have no evidence that there is in fact any increase in scrapie in sheep at all. It is a notifiable disease but that notification is for the purposes of assessing the incidence of disease on a flock or herd basis rather than on a national, individual animal basis, so we know how many flocks have had a confirmed case in the last two and a half years, but further than that we cannot go. Certainly, from the information that I am receiving from veterinary surgeons and so forth, there are no indications of any explosion. In fact, I would hope that there would be a contrary effect. The ruminant protein ban brought in in 1988 also applies to sheep. If therefore that is a route of infection of sheep as against maternal transmission, we hope there will be an effect from that ruminant protein ban.

135. Looking at a statement that you made, Mr Meldrum, about it might not be scrapie which has caused BSE in cattle anyway; it might be a cattle disease, just exploring that a little bit, how seriously is that a possibility? If that is the case, there is presumably some reason why this cattle disease has suddenly escalated. What research, if any, is being done into exploring what the cause of BSE and its level is?

(*Mr Meldrum*) There have been a great number of investigations carried out into the epidemiology of BSE by colleagues elsewhere in the Ministry, based on information that is obtained at the time of any case being confirmed or suspected, so we do have a vast amount of data obtained from farmers and others which is computerised and which is available for analysis. That is an ongoing process. I simply and solely introduced the alternative hypothesis because that is on the record in papers that we have produced and published. We have indicated that the two hypotheses go side by side. I suspect that John Wilesmith, who was involved in the first investigation, would still tend to the view that in all likelihood BSE came from sheep scrapie. He and I

[Audrey Wise *Cont*]

often discuss it and I hold the alternative view but it does not matter all that much at this time because we now know that the agent of BSE has a singular characteristic. All the strains that we have examined so far, through mice, appear to be the same and none of the scrapie strains that have been put through mice have the same characteristics. That does not help us very much. Maybe, in due course, we will obtain further information on BSE from outbreaks that occur in other countries, due to the collaborative research programme going on with these countries, comparing information, data and the strains that are identified. So far, the strains both in the UK and in Switzerland are the same, so far as we can tell.

136. If there is an alternative cause, it just seems to me that somebody should be doing something to find it. I am a bit disturbed at what I felt was some complacency there.

(*Mr Meldrum*) I am not complacent. I can assure you, on BSE of all things, I am not complacent. I hope I was not indicating I am being complacent either. That also is far from the truth. I was just talking about what might have been the original source of BSE. The original agent may have been recycling in cattle at a very low level. This morning we talked about rendering systems that were introduced in the 1970s that may have allowed the agent to recycle through feed and as recycling took place the level of the agent increased year upon year.

137. There may be other factors of intensive farming. All I am asking is: is anybody looking at anything else?

(*Mr Meldrum*) Yes, indeed. I was trying to indicate from my earlier answer that we do have a vast amount of information in the public domain, that has been published by Dr Wilesmith and others from the Central Veterinary Laboratory, indicating the lengths to which he has gone, both in 1987 and, more recently, to analyse cases of BSE to determine the cause, and more recently to identify and ensure that there are no alternative hypotheses. He has in fact looked specifically at the possibility of maternal transmission. He has concluded that there is no evidence at this time of maternal transmission, cow to calf, although that work is ongoing and the final result from other experiments will not be available until the early part of 1997.

Mr Pickthall

138. A few moments ago, Professor Pattison, you said in passing that, for some reason, there is a higher incidence of CJD amongst dairy farmers across Europe, not just in this country. It seems to me the public do not really believe in coincidences of that kind. It is precisely that sort of information. We have heard similar things in the media in recent years about butchers, for example. Is there nothing that you could tell us? You say "for some reason"; is there nothing that you could tell us, beyond the contact with cattle, that would account for that?

(*Professor Pattison*) There is nothing at the moment. I can assure you that all the material from all the cases has been inoculated into the strain typing tests. That of course ultimately will show where there is anything BSE-like in the dairy farmers. Actually, it is not just that there is a higher representation of dairy farmers in other European countries. I should have also added: and it is unrelated to the incidence of BSE in those countries, so it is not mapping onto the incidence of BSE. In terms of looking for other risk factors, that has been done with those cases. They are not this variant of CJD that we are talking about. Dr Will will confirm that there are no obvious aspects of those four cases which seem to connect them, other than their dairy farming operation.

(*Dr Will*) That is right.

139. Is this statistical difference for people occupied in working with animals, for CJD or whatever strain, significantly different from the rest of the population?

(*Professor Pattison*) Dairy farmers are over-represented.

140. By how much?

(*Professor Pattison*) The numbers are very low indeed. As to the total number of cases, we have some rather odd features there. The church unfortunately is over-represented but it is just about two versus one or none in a whole variety of occupational groups. The numbers are so small and you have to put such a huge confidence intervals into the statistics that you cannot really satisfy yourself that you have proved beyond doubt that there is a statistically significant association.

141. I take what you are saying but nevertheless you chose to say, in passing, that this was of significance to report, the fact that they were dairy farmers.

(*Professor Pattison*) There are more in many European countries than you would expect by chance.

Mr Whittingdale

142. I do not want to continue to press you to make predictions that you do not wish to make, but you will appreciate that your reluctance allows others who are less scrupulous to grab headlines. Can I therefore ask you: if it is the case that the new strain of CJD is linked to BSE, would it be probable that the spread of this new strain would follow roughly the pattern that the spread of BSE in cattle has been?

(*Professor Pattison*) I think it is probably more likely that it would follow the pattern that the epidemic in cats due to BSE has followed because you would have the equivalent then of a jump across a species barrier, cattle to man rather than cattle to cat, and you do not recycle cats or human beings back to cats or human beings respectively. Irrespective of whether it came from scrapie or BSE, the recycling of cattle remains back to cattle ultimately led to the huge amplification in the cattle epidemic. We do not think that will happen in the human, even if it is linked. It did not in the cat. The incidence, even if you put in a factor of ten times under-reporting in cats—because of course it depends how near to a veterinary school you are for the diagnosis in the cat—is 14 per million cats in the

[**Mr Whittingdale** *Cont*]
country, but please do not translate that in any way to the human population. It is just that the pattern would be the same.

(*Sir Kenneth Calman*) Just to add very briefly to the overall numbers issue, I want to say this so that you will be aware of the implications: clearly, by alerting doctors—and indeed that is part of the process—a number of individuals will be suspected to have CJD who will not have CJD, which is why there always will be more suspected than confirmed. Of these, a small number or none at all will have the new variant. I think over the next few weeks or months in the press you will see emerging a number of individuals who have diseases which may or may not be CJD. I just hope you will keep that in mind as you watch the press.

143. I appreciate that we are in the realm of uncertainty here, but precisely because of this can I ask the Secretary of State whether any contingency plans have been drawn up in case there is a significant rise in the number of CJD cases—contingency plans within the NHS to deal with that eventuality?

(*Mr Dorrell*) It is something we clearly have under active consideration. I cannot say to the Committee that there is a plan to which I could draw your attention, but clearly there are many consequences that flow from the SEAC findings, and one of them is that those responsible for Health Service planning will need to keep themselves abreast of the latest interpretation of the SEAC findings, the SEAC findings being, I remind you, that at this stage the risk associated with eating beef and beef products is extremely low. That would be the assumption on which a Health Service panel would base its planning.

144. Finally, to address those who are willing to make predictions about this, has anything happened which would change the view about those kinds of statements such as that made to the Agriculture Committee in 1990 by Professor Lacey that "If our worst fears are realised we could virtually lose a generation of people", or indeed the headline quoted by Mr Marshall, "This could be worse than AIDS"? Has anything happened to change your view that that kind of statement is alarmist and based in no way on scientific fact?

(*Mr Dorrell*) My judgement, if I may say so, as the member of Government who has received the advice, is that that remains extremely alarmist, and that what we need to focus on is the advice we have been given about the most likely scenario, and certainly to contemplate some variation from that, but to focus on the advice we have been given about the most likely scenario.

Mr Austin-Walker

145. Perhaps the first part of my question is to ask you to answer the unanswerable, which is to ask you what is the level of risk? I think there is a question of public confidence here. What is the level of risk, in your view, attached to consuming not merely beef but other products such as drinks of bovine extraction? For example, I think people want to be assured that it is safe to eat jelly babies and wine gums. I would also like to ask you a question about the risk from serum vaccinations. In answer to a Written Parliamentary Question last week the Secretary of State informed me that currently licensed vaccines do not contain any bovine material sourced from the United Kingdom. So we are in fact importing serum albumen in from elsewhere. Could I ask the question I did not get an answer to, which is when that restriction on the use of United Kingdom-derived serum occurred, and whether that was taken as a decision by the British Government or whether it was as a result of the policy of the European Committee on Proprietary and Medicinal Products? Could you tell us when that decision was taken and what was the scientific advice at the time which caused the decision to be taken?

(*Mr Dorrell*) The answer is 1989 and the Southwood Report.

(*Sir Kenneth Calman*) If I could expand beyond that, there are quite important technical issues here. If you wish, Chairman, to save time, to have a fuller draft on all of that, we would be delighted to provide that to you, because I think that would answer the question of Mr Austin-Walker in more detail.

Chairman

146. I think Mr Austin-Walker did ask whether we could take it that milk, gelatine and other bovine-extraction materials were safe.

(*Sir Kenneth Calman*) Yes indeed, that is the second part of the question. I was really responding to the vaccine issue, if you will allow us to present you with further detail on that. Professor Pattison will respond to the other part.

(*Professor Pattison*) The Advisory Committee was asked specifically to work through those questions again at their meeting this weekend. On gelatine we came back to the conclusion that we always have: that in the process of manufacture of gelatine, if any agent is there at the beginning then the process itself results in a drop by 100,000-fold, so if you put in raw material in which it is most unlikely to be there anyway, then drop it 100,000-fold we say that it is safe for oral ingestion. That, of course, applies to many of the popular foodstuffs. May I just say that the Scientific Veterinary Committee in Brussels looked at the same evidence and came to the same conclusion that we did. That is why it is so bizarre that they have included a ban on gelatine in their current bans. It just does not make sense. Milk also we worked through specifically and again came to the same conclusion. That is one in which Brussels agrees with our conclusion.

(*Sir Kenneth Calman*) At the meeting in Brussels yesterday both Professor Pattison and I raised that at the meeting, so we hope that there will be a response at some point to that.

Mr Austin-Walker

147. We will obviously wait for the detailed material on the serum, but does it indicate that in 1989 there was a perceived risk from the use of bovine serum?

[Mr Austin-Walker *Cont*]

(*Sir Kenneth Calman*) No. I think you do have to return to the Southwood Report which considered a wide range of things. There is no evidence whatsoever from the Southwood Report that there was perceived to be a risk, but it seemed to be prudent, and therefore the industry itself I think wished to make sure that any possible, theoretical, potential risk was eliminated. But I think that by providing you with the information on that it will make that clearer.

148. What I was seeking was an assurance, for people who might have been vaccinated or immunised before 1989, that you consider that they are not at risk.

(*Sir Kenneth Calman*) Yes, and I think I can give you that assurance.

Audrey Wise

149. On 25 March the Secretary of State for Health said that infants and children were not likely to be more susceptible, and we have had a question about that, but it was not pursued. I would like to ask perhaps Professor Pattison on what basis and with what kind of evidence that statement was made, and how long and to what extent have infants and children specifically been studied in relation to BSE and CJD?

(*Professor Pattison*) Of course, in relation to the latter point, we have no direct evidence because we have no BSE in the human population to study. The Committee were joined at the weekend by three outside experts, covering paediatrics, gastroenterology and infection and immunity. We talked through what is known about the normal handling of proteins (because this is a disease which is driven by proteins, and that is the transmissible agent) and the handling of proteins by the digestive tract. We talked through everything we know about the animal models and the transmissible encephalopathies in animals and age susceptibility which is not great, I have to say. There is no evidence of age related susceptibility. We talked through the human transmissible encephalopathy—Kuru—which again is not experimental data, it is observed data based on ritualistic cannibalism, so it is not really quite accurate for the United Kingdom population. We talked through then the defence mechanisms all the way from the mechanical barriers that the gums and the like provide right down to the other end of the digestive tract. We then talked about the non-specific immunity and then the specific immunity. We noted that if we assume an incubation period of ten years in these cases—and you could assume five, it just changes the numbers slightly, but if you assume ten years—which would make it worse because it is younger, then actually exposure would have been aged between eight and 31, and quite clearly there is not a greater representation of children in that group. So we, with our outside help, reached the conclusion that there was no evidence at all that children and infants would be more susceptible than adults, if indeed there is any risk at all to the human population.

150. What about foetuses? Can I also take the opportunity of asking this. I am not sure whether I picked up Mr Meldrum right on the transmissibility. It is this point about foetuses. What happens with sheep? Do ewes transmit to lambs? I gather that cows are not thought to transmit to calves. What thoughts are there about foetuses? Are they different from either of the other animals? If so, why? In looking at these things, are you taking into account the considerable differences in the way children's bodies react to things, and the younger the child the more the difference? How positive is your statement and how much is it just "Well, we've no reason to think so"?

(*Professor Pattison*) I think the statement is positive. We looked at that in relation to those things which we consider to be relevant, and of course in which studies have been done. The handling of protein is one such, and there is good evidence that that very rapidly gets up to adult levels. There is not a sort of gradual changeover in infancy and children, and for most of the factors of normal physiology that we talked about that is also true. In talking about pregnant women we did, of course, think about the pregnancy as well as the maternal health. There actually the evidence from other animal transmissible encephalopathies is more helpful and led us to the conclusion that the foetus as well as the mother would not be more susceptible, even if there is a risk. We can answer the specific points about sheep and cattle, if you wish, but you may want that outside the meeting if you wish to press on.

Audrey Wise: I would like it inside the meeting.

Chairman: Mrs Wise, we have been over these things many times in the past.

Audrey Wise: Not that particular point, and not the health point. We have had, Chairman, four questions on health out of a total of 16, and I do not think that we should skimp on the health issue.

Chairman: Perhaps you could be brief, then.

Audrey Wise: I was going to have an answer to the question I have already asked, which has not been fully answered, about transmission.

Chairman

151. Can you answer briefly, or would you like to write about it?

(*Dr Kimberlin*) Amongst all the transmissible spongiform encephalopathies scrapie in sheep is the only one where there does seem to be evidence of some kind of maternal transmission when there might be a risk to the foetus. That is one reason why we had to be concerned about the possibility of natural transmission of BSE, but of course we have reached the stage now of recognising that that is not likely to occur to a significant extent. In the human context there are no indications to show any kind of natural transmission from mother to offspring either via the foetus or by other routes.

Audrey Wise

152. Have you managed to find any reason why the scrapie is transmitted that way and the others are not?

(*Dr Kimberlin*) That is a good question. The answer is, we do not know what the rules are that determine maternal transmission in sheep and seemingly not in anything else. It is not even clear whether it occurs in goats. So sheep really do seem to be the exception, for reasons we do not understand.

Chairman: We did actually make a recommendation on external transmission in our 1990 Report, and I think you will find, Mrs Wise, if you look at that, that it will enlighten you on some aspects of this. Perhaps we may now go on.

Sir Roger Moate

153. Could I turn to the important questions about the action programme that is seen to be needed to restore confidence? Could I preface the question with a question about the supposed letter—I say "supposed", it is in the newspapers so it must be true—from Herr Fischler, rather critically suggesting to the Minister of Agriculture, as it says here, that "If your findings do not add much to the existing body of knowledge about a link with BSE then a more careful reaction might have been preferable." Assuming that to be a fair quotation, and I suspect it is, what on earth could the Commissioner have been suggesting—that we did not reveal this information immediately? Or, if not, what else might he have been suggesting?

(*Mr Hogg*) I spoke to Commissioner Fischler yesterday, and of course you are right, he did write. I have a great deal of respect for Commissioner Fischler, and I actually like him as well. He was disappointed that I did not give him extended notice of the statement that the Secretary of State and I made in the House of Commons on the 20th. I did in fact talk to him about 20 minutes or so before we made the statement. Chairman, the reason I did not do that was that I thought that on a matter of this magnitude Parliament and thus the country should hear it first from the Ministers rather than from discussion in Brussels. I make absolutely no criticism of Commissioner Fischler, but it seemed to me quite likely that if I explained to him on the Tuesday, say, or the Monday before the statement what exactly we had been told at that stage, it is very likely that that would have leaked out in a way that the House of Commons would have thought inappropriate. So I thought it was right to come to the House first.

154. That was not the point I picked up. The point was that he was actually suggesting a different reaction altogether, which leaves me puzzled. I cannot think that one could have concealed this information from the world once it was in your possession?

(*Mr Hogg*) Certainly not. We had a clear duty to make the House aware of the information regarding the ten cases.

155. Thank you. Can I turn to what I call the action programme. I think we fully understand why, as Minister of Agriculture, you cannot today announce to this Committee an action programme based upon a partial slaughter policy, a culling policy, whatever. One feels that the longer we keep you here, the harder it is for you to go back and bring this policy to fruition, but I hope that is the situation. We do have a debate on Thursday. I think you will understand that there will be many people who will be very surprised indeed if, within the next few days, some sort of action programme is not announced. If it goes on beyond Thursday, I think you will understand that confidence, so far as it exists, will have disappeared very fast indeed. Every day counts. You have already said, with regard to questions about the National Farmers' Union, that the culling of the old dairy cows when they become life-expired, when they have finished productive life, would cost something like £550 million. It was not quite clear whether you thought that was a one-off, whether you thought that would be an annual expenditure, or for how long.

(*Mr Hogg*) The annual expenditure.

156. The only question then is for how long? There are a whole series of other things that have to be done. Clearly, you are not going to spell out that programme to us today. Can you therefore tell us whether other things are under active consideration? You have said that the culling of the older cattle is seriously under consideration. It has already been suggested that there should be a calf slaughter premium. Even those steps do nothing to restore the prices in the market over a period of time, so there has then to be consideration of a wider intervention scheme than the one that exists at the moment which applies, as I understand it, only to prime steers going into the market. It would have to be much wider than that. So is serious consideration being given to that? That is also a feature of the NFU package, and storage aid is another item mentioned. Having done all that, that still does not actually help to restore the name and reputation of British beef. Therefore, I was a bit worried earlier when somebody asked about an advertising scheme and you said, "Well, perhaps, if and maybe". I think you rather implied that that would be up to the industry, but really that is not good enough, is it? Surely we have to have immediate support mechanisms, we have to have a national effort involving Government for advertising, and probably some sort of certification scheme to certify just how good British beef is and to restore confidence at home and abroad. So my question really is, are all these matters under active consideration, and is it fully understood that we really cannot wait day after day after day before we know positively what is going to happen?

(*Mr Hogg*) There are a whole range of matters wrapped up there. First, as regards the implementation of recommendations already made, you will recall that last week I announced that we accepted two recommendations with regard to the de-boning of cattle over the age of 30 months and also as regards the prevention of mammalian protein being incorporated into any feed fed to any farm animal. Now the industry, of course, is aware of those proposals partly because they, like anybody

[**Sir Roger Moate** *Cont*]

else, hear the statements, partly too because Angela Browning and my other colleagues in the Ministry have had meetings with them. We have to incorporate those proposals into regulations. Those regulations are made under Section 16 of the Food Safety Act. They cannot in fact, for purposes I can explain, be made under Section 13 of the Food Safety Act, and consequently we have to go out to consultation.[2] We have therefore to incorporate proposals of those sorts into consultation documents which I hope will go out very shortly, in a matter of a very few days—unless, of course, there are any further changes we wish to make which might in any way alter the content. At the same time Professor Pattison recommended, and we accepted, that there should be yet more vigorous supervision of the existing SBO controls—that is, not the new ones, the existing ones—and in that respect I gave a formal direction on Wednesday last to the Chief Executive of the Meat Hygiene Service to do just that. So in implementation of the action programme, as opposed to the other things you were talking about, we are getting on with it. You then raised a number of other questions. One class of questions relates to market support, and the other class of questions relates to compensation, which is different from market support. As far as market support is concerned, most of the provisions—not all, but most—would be done under the Common Agricultural Policy mechanisms. There, as I mentioned earlier, officials are currently in Brussels talking with Brussels officials as to what particular range of market support measures might be most appropriate. I discussed that briefly with Commissioner Fischler when I spoke to him. There are also a range of other measures which come not under market support, but which you could call compensation or narrow support if you wanted to. Many of those would be of a national kind, and if there is a requirement we will then have to formulate express proposals involving spending, and inform the House of that. The Committee, Chairman, can be sure that we are looking urgently at both classes—both market support and any necessary compensation that might be due, or support that might be due if the present market difficulties continue.

157. Could I ask one supplementary question? I realise that there were a number of points there, and it would be wrong to pursue all of them. Are you satisfied that with regard to market support the Community could respond swiftly enough to what would presumably have to be a nationally-limited intervention scheme?

(*Mr Hogg*) Certainly if the market remains very weak we would need to get, as quickly as we could, market support in place, and it can take a variety of forms. I think that if you would look, Chairman, at the statement I made you will see I talked about support for private storage, for intervention, and you mentioned the possibility of the enlargement of classes, support in respect of the young male dairy calves. So there are a range of measures there, and if the market remains very weak we will have to act very quickly.

Mr Jones

158. I had some very interesting and alarming news passed to me just now which you may or may not have heard.

(*Mr Hogg*) Is this to me?

159. To you, yes, Minister. That news was that the Italian authorities are already impounding British lamb, never mind British beef, which shows a huge lack of confidence in British products, to say the least. As well as talking about market support measures, Minister, will you also be discussing with the European Commission what kind of measures we could implement in the United Kingdom, after which they would lift their ban?

(*Mr Hogg*) That goes back, if I may say so, Chairman, to where we started this afternoon's session. You will recall that I had a fairly vigorous exchange with, for example, Mr Leigh but also with Mr Ieuan Wyn Jones, and I would simply be repeating myself if I went through that again. Chairman: I think we have dealt with that one.

Mr Campbell-Savours

160. Can I ask you, Professor Pattison, why do you believe we are burning cattle? It seems an elementary question to ask you. Why is there a policy of incineration and destruction?

(*Professor Pattison*) Of BSE-infected cattle?

161. Yes. In principle, why is there a policy of destruction?

(*Professor Pattison*) Because once you get to a clinical disease state in the cattle, that is your only option. You would have to say that that animal has the highest concentration of the BSE agent that it will have at any time of its life. There has always been the theoretical possibility that this would pass into the human population if the exposure was high enough, so you must take them out.

162. Why in principle is it not simply sufficient to remove the offal and still leave the animal?

(*Professor Pattison*) Because there is quite a lot of evidence in other spongiform encephalopathies of animals that once you get to that end stage of clinical disease you cannot actually be certain that it is limited to the brain and the spinal cord.

163. So it may have got into the tissue?

(*Professor Pattison*) Mink and goats are ones that come to mind in particular with their own respective transmissible encephalopathies where, right at the end of the clinical disease it seems to be spread out from the central nervous system.

164. Can I ask you a question which calls for a fairly courageous response from you really? It is about the membership of your committee and their attitudes. Is it true that a number of members of your

[2] *Note by witness:* Further consideration led to a revision of the position and the legislation was introduced under Section 13 of the Act.

[Mr Campbell-Savours *Cont*]
committee have expressed a lot of concern about the inadequate policing arrangements in Britain's abattoirs?

(*Professor Pattison*) I think that is the committee's point. That is exactly the point we have been trying to make ever since we began.

165. What were they particularly worried about, then?

(*Professor Pattison*) Once we began to get the audit reports back last year—and it is difficult for me to go back into previous years, but once we began to get the audit reports back—that indicated collapses in the implementation of the regulations at the level it was, that was when the committee became very worried indeed.

166. So there may well have been animals with clinical symptoms who were going through into the food chain?

(*Professor Pattison*) I have to fall back and say we have no evidence of that. It is quite easy, I think, for a not very experienced vet now actually to pull out animals with that clinical disease, of course.

167. Yes, but I understand that that is the reservation that some of them were expressing, and that is that animals were going through with symptoms?

(*Professor Pattison*) No, it has always been about the clinically well animal, but possibly at the late stages of the incubation period, and banned specified offals, particularly the brain and spinal cord.

168. Can you see what I am driving at? That is, that that may well have been happening, based on observation. I understand that the measure of this is observation—I will come to why we know that—because of what was said in the *World in Action* interview by Mr Meldrum. Does it not suggest, though, that there may well have been, if the system was not working, animals that may have had BSE in their tissue, on the scale that we are talking about, that managed to go through the system? Was not that view expressed by some of the people on your committee?

(*Professor Pattison*) No, I do not think it was actually. If you ask me if it is possible that that happened, in all honesty I would have to say yes. If you ask me if it may well have happened, I think that puts a different emphasis on it.

169. Thank you. Now can I turn to you, Mr Meldrum. Can I ask you about the *World in Action* interview and what happened in the studio, so we can be sure as to whether you feel you were in any way misrepresented. What we saw on the film—and I have a copy of the video here—was an auction in Mold in north Wales where animals that had been cleared after the recent changes of the last year were auctioned, they were cleared by the abattoir inspectors, were purchased by a Mr Moss on behalf of Granada Television, and when they were analysed they were found to have BSE. Indeed, on the video which we have here, we actually see the animals slightly staggering in the ring where they are up for auction. Could you tell us whether those animals could have got into the food chain?

(*Mr Meldrum*) I think it would assist you greatly, and me as well, if you were able to see the full interview which took place with me.

170. I am sure that is right. That is why I am being fair to you.

(*Mr Meldrum*) There was a great deal of cutting of the actual full interview, as I was trying to explain to the programme makers the whole of our control programme to protect the public. Unfortunately, the interviewers did not make it clear to me, even as we went along, exactly what they were driving at, and it was only after the event that I was able to ask further questions, if you wish, about Mr Moss and the animals that he purchased on behalf of the programme makers. It was quite clear to me afterwards, though, on the information that I had received, that Mr Moss, after he had purchased the animals in a market, reported those animals to us as suspect cases and they were in fact taken out. So he did his duty under the law. He reported a suspect case to us. These animals, so far as I can determine, therefore did not go into an abattoir, into a lairage, and therefore were taken out of the food chain after Mr Moss had purchased them. I think the programme was trying to give a totally different impression.

171. No, that is how I understood events. I just wanted to check with you.

(*Mr Meldrum*) I think my colleague Mr Eddy is making a good point to me about the procedures that we have in place, that in markets we do have on occasions, but not necessarily as a routine, veterinary attendance for general disease surveillance purposes. It is not part of our checking system, but there is a formal veterinary inspection of all adult cattle in the lairage in all slaughterhouses in the United Kingdom before the animal is killed and goes forward for human consumption. Any animal found to have symptoms which are suggestive of BSE will be pulled out, will be dealt with as a suspect case and will not enter the human food chain.

172. Is it not also true that during the course of the programme, when a food adviser, I cannot remember his name, was actually asked about his experiences on what you referred to as "the back-up check"—your phrase in the interview—or the back-up arrangements, he said that the actual checks that took place in the slaughterhouses were minimal, and that very often animals still went to slaughter that were carrying symptoms that had not been identified when they were actually at slaughter? Is that not true?

(*Mr Meldrum*) No, it is not true. I have no evidence that animals showing evidence of BSE are not being identified *ante mortem* in slaughterhouses. Recently, as a result of various other enquiries that we were making, we were double-checking on this. I asked the Meat Hygiene Service in particular as to whether or not they could confirm that all adult cattle were having a veterinary *ante mortem* inspection, and they confirmed that they were. Therefore, I have no evidence whatever that animals are going into the human food chain showing symptoms of BSE. It is wider than that. The slaughterhouse management, with the Meat Hygiene Service, work as a team. I

[Mr Campbell-Savours *Cont*]
would expect that it is normal procedure that any animal going into a lairage that shows any abnormality in behaviour would routinely be reported to the management and to the official veterinary surgeon in the slaughterhouse. That is part of the team approach to ensure that only healthy animals are slaughtered for human consumption.

173. Can I put it to you that there is a lot of concern? I am not saying that the Department is now not intent on tightening up—I am sure that it is—but there is a lot of concern that there has been a fairly slack regime in many of Britain's abattoirs over recent years. I think that in part that is why we have this sense of great insecurity in the industry and lack of confidence. Can I put to you the question when will all the new SEAC recommendations on the treatment of carcasses in abattoirs be implemented? Do you know when that might be?

(*Mr Meldrum*) I do not yet know. It depends on how soon the regulations are made.

174. Could the Minister give us an idea perhaps?

(*Mr Hogg*) Yes. We have a consultation period, Chairman. The normal consultation period that we operate under Section 48 of the Food Safety Act is six weeks. I would like to abbreviate that, but clearly one has to give time for a proper consultation process both on the merits and in order to address the legal requirements.

175. Then can I ask you to give us a statement of assurance that if there have been difficulties in the past—they are now on the record in public session—in terms of enforcement of the regulations that there have been in the slaughterhouses, there will not be a slackness in the future, and that you are absolutely determined to ensure that they are enforced in every way so that there can be no mistakes?

(*Mr Hogg*) I, as Minister, regard full implementation of all the requirements which govern the operation of slaughterhouses, but in particular regarding those which touch on BSE, as being of cardinal importance. I cannot over-emphasise how much weight I place upon the importance of full implementation. It is for that reason, if I may remind the Committee, Chairman, that I called in the industry on at least two occasions at the end of last year, and the Parliamentary Under Secretary, Angela Browning MP, spoke to other parts of the industry to reinforce this message. I have done it with the Meat Hygiene Service in person and by writing as well.

176. Yes, but you are absolutely determined—that is what you are telling us—to ensure that these regulations are now going to be enforced, and that there will be no future problems?

(*Mr Hogg*) I think I am repeating myself. We will do our utmost to achieve it because of its very high importance. I cannot over-emphasise how important it is.

177. Can we now turn finally to the question of cross-infection which I think is a particularly interesting subject. I wrote to your Department on 9 August 1994 drawing attention to statements which had been made to me about cross-infection between calf offal and other offal. I was informed, in a reply from the Earl Howe, that "We are generally satisfied with the effectiveness of these provisions and are unaware of any evidence to suggest that potentially contaminated SBO material is illegally entering the human or animal food chain. Indeed, the European Commission recently carried out an assessment of the collection, movement and disposal of SBO material in the United Kingdom and were content with the existing measures." Then it had gone on about how there had been tests to ensure that there was no contaminated animal feed in the system, and how all tests had shown negative results. That was on 23 September 1994. I then notice that in April in the following year, 1995, new measures were introduced on the staining of specified offal—SBOs—reviewing the controls on SBOs to consolidate and strengthen them, introducing new EC standards at specified plants. In the library brief it comes after the statement: "However we are continuing to see cases of BSE in animals born after the introduction of the feed ban in July 1988. This suggests that there has been some continuing leakage of BSE-infected material into the animal feed system." In other words, I was being assured by Ministers, following upon anonymous information—I will not explain to you who came to me with the information—about the allegation that there had been cross-infection. Ministers were denying it, and yet within a matter of six months we have regulations introduced actually to separate with dye the different forms of offal because of the problems of cross-contamination. Is this normally the way Members of Parliament are going to be kept informed of difficulties that might arise?

(*Mr Hogg*) Chairman, there are two points which come under the sort of general mantra of cross-contamination. I shall ask Mr Meldrum to go into the detail. As far as the SBOs, the offals themselves, are concerned, they are wholly removed and destroyed, but you will have noticed last week, in the recommendation that we brought to the House from SEAC, that we shall eliminate from the feed rations for all farm animals mammalian protein. The reason for that is the risk of cross-contamination. It can happen in two ways. It can either happen in the feed mills themselves, or it can happen on the farm in the sense that rations, say, for pigs or poultry, not intended for cattle, could conceivably be used for cattle, either intentionally or accidentally. Now that is an area of cross-contamination that we have expressly addressed in last week's statement, and we will be moving with regulations as fast as we can. On the narrow point of the staining of SBOs, if I might I will ask Mr Meldrum to respond.

178. Were you responsible for the reply to me from the Earl Howe, giving me his assurances?

(*Mr Meldrum*) I am happy with those assurances, although I was not providing them myself. I will tell you why. Because from the point of view of human health, first of all, we have no evidence of SBOs getting into the human food chain until the first fragment of spinal cord was found in a carcass in July 1995. As the Minister has just said, the problem that we were facing was a cross-contamination problem in feed mills. I was criticised roundly by an agricultural organisation two years ago for suggesting that there could be accidental cross-contamination in feed mills

[**Mr Campbell-Savours** *Cont*]

because meat and bone meal was getting into cattle rations when it was intended for use in pig rations. We have developed since that time some additional testing regimes on a totally novel testing system which gives a very clear, though small, indication that on occasions there is cross-contamination in feed mills. Therefore, I am content with and support therefore the recommendation of Professor Pattison's committee, the SEAC, that we should ban meat and bone meal in any rations for farm livestock for that reason. So there was no attempt at all, I can assure you, by those who drafted the answer, to mislead. As far as the new stain was concerned, that was done so we did have a very definitive means by which we could separate, if you are doing a check, those SBOs which are stained with one dye—a blue dye—and those other materials, waste materials, which are used to produce meat and bone meal with a different dye. We wanted to ensure that that separation took place and could be checked the whole way through from the slaughterhouse, in transit to the processing plant. As to why we brought in new standards, those new standards were brought in on a European basis because of the studies that had been commissioned on the inactivation of the BSE agent in a variety of types of protein processing systems. Quite clearly, some were deficient, and a Commission decision was taken in Brussels, applying to all Member States, which we implemented and which came into effect by the end of last year, 31 December 1995. That is why we brought in a new processing standard, bearing in mind, of course, that we still had in place a ruminant protein ban for ruminant rations.

179. Can you see what I am driving at? That is, that there may well have been contamination of feed—not feed stored in 1989, but feed actually produced after that date—contaminated feed getting into the animal food chain, therefore that date in 1989 may not actually be as valid as we are led to believe. It might be that some cases of BSE arise out of the feeding of this feed, contaminated and produced after 1989, to animals, and that might even lead to a later incidence of CJD.

(*Mr Meldrum*) I will not comment upon CJD, that is not my province. As far as the disposal of the SBOs is concerned, those tissues are likely, based on our work, to contain infectivity. There are three stages to the system. They are separated in the slaughterhouse and should be kept separate both from the human food supply, which they are, and from the animal food supply. Thereafter they go into a protein processing plant where they are heat-treated. Then they are moved into a factory where they are mixed. Yes, we believe that there have been problems in protein processing plants. That is why we have tightened up the system. We are not totally content yet, though, that those systems will inactivate all agents present, BSE or scrapie. Also we found cross-contamination in the feed mills. But it is fair to say that if there had been a perfect separation of the materials, which can cause cross-contamination, in the slaughterhouse, and they had been kept totally separate from other materials going into meat and bone meal, that cross-contamination should not have occurred. We certainly accept that there had been some leakage, and that is why we tightened up so much on the production of meat and bone meal certainly and we ensure that SBO material is destroyed.

Chairman

180. I realise that I am at great risk in inviting any other questions, but does anyone have any more questions to ask?

(*Mr Hogg*) Can I make point here, Chairman? I have a bit of information in response to Mr Martyn Jones. You may recall that he suggested that the Italians had seized two consignments of lamb. Would it help the Committee if I indicate my understanding of the position?

181. Yes, please do.

(*Mr Hogg*) It is true that there were two consignments of lamb which were stopped in Italy. I understand that these were routine checks carried out for the purposes of checking for salmonella. The checks proved negative, the consignments were released, and the checks had nothing to do with BSE. That is my information today.

Chairman: Thank you.

Mr Bayley

182. Perhaps it is appropriate that this is the last question, Chairman. On 7 December the Prime Minister told the House of Commons, "There is no scientific evidence that BSE can be transmitted to humans or that eating beef causes CJD. That is not in question. The Chief Medical Officer's advice is clear. There is no evidence that eating beef causes CJD in humans." Given the information we now have from SEAC about the ten cases where the most probable explanation is that there has been a transmission of BSE to humans in the form of CJD, would the Chief Medical Officer now modify the advice that he has given, or qualify the advice that he was giving at that time in December?

(*Sir Kenneth Calman*) The identification of the ten cases which has been produced over the last two or three weeks I think is an important issue for us to look at. That in itself does not change the advice which the Prime Minister gave, because there still is no evidence—indeed, Dr Will has confirmed this—that although a possibility of a link exists, and there always has been the possibility of a link from the very beginning, from the Southwood Report, there is no evidence that that link is there, and the statement which the Prime Minister made in December, as far as I am concerned, still stands.

183. By "evidence" do you mean a 100 per cent proof or something different?

(*Sir Kenneth Calman*) I do not think 100 per cent proof is necessarily evidence. I think what you are asking me is, is there any evidence which I have seen which shows that there is a clear link between BSE and human disease, particularly CJD. The answer is, I have seen no evidence for that.

Audrey Wise

184. This is purely for factual information. On 20 March, Mr Dorrell, you announced that £4½ million would be authorised out of the NHS research budget for research on this matter. Was that the first money to be so allocated? Then on 25 March, in your further statement, you also mentioned research funding. Was that a reference to the £4½ million, or was it to some other additional money which is planned?

(*Mr Dorrell*) The only specific additional money that I have authorised for this so far is the £4½ million that I referred to in the first statement. In the second statement I stressed that we were going to continue to support Dr Will's surveillance unit at Edinburgh, that being part of continuing to ensure that we have the information available to allow us to improve our understanding of these conditions; and that I had also asked Professor John Swales, who is the Director of the NHS Research and Development Directorate, to prepare a planned research programme to ensure that we do not merely adopt a reactive role to the financing of research projects in the field, but that we programme research and we indicate clearly the areas of research activity where there is a priority to have some questions answered. He is in the process of preparing that plan. Indeed, I expect to receive, at least in outline, details of that during the month of April. I have allocated the extra money—the £4½ million—but I have also made it clear that if there are necessary and important research projects that take us above that figure, then the Government stands ready to finance it.

185. I am sure we welcome the £4½ million. Could you just tell me, was that the first specific funding for research in this matter?

(*Mr Dorrell*) No, it is not. We currently spend around £9.3 million—and I have a sheet of paper in front of me on this—which is roughly £½ million from the Department of Health, £5.4 million from the Ministry of Agriculture, £2.6 million from the BBRC which is the Biosciences Research Council, and a further sum from the Medical Research Council (I do not have the figure that they are spending in the current financial year, but last year it was £800,000). The total, we estimate, if the MRC spend in the current year the same as they spent last year, would amount to £9.3 million.

186. So that is in one year?

(*Mr Dorrell*) That is a single-year spend.

Chairman: Ministers, gentlemen, thank you for tolerating us for a very long day. I would hope you might think we have saved time, although it does not seem like it at the moment, in having a joint meeting. At least we have saved you having to be asked the questions twice over. We have been very widely informed, and we are very grateful to you indeed for that, on what is a very important issue. Thank you very much indeed.

Joint memorandum by the Ministry of Agriculture, Fisheries and Food and the Department of Health

BSE and CJD (T4/BSE11)

INTRODUCTION

1. This memorandum is in response to points raised by the two Committees during oral questioning on 27 March, concerning BSE and CJD.

FURTHER INFORMATION

2. The Committees have raised five further areas upon which they required information.

Rationale for Mouse Bioassays

3. First, the Committees asked about the financial and scientific rationale for the two-stage experimental process to determine the infectivity of the tissues of clinically affected cattle as described by Dr Kimberlin. The decisions as to what model should be used were not primarily directed by cost. The most important factor, once strains of mice had been identified as being susceptible to both scrapie and BSE, was that we had a model that would permit direct comparisons between scrapie and BSE infection. In addition, subject to the limitations of accommodating large numbers of transmissions and establishing them rapidly, the Ministry was able to make use of the expertise of the experts at the NPU by doing the work in mice.

4. Another major factor for the use of mice meant that results could be obtained quickly, between one and three years in mice, whereas similar studies in cattle would have taken between two and seven years. The main concern with using the mouse model as opposed to cattle was the possibility of missing out on some potentially low levels of infectivity. We are now attempting to quantify the degree of sensitivity lost by using mice rather than cattle, and will be putting some additional tissues into cattle from the pathogenesis study. It is unnecessary and would prove extremely expensive to use cattle routinely for all tissue studies even if suitable accommodation could be identified.

Compliance of Slaughterhouses with SBO Controls

5. Second, the Committees asked for information on the level of compliance at slaughterhouses with all the regulatory controls relating to the prevention of SBOs entering the human food chain since 28 November 1995.

6. During the period 1 December 1995 to 31 March 1996 there have been 1,224 unannounced SVS inspections of slaughterhouses of which 67 (5.5 per cent) were unsatisfactory. The following were the reasons for failure as at 25 March 1996:

13 inspections indicated that SBO was not adequately separated from other animal by-products.

11 inspections indicated that the storage of SBO was unsatisfactory.

18 inspections indicated that staining of SBO was unsatisfactory.

11 inspections indicated that SBO was not adequately separated from material for human consumption.

16 inspections indicated that record keeping was unsatisfactory.

3 inspections revealed that SBO was not moving to appropriate destination.

8 inspections indicated that the weight of SBO removed was not consistent with the number of animals processed.

The 11 inspections which identified SBO as not being adequately separated from material for human consumption can be broken down as follows:

6 inspections identified that the spinal cord had not been properly removed from the carcases (4 inspections identified problems with only 1 carcase, the other 2 inspections identified problems with 2 carcases);

Three inspections identified that small portions of tonsils were still attached to heads;

One inspection identified that small parts of the thymus were still attached to the trachea; and

One inspection revealed that small amounts of intestines were not being properly separated from associated fat.

7. One plant owner is facing prosecution and a further four are under investigation. Where appropriate, representations have been made by the MHS to plant operators to improve plant conditions and inspection facilities, eg lighting.

8. The MHS currently employs 510 red meat inspectors on a full time basis. In addition it utilises 57 red meat inspectors on fixed term contracts of 100 weeks duration. There is also a pool of 210 red meat staff used on a casual basis. These last two figures in particular fluctuate according to demand. These meat inspection staff are supervised by Official Veterinary Surgeons (OVS). The MHS directly employs 39 OVS and in addition contracts out work to 110 full time equivalent vets to supplement them. Three vets are used on a casual basis. According to MHS data, 412 slaughterhouses deal with cattle.

Bioassay of faeces of clinically infected cattle

9. Thirdly the Committees asked for confirmation that tests on the infectivity of the faeces of clinically affected cattle have proved negative. These tests have not yet been carried out on faeces from clinically affected cattle, but will be included as part of the next phase of the pathogenesis experiment. It was not possible to test all tissues at bioassay at one time, and consequently the work on tissue infectivity had to be prioritised in terms of identifying tissues that were most likely to present a risk to consumers or most likely to carry infectivity. Nevertheless, the epidemiology of the disease in cattle shows no evidence of horizontal cattle to cattle transmission as would be the case if faeces or urine were infective.

Development of live tests

10. Fourthly, the Committees requested further information on research carried out over the last 10 years into a live test to detect BSE in cattle or CJD in humans and the prospects for developing a live test.

Background—post mortem tests

11. Histological examination of a brain section, taken from the brain stem, is necessary for diagnosis of most cases of BSE. This is of course done after death. Diagnosis of CJD can sometimes be confirmed while human patients are alive using brain biopsy, but this option is not appropriate in the case of animals. Most importantly, it is clear from studies on field cases, and more particularly the pathogenesis experiment where cattle were slaughtered sequentially after experimental oral infection, that detectable changes only occur in the brain tissue within 2-3 months of onset of clinical disease. Histological examination would therefore be of little value in young animals (prime beef). Brain biopsy would therefore not enable potential cases to be identified, even if it were ethical to use the procedure in this way.

12. It is possible to carry out tests which involved detection of abnormal PrP (or prion protein) in the brain tissue of infected animals. From experimental models this would be detectable before the normal pathological changes are detected, but these involved use of limited resources of specific antibodies. They therefore tend to be used as research tools. In addition, unless the abnormal PrP can be regularly identified in easily accessible non-neural tissue this test could only be used post mortem.

Tests in live animals

13. Research is in progress with a view to developing tests that will identify clinically normal but infected animals, but these are a long way from being of use in screening animals prior to slaughter. In February 1995 external reviewers of the MAFF funded research were not optimistic about the success of this work in the immediate future.

14. There are several strands to this work, and the following briefly identifies these areas and the likely success of a breakthrough.

(a) Urine test[1]—changes have been detected in the urine of CJD patients, sheep with scrapie and cattle with BSE. The changes have not been fully characterised, but significant progress has been made with respect to BSE. Three molecules appear in urine in quantities that are not seen in normal, unexposed, cattle. Two of the molecules have been identified, and the third is in the process of characterisation (there are some 150 candidate molecules). Once this has been done it should be possible to identify the source tissue, and to identify a more appropriate fluid to collect for analysis (such as blood). Most of the work has been done on clinically affected animals so far, but it can produce a small number of false positives and false negatives. Further work is in progress to analyse a large number of samples from field cases to validate the test, but this will still not confirm that the test can be used with confidence before the onset of clinical disease. This is likely, but progress will be partly dependent on the resources applied to it.

[1] The urine and CSF tests are the most likely to prove of use for testing of clinically normal cattle, but we still face a considerable lead time in validating the research and developing tests.

(b) **Dr Harash Narang** has stated that he has developed a test which involves examining a specimen of concentrated urine under an electron microscope to detect a structure called nemavirus. The Committees discussed this with Dr Narang at the hearing on 17 April. Arrangements are in hand for Dr Narang to be supplied with samples of both human and bovine urine. MAFF has proposed to Dr Narang that these should be used in a blind trial.

(c) **Cerebro-spinal fluid (CSF),** which nourishes brain tissue, has been identified as containing some abnormal proteins in the event of damage to the brain or spinal cord. Some work has been done in humans with CJD. This was pioneered in the USA about 10 years ago but has only recently been successfully replicated and extended. The researcher concerned has been supplied with samples from the National CJD Surveillance Unit to test. At the VLA, Weybridge efforts are concentrating on identification and characterisation of proteins in the CSF of cattle affected with BSE. Some of this work has been in collaboration with Electrophoretics International, but significant advances had been made by staff at VLA beforehand. There appear to be changes in the CSF in BSE cases, which appear not to be present in cattle in New Zealand (assumed to be unexposed), or in cattle affected with other neurological diseases similar to BSE. The tests used are laborious and expensive, so the aim is to characterise the disease specific protein, if that is what it proves to be, and identify its source so that samples can be collected elsewhere (hopefully from blood). Once the protein is characterised it should be possible to produce antibodies to the protein, and develop a serological test for infection. This will however take several years.

(d) **Changes in blood metabolites**—while there are fluctuations in quantities of certain metabolites in blood in clinically affected animals, and they are different in degree from animals affected with other neurological diseases, they are unlikely to be diagnostic in themselves. Their promise is that they offer a means of differentiating between true BSE affected suspects and those affected with other neurological diseases.

(e) **Detection of abnormal PrP**—as mentioned above, by producing antibodies to the abnormal protein it should be possible to detect it in peripheral tissues. Unfortunately the sensitivity of this approach is limited due to the fact that there is no difference in protein composition between normal and abnormal PrP. The difference appears to be entirely structural, with the proteins being folded differently, and the crucial breakthrough will be production of an antibody that can differentiate between the two. This is not on the immediate horizon although work is in progress at the VLA and IAH along two different avenues of investigation.

(f) **Detection of nucleic acid**—no nucleic acid that is specific to BSE or scrapie has not yet been detected. Should it prove to be possible to identify a DNA or RNA that forms part of the agent there are very sensitive techniques (PCR) that could then be applied to detecting the nucleic acid in tissue. Since no agent genome has yet been identified there can be no diagnostic test available in the immediate future.

(g) **Heart rate and rumination monitors**—two relatively crude tests but which appear to be very effective have been developed at the VLA for use on clinically affected animals only. The first monitors heart rate, which in BSE cases is depressed despite their excitable nature. The second monitors the frequency of rumination, which is also depressed. BSE affected cows eat and swallow normally but seem unable to regurgitate a bolus to chew the cud as easily as the normal cow. Neither of these is likely to be of use in preclinical cases.

(h) **Clinical signs**—In humans diagnosis can be made on the basis of clinical presentation, although this requires confirmation by biopsy and finally by post mortem. In many cases of CJD, the electroencephalogram (EEG) recording of the brain is characteristic and is a useful adjunct to diagnosis. Although a rare disease, the clinical signs of classic CJD are documented in medical literature. The national CJD Surveillance Unit recently wrote to all neurologists giving details of the presentation of the new variety of CJD to assist in early identification of possible cases.

Research

15. It is not possible to provide details of all the research that has been undertaken on a live test since computer records are only available from 1991. A list of research for a BSE test for use on the live animal is attached at annex A. It indicates the title of the research, the cost, the date and the place where the research is/has taken place. Much of the research detailed in the table is a carry over of research which was underway prior to 1991. Research into a test for humans is briefly described above. The establishment of the National CJD Surveillance Unit in 1990 has greatly improved the ascertainment of the disease.

Bovine Serum

16. Finally, the Committees requested information on the position regarding the use of bovine derived serum. The position here is the same for other pharmaceutical products using bovine derived materials. In February 1989 the Southwood Report recomended that the Committee on the Safety of Medicines and the Committee on Dental and Surgical materials should consider the implications of BSE for medicines. As a result the BSE Working Party was set up as a joint Working Party of the CSM and the Veterinary Products Committee. Guidelines on the sourcing of bovine materials were subsequently issued to the industry later in 1989. These said that all bovine materials used in medicinal products, including serum, should be sourced from outside the UK and should come from healthy herds which had not been fed material of animal origin. Guidance from the European CPMP on the use of bovine materials in medicinal products was not received until 1991. By this time UK companies had, where necessary, changed their sources.

8 May 1996

ANNEX A

APPROVED RESEARCH SINCE 91/92 CONTRIBUTING TOWARDS THE DEVELOPMENT OF A LIVE TEST FOR BSE

Project Code	*Title*	*Contractor*	*Start date*	*End date*	*Costs 91/2*	*Costs 92/3*	*Costs 93/4*	*Costs 94/5*	*Costs 95/6*	*Costs 96/7*
SC8954	Large scale isolation of abnormal prion protein from BSE-infected tissue	Birmingham University, Chemical Engineering	1st June 1991	31st May 1993	209,213	120,242	45,416			
SE0214	Selective studies of neurological disorders in cattle to aid the clinical differential of diagnosis of BSE	Central Veterinary Laboratory	1st April 1996	31st March 2000						296,604
SE1408	Diagnosis of BSE by detection of abnormal deposits of PrP and other BSE-infection specific antigens	IAH, Neuropathogenesis Unit	1st April 1990	31st March 1994	227,000	238,000	248,000			
SE1409	Development of an antemortem test for BSE and natural scrapie infection through the detection of abnormal deposits of PrP	IAH, Neuropathogenesis Unit	1st April 1994	31st March 1998				250,000	288,260	341,312
SE1411	Further analysis of nucleic acid differences between control and scrapie/BSE infected animals	IAH, Neuropathogenesis Unit	1st April 1994	31st March 1998				85,000	106,575	125,932
SE1702	Evaluation of electrophysiological methods for the study of BSE	Central Veterinary Laboratory	1st April 1992	31st March 1993		20,000				
SE1703	A study of the diagnostic potential of behavioural abnormalities in BSE	Central Veterinary Laboratory	1st April 1992	31st March 1993		59,000				
SE1704	Diagnostic potential of the pharmacological manipulation of clinical signs of BSE	Central Veterinary Laboratory	1st April 1992	31st March 1993		45,000				
SE1705	Electrophoretic analysis of body fluids to identify diagnostic markers in BSE and scrapie	Central Veterinary Laboratory	1st April 1992	31st March 1996		90,000	96,000	115,000	138,489	28,300
SE1706	Identification of BSE and scrapie infected animals by the detection of a urinary metabolite	Central Veterinary Laboratory	1st April 1992	31st March 1995		123,000	85,000	86,000		
SE1708	Biochemical approaches to the differential diagnosis of BSE in the live animal	Central Veterinary Laboratory	1st November 1992	31st March 1995		38,000	74,000	75,000	0	
SE1711	Establishment of in-vitro culture of bovine cells for veterinary research	Central Veterinary Laboratory	1st January 1993	31st March 1994		87,000	0	0		
SE1712	Production of sera suitable for the development of immunodiagnostic tests for transmissible spongiform	Central Veterinary Laboratory	1st April 1992	31st December 1993		18,000	15,000			
SE1714	Scale up of isolation of abnormal PrP from BSE infected tissue	Birmingham University	1st June 1993	31st May 1994			112,028	80,022		

[Continued

APPROVED RESEARCH SINCE 91/92 CONTRIBUTING TOWARDS THE DEVELOPMENT OF A LIVE TEST FOR BSE

Project Code	Title	Contractor	Start date	End date	Costs 91/2	Costs 92/3	Costs 93/4	Costs 94/5	Costs 95/6	Costs 96/7
SE1715	Molecular characterisation of a single stranded (SS) DNA observed in scrapie infected hamster and mouse brain	London Hospital Medical College	1st August 1993	31st July 1994			0	59,900		
SE1718	Identification and characterisation of the scrapie agent from a low protein, high infectivity fraction of brain	IAH, Compton Laboratory	1st October 1995	30th September 1999					51,765	123,760
SE1720	Antibodies against the BSE agent	Reading University, Department of Microbiology	1st February 1994	31st January 1995				0		
SE1722	The molecular nature of the agents of transmissible spongiform encephalopathies	IAH, Compton Laboratory	1st October 1994	30th September 1995				15,258	46,740	
SE1723	Production of polyclonal antisera to highly purified bovine PrP scrapie	Central Veterinary Laboratory	1st April 1995	31st March 1996					167,155	
SE1724	Biochemical changes in the spongiform encephalopathies	Central Veterinary Laboratory	1st April 1995	31st March 1996					191,057	
SE1725	Studies of the enhancement of reproducibility of PrP Scrapie detection after cold storage of scrapie affected tissue	Central Veterinary Laboratory	1st April 1995	31st March 1998					50,103	19,302
SE1726	Continued large scale isolation of abnormal PrP from BSE affected tissues	Birmingham University	1st June 1994	31st May 1995				69,358	49,542	
SE1727	Identification of putative NA components of the etiologic agent of the transmissible spongiform encephalopathies	University of Edinburgh, Centre for Genome Research	1st June 1995	31st May 1996					86,658	34,658
SE1728	Production and properties of PrP:EC IVth framework collaboration	Central Veterinary Laboratory	1st April 1996	31st March 1999						92,493
SE1730	Electrophoretic analysis of body fluids to identify disease-specific proteins in ruminant spongiform encephalopathy	Central Veterinary Laboratory	1st April 1996	31st March 1999						151,520
SE1731	Production of polyclonal antisera to highly purified bovine PrP scrapie [was SE1723]	Central Veterinary Laboratory	1st April 1996	31st March 1997						167,022
SE1732	Collaboration agreement with Electrophoretics International to develop 2DE techniques for the diagnosis of BSE	Electrophoretics International Plc	22nd December 1995	31st December 1996					0	0
SE1914	Sequential observations of neurological signs in BSE	Central Veterinary Laboratory	1st April 1992	31st March 1995		37,000	175,000	91,000	0	
SE1917	Scrapie infection of sheep neural and extraneural cell lines	Moredun Research Institute	1st January 1994	31st December 1996			0	33,538	32,200	33,920
				GRAND TOTAL	436,213	788,242	938,444	960,076	1,208,544	1,414,823

[Continued

WEDNESDAY 17 APRIL 1996

Members present:

AGRICULTURE COMMITTEE	*HEALTH COMMITTEE*
Mr Richard Alexander	Mr Hugh Bayley
Mr D N Campbell-Savours	Mr David Congdon
Mr Ieuan Wyn Jones	Alice Mahon
Mr Martyn Jones	Mrs Marion Roe
Mr Edward Leigh	Rev Martin Smyth
Sir Roger Moate	Mr John Whittingdale
Mr Colin Pickthall	Audrey Wise
Mr William Powell	
Sir Jerry Wiggin	
Mrs Ann Winterton	

Mrs Marion Roe was called to the Chair

Memorandum by Dr Stephen Dealler [T7/BSE4]

1. It was possible to calculate fairly inaccurately the number of infected cattle that were being eaten in 1989. Much better data was available later and hence much more precise figures could be produced by 1992. My own data became available in 1993 and, taking underestimates whenever there was any doubt about accuracy. This showed that approximately seven infected cattle were being eaten for every one that was slaughtered with the disease. It agreed with the data from Dr Richards at CVL that showed that two adult cows were slaughtered while infected for every case seen.

2. It was possible to estimate the amount of infectivity that was being eaten by the human population in 1989 also and much more accurate figures produced by 1993. These data were made more accurate again in 1995 and the data are enclosed. It should be made clear that underestimates are used throughout the mathematical procedure and that statistical work was fully reviewed. This shows that, even taking the SOB into account, a major risk to human life was being taken.

3. It was possible to realise that the mouse inoculation test to assess the amount of infectivity present in tissue would be inadequate from the beginning of the experiment. Even if a mouse, inoculated with some tissue did not die, there could be 300,000,000 IU present in a single meal made of it. The mouse inoculation tests depend on the mouse being highly sensitive to the disease and unless this is known, it is impossible to state that there is no infectivity present. Specific chaemeric mice being grown by the Californian group may be able to produce highly sensitive mice. Hamsters are not infected by BSE at all. If they had been used for the experiments then it would have indicated that there would have been no infectivity in brain or spinal cord. Despite the simple mathematics statements were still made that there was no infectivity in various tissues. Infectivity has been found in almost all the tissues tested of various species. This includes meat, liver, lung, kidney, nerve and other tissues that continued to be eaten after the SOB. My calculations were carried out in order to assess whether the infectivity that should have been expected to be in the tissues would represent a risk to humans. It is unlikely that the mouse inoculation test would show any infectivity in the feed given to cattle.

4. MAFF currently claims that vertical transmission is not taking place to any great degree in BSE and that the results of the experiment looking at the offspring of BSE cattle to see if they will die of BSE will be able to decide this. It should be made clear to the Committee that this is not correct. This will probably have to be explained. I include information showing that the epidemiology of BSE currently fits much better with the cases of BSE that we see being the offspring of dams that had become infected from feed. MAFF experiment will not be able to separate this potential epidemiology from that of cattle being infected after the feed ban from feed.

5. The evidence concerning the apparent under-reporting of cases of BSE to veterinary officers should be explained. It depends on statistical evidence derived from MAFF data and using standard techniques. As a result of these data it is now difficult to state that the number of cases of BSE is in fact actually falling. The statistical estimate figures suggest that it is but at a lower rate than suggested by MAFF data.

6. It can be shown statistically that (assuming that there will be no more cases of BSE born after 1991, that all cattle with BSE were reported, fully diagnosed before 1992, assuming that humans have only ever eaten similar tissues to those eaten after the SOB, and assuming that the disease is cumulative) there is minimal

advantage for UK adults to stop eating bovine products, whatever the level of infectivity in the tissues, and whatever the amount needed to infect the human. This cannot be shown, however for children and would not be valid if the disease was not cumulative.

7. The level of infectivity that would be considered possible to be in the bovine tissues that were eaten in the UK would be expected to drop dramatically if simply cattle were slaughtered at the end of their working life as dams. Attempts at calculating risk remaining have shown this to be very much lower.

8. The report by the Advisory Committee on Dangerous Pathogens (DoH) on Transmissible Spongiform Encephalopathies in October 1994 showed, quite correctly that we did not know the amount of infectivity in specific tissues of the cow. It required that, because of this the tissues should be handled in a method necessary to protect the worker (this was involving Health and Safety at Work, and should not be considered to apply merely to laboratories), the DoH was aware that specific tissues represented a risk (eg liver). It is not clear why liver (and some other tissues), which had been found to be infective in all species tested except cattle (in which a test of unknown sensitivity was used) was not included in the specified offals in 1989.

9. Various groups have been in touch with me having been told by MAFF that certain tissues would not be expected to contain any infectivity at all. They told me that Mr Taylor has been telling them that liver was not found to be infective in any other species. As this is the actual opposite of the truth, could I ask that the Committee ask for data from MAFF indicating which tissues have been found to be infective in which species. I include a simplified form of the data that I have available. It was given, as scientific papers to MAFF in 1990 when I delivered it to Whitehall personally.

10. I should make it clear that I have tried repeatedly over the past 6 years to provide information to the DoH department involved in BSE. This has been extremely difficult. I have been very worried that the information that they had been receiving was misleading in that the announcements from Sir Kenneth Calman were not in keeping with the science of the subject. The reason for this was unclear and attempts to find out why were unsuccessful. Attempts to involve drug companies in research into methods of treatment for BSE were unsuccessful also, specifically because they had been advised by a Tyrrell Committee member that it was not worthwhile. Attempts to approach the Agriculture Commission at the EU were unsuccessful as they also took information from MAFF and advice from similar advisors. Attempts were made to involve the BMA in the ethics of the disease. They were interested but said they would rather keep out of the subject. The same story continued with Royal Colleges, the Central Public Health Laboratory (which appeared to have been told to keep out of the subject), Environmental Health, Public Health groups, the Church of England etc. Considering I am highly qualified and have been working extremely hard on the subject, I was surprised to have only got helpful responses personally and not officially. Although I cannot be sure of this, it seemed that the DoH had taken a particular position and other groups felt inadequate at making any stand. The only group that appeared to be helpful was the Consumers Society. Could I ask the Committee to consider how a researcher should go about getting something done when specific stands have been made by official groups and Government Departments working in private?

11. Specific assumptions taken initially by the Southwood and Tyrrell Committees were reasonable but were unacceptable in public health terms. They have now gradually been shown to be invalid assumptions. When a large proportion of the population may be exposed to a fatal condition to which there is no treatment, assumptions should be made that would avoid potential risk. For instance, if there is inadequate data proving that BSE is either infectious or non-infectious to humans, it must be assumed to be so rather than the reverse.

(a) BSE was derived from scrapie. This now seems unlikely and the pretence that because BSE was just scrapie humans would not get BSE either was always invalid. The repeated announcement that BSE was derived from scrapie should now be withdrawn. Scientific information was available at the time of the Southwood Committee showing that such a logical deduction was never valid.

(b) BSE is derived from the feed that the cows eat. The most fitting epidemiology is now that the cattle we see with disease caught it from their mother and that the mothers caught it from the feed. Further data is required. There may be now data that a second factor is required for infection to take place.

(c) The change in the feed manufacture processes that took place at the beginning of the 1980s was the cause of BSE. There was never any evidence for this and the current evidence from Taylor in Edinburgh shows that this may well not be true. It may simply be that the carcass of a cow with a sporadic spontaneous case of BSE was fed to other cattle.

(d) The feed ban made a large effect on the number of cases of BSE. It is actually quite difficult statistically to show any effect at all initially. The feed ban was in fact just too late when introduced in 1988. By that time the disease was actually running out of cattle to infect in England. I would expect that this will need to be explained as the Committee will probably have been shown data from MAFF suggesting the opposite.

(e) The level of infectivity that should be thought of as acceptable to humans in diet should be 10,000 IU (Tyrrell, 1993, personal communication). This did not seem to take into account specific directives concerning the ration between a dose required to cause disease and the dose that is acceptable. Most work has been done on chemicals in this respect and the ratio between the inoculum for chemical

that caused a fatal, untreatable disease in a test animal and the acceptable dose for a human may be 10,000 to one, ie if 10,000 IU of BSE caused disease in a test animal then an acceptable level in human food might be 1 IU. This clearly would be open to discussion but not seem to be discussed.

(f) That BSE would not be transmitted to further animals and that BSE in cattle would be a "dead end host". This was always incorrect and it is not clear how the Southwood Committee came to the decision. TSEs generally transmit to about 70 per cent of other species. In fact BSE has now transmitted to 19 different species and 17 of them by mouth. Unsupported reports of BSE syndromes in dogs, and cattle have been made.

(g) That BSE would start decreasing in 1992 and would be disappearing by 1995. The peak of the epidemic was probably in 1994 and we will be seeking cases after the year 2000.

(h) That BSE would not be cumulative, ie the infectivity would not build up in the body of a human eating the disease. This has still not been investigated.

(i) That BSE infected cattle would not be entering the human food chain. This was quite ridiculous and the lack of attempts by either the Southwood or Tyrrell Committees to calculate either the number of cases eaten or the risk that this represented must be considered unacceptable. Although the minimum number of cases of BSE entering the food chain could be calculated statistically, this was as such the minimum. To find out the accurate number would require the testing of random numbers of cattle at abattoirs and was suggested by the Tyrrell Committee in 1989. It was never carried out until Narang did so in 1995.

(j) That the regulations introduced by MAFF would be adhered to. Would farmers that knew there was a chance of neurologically affected cattle not being accepted by the veterinary officer always take a cow to the VO? My own experience is that this was never likely and that some would be taken to market because if the VO turned it down it might be worth less than £50. Farmers have been in touch with me stating that VOs have looked at cattle born after the feed ban and actually told the farmer to take them to market as they "couldn't have BSE'. The sheer will of both the MAFF and the trade that BSE would go away must have had an effect. My data (derived from MAFF and using standard techniques) now suggests that large numbers of cattle with symptoms of disease may have been taken to market and these numbers expanded as farmers simply became better at diagnosing the disease early.

(k) That humans would not have been at risk before the SOB. It was always doubtful but the Committees did not seem to ask for any work to be carried out looking for potential methods of treatment.

(l) That the soil on which the animals grazed would not be infected. It is considered that 99 per cent of the oral dose of disease is actually excreted in faecal matter. The possibility that infectivity would remain in the soil must be considered following Brown's report that the agent does not seem to be destroyed in soil. Work from Icelandic groups suggests that infectivity associated with the environment does decay but slowly. The group at the London Zoo realised this fairly early and it has been organised that the top foot of soil from the kudu pen will be removed (personal communication, 1995)

(m) That humans would not develop BSE. Although this has not been proved, it would be a mistake to now not assume it until evidence shows it to be otherwise. The major problem that resulted from this is the advice given to the drug companies and food companies. For instance Glaxo Wellcome were advised not to carry out any research into potential methods of treatment. I attempted to find out how many humans were already infected in 1990 and found it impossible to organise the research. Apparently no similar attempt was being carried out by the CJD Surveillance Unit in Edinburgh.

The thing that joins all of these mistakes is that they were reasonable "best guesses" at the time but were not decisions that would have been taken on public health, medical ethics, infectious disease or medical microbiology grounds. Indeed, at the times, experts in these fields were not present on the committees. The changes in SEAC that took place in December 1995 may have altered this lack of public health advice to the Committee.

Because of the optimistic assumptions apparently taken by the Committees and my own inability to advise the various Government Departments it has appeared that errors have resulted in the current situation.

12. The suggestion that infection would be taking place in "packets" fits the epidemiology. This is, I feel, incorrect. If this was true, the rate in individual herds would rise in parallel to the national rate. Also, as anyone knows who has seen the meal manufactured, the powder that is produced would not contain "packets" but would be spread amongst many members of a herd that was being fed.

13. Mean age showing disease in cattle born after the feed ban has indeed fallen. This was very strange indeed if only becaue the incubation period is inversely proportional to the dose given. By rights, if "packets" were involved there should be no change, and if no "packets" are involved the cattle should have been getting older.

14. "The number of cases of cattle dying of BSE since the feed ban is dropping as a result of the feed ban." There is insignificant data to show this. My own data with Professor Kent showed that this could not be stated and that large numbers of cattle with clinical symptoms of BSE may be entering the food chain. I have had no response from MAFF since that was published. What seems now is that much of the epidemiology data from MAFF since 1992 in cattle born after the feed ban is actually quite difficult to interpret as only until 1993 can be corrected data be calculated.

My own statistics agree that the case numbers are dropping (estimate data) but at a much lower rate than suggested by MAFF. The reason for this can be discussed.

TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES ARE

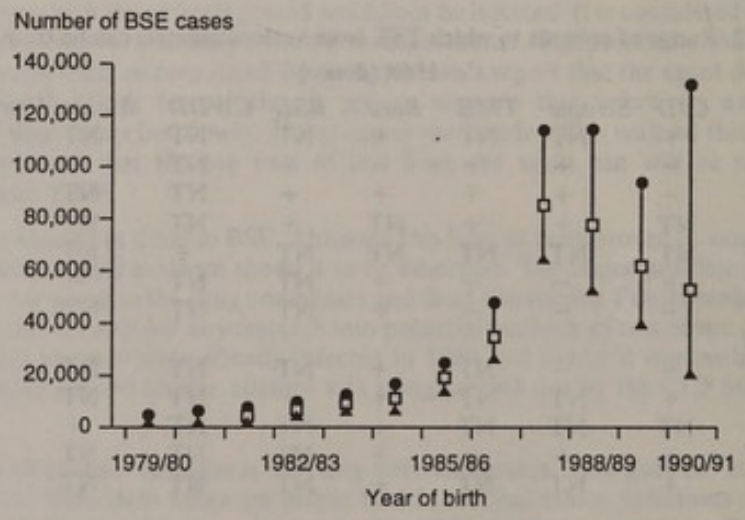
1. Fatal
2. Untreatable
3. Difficult to diagnose
4. Long incubation period
5. Long period of illness
6. Transmissible between species
7. Transmissible in food
8. Not destroyed by domestic cooking
9. Infectivity may be found in all tissues of the body but in much greater quantities in some tissues than in others, eg meat, liver lung, kidney, nerve, blood
10. If adequate amounts are present in the food, a large percentage of the population become infected.
11. Infectivity rises in tissues long before symptoms appear
12. Infectivity is difficult to destroy in the environment
13. May be passed from mother to offspring
14. Animals that are infected may remain asymptomatic throughout their life.

Table 2. Range of animals to which TSE from various animals can be transmitted.

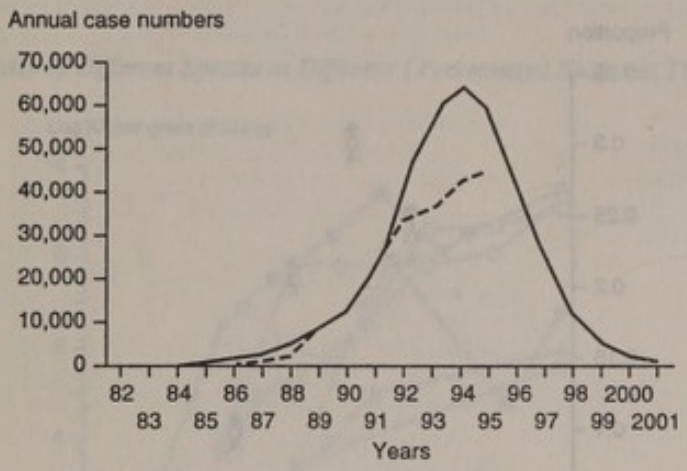
	Host (donor)									
Recipient	*CJD*	*Scrapie*	*TME*	*Kuru*	*BSE*	*CWDD*	*Mouse*	*Hamster*	*Goat*	*Rat*
Human	+	NT	NT	+	NT	NT	NT	NT	NT	NT
Sheep	–	+	+	–	+	NT	+	NT	NT	NT
Mink	–	+	+	+	+	NT	NT	NT	NT	NT
Cow	NT	+	+	NT	+	NT	NT	NT	NT	NT
Deer	NT	NT	NT	NT	NT	+	NT	NT	NT	NT
Chimpanzee	+	–	–	+	NT	NT	–	NT	–	NT
Gibbon	–	–	–	+	NT	NT	NT	NT	NT	NT
New-world monkey										
Capuchin	+	–	NT	+	NT	NT	NT	NT	NT	NT
Marmoset	+	NT	NT	+	+	NT	NT	NT	NT	NT
Spider	NT	NT	NT	+	NT	NT	+	NT	NT	NT
Squirrel	+	+	+	+	NT	NT	NT	NT	+	NT
Woolly	+	NT	NT	+	NT	NT	NT	NT	NT	NT
Old-world monkey										
African green	+	–	NT	–	NT	NT	–	NT	NT	NT
Baboon	+	NT	NT	NT	NT	NT	NT	NT	NT	NT
Bonnet	NT	NT	NT	+	NT	NT	NT	NT	NT	NT
Bush baby	+	NT	NT	–	NT	NT	NT	NT	NT	NT
Cynomolgus	–	+	NT	–	NT	NT	+	NT	NT	NT
Managabey	+	NT	NT	–	NT	NT	NT	NT	NT	NT
Patas	+	NT	NT	NT	NT	NT	NT	NT	NT	NT
Pig-tailed	+	NT	NT	+	NT	NT	NT	NT	NT	NT
Rhesus	–	–	+	+	NT	NT	–	NT	NT	NT
Stump-tailed	–	NT	+	–	NT	NT	NT	NT	NT	NT

	Host (donor)									
Recipient	*CJD*	*Scrapie*	*TME*	*Kuru*	*BSE*	*CWDD*	*Mouse*	*Hamster*	*Goat*	*Rat*
Talapoin	+	NT	NT	NT	NT	NT	NT	NT	NT	NT
Goat	+	+	+	+	+	NT	+	NT	+	NT
Ferret	+	NT	+	+	NT	+	NT	NT	−	NT
Cat	+	−	−	−	+	NT	−	NT	−	NT
Dog	NT	NT	NT	−	NT	NT	NT	NT	NT	NT
Racoon	NT	NT	+	NT	NT	NT	NT	NT	NT	NT
Skunk	NT	NT	+	NT	NT	NT	NT	NT	NT	NT
Mouse	+	+	+	+	+	NT	+	+	+	+
Rat	−	+	NT	−	NT	NT	+	NT	NT	+
Hamster	+	+	+	−	−	NT	+	+	NT	+
Gerbil	+	+	NT	−	NT	NT	+	NT	NT	NT
Vole	NT	+	NT	NT	NT	NT	+	NT	NT	+
Guinea-pig	+	−	+	−	NT	NT	+	NT	NT	+
Rabbit	−	−	−	−	NT	NT	NT	NT	NT	NT
Pig	NT	NT	NT	−	+	NT	NT	NT	NT	NT
Puma	NT	NT	NT	NT	+	NT	NT	NT	NT	NT
Cheetah	NT	NT	NT	NT	+	NT	NT	NT	NT	NT
Kudu	NT	NT	NT	NT	+	NT	NT	NT	NT	NT
Nyala	NT	NT	NT	NT	+	NT	NT	NT	NT	NT
Gemsbok	NT	NT	NT	NT	+	NT	NT	NT	NT	NT
Eland	NT	NT	NT	NT	+	NT	NT	NT	NT	NT
Oryx	NT	NT	NT	NT	+	NT	NT	NT	NT	NT

A: Estimated number of cattle dying of BSE according to year of birth. Bars show 95 per cent confidence interval



B: Predicted cases of BSE reported in specific years assuming that no cattle become infected after 1991.



Key: ——— Calculated number of cases ------ Reported number of cases

Graph A shows that in 1995 it was impossible to say, because of the increasing wideness of the 95 per cent confidence intervals that BSE born in successive years was decreasing at all but it was possible to state that the estimated number was dropping.

Graph B indicates that the number of cases reported in each year would continue to fall but depends on no cases being born after 1991, clearly an underestimate.

Numbers of infected cattle eaten and the point in the incubation period at which this takes place can be calculated using standard methods.

Table III

Ages of Cattle in the UK

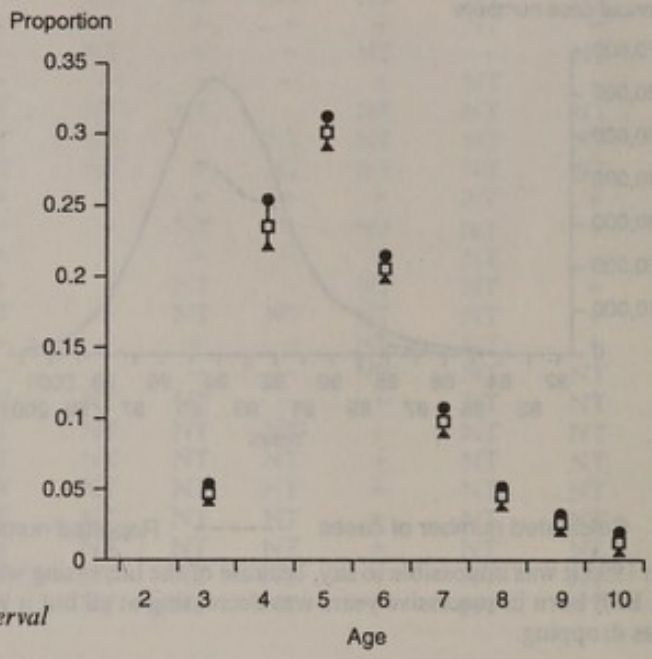
Age (years)	*Number*
<1	3,367,500
1 – 2	2,615,7500
2 – 3	1,420,500
3 – 4	1,285,000
4 – 5	975,000
5 – 6	709,000
6 – 7	531,000
7 – 8	354,000
8 – 9	265,000
9 – 10	133,000
10 – 11	88,500
>11	88,500

Notes:

Data for milking cattle are derived from the Milk Marketing Board National Milk Records Census 1988 and are divided into numbers of cattle in each lactation. The first lactation is taken to be at approximately three years of age. Cattle which are not in milk but pregnant are taken to have a similar age distribution as those which are lactating.

Data for the number and age of young cattle and cattle in beef herds for slaughter are taken as averages from the Meat and Livestock Commission December and June Census reports 1986–1992. Cows and heifers in milk from beef herds are taken to have a similar age distribution as those from milking herds. The age distribution of bulls for service is unclear but they represent less than 1 per cent of the bovine population.

Figure 2. *Proportion of Cattle dying of BSE at different ages (Appendix 1). Bars show 95 per cent confidence intervals.*



Key:

● *Top of confidence interval*
□ *Estimated value*
▲ Bottom of confidence interval

NOTE: For a statistical analysis see Appendix 1

The age distribution of cattle is known and hence the ages at which cattle are slaughtered. The age distribution of cattle developing BSE has remained remarkably similar during the epidemic. Hence the numbers of cattle that would have developed BSE, had none been slaughtered for consumption can be easily calculated.

Levels of SE infectivity in animal tissues

Tissue	*Species tested*					
	Sheep	*Mouse*	*Hamster*	*Goat*	*Mink*	*Human*
Muscle	< +	NT	+	+	+ +	NT
Brain	+ + +	+ + +	+ + + +	+ + +	+ + +	> +
Spine cd	+ + +	+ + +	NT	NT	+ + +	> +
Nerve	+ + +	+ +	NT	> +	> +	> +
Adrenal	+ +	NT	NT	> +	NT	NT
Spleen	+ + +	+ + +	+ + +	+ + +	+ + +	> +
Pituitary	+ +	NT	NT	> +	NT	NT
Lymph	+ +	+ + +	NT	+ + +	+ +	> +
Liver	> +	> +	NT	> +	+ +	> +
Kidney	< +	> +	NT	NT	+ +	NT
Gut	+ + +	+ + +	NT	+ + +	+	NT
Salivary	< +	NT	NT	> +	+	NT
Pancreas	< +	NT	NT	> +	NT	NT
Thymus	NT	+ + +	NT	+ +	> +	NT
Lung	< +	+ + +	NT	NT	+ +	NT

An attempt has been made to indicate the approximate levels One + has been added when the level is measured in a different species < + indicates that the test has been carried out in another species and hence cannot be said to be negative > indicates greater or equal.

Levels of infectivity found in the brain and spleen during the incubation period of spongiform encephalopathies in animals.

Figure 1. *Brain Infectivity of Different Species at Different (Percentage) Stages in Their Incubation Periods*

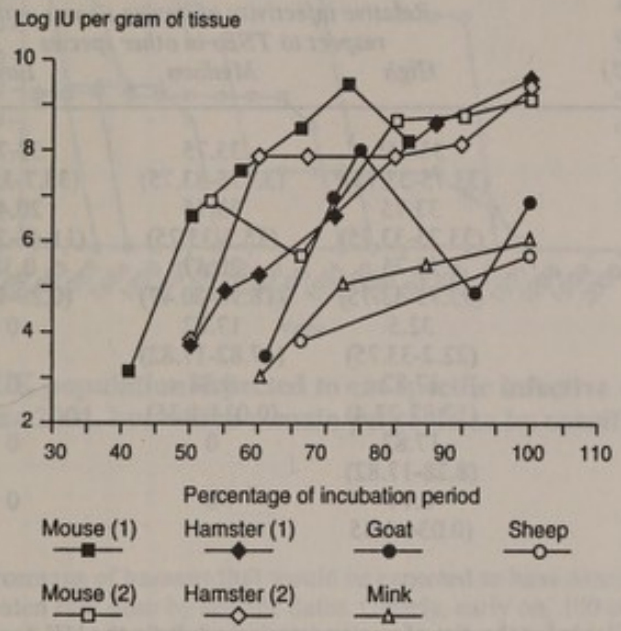
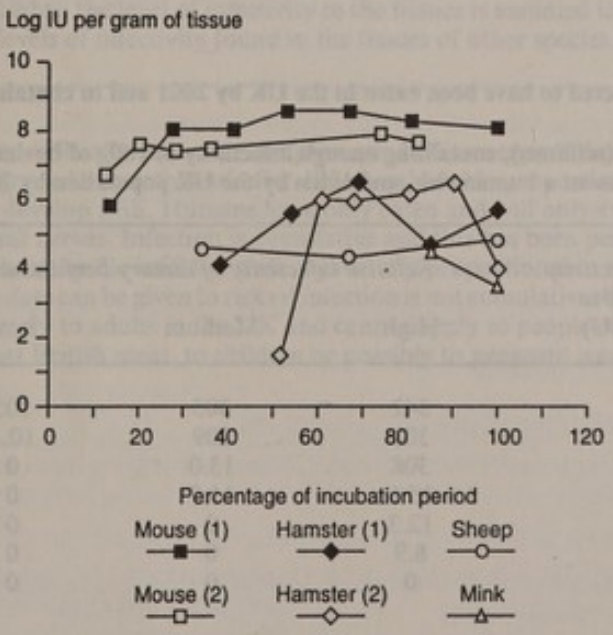


Figure 2. *Spleen Infectivity of Different Species at Different (Percentage) Stages in Their Incubation Periods*



The levels are measured in infective doses (IU), where one IU is the minimal amount of infectivity required to transmit the disease by injection into another animal of the same species. N.B. results for sheep and goats were found by inoculation into mice and so the true levels should be considered to several orders of magnitude higher than shown.

The levels in other tissues are not known through the incubation period to any accuracy but should be considered at this point to rise in parallel to either brain (nervous tissue) or spleen (tissue outside the nervous system).

Numbers of adults in the UK expected to have eaten a specific cumulative dose of BSE by 2001

Table IX. Adult population numbers (millions) in the UK that would be expected to have eaten a potentially fatal dose of BSE by 2001

Infective dose (IU)	*Relative infectivity of bovine tissues with respect to TSEs in other species*		
	High	*Medium*	*Low*
10^3	33.75[a] (33.75-33.75)[b]	33.75 (33.75-33.75)	33.75 (33.7-33.75)
10^4	33.75 (33.75-33.75)	33.75 (25.5-33.75)	20.47 (11.44-28.35)
10^5	33.75 (33.75-33.75)	20.47 (18.98-20.47)	0.38 (0.29-1.35)
10^6	32.5 (22.2-33.75)	17.82 (17.82-17.82)	0
10^7	17.82 (17.82-23.4)	0.38 (0.034-1.35)	0
10^8	17.82 (8.28-17.82)	0	0
10^9	0.14 (0.034-1.35	0	0

Notes:

[a] Statistics are only available for the diet of non-vegetarian adults in the UK aged between 16 and 59 years (33.75 million).

[b] Figures in brackets represent the upper and lower 95 per cent confidence intervals. Figures are often the same due to the effect of exponential change in doses.

Numbers of meals expected to have been eaten in the UK by 2001 and to contain specific doses of BSE

Table VIII. UK meals (millions), containing enough infectivity in 100g of bovine tissue to individually represent a human risk, and eaten by the UK population by 2001

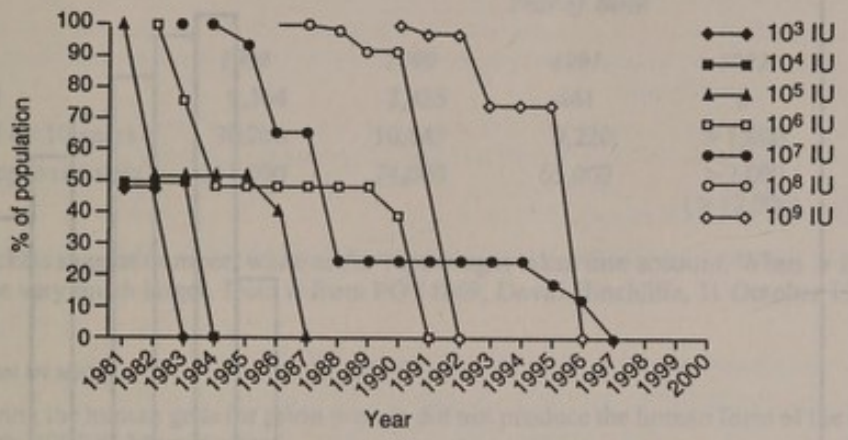
BSE infectivity contained in a meal (IU)	*Relative infectivity of dietary beef tissue*[a]		
	High	Medium	Low
10^3	342	305	305
10^4	308	209	10.3
10^5	308	13.0	0
10^6	15.0	11.7	0
10^7	12.3	0	0
10^8	8.9	0	0
10^9	0	0	0

Note:

[a] The figures represent the number of human meals calculated to contain more than the number of IU shown in the left hand scale. For instance, if 10^7 IU is the infective dose for humans then 12.3 million individual meals would be expected to contain more than this if bovine tissue is relatively highly infective when compared to TSEs of other species.

Assumptions made up to 1991 all cattle with symptomatic BSE are reported to MAFF, are accepted by veterinary officers are diagnosed correctly and after 1991 a calculable proportion are under-reported. No cattle born after 1991 ever develop BSE. Humans have only eaten and will only ever eat meat, liver, kidney and 10 per cent of peripheral nerves. Levels of tissue infectivity are assumed to be either the highest, lowest, or geometric mean of those found in animal tissues and to rise either in parallel to spleen or brain.

If BSE risk to humans is cumulative then little extra risk remain to adults in the UK from eating beef. This statement does not depend on either the infective level in the tissues or the dose required to infect.



Percentage of UK population expected to eat specific infective doses of BSE by the year 2001, but which remain yet to do so by specific years

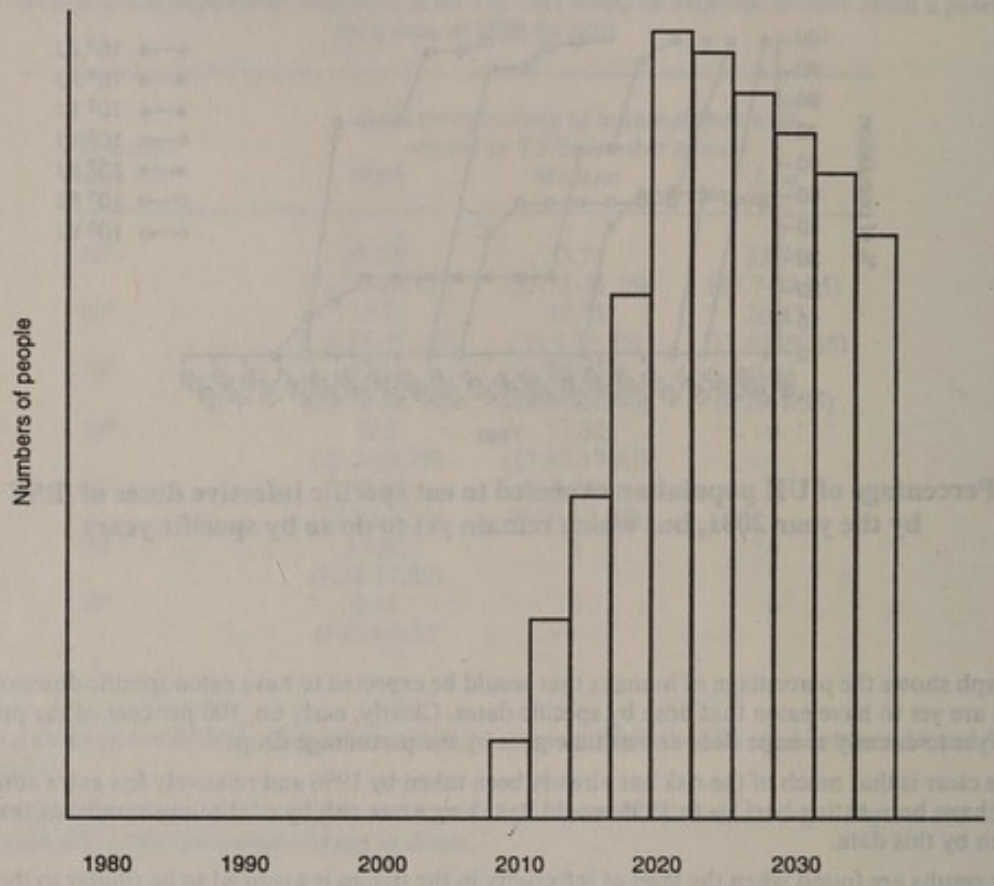
The graph shows the percentage of humans that would be expected to have eaten specific doses of BSE by 2001 that are yet to have eaten that dose by specific dates. Clearly, early on, 100 per cent of the population would be yet to eat any specific dose and as time goes by the percentage drops.

What is clear is that much of the risk has already been taken by 1996 and relatively few extra adults in the UK, that have been eating beef up to 1996 would be taking extra risk by continuing ie most of the risk has been taken by this date.

Similar results are found when the level of infectivity in the tissues is assumed to be similar to the highest, lowest or geometric mean levels of infectivity found in the tissues of other species.

Assumptions made: Up to 1991 all cattle with symptomatic BSE are reported to MAFF, are accepted by veterinary officers, are diagnosed correctly and after 1991 a calculable proportion are under-reported. No cattle born after 1991 ever develop BSE. Humans have only eaten and will only ever eat meat, liver, kidney and 10 per cent of peripheral nerves. Infection is cumulative and this has been persumed as it is considered correct by many scientists because the infective dose is inversely proportional to the incubation period and no immunity is formed. No date can be given to risks if infection is not cumulative and risk must be considered to continue. Results only apply to adults in the UK and cannot apply to people entering the UK, to people in countries that may import British meat, to children or possibly to pregnant women.

Estimate graph of BSE disease in humans



The variation in human dose may give rise to a long slow rise to the condition before a much faster rise takes place due to smaller doses that very much large numbers of people have taken in.

The graph of human disease distribution above is derived from the year at which specific doses were present in human food but the graph would be further to the right or left depending on the ease of human infection that is present. No absolute figure can be given for the number of people that would be affected.

QUESTIONS IN THE HOUSE OF COMMONS

N272 Mr David Hinchliffe (Wakefield) 18 December 1995.

. . . what account was taken by the Advisory Committee on Dangerous Pathogens recommendation on the handling of liver in cattle . . . in the CMO's advice on eating bovine liver.

Answer.

. . . affected cattle do not enter the food chain. In addition, all the evidence supports the view that liver does not transmit the infective agent.

BUT:

1. Liver *has* been shown to transmit spongiform encephalopathy and for infectivity to be present.

2. It is the number of *infected* cattle not the number of *affected* cattle that decides the risk that is being taken. MAFF were aware that very large numbers (1.8 million) infected cattle were being eaten.

3. It seemed that large numbers of affected cattle *were* being eaten, as had been demonstrated by World in Action and by Kent's statistics published in November 1995.

BSE cattle born after the feed ban (July 1988)

	Year of Birth				
	1989	*1990*	*1991*	*1992*	*1993*
Numbers reported	9,364	2,425	461	6	1
Numbers expected by 10 years	30,206	10,543	9,220	>1,000	>1,000
Numbers eaten (approximately)	211,000	74,000	65,000	>7,000 (>12,000)	>7,000 (>28,000)

Numbers in brackets suggest numbers when under-reporting is taken into account. When > is present, the true figure could be very much larger. Data is from PQ 11869, David Hinchliffe, 31 October 1995.

PRION PROPAGATION IN MICE

"Mice carrying the human gene for prion protein did not produce the human form of the disease when inoculated with BSE" St Mary's group.

. . . MAFF: Therefore humans will not get BSE.

. . . Prusiner: Mice would not have been expected to develop the human form of the disease. They would require a chimeric prion gene for this to be expected.

DON'T STOP EATING BEEF

Because it is too late

Unless . . .

1. Young children
2. Visitors to the UK
3. Importers of UK beef products
4. ?pregnant women.

DON'T STOP EATING BEEF

Because it is too late

1. Research funding external to Government. SERC.
2. Separate MAFF and create a Food and Drugs Administration
3. Take pressure away from researchers to find what the politicians and the economists want them to.

BOVINE SPONGIFORM ENCEPHALOPATHY (BSE)

Remember: this a fatal disease with no method of treatment, inadequate methods of diagnosis, and no method of prevention in any animal after infection has taken place. Infectivity is presumed to be found in many tissues.

Information is being published tomorrow in an international scientifically reviewed journal showing:

1. Cases of BSE are becoming severely under-reported. For instance only 40 per cent of clinical cases reached UK Government statistics in 1993.
2. BSE may continue in the UK for many years, with cases born each year, showing symptoms three to eight years later.
3. BSE may not be derived directly from the disease in sheep (scrapie). The UK Government used this originally to suggest that BSE would not infect humans.
4. 1,800,000 cattle incubating BSE will have been eaten before 2001 even if no cases are born after 1991. Many of these will have been imported into Europe, particularly Belgium, France, Holland, Italy and to a smaller degree to Spain, Germany and Portugal.
5. The epidemiology of BSE in the UK is that of an infection that is passed down from the mother to the child but where the mother would show symptoms later in life. It may be that BSE cases we see are derived from infection from their mother.

6. UK Government advisors have suggested that there is little risk from eating liver, kidneys, nerves and muscle from infected cattle. The article shows that this cannot be true if these tissues contain the same amount of infectivity as is found in other species with a similar disease.
7. The acceptable levels (UK Government) of infectivity to be found in food may be 10,000 times the amount that could be seen as acceptable under World Health Organisation directives used for other potentially fatal diseases.
8. The UK Government Advisory Committee on Dangerous Pathogens reported in October 1994 showing that cattle that may be infected should be treated as if they are infective. They say that some tissues should not be even touched (eg liver) that the UK Government continues to tell its population that it is acceptable to eat.
9. The risk to humans in Europe from BSE is unacceptably high but cannot be stated precisely at this time.

ESTIMATES OF THE NUMBER OF PEOPLE THAT WOULD DIE OF BSE

(ASSUMING THAT THE RECENTLY REPORTED 10 CASES TO HAVE BEEN BSE IN HUMANS)

This can be estimated by looking at the potential times when the people became infected and extrapolating forward according to the number of infected cattle eaten in following years.

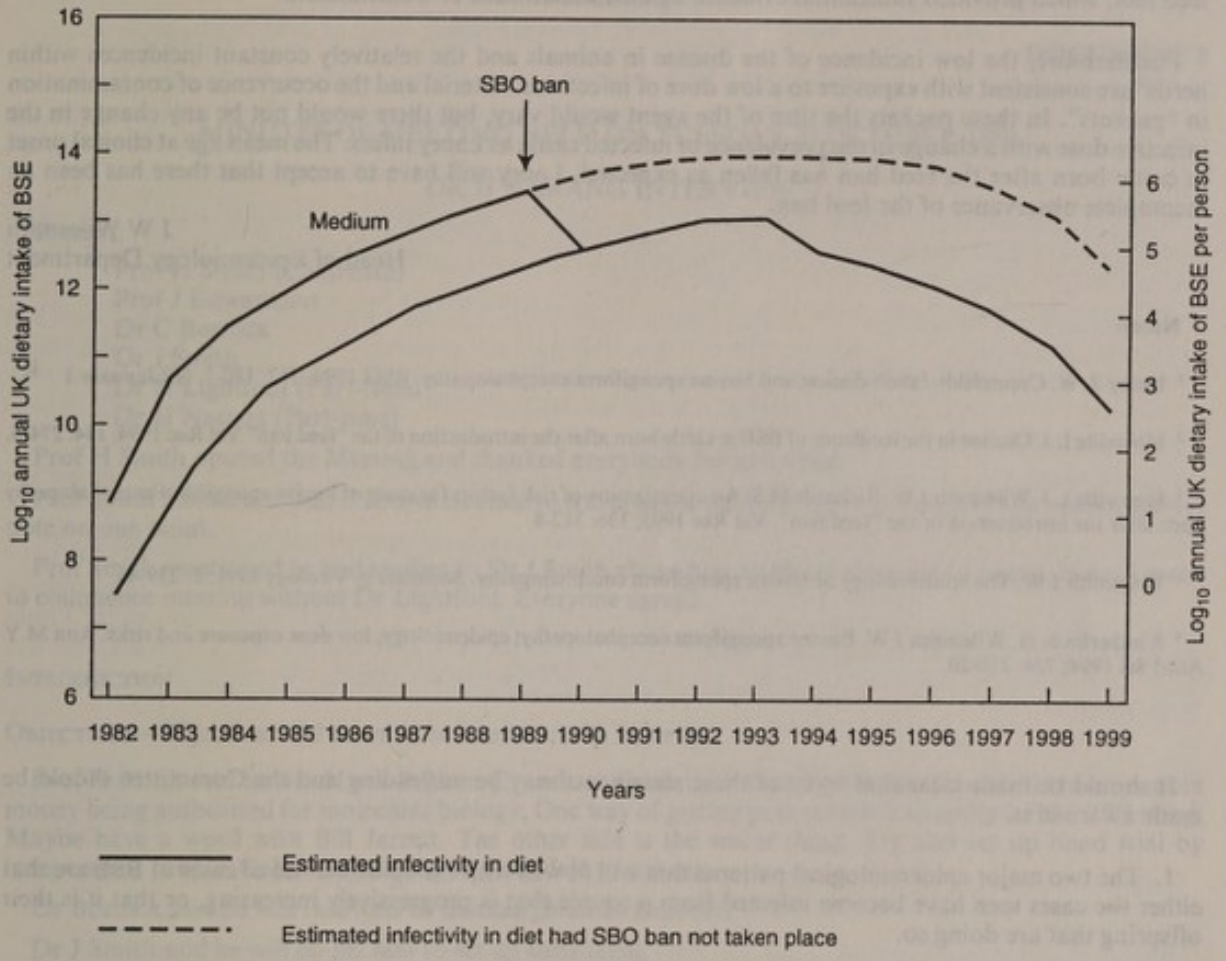
It is assumed in the model that in every year a certain number of humans become infected. They would show signs of CJ disease as a peak several years later. Obviously the CJD peaks resulting from humans infected in later years will appear themselves in later years, and add up to each other.

Year	*Number of cattle eaten by the human population in specific years at 50 per cent or greater of incubation period*	*Total number of people expected to die of CJD assuming that the 10 cases represent specific percentages of those infected in this, the first year of infection*			
		100 per cent	*10 per cent*	*1 per cent*	*0.1 per cent*
1982	274	2,141	21,440	214,100	2,141,000
1983	675	617	6,170	61,700	617,000
1984	1,415	248	2,480	24,800	248,000
1985	3,012	109	1,090	10,900	109,000
1986	5,510	54	540	5,400	54,000
1987	8,908	30	300	3,000	30,000
1988	14,279	17	170	1,700	17,000
1989	24,606	10	100	1,000	10,000

(these figures assume that nobody ever becomes infected with BSE after the Specified Offals Ban was introduced in November 1989, figures are approximately 10 times higher if infection continued after the offals ban).

Further calculation must be used by considering that all the cases tested so far have one specific gene, which is only present in 38 per cent of the population (met-met homozygous).

Infectivity expected to be in the human diet during the BSE epidemic



THE COHORT STUDY INVESTIGATING VERTICAL TRANSMISSION

(looking into the number of cattle that die of BSE when they are the offspring of infected mothers vs the offspring of others mothers from the same herd)

COHORT STUDY OF COWS IS IN PROGRESS

Editor,—R W Lacey's letter makes no reference to any scientific paper on the epidemiology of bovine spongiform encephalopathy.[1] As a result it contains too many omissions, errors, and misconceptions to pass into the literature unchallenged.

Lacey's description of the cohort study to examine the risk of maternal transmission is incorrect. This study is comparing the incidence of bovine spongiform encephalopathy in offspring of dams that developed clinical signs of the disease and in offspring of dams that reached at least six years of age without developing clinical signs. Three hundred pairs of animals are involved, and the members of each pair were born in the same calving season and herd. The criteria for purchase, between July 1989 and February 1990, were that the animals had been weaned, females were virgin, males had been castrated, and documentation for the animals' provenance was available. Age at purchase ranged from two to 24 months; most of the animals were yearlings. The study population therefore comprises some animals born before the ban on ruminant feed containing recycled animal remains in July 1988. The animals are being kept until seven years of age; the youngest will reach this age in November this year. The sample size of the study precludes interim analyses because of the inevitable loss of statistical power. The results of the study will be reported in due course; in the meantime not even Lacey can draw any conclusions.

Susceptibility to bovine spongiform encephalopathy is independent of age, but the risk of infection has undoubtedly declined as a result of the feed ban.[2] Using a crude, inappropriate comparison of the age at which bovine spongiform encephalopathy has occurred in cattle born before and after the feed ban, Lacey claims

that vertical and maternal transmission has occurred. He ignores a large case-control study that examined the risks of maternal and horizontal transmission in cattle born more than three and a half months after the feed ban, which provided substantial evidence against such means of transmission.[3]

Furthermore, the low incidence of the disease in animals and the relatively constant incidences within herds[4] are consistent with exposure to a low dose of infectious material and the occurrence of contamination in "packets". In these packets the titre of the agent would vary, but there would not be any change in the infective dose with a change in the prevalance of infected cattle as Lacey infers. The mean age at clinical onset in cattle born after the feed ban has fallen as expected. Lacey will have to accept that there has been an incomplete observance of the feed ban.

J W Wilesmith
Head of Epidemiology Department

Notes:

[1] Lacey R W, Creutzfeldt-Jakob disease and bovine spongiform encephalopathy. BMJ 1996; 312: 180-1. (20 January.)

[2] Hoinville L J. Decline in the incidence of BSE in cattle born after the introduction of the "feed ban" Vet Rec 1994; 134: 274-5.

[3] Hoinville L J, Wilesmith J W, Richards M S. An investigation of risk factors for cases of bovine spongiform encephalopathy born after the introduction of the "feed ban". Vet Rec 1995; 136: 312-8.

[4] Wilesmith J W. The epidemiology of bovine spongiform encephalopathy. Seminars in Virology 1991; 2: 239-45.

[5] Kimberlin R H, Wilesmith J W. Bovine spongiform encephalopathy; epidemiology, low dose exposure and risks. Ann M Y Acad Sci 1994; 724: 210-20.

It should be made clear that some of these statements may be misleading and the Committee should be made aware of it.

1. The two major epidemiological patterns that will fit well with the epidemic rise of cases of BSE are that either the cases seen have become infected from a source that is progressively increasing, or that it is their offspring that are doing so.

The cohort study cannot tell whether those that are dying of disease are from either group and the MAFF should not suggest that it can. When the study was started this could not have been predicted and no blame should be put on the scientists involved. Unfortunately now the study may be worthless.

2. The suggestion that infection would be taking place in "packets" fits the epidemiology. This is, I feel, incorrect. If this was true, the rate in individual herds would rise in parallel to the national rate. Also, as anyone knows who has seen the meal manufactured, the powder that is produced would not contain "packets" but would be spread amongst many members of a herd that was being fed.

3. Mean age showing disease in cattle born after the feed ban has indeed fallen. This was very strange indeed if only because the incubation period is inversely proportional to the dose given. By rights, if "packets" were involved there should be no change, and if no "packets" are involved the cattle should have been getting older.

4. "The number of cases of cattle dying of BSE since the feed ban is dropping as a result of the feed ban". There is insignificant data to show this. My own data with Professor Kent showed that this could not be stated and that large numbers of cattle with clinical symptoms of BSE may be entering the food chain. I have had no response from MAFF since that was published. What seems now is that much of the epidemiology data from MAFF since 1992 in cattle born after the feed ban is actually quite difficult to interpret as only until 1993 can the corrected data be calculated.

My own statistics agree that the case numbers are dropping (estimate data) but at a much lower rate than suggested by MAFF. The reason for this can be discussed.

Extract from memorandum by Dr Harash Narang [T8/BSE5]

Document 1[1]

DOCUMENT 2

MINUTES OF MEETING HELD ON TUESDAY 23 OCTOBER 1990

DR H NARANG INTERVIEW

Present:

Prof H Smith (Chairman)
Prof J Edwardson
Dr C Bostock
Dr J Smith
Dr N Lightfoot (Part-time)
Dr H Narang (Part-time)

Prof H Smith opened the Meeting and thanked everybody for attending.

Prof Smith mentioned that F Brown has had to travel to the States at short notice, and has written a short note on one point.

Prof Smith mentioned he had spoken to Dr J Smith about how to set up the meeting today. It was agreed to commence meeting without Dr Lightfoot. Everyone agreed.

INTRODUCTION

OBJECTIVES—Objectives laid down in two sentences in letter to.

Prof H Smith said he would ask Prusner (?) and . . . Confirmed that Dr J Smith has said there is no more money being authorised for molecular biology. One way of getting peer-review is to apply for the BSE thing. Maybe have a word with Bill Jarrett. The other side is the smear thing. Try and set up blind trial by independent people. Colindale is told and Colindale tries it out.

Dr Bostock said he will raise this in Edinburgh on 24 October.

Dr J Smith said he will do his best to set up something.

Dr Lightfoot said that Dr Narang is working away and it is very difficult to stop him. Do we need to stop him? Money is coming from support grants at present. David Clark fixed his present money.

Prof H Smith asked Dr J Smith whether Dr Narang applied for this money without permission.

Dr J Smith replied that he had to decide whether to let him accept the money—political.

Prof Edwardson said that others are getting this type of help all the time. Narang has responsibilities to the service and he must be told clearly. There is a lack of critical judgement and this Committee is concerned about this. This must be looked at closely.

Dr Bostock said one cannot stop him doing his work.

Prof Edwardson commented that no-one else will support him.

Prof H Smith said that Dr Narang could not be sacked.

Dr J Smith said he has created a terrible embarrassing situation with the press.

Prof H Smith said that if Dr Narang spends 20 per cent of his paid time on slow virus work, that is all right. The financing of molecules is all right. A critical look at his technique should be made in a blind trial by independent persons, set up in a way previously mentioned.

Dr Bostock stated that there are probably at least 50 papers which claim to find nitric acid. People have cloned sequences from scrapie-infected material. Nothing to do with agent.

Prof Edwardson said some managerial talks about talking to the press. CJD in northern region—scientific scare-mongering.

Dr Lightfoot said that Dr Narang's attitude had improved over the past year. He is listening more to us.

Prof Edwardson said he acknowledged that Dr Narang is an excellent microscopist, but it must be indicated clearly to him exactly what is needed.

[1] Not Printed on the grounds of illegibility.

DOCUMENT 3

Letter to Dr E Mary Cooke from Dr Harash Narang, dated 24 May 1994

Thank you for your letter dated 18 May 1994.

I wrote to you on 11 April 1994, that there are problems in organisation in keeping the inoculated animals during the critical development of the disease. These problems are being resolved very slowly with the Home Office. If agreed, the first group of animals which would be ready will be on 27 June 1994.

However, I have now resolved the problem with HSE. My work involves Group I, GMMs in Type A operations and therefore does not require prior notification to HSE. The genetic manipulation experiments which I will be doing, fall into a very low risk category.

It is important to obtain ssDNA from different strains of scrapie for further analysis. This is a high risk work, purification of DNA could be done in USA. This material at present is not available at the London Hospital and therefore collaborative work with Professor Dmitry Goldgaber would be essential. Professor John Oxford is away, when he comes back, I will go through in detail the work which I would undertake at Stony Brook.

The experimental costs involved would come out of a Grant from USA. I would not get a salary subsistence of travelling costs. To meet all costs I will have to apply at least two years in advance.

Starting date would depend on mutually agreed date and on the availability of scrapie-infected mice.

The Registration fee (Lit. 600.000 plus VAT 19 per cent) for 9th Mediterranean Congress of Chemotherapy in Milan. I have not obtained funding from any other source and have not applied to any other source. Costs: flight is £230 plus £5 for tax and have to pay £75 per night. As yet I have not got the full programme and would not know the time and date of my presentation. I would be grateful for the reimbursement of costs from PHLS.

DOCUMENT 4

Letter to the Rt Hon William Waldegrave MP, the then Minister for Agriculture, Fisheries and Food from Dr Harash Narang, dated 2 September 1994

Thank you for the reply of my letter dated 1 August 1994 by Mrs S C Townsend.
In your letter you write that PHLS has been in regular correspondence with me regarding the disciplinary proceedings. You can see from my letter to Rt Hon Jim Cousins MP, that PHLS have not been in regular correspondence with me regarding the disciplinary proceedings.

Sorry to bring this point again, I submitted two papers to Dr E Mary Cooke for an internal review on 4 July 1994, to this day have not received a reply. I do not know how to explain this, but to say, in my case there appears a great deal of deep seated discrimination and prejudice towards me and my scientific work which I cannot finish.

Ten cow brains were examined by this method and the paper was submitted on 20 March 1991 to Dr N F Lightfoot, Director PHL, Newcastle. The paper however, was held for internal review for months. Further, the results supplied by MAFF show different dates for two of the specimens, to those on the Forms supplied the specimens were collected. These differences would mean that the two laboratories have examined different specimens, therefore any fair minded Editor will tell you to start the study again.

Regarding the work in the London Hospital: I was moved to The London Hospital in August 1993. For one reason or another, permission to start work was not granted by PHLS till end of March 1994. I started ordering chemicals and equipment, in April 1994 hoping to do some work. My equipment arrived on 19 July 1994.

For the first time sick animals were made available to me on 18 July 1994. I was half way through the experiment when I was told by Professor Williams on 21 July 1994, that I have to stop my work, not to do any more, Sorry to bring this point again, I submitted two papers to Dr E Mary Cooke for an internal review on 4 July 1994, to this day have not received a reply. I do not know how to explain this, but to say, in my case there appears a great deal of deep seated discrimination and prejudice towards me and my scientific work, which I can not finish.

At the Progress-meetings with MAFF and the BBSRC jointly funding bodies, the work was outlined. However, from the start, the project as outlined at the first meeting not followed. In September 1993, when I met Dr D A J Tyrrell in Brussels, I told him and Mr Ray Bradley about my concerns and these problems. There appears that there has been a lack of co-operation from the start. Work was being done without my supervision.

For my experiments, I had been waiting for the scrapie-infected brains for months, later I was told, that materials were being used for RNA preparations rather than DNA. Research was not funded for RNA work. It appears that, work on the project has never been done, as proposed in the original Grant Application.

There are two tests:

(1) A diagnostic test which would reveal within an hour if the tissue was infected. Although, at the first Progress-meeting, it was discussed that this part would be done, but no attempt was made to do this test.

Second part of the experiment which would reveal the nature of the spongiform encephalopathy agent. Purification and characterisation of a specific ssDNA would lead to development of a polymerase chain reaction. The polymerase chain reaction technique will be used to amplify the ssDNA from a peripheral tissue that could be samples from living animals.

The proposed work, will demonstrate, nature of the agent, strain variation, molecular basis of pathogenesis.

1. ssDNA will be purified from scrapie-infected brains and control brains from uninfected animals (3–18 months). I already have small amounts of purified ssDNA.
2. Hamsters will be inoculated with (a) ssDNA. (b) Transfected cells with ssDNA. Infected animals will develop the disease (3–36 months). A positive result in each case would indicate that ssDNA in question is the scrapie agent.
3. Complete sequence of the scrapie associated ssDNA will be determined. Sequences obtained from hamsters and mice infected with different strains of scrapie and compared with CJD and BSE (6–36 months). This would form the basis of a live test.
4. A unique sequence will be used to develop a polymerase chain reaction using tissues of affected animals, including blood, cerebrospinal spinal fluid and urine specimens at different stages of incubation period (4–36 months). Once the test is developed it would take six to seven hours for the diagnoses). It will be used as a live test.

As I was getting ready and ordering the materials for some of the work, I was told to stop further experiments, but pack so that materials could be transported back to Newcastle laboratory. (See copies of letters to Professor Almond, enclosed).

DOCUMENT 5

Notes of a telephone conversation with Mr Ray Bradley, CVL, 2nd August 1994, 9.30 am

We discussed the publication of the work done by Professor Oxford and Lynne Bountiff following Harash Narang's protocol.

The formal situation is that Professor Oxford should write a report of the work and the paper for publication and this should go to the Chief Scientist (MAFF) and AFRC. They would then expect to approve these and the work would be published in the normal way.

I pointed out to Ray that we might get into the situation where there was enormous media interest in the results of this work and, of course, there may be PQs and it would be quite difficult to maintain the formal position when we knew the results. He quite understood that point, which will affect them as much as us, and he will get as much urgency into the situation as he can. However, it does not alter the fact that nothing can be done until Professor Oxford has produced a report and we have no guarantee as to the speed at which this will happen.

From a practical point of view I think it means that we cannot rely on being able to make public the results of the work within the next few months. We, of course, could not publicise these results independently. Indeed, we have never though that we would or could and I have told Ray Bradley that we would not do this. It is going to make the whole process very difficult, and it means that if we are planning to take any steps in relation to Harash Narang, then we must keep MAFF and DoH fully informed as there will be pressure in relation to this work.

Notes of a second telephone conversation with Mr Ray Bradley, CVL, 2nd August 1994, 10.20 am

I informed Ray Bradley that the things I had been anxious about in our previous conversation had, in fact, just happened. That we had had a letter from an MP asking for the results of the work.

Meanwhile, Ray had gone back and talked to other people in MAFF and the situation remains as he initially described it, that the report has to go to MAFF, AFRC (now BBSRC) and be submitted in the normal way to a journal. We could ask the editors of the journal to leap the queue if appropriate and, once

it had been submitted, with the journal's agreement it would be possible to release a statement giving in outline the results that had been obtained. At the present time they do not see any possibility of a variation on this.

I have told Ray that we shall refer all requests to MAFF about the publication of the results and I have also told him that he may be receiving a formal letter from me on this topic.

To: Mr K Saunders
Dr R Gross
Mr J Phipps
Mr M Guest
Dr D Walford
Ms C Murphy
Mr C Osman

DOCUMENT 6

Letter to Dr Harash Narang from Mr John H Phipps, Head of Human Resources, Public Health Laboratory Services, dated 26 August 1994

STRICTLY PRIVATE AND CONFIDENTIAL

Thank you very much for meeting with Dr Freeman and I on the 19 August at Colindale. We both found the conversation very useful and greatly appreciated your constructive contribution.

As we agreed, in accordance with your request, I shall summarise the four key areas of our discussions.

1. Your absence from the Public Health Laboratory in Newcastle, whilst pursuing research at the London Hospital for a year has seen:

(a) A reduction in workload at Newcastle, together with diminishing income.

(b) Reductions in central funding.

(c) Substantial equipment and staff rationalisation.

(d) The redistribution of your specific scientific work in diagnostic services to other staff at the laboratory.

(e) The appointment of a Consultant Microbiologist to co-ordinate the planning of research and development.

This has meant that there is no longer a requirement for a Clinical Scientist at your grade to undertake your former responsibilities at Newcastle PHL as they have now been redistributed.

2. However we did consult with you on our intentions to explore reasonable alternative employment consistent with your skills and experience. We shall therefore determine if there are any substantive vacancies within the Service to meet this specification and liaise with you accordingly. Your preference to stay in Newcastle was noted, although as we pointed out, the likelihood of a suitable permanent vacancy arising was most improbable.

3. We also invited you to make any contribution to the consultation process by identifying or considering any preferences you would wish to discuss, within the remit of the PHLS.

Naturally, we accepted your point that you had no preferences, and it was our managerial prerogative to put forward potential alternatives. However we would value your involvement in the consultation process and are very happy to consider realistic alternative proposals on how we can continue to use your capabilities in a permanent role.

4. Finally, it was pointed out to you that you would have to be declared redundant if we were unable to redeploy you into a mutually acceptable permanent position, elsewhere within the PHLS.

In such an event you would enjoy the benefits of premature retirement on the enhanced terms available in such circumstances. I can provide you with an estimate of these benefits when we next meet.

You personally raised two matters. Obviously you were concerned about specimens relating to your research at The London Hospital. It was agreed that Dr Mary Cooke of the PHLS would contact Professor Williams at The London Hospital to arrange safe storage of any materials relating to your recent research pro tem at The London Hospital. It was not felt acceptable that any materials or samples from this work could be transferred to Newcastle PHL.

We also noted your second query on how these immediate discussions would affect or would relate to the appeal you have currently lodged against disciplinary action initiated by the Service.

As I pointed out to you, and would now confirm, the issues are completely separate and unconnected. Arrangements for the disciplinary appeal are proceeding I understand, whilst our next meeting, scheduled for the 9 September 1994 will be continued consultation on minimising the possibility of redundancy caused by organisational change. I would regard the consultation on your employment with the PHLS as being our first priority.

You indicated your willingness to meet again on the 9 September in Colindale at 2 pm and unless you hear to the contrary in the interim, this letter is confirmation of that meeting. On this occasion I shall be accompanying Dr Lightfoot, who will have returned to work, and as in my last letter, renew the invitation for you to be accompanied by a colleague or representative if you so wish.

I hope that we shall be able to jointly find a solution to our current dilemma. In the light of the situation explained at our meeting and confirmed above it will not be necessary for you to report for work at the Newcastle Laboratory before we meet again on 9 September, although you will naturally continue to be remunerated as usual.

As promised I am sending copies of this letter to both your London and Newcastle addresses.

DOCUMENT 7[1]

Examination of Witnesses

SIR KENNETH CALMAN KCB (Grade 1A), Chief Medical Officer, Department of Health, PROFESSOR JOHN PATTISON, Chairman, Spongiform Encephalopathy Advisory Committee, and MR K MELDRUM CB (Grade 3), Chief Veterinary Officer, Ministry of Agriculture, Fisheries and Food, were further examined; PROFESSOR JOHN BOURNE, Director, Institute for Animal Health, Compton, DR STEPHEN DEALLER, Consultant Microbiologist, Burnely General Hospital and DR HARASH NARANG, were examined.

Chairman

187. Good morning, I would like to welcome you to this third session of evidence on BSE and CJD held jointly by the Agriculture and Health Committees. This morning we are taking evidence from two scientists who have been critical of some aspects of the Government's policy on this issue, Dr Stephen Dealler, of Burnley General Hospital, and Dr Harash Narang, formerly of the Public Health Laboratory Service. We also have present as witnesses Sir Kenneth Calman, the Chief Medical Officer, who is accompanied by Professor John Pattison, the Chairman of SEAC, and Mr Keith Meldrum, the Chief Veterinary Officer, who is accompanied by Professor John Bourne, the Director of the Institute for Animal Health. Dr Dealler and Dr Narang, our questions will be directed chiefly to you this morning, as Sir Kenneth and Mr Meldrum have had an earlier opportunity to give evidence to us; but I will give them now a general invitation to intervene if they wish to give their comments on anything which arises in the questions. I would also like to mention that there will be a lobby of disabled people taking place in this room this afternoon, so we shall need to finish at one o'clock sharp. I hope that questions and answers will be brief so as many Members as possible can take part. I shall start the questioning, which will be directed to Dr Narang. Dr Narang, can you give us details of your work on developing a live test for BSE? Can you clarify what response you have made to recent requests from the Department of Health to inform them about your work?

(*Dr Narang*) Thank you. I have been working for some 26 years with the Public Health Laboratory, and of my own accord, when I was made redundant, I was contacted by relatives who believed in my work and they trusted me, and they gave me the authority to examine the urine specimens of their relatives. I developed this test by concentrating the urine and then examining the specimen in an electron microscope. As people will be aware there is a unique structure which we see. If the structure is present called a nemavirus, and SAF in the specimen then the specimen is positive. As you will recall in the last case I examined urine specimen from Peter Hall, he had particularly diagnosed not to have CJD by his neurologist and yet my test came out to be positive. Even when the neurologist knew it was positive he said, "No, it is not CJD". When the patient died and the postmortem result came out it was CJD. This happened to be an atypical case. What I have to say briefly is, it does not matter what strain of the agent is lurking around, this test is positive.

188. Thank you. Can you tell us what response you have made to recent requests from the Department of Health to inform them about this work you are doing?

(*Dr Narang*) I have written to Mr Stephen Dorrell; I have written to Mr Douglas Hogg informing him about my test and offering my services. In short, when I get a reply, it was the usual civil servant reply, giving you the stinging impression that you do not have to reply because they are clearly not thinking of considering your proposals. At one stage I was told by a journalist that the SEAC Committee would contact me, and SEAC wrote me a letter which arrived nearly a month later on my desk. A phone call came and as required by SEAC, I submitted all my published work. I said I would be prepared to discuss anything further, but I have not heard anything since then.

Chairman: Thank you very much. I will try and get some response from the Government side in a moment.

[1] Not Printed: See Agriculture Committee, Fifth Report, Session 1989–90 BSE, pp. 237–240 (HC 449)

Mr Campbell-Savours

189. Could I ask for a response from Sir Kenneth to that particular point Dr Narang's has made. He said he wrote to the Secretary of State; he included papers which provided, he believed, to be his evidence; what happened to that letter? In the light of that letter, why did we receive a response from the Secretary of State direct last week in this Committee?

(*Sir Kenneth Calman*) I am obviously aware of the correspondence between Dr Narang and the Secretary of State for Health, and the Secretary of State's response for further information about the test itself.

190. I am sorry, we were told last week that he was still awaiting information. The tone of the statement last week was that there had been no response at all from Dr Narang.

(*Sir Kenneth Calman*) I think what Dr Narang has just said is in fact news to me. If papers had been sent then we have been anxious through SEAC, and SEAC has been one of the vehicles through which we have asked for information about the test— If I could make it very clear, and I did make it very clear the last time I came to this Committee, there is an urgent need for a test in live animals for BSE.

191. I am sorry, we understand that, that has been repeated to us on many occasions. You have just said to us, "... what Dr Narang has just said is ... news to me". Can I put it to you, it should not be news to you. You should know precisely what is happening in relation to Dr Narang's work. One of the reasons why we are involved in this scare today is, it has been alleged there has been a breakdown in the scientific community in work being done in the Department. As Chief Medical Officer you should know precisely what he is doing.

(*Sir Kenneth Calman*) I am trying to sort out the questions. I am entirely aware of the correspondence between Dr Narang and the Secretary of State. We have been waiting—and I say that—for information from Dr Narang which he has given us just now; in fact he responded with papers very recently. Is that correct?

(*Dr Narang*) No, that was in January.

(*Sir Kenneth Calman*) We responded to that. We have asked for further information about the test.

192. Are you saying that in giving that reply to that question, when Mr Dorrell gave his reply to that question last week he also knew that this material had been supplied in January by Dr Narang?

(*Sir Kenneth Calman*) That material was not sufficient for us to make any decisions, which is why we have asked for further information through SEAC. We have also asked for further information about the test itself.

Chairman

193. Has that further information now been received by you? If so, when was further information received?

(*Sir Kenneth Calman*) I will have to respond to that by letter, because we have had a whole series of letters from Dr Narang, and it will be very important—If this is the sum of the questioning today, should you wish a precise note of every letter which has come and gone between the Secretary of State for Agriculture and the Secretary of State for Health, we will be very pleased to provide that to you.

(*Mr Meldrum*) Could I add a comment from the animal health side because, like Sir Kenneth, I am also very keen indeed to pursue any avenue which could deliver a diagnostic test we could use for living animals. I have been constrained in responding to any sensible questions on this subject because I simply did not know what test Dr Narang was using in urine. The only information I have is contained in three and a half lines in a letter he sent to Mr Hogg on 9 December last, in which he talks about the test looking for nemavirus. Understandably, Dr Narang has not provided details of that test because he wishes to keep the information to himself—I assume because of copyright or patent issues. The only other point I would make is that we have only just received a letter from Mr Bell, who is Dr Narang's sponsor, asking us whether we would co-operate with some testing of urine obtained from a variety of cattle to validate the test for BSE in cattle. I only saw that letter yesterday morning, and we will respond positively to it. We are certainly very happy to set up a validation system with Dr Narang under controlled conditions, under independent audit, to determine whether or not his urine test will deliver what we would like it to deliver for live cattle. If that test is based on the nemavirus theory then I have severe doubts as to whether it will be able to work, for reasons that Professor Bourne can explain if you so wish later on.

Mr Campbell-Savours

194. Dr Narang, do you accept you are trying to protect for commercial reasons work you are doing?

(*Dr Narang*) I am a scientist as I always clearly say and I work according to the rule of science.

195. Are you trying to protect it for commercial reasons? Can you give me a clear answer?

(*Dr Narang*) No, I am not trying to protect it. Dr Robert Will came to see me and we discussed this test and I offered to give him specimens. All I asked was that we sign an agreement between the two parties saying, "This is your intellectual property", then I can put every card on the table.

196. You can put what?

(*Dr Narang*) I can give every detail of the test. Once we have made an agreement that this is my intellectual property then I can give all the details.

197. So in no way have you at any stage sought to prevent any of the work, or detailed information about the work you are doing, from coming into the public domain so far as you are prepared to give it to Sir Kenneth?

(*Dr Narang*) No, I am prepared to give it to anyone. As I have said many times, I am not after money. Money was not my objective. If I was working for Public Health then Public Health would have owned this test or royalty, whatever it was. To me it is realising I want to have a control of how this test should be organised to begin with so there is no problem later on.

[**Mr Campbell-Savours** *Cont*]

198. Mr Meldrum, why is there a gap, let us say, of three or four months between Dr Narang making his theories known to you generally and you now offering before this Committee to carry out further work on urine tests. Why is there a delay?

(*Mr Meldrum*) Quite simply, any test which is put on the table must be validated. There has been an extensive amount of information exchanged between my Department and Dr Narang over a period of time. Quite frankly, you cannot expect, nobody can expect, my Department to get into a situation of validating a test where you have no information at all on what test is being put on the table for validation. Until I saw that letter yesterday morning I had no proposals from Dr Narang as to how we could co-operate with him on validating a test on urine from cattle. In fact, in that particular letter I saw yesterday it made reference to obtaining samples from a nearby experimental farm. By that I know now he means the High Mowthorpe Farm. You cannot just obtain samples of urine from cattle without knowing whether or not they are infected, and that you can only determine after slaughter. That experiment will run on to 2001. That particular suggestion falls. We are certainly happy to co-operate if we can have information with which we can work. In the absence of information, I am afraid I am fettered in what I can do. I stand by my comments made to this Committee, Madam, last time that we are looking for a live diagnostic test and will work with anybody where we believe there is a reasonable chance of producing a test that will work in the field. I want that test like everybody else wants it.

199. Can I ask you, Dr Narang, something which I think might be a little more difficult for you to handle, because I do believe there might be some criticism of your position. When Professor Oxford applied for the grant for you to do work in the London Clinic that grant was paid to you on the basis you would take a hands-off approach, if I understand it; that Dr Lynne Bountiff would carry out research, and you were prepared to accept their findings. Professor Oxford, is a fine defender of Dr Bountiff as you know—she is a very eminent post-doctorate research scientist, I do not know the exact terminology—yet this was work verifying what your findings were. When they set out to verify your work they could not verify it. They found that, in their view, there was nothing scientifically which they were able to establish to underwrite the work you were carrying out. To some extent that might be responsible for some of the loss of confidence within the Department as to what you are doing?

(*Dr Narang*) Can I point out that this work was nothing to do with diagnostic work whatsoever. I was asked to go to London Hospital on paper on 1 July 1993, but I sent some correspondence to you—and probably all Members have it in front of them—which I refer to in Document 3[1]. If you look at this document, dated 24 May 1994, it clearly shows you in the first paragraph, last line, "If agreed, the first group of animals which would be ready will be on 27 June 1994". Although the work was started in July 1993, this is the predicated date when the animals would be available. If you look at the next document[1], which I wrote to the then Agriculture Minister, Mr Waldegrave, in the last paragraph, starting on the page marked "49", I say, "For the first time sick animals were made available to me on 18 July 1994. I was halfway through the experiment when I was told by Prof. Williams on 21 July 1994, that I have to stop my work". If you calculate that date from there to there, this is the time it took to finish the experiment in practical terms, in 3 days. If people want to believe that the work was not done in the way it should have been done, I do not really believe you can do one whole year's experiment in those three or four days when the experiment was not even finished. My summation is, the work was not done. If you look at my published work, it matches nowhere with what was done.

200. Dr Oxford, whom you greatly respect—you and I have spent a lot of time together over the years and you greatly respect him—he maintains that the work was properly done. He would argue perhaps there might be the scope for further work. He says that it was properly done and he has utter confidence in Dr Lynne Bountiff's ability to verify your work.

(*Dr Narang*) If that was true—and, as you see, the work was finished in that time—then work is still not being published. It is nearly two years on; if the work was not done it should have been published. Research takes a longer time to publish the result. If I refer you to Document number 5[2] in your bundle it says, "Notes of a telephone conversation with Ray Bradley", and this is with Mary Cooke. If you read it carefully then here they are trying to get these results published as soon as possible. In the last but one paragraph they even want to jump the queue and publish these results. That was the urgency then, but the results are still not published.

201. Sir Kenneth, why have you not expedited this process of publishing this work, which did not endorse work done by Harash Narang?

(*Sir Kenneth Calman*) If I could make one brief comment, and I think others might respond to that more effectively. When I said there was new information, the new information Dr Narang has just given us is that this is a concentrated urine test looking for nemaviruses via an electron microscopic technique. That is the first time he has said this, about this live test. The only other piece of information we have had is a cutting from *The Times*. That is quite new information. I assumed that is what it was, but we had not been able to get that information. That in itself was the new bit I mentioned. I think Professor Bourne might appropriately respond to your question.

(*Professor Bourne*) The work Dr Narang described arose out of an observation he made and subsequently published on a demonstration of what he described as a nemavirus, which was based on electron microscopic morphological studies on brain tissues. As a result of that work he further described a single stranded DNA as part of the nemavirus preparation he initially observed and hypothetically indicated was a virus. The study described by Dr Bountiff was carried out in Professor Oxford's lab. There is no question about the credibility of that

[1] See p. 66.

[1] See p. 66.

[2] See p. 67.

[**Mr Campbell-Savours** *Cont*]
laboratory, the credibility of Professor Oxford, or, indeed, the credibility of Dr Bountiff, who I might say was a very strong supporter of the nucleic acid theory with respect to the casual agent of the TSEs. This work was carried out with the involvement of Dr Narang, who was involved at every stage of that work. I have a detailed report here indicating the close involvement of Dr Narang in that work, who, throughout the work, made observations and some criticisms which were always attended to. Nonetheless, the finding of a single stranded DNA could not be confirmed by Dr Bountiff. The paper describing this work has now been submitted to the Journal of General Virology and has been accepted by that journal and we are now awaiting publication of that paper.

202. Will it be peer reviewed?

(*Professor Bourne*) Absolutely. It may be appropriate if I read just one paragraph from this paper, which refers to the modifications which were put in place as a result of Dr Narang's concerns, and also indicate the general findings. It reads: "Despite all the modifications to the preparation of brains, ie, using late-stage disease animals, using increased numbers of brains, inhibition of nucleuses, examining fractions from sequential steps throughout the preparations, and preventing adherence of nucleic acids to vessel walls, there were no bands seen in TAE gels of a scrapie preparation that were not present, albeit in lesser amount, in the equivalent normal preparation." The finding of the paper is that they could not find the single strand DNA and they could not confirm the original hypothesis and finding of Dr Narang.

203. That is what I have already said, but I have said it in one sentence.

(*Professor Bourne*) Can I also add that this work was supervised by Professor Jeff Almond, another noted molecular biologist. I think one cannot discredit this work.

204. Can I put to you what I think, essentially, the problem is here? We have a lack of confidence in the beef market. Farmers are in trouble, Parliament is in uproar and the public is concerned. The public does not know, really, what to do. What is happening is that the media is latching on to particular individuals who, because their questions are not being answered and because they are not being taken seriously, provide pegs on which this whole issue can be exploited. Dr Narang is one of those pegs. I am not criticising Dr Narang here; he is entitled to go out and advocate his case. All I am saying is that people like Dr Narang should be working for the taxpayer and not for Ken Bell International, a firm of fish processors up in the north east of England. Whilst they work out of the system they are game for exploitation and undermining the sensible debate that we believe—and certainly farmers believe—should be taking place on this issue. To some extent I hold the Department of Health officials responsible for this because they have been isolating the likes of Dr Narang, saying, "You are on your own". I have got reams of paper showing, over the years, there was a lack of preparedness within the Department to take their work seriously, for all sorts of reasons. It may well have been that some of them were legitimate—like the fact you could not verify this recent work. Is it not now time that these people should be brought under the umbrella of the taxpayer whereby their work is being monitored all the time, whereby it cannot be exploited by the media and by others, and whereby we can see that any advances that they make are treated seriously, and any work which they do which is not serious should be invalidated immediately, before the press and the media can run away with it? That is my argument. I think Dr Narang has been wrapped up in this whole affair. If I could ask Sir Kenneth and Mr Meldrum, please, to respond to that statement?

(*Sir Kenneth Calman*) First of all, Dr Narang's work has been taken seriously: the Touch method has been looked at and the methods which Professor Oxford has been involved in has been taken seriously. I agree that taxpayers are very, very important in this, and normal methods of reviewing and paying for research work would be through a process of peer review. Then you and others would be satisfied about the quality of that research work to the research councils, or whatever. We require, for that, information about the test and the purpose of the experiments to be done. That was part of our discussion. I have in front of me a large number of letters from Mr Hogg and other Secretaries of State (going back to Mr Waldegrave) to Dr Narang, which we can make available to you, if you have not already seen those letters. I think we have taken it very seriously indeed, and if Dr Narang has a test in urine looking for nemaviruses which has not been substantiated elsewhere then that work for the taxpayers' benefit can go through the process of peer review to the Agriculture Research Council, or the BBRC or the MRC or through the Department of Health's process or through MAFF's process. Is that not an appropriate response?

(*Mr Meldrum*) Chairman, I did comment upon this last time. We have co-operated with Dr Narang, we have co-operated with the Touch Test referred to just now by Sir Kenneth and I have a copy of the draft paper in front of me which was submitted in March 1991. I have been through the files yesterday in great detail to make sure we understood. We have also co-operated with the work done by Professor Oxford, insofar as the work that Professor Bourne has described was funded by my department. We wanted to determine whether or not the nemavirus detection system would work in independent laboratories carried out by an independent team, and Professor Bourne has indicated it does not. Thirdly, on the urine test, yes, we are very happy to work with Dr Narang on this urine test; we are happy to set up a validation system, but I must emphasise, so far as I am concerned, it is absolutely of paramount importance that any test that is used in the field so far as BSE is concerned has been validated. If we use tests that are invalid there is a major problem over public perception and understanding of what we are doing. We have done this step by step. We have attempted to determine whether the nemavirus theory stood up, and it does not.

205. But why does a fishmonger from the north east of England have to fund this work? Why?

(*Mr Meldrum*) We have already funded the nemavirus work. With great respect, the work that Professor Bourne has just described was in fact

[**Mr Campbell-Savours** *Cont*]
funded by my department. Unfortunately, from the point of view of a diagnostic test, it did not turn up trumps. If we have a urine test we are also prepared to work with Dr Narang to see whether that test can be validated and work in the field, but I must say that if that test is based on the same nemavirus theory that Dr Bountiff has commented upon in her paper, which is to be published shortly, then I have severe reservations as to whether it will work, but we are still prepared to work with Dr Narang and provide the specimens that he needs under controlled conditions for testing under independent audit to make sure that we have got verifiable results.

(*Dr Narang*) If you look at Document no 1[1], this was the first test which I carried out. You will see on the left-hand side the results given with the dates. If you look at the right-hand side, they were the documents provided to me and you can see for yourself that the specimens being examined by one laboratory are not the same as examined by me. What it shows you is that Keith Meldrum's department has mixed those specimens up—and if you mix the specimens up, then who is examining which specimens? So the validity of that test is not there.

Mr Powell

206. Mr Campbell-Savours says that people are not answering questions. Mr Meldrum, I wonder whether you can confirm that I heard correctly when you told us a few minutes ago that until yesterday you had no information at all—I think the words you used were—about the test about which Dr Narang was talking. Is that correct?

(*Mr Meldrum*) No, I said, in fact, I have a letter in front of me of 9 December 1995 to Mr Hogg[2] in which the test is simply described, in three-and-a-half lines, as a test to determine nemavirus. I have had no further information on what validation work Dr Narang's would like us to do with him, although I saw a letter yesterday that had been in the pipeline about a week or so. I think it got caught up in all the letters we have been receiving in the department, since the BSE crisis.

207. I am grateful for that clarification. Dr Narang, the Secretary of State for Health told us when he was before this Committee that on 4 December SEAC wrote to you asking for information about the test upon which you have been talking. Did you receive that letter?

(*Dr Narang*) I received that letter nearly a month later.

208. Have you replied to that letter?

(*Dr Narang*) I have replied to that letter. I had a 'phone call from SEAC's secretary and I said I am sending all the relevant papers which I have published—

209. Can you let us have a copy of your reply, please?

(*Dr Narang*) I do not have it with me here.

210. Can you let this Committee have a copy of your reply?

(*Dr Narang*) Sure.[1]

211. Thank you very much. You said that you received that letter after a month, so in the early days of the new year?

(*Dr Narang*) That is right.

212. We were further told that the Secretary of State's private secretary wrote to you on 29 January of this year because no reply had been received from you from the letter to you from SEAC of 4 December. Did you receive the letter of 29 January?

(*Dr Narang*) Yes, I did.

213. Did you reply to that?

(*Dr Narang*) No, I did not reply because as I told Mr Powell, I already sent the information to Mr Hogg. What I had with SEAC was a telephone conversation. I was able to send the information—to SEAC Committee.

214. Did you put in writing that you were not going to reply because you had already given the information?

(*Dr Narang*) No, I did not write a letter I just enclosed a compliment slip and published paper. I wrote a letter to Mr Douglas Hogg instead. If you look at the letter I was having correspondence both ways. I offered my services again that I have a test here.

215. What was the date of your letter to Mr Hogg?

(*Dr Narang*) I cannot remember.

216. Can you let us have a copy of it, please?

(*Dr Narang*) Sure[2].

Mr Powell: Thank you.

Sir Jerry Wiggin

217. Professor Bourne, you have seen the papers associated with our interview with Dr Narang. Have you seen any—what I would describe—as proper, scientific papers from him at any time, including his statement to the Agriculture Committee in 1990, which struck me as being supposition rather than a scientific paper?

(*Professor Bourne*) If I could comment, there are two tests on record by Dr Narang. One relates to the Touch test, to which reference was made a moment ago. The other relates to the single strand DNA. I have commented on the single strand DNA and I can comment on the Touch test if you wish me to do so. The third test, which was a hypothetical one from my perspective, because it has not been described in scientific literature, is that on urine. It was only this morning that I learned that that was based, as was the Touch test, on the use of electron microscopy. Only in the last few minutes have I learned that was later linked to identifying putative hypothetical nemaviruses.

218. You and I have known each other a long time and I have very high regard for your work in a number of institutes. Would you be prepared to say that in normal circumstances you would not consider employing Dr Narang on the evidence you have seen

[1] Not Printed.
[2] See p. 102.

[1] See p. 110.
[2] See p. 106.

[**Sir Jerry Wiggin** *Cont*]
of his scientific work? I should remind you that this is a privileged Committee and you can say what you like.

(*Professor Bourne*) Well, it is a very leading question and it is a rather embarrassing one to answer.

219. I have some minutes of a committee meeting held at the Public Health Laboratory Service[1] dealing with the dismissal of Dr Narang very convincingly, which you were kind enough to provide.

(*Professor Bourne*) I am aware of that document. I am bound to say I would find it very difficult to employ Dr Narang in my institute.

Sir Jerry Wiggin: I am sorry, I am required to declare a distant interest in this matter, and I should have done so before asking my question. I work for British Sugar, British Sugar are the same management as a firm of animal feed manufacturers.

Alice Mahon

220. I think, with other Members of the Committee, we are all a little bit concerned about what seems to be a breakdown between the leading scientists in the two departments. Dr Narang, can I say, it has been claimed in the press that public funding was withdrawn from your work in developing a live test because the Government was actually adverse to having the full extent of the BSE epidemic revealed. Is this your view? If so, do you have any evidence to back up what is really a very serious allegation?

(*Dr Narang*) I had been working for a very long time for Public Health. I joined them in 1977. Public Health gave me some assistance to go to America in 1989 and to undertake some CJD related work. At that time everything was fine—I was the best person in the Public Health, they could not have afforded me a few weeks' holiday if I wanted one! After I came back everything turned sour; sour in the sense that I was finding whole cases of CJD every year from 1988 onwards—four the first year, four the second year. We were having a meeting in Newcastle, and somehow or other I was told "Stop this kind of work". I said "Do you want me to stop work?" "No, we do not want you to stop the work, but do not do this work". From then on, for one reason or the other, this has been rumbling on. If you look at the minutes of this meeting on Document No. 2 of 23 October 1990[2]—I can supply you the whole lot of it if you wish, because it would be very entertaining.

221. I would like to ask Mr Meldrum, or perhaps Sir Kenneth, about that, because I found it extraordinary, if we look at the first paragraph on page 2 of those minutes, that there is a statement here: "... One way of getting peer review is to apply for the BSE thing. Maybe have a word with Bill Jarrett. The other side is the smear thing". Could either Mr Meldrum or Sir Kenneth tell us what they thing that means?

(*Sir Kenneth Calman*) I want to make a preliminary point and that is just to challenge Mrs Mahon's assertion that there has been poor communication between departments and leading scientists. I think what you have seen this morning is a great deal of communication between the departments and their leading scientists, and I would not like that to go unchallenged. That is the first point. I think what you see are multiple letters backwards and forwards between scientists of all sorts in this particular area. I think it is inappropriate to say there has not been good communication—there has been a great deal of communication. Secondly, I am not sure I have in front of me minutes of that meeting. Is that the minutes of the—

222. It is the Review Group, I think, considering Dr Narang's work in 1990. Professor Smith was in the chair.

(*Sir Kenneth Calman*) I am not a member of the PHLS and I was not on the board. "Maybe have a word with Bill Jarrett"—I happen to know Bill Jarrett—"The other side is the smear thing". I could not comment at all. I am not sure what that means at all.

223. It is a fairly sinister sentence to appear in some minutes. It looks to me, as an outside observer, as if all of a sudden—to quote Dr Narang—"when I came back from America I was no longer the best thing since sliced bread and my work had to stop". Then we have this extraordinary statement that refers to a "smear thing". Mr Meldrum?

(*Mr Meldrum*) I am sorry, I cannot help in this respect at all, because I have nothing whatsoever to do with the Public Health Laboratory Service. I have explained our work and the commitment that we have to work with Dr Narang and my comments still stand on the record.

224. Dr Narang, I would like you to comment on those minutes, please.

(*Dr Narang*) If you refer to this document dated 4.3—Document no 1,[1] which refers to MAFF providing me with specimens, and look at the date when the mix-up of these specimens has started, they coincide with each other. If you read the next sentence after the "smear", "Try and set up blind trial". This was the blind trial which was going on at that time.

225. Would you like to explain to the Committee a bit more?

(*Dr Narang*) It looks to me that this blind trial which was undertaken was not really a blind trial at all; they were mixing up specimens, one way or the other. If you can mix up even the dates—these people are holding 20 specimens a day, how do I know they have given me number one, or number two or number three? It has come out of the fridge.

Chairman

226. I have to say, Dr Narang, I do find these documents you have presented here extremely confusing myself. I have just got pieces of paper strung together, and half the time I do not know which bits refer to what. Therefore, I can understand there is confusion in some Members' minds as to what exactly we are looking at.

[1] See p. 65.

[2] See p. 65.

[1] Not Printed.

[**Chairman** *Cont*]

(*Dr Narang*) There is the Document No. 1, Chairman.

Alice Mahon

227. I am not confused. I think I can follow.

(*Dr Narang*) In Document No. 2 the dates are very, very close after that. So what was being discussed at the PHLS—the "smear thing" happened to me a few days later. Therefore, to evaluate in an independent way—to me it was not independent at all. It was a mix-up.

(*Mr Meldrum*) I am sorry, I have to dispute that very forcefully indeed. That is not true. That is, in fact, an incorrect statement. If I went through the files again I could demonstrate quite clearly that there was some interim confusion on the dates of submission of samples from our Veterinary Investigation Service Laboratory in Newcastle, but those were resolved. I say that in part because I have in my hand a letter from Dr Narang to Dr Lightfoot who was director of the PHLS at Newcastle, dated 20 March. That was, a draft paper to go into the Veterinary Record on this particular blind trial to determine whether or not Dr Narang's test would identify cattle infected with BSE after death. In the words that he has used in the draft he was able to pick out three out of five of the positive animals that we had identified by both post mortem examination of the brain sections and by examinations for fibrils. On the other side, all the samples that were negative were also found negative by him. Therefore, on the positive brain submitted to him, only 60 per cent were found positive which we had found positive and in one case the brain was very positive indeed. It was a three plus positive on fibril detection. Certainly I would dispute that the minor difficulties with dates of submission of samples had any bearing on that particular experiment. It is only a pity that particular information was never published. I have in front of me, as I say, a copy of the original draft.

Chairman

228. May we see a copy of that document please?

(*Mr Meldrum*) Of course.[1]

Alice Mahon

229. Dr Narang, how would you comment on the allegations which have appeared in the press that you have been obstructed in attempts to obtain heads from abattoirs for random sampling to determine numbers of BSE infected cattle which might have been getting into the human food chain?

(*Dr Narang*) Yes, this is what happened. I applied for the Ministry of Agriculture grant and this was something like £9,000 for a year and I would obtain 100 heads from a local abattoir to see what percentage I would find positive. On this statement, after the submission of the grant, I had a word with Mr Ray Bradley and he told me: "Harash, your test is very sensitive. It will make most of them positive. I know it, you know it, the minister knows it, he does not want your rubber stamp".

[1] Not Printed.

230. Which minister are we concerned with here?

(*Dr Narang*) I cannot remember who the minister was but this was Ray Bradley who told me this, he made this statement.

(*Mr Meldrum*) Madam Chairman, I am sorry, this is quite ridiculous. We are making statements—

Mr Alexander

231. It is hearsay.

(*Mr Meldrum*) Thank you very much indeed, I was going to say that myself. Before we would put money into a screening test in abattoirs we would have to be certain that a particular test had been validated and would deliver correct results. I think so far this morning we have indicated to this Committee that the tests that had been used by Dr Narang—both the touch test and the nema virus test—do not deliver that particular level of sensitivity. Therefore we would not wish to use a test as a survey in slaughterhouses unless we had confidence in that test. It must be a sensitive test to detect animals both affected by and incubating BSE. We have to go through this stage by stage. To do a survey without a validated test would give totally invalid results.

Chairman

232. Sir Kenneth, have you anything to add to what has been said?

(*Sir Kenneth Calman*) Yes, I have, it is about the minutes on Document 2. I saw these for the first time last night, I am not sure whose minutes they are. It would be helpful, through you, if we could clarify who wrote these minutes and under what circumstances were they written. I am not at all clear.

233. Thank you very much. We will try and find out.

(*Sir Kenneth Calman*) Perhaps if Dr Narang could respond to that, whose minutes are they?

234. This is document 2. This is what I was saying earlier, we have documents here and quite honestly I do find it confusing because there is no heading to it, it is just minutes of a meeting. We do not know why it was held or anything. Can you please clarify Dr Narang?

(*Dr Narang*) This meeting was held by PHLS to peer review my work.

235. By whom?

(*Dr Narang*) Public Health Laboratory Service.

(*Sir Kenneth Calman*) Who wrote these minutes?

(*Dr Narang*) Public Health Laboratory Service have these minutes and they are in their documentation. They took the notes and they have come out as a consequence of PHLS being asked to disclose them.

Mr Whittingdale

236. One thing which it appears everyone is agreed about is that the development of a live test would be a major breakthrough. Dr Narang, you have indicated you believe that you are developing such a test but clearly you have failed to convince our witnesses, the Chief Medical Officer, on the Government side that your test has any validity. Can you say whether any

[**Mr Whittingdale** *Cont*]

other research group anywhere in the world is conducting research along the lines that you are doing and whether there are any other findings which bear out the kind of successes that you are claiming in your test?

(*Dr Narang*) What I would say in very simple terms is: in the test which I use, the procedure is different from other tests but the end product is the same. If you ask any scientist round the world and say: "If you see nemavirus/SAF scrapie associated fibrils in this sample is it a positive specimen or not?" they will say it is a positive specimen. So the procedure is different, that is the only difference here, I take a short cut, and their people, they purify it and it is a much longer procedure.

237. But you have given an indication that this is a general approach you are adopting. Are you aware of anybody else who is seeking to duplicate your work in this area or validate it?

(*Dr Narang*) I would say in this country if the Government is trying to stop my work one way or the other I do not think they will be encouraging anybody else to do similar tests.

238. What about other countries?

(*Dr Narang*) Other countries? You see you have to look at it. In America there is no BSE. I have approached a number of other people in a number of other countries. They do not have BSE. The problem is here in this country. People often say to me: "Why do you not go and work abroad" but the problem is here and we have to work here.

239. Could I just ask Mr Meldrum: you appeared to indicate earlier that you had examined this route as a possible method of developing a live test and you had essentially reached the conclusion that it was a blind alley. Is it your view that the kind of test which we are hearing about from Dr Narang is not worth exploring further?

(*Mr Meldrum*) As I understand it, Chairman, the two tests that we are talking about, the Touch test and the electron microscopical examination for presence of nemavirus, are both post mortem so yes, we are looking for a test which will be available ante-mortem. As we said last time we gave evidence to the Committee we are looking for a test which will give us a differential diagnostic test that we can use in a live animal: those that are or are suspected of being infected with BSE and those that are not. So that is one purpose. Then you can use the same test for screening purposes if you wish, bearing in mind that we know the public health angle of this is dealt with by the programme of controls that we have in place to protect the consumer, which I will not comment upon again, which we discussed last time we met in some detail. I think Professor Bourne has a comment he wishes to add.

(*Professor Bourne*) I wish to comment that there are two approaches to developing a test. One is a specific test and the other is a non specific test which nonetheless may have a large degree of usefulness. A specific test is based upon identifying the infectious agent or the abnormal prion protein. The golden egg would be to identify nucleic acid which is associated with infectivity. Dr Narang suggested that the nemavirus was such a nucleic acid, as I have indicated scientists have been unable to prove his finding. Other work is going on in the UK and the States and elsewhere looking for the putative nucleic acid. There are tests also going on in the UK and elsewhere trying to identify the changed prion protein. The approach to that would be by developing appropriate antibodies. The non-specific tests relate to identifying other proteins which would simply be associated with BSE, CJD or neuro degenerative disease. There is work going on mainly in the States as far as I know in that particular direction with respect to CJD and some work also with BSE. I think the problem with BSE is that thus far we have been unable to demonstrate infectivity outside of the brain or spinal cord. So it is unlikely that any test would be applicable unless one uses brain or spinal cord tissues. I think having a test on a live animal is going to be extremely difficult and certainly beyond scientific knowledge at the moment.

Mr Martyn Jones

240. Can I just clarify, I have been given to understand that the accepted wisdom is we do not know what the agent is causing any of these transmissable spongiform encephalopathies and that Dr Narang's suggestion is that nemavirus is actually the causative agent. Are there any other eminent people who think nemavirus is the agent that is causing this?

(*Professor Bourne*) I am unaware of anyone who supports the nema virus theory. What we do know at the moment is that infectivity is associated with the prion proteins. There are two schools of thought, and I am sure you know, that it is the protein itself which is the infectious agent and another school of thought which is the protein has within it a nucleic acid core. There is a lot of experimental evidence to support one or the other theory. There is a lot of work going on looking at this putative nucleic acid. To find that would be a really major achievement and there was no doubt that when the experimental work was embarked upon trying to substantiate Dr Narang's findings it was done with a great deal of enthusiasm and hope that one would find such a nucleic acid and associate that with the disease. It failed to do that.

(*Dr Narang*) The difference between nemavirus and prion protein is that prion protein is the central core, like in a pencil lead, and the wood outside is the DNA wrapped around it. Therefore if you purify one, the inside one, the outside one goes with it. That is the difference between my hypothesis and now a prion person Dr Prusiner also believes what I said in 1992 that after all, nemavirus is the agent.

Chairman: Thank you very much. We now move on to questions to Dr Dealler, Mrs Wise?

Audrey Wise

241. Dr Dealler, in your written evidence you cast doubts on several aspects of the received wisdom as to the way this problem has arisen and the way the disease is transmitted. Could you elaborate in particular on why you claim that it now seems unlikely that BSE was derived from scrapie and that it may well not be true that the change in food manufacturing processes at the beginning of the 1980s was the cause of BSE?

[**Audrey Wise** *Cont*]

(*Dr Dealler*) I should say that a lot of the assumptions made back in 1987/1988 by the group that were deciding on this were very reasonable, very rational and were the best guesses you could make. The problem was that as information arrived later on it was very difficult, for reasons I am not sure, to change the opinion and the original best guesses were backed up and kept solid right towards the end until they were absolutely collapsing. One of them is, for instance, that the disease was derived from scrapie. This is not the only one, all the ones we are talking about here are all collapsing. We were expecting for instance that when the disease goes from one species to another the incubation period would be relatively long and then when it went from one cow to the next cow, the incubation period would drop and drop again and drop again. As we saw the epidemic the incubation period would get shorter and shorter; that has not happened, the incubation period has remained relatively solid. The next thing that they found was that they expected there to be an outbreak to some degree of scrapie in sheep because similar food was fed to cows as was fed to sheep but not in such large amounts. 400,000 tonnes of added feed was fed in fact to sheep in 1994. There has been no outbreak in sheep and if this was the disease of scrapie we were expecting there to be an outbreak. The next thing was that we expected that the distribution of disease inside the brain of scrapie and BSE when inoculated into a similar animal would be very similar. In 1991 it was announced that when these two diseases were inoculated into the same kind of mouse the distribution of the disease inside the brain was different in fact. The next thing that happened was that scrapie was inoculated into cows and this experiment for some reason—I am not sure why it was only done in the States—and the disease caused in the cows did not turn out to be like BSE and that experiment has been repeated in the States. The next thing they tried was inoculating BSE into sheep, that took place in the UK. Again in the distribution of the disease inside the brain of the sheep—this has not been published by the way—the sheep was different in fact. In other words, all the things which were expected did not happen. I think at the moment we cannot say there is any evidence at all that BSE was derived from sheep. All we can say now is that if it was derived from sheep it would have been from a very uncommon strain of disease. Although we cannot state one or the other, I think at the moment it would be unwise to say that BSE was derived from sheep. I took a small survey of researchers at Gottingen in Germany at the last major conference and I found nobody who thought that BSE was derived from sheep, apart from people at the Ministry of Agriculture.

Chairman

242. The second part of the question was concerning food manufacturing processes.

(*Dr Dealler*) Food manufacturing, some work was published by Taylor in the veterinary records in December of last year. After a lot of very hard work—and I think he should be congratulated for his work—basically what they expected was that the feed manufacture that took place before the change, which we perhaps say took place in 1980/1981, a lot of fatty material was extracted using solvents—acetone, trichloroethylene—and afterwards that fatty material was separated by heat and centrifugation. They thought that change went from methods before that date, infectivity was being destroyed in some way, and after that date it was not, so infectivity managed to get through after that date. What these results show is that it could not demonstrate that at all and in fact we found it was the other way round, we have had to repeat a certain amount of the work but at the moment it appears the other way round, infectivity was not destroyed in 1991 and was being destroyed afterwards. I think this work has to be repeated. At the moment again the point comes out that only 80 per cent of the tissue was in fact being treated in such a way as solvent extractions before 1980, 20 per cent was not of course.

243. Can you bring your answer to a close.

(*Dr Dealer*) That is basically the reason, the science came out the wrong way round.

244. Mr Meldrum?

(*Mr Meldrum*) I will be brief, Chairman, we discussed this last time also when we gave evidence. We have taken the view for some time that the two hypotheses that BSE may have come from scrapie in sheep or may have been a natural strain occurring in cattle run side by side; that has been the Ministry's view for some time. There is nothing new. I can recall a number of statements made to that effect and also articles written by researchers within my Department. On the second point, I would not dispute work done by Dr Taylor from the NOU in Edinburgh which has a bearing on the origin or the reason as to why we have so many cases of BSE but that also should be seen against the comments that I made at the last meeting of this Committee when I was talking about the continuous rendering systems introduced in the early 1970s. One of those in particular we know now has no effect whatsoever upon the agent of BSE and it would survive that particular process.

(*Dr Dealler*) Could I answer one thing in this. Throughout the early 1990s we have been having statements coming from the Ministry of Agriculture that because BSE was derived from scrapie therefore there would be no risk to humans from BSE. I think that should be made absolutely clear.

(*Dr Narang*) Chairman, can I make a point: I have done an experiment heating a brain at 121 degrees for 15 hours and then inoculating it back into animals. One of the eight animals developed the disease. Imagine this is a temperature of 121 degrees for 15 hours and still it did not manage to kill the agent. You have something like 12 per cent of the animals developing such a disease.

Chairman: Dr Narang, you have had your turn earlier, let us concentrate on Dr Dealler. Mrs Wise?

Audrey Wise

245. Except, of course, that Mr Meldrum also gave evidence before the recess and these are the only two people that we are having who actually have a different point of view. I am anxious to understand their points of view to make some judgments. Not

[**Audrey Wise** *Cont*]
just have a comment on it all the time, I want them to develop their point of view. It seemed to me from reading your material, Dr Dealler, that although, as you have said, these were best guesses and not unreasonable and you are not critical of them in that sense as scientific hypotheses, you are critical, it seems to me, of the things which follow from that in the public health aspect in that you say: "When a large proportion of the population may be exposed to a fatal condition to which there is no treatment, assumptions should be made that would avoid potential risk". So best guesses really are not enough, you are saying, when public health is involved. Are you saying that there has been an over-optimistic approach, perhaps even a somewhat complacent approach, because it was felt that it was derived from scrapies so we knew that, it was the change in the feed so we knew that and that this might get in the way of actually finding other answers?

(*Dr Dealler*) I am afraid that is what I basically found out. This is why I carried out the research looking into the number of infected cattle that we were eating. We have been given information continuously by the Ministry of Agriculture about the number of cases we were not eating but that did not matter, it was the number of cases we were eating that mattered. From there I had to do calculations on the risk assessments to try to work out the risk that that represented to the population in the UK. I can find no evidence that this information was being given to SEAC. I can find no evidence that the Ministry of Agriculture were doing this themselves. I can find no information of this coming through to the House of Commons. Questions were actually asked in the House of Commons asking for data on the number of infected cattle we were eating and they have been basically avoided and misleading data has been given as a result. I felt I had to do it. Really as an independent scientist I thought that was necessary.

246. You have said in your paper to us as well that the decisions were made on best guesses which were not decisions which should have been taken on public health grounds but that experts in the field of public health, medical ethics, infectious diseases, medical microbiology, were not present on the committees at that time but that has been remedied. How serious do you think this omission is and why do you think it came about?

(*Dr Dealler*) I can only go on the seriousness of it by looking at the potential risk that has been taken to the population. I think changes have been made, and full congratulations must go to Professor Pattison in this respect, a large change has been made in that now a lot of information is arriving at the committee concerning public health. One of the major ones, I think, to do with this is to do with the fact that if a risk is taken when it is known that a large proportion of the population will be exposed to infected animals you have to work out what is an acceptable risk level in public health terms. For instance, a good example is with chemicals. If I tell you that a certain chemical at 1 mg will kill a mouse, the amount that you would allow to be exposed to a human may be one hundred times less than that. If it produced a fatal disease with no method of treatment, no method of diagnosis, passed from one species to another, not destroyed by cooking and was going to be exposed to the entire population then you are talking about probably 10,000 times less than that amount being acceptable as the amount that the human population should be exposed to. I could find no evidence that that kind of public health work was taking place. They were not allowing for the fact that you had to put a large barrier between the risk to the animal in the experiment and the risk you would take with the human population.

247. Thank you. The question of the cumulative nature or non-cumulative nature, could you explore that a little bit because a very great deal seems to hang on whether this is cumulative or non-cumulative?

(*Dr Dealler*) My initial experiments were done assuming that it was cumulative basically because when I visited a lot of experimenters in the United States that is what they told me they assumed it to be and a lot of the researches were not done to decide whether this current disease was cumulative or not, a little bit today, a little bit tomorrow and a little bit the next day and it built up, they just decided that it was. It was an assumption. When I initially carried out my calculations in terms of human risk I assumed that it was cumulative. Dr Tyrrell wrote back to me and said that there was no evidence that it was cumulative and they were going to assume that it was not cumulative. I cannot see any justification for this in public health terms. What I had to do then was to do the same kinds of calculations assuming that it was not cumulative. This data was also sent to Dr Tyrrell and I had no reply.

248. When was that?

(*Dr Dealler*) That was in 1993. Information was given to the Tyrrell Committee, initially via Ray Bradley, in the middle of 1993. It was only given to the committee five days before the committee was going to meet even though Ray Bradley had it three months in advance. It was complex statistical information, it would be very difficult to understand in such a short period. It was difficult to see how the committee at the time could make decisions on it without making specific assumptions which at that time would not be the assumptions made for public health.

Sir Jerry Wiggin

249. Dr Dealler, I do not know how much of your time as a consultant medical microbiologist is spent on research but you must *per force* of your qualifications have observed scientific research presented, proven and published. In the four pages of statement which you have put in your paper you have simply made a number of assertions without the slightest technical proof. Is it any surprise to you that the real experts who studied this matter and spent many millions of pounds and hundreds of thousands of man hours in coming to the conclusions that they have actually reject your assertions because I can see them as no more than assertions?

(*Dr Dealler*) Well, this is a problem that has taken place with not having people outside MAFF actually carrying out the epidemiology on it.

250. Oh, come on.

[**Sir Jerry Wiggin** *Cont*]

(*Dr Dealler*) It is very difficult. I would give them full marks, I very much appreciate John Wilesmith's work, but what has happened is that they have taken specific assumptions all the way through where the number of assumptions that could be taken is really very wide. What I had to do was to look not just at the direction that was assumed there but the various other groups it could be.

251. Would Professor Pattison like to comment on that remark?

(*Professor Pattison*) I can only comment about the time that I have been on the committee and in particular its Chairman. Dr Dealler and I share quite a long correspondence. It is always helpful to have his letters and his publications and actually in fairness to him probably all of what he is saying has been published in the scientific literature. I think the difficulty is, as he himself said and SEAC would also admit, that there are just one or two very important unknowns in this area so you have to make assumptions. When you make assumptions of course you do not always know that actually they are going to be proved to be the right assumptions in the end. I think we have moved on now and we have got to the stage, having described the ten cases of the new variant, where it is only continuing surveillance and accumulation of actual data which is actually going to lead us to the right conclusions. I would not disagree from the broad limits of possibilities that Dr Dealler sets on many things.

Mr Bayley

252. To try and make it simpler for us non-scientists, as I understand your work you have not established that the infective agent jumps the species barrier but you instead are assessing what the risk would be if that happens against a number of assumptions?

(*Dr Dealler*) That is correct.

253. At the earlier two evidence sessions a number of members of the Committee sought from Professor Pattison and others an estimate of the level of risk from eating beef and some Members put in assumptions of their own that it was one hundred thousand times less dangerous than smoking a cigarette. I forget what the assumptions were but they were of a popular sort which have been printed in the newspapers. Professor Pattison said that he was unable to put a figure on the level of risk although the Secretary of State has said that it was extremely low—I forget his precise words. Does your work suggest that that could be the case but the level of risk could also be higher? My question is, am I representing your work reasonably and perhaps Professor Pattison afterwards could comment?

(*Dr Dealler*) I should think Professor Pattison and I would agree all the way through this one in that it is actually very difficult to be sure of the risk that has been taken. The problem is that we do not know when the recent ten cases became infected and although we can calculate the number of infected cattle that would be eaten at different times if, for instance, these ten patients became infected in 1982 there were only 274 infected cattle over 50 per cent of their incubation period that were eaten and those ten cases might only be one per cent of the ones that died having eaten the tissue in 1982. If that is the truth then the number of humans that will be incubating the disease at the current time will be very large and we are talking about millions. Whereas if these ten patients had become infected in 1989 when there were relatively large numbers being eaten, and we are talking about hundreds of thousands of infected cattle being eaten each year by 1990, for instance, what we can then say is that the relative risk of people incubating the disease would be very low indeed. At the moment there is inadequate data. That is one method of calculation. The other method of calculation depends on working out the amount of infectivity present in the tissue that we are likely to have eaten and looking at other species to see the chance of infectivity being passed over in them. I am afraid when that is done again the potential risk comes out to be unacceptably high in public health terms.

(*Professor Pattison*) At least in the first part of his statement Dr Dealler is correct and we would agree. You have got the table that he included in the submission to you and you have probably heard the comments that I have made on television that in fact we may have no more cases, and that is the ten in the bottom left-hand corner, and we may have a lot more cases and those are the figures in the top right-hand corner. As I said before, I would not disagree at all that this is currently within the possible range of what might happen but actually we need now to find out what is going to happen. I think the longer we go on without a very significant rise in the number of cases, the more it would tend to put you towards the bottom left-hand of that table rather than the top right. Perhaps I should also say that of course we are just dealing with the time period before the offals ban so we will always need to distinguish between that risk and the current risk. Very often those two things are elided together. Our estimate I do not think is as high as Dr Dealler's of the risk that has been run. For one thing it would be very surprising, although nobody can prove it to you at the moment, if there is no species barrier between cow and man. As I say, we cannot prove that at the moment but it would be very surprising indeed. It would be very surprising if muscle meat turned out to be an important source of replication of the agent of this disease because it is not in any other spongiform encephalopathies. So even though we have relatively insensitive tests, the mouse bioassay being least sensitive and the cattle bioassay being of course much more sensitive, you can only say that the figure is less than ten to the minus six, less than one in a million. It would surprise us enormously if it was just under one million. That was the point that Sir Kenneth Calman made last time to you about the floor. I think there is not a major disagreement about this but in relation to the level of the risk, particularly the level of the risk now, there may be disagreement.

(*Dr Dealler*) Can I say that I find this completely correct. My own publications have shown that the added risk to adults in the UK who have continued to eat beef in the UK must be absolutely minimal. That has been published in *The Lancet*.

[Mr Bayley *Cont*]

Mr Martyn Jones

254. Can I come back to the original challenge you made to MAFF's assumptions that scrapie jumped into cattle. Obviously scrapie was not the reason for all the epidemic within cattle but it was recycled BSE effectively and that means there has not been a species barrier jump and presumably the risk will be much less. Perhaps you could comment on that? Also, if it was not a feed problem, if it was not the recycling of the feed, how do you explain the epidemiology of the BSE in cattle given that there was a rapid rise and it is now falling steadily?

(*Dr Dealler*) I think it is the recycling of food that is involved here. The epidemic rise follows two patterns. It follows the pattern that the cows themselves that are showing signs of the disease ate infected material themselves but it also follows the pattern that it is the offspring we are seeing with the disease, it is not the cows themselves that we are seeing going down with the disease. Do not forget that it is milking cows that get fed by far the excess of potentially infected material late in their lives and the cows we are seeing going down with the disease are potentially their offspring. The second thing is where did this disease come from? The idea held really by most of the experts in the world is that it was from a disease in cows which was transferred to other cattle. This does not really make much difference to the potential risk. Do not forget that BSE has now been transferred to 19 different species and 17 of them by mouth. If anything I would say it is that we cannot make a statement as to whether it is highly infectious to humans or it is not.

255. You are saying maternal transmission basically? If that is the case why is there a decrease in cases now?

(*Dr Dealler*) One thing I should say is that there is an argument as to whether the numbers are dropping very fast at all. Information was published in 1995 showing that the numbers probably are not dropping at the rate at which we are being told by the Ministry of Agriculture. The next thing is that when the numbers, the estimated numbers, for the drop in the number of cases of BSE is looked at compared to what the number would be that we would expect if it was vertically transmitted, unfortunately it is exactly the same. The number we are seeing is the number we would expect from maternal transmission.

Chairman

256. I would just remind everybody that we have ten major questions still to ask in an hour and a quarter, please short questions and short answers. Mr Meldrum?

(*Mr Meldrum*) We have no evidence of maternal transmission. I can provide evidence of that; it has been published already. I would just challenge the statement made by Dr Dealler that there is such a phenomenon occurring. In fact, we take the contrary view that if it is occurring it is at a rate of less than five per cent. The information he gave you on the number of species affected is massively misleading; most of the species that he has commented upon were experimental animals. There is in fact another comment he has made on under-reporting and he is challenging the data that we provide. I would challenge him formally to provide any evidence that the information we have provided to the public and to this Committee is incorrect. He is suggesting that we are in fact not giving the full information on the number of cases reported to us and the number of cases confirmed. I can assure the Committee that is not the case and that all information is provided to the public and there is no evidence of under-reporting. If so we would know about it from other sources and from the audits and the checks that we do elsewhere.

(*Dr Dealler*) I think it is quite important that it is made absolutely clear that I have been phoned up by numerous farmers telling me that they did not report cases of BSE, they just took them to market. I watched a very good tv programme that was produced by *World In Action* about people picking out cases of BSE that had been taken to market. What my data does not say is that the Ministry of Agriculture is not reporting data to the House of Commons, I have no evidence for that at all. The exact reason why the under-reporting appears to have taken place, I cannot state. That it actually is seen is because it is a statistical anomaly and where the difficulties take place I do not know.

Mr Pickthall

257. Dr Dealler, would you like to comment on what Mr Meldrum has just said about maternal transmission, or the possibility of it, as I understood him to say that it is not occurring and if it is occurring it is less than five per cent?

(*Dr Dealler*) The information was published at the international conference in Gottingen last year, it was also published at the Society of Veterinary Epidemiology about three weeks ago. This is the data concerning vertical transmission and the evidence for it. The evidence we have got at the moment is desperately statistical. One of the things that we would expect, for instance, is that if it is cattle going down with the disease the incubation period would be gradually dropping and it is not, it is as if the cattle after we are seeing the disease are becoming infected from a similar source throughout the epidemic. The next thing we were expecting was that as the epidemic went on it would be like winning on the Lottery, a cow wins the Lottery and goes down with BSE. The more Lottery tickets there are as the epidemic rises the number of cattle going down with BSE in any particular herd is expected to rise in parallel with the national rate but in fact it rises to a certain amount and just stops. It is as if there is only a certain number that could be infected within a herd. That is exactly what you would expect is the appearance of vertical transmission. The next thing is that of the cattle exported to Portugal with some of their offspring which have gone down with BSE we have no evidence they have been fed any infected material at all, and it is the offspring which have gone down with the disease. With some of the cattle which have been on organic farms where the mother has been brought into the herd, it is the offspring which have gone down with the disease. When we look at the age distribution of cattle going down with BSE we expect

[**Mr Pickthall** *Cont*]

there to be a single great big peak and in fact that peak is at age five. But when you correct this for the number of cattle in the bovine population you find there are two peaks, there is a great big peak at age five, then it goes down and goes back up again, and we have now got good statistics to say there appears to be two populations which are involved here, and that is what you expect with vertical transmission. I have offered to give this information to the Ministry of Agriculture but have had no reply.

258. So your argument about the possibility of vertical transmission is entirely based on statistical patterns that you have put together?

(*Dr Dealler*) It is based on the Ministry of Agriculture data, yes.

259. You are not aware of scientific evidence that demonstrates vertical transmission? You are very critical of the experiments which are going on at the moment?

(*Dr Dealler*) What I would say is that I am very worried about the experiments which are taking place. If they had taken the offspring of cattle they know have got BSE and at the same time taken offspring of cattle from the same herd, my data would suggest that not just the cow you know has got BSE will have BSE but also the cow you did not think had BSE because it is from the same herd. Why I am very worried is that you will find no difference between the two groups. In fact it would be very difficult now to justify the results of that study.

260. And being from the same herd, how would they have contracted the disease?

(*Dr Dealler*) In the same way—

261. From feed?

(*Dr Dealler*) From feed, yes. What would happen is that both the offspring from the cow known to have BSE and the cow not known to have BSE, would have the same distribution in the study, so there would be no difference between the controls and the tests. Obviously they did not know this when the experiment started, they could not have known this, and nobody must say anything against the epidemiologists who organised this, but that has now come out to be a good possibility. This means it would be very difficult to say vertical transmission was not taking place using this study.

(*Mr Meldrum*) We have reported to you already a paper published in the *Veterinary Record* on 1st April 1995 which looks at the possibility of maternal transmission, and the conclusions I have already given you this morning. On the maternal transmission experiment to which Dr Dealler referred, I would not accept the criticisms he makes of it, for very detailed reasons, but the results of that experiment will not be available until early 1997. To put it simply, I have in front of me some figures from one particular herd of pedigree cattle, one of the first herds which had BSE in the UK. The first cattle were probably exposed in 1980 and when the feed ban came into effect in 1988/89 there were no more cases born thereafter; there was a complete cut-off. There were 83 cattle with BSE in that herd. If there had been maternal transmission, surely one would have expected to find animals in subsequent cohort calving patterns going down with the disease. That has not happened. This is a herd of very long standing and very good records.

Mr Bayley

262. Why do you draw the conclusion that you do from a sample of just one herd out of the whole of the national herd? Surely one must deal with a larger sample than generalise from one herd to the whole of the cattle population of the UK?

(*Mr Meldrum*) I thought I said in my introduction that was by way of an example when I mentioned that herd just now.

263. But on its own it tells us nothing, does it?

(*Mr Meldrum*) It is an example of what I am talking about. I was giving an example of why we do not believe maternal transmission occurs. The article I referred to was an investigation into about 290 herds, and the article I referred to has been published in *Veterinary Record* last April, and the conclusions are already reported to you. I was using that single herd as an example as to why we do not believe maternal transmission exists.

264. Was that representative of the entire cattle population of the UK?

(*Mr Meldrum*) No, that is just an example of one particular herd where I would have expected to have had other cases born later if maternal transmission had been occurring in that herd. There is certainly no evidence of maternal transmission occurring in that particular herd.

265. With respect, what has happened in one atypical herd cannot possibly be the basis for generalisation?

(*Mr Meldrum*) I am sorry, I do have to say I thought I had made that point already. The case control study to which I referred and which was published in the *Veterinary Record* concerns investigations into about 290 herds in detail to determine whether in addition to feed there could be any other factors involved in BSE, and they have concluded they can find no such evidence.

(*Professor Bourne*) I wish to respond to the comments Dr Dealler made on the incubation period. It is true experimentally one finds when one crosses the species barrier initially one has a long incubation period. It subsequently reduces, but it does not go down from that period, for it remains constant. The second point I wish to make is that the incubation period has been shown experimentally to be related to dose, the smaller the dose the longer the incubation period. I believe the field findings which are far from an experimental situation are not inconsistent with the experimental findings, and they are not inconsistent with MAFF's epidemiological data.

(*Dr Dealler*) I would argue with that in that the work done by Marsh in 1987, the work done by Hadlow in 1982, the work done by I think another group in Wisconsin during that time, all showed that although it drops initially it did not go down much further. The work done by people in Edinburgh and particularly Kimberlin showed a drop but the incubation period did not go down much further. His work has a wide confidence interval and I would say at the moment we have to argue with it.

Sir Roger Moate

266. Dr Dealler, what alarms me, and others might feel the same, is the extent to which we wander far away from science into the realms of hypothesis, speculation and then the suggestion the statistics which are necessary to prove the initial hypothesis are not true or are altered. It seems to me that any commonsense view would tell us that if there is maternal transmission we would not be seeing a significant fall now in BSE cases. You say there is not because the statistics we have are wrong, that we still have a much higher incidence of BSE than we are being told. You do not doubt the Ministry's statistics but you say that farmers are suddenly cheating, because if they had always been cheating the statistics would be the same throughout. You say it is based on the number of farmers who have phoned you up. I find this very unconvincing. How many farmers have phoned you up saying they have been cheating the system?

(*Dr Dealler*) About five have phoned me up. I did a survey at the Yorkshire Show going round asking people, and it is worrying. What has happened is that it appears that in about 1992 for some reason, and I am not sure what it is, the marketeers, the auctioneers, for some reason, and I cannot pretend to know the reason for this, decreased their asking of farmers for information about the cows themselves and whether they were from an infected herd. This might have been due to the introduction of the computerised system for finding which cows were from an infected mother and which were not for export. It is difficult to try and work out why these numbers have taken place. I am sorry but the numbers are really very large indeed which I am talking about. These were carried out by a professor of statistics in Leeds and we decided that because it was going to be difficult they would be calculated in two different ways, using two different sets of data derived from the Ministry of Agriculture, because we knew there would be this problem, that it would be difficult to believe such a large amount of under-reporting was apparently taking place. So there is my information from the Show and from people ringing me up, and from one vet going through this quite clearly with me, he was asking his farmers, "Why are you not telling us about cases of BSE any more?" and they said, "Because we are taking them to market". I can give you his name if you are interested. This was, I must admit, out of the blue, but when we did the statistics we found this would fit very well and was derived from two sources.

267. It sounds more politics than science.

(*Dr Dealler*) It is good statistics.

Mr Congdon

268. We have had a long series of questions about a whole variety of things but what I do not totally understand is that the intensive methods of farming are not fundamentally different in this country from other European countries, as I understand it, so why does there appear to be no BSE problem of comparable severity in other countries?

(*Dr Narang*) You can find the details in my recent publication 'Origin and Implications of Bovine Spongiform Encephalopathy' which I would be glad to send anybody. In America I know the cattle were not being fed with this meat and bone meal until they were about two years old. Here cattle are being fed when they are calves of no more than four to six weeks old, so the incubation period starts from that day on. Also I have seen cattle heads going for recycling for meat and bone meal in 1970, 1971 in this country. We do not know whether this process and this practice occurred in other countries, whether they incorporated cattle brain with this material. That would account for the difference. In the Republic of Ireland, as soon as they have seen one or two animals in a herd, they have culled the rest.

(*Dr Dealler*) I do not know the reason for this. A number of ideas were put over and one is that there is another factor involved, perhaps a toxic factor. Another factor might be that we use large amounts of rape seed for feeding cows, which has a lot of selenium in it, and a lot of the offal which was going to recycling was in plastic bags at the time and the idea was that maybe it got through because the actual agent itself is very hydrophobic and would stick to the plastic bags; we just do not know.

269. Do we know the extent to which any of this is different here compared with other European countries?

(*Dr Dealler*) I do not, I am sorry.

(*Dr Narang*) Those are the two points which I know about in America and in the Republic of Ireland.

270. I was deliberately asking about Europe for obvious reasons. As a lay person I must confess I am probably even more confused now than I was two or three weeks ago about the scientific situation. This issue of the so-called new strain of CJD, is it possible that it has appeared in other countries and not been noticed? Linked with that, my understanding is that if you look at the CJD cases in totality, new strains or otherwise, we have no higher rate of CJD in this country than elsewhere. Have you any explanations for either of those?

(*Dr Narang*) I can give you one indicator. In the Far East and India, I have personally seen rabies vaccines being used for very large human populations which have been prepared with sheep's brains, and if you get that genus of sheep's brains along with the vaccine, naturally you could develop CJD at some stage or the other. That explains why we get CJD in vegetarians. Food travels everywhere, you do not get this disease by sleeping with the cows, which is a very important issue. In 1990 I presented a memorandum to the House's Select Committee in which I pointed out two cases, I had discovered of atypical pathology of a CJD. So if we had been informed, we would have known more whether such cases were occurring in other parts of the world or whether this was a new form.

(*Dr Dealler*) I would say that the work of Dr Ironside is absolutely excellent and if he says something new is appearing, I would believe him. When we try to work out when we would expect numbers of cases of BSE, if indeed any appear in humans, to start, all we can do is look at the way this kind of disease passes from one species to another. To do this I would look at the time taken for a mouse to infect a hamster or something like that. This incubation period depends on the normal life

[**Mr Congdon** *Cont*]

expectancy of the animal which receives the infection, it depends on the root of the infection, whether it comes orally or by injection. It depends on the dose of the injection that is received. When all the figures you get from other animals are applied to BSE in humans, it is really unlikely to see any cases of BSE in humans. I would be surprised if we saw any before the year 2000, looking at other species. In fact the numbers we have seen may well be quite early.

(*Sir Kenneth Calman*) Just a factual response to the incidence of CJD in European countries. It is very similar at around one per million. There has been one case of the CJD variant reported in France over the last few weeks and clearly part of the reason for making all this public as soon as possible is to allow as much international dissemination as possible and indeed to look for such cases across the world.

Mrs Winterton

271. Dr Narang and Mr Dealler, would either of you challenge the integrity and scientific credibility of the chairman and members of SEAC who are advising the Government on its policy as far as BSE is concerned?

(*Dr Narang*) What I would say is what I said in my statement, that there seems to be a wide gap in understanding of the science. We have been talking a great deal about the science and as one member has said, they are more confused today than two or three weeks ago. This is one of the big gaps, that we try to interpret the results of science experiments the way we want to, but there is always the other side of the coin. For example, I know of experiments where when you feed the scrapie agent to animals and they do not develop the disease every time. You feed it with BSE and they will always develop the disease.

272. Dr Narang, may I interrupt your answer because you seem to be going all round the Wreckin. Are you saying that in fact SEAC have got it all completely wrong and they are looking at all these matters with their experience, and that actually their theories and their research are wrong and they are giving wrong advice to Government? I asked you about the integrity and the scientific credibility of members of that committee.

(*Dr Narang*) So far as I know, only two members of SEAC have actually worked in terms of experiments with this disease, the others have not.

273. Do you believe that they have integrity and their scientific background is such as to make their work and their advice worthy?

(*Dr Narang*) Not all the members of this committee. I do not really believe they have the scientific integrity of this special subject.

274. Thank you. Dr Dealler, may I have your response?

(*Dr Dealler*) May I say the current members of SEAC I am very impressed by and they have worked very hard and I would be impressed by their integrity. Unfortunately, until Professor Pattison arrived, I would say I felt there was inadequate experience on that committee to know the public health, the medical ethics and the medical microbiology which was required in order to be able to assess the risks to humans, and that was the problem. It was not integrity in any way which was the problem, it was the lack of experience.

(*Sir Kenneth Calman*) To make one fairly brief point, and that is that SEAC provides the Government with advice but they are of course not the only source of advice on a wide variety of issues. In relation to medical ethics issues, for example, we have lots of other people who provide that advice, and similarly in relation to wider public health issues again there are a wide range of people who provide that advice and have done so in relation to this issue, and I would include part of my own function as giving that public health advice, indeed the medical ethics advice as well. SEAC has an important role but it is not the only source of advice to Government in relation to BSE. It is a crucial source but clearly we need to think much more widely about some of these issues.

275. Dr Dealler, Dr Narang, upon what evidence or research do you base your view or allegation of the possibility that the soil upon which animals graze may be contaminated?

(*Dr Narang*) I have never said the soil is contaminated and animals will get BSE from eating the grass from that soil because the agent is more likely to be present in the urine. Somebody asked me if milk could contain the agent too, but as the milk is swallowed—because I believe it is not just simply that you eat the contaminated material that you get infected, you need secondary conditions. For example, you have to have some kind of abscess in the mouth, teeth must be missing and there are cavities in the mouth, so if you swallow the thing straight into your stomach, then it is unlikely that this will get a hold in your body and this is where the difference is. Therefore, by eating simply or drinking urine—and animals do that all the time—and people who drink milk will be not getting this disease.

(*Dr Dealler*) As I say, the evidence from the scrapie study in Iceland in which they found that flocks of sheep which went down with scrapie, is that if they slaughtered these sheep and then brought in replacement sheep from a herd which had no scrapie, they would find that a certain number of the sheep brought in would then develop the disease, and they could do it again, take the sheep away and bring in a new set and some of them would develop scrapie. That experiment has been followed up since the middle of the 1960s, I think, by the Icelandic group and the amount of infectivity that appears in the environment in some way—I do not know whether it is in the soil or not—appears to drop over a long period, but work that has been carried out by Paul Brown in the National Institute of Health showed that if infectivity was put into the soil and if you came back three years later, a very large amount of the infectivity remained there. The next work that was done looked at the presence of infectivity in faeces and infectivity was found in the faeces of one particular species that was tested. The next thing involved the proportion of infectivity taken in by mouth that appears to get in some way into the infected animal, and the ratio between an orally infective dose and an inoculated infective dose may be 10,000:1, and it assumed—I am sorry, it has to be an assumption—by a large number of members of

[**Mrs Winterton** *Cont*]
the research field base that in fact, if you take in 10,000 infective units, 9,999 of them will appear in the faeces, and the worry, of course, is that infectivity would remain in the soil. The worry about this has not been got around. I know certainly that London Zoo were worried about the kudu pen—do not forget the greater kudu has largely gone down with BSE and they have been picking up all the faeces pats and taking them away and incinerating them—they are expecting to remove the top foot of soil from the kudu pen when all the kudus have died of BSE in order to prevent that disease being passed on to any further animals that appear in the field. At the moment there is inadequate data to show that the agent remains in the soil in this form in BSE. Again it comes down to statistics. I am worried that if this is happening in the kudu pen in the Zoo, what about all farmers? The farmers do not seem to be being told that there is a risk of this.

276. On your first point about the experiment in Iceland in scrapie, of course that could have a million and one different explanations, including genetic predisposition?

(*Dr Dealler*) Indeed.

277. But what you have said is that there are obvious differences between species and I would like Professor Pattison, if he will, to respond to what has just been said, bearing in mind my question in the last session about contamination and/or transmission through the soil?

(*Professor Pattison*) Yes. I did say at the last session that we have not actually directly tested soil, so we do not have the experimental data on that. I take all your points about there being differences in species and eventually we have to look at what BSE does in cattle and the soil. All I can say is that you would expect rather a dramatic amount of horizontal transmission, it seems to me, within herds if the pasture was contaminated and that was a ready source of infection for other animals, and whilst I accept all the caveats about whether or not there are minor components of transmission which are not food-borne, it would be very minor and it does not suggest to me that in the natural situation the pasture is a source of infection.

Mr Alexander

278. We have been listening to a lot of experts this morning and we are trying to weave a way through it all and reach a personal conclusion. I have two short questions and the first arises out of—I think it was Dr Dealler who said that BSE had been transferred to 19 different species and Mr Meldrum said that was largely under experimental conditions. Could you elaborate on whether that is the case or whether, in your view, it has been transmissible to different species otherwise than deliberately?

(*Dr Dealler*) What appears to have taken place is that a certain amount of the infectivity present in something like feed and meal has, in fact, been given to animals in zoos and these have gone down with what has turned out to be BSE by further experiments. These would be gazelles, impalas and like animals. A certain amount of infectivity probably has been transferred to zoo animals by mistake through feeding bovine tissue to big cats in zoos. In the experiments that have been carried out actually by either inoculating or feeding bovine tissue to sheep, goats, pigs, monkeys, these have been carried out by action as an experiment.

279. The other question I have is this: how confident can we be—I hope we can be totally confident but please say so—that transmissible spongiform encephalopathies which might affect human health have not been transmitted to other edible species?

(*Dr Dealler*) I have inadequate data in this respect. I would ask Mr Meldrum to answer this.

280. This is what we really want to know, is it not?

(*Dr Dealler*) Yes, it is. As I say, they were expecting a certain amount of infectivity to have increased the amount of scrapie in sheep but that does not appear to have taken place because it is actually difficult to get farmers to report cases of scrapie. That is the worry, that only a proportion are, in fact, being reported. The experiments that took place in pigs, in which pigs were fed infectivity, I believe they are still waiting for reports on this and there are things under the table but I am not sure we can take it as being true. I think at the moment the worry is that if you do not know whether something is a risk or it is not, if it is a fatal disease with no method of treatment, do you assume it to be a risk at present or do you assume it not to be a risk at present, and this has been one of the problems with BSE from the beginning. Being on the medical side, we always assume something to be a risk until there is adequate evidence that the risk is not high or the risk is not there, which does not seem to be so much the position that was taken by the Southwood Committee, for instance, which recommended that it was okay to continue eating bovine brain, which is obviously a different line of position being taken, and I think in some way the agriculture and the health people must come together to decide what the acceptability would be for risk to be taken and a risk assessment must be done.

Chairman

281. Have you anything to add to that, Dr Narang?

(*Dr Narang*) Yes. What I have to say is this, on the issue of brain. Stemp in 1959 published his research on brain studies whereby he inoculated lambs and after every two months he killed the lambs and passed the material on to other fresh lambs and in six successive passes he showed that after two months of test study the agent had decayed and he was not able to reproduce the disease again in the seventh pass. So if one is using cow brains in the food chain under six months, I think that is correct.

(*Mr Meldrum*) If I can be brief on the issue of species affected, the only species we know that have contracted BSE by mouth naturally based on examination of the brain and typing of the agent found are cats and kudu. Of course, there have been other species, as Dr Dealler has said, under zoo conditions which have succumbed to a transmissible encephalopathy or an encephalopathy but we do not know if it is BSE. I was trying to be exact in my answer. So far as pigs are concerned, we have had no naturally occurring cases of an encephalopathy in pigs. Experimentally they have succumbed after

[**Chairman** *Cont*]
intracerebral inoculation but given BSE brain material by mouth they have not so far succumbed and that experiment is incomplete.

(*Sir Kenneth Calman*) Simply to reassure Dr Dealler, in terms of human health issues the two departments, Agriculture and Health, work very closely together. Indeed, that is part of my responsibility, and to answer the question, we have no evidence with any other edible species that there is any BSE-like epidemic or any risk to human health.

Rev. Smyth

282. I have been thinking today of that old saying, "Lies, damned lies and statistics". There does seem to be some problem about getting the facts and the data. I wonder if our witnesses could tell us what estimate do you actually make of the number, of the current extent of BSE amongst British cattle?

(*Dr Dealler*) There are two forms. One is that the disease is vertically transmitted, and we do not know whether it is or not, in which case the numbers I would have expected to be dropping fairly rapidly, to be down past 8 per cent. of the dairy population in the United Kingdom. If it is not vertically transmitted it would not be dropping as rapidly as would be apparent by a vertical transmission method; it would still be down to about the same population. Do not forget, at the peak about 25 per cent. of the dairy cattle born in 1988 would have been expected to die of BSE had they not been eaten first or for some other reason.

283. I asked the question because there was a difference on under-reporting and it appears you have a different opinion as to the extent of infected cattle that, say, MAFF have been reporting, yet when you reported the Professor from Leeds, you said he did the statistics upon data provided by MAFF?

(*Dr Dealler*) I am really not at all sure how MAFF can justify the statistics that they have given. Statistically it is very difficult, unless you actually look at them, to decide why a very strange change has taken place. It is not due to fewer cases. That is not the reason it has happened. It is due to a change in the age distribution in cattle born after the feed ban and so it is not to do with just a change in numbers, and without a reason for this it is very difficult to be able to be sure what is going on. I have discussed this with a number of epidemiologists and they really do not know, and the idea put forward by Professor Kent of its possibly being due to under-reporting fits so well with the other data that I think at the moment with being told that under-reporting was taking place, I must assume that to be so until further data shows it was not.

284. We understand that there are cases this week in the press of people who sold cattle having denied that they were from an infected herd. What I would ask you is what further steps do you believe could be taken to find out more accurate data?

(*Dr Dealler*) Certainly one of the experiments that needs to be undertaken, and I am sure should have been done earlier, is to look into the actual number of infected cattle that are reaching the slaughterhouse. Dr Narang provided a test which I have to say has been repeated at a major research station in Edinburgh and by the Ministry of Agriculture's own people at the Central Veterinary Laboratory, in which you get some idea. Maybe it would give an under-estimate itself, this test, but it would give some idea, and the World in Action group who actually sponsored it to find out the number of cattle that were infected found that it was around 30 per cent. of the dairy cattle that were being slaughtered. Not a very large study was actually done in order to see, but that could be repeated. I think we ought to know how the numbers are dropping, although I must admit it is very difficult to find the same kind of abattoir group now as we would have done then, but we can calculate the number of infected cattle we are eating. That is not difficult. You can do that with MAFF's own data but in order to find the true number it would have to be done by experiment.

(*Dr Narang*) Can I make a very quick remark, that 60 per cent. of the cattle that are being confirmed with BSE were born after the feed ban and, therefore, you can make your judgment or calculation.

(*Mr Meldrum*) I admit I did indeed make a calculation on that and I would say it is feed-borne and I have clear evidence of that. We have said before that there is evidence of feed-borne transmission occurring on occasion since the feed ban. That is why Professor Pattison's committee recommended that meat and bone meal should be totally withdrawn from rations used on farms to avoid the possibility of cross-contamination. We have clear evidence on occasion of cross-contamination in feed and that would explain the born after the ban cases.

Mr Powell

285. Dr Dealler and Dr Narang, how would you comment on the most recent advice by SEAC to the Government on the implications for public health of the so-called new strain of CJD? Do you agree with SEAC that the most likely explanation for the new strain is BSE infection? What would be your calculation of the worst-case option in terms of the number of future sufferers from CJD?

(*Dr Narang*) To answer the last question first, I would not make any assumption. I am a scientist and I would not say how many people would die of the disease because I would not like to scare anybody. However, I have said that there is a blessing in disguise, and this is the base of it: that if people have been infected with the scrapie strain, which has a very long incubation period, as we know, those people would be better protected from the BSE strain because one strain of spongiform disease will block another. This has been well established in scientific literature, that we have two types of CJD, one we call trembly and one we call scrapie. If you inoculate animals first with one and then with the second after a few weeks the other does not take hold, and we have shown that by this method. So to me, to calculate based on assumptions, I do not like to give this figure and I think people should be assured and not panic that they are going to die tomorrow because they are going to develop the disease. Coming back to the strain, in 1990 I was doing these experiments. By simply looking at the pathology in the brain you can see it is a different strain. You cannot see what that means actually and this can only be done by

[**Mr Powell** *Cont*]

inoculating animals. Like when you have influenza coming from Australia, you do not look at many people. You go to the laboratory and do a simple experiment. And these experiments were under way—I was doing them in 1989-1990—to pin it down whether the strain comes from BSE or scrapie or CJD or none of those, and that would have been shown somewhere round about 1991-92 had these experiments had been allowed to continue.

(*Dr Dealler*) I would say that the data I gave here, which has been calculated as well as I could do it, shows that the worst-case scenario is millions but that would be a worrying thing. I really do not feel that that would be helpful at the moment, also that the 38 per cent. of the now cases of CJD carried a particular gene which may make them more likely to go down with the disease. So all these would relatively decrease the numbers. I would say also that we have been exporting quite large numbers of infected cattle to Europe and figures can be quite easily produced from the Ministry of Agriculture's own data in terms of the number of infected cattle exported, and I think I almost have a greater worry for the risk that has been taken in France.

(*Professor Pattison*) I wanted to say that Dr Narang said that we should strain-type these cases. That has been done, of course. He then went on to say that it could have been done in 1990-92. Since the first case started symptoms in 1994, I cannot actually see how that could have been done.

(*Dr Narang*) Let me make a point. I believed in my report in 1990 to the Agriculture Select Committee I was seeing a particular pathology at that time. Your people have been working retrospectively, going backwards to find out whether such things have happened, and I have consistently thought for a very long time now that at one time we used to get patients coming when the leading symptom was Alzheimer's but now they are coming with Shakes and Shaky ataxia, which is the trembly type, and these are the differences clinically. As well and I can say, there are other cases, and probably you will hear in due course that they are coming.

Mr Leigh

286. Dr Dealler, would you eat British beef?

(*Dr Dealler*) No, I stopped in 1988, but for the rest of the population if they are continuing to eat beef, my statistics show there will be no advantage in their stopping.

287. You stopped eating British beef in 1988; your statistics, which you have quoted endlessly this morning, prove that there is no advantage for them stopping?

(*Dr Dealler*) That is assuming it is a cumulative illness, which it may not be.

Mr Bayley

288. If your fears are right, people will already have eaten too much, enough to have been infected?

(*Dr Dealler*) Because of the peak in the epidemic of BSE—I do agree, I think this probably is dropping, the number of cases. It is possible to calculate the added risk for continuing to eat anything particular, like beef, or stopping. The advantage for stopping is really very small at this moment in time; whereas if we had stopped eating particular tissues earlier on in the epidemic of the disease it was still worthwhile. It would not be worthwhile now anyway. Although infectivity has been found in the meat of goats, mink and of hamsters with spongiform encephalopathies, the amount that is likely to be present in meat is likely to be very low. Although we do eat very large amounts of meat, and would be from infected animals, the actual risk that represents must be very low.

Mr Leigh

289. As a beef eater myself since 1988, what are my chances of having caught this disease?

(*Dr Dealler*) We are going through another great big chessboard of statistics, which I ought to give you separately.

290. You are appearing before a Select Committee, this is an important occasion and the public could be alarmed. You have said it does not make any difference now whether I stop eating it or not, but you cannot in any way answer me?

(*Dr Dealler*) Yes, it is somewhere between zero and 100 per cent. That gets you nowhere, and that is why I say you need a chessboard of statistics.

291. You cannot give me any idea what I would save by stopping now?

(*Dr Dealler*) You will save nothing.

Mr Bayley

292. Would your advice be the same for a mother weaning her child on to food for the first time?

(*Dr Dealler*) Again, I have to go back to my statistics. I know statistics are perhaps not desired here, but by looking at the amount of infectivity present in specific tissues in animals—we do not know how much infectivity there is in the tissues of a cow—in animals we have an idea of the amount of infectivity present in any particular meal fed to a child. As such, you can show there is a risk—a risk is involved. There is a statistical advantage for not feeding children who have not been fed beef before. The problem is; how big is this advantage, whether this advantage is worthwhile, and whether it is ethically acceptable. This is why I was going back to the problem, as there did not seem to be ethical advisers on SEAC to decide what would be an ethical level of risk to be taken. This is why I say it is a problem and why it needs to be between the Ministry of Agriculture and the Department of Health.

(*Dr Narang*) May I make a point which is very, very important. I would like to offer an example, an experiment and people can make their own judgment. If you make a mouse pregnant first, and then inoculate its body, you will find the litter will develop the disease as the mother will develop the disease. If you inoculate the animal first and then, three weeks later, make the mouse pregnant the litter may or may not get the disease. Cows have been eating this meat all the time, contaminated, but they are not contaminating themselves, they have been contaminating the foetus at the same time. I leave people to make their judgment, whether they want to eat it or not.

Mr Ieuan Wyn Jones

293. The Government's present advice to the public is made on the assumption that the Specified Bovine Offals Ban introduced in 1989 has been largely efficacious. In your evidence, you seem to cast doubt on the efficacy of the ban by claiming that liver and other tissues which were not covered may carry the disease. Where is the evidence which supports this claim?

(*Dr Dealler*) At the beginning in 1987 we actually knew nothing about tissues of the cow. All we could do was look at the tissues of other species, and look at their tissues when they were infected with a spongiform encephalopathy to decide which tissues were infected and which ones were not and from that we could make an assumption in cattle. This is very reasonable—I am not arguing with this. What you find is, in every other species which has been tested, the liver is infected. Lots of different tissues have not been tested at all. You find three out of five of the meat of animals were infective; of peripheral nerves, every one was found to be infective. It then comes down to, if those are not included in the Specified Bovine Offal Ban, is the amount of infectivity that will be present in human food—or we should assume to be in human food—enough to be a risk. That was the work I carried out in 1993.

294. Are you saying there is scientific evidence that those tissues could be affected, or are you saying you are basing that on the assumption they might be?

(*Dr Dealler*) They might be. We must look at other species. At the moment we have not got an adequate test in cattle. We will have one, of course, but it is not there yet.

295. Currently would I be right in saying there is no scientific evidence to support that assumption?

(*Dr Dealler*) That needs scientific evidence.

296. In cattle?

(*Dr Dealler*) Yes.

297. But, are you saying there is scientific evidence which indicates that tissues you have outlined do have the disease?

(*Dr Dealler*) There is no proof, there is evidence.

(*Dr Narang*) When politics was not involved, it has been shown that scrapie sheep muscle contained the infective agent. Secondly, to do this experiment—because you covered a great deal in the last hearing about what sort of tests should be carried out to demonstrate whether the red meat carries the agent or not, that is the big issue here—those experiments should have been done using mink. Mink should have been fed with this red meat from the BSE cattle and you would have seen in under three years' time whether the infective agent was present in the meat or not. That was the vital experiment, and I do not know why it has not been done.

Mr Bayley

298. Sir Bernard Tomlinson is a very eminent neuropathologist who I spoke to three weeks ago, and he felt there was a case for extending the selected offals ban to all offal, including liver, and he suggested if Select Committees wanted advice on this matter they should ask you to come and give that evidence, Dr Dealler. Do you share Sir Bernard's view, if only as a precautionary measure, it would be sensible for the Government to extend the selected offals ban and, if so, to what other parts of cattle?

(*Dr Dealler*) Similarly, all you can do is calculate risk statistically. This was done by the Advisory Committee on Dangerous Pathogens, who looked into which tissues of a cow they would assume to be infective, because there was inadequate evidence to show that they were safe, and they included: liver, peripheral nerves, the lung, and some degree of blood. Unfortunately, if you take action like that you virtually rule out all cattle from being eaten completely. I personally would have said I was very surprised that liver was not included in the original Specified Bovine Offal Ban, and lung. Lung is also found in mice to be highly infective. It is not just me giving evidence out of the blue, this is a large proportion of the scientific population. When I told Professor Minson this he absolutely jumped backwards saying, "I can't believe we are still eating it", and this was a Professor of Virology in Cambridge.

(*Professor Pattison*) It came up, of course, when we met. I think the problem is, as Dr Dealler has just said, if you take a single piece of data from a single experiment from clinically sick animals of any species with any TSE and apply that across the board you stop eating everything. There is nowhere to draw the line if you are going to take those isolated pieces of data and apply them generally. We have thought very carefully indeed about liver. As I said the last time, it is quite true, in terminally sick animals, in sheep scrapie, goat scrapie, transmissible link encephalopathy you do find it in peripheral tissues when the animals are almost dead of the disease. One thing we always gloss over is, before the offal ban, there was a ban on clinically sick animals going into the food chain. That was a very, very important item of legislation, and it would actually have reduced the risk very considerably. So, if you look at the evidence for sub-clinical animals—those that have not yet developed the disease—the liver does not spring out (and neither does any other tissue, apart from lymph node spleen and central nervous system) as important sites of replication and infectivity with these agents. That is why we came to the conclusion we did.

Mr Ieuan Wyn Jones

299. In a sense, Professor Pattison has answered the second part of the question. I just need confirmation that there is a difference in the age of the animal as to the risk which might be involved in certain tissues. Would that be right?

(*Professor Pattison*) Yes, because the older the animal gets the more likely it is to develop the clinical disease if it were an infected animal. Just on basic principles we would assume that towards the end of the incubation period, just before they have symptoms, there has been a significant build-up of the agent in the tissues that we know contain it.

300. Do you think there is any need for you to reconsider the Specified Bovine Offal ban in the light of the evidence which has been given to the Committee this morning?

[Mr Ieuan Wyn Jones *Cont*]

(*Professor Pattison*) I do not, but let me hasten to add that we tend to reconsider everything continuously. We do not feel that the state of knowledge and the accumulation of experimental data is such that we can close off any consideration. But I have not heard anything this morning that makes me immediately want to go out and reconsider it.

(*Dr Narang*) In animal experiments it has been shown that the glands, lymph nodes, spleen and liver contain the infective agent, and this comes up very, very early. This never increases thereafter in those tissues, except in the brain. Even if an animal is clinically ill, these organs will contain the same amount in the beginning as at the end.

(*Dr Dealler*) I would agree with Dr Narang, that is exactly what I have found. In lymph node tissues and peripherals like this, infectivity arises relatively early and stays at a sort of static level. I would say that by slaughtering cattle at the end of their milking lives would have had a far greater effect than taking out livers or anything else. So I think there is no need for a change in the specified bovine offals.

Mr Pickthall

301. I am trying to get my head round the arguments about whether or not BSE is cumulative. If your argument, which seems to me paralysingly logical, is followed up, then it would seem to be very important to the beef industry to prove that the disease was cumulative.

(*Dr Dealler*) That is right.

302. What research is being done? Do you know?

(*Dr Dealler*) As far as I know, none.

303. Would you confirm that, Sir Kenneth, or Mr Meldrum? Is any research at all being done into whether the disease is cumulative or not?

(*Professor Bourne*) If I could answer that? The short answer is no, but that is not a complete answer. For many years it has been recognised that in scrapie and in TSE you have no immune response, and it was thought the immune system was not involved in the disease pathogenesis. We now know it is. The assumption is that this hypothesis—the cumulation of infectious agent—would occur presumably within an immune cell. Immune cells do not last forever; they are destroyed and they are replaced, but there is work going on into studying the pathogenesis of the disease through the immune cells, so indirectly one is working towards providing the answers to the question you are asking now. I would not suggest we are anywhere near answering the question but certainly work is in place directed towards it.

304. What would, Dr Dealler, be the implications for your argument? What Professor Pattison has said earlier, and I think you agreed, was that the smaller the dose the longer the incubation period. You were talking about advice to citizens. Now, the smaller the dose the longer the incubation period does mean the larger the dose the quicker you get it?

(*Dr Dealler*) That is right.

305. So if you want to postpone the minute risk of getting this disease you would need rather less?

(*Dr Dealler*) In my data it shows that we were past the point to make much difference in this. By stopping at the moment eating bovine products we might halve the amount of total infectivity that we have eaten, and halving does not make that much odds. Making a ten-fold difference is the sort of effect you can see making a big difference in the incubation period. I would say that the incubation period of this disease is likely to be so long that if we do see an epidemic of BSE in humans we will see it over the next 50 years—it will not be something that appears fairly shortly. This was published in work for the Federation for Infectious Societies last year. So what we have not done, of course, is look into methods of treatment. What we would love to do is lengthen any kind of incubation period, just so we can do the experiments. You do realise, of course, that we have not been doing the experiments, assuming that everything was going to be OK, and as far as I can see I am the only person that has done any research into potential methods of treatment.

Alice Mahon

306. Just to move on in your view of the likely length of the incubation period. Can you tell us what your view is of the likely incubation period of the new strain of CJD? Is there any means of knowing when the ten victims so far identified were infected? Might large numbers of people have been infected before the first case of BSE was identified in 1986?

(*Dr Narang*) You cannot really know when people were infected because they were infected at some time. What we know from animal experiments is that the incubation period for the BSE strain is much, much shorter. I would predict that in humans it will be around about 5 to 10 years, not 20 years as previously thought. To say when they were infected—the important thing is to tie this strain out and relate it to whether it has come from BSE—like we have done in cats—and then we will know whether it ties up with that issue or not.

Mr Bayley

307. In view of the fact, Dr Dealler, that you said that infected material injected might be 10,000 times more likely to infect the recipient than, say, material introduced orally, do you think there is any significant risk that animal serum used in inoculations might be infected by spongiform encephalopathy, or might in the past have been infected?

(*Dr Dealler*) This would be something that took place before 1989 and the worry is that bovine serum, which was used as a carrier for various things for inoculations, might turn out to be infective and transfer the disease. As far as I know, that was completely stopped at that time, and the pharmaceutical industry that was involved in this made sure of that. All I would say is I worry that we may not just have BSE in our own country but we have exported calves infected with BSE and bovine serum is often taken from calves. The worry is that maybe some of those will actually carry the disease, and we will find this in Europe as well.

[**Mr Bayley** *Cont*]

308. In 1989 the use of bovine serum for vaccinations for human beings was banned. On what date was the use of bovine serum banned for animals—for veterinary medicines?

(*Dr Dealler*) I do not know the answer.

(*Mr Meldrum*) It was roughly the same time for assessment for both human and veterinary medicines but I cannot give you the exact date.

(*Sir Kenneth Calman*) This was an issue raised when we met before, and I can just confirm again that vaccines used in the United Kingdom contain no bovine material, other than some gelatine obtained from sources outside the United Kingdom. The manufacture of vaccines involves some bovine materials, but all materials come from BSE-free sources outside the United Kingdom. There is quite a long statement on this subject: and the details, pre-1989, are confirmed within it. The issue pre-1989 is that some may have used the bovine serum albumen, but that is classified as a source which actually contains no risk according to the CPMP. I can provide all of that, which I think will answer the question pre and post-1989.

309. A fortnight ago I spoke at a meeting with a number of farmers from north Yorkshire and one said to me that, observing his own herd and thinking back to the time when infective feed was being fed to the cattle, he observed that those cattle in his herd which had subsequently developed BSE had all been young cattle at the time that infective feed was being fed, and none of the older cattle succumbed to BSE. He suggested that perhaps the route of transmission was not the feed at all but the veterinary medicines used to treat young calves against TB, Foot and Mouth and the various other things calves are treated for. I wonder whether that as a hypothesis is one which has been examined by MAFF scientists; if, so, is it one that should be treated seriously, and is work carrying on in that field at the moment?

(*Mr Meldrum*) Yes, that was looked at in very great detail soon after the very first cases of BSE had been identified. A detailed study was carried out by John Wilesmith. He published those results thereafter and could find no evidence of any other cause, apart from feed. He did look specifically at medicines used in cattle. He looked at a variety of possible causes and concluded at that time that there was no correlation with the use of any vaccine. In passing, we do not use foot and mouth vaccines in the United Kingdom and never have done—I hope.

310. Subsequently you banned the use of bovine products in vaccines for cattle, so something must have changed to make you feel that the results of that research perhaps were not as conclusive as you felt at the time—otherwise you would not have banned the use of bovine products in veterinary vaccines at a later date?

(*Mr Meldrum*) An interesting question you ask, but I would offer a different answer. The answer I would give you is that we have all along taken an ultra precautionary route as far as BSE is concerned, contrary to what Dr Dealler may suggest. We have done it all along, right from the word go. We have assumed that BSE might affect man and, on that basis, we have built up our controls. We also have to assume the possibility exists that vaccines might be contaminated with the BSE agent; and to avoid the possibility of exposure through that route, guidelines were drawn up through our Veterinary Medicines Directorate to ensure that such bovine materials included in vaccines were sourced outside the United Kingdom. It is purely and simply an ultra precautionary measure.

311. I understand that the national herd is now protected because of the measures you have undertaken. Nevertheless, if there is a risk of infection and transmission of the disease through vaccines it would be important to know it because it would help you to understand the epidemiology within the herd. Do you think it would make sense to do some more research now to re-visit the hypothesis that in the past animal vaccines might have been a carrier, a transmitter, of the disease?

(*Mr Meldrum*) We cannot reinterpret the results from the first investigation because I do believe they were sound, and that particular option was looked at and listened to very carefully by the researchers. Since that time, yes, we have looked at the possibility that other factors, other sources, could in fact expose cattle to the agent of BSE. We have concluded, with the exception of the possibility of maternal transmission, that feed is the only source, but we do keep those possibilities under very careful review. The majority of the material that would have been removed from animal vaccines in 1989, or thereabouts, was derived once again from very young animals, and blood products; and there is a very low risk from such products, even if there were to be an extensive encephalopathy of animal at the time of collection. These are calves in the main that are either newborn, or material is obtained very soon thereafter and sources outside the United Kingdom. Certainly all these issues are kept under constant scrutiny. As Professor Pattison has said, from the point of view of SEAC we do not close off any particular option and we consider any possibility as time goes by, and reconsider them and reconsider them.

Mr Alexander

312. Dr Dealler, you have said that in 1988 you gave up eating meat. Did you become vegetarian then, or was it just beef that you gave up?

(*Dr Dealler*) It was just beef.

313. At the end of the day our public out there are going to read this evidence and they are still going to want to know from you two, in particular, this one question: do you think that beef and beef related products are safe to eat. If you do not, what is the brief, scientific evidence, on which you hold that view?

(*Dr Dealler*) I consider beef products to be safe to eat in the UK for adults. Unfortunately, I cannot say statistically that it is safe for children. But the biggest thing that will make any difference now would be the slaughter of cattle at the end of their working lives, rather than them being used for human food. That was the thing that made the big difference.

(*Dr Narang*) If beef is coming from an infected animal, whether it is asymptomatic or symptomatic, I personally would not eat that meat, because to me I am taking no risk. It is entirely up to the people whether they want to eat it or not. As I said, I have a

[**Mr Alexander** *Cont*]
test, other people believe in my test and if this test would be done it would create confidence in the consumer and in the farming industry.

Chairman: Can I say how grateful I am to my colleagues on the Committees and also our witnesses for responding to my cajoling this morning. We are actually finishing dead on time. I am most grateful to our witnesses for responding so fully to the questions that have been put to them, and for helping us with our evidence on this very, very important matter. Thank you all very much indeed for coming before us. We shall have another session tomorrow when we shall again ask further questions concerning BSE and CJD. Thank you all very much indeed.

Extract from memorandum by MAFF, (Letter to the Clerk of the Health Committee from Mr R Bradley, Central Veterinary Laboratory) (T10/BSE1A)

EVIDENCE TO THE SELECT COMMITTEE, APRIL 1996—DR NARANG

I refer to the House of Commons Agriculture and Health Select Committees attended by the CMO, CVO, Professor Pattison, Professor Bourne, Dr Harash Narang and Dr S Dealler and the transcript of the evidence session on 17 April 1996 and specifically pages 20 and 21, paragraphs 227 and 229.

Q 227 I can support the statements made by Mr Meldrum in regard to the tests done on 10 brain samples (five from cattle with BSE and five controls) by Dr Narang using his "Touch Method". However, initially Dr Narang reported only two of the five brains from cattle clinically affected with BSE as positive. He later changed one of his negative results to positive, but outside the time limit agreed for the study. Thus, as Mr Meldrum stated his best result was a 60 per cent accuracy in detecting clinically and pathologically positive cases of BSE using his "Touch Method". This contrasted with 100 per cent accuracy in this study using the existing SAF test (please see below).

Q 229 I have no knowledge that Dr Narang was obstructed in attempts to obtain heads from abattoirs by anyone. Indeed, the statements made by Mr Meldrum in Para 227 make it clear that there was no obstruction since a joint study between MAFF and Dr Narang using heads from abattoirs was conducted to evaluate his "Touch Method".

I have no direct recollection of the alleged incident or when it was supposed to have taken place. I have had several meetings with Dr Narang over the years and held scientific discussions with him on spongiform encephalopathies and methods of diagnosis. However, what I can say is that I would not have said that his test (The "Touch Method" I presume he means) is very sensitive because it is not (see Para 227). It is not as sensitive as the existing SAF test which, like the "Touch Method"; uses electron microscopy.

I certainly would not have said that most of the 100 cows tested would be positive for BSE. I believe there is a misunderstanding here. Most animals slaughtered for beef are less than 2½ years old and BSE is rare in this age class and even more rare if derived from beef suckler herds. Even if the animals targeted are adult dairy cows only a small proportion would be infected and few of those are likely to show evidence of disease by a test such as Dr Narang's "Touch Method", the SAF test, immunoblotting or histology. The only test for infectivity is a bioassay and this is not practical to use in the abattoir situation.

The study proposed by Dr Narang could not have achieved the objectives of the Ministry since the test he proposed to use (The "Touch Method") was insufficiently sensitive to detect all clinically positive confirmed cases of BSE in the controlled study. It would be even less likely to detect cases during the incubation period. Cattle clinically suspected to have BSE are not, of course killed in abattoirs but are compulsorily slaughtered and destroyed so that no part of them can enter any food or feed chain.

I believe Dr Narang misunderstands both the epidemiology of BSE and the scientific basis of the control measures used to protect public (and animal) health from any risks there may be from infected tissues derived from clinically healthy animals. Since there are no practical tests to detect such animals it is assumed that all cattle may be infected, though probably only a small proportion actually are. Thus, we could be reasonably confident that some clinically healthy cattle entering an abattoir could be infected with the BSE agent but there is no practical way of identifying them. That was why the SBO ban was introduced. The SBO ban is an effective method for separating infected tissues from uninfected tissues. Meat derived from this process is, in my opinion, safe to consume, provided all SEAC recommendations are followed.

Whereas much research effort supported by Ministers, is being devoted to developing a test for identifying infected, live animals before clinical signs are evident, none has yet reached the stage where they could be effectively and practically used. Thus the SBO ban is an important control measure and removes potentially infected offals from all cattle whether or not they actually harbour infectivity.

29 April 1996

Letter to the Clerk of the Health Committee from Dr Stephen Dealler (T12/BSE4B)

I am returning the document that you sent for changes.

There were few changes that were required. One did alter the meaning in that I think that the person writing down the statements missed a "not" out from what was written. If this is not so then I made an error myself in the statement.

I was asked to submit further information to the committee that I felt was asked for.

I include a copy of the information suggesting that vertical transmission of BSE may have been taking place and a longer article going through it in explanation (as it is complicated). A copy has been sent to Professor Pattison, to the Department of Health and to Professor Almond for their opinion.

23 April 1996

Memorandum by Dr Stephen Dealler (T10/BSE4A)

INFORMATION CONCERNING THE MINUTES OF EVIDENCE TAKEN BEFORE THE AGRICULTURE AND HEALTH COMMITTEES. QUESTIONS 78-186

Mr Hogg said that information from Professor Pattison, Sir Kenneth Calman and Mr Meldrum was put to the EC and was not taken as full information.

This can hardly be surprising as MAFF have been repeatedly telling the EC information that was misleading (examples). Surely their credibility must be low. The advisers to the SVC at Brussels did in fact include a member from SEAC. Can it be surprising that, when SEAC was found to be wrong in its advice that the SVC no longer accepts our advice.

Dr Calman again says that there is no evidence that BSE has infected humans in these cases. This depends on the definition of "evidence".

Professor Pattison attempts to indicate the number of cases of BSE that might appear in humans. His information using the methionine homozygosity found in all the patients (and in 38 per cent of the population) was useful.

It is not made clear, however that attempts to indicate the number of people already infected can be carried out (and indeed will have been). He states that . . . . . the dose required for humans to be infected may be at least 100,000 infective units. Kimberlin's research showed this to be actually around 10 fold lower. His information is otherwise good.

The impression given by Dr Will was that all cases of potential CJD were currently being reported to him.

My own experience is that this is not true. Indeed in the article in the Lancet on the 6th April this is admitted by Dr Will in that a large percentage of the 10 cases now identified with possible BSE would not have even been classified as possible CJD. **Surely.**

Dr Kimberlin makes it plain that the apparent exposure by humans to scrapie may have been high and yet we did not become infected.

The problem with this is that sheep are eaten at a different point in their life expectancy than cattle. When carrying out calculations it would appear that humans would have been exposed to between 10,000 and 100,000 times the level of dose to BSE than to scrapie. The risk to scrapie should be discussed. One of the best evidences that scrapie is not a risk is that the sheep-produced vaccines against rabies do not seem to represent a risk and they have been used widely.

Mr Meldrum put the possibility that BSE was derived from cattle as "only a thought".

It should be made clear that this is now the main idea that is held in the research community and was made clear in a letter by Roger Eddy in the Veterinary Record earlier this year. My own letter giving similar data was not even replied to by the VR.

Calman giving an assurance that there was no risk from bovine serum used in inoculations before 1989. He had no right to do so.

Quantity found in blood. Blood transfusion risk.

Kimberlin told you that there was no evidence that TSEs were transferred in other animals apart from sheep.

Vertical transmission has been suggested in mink, and humans. The reason why there is little information about various species (for instance there is information suggesting no vertical transmission in mice) is that it is difficult, time consuming and expensive to carry out.

Pattison said that infectivity in mink and goats have been shown to spread out from the nervous system shortly before clinical symptoms appear.

This is not the whole story in that many animals have been tested and the appearance of infectivity around the body has been shown in many tissues before symptoms appear. Also, infectivity appears to be associated with the logarithm of the level and hence, when this is taken into account significant infectivity is seen in many tissues of the body early in the disease. Inadequate information is available.

Pattison told you that vets should be able to pull out cattle with early symptoms of BSE at a market.

The presence of vets at markets has varied dramatically during the period of the BSE epidemic. Initially there were few, then MAFF demanded many, then, in around 1992–94 the numbers decreased dramatically. For instance in York a local vet said that a vet was there for around 20 per cent of the time of the market sale. Also, much of the initial symptoms are almost personality changes in the cow. Only the farmer will recognise these. Cattle are often herded into a market and out again. They are bound to be frightened and, as this is one of the symptoms of BSE it is actually quite difficult to pick the normal ones from the BSE infected cattle.

Meldrum suggested that, as Mr Moss had removed cattle with BSE from the market they therefore did not go to slaughter for human food.

This is ridiculous. The point made by the World in Action programme was that cattle with symptoms were going to market. This would have been happening up and down the country. For a few days, for the purposes of television he picked some from a market. What about all the other markets, what about the days he wasn't there?

Mr Meldrum says that he has no evidence that animals showing evidence of BSE are not being identified antemortem in slaughterhouses.

My evidence was published in 1995 showing that this might be taking place in large numbers. I have had no reply from MAFF and I presume that he must have been shown this data. The evidence was based on MAFF's own statistics using standard statistical methods and followed my own experience of farmers telling me that symptomatic animals were being taken to market.

Mr Meldrum gives the impression that the separation of SBOs from the carcass was clean and that they had no evidence that infective material would remain in the tissue being used for human food.

It was made quite clear to Mr Meldrum in 1988, 1989, by the Parliamentary Committee in 1990 and at many other times by myself that this could not be accepted. The sheer difficulty in carrying out the procedures of removing the tissue would be bound to leave some behind eg cutting skull with a band saw, trying to cut the spinal column in such a way as to miss the spinal cord but cut the carcass in half. Many of the tissues not included in the list of SBOs have been shown to be infective in other species.

Dr Calman. His claim that there is no evidence that BSE had infected humans.

This was quite unacceptable. There is evidence but not proof. As proof requires inoculating into humans and then waiting for 15 years this cannot be carried out. Evidence can only be statistical, pathological and epidemiological at this time and those are exactly what are present. Using a strange definition of "evidence" does not get around this.

Mr Dorrell is glad that the 4.5 million pounds has been put towards BSE research.

This is simply taken from other research and this is the same procedure as took place with BSE research organised by MAFF. They simply took it from other research funding. For such an important disease this was difficult.

ANNEX A

VERTICAL TRANSMISSION OF BSE: EPIDEMIOLOGICAL EVIDENCE

This is a letter to explain more clearly the images on the attached papers. The reason why explanation is required is that the images are derived directly from the posters shown at the Gottingen Conference and from the Society for Veterinary Epidemiology that took place in Glasgow at the end of March. At the conferences I stood there and explained the poster.

The problem with the epidemiology of BSE is that the models that have been used concerning "nuggets" of infection causing the cases we see by the cattle eating them have had problems in explaining all of the findings.

Attempts have been made to try to produce better models that would fit the oddities but it can always be said that models fitting does not infer that the models are correct and in fact further better models may appear later as information appears.

CURRENT EPIDEMIC RISE OF BSE AND EPIDEMIOLOGICAL MODELS

This has risen in the way that would be expected of a disease that was infectious, was transmissible between animals relatively slowly, and in which the relatively slow rise (taking place over apparently 6–7 years) suggests that the point between infection and an animal becoming infective to another is long.

Initially it was assumed that the disease was derived from scrapie in sheep and that this was presented to cattle as part of their diet. The spread of the disease was considered to be due to the cattle remains being fed to further cattle and the number of cattle that became infected in this way from one infected cattle being slaughtered could be estimated.

Specific problems arose in this epidemiological model and other models were chosen to try to explain this:

The first was that the infection was present in "nuggets" that were present in the food. This model was required to explain why the age distribution of cattle infected in different years should be similar. If infection was not in "nuggets" then the age distribution of cattle dying of disease would have been expected to grow younger. The nugget hypothesis would suggest that an infected animal passed infection to a limited number of bags of cattle feed and that throughout the epidemic there was not so many cattle slaughtered while infective as to cause the number of infective cattle *per bag* to be multiplied. This model also required that infective nuggets were relatively uncommon and that a bovine would not be eating multiple numbers, this also suggests that infectivity was passed almost entirely when the animal was young.

The next problem was that a "nugget" model was simply not what one would expect from looking at the method of manufacture of the meat and bone meal. Also, the number of infected cattle involved in specific batches of feed might actually have multipled during the epidemic according to the mathematics. To get around this a different model was considered in that the infectivity was not so much in nuggets of the same size but rather of infectivity spread throughout a batch (eg a bag of meal). Within that batch there would be some parts much more infective than others and as such the distribution of infection would be such as to produce the same effect as a "nugget" and as such there would be a wide distribution of incubation periods but that this distribution would not be expected to change as the epidemic progressed. This would also depend on the number of infected cattle per batch not multiplying or at least the effect not being great enough to alter the age distribution.

Specific problems have arisen with these models and the possibility of both horizontal and vertical transmission must now be taken into account.

Here I put forward the epidemiological model of vertical transmission and the possibility that the cattle that we see with BSE are actually the offspring of infected mothers (model 2) and try to compare it with the previous model above (model 1).

The first thing I must ask you to do is to clear your mind that MAFF/Wilesmith's model is correct and try to look again at the data with open thoughts. This is by far the most difficult thing and I found it much more difficult than anything else.

Problems with Model 1

A. CALCULATED NUMBERS OF CASES OF BSE BORN AFTER THE BAN COMPARING THE TWO MODELS

The calculated number of infected cattle becoming infected in specific years and dying by the age of 10. It was expected that the number born in these years would drop dramatically (line W) but in fact they did not (line C). When calculated, the number expected if the disease was vertically transmitted was really very similar (lines A or B) and these were calculated as the number of offspring of the proportion of adult dairy cattle at the time of the feed ban compared with the following years. As these cattle were slaughtered at the end of their milking lives, fewer and fewer offspring would be of cattle born before the feed ban. Assumptions used were those used in the statistical analysis of Dealler and Kent (ie that all cattle with symptoms prior to 1992 were reported, and that the age distribution of disease remained steady).

Various explanations have been given for the slow drop in cases eg that pig meal still containing bovine material entered bovine food (with a nugget model this would have to be such a high proportion that it is impractical) eg that farmers retained feed from prior to the feed ban on the farm. Farmers have a monthly turnover of feed and in order to keep the level of infectivity at this rate this hypothesis would require that such an enormous percentage of meal was retained that the hypothesis could not be practical. Eg that the meal manufacturers were not carrying out the regulations and that infectivity continued in the feed. My own experience is that renderers were quite determined to keep infectivity out of the feed in order to keep their industry going eg that other parts of the cow, not thought of as infective were entering the bovine feed (tallow?). Again the level of infectivity in the tallow would have had to be very high indeed to produce the effect. A "nugget" model and these are all difficult to fit in with the number of BABs.

B. CATTLE DEVELOP BSE IN PORTUGAL AS OFFSPRING OF UK DAMS.

The number of cases of BSE in Portugal has in fact gone up to around 35 but, unlike Germany, some of the cases seen are the *offspring* of the cattle exported. Further investigation into this by UK journalists came out with the indication that at least 12 were in this category, and that one was the offspring of a Dutch cow and one was the offspring of the offspring of an imported dam. One BSE case has been reported in the offspring of a dam imported to an "organic farm" in the UK. Clearly more information is required.

C. THE DISTRIBUTION OF CASES OF BSE IN THE NATIONAL HERD.

What was expected with the "nugget" hypothesis was that one infected batch of feed might go to one herd but the next infected batch was just as likely to go to that herd again as to any other herd, ie in the cases of BSE would appear in the herds like the winners of the lottery, and cases would appear randomly. What seems to have happened however, is that once a herd has had one case, it is more likely to get another than a herd that has not already had a case, ie it is as if winning the lottery once made you more likely to win it again. One explanation of this is that once a farmer had seen one cow with BSE, he was more likely to recognise it again. However, this information that I get is the opposite; once a farmer has recognised the first case he knows what to look for in a second case and takes it to market ie if you've won the lottery once you would be expected to be less likely to win it again. Farmers talk about "infected herds". It is difficult to explain the appearance of cases in apparently previously unaffected herds (this is still happening). Model 2 will explain these cases but only in small herds would this be statistically valid.

Epidemiology seen fits precisely in what would be expected with vertical transmission. An infective batch would affect the mothers and hence the offspring in later years *would* be more likely to develop disease.

My own attempt at doing a sibling study was poor. Although there was an excess of siblings going down with BSE ($p = 0.05$) I am unhappy with the size of the study (30 affected herds) and neither model would expect an excess (except the vertical transmission model would expect an excess born early in the epidemic).

D. STEADY AGE DISTRIBUTION OF BSE IN CATTLE BORN IN DIFFERENT YEARS.

The surprising steadiness of the age distribution (until the feed ban, after which the cattle were apparently showing symptoms when younger). It was expected that the age distribution peak gradually drops as the amount of infectivity present in the feed increases. This would depend on the number of infective animals per batch reaching multiple levels, or that the disease came from scrapie originally.

The steady age distribution is exactly what would be expected with vertical transmission, however.

E. STEADY IN-HERD RATE OF BSE

As more lottery tickets are sold, there should be more winners, and if they are distributed randomly then the numbers per herd should rise in parallel (statistically) the national rate. What actually happened was that the rate stayed relatively steady. After "under-reporting" is taken into account the figure stays between 2 and 3 after 1089. In other words to have an epidemic what was required was that more herds would be infected. By 1988 around 90 per cent of the dairy cattle in the UK were in an infected herd, ie the annual national rate could rise little further. The rate represents a much higher rate seen in cattle that are infected but slaughtered before symptoms appear (around 20-25 per cent of the dairy cattle born in 1988). (This agrees with the data from the Statistical group at Reading University.) It is as if only 20-25 per cent of the cattle in a herd could be infected with BSE (no matter how much infection is given). This was agreed by Reading, who were involved in looking at the genetics of the cattle (they only looked at the PrP gene) for a reason why one cow did develop disease and another did not. They found that although there was genetic variation, it did not correlate with disease.

What the pattern fits well with is vertical transmission. It would be the mothers that were becoming infected because they were fed relatively large quantities of infective feed and a percentage of their offspring would develop disease. This would fit with the data concerning scrapie and vertical transmission of Dickinson. The reason that only a certain percentage "can" be apparently infected is that *all* the mothers are infected to produce the annual steady in-herd rate.

F. AGE DISTRIBUTION OF BSE IN CATTLE HAS APPARENTLY 2 PEAKS

Initially it was difficult to demonstrate the graph accurately because the confidence intervals for cattle dying at ages over 7 were becoming unacceptably wide. However, with further statistical analysis it was possible to show that 2 peaks existed. This is under argument in that the number of cattle in each age group in the national herd is poorly known. My data is derived from the former Milk Marketing Board and includes information on beef sucker dams and bulls. When these last two groups are removed the second peak still appears to rise

at the age of 10. It has been suggested that the effect is due to the younger group becoming infected when very young and open to infection more easily (the first few days of life?—this seems unlikely as much of the bovine meal used at that time does not contain MBM to maintain milk yields.

What fits well, however, is that the young group are the offspring of the older group.

Further research is required into statistical analyses and it is suggested that these are carried out by group independent of MAFF.

Other models have not been adequately investigated. For instance the possibility of environmental transmission. The association between BABs and the use of MBM for pigs has been shown but is inadequate to explain the high numbers. The possibility that the association is due to wealthier farms having pigs and cattle together (remembering that wealthier farms are the ones that used more of the MBM originally) should be taken into account.

Could I ask you to remember that the original model was a "best guess" and quite inadequate amounts of research has been carried out to be certain of it. As such, it should not be assumed to be true. Please look at the epidemiology of disease that is seen, assess what pattern this fits and try not to assume the original model to be true until proved false.

ANNEX B

Vertical Transmission of BSE: Epidemiological Evidence

SUMMARY

MAFF data for BSE cases in the UK was used to compare two models that may represent the epidemiology of the outbreak of BSE in the UK. The first, as suggested by MAFF epidemiologists[1], is that BSE was derived from scrapie and was transmitted to cattle by randomly distributed infective fragments in feed. Cattle that became infected were then fed to further cattle and an epidemic took place. The second model is similar to that above but suggests that the cattle that develop symptomatic disease, are mostly the offspring of cattle that had become infected through eating infected feed[2] (as in model 1). These dams would not show signs of disease until an older age than their offspring.

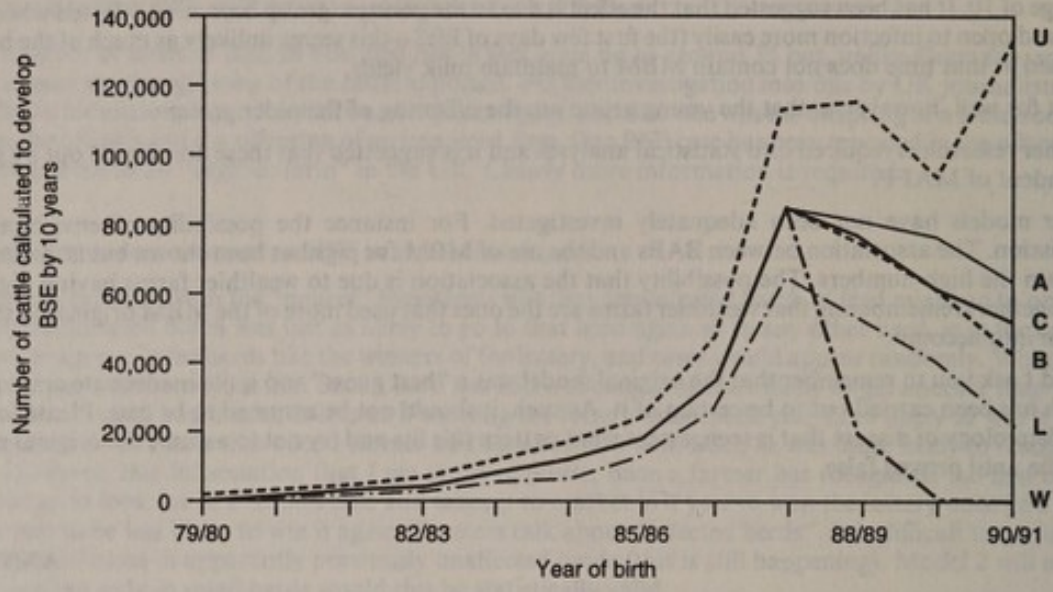
Table 1: Phenomena that separate the epidemiological models

	Phenomena	*Model 1*	*Model 2*
	Epidemic rise of disease.	✓	✓
	Epidemic rise stops when food ban introduced.	✓	✓
A	Slow down of BSE rate in cattle born after food ban.	×	✓
B	Offspring of asymptomatic UK cattle develop BSE in Portugal.	×	✓
C	New BSE case in a herd already affected is more likely than in a herd not previously affected.	×	✓
D	No significant change in age distribution of BSE cases during epidemic rise of disease.	×	✓
E	Steady in-herd rate of disease during epidemic rise.	×	✓
F	Apparent two peaks in age distribution of cases that would have appeared without bovine slaughter for human food.	×	✓
	Control trial shows little difference between BSE rate in the offspring of symptomatic and asymptomatic dams.	✓	✓

× = poorly compatible or requires model alteration ✓ = compatible.

The phenomenas are discussed on the various sheets of the poster and possible changes in model 1 that would be required to permit compatibility are considered. Arguments for and against model 2 are included.

A = Case numbers expected to take place if cattle infected individually (vertical transmission).
B = Case numbers expected to take place if cattle infected as a herd (vertical transmission).
C = Case numbers reported (derived from MAFF data).
U and L are confidence intervals for C.
W = Case numbers expected if feed ban was fully effected and no vertical transmission.



Specific models suggested for the BSE epidemic:

Model 1. Transmission of infection from the tissue of one cow to the feed of another through inadequate sterilisation practices.

Model 2. The bovines seen with clinical signs of BSE became infected vertically from generally asymptomatic dams, which themselves became infected as in Model 1.

(A) *Calculated case numbers versus reported numbers for models*

The number of cases reported fits remarkably well with the number of expected if whole herds become infected, vertical transmission takes place and the cases we see are those of the infected offspring.

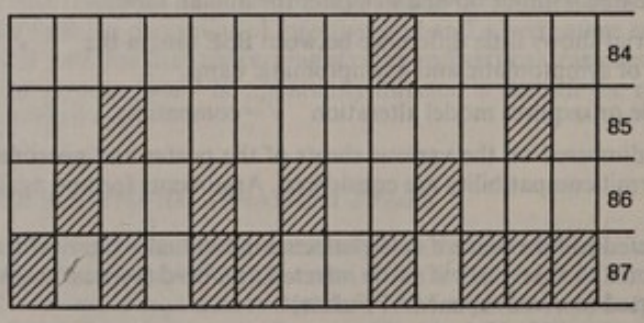
(B) *Cattle develop BSE in Portugal as offspring of UK dams.*

These cattle developed the disease although they were born in Portugal. This has been reported for 12 cattle (Portuguese Government statistics derived from Agricultural Ministry 1995). It appears that all but 3 were the offspring of UK dams that had been exported to Portugal but showed no signs of BSE. One was thought to be the offspring of a Dutch dam, another the offspring of a Portuguese dam, and another the offspring of the offspring of a UK dam*.

Statistically this cannot be due to the export of infected meal from UK to Portugal as there has been no reason why it should selectively infect the calves of UK derived dams.

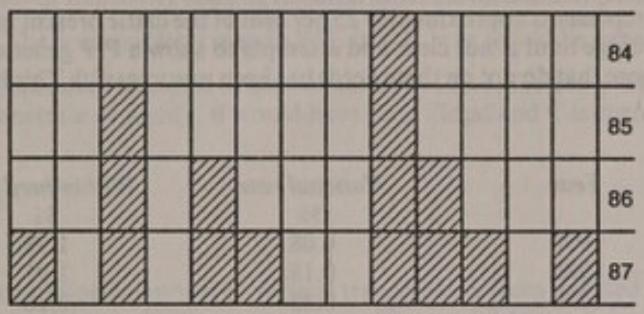
* Further data is required and has been difficult to obtain.

(C) *Distribution of farms in which a bovine becomes infected that is expected assuming random distribution of infection in feed.*



On this cartoon representation, a box indicates an individual farm and a hatched box is one in which a case of BSE has become infected in that year.

Distribution of farms in which a bovine becomes infected that is found in UK.



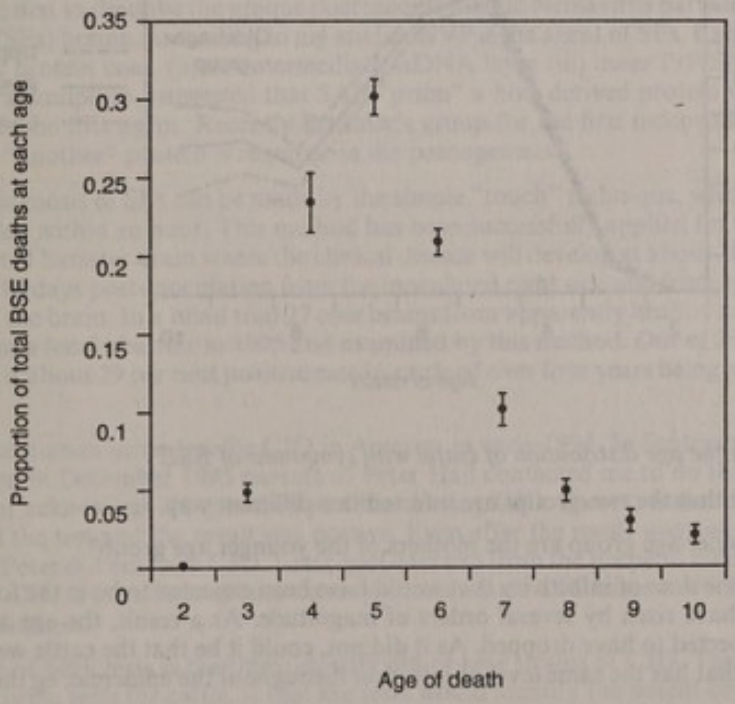
On this cartoon representation, a box indicates an individual farm and a hatched box is one in which a case of BSE has become infected in that year.

This would suggest that the reason why a bovine on a farm was selectively affected by BSE in a single year is also true for the remainder of the epidemic.

Various reasons for this phenomenon have been given eg a farm commonly is supplied by the same producer of bovine meal for many years and the epidemic may be closely related to a specific supplier (statistical demonstrations of this have not been produced).

As many of the dams in a herd would be expected to become infected concurrently and to give birth to a proportion of infected calves during the following several years the pattern seen is exactly what would be expected from model 2.

(D) *Steady age distribution of BSE in cattle born in different years.*



It appears that, as the epidemic has progressed there has been little change in the age distribution until the "feed ban" in 1988. Cattle born after this date were progressively younger, a factor that cannot be justified by the science of the disease and may suggest under-reporting.

(E) *Steady in-herd rate of BSE*

The in-herd rate is the number of cattle developing BSE per 100 of the bovine population in infected herds.

This would be expected to rise as the amount of infectivity in the feed of the cattle rises during the 1980s. The in-herd rate might be expected to rise in parallel to the national rate.

What is found, however, is that the in-herd rate rises quickly to a specific level and does not rise further. The rise in the national rate of BSE is due to an increase in the number of herds becoming infected.

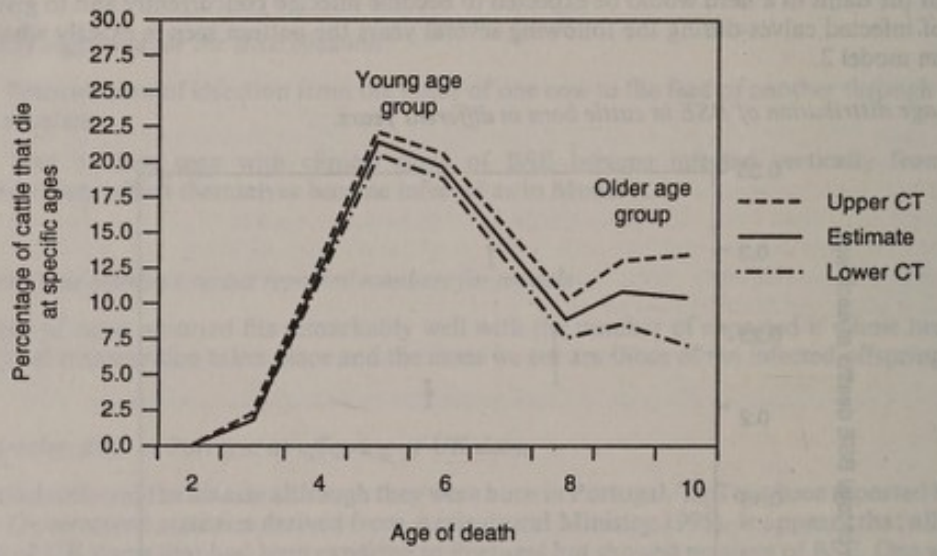
By 1988 approximately 85 per cent* of the dairy cattle in the UK were in an infected herd although many of the herds had cattle that were incubating BSE and showed no signs at this time.

Hence the national rate of BSE *could not* rise much past the level seen for cattle born in 1988.

The in-herd rate only represents approximately 25 per cent of the cattle present in the herd. The reason why this appears to be an average limit is not clear and attempts to show a PrP genetic difference between cattle that develop BSE and those that do not on these herds has been unsuccessful. This is not as would be expected in Model 1.

Year	*National rate* %	*Within-herd rate* %
1998	0.08	1.78
1989	0.18	1.91
1990	0.35	2.16
1991	0.63	2.44
1992	0.92	2.72
1993	0.86	2.43
1994	0.59	2.05

(F) Age distribution of cattle that would have died of BSE when slaughter for beef is taken into account



Apparent double peak in the age distribution of cattle with symptoms of BSE

It must be considered that the two groups are infected in a different way.

Could it be that the older age group are the mothers of the younger age group?

During the epidemic the dose of infectivity that would have been expected to be in the food of cows would have been expected to have risen by several orders of magnitude. As a result, the age at which BSE was diagnosed would be expected to have dropped. As it did not, could it be that the cattle we see with BSE are infected from a source that has the same level of infection throughout the epidemic, eg the mother.

Adequacy of epidemic models

Model 1 . . . Adequate but dependent on specific apparent requirements:

A* Large amounts of infectivity continued to be present in bovine food for several years after the feed ban. The ban would have made little difference initially.

B* UK bovine feed must have been exported to Portugal.

C* Either BSE was derived from an uncommon strain of scrapie that had no species barrier between sheep and cattle, or BSE was derived from a spontaneous TSE in cattle, or BSE derived from scrapie many years earlier than currently considered (eg 1970).

D* All the cattle that could become infected in a particular herd and year do so. There is an apparently inherent limit on the proportion of the herd that this represents.

E* Two groups of cattle are infected with BSE either in specifically different ways or with specifically different quantities of infectivity such as to cause separate incubation periods.

Model 2 . . . Adequate. The epidemiology seen in BSE in UK is as would be expected.

* There has been no independent scientific evidence that any of these factors is taking place. Indeed, A, D and E are difficult to demonstrate or justify. B would have been illegal and C is currently denied by Model 1 epidemiologists.

DISCUSSION

This shows that the epidemiological model of vertical transmission from infected cattle will explain many of the anomalies in the epidemiology of BSE currently seen. This model does not deny transfer in feed of BSE as the cause of the epidemic but suggests that many of the cattle seen with the disease are in fact the offspring of infected mothers.

Supplementary memorandum by Dr Harash Narang [T12/BSE5B]

ORIGIN AND IMPLICATIONS OF BOVINE SPONGIFORM ENCEPHALOPATHY

Based on experimental results a number of points were made in my memorandum which I submitted to the Agriculture Select Committee in June 1990. I am a scientist and I have a strong feeling that science should not mix with politics. True story of harsh treatment can be only told from written documents of Public Health Laboratory Service Board. I have provided facts without making predications on numbers of BSE or human cases.

In 1972 I was the first to describe the unique microscopic telltale Nemavirus particles (NVP) in spongiform encephalopathies (SEs) brains. According to my studies NVP is the agent of SEs. Each NVP consists of three layers: (i) an outer protein coat, (ii) an intermediate ssDNA layer (iii) inner PrP/SAF (Fig). Another view which is based on assumption, suggested that SAF/"prion" a host derived protein which forms the central core of the NVP, is the SEs agent. Recently Prusiner's group for the first independent have confirmed my work of 1992 that "another" protein is required in the pathogenesis.

The definitive diagnosis of SEs can be made by the simple "touch" technique, where both SAFs and NVP can be demonstrated within an hour. This method has been successfully applied for the diagnosis of human CJD Scrapie-infected hamster brain where the clinical disease will develop at about 100 days, NVP and SAF, however, are seen 10 days post-inoculation from the inoculated right side and from 18 days post-inoculation from both sides of the brain. In a blind trial 27 cow brains from apparently healthy cattle over four years old were collected from a local abattoir in 1995 and examined by this method. Out of 27 brains examined eight were postive. This is about 29 per cent positive rate in cattle of over four years being processed for the human food chain.

I deleveloped the human urine test for CJD in America in early 1994. In September 1995, I used this test in the UK and then in December 1995 parents of Peter Hall contacted me to do this test on their son. The neurologist did not acknowlege the possibility of him having CJD. Parents insisted that the test should be done. I performed the test and the result was postive. Even after the result was given to the neurologist, he was adament that Peter did not have CJD. Later post-mortem from the hospital, indeed confirmed that Peter died of new strain CJD. I have been able to confirm further three CJD cases and have urine specimen from six cases for examination.

The importance of both tests is that they identify old or new strains of CJD. The other advantage of the post-mortem and urine tests for cattle, is that the tests would identify the parent cows, their progeny could be traced to their farm and selectively culled. Random culling of cattle of certin age group may temporarily remove the pain, but will not control the spread of the disease. The advantage of these tests are that it will create public confidence, save the farming and associated industries, help to eradicate the disease saving tax payers millions. MAFF have denied me access to urine specimens from cows. From 1990 Public Health Laboratory Service Board, one way or other have stopped my work and in November 1994, with an excuse because of reduction of the Central Government funds, made me redundant. I have been working by begging and borrowing laboratory facilitied, while Ken Bell International, Newcastle, kindly provided funds to do this work.

All SEs are slowly developing infectious diseases, however, the titre reaches up to 10 million to 10,000 million dose/gm of brain tisue, while in other tissues of body it remains constant in the order of 1,000 to 100,000 dose/gm. The SE agent, even small amounts, replicate in a new host after a long incubation period, to produce a most devastating disease. The ability of the transmissible agent of SEs to remain remarkably stable over a wide range of physical and chemical conditions, is an interesting yet worrying. A dilution was

used to inoculate eight hamsters with scrapie-infected hamster brain which was left for 15 hours at 121°C. Out of eight hamsters inoculated, one developed scrapie. Similar studies have shown that the infectivity decrease by 10,000 to 100,000 dose/gm following treatment at 80° to 100°C for 30 minutes.

Over 20 Scrapie strain have been described which show a phenomenon analogous to "interference". The inoculation of mice, first with a long incubation strain of the scrapie agent followed by inoculation a few weeks later of a short incubation strain of the scrapie agent into the same mice. Inoculation first with the "long" strain inhibits replication of the "shorter" strain or vice versa, that is inoculation, first with the "short" strain inhibits replications of the "long" strain. Based on their response to the scrapie challenge, sheep are divided into "positive" (susceptible) and "negative" (resistant) lines. Comparative studies clearly demonstrate host PrP gene plays no role in the outcome of susceptibility with BSE agent. Almost every mammalian species can be infected by the SEs agent, whilst the host genetic make up may influence length of the incubation period which usually bears a relationship to average life expectancy of the species. All scientists agree on one point, all SEs, whether human or animal, are essentially the same disease. BSE is naturally transmitted with food from one species to another jumping species barriers and within a few years has infected domestic cats and 16 other exotic zoo animals. Since the link of BSE to CJD is still disputed, it is not wise to consider that the disease will die by itself and will have no effect on humans.

One question is often asked "why does it take 15 to 20 years to develop the disease". In case of BSE the incubation period wil be five to 10 years. The answer is—the replication of the virus is not slow—the clinical symtoms develop after a long incubation period. The disese process first starts when a virus enters host cell. It replicates, building itself, it requires the PrP protein molecules which are essential components of a normal cell wall. Like bricks are put together with sand and cement to form a wall, PrP molecules join together with the help of *Nemo corrupta* a protein to form the morphological structure SAF, whilst ssDNA wraps around SAF and, after acquiring a protein coat, form the NVP. This results in a gradual weakening of cell membranes. Vacuolation occurs and the clinical symptoms become evident after a long incubation period. In some hosts infected with a low dose of SE agent, the breaking point of cell membranes may not be achieved in the life span of the host and the disease remains subclinical.

Up until March 1995, over 17,000 BSE cases had been confirmed in the United Kingdom, in cattle born after the feed ban(BAB) was imposed. However this figure represents less than half of the 35,000 suspected cases reported by vets. No alternative diagnosis has been made for this large number of cattle, it is important that a second line of tests should be used to determine the nature of this mystery disorder. If half of the cattle did not have BSE, it is surely in everyone's interests to establish the cause of the symptoms which has resulted in an economic loss of over £8.5 million.

The evidence implicating a hereditary or congenital mechanism at present it is difficult to prove, if there is vertical transmission in BSE, as seen in scrapie sheep, although about 75 per cent of calves are destined for slaughter for beef at 18 months to two years whilst the remaining 25 per cent of animals to be used for breeding replacement, will they be free of the BSE agent? From the results available so far, the occurence of maternal or horizontal transmisssion appears to be the case therefore, BSE will become a permanent feature of British cattle.

BSE is the same as the scrapie agent in sheep. Most experts will say scrapie had been common in sheep in the UK since the 1750's—of course—we have been eating sheep with no apparent harm to our health. If it is all right to eat sheep, what harm could come from BSE? The most important feature the strain BSE agent has a short incubation period and high efficiency of primary transmission to other mammals, is a big cause for concern. Further, that transmission was equally effective in "postive" and "negative lines" of sheep by oral route, suggests that the BSE agent is more virulent than from the scrapie sheep.

A large section of the population has been exposed to the BSE—infected foodstuff for a long period. There are two questions, because of the long incubation period—we will not know for some time—how many humans are affected without clinical symptoms—how many human are subclinically affected—how many of these will develop the clinical disease remains unknown. Studying effect of gingival (gum) scarification on oral route of infection, demonstrated that 70 per cent of nonscarified compared with 100 per cent of scarificated mice developed the disease and with a significantly shorter incubation time. This suggests that the agent may enter the body via breaks in the skin. These cuts in the skin would serve as ideal portals for entry of any infectious agent in contact with the skin therefore food handlers, including housewives, with hand wounds such as cuts and cracked or may be more at risk. Single episodes of oral ingestion of the agent alone therefore may not transmit SEs, and it is likely that other factors play an important role.

April 1996

Further supplementary memorandum by Dr Harash Narang [T13/BSE5B]

Letter to Dr Narang from Dr R G Will, Consultant Neurologist, National Creutzfeldt-Jakob Disease Surveillance Unit, dated 16 February 1996

You may remember we were in discussion late last year regarding your urine test and I am glad to hear that you managed to get something set up without our help in Edinburgh.

I note from recent press reports that you have had some apparent success in using the urine test for diagnostic purposes in CJD. As I am sure you will recall from our previous discussions, it would seem to me that such a test is exceedingly unlikely to be helpful in CJD because of the wealth of evidence that suggests that conventional viruses are not involved in the pathogenesis of the TSEs. However clearly if there was any suggestion that there is a test that is of positive predictive value in the diagnosis of CJD this is something we must seriously consider here at the CJD Surveillance Unit. Accordingly, I do think it would be sensible for us to attempt here to replicate your test on samples of urine from patients with CJD.

I am sure you will agree that with any diagnostic test or development it is very important that results are replicated in other laboratories in order to achieve independent validation of any diagnostic procedure. What I am therefore writing to ask you is whether you would give us details of your procotol which I understand at least in part involves PCR of urine in order that we can test your interesting findings on an independent basis.

I realise there may be some sensitivities here in relation to confidentiality of your methodology and I can assure you that any information you give us will be treated entirely confidentially and would not be passed to any third party.

I would be very grateful for your help with this as although my own personal view is that the test is unlikely to be helpful for diagnostic purposes in CJD it is nonetheless very important for us to consider any putative diagnostic test for CJD. You may remember a few years ago you very kindly came up to Edinburgh to visit the CJD Unit and we had a very interesting discussion at that time. If you thought it would be helpful, I would be very happy to come down to Newcastle to discuss this and other issues with you directly and indeed it would be a great pleasure to have the chance to meet you again.

Copy of a letter to Dr R Will from Dr Harash Narang, dated 21 February 1996

Thank you for your fax dated 16 February 1996. In our previous telephone discussion you told me that because of danger of infection from urine specimens, you were having difficulty finding a suitable licensed laboratory in Edinburgh for this work. I am glad to know that you would be able to do this work now.

The urine test which I have developed for CJD does work. Recently, MAFF has granted a licence to Electrophoresis Ltd to develop a urine test for BSE. I do not know where you got the notion that the urine test for diagnostic purposes would be exceedingly unlikely to be helpful in CJD cases.

The nature of the agent remains controversial. Dr Prusiner believes it is a protein but too much of the evidence so far presented has been based on assumptions. I have demonstrated that the Nemavirus is uniquely typical of all spongiform encephalopathies. These findings have been independently confirmed by others.

I note, too, with interest, that Prusiner's group has recently concluded that "another" macromolecule—other than the "prion"—is required in the post-translational process, which seems to undo the PrP gene mutation hypothesis. In reality, there are two requirements for this process to occur. (i) To code for another protein, a DNA is required; (ii) since all hosts have the PrP gene, to code for another protein, DNA has to be non-host. In my previous studies it had been demonstrated that this macromolecule is coded by the ssDNA of the Nemavirus. There is nothing unconventional here. This is not a slow virus. Only the disease process is slow.

I well remember the interesting discussion we had in Edinburgh in 1989. At that time, I was given the impression that I would play an active part in CJD Surveillance. I would be testing brains from every suspect case of CJD using my "touch technique" which would complement the histological test. As you know from my published work, this test works both for biopsy and autopsy tissues. I confess that I was dismayed when you failed to contact me again. I am sure you had a great deal of correspondence on these lines with Dr J W Smith then Director of PHLS. Subsequently, I received minutes of a PHLS meeting dated 23 October 1990 stating: "Dr Smith clarified the PHLS position on slow virus work, namely, that PHLS did not wish specifically to engage in this area at present, because it was being adequately addressed by other excellent research groups".

In your report "Creuzfeldt-Jakob disease surveillance in the United Kingdom" you reported 260 cases. Of these 76 are definite, 62 probable, 48 possible, 48 others, 25 unclassified and one GSS for the period of 1985 to April 1990. Further classification of 139 notified cases for the period of May 1990 to 30 April 1992, you reported 43 definite cases, 11 probable, 20 possible, 63 other and two not classified. From these figures you can see that if my simple touch technique was used for confirmation, we would have better scientific

information and publish the accurate incidence of CJD. Since my test can be done on the same day of postmortem, correct death certificates could be issued. Further, this test would be useful for screening tissue donors so that tissues from CJD cases are not used for transplant.

I note from your many public statements that you do not believe that BSE can be passed to humans. You also say in your fax that the urine test would be "exceedingly unlikely" to be helpful in CJD cases. Given your clear beliefs in this regard, and your own department's lack of experience in dealing with the Nemavirus, I think the best way forward would be for you and I to meet, with a view to discussing terms and conditions for having my test validated. I'd strongly propose, for example, that the management and scrutineering be handled by an impartial third party, chosen by mutual agreement. As for the validation process itself, I'd suggest that I first demonstrate my test on 10 CJD positive samples and 10 Alzheimers (CJD negative) ones. In the second place, the impartial third party would select, say, 10 of those samples in whatever combination he chose, and then I'd test those, blind.

With reference to paragraph three of your fax, I am at a loss to know how you could guarantee that the details of my test would not be passed to any third party. But we can discuss that too. For the moment, allow me to explain that my test has been developed with private financial backing, and that the test itself is my intellectual property.

When we meet, I think it might prove helpful if detailed minutes be taken.

Copy of a letter to Dr R Will from Dr Harash Narang, dated 19 March 1996

Thank you for coming to see me here on Friday 13 March with Dr Martin Zeidler. We had a good discussion about the use of my urine test for CJD. It was very heartening that you acknowledged the importance of my test in both the clinical diagnosis and surveillance of CJD.

To recap, we agreed four points:

1. You would supply frozen urine specimens from confirmed cases of CJD to find out if frozen nature of the specimens does not interfere with the test result.
2. That you cannot provide funding for this research. Mr Ken Bell of Ken Bell International has agreed that he would provide immediate financial needs.
3. That you have no influence in securing lab facilities. Therefore, in order to arrange for lab space, I will write to individuals who may be able to help.
4. If everything goes well, we could then arrange a blind study by coding specimens from suspect CJD cases mixed with normal specimens.

Thereafter, as I suggested to you. I hope we could begin the work of testing urine from CJD suspected cases at intervals of one month (while patients are still alive) in order to understand more about the process of the disease and at the same time to progress the Science to a significant degree.

In the light of your membership of the Spongiform Encephalopathy Advisory Committee (SEAC), and your having realised the significance of my test, I am little surprised that you feel as you said, that you have no power to recommend, through SEAC, that financial help and laboratory space be provided for this work.

I look forward to hearing from you and receiving the frozen specimens soon. In turn, I shall advise you immediately I have secured laboratory facilities.

ANNEX A

Copy of a letter to Mr Douglas Hogg MP, Minister of Agriculture, Fisheries and Food from Dr Harash Narang, dated 9 December 1995

I heard your statement on Channel 4 News on 6 December 1995 to the effect that you would like to try the brain test which I have developed for BSE.

I have been working in the field of scrapie and CJD for over 25 years. In 1972, I described virus-like tubulofilamentous particles, now known as "Nemavirus." Since my first report, I have followed my own direction of work (see Agriculture Committees Report of Bovine Spongiform Encephalopathy (1990) and have published results of my findings in International Journals which have been confirmed by others. There are two tests:

Postmortem test:

In order to purify Nemavirus for further characterisation, in 1987, using infected brain tissue, I developed a simple touch technique for spongiform encephalopathies. This test provides a means of rapid diagnosis with very little tissue handling and risks of exposure. A piece of bovine brain tissue is removed after slaughter and examined by electron microscope. Using this test an operator can examine 25-30 cows a day.

Urine test:

The second test involves the purification and concentration of the virus from urine specimens. I have used this test to demonstrate Nemavirus in the urine specimens collected from a human case. The urine test could be used for cows while they are still on farms. This test will allow removal of affected animals from herds.

I trust that I may be of some service to you.

Copy of a letter to Mr Douglas Hogg, MP, Minister of Agriculture, Fisheries and Food from Dr Harash Narang, dated 27 December 1995

Thank you for the reply of my letter by Mr Marcus Nisbet, your Private Secretary dated 21 December 1995. You make a point that "as a scientist you will appreciate that all claims need to be rigorously tested and receive sound experimental support." I would ask you, since MAFF have spent millions on this research, I do not see another instance where such claims were tested before grants were awarded. With all the effort so far no real progress has been made. Recently, Electrophoretic International has been given a contract to develop a live test, as far as I am aware this firm has not developed a test yet or even done any work in relation to spongiform encephalopathy research. There appears to be different criteria to assess who should be supported? The Spongiform Encephalopathy Advisory Committee has asked for my experimental details of the live test. I need facilities so that I can make it available to farmers, I do not want some one else to exploit my test while I take a back seat and suffer. If the tests were used a few years ago, they would have helped to eradicate the disease.

I am surprised, that you are not aware of my published results in independently refereed International Journals (List of publications and copies of these papers enclosed)[1]. Details of my "post mortem test" were first published in 1987 (Narang, Asher and Gajdusek. Tubulofilaments in negatively stained scrapie-infected brains: relationship to scrapie-associated fibrils. Proc Natl Acad Sci 84, 7730-7734). Tubulofilamentous particles known as "Nemavirus" are only observed in tissues infected with the spongiform encephalopathies agent, these findings have been confirmed by others.

Over 25 years of research has revealed that all spongiform encephalopathies (SEs) are slow virus transmissible infectious disorders of the brain. It is a fact that Nemavirus/scrapie-associated fibrils (SAF) are ultrastructural markers (electron microscope), whilst protease-resistant protein (PrP) is a protein marker (electrophoresis). The PrP molecules aggregate to form SAF which occur as an internal part of Nemavirus. Each Nemavirus consists of three layers: (i) an outer protein coat, (ii) an intermediate ssDNA layer (DNA marker) and (iii) inner PrP/SAF. One of these markers has to be used for diagnostic test and I have them all.

It is not only that you have been given wrong facts by people who have no working experience with the disease the "post morten test" study was a real mixup. According to my record I was given 10 brains in 1990–91 and the information supplied by MAFF in 1991, I identified eight out of 10 correct (not three out of five as in your letter). There were no false positive results. The paper was submitted on 20 March 1991 to Dr N F Lightfoot, Director PHL, Newcastle, however, it was held for an internal review for six months, during this period he also destroyed very valuable research materials. I was told by Dr Lightfoot to ignore the information given in the original Forms.

At this stage I found the results supplied by MAFF show different dates for the specimens to those on the Forms supplied with the specimens. Obviously this revealed that the same specimens were not tested by both the participating laboratories. The false negative results were due to a mixup of specimens by MAFF, which invalidated the whole study. Although dates are correct for the remaining seven specimens, under such circumstances it would be hard to believe that there was no mixup in these specimens. Mr Ray Bradley's letter to Professor Edwardson, dated 27 November 1990, page 1, paragraph 5, demonstrates that the MAFF study was not a blind study. In another study 27 cow brains from apparently healthy cattle over four years old were collected from a local abattoir for examination by EM as a part of the World in Action Programme. Out of 27 brains examined eight were positive for Nemavirus. May I also point out, that at the request of the families I have done this test on a number of patients including, Stephen Churchill and Jean Wake, all cases confirmed histologically months later. I have also been approached by a number of other families of CJD sufferers to conduct both "post mortem test" and urine test. I have been also contacted by a number of farmers to do a similar test on their cattle.

[1] Not Printed.

At the beginning of the MAFF experiment in 1990–91, I was under the impression that this was an honest evaluation of my test. Recently I found minutes of a Public Health Laboratory Service Board review meeting held on 23 October 1990, where my scientific work was discussed. Sir Bernard Tomlinson was supposed to represent me and was asked to pick the best out of a number of dates. He wrote back, "only 23 October 1990 is not suitable". Sir Bernard Tomlinson was greatly surprised that PHLS picked the day not suitable to him. The obvious reason appears on page 38 of the minutes of the committee meeting in which Dr J Smith said "The other side is the smear thing. Blind trial by independent people. Colindale is told and Colindale tries it out". It continues "Dr Bostock said he will raise this in Edinburgh on 24 October". I had received two specimens before 23 October 1990, and others after 16 November 1990 and proof of this mixup is on the forms which came with the specimens.

The Public Health Laboratory Service was originally established for the control of the spread of infectious diseases. It is strange that one of the reasons given for my suspension was "that you had witnessed cows with clinical features of BSE passing through markets and into food chain in Cumbria". PHLS rather than investigating this issue further, suspended me. It appears to be all part of the smear campaign, it is not only myself who has suffered for the last six years, the public have been misled. Only an independent inquiry can clear the mistrust.

After this experience I find it very difficult to trust those who have limited knowledge of the published work and have not done a single slow virus experiment, are in best position advising how to conduct the research work.

Copy of a letter to Mr Douglas Hogg MP, Minister of Agriculture, Fisheries and Food from Dr Harash Narang, dated 29 December 1995

Since my last letter to you dated 27 December 1995, a family has contacted me after they read the article in "Night and Day". The parents consider their son is suffering from suspected CJD, and were told in July 1995 by a consultant that their son has some incurable degenerative brain disease. He may live for about six months. If he lives, will make his 20th birthday in early 1996. This will be the fourth known person under 20 in the UK to contract the disease. It is strange that a number of patients under the age of 40 have contracted this disease in the past few years in the UK and therefore it is important to realise that this is a new phenomenon which suggests that there is a different source of infection in these patients.

I went to see his parents last night, urine specimens from the patient were given to me to do the live test. Unfortunately, as usual I do not have the laboratory facilities to do the tests. I hope you are aware that recently I diagnosed CJD using Mrs Jean Wake urine specimens. Dr Robert Penny consultant neuropathologist by post mortem has also confirmed the diagnosis of CJD in this patient.

I am sure you have seen BSE cows staggering and being put down on television. Parents of the boy have asked me to invite you and Rt Hon Stephen Dorrell MP, Secretary of State for Health, to come and meet their son to see the human suffering, so that you can realise the need of developing a rapid test for this devastating disease. They would also like to extent this invitation to Rt Hon Gavin Strang MP, and Harriet Harman MP.

Copy of a letter to Mr Douglas Hogg MP, Minister of Agriculture, Fisheries and Food from Dr Harash Narang, dated 5 January 1996

Thank for the reply to my letter from Mr Marcus Nisbet, your Private Secretary dated 4 January 1996. I have no objection to you sending relevant published technical information to all of the recipients of my letter.

Since my last letter to you dated 29 December 1995, the parents who suspect their son is suffering from CJD would like to hear as soon as possible from you and they have asked me to act as an intermediary.

Further to the information which I supplied to you on 27 December 1995, I received a copy of a paper from the Prusiner's Group. From the title of this paper, it is evident that these authors have been doing experiments using transgenic mice similar to those used by Dr Collinge.

As you will see after five years experimental work with these mice, Prusiner has concluded in this paper that "another protein" is required to produce the disease. I published this original finding in detail in 1992.

"Another protein"—I call it an "accessory protein"—requires a DNA to code for it. This DNA is not a host DNA, otherwise we will all get CJD. It is non-host virus DNA. It leaves you with "Nemavirus".

Similarly the work on the human transgenic mice by the Collinge Group revealed that animals inoculated with CJD and BSE tissues do develop the disease, but with variable incubation period.

You have to remember that humans do live longer than 300 to 600 days. Since these mice carry an additional human PrP gene (the role of which we do not know) your scientists have yet to discover if they will produce the human form of PrP. The answer will be negative. I would happily explain why this is bound to be the case.

Copy of a letter to the Rt Hon Stephen Dorrell MP, Secretary of State for Health, Department of Health from Dr Harash Narang, dated 8 January 1996

I have been advised by Mr Douglas Hogg MP that the matter of Creutzfeldt-Jakob disease (CJD), being a human disease, is something which I should properly bring to your attention. Enclosed is a copy of the letter which I originally sent to Mr Hogg.[1]

I would be grateful if you would give the matter your close attention, and I very much look forward to your response.

Copy of a letter to the Rt Hon Stephen Dorrell MP, Secretary of State for Health, Department of Health from Dr Harash Narang, dated 28 January 1996

Thank you for the reply to my letter from Mr Shaun Gallagher, your Private Secretary, dated 19 January 1996. I do understand, that you have a very heavy pressure on your diary and personally cannot come to meet the parents of the CJD case.

This will be the fourth known person under 20 in the UK to contract the disease, although a case has been found in the age group in another country. For a long time and during the 1960s rabies vaccine was prepared from sheep's brain and extensively used in human medicine. BSE/scrapie is not spread by physical contact but either by inoculation or eating contaminated food. In the UK where most of the BSE exposure has occurred, there will be more cases among young people over a period of time. This is what appears to be happening. Certainly, there already are an appreciable number of patients under the age of 40 those who have contracted this disease in the past few years. This to be a new phenomenon in the UK.

Given the current limited knowledge, it is hard to conclude, that there is no risk to humans. Unlike scrapie, when fed with known BSE tissue 100 per cent mammals developed the disease. To appreciate the danger for humans, any miscalculation would spell disaster. Simply look at the rise in the number of BSE cases from month to month during the period of 1985 to 1990 (copy of the chart enclosed)[2]. I hope that in the case of humans, we do not see a similar situation.

The parents of the 4th teenager CJD victim have asked me to enclose their letter with mine[3].

[1] See pp. 103.
[2] Not Printed.
[3] Not Printed.

Copy of a letter to Mr Douglas Hogg MP, Minister of Agriculture, Fisheries and Food from Dr Harash Narang, dated 2 April 1996

Thank you for your letter dated 21 February 1996. As we are all aware, the situation has changed dramatically since then. In the current crisis it appears that you are becoming increasingly committed to a large-scale slaughter policy. This would be too much like killing a number of Aids patients and then claiming we'd got rid of Aids.

In the crucial matter of restoring world wide confidence in British beef, your chief priority must surely be to clean the National herd of BSE. To this end, I suggest a two part plan of action.

1. The testing of all cattle brains from animals gone for slaughter and, of those found to have BSE, to trace and to slaughter their progeny.
2. On the farms themselves to urine test all herds and thereby erradicate whatever BSE remains.

I have been asked by a very large number of farmers and abattoir workers that I should test their cattle, and assure the public that their meat is not contaminated. They would like to undertake this voluntarily as they understand from the media that the public wants the animals tested before the meat goes into the consumption cycle. I am prepared to help these farmers and restore confidence by testing the cattle and moving the agenda forward. This would save animals, which would be otherwise slaughtered, even if they are not incubating the disease, therefore saving a huge amount to the country.

As requested I have sent all my published papers to the SEAC Committee in December 1995, and up to now I have had no further communication from any of the committee members. Although I have heard from the media that I have not bothered to answer Professor Pattison's letter, I have yet to receive one.

As to scientific advice you have been receiving about my work, I take it that you are not aware that it largely comes from people not involved in SE work.

ANNEX B

Copy of a letter to Dr Harash Narang from Mr Marcus Nisbet, Private Secretary to the Minister of Agriculture, Fisheries and Food, dated 21 December 1995

Thank you for your letter of 9 December to the Minister of Agriculture, Fisheries and Food, Mr Hogg which gave brief descriptions of the two tests you are working on for the identification of BSE and CJD; I have been asked to reply.

We are grateful for your approach and offer on these tests. However, as a scientist you will appreciate that all claims need to be rigorously tested and receive sound experimental support. This is, of course, usually seen to be achieved by publication of papers in independently refereed scientific journals. We are not aware that details of your tests have been published in this way and so would be grateful if you could provide more detailed information of a standard which can be properly assessed.

We would be grateful if you could confirm that what you call the "post mortem test" is the same test which you demonstrated to the Central Veterinary Laboratory in 1989. According to our records, when you used this to examine brains provided by MAFF from affected and unaffected cattle, the test correctly identified only 3 out of 5 positive brains. We also note that you have never published details of this test in a refereed journal. It may, of course, be that you have collected further evidence to indicate that the test now performs better than it did in the earlier trials; if so we would be interested in the details of what it is and where it has been published.

I understand that the Secretariat of the Spongiform Encephalopathy Advisory Committee has already written to you to ask that you provide that Committee with full details of your "urine test". The Minister has asked me to urge you to provide them with full details to enable them to evaluate the test to see if it could prove clinically reliable and robust. The sort of details needed are the exact protocol; information on how the results have been validated, in both humans and animals; information on the stage during the incubation period of the disease that the test first detects positives; and details of the reliability of the test in terms of the percentages of false positives and negatives found. To be usable in the field any test for BSE would have to distinguish successfully between animals with and without BSE but which are showing clinical signs of disease. If your test can also be used to detect sub-clinically infected cattle, as has sometimes been suggested in the media, we would be interested to know how you have subsequently verified animals as suffering from BSE or as being BSE-free. We would also be interested to know if the results of your studies on these tests have been published, or are submitted for publication, in a refereed journal.

We are interested to see in more detail the results of your work and would welcome a reliable test for diagnosing BSE in live cattle, especially if it could detect sub-clinical cases. However, any such test must be validated to the normal, accepted levels of scientific proof.

Copy of a letter to Dr Harash Narang from Mr Marcus Nisbet, Private Secretary to the Minister of Agriculture, Fisheries and Food, dated 4 January 1996

The Minister has asked me to thank you for your further letter of 27 December. You attached to that letter a very large number of technical documents and it will obviously take some time to assess these fully. I have not copied this letter to recipients of yours but would be grateful for your confirmation that you would be happy for the final reply, which may well contain our assessment of the relevant technical papers you have published and other results, to be copied to some or all of the recipients of your letter at that stage. I am afraid that it is likely to be some little time before we can come back to you on this.

Copy of a letter to Dr Harash Narang from Mr Marcus Nisbet, Private Secretary to the Minister of Agriculture, Fisheries and Food, dated 5 January 1996

Thank you for your further letter of 29 December to the Minister.

As I hope you will appreciate CJD is a human disease and this is really a matter for the Secretary of State for Health and not the Minister of Agriculture, Fisheries and Food. I note that you have copied your letter to the Secretary of State who, I am sure, will be replying in due course.

Copy of a letter to Dr Harash Narang from Mr Shaun Gallagaher, Private Secretary to the Minister of Agriculture, Fisheries and Food, dated 19 January 1996

Thank you for your letter of 29 December to the Minister for Agriculture, Fisheries and Food, which you copied to the Secretary of State for Health, about the development of a rapid test for Creutzfeldt-Jakob Disease (CJD). As you known, your letter has been referred to this Department because CJD, as a human disease, falls within our remit. Thank you also for your further letter to the Secretary of State of 8 January.

Mr Dorrell was very sorry to hear of the sad case of the young man you mention in you letter whose parents are concerned that he may be suffering from CJD. Your letter says that the young man's parents would like Mr Dorrell to meet him. I am afraid that Mr Dorrell's diary is under heavy pressure and it will not be possible for him to accept this invitation.

The Government appreciates fully the seriousness of CJD. It is a rare disease and, if a validated test were to become available, it would overcome current diagnostic difficulties. I understand the Spongiform Encephalopathy Advisory Committee (SEAC) wrote to you on 4 December seeking details of your urine test. The Committee still wishes to give the Government an independent assessment of its clinical robustness and would welcome receiving the details necessary for this.

SEAC has recently considered the cases of the two teenagers with confirmed CJD. The Committee concluded that it was not possible to draw conclusions because cases of sporadic CJD in teenagers, although rare, have been found in other countries before the appearance of BSE and the two cases had no exposure to risk factors for CJD and no contact with BSE.

Copy of a letter to Dr Harash Narang from Mr M T Skinner, Health Aspects of the Environment and Food Division, Department of Health, dated 12 February 1996

Thank you for your further letter of 28 January to the Secretary of State for Health about BSE and CJD, enclosing a letter from Mr P Hall.[1] I have been asked to reply to your letter and will also be sending a separate reply directly to Mr Hall.

I can only repeat what Mr Gallagher said in his letter: the Government takes BSE very seriously and has introduced measures to safeguard public health. These measures have been based on the assumption that BSE could be transmissible to man, even though there is no current scientific evidence to support this. One of these measures has been to establish the National CJD Surveillance Unit which investigates the incidence and epidemiology of CJD in the UK since the BSE epidemic began, paying particular attention to occupation and eating habits so that any change in the pattern of CJD. This includes detailed investigation of individual cases of suspected CJD referred to the Unit.

The Unit has so far found no conclusive evidence of any change in CJD that could be attributable to BSE. The Government recognises the importance of continuing the Unit's activities for some years to come because of the potentially lengthy incubation periods for CJD. As I am sure that you are aware, the measures to safeguard public health have recently been enhanced and I am sure these, too, will need to remain in place for some years.

The independent Spongiform Encephalopathy Advisory Committee (SEAC) gives special consideration, drawing on reports from the Surveillance Unit, to cases of CJD where particular risk factors might be involved, eg farmers, or where the cases are unusual, for example teenagers. In the cases of the two teenagers in the UK with confirmed CJD, SEAC concluded that it was not possible to draw conclusion because cases of confirmed CJD, although rare, had been reported in teenagers and young people in other countries before the appearance of BSE (four cases in teenagers in the USA, France, Canada and Poland, and two cases in their twenties in Poland). In addition, the two UK cases had no exposure to risk factors for CJD and no contact with BSE.

In relation to Peter Hall, I understand that the clinicians caring for him have yet to decide whether to refer the case to the National CJD Unit for investigation.

Copy of a letter to Dr Harash Narang from Mr Douglas Hogg MP, Minister of Agriculture, Fisheries and Food, dated 21 February 1996

Thank you for your letters of 27 December and 5 January. As you know from my Private Secretary's replies to you, I have sought expert advice on the highly technical issues you raise in your letter. I attach a copy of the advice which has been sent to me. I am copying this to recipients of your letters.

I have to say, on reading the technical advice that I have been given, that I do not see any reason why we should depart from the long established mechanisms under which this Department funds research.

MAFF is planning to spend £6.4 million on research into BSE and related diseases in the coming financial year. Furthermore, we are not the only funding bodies. The BBSRC, MRC, private trusts and individuals also fund work and the SEAC is consulted. So far as MAFF research is concerned the Policy Division determines the field of research and the Chief Scientists Group organises the proposals and ultimately issues contracts. All proposals are carefully scrutinised by the Policy Division, CSG and sometime others, including independent scientists. There is ongoing liaison with the Research Councils concerned with TSE research and

[1] Not Printed.

we also hold quinquennial reviews (that last in early 1995) in which scientists independent from MAFF are referees. Feedback is constantly received from expert committees including the SEAC as projects are established and current ones come to an end.

This system applies to all MAFF research and we are constantly refining the procedures to make them more efficient and to secure the best return for the tax payer. I reject completely the accusation that no real progress has been made. This is at variance with the views of all the major organisations in the world who have an interest in TSE research and include The World Health Organisation, The European Commission and the Office International des Epizooties. Two researchers at CVL, Mr Wilesmith and Mr Bradley have received important awards from national and international organisations for their research and other achievements in the TSE field. Most of the project leaders of work funded by MAFF are in national and international demand for presentations at scientific meetings.

I certainly agree that fairness is one criterion for awarding research funds but of equal or greater importance are the need for the work, the skill of the team, the resources required and available, the design of the study, its costs and priority in relation to other studies. Work with TSE is necessarily long term and usually demands a team approach in a centre of excellence though new groups have been encouraged to initiate work and some have creditable results.

I understand that any proposals from yourself have been examined in the same way as those from other individuals and institutes. If they have not been funded this is because they lacked sufficient merit or were not of sufficient priority.

In your letter of 27 December 1995 you appear to suggest that research grants may not be awarded fairly and as an example you state that Electrophoretics International Ltd has been given a contract to develop a live test without previously having experience in TSE research.

You are wrong to suggest this Company has no experience of TSE research. They have been previously working with blood and cerebrospinal fluid (csf) from patients with CJD using a technique known as 2 dimensional electrophoresis. They wish to explore the same line in cattle with BSE and to do this they need samples and access to the appropriate equipment. This can be provided at Weybridge where the staff have, for some time, been developing a similar method using funding provided by MAFF. However, Electrophoretics International Ltd are not being funded by MAFF for this work.

It is clear to me from reading the papers that this Department and the BBSRC have gone to considerable lengths to try to confirm some of the experiments which you claim underpin your theories about the agents causing BSE and scrapie. I have to say that, despite the expenditure of considerable sums of taxpayers' money the results remain disappointing and I see no justification and none of my scientific advisers have proposed that we should spend more money to develop tests based on theories which are not accepted by the majority of workers in the field and which are not substantiated by research findings. I know that this will be a disappointment to you and I am prepared to ask my scientific advisers to look again at the matter if you can give us sufficient technical background for a proper research proposal. However I am not prepared to ask them to depart from the normal procedures and to underwrite work which has only been reported in very general terms in the national press and not in sufficient detail in the peer reviewed scientific press and which you have subsequently refused to explain both to Departments and to the independent advisory committee, SEAC.

You will also I think see from the scientific advice that I have been given that there is no evidence from Dr Prusiner's work, as reported in your letter of 5 January, to substantiate the nemavirus theory nor of course does Dr Prusiner himself and his co-workers suggest this explanation of their findings in the paper you sent to me.

Extract from supplementary memorandum by the Department of Health [T6/BSE10]

Letter to the Clerk of the Health Committee from the Parliamentary Clerk, DoH

Thank you for your letter of 22 April about the further information referred to by Government witnesses at the oral evidence session on 17 April in Questions 193, 204, 228, 240 and 256.

Angela Evans at MAFF has already provided you with the information referred to by Mr Meldrum at Questions 228 and 256.

I attach a list and copies of the correspondence relevant to Sir Kenneth Calman's answer to Question 193. Sir Kenneth's answer to Question 204, referred to correspondence between MAFF Ministers and Dr Narang. We believe that enclosure 10 of the documents provided for Question 193 fulfills the undertaking given as it contains detailed comments on Dr Narang's views and a summary of MAFF's involvement with his work.

The Government's views on Dr Narang's comments in reply to Question 240 are as follows. There are a number of theories about the nature of the agent which causes spongiform encephalopathies. The most widely held theories are summarised in section 1.19 of "Transmissible Spongiform Encephalopathies: A Summary of Present Knowledge and Research" published by the Spongiform Encephalopathy Advisory Committee in February 1995. Copies of this publication are available in the Library of the House.[1]

Dr Narang proposes an alternative theory on the basis of his paper of 1992 (see document 4 in response to Question 193), involving a nemavirus, a combination of PrP molecules and single-stranded DNA. So far as we are aware, no one has been able to reproduce Dr Narang's experimental results or demonstrated that the spongiform encephalopathy could be produced in the way he suggests. However, if his test for CJD is proven effective, it would be of great value whatever its theoretical basis might be.

9 May 1996

Q 193 (Sir Kenneth Calman's Answer): List of copies of correspondence between Dr Narang and the Department of Health and MAFF in relation to Dr Narang's alleged urine test for CJD—4 December 1995 to 21 February 1996

1. Copy of letter of 4 December 1995 from G M Robb, Department of Health official to Dr Narang on behalf of the Spongiform Encephalopathy Advisory Committee (SEAC) requesting details of his urine test for CJD (See below).

Note:

Mr Robb spoke with Dr Narang on the telephone on 21 December 1995 as a reply had not been received. Dr Narang said that the test was under commercial development and he was not, therefore, prepared to let SEAC have any detailed information about it, even on a confidential basis. He stated that he had not published any papers about the test. He was willing to give SEAC details of the papers he had published.

2. Copy of letter of 9 December from Dr Narang to Douglas Hogg giving brief outline information about his post mortem and urine tests for BSE, and offering his assistance. (See Annex A on page 102)

3. Copy of letter of 21 December 1995 from M Nisbet, Douglas Hogg's Private Secretary to Dr Narang thanking Dr Narang for his offer and asking for detailed published information about his tests. (See Annex B on page 107)

4. Copy of letter of 27 December 1995 from Dr Narang to Douglas Hogg copied to G M Robb, Department of Health official for SEAC, containing information about Dr Narang's research and a list of published papers. (See Annex A on page 103)

5. Copy of letter of 29 December 1995 from Dr Narang to Douglas Hogg (copied to Stephen Dorrell) claiming that he has successfully used his urine test for CJD on two patients suspected of having CJD. Dr Narang's letter invites Mr Hogg and Mr Dorrell to visit one of these patients. (See Annex A on page 104)

6. Copies of a paper published by Dr Narang in 1992 and of the front page of a paper published by Glenn C Telling et al in 1995, faxed to G M Robb, Department of Health official for SEAC, on 4 January 1996.*

7. Copy of letter dated 5 January 1996 from M Nisbet, Douglas Hogg's Private Secretary acknowledging Dr Narang's letter of 29 December which was passed to Mr Dorrell to answer as it concerned CJD. (See Annex B on page 107)

8. Copy of letter of 5 January 1995 from Dr Narang to Douglas Hogg enclosing copies of the same papers* referred to in 6 above. (See Annex A on page 104)

9. Copy of letter of 19 January 1996 from S Gallagher, Stephen Dorrell's Private Secretary responding to Dr Narang's letter of 29 December encouraging Dr Narang to provide details of his test for CJD to SEAC so that it can be independently assessed. (See Annex B on page 107)

10. Copy of letter of 21 February from Douglas Hogg to Dr Narang in reply to his letters of 27 December 1995 and 5 January 1996. The letter includes an expert scientific critique of Dr Narang's letters. (See Annex B on page 108)

(**Not printed*)

[1] Not Printed.

Copy of a letter to Dr Harash Narang from Mr G M Robb for the SEAC Secretariat, dated 4 December 1995

I am writing to you on behalf of the Spongiform Encephalopathy Advisory Committee (SEAC) which advises the Government on transmissible spongiform encephalopathies, including BSE and CJD.

The SEAC has seen reports in the media of a potential diagnostic test for CJD which you are reported to have developed. Any such test, if clinically robust, would be valuable, and the Committee have therefore asked if you would be willing to send them details of this test, and, if possible, also of its use so far. Moreover, they would be interested to know whether you have yet submitted any papers on the test for publication, and if you have, whether you would be prepared to let them see copies.

The Committee will next meet early in January 1996, and it would, if possible, be very useful to have an initial response from you by the end of the year.

Extract from a letter to Dr Harash Narang from Mr M A Anderson, Senior Veterinary Investigation Officer, Ministry of Agriculture, Fisheries and Food, dated 15 March 1991

Please find below the results of the various examinations and tests performed on 10 samples of brains from 10 cattle supplied to you. *(Certificates not printed.)*

ANNEX 1

No.	Date	Clinical BSE Signs	Electron Microscopy†	Histopathology¶	SAF*
1	19.9.90	+	+	+	+ + +
2	20.9.90	+	+	+	+ + +
3	16.11.90	–	–	–	–
4	20.11.90	–	–	–	–
5	23.11.90	+	–	+	+
6	23.11.90	+	–	+	+ + +
7	19.12.90	+	+	+	+ + +
8	20.12.90	–	–	–	–
9	3.1.91	–	–	–	–
10	10.1.91	–	–	–	–

Date = Date of collection and submission to Dr Narang.
† = Test performed by Dr H Narang using electron microscopy.
¶ = Histopathology performed at Newcastle VIC.
*** = Scrapie associated fibril examination at CVL.**

ADVICE TO THE MINISTER ON DR NARANG'S LETTERS OF 27 DECEMBER AND 5 JANUARY

The main thrust of Dr Narang's letters addresses two points: (a) the comparative blind study of brains from BSE-affected and control (unaffected) cattle using electron microscopy (two methods) and conventional histopathology. I will deal with this study below, including sufficent detail so that others reading this note are clear as to the aims, methods, results and conclusions, and (b) evaluation of Dr Narang's work in general.

(a) COMPARATIVE BLIND STUDY

Historical

BSE is identified clinically and, being a notifiable disease in Member States of the European Union (EU), is reportable to the relevant authorities. Failure to do so is an offence. Since compensation at 100 per cent of valuation (subject to minor adjustment) is payable in the UK, there is a strong incentive to report cases independently of the legal requirement to do so. Following notification, in a straightforward case, suspects are visited by a Ministry Veterinary Officer. If he or she believes BSE is the clinical diagnosis the animal is humanely killed, the carcase is taken to a secure site and the head removed for microscopic examination of the brain at a Veterinary Investigation Centre. The remaining parts of the carcase are incinerated.

Statutory Confirmation of BSE

This is done by microscopic examination of the brain by the standard techniques which are agreed by Member States of the European Union, The Office International des Epizooties and World Association of Veterinary Laboratory Technicians. Veterinary neuropathologists from the EU and several countries round the world have been trained to a common standard using the methods developed at the Central Veterinary Laboratory, Weybridge. This method can be regarded as the "gold standard".

Alternative Methods in Use

Detection of the disease-specific form of PrP (PrP^{sc}) by immunoblotting (Western blotting) or by visualising scrapie-associated fibrils (SAF) using electron microscopy are methods supported for use in particular circumstances by the above-mentioned organisations. Approved protocols exist. Immunocytochemistry is used by some laboratories and protocols are being developed for international approval and use. There is a sound correlation between the results derived by microscopic examination of the brain and SAF detection using these protocols though autolysis may affect the results from the former method in a small number of cases.

Other Methods

The "Touch Method" (Narang, Asher and Gajdusek, 1987) has been used by Dr Narang in experimental scrapie in hamsters and rats, in experimental and natural CJD and in other species with transmissible spongiform encephalopathy (TSE), including cattle with BSE. Our own evaluation of the method is that it is specific for TSE, at least in regard to scrapie and BSE but that it shows no advantage over the conventional SAF method and is significantly less senstive. It is however, a quicker method. This was one reason why the comparative study reported below was initiated in 1990–91. The other was that, at that time, we did not know the comparative sensitivity between the conventional SAF method and Dr Narang's "Touch Method". No-one has, to our knowledge, published successful results from a peripheral tissue using the "Touch Method". This would be an essential requirement if it was to have any value as an *ante mortem* test in a clinically or pre-clinically affected animal. Mr Bradley did mention this important aspect to Dr Narang at the seminar held in London on 17 December 1992 and suggested he tried the method on spleen and lymph nodes of hamsters terminally affected with scrapie. We are not aware of any publications from Dr Narang on this aspect reporting work he may have done. We also point out that *even* if a more rapid and effective test was available to detect PrP in the brain (which it is not) the organs which are the most likely to contain infectivity in an infected animal (whether or not they are from an infected animal) are removed at source under the Specified Bovine Offals Order, so they can *not* enter any food or feed chain.

We know of no laboratory which uses the "Touch Method" for the routine confirmation of TSE in any species. Indeed, when the protocols for the three standard confirmatory methods were circulated for comment to a number of laboratories round the world concerned with TSE diagnosis and research, none even mentioned the "Touch Method".

Electrochemistry of urine as a means of clinical diagnosis of Alzheimer's disease and CJD was originally used by the French and has been developed further since, at CVL Weybridge. It is not yet developed to the stage where it can be used in the field.

Detection of SAF in urine has, so far as we are aware, only been reported by Michael Hornsby ("Times", 6 November 1995) referring to work by Dr Narang on the basis of information presumably supplied by Dr Narang. We know of no other published report on the subject and are disappointed that Dr Narang is unable to supply, even in confidence, any information to SEAC on the protocol of data on the validation of the method.

Detection of TSE-specific protein in cerebrospinal fluid is another method that is under investigation by various groups, but has not yet reached the stage of publication of validation.

The Aim of the Blind Study Involving Dr Narang

The aim was to determine the sensitivity and specificity of the "Touch Method" by comparison with microscopic examination of the brain and with the conventional SAF detection method in a blind study. In this context "blind" means that the operators (Dr Narang, PHLS Newcastle—("Touch Method"); pathologists in the VI Service (microscopic examination of the brain); and electron microscopists at CVL Weybridge (conventional SAF method)) were unaware of the clinical status of the cows donating the brains or the results of tests by their opposite numbers until all the tests were complete. At this time the complete results from each study were communicated to all participants by the key holder, who played no part in the practical aspects of the study.

The Methods

Brain samples were supplied from ten cattle, five suspected to have BSE and five clinically healthy controls which were homebred, and came from farms without a history of BSE. No participant knew the history of any case.

Each test was carried out by the standard protocols of the laboratory concerned. Dr Narang reported the results to the SVIO, Newcastle VIC within half a day. Final results for all studies were tabulated and sent to each participant.

The Results

These are listed in Annex 1. Mr Bradley wrote to Dr Narang on 20 March 1991 five days following communication of the results (Annex 2). Dr Narang did not reply to this letter. However, he did point out an error to Dr Lightfoot on 20 March 1991 in whch he noted that brain 4 had been received with brain 3 on 16 November not 20 November as stated in the report. This is a simple error of transcription, not an error in the brain samples submitted to each participant. This has been confirmed both verbally and in writing by the SVIO, Newcastle VIC. The brain in question was negative in all three studies and supports this interpretation.

Comments of the Results

1. There was complete agreement between the results obtained from clinical examination, microscopic examination of the brain and the conventional SAF method.
2. Dr Narang's "Touch Method" gave no false positive results, ie there was complete agreement between all tests in all BSE-negative controls.
3. The "Touch Method" correctly detected two BSE positive cases out of five. After re-examination Dr Narang subsequently changed one negative diagnosis to positive, but outside the agreed time interval.
4. The "Touch Method" appeared, under the conditions of the study, to be specific for BSE. However, its sensitivity was inadequate since only two out of five (40 per cent) or at best three out of five (60 per cent) of positive BSE cases were correctly identified.
5. The only advantages of the "Touch Method" over the conventional SAF method are speed (20 minutes versus 3 days at that time) and pershaps cost. The SAF method can now be completed in 3 hours.

Subsequent Developments

A recent paper by Stack *et al* (1995) has evaluated the "Touch Method" using the cervical spinal cord and brains from 12 sheep with natural scrapie and six scrapie-negative controls, by comparison with the conventional (centrifugal extraction) technique for SAF and microscopic examination of the brain. The conclusion of this study is that the "Touch Method" did not offer any advantage over the conventional SAF method for the diagnosis of natural scrapie. Furthermore, the SAF method was clearly more sensitive since it gave consistently higher SAF scores in more brain regions than the "Touch Method". No false negative results were reported supporting the view that the "Touch Method" is specific for TSE in sheep.

Overall Conclusion

Dr Narang's "Touch Method" appears specific for natural TSE in sheep and cattle and in the limited studies undertaken (17 affected animals and 11 controls in total). However, to be of value in the field it must have a sensitivity, at least comparable to existing methods (eg the conventional SAF method). Its sensitivity is substantially lower, and could not be used with confidence in a practical way to protect public or animal health when more sensitive methods are available.

The method might be of use in situations where speed of diagnosis is paramount (eg in some research situations) but on the understanding that a negative result could be false and that the case in question may be infected and might be shown to be so by use of a more sensitive method.

We are convinced that there is no justification for conducting any further evaluation of the "Touch Method" as it is inferior to methods already available, and protocols for which are adopted internationally. We reject Dr Narang's current accusation that the study was invalidated. If he thought this why did he write a draft paper for publication describing the study in 1991 using the table illustrated in Annex 1 (but with case 7 altered to produce a positive result without comment)?

(B) EVALUATON OF DR NARANG'S RESEARCH

Dr Narang's Papers

Dealing with the two files of papers not related to spongiform encephalopathy and the bibliography we note an impressive number of publications in refereed journals of repute and sometimes with eminent co-authors. Most of these papers relate to ultrastructural or electron microscopical studies in regard to conventional microbiological agents or diseases caused by them. Other than demonstrating a wide experience and a competence in the techniques, they are not directly relevant to spongiform encephalopathy (SE) research. We note that Dr Narang's main bibilography list contains 87 items which presumably includes all the 32 SE papers listed separately.

Examination of the 32 SE papers shows that 24 of them, and most of them since 1990, are single author papers. Some of the others, particularly the earlier ones are in co-authorship with scientists of distinction notably Dr E J Field, Dr R L Chandler and Dr D C Gajdusek. Certainly the papers are focused on two main areas, infection-specific particles as determined by electron microscopy and infection specific ssDNA and the connection between these two features. The greater majority of the SE papers are well known to the scientific colleagues I have consulted.

There are two original contributions that Dr Narang has made to the literature. Firstly the morphological description of nemavirus and secondly the evidence for the presence of homologous ssDNA. He has also contributed to the diagnostic technique known as the "Touch Method" and this has been critically reviewed above.

Tubulovesicular Structures (TVS) and Tubulofilamentous Particles (TFP)

It is very clear that the particles we now know as TVS and first described in ultra-thin sections by David-Ferreira *et al* (1968) and by Bignami and Parry (1971) are genuine TSE-associated structures. Narang and/or others have extended these observations to more species and diseases including CJD and BSE. That is commendable. There now becomes a dispute between Dr Narang and other researchers when he claims that TFP, or nemaviruses, are TVS cut in the longitudinal plane. This is not supportable either on the grounds of size or shape. (For the arguments see Liberski P) (1995) *Acta Neurobiol Exp 55, 149-154).*

His illustration of nemavirus (TFP) in more than one article, perhaps the best is Fig 2a in *Intervirology* (1993) 36, 1-10, has the morphological appearance of a doublet microtubule from 9+2 ciliary or flagellar axoneme (Chasey D (1994) *Intervirology* 37, 306). A possible origin for such structures is the ciliated epithelium of the ependyma. Cilia also occur in the grey matter of the spinal cord and brain. Such structures appear to be quite different from the intra-axonal tubulo'filamentous particles illustrated in his 1992 paper in *Intervirology* 34, 105-111. Similar structures of the latter have been found in myelinated axons and dendrites of scrapie-inoculated hamsters, in hamsters injected with a mercury salt and in sham, saline-inoculated control hamsters inoculated in a scrapie-free laboratory (Liberski P (1995) ref as above). Liberski concluded that TFP are swollen microtubules unrelated to TVS. We remain unconvinced that TFP (nemavirus), as described by Dr Narang are TSE-specific structures. The artistic drawings he has made are ingenious, but hypothetical and there is no morphological or immuniological evidence to support their existence as TSE-related structures.

TFP and the Touch Method

I now turn to the use of the "Touch or impression Method" and the identification of TFP on such grids. This would not be surprising either in the form of ciliary or flagellar axonemes or swollen microtubules. However, they would not be TSE-specific for the reasons stated above. However, we agree that TSE-specific fibrils can be observed on such grids, both in BSE and scrapie, from our own experience. We have no difficulty in accepting that the method is disease specific but are critical of its sensitivity when compared with the conventional SAF method.

Single Stranded DNA

I now pass on to the work on single-stranded DNA. As noted above, Dr Narang's model of a nemavirus (Fig 3 of Dr Narang's 1992 *Intervirology* paper or in his 1993 Brussels paper (VI/4131/94-EN) is ingenious. However, because we believe this to be based on the structure of a ciliary or flagellar axoneme, structures which are found in normal brain, the model is flawed. This does not itself exclude there being a disease-specific ssDNA in scrapie. Studies to investigate the existence of the specific ssDNA Dr Narang claims is part of the nemavirus, have been conducted by Dr L Bountiff in Professor Oxford's laboratory in association with Dr P Levantis. These studies could not substantiate Dr Narang's claim, even though his precise protocol was used under his guidance. The study was jointly funded by the BBSRC and MAFF and referred by Professor J Almond (University of Reading) and Mr R Bradley (Central Veterinary Laboratory). Dr Helen Grant, a

notable independent expert in the human disease CJD, was invited, at Dr Narang's request, to the final project meeting and she concurred with the results and conclusion namely that scrapie-specific ssDNA could not be identified.

Dr Bountiff concluded specifically in her report to MAFF and the BBSRC as follows:

"In the light of the data presented above". *(ie in the report)* "using protocols that were as consistent as reasonably possible with those used by Dr Narang previously and with due consideration of the criticisms made by by Dr Narang, we" *(Professor J Oxford, Dr P Levantis and Dr L Bountiff)* "were unable to detect a scrapie-specific DNA 1.2kb in length.

Since the previous reports of detection were inconsistent, and have only been made in nucleic acids from scrapie infected animals at a very late stage of clinical disease, we conclude the 1.2 kb band is:

(a) very unlikely to form the basis of a useful diagnosis test for scrapie:

(b) very unlikely to cast any new light on the pathognesis of scrapie.".

The "Touch Method" Re-Visited

Also at the London Hospital an opportunity was taken to examine brains of some of the scrapie-affected hamsters used in Dr Bountiff's study by the "Touch Method" which was first described by Almeida and Howatson about 30 years ago. Nemavirus (TFP) was found in scrapie-affected and in control brain testifying again to the non-specificity of the method. The particles were considered to be microtubules.

Probe for ssDNA

Dr Narang has claimed to have isolated the presumptive scrapie-specific ssDNA and has prepared and patented a probe that he claims can specifically detect it. This was done some years ago. We understand he supplied some of this probe to an American institute, experienced in scrapie research and molecular biology, on numerous occasions. During this period Dr Narang spent some days at this institute and personally supervised the preparation of nucleic acids from coded brains from scrapie-infected and control (normal) animals used for hybridisation with his probe. Hybridisation experiments were performed numerous times with different preparations of his probe. These probe preparations were either brought to the institute by Dr Narang or were subsequently sent by him after his departure. When one of his probe preparations was used no hybridisation could be detected. With a different probe preparation the results were totally inconsistent with the brain samples tested; that is the probe hybridised to both normal brain and scrapie brain material and could not discriminate between them. In simple terms the probe did not work. His clone was requested but was not received. If the probe is as effective as he claims why had it not been used to detect infected animals and people? Indeed, instead of using his "Touch Method" or the newly reported urine test, why is this probe not used even by Dr Narang? If a scrapie specific nucleic acid exists then it is probable that an effective test could be developed quite easily by using a specific probe and the polymerase chain reaction. It is incongruous that he claims to have such a probe and that it is not being used by him or anyone else so far as we can tell.

Urine Test

We are unable to comment upon Dr Narang's newly developed urine test since insufficient detail is published. We appreciate that he may wish to protect his discovery. This can be simply done at modest cost by patenting. However, if it is an effective test, we urge him to publish the protocol, and the validation procedures that have been completed (ie a blind study of urine from confirmed cases of SE and of urine from healthy controls determined by an approved method), in order that it can be used to benefit humans and animals as soon as possible. It is disappointing that he claims that his test could have contributed to the eradication of BSE much earlier, but he has kept the data to himself seeking to publish only the bare essentials in a daily newspaper rather than in a refereed scientific jurnal. If his test depends on the identification of nemavirus, the same questions will arise as those referred to in section (a) above. However, it is not clear what TSE-specific particles he claims to have discovered.

Protein "X"

Dr Glenn Telling and his colleagues propose (in the paper Dr Narang kindly sent) that a *species-specific* (or host) macromolecule, they called protein X, participates in prion formation and may function as molecular chaperone in the formation of the disease-specific form of PrP (PrP^{Sc}).

In Dr Narang's 1992 paper (*Res Virol* 143, 381-386) he mentions a *non-host* peptide "Accessory Protein" which is *coded for by the putative ssDNA* already discussed above. He indicates that the protein acts as an enzyme and described its (hypothetical) function.

There is thus a major difference in the two hypotheses. Telling *et al* (1995) are quite clear that the hypothetical protein X is *a host protein.* Furthermore, they state that, like the binding of PrP^{C} to PrP^{Sc} which is most efficient when the two isoforms have the same sequence, the binding of PrP^{C} to protein X seems to exhibit the highest affinity when these two proteins are from the same species. By contrast, Dr Narang is equally clear that his unconfirmed ssDNA and the hypothetical "Accessory Protein" are *non-host* and the former at least is scrapie-specific. The two observations are therefore quite independent and unrelated.

Telling *et al* (1995) developed the protein X hypothesis to explain an apparent interference phenomenon in transgenic mice carrying human and mouse *PrP* genes when challenged with CJD (human prions). Such mice did not develop CNS dysfunction any more frequently than non-transgenic controls. However, when the mouse *PrP* gene was ablated transmission resulted without difficulty and it was concluded that mouse PrP^{C} somehow in inhibited conversion of human PrP^{C} into human PrP^{Sc} in the original transgenic model.

It is noteworthy that a study in the UK (Collinge *et al* 1995 *Nature* 378 779-783) using the same transgenic mouse model (one containing both mouse and human *PrP* genes) CJD did transmit quite easily. Therefore it is quite probable there are other possible explanations which do not require the introduction of the protein X theory.

Decontamination Studies

Dr Narang's 1987 paper in the PHLS *Microbiology Digest* 4, 64-67 was an important paper because it included information on laboratory safety and agent decontamination and would be widely circulated within hospitals and laboratories, particularly those of the Public Health Laboratory Service (PHLS) in the UK. Unfortunately it was out of date and inaccurate. The issues in question relate to:

(a) Listing four autoclaving procedures recommended by the DHSS in 1981 (though Dr Narang claimed this was 1984), two of which were suspended by new information resulting from research and published by the DHSS in 1984.

(b) An incorrect statement relating to the effectiveness of steam at 132°C.

(c) An overcautious statement in regard to the concentration of sodium hypochlorite required for effective decontamination of the scrapie and CJD agent.

(d) An incorrect statement relating to the concentration of sodium hypochlorite required for effective decontamination of the scrapie and CJD agent.

The full critique was in The PHLS *Microbiology Digest* (1988) 5 (1), and the author was Dr D M Taylor of the then AFRC/MRC Neuropathogenesis Unit (NPU) in Edinburgh.

REFERENCE

STACK, M J, ALDRICH A M, KITCHING, A D AND SCOTT, A C (1995). Comparative study of electron microscopical techniques for the detection of scrapie associated fibrils. *Res Vet Sci* 59, 247-254.

ANNEX 2

Copy of a letter to Dr H Narang from Mr R Bradley, Central Veterinary Laboratory, dated 20 March 1991

TUBULO-FILAMENTOUS STRUCTURES IN CATTLE BRAINS

By now Mark Anderson of Newcastle VI Centre will have communicated to you the full results on the ten cattle brains (five from clinically normal animals and five affected by BSE). BSE was confirmed in each case by brain histology and supported by the finding of scrapie associated fibrils in each BSE case. All studies were done blind as were your own.

In contrast your own studies aimed at detecting tubulo-filamentous structures failed to identify two brains as being from BSE-affected animals. The other animals were correctly identified.

I am quite willing to discuss the results with you but consider that the failure to identify two BSE positive animals seriously detracts from the value of your test as a practical substitute for brain histology in BSE diagnosis.

Please give me a call if there is anything you wish to discuss otherwise I feel we should consider the study concluded.

THURSDAY 18 APRIL 1996

Members present:

AGRICULTURE COMMITTEE	*HEALTH COMMITTEE*
Mr Richard Alexander	Mr John Austin-Walker
Sir Roger Moate	Mr Roger Sims
Mr William Powell	Mr Hugh Bayley
Mr Martyn Jones	The Reverend Martin Smyth
Sir Jerry Wiggin	Alice Mahon
Mr Edward Leigh	Mr Richard Spring
Mrs Ann Winterton	Mr John Marshall
	Mr John Whittingdale
	Mrs Marion Roe
	Mrs Audrey Wise

Sir Jerry Wiggin was called to the Chair

Memorandum by the Federation of Fresh Meat Wholesalers

PRIORITIES FOR THE BRITISH BEEF INDUSTRY IN THE CURRENT CRISIS (T2 BSE)

Eliminate BSE

1. Our overriding concern as an industry must be to eliminate BSE. Only by doing this will we finally restore consumer confidence at home and abroad. It follows that all proposals to this end must be both practical and supported by scientific evidence, SEAC and WHO. Our aim must be to seize the long term high ground which will enable us to develop "Super Premium Beef", and rebuild our industry for the future with confidence.

Slaughter of Cows

2. Current proposals from MAFF/NFU to slaughter cows and prime beef cattle over 30 months old, and calves, are both unnecessary and economically devastating. On the other hand, the proposal only to slaughter cows at the end of their working life and to prevent the meat from reaching the human food chain will not eradicate BSE quickly, or provide the consumer with immediate assurance that British food is safe.

3. To this end we strongly commend the slaughter of all cows born on farms with an incidence of BSE before 1 January 1991 by the end of 1997. Immediate action is needed because culling of cows will increasingly become a welfare problem.

Slaughter of Beef Cattle over 30 Months Old

4. It is both unscientific and economically unsound to destroy steers and heifers between 30 and 42 months old. SEAC and WHO have both stated that such animals boned-out in line with current regulations are perfectly safe to enter the human food chain. This measure specifically excludes the very cattle that we have been trying to encourage—extensively and naturally produced low input cattle from native (slow maturing) breeds. Forcing farmers to send cattle for slaughter before 30 months will lead to greater intensification, more bull beef, and more factory farming with increased use of supplements—and consequently more expensive beef.

Slaughter of Calves

5. Proposals for a calf slaughter scheme should be discontinued. Calves will be needed for the human food chain as manufacturing meat to replace cows lost to the human food chain. Low calf prices would have an impact throughout the beef chain and would be reflected in store prices, keeping finishers in business.

DISTINCTION BETWEEN BSE IN BEEF AND DAIRY CATTLE

6. There has been too little distinction between the beef and dairy herds. 85 per cent of beef herds remain BSE free. It is both unnecessary and unfair to consider the incidence of BSE in the beef and dairy herds together. Equally there is no distinction between young and old dairy cows. The priority here must be to remove only cows at high risk from BSE, not those which are safe.

EXPORTS

7. While there is evidence of some recovery of domestic confidence in British beef, we believe that the likelihood of reopening export markets is remote for some considerable time. The need to remove the EC ban is no longer urgent. Even if there was a political will, overseas consumer concerns will remain, and restrictions on British beef availability will make our product uneconomical overseas for the foreseeable future.

IMPACT OF MEASURES PROPOSED SO FAR

8. So long as the current import ban remains the effect of measures proposed so far will be as follows:

— The UK may actually become short of beef as we are prevented from processing all cows and beef cattle over 30 months old. Average beef cattle weights could actually fall as farmers adjust to selling animals at under 30 months of age.

— We believe that the proposed support level for farmers, either for cows or clean cattle over 30 months, may be set at a rate which will lead to clean beef animals being worth more to farmers if grown on to the age at which they can be destroyed to attract compensation, rather than sold earlier at the market price for animals entering the human food chain. Price will be a critical factor in recovering the market share of beef in domestic consumption. Any further cutback in the availability of beef animals is bound to increase prices.

— We shall immediately lose exports valued at £520 million per year in addition to the production from 700,000 cows in the current year with a value of approximately £450 million. In the first year of implementation the current measures will lose the value of 350,000 cattle over 30 months old, worth approximately £300 million. These will be replaced in part by imports worth possibly another £400 million, with an impact to the balance of payments in the current year in excess of £900 million and an on-going loss of £500 million per year.

— With 25 per cent fewer cattle going through slaughterhouses while the cow ban remains in place, we must shed 25 per cent of jobs in slaughtering, boning and further processing, unless this work is replaced by imported carcases. This means a loss of approximately 4,000 jobs in our immediate sector, people who will not be paying tax, but will be collecting unemployment and other benefits.

SUPPORT AND COMPENSATION MEASURES NEEDED

9. Loss of Stock Value. Industry must have urgent compensation for unsaleable stocks held or being returned from overseas customers.

10. Employment Legislation. There is a need for urgent decisions and a relaxation in current employment legislation related to redundancy, or immediate assistance with redundancy costs.

11. Selective Slaughter. A selective slaughter policy should remove cattle most likely to develop BSE in the immediate future. These will be from higher risk dairy herds; presumably current statistics would allow these to be targeted so that a dramatic fall in the number of BSE cases would become evident. Older cows born before 1 January 1991 should be progressively slaughtered, oldest first, so that they are all removed by 1 January 1998.

12. BSE Free Cattle. All cattle, including cows born after 1 January 1991, from BSE free herds should be destricted and should be able to be traded. For the sake of uniformity all animals over 30 months old should be boned. These animals should be properly registered by the veterinary authorities and moved only with appropriate licensing documentation.

13. Beef Premium Top Up Scheme. The market for young cattle should be allowed to fall and farmers' returns supported by a Beef Premium Top Up Scheme. Lower prices will encourage consumption of beef so that after a short period payments needed would fall away. Since there are bound to be further statements about BSE, such a scheme would create a safety valve factor for industry in future.

14. Intervention. In the short term we may require intervention measures for 30 month plus cattle while manufacturing or catering markets are developed. Intervention grades must be widened to include "O" grades for young bulls in the UK, and the top weight limit increased to 420 kgs.

15. Traceability. We must have an immediate traceability scheme with public access to the database. Meat plants must be able to develop their own traceability systems. Any national scheme must be "copper bottomed" and auditable. Such a scheme might include use of branding, electronic tagging, or passports.

16. QA Scheme. We must immediately have a total farm assurance QA scheme that is accepted by all sectors of the food chain, with the minimum amount of bureaucracy associated with it. The total removal of the spinal column should be associated with this scheme.

17. Import Measures. Beef imports should continue to be subject to the same restrictions currently imposed on British beef.

18. Canning. Immediate consideration should be given to supporting the canning of surplus unsaleable meat from either young cows or beef cattle over 30 months old which could be retorted to kill any BSE agent. Such meat could be offered for sale to the third world, or form part of an Overseas Aid policy.

12 April 1996

ANNEX

A BRIEF INTRODUCTION TO THE FEDERATION OF FRESH MEAT WHOLESALERS

Our Federation has represented the interests of fresh meat wholesalers and slaughterhouse operators of all sizes in England and Wales since 1934. Our members are at the heart of the meat chain, and as such we are the "marketeers" for the beef, sheep and pig producers of this country. We feel the pulse of our suppliers, and of retailers both at home and abroad. By investing in improving the quality of our product to meet an ever increasingly sophisticated market, the UK has moved over the last 20 years from being a net importer of meat to being a net exporter of 300,000 tonnes in recent years.

The Federation aims to ensure that members' interests are represented continuously and properly at Government and all other levels empowered to initiate regulations and manage schemes affecting the meat industry. We maintain links with other trade organisations within the meat industry, and with producer associations, with the aim of harmonising and unifying industry's response to changing market conditions and proposed legislation.

Our membership currently includes 111 meat plants and depots of all sizes, representing 80 per cent of the throughput in red meat slaughterhouses. Our members have a collective annual turnover of £2.5 billion, of which exports account for £0.5 billion, within a total UK red meat wholesale market worth £6 billion each year.

The Federation has consistently supported the EU single standard concept to be achieved by all meat plants, and we campaigned in support of a national Meat Hygiene Service in order to achieve one consistent hygiene standard and even-handed implementation of the Fresh Meat (Hygiene and Inspection) Regulations 1995.

The current crisis in the meat industry has had a devastating effect, most particularly in the export sector which is now at a standstill. The EU ban on beef exports from the UK, is having a very serious knock-on effect in the sheepmeat and pigmeat sectors for European sales, and even as public confidence recovers the effects of lost sales will be long term and very serious. In this situation, we keep our members continuously updated on developments and aim to inform and advise those making decisions on behalf of our industry.

Examination of Witnesses

PROFESSOR TIM LANG, Professor of Food Policy, Thames Valley University, MR PETER SOUL, (Grade 5) Head of Operations, Meat Hygiene Service, Executive Agency, Ministry of Agriculture, Fisheries and Food; and MR ASHLEY BOWES, President, MR JOHN BAKER, Chairman, Midland Meat Packers, and MR RICHARD CRACKNELL, Director and Deputy Chief Executive, Anglo Beef Processors, Federation of Fresh Meat Wholesalers, were examined.

Chairman

314. Good morning, ladies and gentlemen. Under the Rules of the House, I am required to declare that I am a consultant to British Sugar, which is a company associated with this and in the same group that manufacturers cattle food. Now, on to our business. Gentlemen, you all come from disparate parts of the world. Would you like to introduce yourselves, say who you are and why you are here this morning.

(*Mr Cracknell*) I am Richard Cracknell, Vice President of the Federation of Fresh Meat Wholesalers, I am also the Deputy Chief Executive of Anglo Beef Processors, beef and lamb slaughterers. We kill about 200,000 cattle a year and about 1½ million sheep.

(*Mr Baker*) I am John Baker, past President of the Federation of Fresh Meat Wholesalers and also an MLC Commissioner. I am also Chairman of Midland Meat Packers, operating probably the largest beef abattoir in the UK.

(*Mr Bowes*) I am Ashley Bowes, President of the Federation of Fresh Meat Wholesalers. I am Managing Director of Bowes of Norfolk and we are in the business of pig production and pig processing of about ½ million pigs a year.

(*Mr Soul*) I am Peter Soul and I am in charge of operations at the Meat Hygiene Service. I am a veterinary surgeon and have been working in the meat hygiene field for a number of years.

(*Professor Lang*) I am the interloper as I have nothing to do with meat, butchery or the meat trade, but my job is Professor of Food Policy at Thames Valley University. Before that I was head of a couple of NGOs, non-governmental organisations, and before that an academic.

315. We do not normally start with any statements in this Committee, but perhaps it would be helpful if certainly the lead organisation would just say a little bit about the situation as they see it at the present time and the problems which have been created for you over the recent BSE scare. Mr Bowes, would you like to start off?

(*Mr Bowes*) The situation, Chairman, in our industry has obviously been very chaotic and we have been faced with a decline of trade that is extremely hard to bear. We have, as you are aware, lost our export trade and we have here representatives of two of the major exporters in this country who have suffered very badly because of this and we have also had the effects of the retail demand that has declined and the issue of a very much changing scenario where the manufactured products have suffered the greatest decline which has been a major problem to us.

316. Mr Soul, do you see your organisation as having played a role in all this matter with particular reference to the events of last autumn?

(*Mr Soul*) Yes, we are responsible for the enforcement of the Order which requires the removal of important tissues from the carcases in addition to our normal role in meat inspection itself and meat hygiene, so, yes, we are very major players, I would say, in the events that have taken place.

317. Professor Lang, we know you have a view that is slightly different from most people's on this which is why you are here this morning. Perhaps you would like to express it briefly.

(*Professor Lang*) In general, yes, I have said all along—and indeed made a submission to the Agriculture Select Committee in 1990 saying what I still believe, that the Ministry acted extremely responsibly and very well and did all that one can do in a crisis, but that since then I think there has been a series of mistakes in terms of public management, information management, consumer sensitivity and that I think we need to learn an enormous number of lessons there. I think there is deep public concern about the smoke signals that have been sent out about BSE over the last seven years. These have constantly indicated that not quite everything is under control. At the same time very contradictory statements were being made adding to the perception of confusion. It was being said beef was 100 per cent safe and there were no risks, yet changes were being made to regulations which suggested that not everything was previously under control. This package of messages, as a psychologist, I know merely adds to confusion, adds to the uncertainty if something goes wrong. So my main criticism would be with the management of the crisis rather than what seemed to create it—the epidemiology or the aetiology of it.

318. Indeed I recall your evidence to the Agriculture Committee in 1990, and you have just said, and I understand why you say it, that you feel that the management of the announcement and so on was inadequate. Would you agree first of all that the Government have been consistently open in this matter, in other words, they have reported whatever the scientists have told them?

(*Professor Lang*) Absolutely.

319. And given that they were faced with this news, how, even with the wisdom of hindsight, would you have handled it differently?

(*Professor Lang*) I think that is perfectly fair, and I am not making any cheap jibes, nor do I think it is appropriate to do so. I think firstly what we have got to do is learn some very sober lessons with regards to the direction of the Ministry. Secondly, I have very specific criticisms which I think, or I know, are widely shared in the consumer group movement. We held a closed meeting only two days ago to discuss the implications of the BSE crisis for consumers and the NGO community. There is a very strong feeling that consumers were being lectured, were being told, if

[**Chairman** *Cont*]
you like, it was the last of the nanny state-ism, saying, "Eat this, it is perfectly safe" rather than actually meeting the concerns and the fears, irrational or not, of consumers. Just how you manage those sort of findings is another issue. And I repeat, I think you are right, the Ministry was open. When those sort of findings are given out, they need to be given out in a different way. I also think we need a different structure—

320. As given out by the *Daily Mirror*?

(*Professor Lang*) The recent ones, yes, but the Government was going to make those announcements anyway and, to be frank, I was not alone in being slightly surprised that meetings were under way with the food industry *en bloc* quite understandably, but no meetings were required or called for with the consumer organisations. Indeed there was no full meeting by the Ministry of Agriculture with the consumer organisations until two weeks to the day, after the 20 March announcement by Mr Dorrell MP. I was invited and I was one of those people. I think that this set of priorities somehow is a symptom or a symbol of what has gone wrong, the sense that the industry comes first and consumers come second. And that I deeply regret.

Chairman: I do not want to get into an argument with you, but bearing in mind that it seems impossible to keep confidential matters within government, I would have thought that open meetings, particularly with the sort of organisations you describe, were an invitation to leak the matter. Of course the history of this is that it did appear in the *Daily Mirror* the day before the Ministers made the announcement and that is a fact.

Mr Marshall

321. Would Professor Lang accept that probably the most irresponsible approach to this issue has been that of Professor Lacey who can go on television and forecast Armageddon and who has failed to give any scientific evidence whatsoever to the Standing Advisory Committee looking into the matter? Should those who claim to have knowledge not have the guts to put it before fellow scientists rather than to scare the housewife into refusing to eat British beef?

(*Professor Lang*) I think that is precisely the sort of difficulty that one gets into if you have not got a proper information flow and if you have not included the sorts of consumer organisations that you and the Chairman were being slightly disparaging about in your procedures. I think whether you like it or not you must meet the consumer half way and there has been a very strong feeling since 1990 on the subject. Indeed, preparing for today I looked up that the Consumers' Association did a poll in 1989 showing that three-quarters of a national representative sample did not trust government to manage food safety and only two weeks ago *The Guardian* newspaper paid for a similar poll and it was an exact repetition. I think purely in terms of food policy, my area of expertise, I have to say that something has gone wrong there, so you are right. I think these sort of excessive extrapolations which have been made by some people maybe have not been warranted, but at the same time epidemiologists would say that one should always have a high case and a low case scenario and I think Professor Lacey always gave the high case scenario, which was perfectly legitimate, but the low case scenario was not given with the same verve.

Mrs Roe

322. Chairman, I would like to put a number of questions to Mr Bowes and his colleagues and at the same time I would put them together and then perhaps invite Mr Soul and Professor Lang to comment on the responses that are received. Can you explain how, after all the publicity that has been given to the SBO ban and all the problems that BSE has caused the British beef industry in the past, that as late as December last year some slaughterhouses were found to have not been implementing the SBO ban to the satisfaction of the State Veterinary Service? How is it that since the summer of 1995 there have been 21 instances detected in which pieces of spinal cord have not been properly removed from the carcase? How likely is it that this problem has been ongoing since the SBO ban was first introduced and that SBO material has been entering the food chain? Regardless of whether this material is or is not infected, the law states that it should be removed. Why have some slaughterers found it difficult to comply with the legislation?

(*Mr Bowes*) Chairman, if I may start on this point, the instances that were found of the spinal cord actually, however undesirable it might be that some are found, number one, we have the Meat Hygiene Service in place in all our plants to inspect our animals also and so there is a system in place where checking the individual plants works, but when one considers when we saw the Minister, I believe it was in November, that he told us that four pieces, the smallest pieces of spinal cord had been found in the previous month and when one considers that possibly during that time there were 250,000 cattle killed, the percentage was indeed very small. Unfortunately, although we do our best, we are not perfect and one can only compare us, I believe, properly to the medical profession because one can see that mistakes are occasionally made in the medical profession also and we as an industry do actually also have to comply with the highest standards set by our retail friends and I can assure you that when you are serving the big supermarkets, et cetera, and retailers in this country, the standards are extremely high and compliance is expected at all times, but the occasional mistake unfortunately cannot be avoided. I would like to hand over to the actual operators of the beef.

(*Mr Baker*) Chairman, I can only really speak from experience of my own plant obviously and in our business it is one of those things that we do not visit one another's plants very often, so speaking of our own plant, we have always taken the spinal cord out even before the SBO ban because it is part of the our own dressing specification and in actual fact it does help to keep the quality of bone in beef to take the spinal cord out and most specifications that we have had over the years have asked for it to be taken out anyway.

Chairman

323. Would that be general in the industry?

(*Mr Baker*) I certainly think so in the bigger plants. I would not like to comment on the smaller ones.

324. So you were doing this before 1990, before the ban came in?

(*Mr Baker*) Yes, ever since I can remember, Chairman, but, as Mr Bowes said, there is human error in this as there is in other things. We had an inspection back in the summer of our own plant and at any one time we would have around 2,500 sides of beef hanging in chillers and I believe that when those sides were checked on a particular day, I believe they found one piece of spinal cord in one side which was just about an inch long and when you consider that most spinal columns are about seven or eight feet long, okay, we admit it should not have been there, but there is human error and it had been passed by the Meat Hygiene Service. We have put a lot more stringent controls in since. It is part of all our employees' responsibility now in any part of our system, whether it is in the boning room or loading bay or whatever, if it is seen or found, to be reported to a supervisor immediately and, as far as I know, we have not had an instance since, but all I can say is that there is possibly a human error problem which has come about or some mistake.

Mrs Roe

325. Would Mr Soul please comment?

(*Mr Soul*) I think that the point Mr Bowes makes is very important, that we have to be very careful when we are using statistics and really question what exactly do they mean and do we know what they mean. I think if you look at these 21 failures over a period of nine months and you look at the throughput of cattle during that period, then the failure rate comes out at something like .001 per cent, so I think we have to have that in mind initially. The other point I think that is important to make is how do you prioritise the work that you are doing and we must not forget in all this that there are many important activities going on in slaughterhouses and that the hygiene of the product and the meat inspection of the product is also very, very important. As Mr Bowes has said, we must not of course belittle the importance of a failure to ensure that all traces of specified bovine material are removed and if some spinal cord is left behind, then potentially that is a serious failure, so we need to put a lot of resources and a lot of effort into ensuring that it is done to the maximum possible extent. Since we have been made aware of these findings by the State Veterinary Service audit, of course we have tightened up in the area and we have focused much more clearly on the SBM rules and we have put a lot more effort into training people, into putting in more inspection resources and in particular I would say in getting the vets who are also involved to look much more closely at that aspect of the enforcement role that we have.

326. Professor Lang?

(*Professor Lang*) I think your question is absolutely right. I think there was a deep shock even amongst seasoned observers of the food and the consumer scene at the MAFF findings from those spot checks. I think the enforcement failures basically added to consumer scepticism. We know that the focus of your inquiries now and six years ago is a very complicated area of science. As far as the consumer level of debate is concerned, either the continued high incidence of BSE shows that there is a failure due to leakage or that the SBO is not being properly enforced or that the theory of the disease's aetiology is wrong, which is contentious stuff, I know, but a possibility, or there is maternal transmission. Eight years on, to have a failure of enforcement coming to light, sent all the wrong smoke signals to consumers and merely added to the scepticism and the unmanageability of consumers. I thought it was very honest, to go back to the Chairman's point, of MAFF to release the figures on those spot checks, but you have to see it from the point of view of the taxpayer and the consumer, that they merely added to their scepticism.

Alice Mahon

327. I wonder if you would like to comment on the recent press report, in the *Mail*, by Lorna Duckworth when she said that Britain's abattoirs were at the centre of a row over hygiene checks and that slaughtermen were refusing to pay fees to the Meat Hygiene Service for the last year to replace the local authority inspectors and slaughtermen, in this report, are alleged to be angry that hourly fees have increased from 15 perhaps to 35. The article then goes on to say that foreign vets employed on the cheap failed to check that offal parts thought to carry BSE are removed and abattoir bosses, and I am quoting from the article, claimed some vets struggled to speak English while others have dealt only with pets. Would anybody like to comment on that?

(*Mr Cracknell*) I can comment on that from the meat industry side. There has been an ongoing struggle, as there always is over any costs, to contain the cost of the Meat Hygiene Service and it has been a new service this year and was instigated very much at our request because we wanted one common standard throughout the country instead of 300 different standards that we had in years gone by and it was introduced by local authorities.

328. They did not have a single standard that they had to abide by then, the local authorities? Surely there was something set down?

(*Mr Cracknell*) There was a single standard of meat inspection, but clearly it was being provided by 323 different local authorities so throughout the country there were variations in application and there is no doubt that we have now since April 1995 had a very much more uniform application of rules and regulations, but there was a row. The facts are that certain of the smaller slaughterhouses have actually withheld payment of meat inspection charges, I think in our view quite unjustifiably because it is actually the farmers who pay half the meat inspection charges and I am advised that they still deduct those contributions from the farmers, so I think the row that you refer to is a storm in a teacup. Whilst there is plenty of anecdotal chat of the foreign vets who cannot speak English, I personally operate

[**Alice Mahon** *Cont*]

eight abattoirs and I have never come across one, apart from maybe the odd Irish one or Scottish one and I have difficulty understanding them.

329. You know of no slaughtermen who are going to challenge the charges, for example, in the European Court, it was said?

(*Mr Cracknell*) Certainly some of these smaller abattoirs are challenging the charges on the basis of, well, I am not sure what basis, I am not personally one who subscribes to that group of abattoir operators, but certainly they are being challenged by in our own courts and in the European Court.

Chairman: I am not sure how entirely relevant it is, but I think I can answer the question because it is to do with the subsidy of meat inspection charges elsewhere in Europe and that is a difference of opinion.

Alice Mahon

330. Well, it is relevant insofar as if the job is not being done properly because there is a question of the charge. I wonder if anybody else would like to comment on that.

(*Mr Soul*) I should point out that the dispute about charging is something that has been going on for quite a long time and before the Meat Hygiene Service came into place. You raised a number of points and one of them was about the dispute over the charges and costs and so on, but in fact overall I do not think the Meat Hygiene Service, I am certain the Meat Hygiene Service has not cost more than the previous system. In fact I am confident that it has managed to deliver a more efficient service at less cost and the rates were fully discussed with ministers and agreed before they were passed. You made the point about Spanish vets. Did you say Spanish or not? I am not actually sure.

331. The slaughtermen said foreign vets.

(*Mr Soul*) They all have to be qualified to veterinary standards and they all have to be members of the Royal College of Veterinary Surgeons. Not only are they fully qualified, but they undergo a training course in this country before they are authorised as official veterinary surgeons.

Audrey Wise

332. The Agriculture Minister called in slaughterhouse operators, I understand, on at least two occasions at the end of last year to impress upon them the seriousness with which the Government regarded failures to observe SBO controls and I am not surprised as I would hope they would regard it seriously since in a parliamentary answer Mr Hogg disclosed that on the spot checks, 48 per cent of the abattoirs were found to be failing in handling of specified bovine offal and 65 per cent of knackeries and hunt kennels were failing to comply with the rules for handling specified bovine offal. Now, it has been suggested that these are small infringements, not terribly important, but I must say that 48 per cent strikes me as a remarkably high percentage to come up on unannounced spot checks and it certainly caused me considerable worry. Now, what procedures have been put in place to eliminate non-compliance now?

(*Mr Soul*) I think we should perhaps start out by pointing out that compliance with the SBO Order, as it was then, is the responsibility of the occupiers of the slaughterhouses and the responsibility of the Meat Hygiene Service is to enforce that Order, but it is quite clearly then the responsibility of the occupiers to ensure that all the specified bovine material is now removed.

333. Is it not your responsibility to make sure that they are complying?

(*Mr Soul*) That is right, yes.

334. So if they are not complying, then the buck stops with you, does it not?

(*Mr Soul*) Yes, we have the responsibility to carry out the enforcement and I think we have already touched on this point earlier, have we not, about the use of statistics and about the priority that you give to different areas of the work and I think it is important that we do not lose sight of all the activities that the Meat Hygiene Service is responsible for enforcing in slaughterhouses. As regards the use of the statistics, you mentioned 48 per cent, I think. I think you have to be very, very careful because what does that actually mean and what were the failures that were being identified? If, for example, it was a failure of slightly inadequate staining of the material, that would go down as a failure. If it was not quite keeping to the correct records, that would go down as a failure. There are a whole series of things which are identified as failures, but which are relatively minor and in terms of enforcement are the sort of things which one could not take before a court and expect to achieve a successful prosecution, but I would not wish to belittle some of the things which are important. I think again earlier we have said that we believe that failures to remove the spinal cord, for example, is a significant failure. I gave you a statistic on that and I believe that the results suggest that the .001 per cent of failures to remove the spinal cord has been identified in the last nine months. I would not wish to belittle that. It is something which we treat very, very seriously and since that was brought to our attention, we, the Meat Hygiene Service, have increased the enforcement effort in that particular area very, very much indeed.

335. I have forgotten that I ought to declare that I might be held to have an indirect, non-pecuniary interest inasmuch as I am President of the Union of Shop Distributors and Allied Workers and I had forgotten to disclose that to the witnesses and those present. I was reminded of my omission by your statement which amounted to the old excuse of, "Well, it is only a little one". I have known members of my Union threatened with losing their jobs for eating a sweet and it being held as theft and "It is only a little one" is not always held to be a valid excuse. When it comes to bits of banned material being found in places that they should not be, then I must say I am not impressed with the excuse. Forty-eight per cent seems to me to be a considerable statistic and it is not mine, but it is the Ministry of Agriculture's statistic, and nor is it mine, the fact that when I asked him on the question of prosecutions, when I asked

[**Audrey Wise** *Cont*]

him how many, he could not suggest even one and I did press him a bit, but he said it was your responsibility and that you were looking at a number of cases to see if you could find sufficient evidence to justify bringing criminal cases. Of course it is serious to bring criminal prosecutions, but it is serious to infringe bans and regulations made for public health. Can you tell us the results of your investigations? Were all of these 48 per cent infringements only "little" ones?

(*Mr Soul*) Chairman, I was not quite clear who you asked or who told you about the prosecutions that have taken place so far.

336. Mr Hogg.

(*Mr Soul*) We have launched as a consequence of raising the profile, if you like, of SBM controls six investigations with a view to prosecutions. There has been a preliminary hearing in one of those cases. Another case has gone to the Procurator Fiscal in Scotland. One of the investigations so far has been rejected as not being viable for prosecution and three cases are still under investigation.

Chairman: I think that is as far as we can probably go on this, particularly if they are *sub judice*.

Audrey Wise

337. Yes, you have given me the information there that I wanted and I do not want to press that particular point any further. I want to go back then to my question about what procedures have been put in place to ensure better compliance because in any case of course even prosecution is locking the stable door a little late, so what are you doing to ensure better compliance?

(*Mr Soul*) Do not forget that we are a very new service and we only came into existence on 1 April 1995. When this problem was brought to our attention we immediately instituted a number of measures in order to ensure that enforcement was carried out more rigorously. Those measures included training of the staff, providing the staff with additional information. It included extra inspection resources in some plants, extra meat inspection time. It included very clear instructions to the staff on how they should carry out their enforcement role. It included additional visits by the Principal official veterinary surgeons who have a group of plants under their responsibility and additional checking by the official veterinary surgeons who are presently in every slaughterhouse. Now, we have a particular problem in the enforcement area because if the meat has been inspected and health-marked by our staff, then we are advised that we cannot take that case forward for a prosecution. That means that we have had to carry out formal disciplinary procedures against those members of our staff that have health-marked carcases where spinal cord has still been present because, as you have said, our role is enforcement, the buck stops with us and we have to inspect the carcases to make sure that all the specified bovine material has been removed and if we fail to do that, then the steps that we have to take then are formal disciplinary steps against our own staff and a number of them, I think 22 of our staff, have undergone formal investigations with a view to disciplinary measures. One member of our staff has actually been dismissed, I regret to say, and a number of staff have had formal final warnings issued to them. You see, the problem for us from an enforcement point of view is that if, despite their best efforts, they fail to spot tiny pieces of spinal cord in a carcase and they put a health mark on it, then the only steps that are left open to us in fact are to discipline our own staff.

Audrey Wise: I think you might be asked about the number of meat inspectors later on, so I will not do that, but I would like to ask that you supply the Committee, if you have not already, with a list, a more detailed one, and I am grateful for the list of measures and obviously you cannot go into any more detail in oral session, but I would like that put into writing for us with a clear comparison with the previous procedures so that instead of more training, what training they did have and what they now have and, likewise, with information and, likewise, with numbers, how many did it used to be and how many is it now, that would be a great help.

Mr Bayley

338. Mr Soul, you have referred a number of times to us not drawing too severe conclusions from the fact that tiny pieces of spinal cord were found, but in Mr Hogg's parliamentary answer of 22 November when he gave those figures about the 48 per cent of slaughterhouses failing to abide by the regulations in handling SBOs, he said that the pieces of spinal cord ranged, and I quote, "in size from one centimetre to one-third of the spinal cord", so in some cases they were small, but in other cases large. I wonder if I could ask you to qualify or to give us some more information. You said to Mrs Wise that since this announcement was made in November last year, additional inspection resources and time and visits have been made available to the Meat Hygiene Service. How much additional time has been made available? How many additional inspectors are available and how much extra time do they have available to inspect carcases?

(*Mr Soul*) Chairman, I have already undertaken to provide the Committee with details of the additional training and the additional resources that we put in.

339. But it is specifically the time, the number of man hours that you had in November and the number of man hours that you have now, or person hours, I should perhaps say, amongst your inspectors and vets.

(*Mr Soul*) There are two areas here. One is that following the Minister's announcement, additional resources were put in. Following the announcement on 20 March, yet more resources have been put in. Now, I am sure that we can provide you with the details of the man hours, the additional man hours that have been put in on both those occasions, but I do not think I am able to give you that sort of figure now off the top of my head.

340. I would be grateful for the figures.

(*Mr Soul*) Indeed we will provide them, but could I just say that following the announcement by SEAC, it has been agreed now that an additional two meat inspectors will be available in every full throughput beef slaughterhouse.

[**Mr Bayley** *Cont*]

341. Of which there are 200? How many are there in the country?

(*Mr Soul*) There are just over 400 beef slaughterhouses in total. I do not think I can break those down into full throughput and low throughput.

342. I am told there are 195 low throughput, so if you are going to have two extra inspectors in each of the full throughput slaughterhouses, you will have something like 450 additional inspectors. Is that right?

(*Mr Soul*) No, the total additional meat inspector resource is going to be of the order of about 300 altogether.

343. And that is on top of roughly 500 at the moment?

(*Mr Soul*) In red meat, yes. There is one other thing, Chairman, if you will bear with me. The two additional inspectors in the full throughput plants and additional inspectors in the low throughput plants are to follow SEAC recommendation which is that there must be constant supervision during the dressing of beef carcases to ensure that all the specified bovine materials are removed.

344. What does constant supervision mean? Does it mean that the inspector watches as your men chop up the carcases?

(*Mr Soul*) It means that the meat inspectors will be present all the time that the dressing of carcases is taking place and that they will be supervising the dressing procedures.

345. Could I ask the meat operators what that means from your point of view? How many people do you have cutting up carcases in an average—well, I do not mean an average abattoir, but if one excludes the small abattoirs which deal with a few hundred carcases a year, but if you talk about an abattoir dealing with an average of 12,000 or 14,000 carcases a year, how many abattoir workers would you have dressing the carcases and how many inspectors will you have and how do they work together as a team?

(*Mr Cracknell*) Can I answer that question. If I could change the throughput to 50,000 cattle a year, which is roughly 1,000 carcases a week, we would need somewhere like 18 people involved in the actual slaughtering operation from the lairage through to the final wash. There would be a team of ancillary people involved in offal removal, et cetera, and we would typically have, depending on the layout of the abattoir, two, three or four meat inspectors, plus an OVS supervising that operation.

346. That is a big abattoir?

(*Mr Cracknell*) Yes, okay. It is what I would describe as a full throughput abattoir, I would think, so there would be a ratio of three or four meat inspectors to 20 of our operators and that has been increased in those abattoirs by a further two in the last four weeks.

347. And at what point do they inspect the carcase? Do they inspect it while the job of butchering is going on?

(*Mr Cracknell*) This is a moving line operation, if I can describe it to you, and normally there would be one meat inspector inspecting the carcase itself, there would be one looking at the offals, including the head, and there would be one looking at the intestines and then there would be an OVS whose job is partly the ante-mortem inspection, but he would be supervising the post-mortem inspection as well.

Audrey Wise

348. I should have gone on to ask as well whether there is anything else which needs to be done in order to ensure that specified bovine offals do not enter the human or animal food chains. Are you satisfied that everything possible is being done or is there anything else that can be suggested by any of our witnesses?

(*Mr Soul*) We have the recommendations by SEAC and those are now in place. We have provided these additional resources that we have talked about, so they are now in place. We have been able to do that because the industry is operating at a low level at the moment, but we have plans in place and we are already recruiting so that we will be bringing in extra staff as we anticipate the production in beef slaughterhouses goes up and we will be able to maintain that level of inspection resource input in plants. Now, I believe that SEAC are in the best position to make recommendations as to what controls are required. We have implemented in full the SEAC recommendations.

Chairman

349. I think that it is just worth observing that the recommendations from SEAC are based on the assumption that maybe every beast has some infected material in it, whereas the actual practical reality is that it is in very, very few animals, bearing in mind that they are inspected before they leave the farm and bearing in mind that they are inspected in the lairage before slaughter, so your odds of .001, whatever it was, is actually multiplied by the chance of their being an animal within a few days of developing the final phases of the disease because it is only in the last few days that the infected material is invasive, so the odds are actually so long that they are virtually negligible and if, on top of that, you then remove all the material, it is fair to say that, as chances go, it is about as long as it could be that anybody will eat infected material as a result of these inspection failures. Would you agree with that, Mr Soul?

(*Mr Soul*) From my position, I see that as being absolutely correct, yes.

Audrey Wise

350. There was a good deal of certainty in that. I would point out that it may be a few animals, but the whole problem is we do not know which animals and I am also very surprised at the certainty of the assertion that it is only in the last few days that there is a danger. I do not think that we have actually had scientific evidence to that effect and again I would think that this is something that amongst all the don't knows, it is another don't know, but I accept the answer fully, that it is primarily SEAC's responsibility to lay down the regulations. I wanted to know from your point of view if there was anything else that you needed and you have answered that. I would like to ask our trade witnesses again on this matter of compliance and implementation, in

[**Audrey Wise** *Cont*]
view of the fact that this is clearly putting a more exacting task on you and your staff because although you did some things voluntarily, it is a little different when you are doing them not voluntarily, but under threat of criminal prosecution, but obviously it is a harder job for all of you and can you tell me whether you are giving your staff extra training to meet these new exacting requirements and whether they are given more time to do the job, whether you are employing more people? We have heard about more inspectors, but are you employing more people so that they have more time to carry out these procedures? Would you like to tell us please?

(*Mr Baker*) Chairman, first of all, the checks that actually go on in the abattoir are only the initial checks. There are further checks, and again I am speaking from my own experience, there are further checks that we have right down the line and do not forget that the majority of this meat, or all of it at some stage or other, but the majority of what we slaughter is actually boned out in our own boning rooms attached to the abattoir and those bones are taken out and they are taken away and rendered, so any instance of spinal cord being left in the vertebrae channel would never get into the food chain anyway. We have tightened up the procedure of the slaughterhouse to the extent that we actually weigh and collect all the material in one single container each day or in a number of containers a day. This holds the spinal cord. That is all weighed and checked to the average weight of whatever we would expect to get off the number of animals we slaughter. We have put a lot more checks on our own procedures, yes.

351. Yes, but my question also was about more training or more staff because it clearly is a more exacting job. Every time you say, "Well, it does not really matter because it is not going to have any bad consequences" then I fear you are hitting confidence. If you could tell us that you had employed more staff or they have had more training then that might do something to restore confidence, but can you tell us that?

(*Mr Cracknell*) Sadly, since the SEAC report clearly all our plants have been working at very much reduced throughput.

352. No, I meant before that in order to comply. I did not mean just with this crisis. Obviously I understand you are not going to take on extra workers at this minute. I meant since the regulations came out originally, did you do extra training, did you employ more staff, did you give your staff more time? That was my question.

(*Mr Baker*) There certainly has been more time. More staff have been allowed into the slaughter operation to make sure that it is taken out. As far as training goes, our own company probably has the best trained meat technicians in the country and they are thoroughly trained anyway. It is just the fact that we have tightened up the procedures.

Chairman

353. I think we must move on now.

(*Mr Bowes*) Chairman, we as a Federation have always supported that if there are malpractices government should prosecute.

Alice Mahon

354. Since the BSE problem was first recognised, critics of Government policy have argued that in the necessarily messy environment of an abattoir, the complete separation of SBOs and material for human consumption is practically impossible. Do you agree?

(*Mr Bowes*) I would say that you are wrong in your statement. It is possible to keep SBO material separate, but at the same time one must consider how you actually take it out of the animals, so that all depends on how far one argues backwards. It is quite possible to keep all this material separate. Mr Baker is in a better position to answer than I am.

(*Mr Baker*) There is no problem at all with that procedure, particularly in the medium to high throughput plant. I cannot answer for the smaller ones, but we have no problem at all in keeping the material separate.

(*Mr Cracknell*) I think we would challenge that it is a necessarily messy environment. Our workforce and ourselves are proud of what we do. We do it carefully. We do it diligently. Our staff know their jobs are on the line just as much as ours are in all of this. These abattoirs are not what they were several hundred years ago.

355. Do you believe that it is possible, in modern mechanised meat cutting plants, to saw a carcase in half without contaminating other parts of the carcase with bits of spinal cord in debris from saws and other equipment?

(*Mr Baker*) Obviously when you saw anything you actually end up with a slight dust, but the modern saws nowadays have washers on them that wash away as you go down the sword and the carcase and it actually washes most of that material away off the bone as well as it goes down.

(*Mr Cracknell*) I think "absolute certainty" is a phrase that has been used a lot in this recent crisis. I would stress again that the removal of the spinal column in the abattoir is the first of many checks. We then remove the whole of the vertebrae in the boning hall which also is specified offal, and that is the second check. Then there are a number of other checks to make sure that this material cannot get into either the animal food chain, which clearly has been the problem in the past, or into the human food chain, which I do not think it has. But it is absolutely true that none of that material through the series of checks will get into either of those food chains.

Mr Spring

356. I would like to ask Professor Lang a question. Hearing what Mr Cracknell had to say just now about the processes and checks in the abattoirs, what we are really looking at here is a question of confidence in safety and that is the bottom line with all the implications that flow from that. We have

[**Mr Spring** *Cont*]
heard about the measures that are in place or are about to be put in place. Are they sufficient, if rigorously enforced, to ensure that the safety question is adequately addressed so that people can have confidence that British beef and beef products are safe? From your point of view, I wonder what your observation is.

(*Professor Lang*) I agree with you. Like everyone, I am fascinated by the minutiae of the details of butchering practices and whether some micro amount—a milligram—of some infected material is left behind. I do not think that is the issue at all. I think you are absolutely right, I think the issue has to be seen more upstream rather than downstream, namely the point at which a consumer goes out and chooses whether to buy poultry or Linda McCartney's latest veggie burger or red beef. At that moment these minute bits of doubt suddenly become a trigger that can push someone from one product to another product. I frankly do not see these issues—the details of butchery—as particularly relevant any more. That is why I said earlier in answer to the Chairman that I think MAFF responded rightly and very well in managing the crisis back in 1987/88 but since then went astray by putting all of its collective efforts into saying that beef is 100 per cent safe when even Tyrrel in 1989 did not say anything as categoric as that. There has been a split between the hundred per cent certainty people and those who say there is some degree of uncertainty. That chasm is what has now widened. I am not certain that even toughening up on beef regulations in the way that the previous questions have been pointing to—welcome though that would be for the epidemiologists—would deal with the psychology of consumer confidence. I think a completely different line of questions is needed to address that. The sort of things we need to recognise are that ultimately time is going to be the only healer in this. Even Professor Gadjusek, the Nobel Prize Laureate in this area, in a paper in 1985 was saying one has to talk in spongiform encephalopathies of a 30-year period of incubation potentially. I think the public now knows that. No amount of tightening up of the regulations is going to alter that uncertainty for the next 14 or 20 years. Even if MAFF and Mr Hogg, repeating what Mr Gummer and Mr Dorrell said back in 1989 when he was a Junior Minister of Health, say that beef is 100 per cent safe, I do not think the public now believes that view. That is why I quoted earlier an opinion poll of only two weeks ago. You are essentially having promises of certainty put into a situation of uncertainty as far as the consumer is concerned. I do not think that tightening up slaughter practices is going to resolve the confidence problem. I think changes at MAFF, changes in the direction of national food policy, reforms in how the Ministry of Agriculture deals with consumers are what are needed. Frankly, the Consumer Panel, that Mr Gummer in my view very honourably set up in 1989, and the Quarterly Meetings with chairs of consumer organisations have all been seen to be complete charades. They were not even called back in this crisis. All the signals that have gone out to consumers, even allowing for a fanning of those smoke signals by some people, have essentially not done anything to assuage the doubt as far as the consumer is concerned. The issue now is the psychology of risk. There is a burgeoning literature by academics that I would recommend the Committee spend some time looking at.

357. I think Members of Parliament know about the psychology of risk.

(*Professor Lang*) I am not certain that is true, with respect.

358. Thank you for your answer. I think one can make this observation: the consumer appears to be making a judgment at this moment actually, despite what you are saying. I would like to ask Mr Soul essentially the same question in the sense that with these changes that are now being implemented, from your perspective, although perhaps this is part of a whole block-building process of confidence, but at the area of inspection of abattoirs, of the whole process involved in beef production or meat production, are you satisfied that there is nothing more that could be done to ensure the highest standards in this particular process now?

(*Mr Soul*) Chairman, I would have to come back again to the SEAC recommendations because I believe that they have had ample time and they are the experts in looking at the science surrounding this extraordinarily complex subject. I do not claim to be an expert in the spongiform encephalopathies, but I believe our role is to implement the SEAC recommendations and I believe that we have done that in full. We are taking measures to ensure that if and when the beef industry does start gearing itself up again we will continue to be able to implement those recommendations in full. I believe that the public should have confidence in the scientists and when they understand that the recommendations of the scientists are all being carried out then that should give the public confidence.

Mr Leigh

359. I am sorry, Professor Lang, I have got to put this point to you, I think your comment you have just made that Ministers such as Mr Hogg and Mr Dorrell should have been casting doubt on the safety of British beef is absurd and irresponsible.

(*Professor Lang*) I did not say that.

360. You seemed to be implying that. I really do think this is ridiculous. Imagine if Mr Dorrell or Mr Hogg had actually in the last few weeks suddenly expressed doubt about safety or had behaved in any different way, it would have fanned the flames of this crisis into a holocaust which would have ended up in the mass slaughter of our entire national herd. There is not one jot of scientific evidence that that is necessary. There have only been ten cases. There is no firm evidence that they are linked to eating beef. For you to come to this Committee and repeat again and again that since 1989 and 1990 the government should have been throwing doubt on the safety of our beef I think is absurd and I think it is irresponsible.

(*Professor Lang*) That is not what I said, Mr Leigh. I am glad you are giving me the opportunity to clarify that. I will repeat it in case you were not here.

361. I was here for the entire session that you have been saying this again and again.

[**Mr Leigh** *Cont*]

(*Professor Lang*) Forgive me. I said that I thought that the Ministry had dealt very well with the crisis in the early stages, and I repeat that I want you to know that. But I do think it was irresponsible of the Ministry to give categoric assurances in an area of public health in which there was huge uncertainty. There was a new epidemic—I am sorry, it is true—a new epidemic, new animal diseases, indeed a new form for humans is now being talked about. To start saying that Ministers giving categoric assurances was the right policy to take is wrong. It was a serious policy mistake in relation to the consumer. I am sorry, it was.

362. What would have happened to our entire beef industry if Mr Hogg had said, "I don't know. I am not really sure"?

(*Professor Lang*) Look what has happened now, it has destroyed it.

Chairman: Order, order. I think this exchange has gone far enough.

Mr Bayley

363. I would like to put a question on a different point to Professor Lang. You were saying earlier that from your point of view the real issue is how the public feels about beef. One of the things that may have undermined confidence is the fact that it is the same ministry that regulates farming in the food industry and acts as the protector of food policy for the public. I know you have had things to say about this in the past. Do you think that a separation of a food standards agency from the Ministry of Agriculture would help to restore confidence? Do you think it would make sense to follow the American model of having a food and drugs administration that would draw together the work of the committee on the safety of medicines and the issue of quality of food standards? Is that a model that we should face up to? Should it be entirely separate from all government departments if you were to go down this road, or should it, under the American model, have a responsibility to the Department of Health?

(*Professor Lang*) I think that is right. It gets back to my answer to Mr Leigh's question. I think public confidence is to do with the mechanisms of government. Let us face it, there is a crisis of confidence in governance here which includes us all. This is a disastrous situation to have put the beef industry into. I used to be a beef farmer so I have deep sympathy with it. They have been made victims. Rather like consumers, the label on the feed did not tell them the whole story. Now their industry has been destroyed in all sorts of long-term ways, or seriously damaged. I think you are right, I think we need in this time of crisis to start looking to long-term solutions. It is always unpopular to look at long-term solutions when you are wanting to prop up a beef market or prop up a textile market or whatever is in crisis. I also happen to agree with the thrust of your question, Mr Bayley, in that I think we need to separate better regulation from sponsorship. I think MAFF—the Chairman knows this from when he was the Junior Minister only too well—often has fraught complications from promoting the interests of an industry and at the same time having to address public health and consumer regulation issues. I personally think that we need to look very carefully at the experience in other countries. A lot of people are currently talking about an FDA, a food and drugs administration. I personally have looked at that system for a decade now. I think initially it is very attractive, but if you look at the United States, they could have had the same sort of problems as we are now in. I am not certain an FDA clone would have resolved this problem. I think much better models, funnily enough, are being explored at the moment in Australia, where from 1990 they have set up a National Food Authority which I think is much more interesting and much more consumer orientated, much less fraught with some of the problems in the FDA that we do not have time to go into. And also the Swedish experience is another interesting model and the Norwegian experience. I would urge the joint committee, perhaps on another occasion if not now, to explore what the differences in this separation of sponsorship from regulation could make to a problem like BSE. I think as far as the consumer is concerned it would make a huge difference because there would be some confidence that when a food standards authority or national food council spoke, it was not necessarily representing the interests of production. That has been a problem in pronouncements from MAFF. MAFF is seen rightly or wrongly as representing the producer interest. That then means that its pronouncements are treated with contempt even when they are true.

Chairman: This is getting a little bit off the subject. We have had Professor Lang's view in detail.

Mr Marshall

364. Professor Lang, you would accept that neither you nor I are scientists and if we were to seek to disagree with Professor Pattison we could produce no good scientific reason for doing so. Therefore, it would surely be irresponsible of you, I, or the Minister of Agriculture to doubt Professor Pattison's statement that British beef is safe. If you disagree with that, if you say that you are uncertain you have to produce reasons and you or I cannot produce one reason why the Minister for Agriculture should have doubted the scientific advice he was given.

(*Professor Lang*) I think that is a very good and sensible question. However, I think the experience is somewhat clouded. Back in 1989 when the Southwood Committee was set up, five people, specialists in encephalopathies mostly, neuro-pathologists, were on it. Many of us at the time said there should be specialists from public health and consumers represented on that committee. I think one has to now say that ever since Professor Pattison was appointed—and I noticed you used him rather than Dr Tyrrel—who seems to me and to many people a deeply honourable man the scientific net on the SEAC committee is being spread more widely. It now includes micro-biologists like Professor Almond and public health specialists and epidemiologists. When the net has been drawn more widely through the scientific pool of expertise you notice that the announcements from the SEAC committee have been somewhat less than 100 per cent certain.

[**Mr Marshall** *Cont*]
Frankly, I do not think this shift came as a surprise to anyone from the area of food policy. MAFF has suffered far too often and far too long from making categoric statements that are seen—and I repeat seen—to represent or to give the answers that vested interests in production want. That merely increases consumer scepticism.

Mr Alexander

365. I would like to go back to the question of the effectiveness and perception of the SBO ban. I hope this may be the last question on abattoirs, but it is one which the Committee would like to have done. If we had had the time I think we would have liked to have visited an abattoir and a meat plant and seen just what does go on. Some colleagues may have, I have not. If we were to visit an abattoir or a meat plant today or tomorrow, what would we physically see going on there which we would not have seen before the SBO ban came in just over six years ago?

(*Mr Cracknell*) Clearly the SBO ban has removed from the animal and human food chain a number of organs and a number of parts of the carcases which traditionally went into the ordinary by-products process. You would not see a lot of difference. In my experience no one ever used brains or spinal columns as food, and I have been in the meat industry all my life. They have never been incorporated. The only physical differences are that parts of the carcase, which now include the whole of the head, including the cheeks, the spleen, the spinal column, of course, and the glands are now put into separate containers. They are stained and in certain cases they are weighed and checked and there is a great deal of attention paid to that part of the operation which in 1989 all went to ordinary by-products.

366. Is oxtail part of the SBO and if not, why not?

(*Mr Cracknell*) Oxtail is not part of the SBO. Mr Soul would be better qualified to answer that.

367. Before he answers, it is part of the spinal column, is it not?

(*Mr Cracknell*) I understand that it is not. I am not a veterinarian, but I understand that it is not.

(*Mr Soul*) The oxtail is not SBO or SBM as we now have it because the spinal cord ends at about the level of the beginning of the sacrum and becomes what is called the cauda equina, which are the nerves which then go to the nether regions, if you like, so they are similar to the nerves that leave the spinal cord all the way down the vertebral column and go off to different parts of the body.

Mr Whittingdale

368. Can I turn to the enforcement of the controls which we have already talked about to some extent. I think to begin with I would like to direct my question particularly to Mr Soul. You have already told us something about the resources which are available to the Service, particularly in answer to Mr Bayley. I do not want you to have to repeat some of the answers you have already given. Our present understanding is that you have 509 full-time meat inspectors and there are 39 full-time official veterinary surgeons who inspect premises. Could you just make some comment about the ability of that number to carry out all the checks that are necessary to ensure that SBO material is excluded from a food chain? How does that number compare to the situation prior to the establishment of the Meat Hygiene Service when responsibility lay with local authorities?

(*Mr Soul*) If I can take the last point first, it is extremely difficult to compare the numbers of meat inspectors working for the Meat Hygiene Service with the numbers that were working for the local authorities. It has proved extremely difficult to obtain that information. You are not comparing like with like. In many cases meat inspectors working for the local authorities would be doing other jobs in addition to meat inspection and might only be spending an hour or so a day, or even an hour a week, doing meat inspection. I am afraid I cannot actually help you with a direct comparison of the number of inspectors that we have now and the number that were working for the local authorities. You asked whether I believe that the resources we have available to us are sufficient. At the moment they are because the industry has turned down so much, but we are confident that when the industry gears up again we will need a lot of additional meat inspectors. We believe we will need something in the region of about an additional 300. We are actively looking at qualified meat inspectors that we can recruit or use on a casual basis in order to ensure that we can provide the resource that we believe we will need. Despite that, we think that we will not be able to get enough and so we are also looking at a role for a meat technician who would not be a qualified meat inspector but who we would put through a special training course so that they are able to look specifically at enforcement of the SBM Order. We have got agreement with the relevant colleges and the meat inspection associations and the veterinary associations to proceed along this track and we have made arrangements with[1] the colleges, to have these courses available. We are already advertising for people because we want to be absolutely confident that we will be able to provide the inspection resources that are necessary as the industry starts to recover.

Rev. Smyth

369. Have you a timescale in mind or are you just playing it by ear?

(*Mr Soul*) I think we are looking at a worst-case scenario. We are planning to be able to resource what we believe are the biggest demands that will made on us as a service. It may well be that as it works out we will not need all the people that we are currently recruiting and training, but we are determined that we will be able to provide the resource that is necessary in order fully to implement the SEAC recommendations.

370. Is this then perhaps the reason why the government has decided on direct compensation to slaughterhouses rather than waiving the MHS charges? Why have they done that, for example? It was previously announced that they were going to do that. Will the assistance—worth about £110

[1]Witnesses footnote: Royal Society of Health

[**Rev. Smyth** *Cont*]
million—be sufficient to address the immediate concerns in the meat industry if you are looking at the worst-case scenario?

(*Mr Soul*) I think that is a question for Ministers, not for me to answer. All I can say is that we have been allocated sufficient financial funding to enable us to recruit and train the staff that we believe we may need.

Mr Austin-Walker

371. Mr Soul, you talked about the procedures for checking that SBO has been removed from the carcases and the procedures that are operated and you have talked about the staffing requirements that have been put forward. Could you tell us how long it takes to check a carcase to ensure that all SBO material has been removed?

(*Mr Soul*) Chairman, I think that is the how long is a piece of string type of question.

Chairman: We do ask questions like that, yes.

Mr Austin-Walker

372. Presumably MAFF has made some estimate?

(*Mr Soul*) We certainly made some estimates of the additional time that we would need to put in when we were asked to tighten up on the enforcement controls, but we have not made any estimate of the time necessary to check removal of all the SBM because it could be so different in different slaughterhouses and you have got different staff doing it. In some cases you would have three inspectors, say, each being responsible for checking a different part of the SBO that has been removed. In another slaughterhouse you might have an inspector who is responsible for doing all of those jobs.

373. If you are making some assessment of the throughput of cattle to be slaughtered and the number of inspectors that you require then you must make some assessment of it?

(*Mr Soul*) I do not think it is possible to compare between different slaughterhouses. Every slaughterhouse is different and because of that, because of the design and layout of the slaughterhouse and the way it operates and so on, the only way you can assess the inspection requirement in a slaughterhouse is to look at that slaughterhouse, to look at the way it operates and to determine on a time and motion basis what inspectors you need in that plant.

374. Can I ask you to comment on the consultation paper that MAFF has put out on the "Funding of the Enforcement of Statutory Controls on SBO in Fresh Meat Premises" because I understand that the assessment is that for budget purposes they have estimated that it would take an average of 30 seconds per carcase for an official veterinary surgeon or meat inspector to check a carcase?

(*Mr Soul*) That was going back to what I said earlier where I was talking about the estimate we have made for the additional resources we wanted to put in to check that the spinal cord had been removed properly. That is just one of the SBM materials. We estimated that in order to carry out that enforcement responsibility more thoroughly we would use as a rough basis, spread over the whole industry, an extra 30 seconds per carcase.

375. In terms of the direction that the SBO control should be more rigorously enforced, could you indicate how you propose to comply with that?

(*Mr Soul*) We are now intending to put two additional inspectors on average in full throughput plants and to provide an inspector in low throughput plants to ensure that there is constant supervision during the dressing procedures to ensure that all the SBM has been removed.

376. And you are satisfied that that is adequate?

(*Mr Soul*) Yes. We have estimated that it is something in the region of an additional 300 meat inspectors, which is a very very significant increase in the number of inspectors we have at the moment. Perhaps I should draw the Committee's attention to the fact that we are now insisting that the spinal canal is thoroughly cleaned out, that all the other soft tissues in the spinal canal are removed because some of the findings of spinal cord that have happened in the past have been because tiny pieces of spinal cord have been hidden in amongst other soft tissues in the spinal canal. There is no statutory requirement that those soft tissues are removed, but we are now saying that in order to comply fully with the SEAC recommendations we will have to insist that the spinal canal is completely cleaned out of all traces of other soft tissue so that we can be absolutely certain that all traces of spinal cord have been removed.

Mr Sims

377. In public discussion a distinction has been drawn between conventional cuts and joints of meat and products such as beefburgers, pies, sausages and pâtés, the implication being that if there is a health risk the latter can carry a higher risk than the former. Could you tell us to what extent the sort of products I have just referred to in the past would have contained material now proscribed as SBOs? Can we assume that branded products from the main burger chains and main processing companies would not have included such ingredients in any case? We are given assurances by some of these companies now that they are not using British beef but imported products. Is it possible that they are importing products from countries that do not in any case themselves remove SBOs?

(*Mr Baker*) Yes, your last point is correct. We believe there are countries that have cases of BSE, although not in anything like the quantity we have and they have no SBO controls at all. Coming back to your earlier point regarding flank meat or trimmings being used in the production of any sort of pies or sausages or burgers, that meat is no different at all to the muscle meat we would recognise as sirloin steak or topside. A perception that has been brought about is that because it is probably trimmings off sirloin steaks or rumps or whatever it is different. It is no different at all.

[**Mr Sims** *Cont*]

378. Does that comply right across the board? Are you saying, therefore, that the sort of meat that is in sausages, pies and so on might equally well have been found on a joint that you buy from the butcher's shop?

(*Mr Baker*) All the SBO material is removed long before it ever gets into meat manufacture. Nowadays there is more trimming to meat than we have ever done because people only want the sirloin steak and they do not want the pieces on the end and so forth, and that is just the same piece of meat but it goes into meat manufacture. Most of the mince products that we do is from forequarter muscles or the flanks of the cattle, whatever. The trimmings off these other cuts are minimal really and there is no difference whatsoever with the normal muscles that we would recognise.

379. Was that the case before the ban on SBOs came?

(*Mr Baker*) Absolutely, yes. As Mr Cracknell said earlier, we cringe when we read in the press about brains and eyes and spinal cords in the past being used in manufactured products. In all my years of experience it has never happened. I do not think anybody would dream of using those things in manufactured products, but that is the way the press brings these things out.

Audrey Wise

380. Mechanically recovered meat; where does that stay in this scheme of things?

(*Mr Baker*) It is now banned from being produced from the spinal column, the vertebrae. It was being produced up to November last year and I never saw any problem with that. Why we have banned it now, I do not quite understand.

381. It is not exactly the same as just a trimming, is it? It is not what you would be able to use of the animal unless you were involved in a lot of technology to recover it. Might it not involve some different sorts of tissues?

(*Mr Baker*) It is basically muscle meat. It is a fact that with the high speed boning knives and so forth the knives often do not take as much of the meat off and as cleanly as we would like. What mechanically recovered meat is is when you put those bones through an MRM machine it forces the soft tissue away from the bone and you end up with that soft tissue. It is a very fine material, but it is just muscle meat that has not been taken off cleanly when they deboned the carcase.

382. When did brains stop being sold? In my young married days you could buy brains and people ate brains on toast. You said they had never been used. I do not understand that.

(*Mr Baker*) Not in manufactured products. Like you, I quite like brains on toast.

Audrey Wise: I did not say I liked them, I said you could buy them.

Mr Marshall

383. You did say, Mr Baker, that these bits of the meat subject to SBO had not been used in the meat processing industry as far as you knew, but you have been talking about brains which were once sold to consumers. Can you tell us what the part of the carcase that is subject to SBO was worth? One assumes that to some extent the SBO ban was really a recognition of previous practices anyway.

(*Mr Baker*) I would suggest that the number of people who eat meat and brains or whatever as a delicacy like that is very few. They do eat more on the Continent, but it is not something that we have been able to export from here because the keeping condition is very short. It is something that needs to be consumed very quickly. As I say, the spleen and so forth that became an SBO back in 1989 was packed and was exported up to that date. I do not know whether it was consumed or not. Very little of the material that is now an SBO was ever consumed.

(*Mr Soul*) Chairman, that is my understanding. The brains that are consumed are usually sheep's brains. Calf brains, yes, are consumed as well, but adult cattle brains I think would be most unusual.

Chairman

384. And now very unwise. They have been prohibited.

(*Mr Soul*) Yes, they are banned now.

Mr Whittingdale

385. I am becoming slightly confused. The ten cases of CJD that have occurred of the new strain, we were given to understand that that was contracted probably as a result of people eating infected product prior to the SBO ban being introduced. Now you are saying that in actual fact people have not been eating SBO in any case because you have never used it in the production of food for human consumption unless people happen to like eating brains. Are we therefore saying that these ten cases are likely to have come from people who happen to like eating brains or how else might they have come to have eaten SBOs which were infected with the agent?

(*Mr Baker*) I do not know, quite honestly. The only thing I can say is that before the SBO ban not every abattoir took out the spinal cord from the vertebrae. It was quite often common for people to have T-bone steaks where the spinal column was still left in. Other than that, I cannot think where that could have come from.

(*Mr Cracknell*) I think even in T-bone steaks it was very unlikely that people ate the bone. I would endorse what my colleague says. In my experience we have never ever recovered the SBOs, with the exception of the new SBO which is MRM. In my experience, we have never recovered those things and sold them into the food chain. I actually did not read into SEAC's report what you did. It said it was a possibility—I cannot remember the exact words now. I think it is true to say that those ten people probably ate carrots as well and that is as likely a cause.

[**Mr Whittingdale** *Cont*]

386. Therefore, on your analysis you are saying that it is very unlikely that the people who have contracted this new strain could have got it as a result of eating SBO prior to the ban being introduced, are you not?

(*Mr Cracknell*) I think that is right. It must be billions to one.

Mr Powell

387. Mr Baker, your slaughterhouse facilities in Crick in Northamptonshire enjoy the highest reputation. Many of the excellent beef farmers in my constituency have their beasts slaughtered there. I suppose it is possible that a number of my constituents work there given the willingness of people to travel long distances to work these days, and clearly all my constituents have the highest confidence in the meat which is actually butchered in your facilities in Northamptonshire. I want to deal first of all, if I may, with all of the stuff we have had about meat inspectors and government regulations and all the rest of it because for the last month we have been told by Marks and Spencer's, by Tesco's, by Sainsbury's, by McDonald's, by just about every single organisation which sells meat, that in addition, over and above and on top of any rules and regulations which are laid down by the government and enforced by government inspectors, the Tescos of this world have additional safeguards. Can you tell us something about those additional, extra safeguards which are over and on top of anything which is done by Mr Soul's organisation?

(*Mr Baker*) Obviously the first and most important thing is this 30-month rule on cattle now being slaughtered fit for human consumption which really came from the supermarkets. It was not only the supermarkets. They set it off and then it snowballed from there to manufacturers and so forth. We have been constantly in touch with them over the last three or four weeks about some of their demands and, as you say, they are visiting us more often and they are doing more checks. They are farm assured. They are now demanding that all cattle being slaughtered come from farm assured farms, which is something we have been setting up with them over the last two or three years but now it is part of their specifications.

388. And those new specifications take the matter further than those which are laid down in law by way of regulations.

(*Mr Baker*) That is right.

389. So the consumer in this country who is shopping in nearly all of the outlet facilities has in addition to anything which the government is able to offer by way of assurance, so to speak, the Marks & Spencer's assurance, the Tesco's assurance and the Sainsbury's assurance over and on top of that?

(*Mr Baker*) That is right.

390. And their inspectors visit your facilities more often than government inspectors, is that right?

(*Mr Baker*) Not more often than government inspectors. We have 11 meat inspectors in our plant plus an OVS.

391. The whole time?

(*Mr Baker*) Yes.

392. But they do more checks, do they not?

(*Mr Baker*) Yes, they are actually coming and checking. Now the specifications have changed they have come to make sure we are using the new specifications particularly as far as farm assurance is concerned. It is to check that they are satisfied the specifications are being adhered to.

(*Mr Cracknell*) As an industry we also work under the Food Safety Act and over recent years we have developed quite sophisticated quality control systems with self-auditing systems on top of those and it is very much an integrated quality system which begins on the farm, of which the Meat Hygiene Service is an integral part, and then is carried on further down the meat chain as the meat is processed, packed and distributed.

393. Of course, and all of that is true and it is not in the slightest bit surprising that my constituents have such a high degree of trust and regard for the safety standards which are applied by your industry. I want to ask Mr Baker about this culling of 30-month old cattle when they have come to the end of their working lives. Do you have the facility for the slaughter of the vast quantities which may have to be slaughtered as a result of this approach?

(*Mr Baker*) Not personally, no.

394. You are the largest slaughterer in the country, are you not?

(*Mr Baker*) Yes, but whether the industry does is debatable because the ministers constantly refer to 15,000 cattle a week.

395. Can you burn 15,000 cattle a week at Crick?

(*Mr Baker*) No, and I would not want to.

396. I am very glad and relieved to hear that.

(*Mr Baker*) But the number is much more than 15,000 a week, of course. When you include the steers and heifers whose weight is such they can be slaughtered over the 30 months, we think there is some considerable time but we need to slaughter nearer 30,000 a week and render them for, we think, at least six months, and then the number will probably drop down to 20,000 this time next year.

397. So 30,000 for six months and then down to 20,000. That is what you are telling us? I want to ask you, finally, about the export ban, which, as you know, extends not merely to meat products but through to confectionery and cosmetics. Have any of the products which have been covered by the European Union export ban been withdrawn voluntarily from sale in the United Kingdom by the manufacturers?

(*Mr Baker*) Not that I am aware of.

398. None of them, so far as you are aware. Does anybody else have any observations about that?

(*Mr Cracknell*) I think a number of manufacturers have withdrawn British beef from sale.

399. I am not asking about that. I am asking you about confectionery products, I am asking about cosmetic products, all the byproducts which, for some astonishing reason, are being banned from entry into the rest of the European Union?

[**Mr Powell** *Cont*]

(*Mr Baker*) It is not something we would specifically produce on site at Crick. There is a number of materials that we would sell that would actually get manufactured into that type of product but I cannot really answer for them.

(*Professor Lang*) Can I comment that I note the thrust of Mr Powell's question but I was not astonished by the European ban. I think again I am back to my main point about the psychology of the consumer. The issue at stake here is trust. It is to do with confidence in the process whereby products are deemed to be safe. I think that many public health specialists have been very reluctant to remove the ban on the grounds that consumers are demanding confidence in the process. They were in a deep shock to find that beef byproducts were appearing in all sorts of things from cosmetics through to children's sweets. So I note what you say as being against that ban but I do not think we should be surprised or shocked by it.

Mr Powell: There are other explanations, of course, which have been put forward, including by the EU Commissioner, for the ban.

Mr Marshall

400. Chairman, I was not shocked by the European ban either. The reason I was not shocked by it is that the Europeans are all too often willing to cheat, but surely the significant fact is that the European Agricultural Commissioner, Mr Fischler, has said there is no scientific justification for the ban. That is the important thing. It is cheating, that ban.

(*Professor Lang*) But the issue is more than science. Clearly science is a major issue but it is not just the issue. Studies of consumers and how they choose show that science is a low issue. Culture, class, symbolic and emotional reasons are much more important in terms of deciding why we choose one product rather than the other, and I think if this Joint Committee comes up with recommendations only looking at science, the crisis will go on.

Mr Marshall: But sociological claptrap is no justification for a ban on British exports.

Mrs Winterton

401. Professor Lang, may I take you to task on what you have just said, actually agreeing in part with it because certainly perception is only based to a certain extent upon science, but the perception by the public of the wholesomeness of British beef has been actually challenged and has been given much publicity by the media, who have behaved in an hysterical fashion with headlines that would frighten anybody. None of that reporting has been based on fact or on science and that hysteria has been whipped up by others who have their own particular axe to grind. Could you tell me, bearing in mind all that you have said, why the European Union does not ban its own beef and beef products? Are you not concerned that in trying to get rid from the food chain of perfectly wholesome British beef we shall import into this country from all sorts of other nations beef which is not reared in a way which our animal welfare people would approve of, where the plants where these animals are slaughtered will not be up to the standards that we have in this country, where the precautions that have been taken in this country for ages will not be undertaken, and yet consumers will then go out and buy beefburgers and other products far inferior to what we produce here? Can you please explain to us this hypocrisy of the EU and the way that you almost seem to support it?

(*Professor Lang*) No, I do not agree with the thrust of your question. I do not support that hypocrisy at all. I would like to make that clear. I think right across Europe there is deep concern by consumers about the intensification of agriculture and the products that come from it. I know from having talked most recently to German consumer organisations just ten days ago, they too feel very strongly that while it is not British beef versus everyone else's, it is about the general intensification of agriculture *per se*. That is what this crisis has always been about and that is what it continues to be about in terms of consumer perceptions. They want a change of direction, I think, in the process of farming.

402. Professor Lang, I was not born yesterday. This is all about politics. You know it and most people know it.

(*Professor Lang*) No, it is not.

403. Yes, it is.

(*Professor Lang*) It is a feature, I am sure.

404. The German government has done nothing to "protect" the German consumer. This is all about damaging our beef industry to benefit theirs.

(*Professor Lang*) I am not aware that the Germans import a very high percentage of British beef. It is only about 6,000 tonnes. That is a lot but it is not a huge amount. What is much more at stake is that the British beef market was expecting to carry on increasing its exports while its home market was gently declining. You know that as well as I do. I think you are right, there is clearly politics to this but I do not see the fissure being the EU versus Britain. I think it is something much more complicated. It is consumers saying they want a different direction for farmers.

Alice Mahon

405. Could I just ask a very quick question to Professor Lang. I am sorry we did not have time to hear you go into more detail about a separate food agency, and just to tell you that we are not all xenophobes, certainly not on this side, but could you tell us, do you think the Department of Health should be responsible for a separate food agency and how would you see it, very briefly? I am quite interested in that subject, and maybe if we do not have time, could you let us have some written material on your ideas?

(*Professor Lang*) Yes, I was going to offer that. Coincidentally, nine months ago I and three colleagues in two different universities, Oxford and Sussex, began to work on proposals for a reform of MAFF to deal with these sorts of issues. That will be in the public arena very soon and I will happily let the Committee have copies of it. Essentially I think we do need to have a very intelligent debate as soon as possible about what sort of mechanisms we can

[**Alice Mahon** *Cont*]

introduce in order to prevent these sorts of problems. But also there has to be a policy shift. I regret to say this but I think on trial is 50 years' experience under both Labour and Conservative Governments -it is not a party political point—who have asked farmers to intensify and industrialise, and I think the public, as so often, is in advance of the political processes and mechanisms and they are saying, "We are not particularly happy about what has gone on." They see the heart diseases under health, which is familiar to the Health Committee; they see the pesticides and the nitrates and the water bills that we are now all having to pay more on to pay for cleaning up and so on. The bill for this industrialisation of agriculture is mounting up rapidly and I think that is why there has been such deep scepticism in the public in response to MAFF continuing to say, "Thou shalt eat British beef," and certainly talking to European consumer organisations, they are not going to be hectored by us.

406. So should it be under Health?

(*Professor Lang*) I think that is one option.

Mr Powell

407. If I may, I want to return to the points which were raised by Mrs Winterton, which were very important points indeed, because we have heard a lot about consumer organisations. Members of Parliament, fortunately, are in daily contact with their own constituents, who are actually the consumers, and I have to say that there is an enormous gap at the moment between what I am told by consumer organisations and what I am told by members of the public, who happen to be our constituents, about this, and what they are very concerned about at the present time is the quality of meat which might be imported into this country as a result of this crisis from other countries in the European Union. In particular I was able to draw the Committee's attention to an article which appeared in the *Daily Telegraph* about a month ago about, for example, Belgian beef, which was fed hormones and, unbelievably, also reared on cement powder. What I am under pressure to do is to try and persuade the Minister of Agriculture to introduce a ban on all imported beef into this country because it is not up to the quality of the beef which is actually reared in this country and available from the British farming industry and the British slaughterhouse industry and the British retail industry.

(*Professor Lang*) Very quickly, I agree with your scepticism. I think one of the morals we can draw from this tale, certainly from my point of view, concerns closed systems of farming. Mr Meldrum since 1991 has been assuring me that there have been no cases of BSE from organic farms except two cases where calves were bought in. The moral of the tale is that we need to get our farmers to farm again, namely, not just to trade, where they do not know what the feed is, where it comes from or where their calves come from. We need to have closed systems of farming where farmers actually rear their own stock and grow their own feedstuffs. I agree with Mr Powell absolutely. I think the criticisms of British intensive farming should not be taken as an excuse to say that on the other side of the Channel things are better. There too they have been going down the same route. That is why I would urge you, as a Joint Committee, not to go into Britain versus the rest. This is a much bigger problem that we are now all addressing.

Mr Leigh

408. You made, I thought, a bit of an alarming statement, Professor Lang. You said that we should not base our recommendations on science?

(*Professor Lang*) No, just on science.

409. I am glad you said that because, for instance, there is one particular problem that comes up with gelatine. Professor Pattison, in his evidence, assured us that this was part of the export ban but even if it was infected, because of the manufacturing process the infective agent would be rendered harmless. So that just shows the danger of basing export bans or recommendations or anything else on matters other than pure science. You say we should not base our recommendations on just science but if we were to start veering off or wandering off into public perceptions based on what they read in newspapers, do you not think it would be very dangerous for the Select Committee to make recommendations based on that?

(*Professor Lang*) I agree. It is not for me to tell you how to do your job. I think you are right, that would be very delicate stuff indeed. I think what I was urging you to do was to take on board that consumer perceptions anywhere in the world—and anthropologists have been clear about this for 100 years—are based on very subtle configurations of meaning. Some people in some circumstances will eat one food and 100 miles away they will not. Even within Britain we have tremendous regional variations in diet, which is remarkable given that we are supposed to live in a globalised market. So I agree with you, you are right.

Mrs Winterton

410. Professor Lang, you have stated twice that you are concerned on behalf of consumers at the way intensive farming is carried on throughout Europe and in this country and you have expressed the view that you would like us to return to more traditional methods?

(*Professor Lang*) To debate it, yes.

411. Are you aware that it has really been the push for cheaper food that has pushed agriculture into intensive methods and the consumers, be those consumers British or European, are going to have to pay more for their food? Are they prepared to do so?

(*Professor Lang*) I think again that is a very pertinent point and I agree with the sentiment that you have expressed. In hard economics, the issue is cost externalisation. What we have done is to have cheaper food in Britain at the cost of a rising health bill and a rising environmental pollution bill. So we are paying for it under a different budgetary heading. One of the things we have to recognise is that when you cross the Channel, the cultures there tend to pay more for their food. Food is a larger element of the French or Belgian or German household budget than

[**Mrs Winterton** *Cont*]
it is in Britain. We spend more on our houses than we do on our food and I think to some extent there is now a recognition that something has gone slightly wrong in the British food culture and many people now see that we must starting giving a greater priority to the amount of money we spend on food. Finally, I would like to say that I am not that convinced that our cheap food policy has worked, even in cheapness. As you know, we have a major problem in one-fifth of the population not being able to afford an adequate diet. I should say I sit on the Government Committee on Food and Low Income which will be reporting very soon, and believe you me, it is a very humbling experience. I do not think the cheap food policy has worked even in meeting cheapness. I think it is better to start talking in terms of affordability and whether or not we choose to spend more money to get better quality. I think there is, frankly, a need for a long-term education process and I think this Joint Committee could do a great deal to recommend a more long-term view in building up consumer education, where I think we have lamentably failed.

(*Mr Cracknell*) Chairman, can I interrupt for one second because clearly it is very interesting. This question of intensification of livestock is a worry to us all and I suspect it is at the back of the worries of many consumers. The actions that have been taken in the last four weeks to ban cattle that are over 30 months old—never mind the cows and the bulls that are coming to the end of their useful lives—clean cattle, prime cattle, our natural breeds, the Continental breeds, where we have encouraged farmers over the last few years to extensify production, to leave cattle longer, to grow them more slowly, to use more organic, traditional farming methods, actions that farmers have responded to very well—those now are the very cattle that are going to be slaughtered and taken out of the food chain. We think that that is a PR problem that is waiting to happen. We are not talking here about old cows. I think the Ministry were perhaps somewhat surprised to find out how many there were, but we have been actively encouraging farmers to de-intensify production methods. The best Charolais cattle do not naturally mature at under 30 months; they have to be fed faster and I worry that this is a problem that is coming.

(*Professor Lang*) I agree. I see absolutely no basis at all -

Chairman

412. I understand in the Minister's statement he did say that he was seeking a way out of that particular problem and I also understand that the 30 months, I was interested to hear one of you say that had come from the buyers or the supermarkets. It was a SEAC recommendation, I believe?

(*Mr Baker*) No. The SEAC recommendation was that beef over 30 months should be de-boned at dedicated de-boning plants.

Sir Roger Moate

413. Can I move back to what we touched on earlier, namely, the methods that we are going to employ to destroy those cattle that even under the present arrangements are going to be destroyed. Very high figures have been mentioned, we know, at least 15,000 a week.

(*Mr Soul*) Possibly thirty thousand.

414. Three-quarters of a million to a million a year, and there has been a lot of media speculation about the options facing us. I wonder, gentlemen, if you could tell us where we have got to, what you see as the principal options available to the country now to dispose of these cattle in a way that does not undermine public confidence. That is the first question, what are the options? Secondly, how critical is it at this minute of time that we come up with options fast? How big is the blockage? There is one other question I would like to add to that: basically, who is going to be responsible for supervising that process to make sure the new system actually works effectively?

(*Mr Cracknell*) The options for disposal are either incineration, for which there is very limited capacity, we understand, or rendering, for which we understand there is some capacity, certainly adequate capacity, we are told, to cope with the culled cows and mature bulls. Whether there is enough capacity also to cope with an unknown quantity, but probably not less than a quarter of a million and not more than half a million, of prime cattle over 30 months which now have to be dealt with in six months, we are told, I do not think anyone knows.

415. I am sorry to interrupt you there. Did you say there is enough capacity now in incineration to deal with the 15,000 old cows coming through currently?

(*Mr Cracknell*) In rendering. We have been told that by MAFF, in rendering, not in incineration, but no-one knows how many of these prime cattle over 30 months there are in the system. So I do not suppose people can speculate on whether we have the capacity and time will tell, but those are the only two options.

416. As far as your Federation is concerned, your concern ends at the rendering process. After that it has to move into the governmental machine for disposal?

(*Mr Cracknell*) Our Federation is concerned that we are seriously taking a wrong step in doing all this, in taking fit animals out of the system, and so we are worried about that. Our Federation would much prefer, instead of this blanket slaughter of everything over 30 months old, to see a more selective slaughter because we think the housewife wants confidence in beef. She will get confidence in beef. Last year there were 11,000 cases of BSE, this year we want 2,000, next year we want 500, the following year we want 400. We want to bring those numbers down rapidly.

417. With respect, I am sorry to interrupt again, but whatever happens with selection later on, at this moment in time there seems very little debate about taking the old culled cows out of the food chain, and even at that figure it is 15,000 a week, as I understand it. So we have an enormous problem of disposal and

[Sir Roger Moate *Cont*]

there is little debate about the need to take out those three-quarters of a million. It is critical, it is urgent. What are the options now? We can render them but can we incinerate? I gather there is not the capacity, so my question is, I believe, a crucial one. What are the options now, who is responsible for introducing them and when might we see those plans, because unless we have them soon you cannot dispose of your rendered material?

(*Mr Cracknell*) We are in consultation with MAFF over starting a scheme of culling cows and clearly the farmers are anxiously awaiting what is to happen. We are somewhat disappointed that we have not been consulted as an industry in this throughout this crisis. In fact, yesterday was the first time we saw the Minister whilst we have seen our industry disappearing, and we want to be involved in the process of culling. We want to help the livestock industry. The farmer has been more or less compensated totally. Many of the members of our Federation are going to go to the wall. There was the day before yesterday a package announced but it is somewhat limited in its scope and it is a bit of an unguided package of aid. But I believe we can get a system up and running fairly quickly as long as the Ministry and the farmers treat us as if they want our help. We have been told that we are going to have to be tendering for this work and it is going to become another difficult bureaucratic process. We have the ability to slaughter these cows but we want to be participating in the scheme that will be used to take them out.

418. Mr Soul, can you help us on this?

(*Mr Soul*) I do not think I am in a position really to help very much with the detail that you are seeking now because it is the core MAFF that is busy working out the details of the slaughter and compensation arrangements. But I think it is quite true that the only really viable options for disposal are rendering or incineration. I understand there is insufficient incineration capacity at the moment but, of course, once the material has been rendered it could then be incinerated. It is much less bulky, so the rendered material could be incinerated or disposed of by burial in an authorised tip.

419. So the major question I am putting about incineration is beyond our witnesses today, is that right? You cannot tell us?

(*Mr Soul*) Yes.

Chairman: I can tell you that it is the Ministry's intention that the renderers should deal with the vast majority of the cattle and there is capacity in the rendering industry to do so.

Sir Roger Moate: Then we have to incinerate the rendered material.

Chairman

420. In the same plant maybe.

(*Mr Baker*) Part of the question as well is, do we actually agree with the policy of slaughtering the steers and the heifers over 30 months? We have been making representations that there is nothing wrong with the cows. Actually to slaughter these steers and heifers as well as incinerate them I think is absolutely criminal. We believe there is a market out there for it, particularly from the manufacturers and catering people, and we do not believe those cattle, what we call clean cattle, over 30 months old should be destroyed at all.

Sir Roger Moate: There is a lot of sympathy with that point of view, I can assure you.

Chairman: I think we have taken on board your views. Gentlemen, thank you very much for the time you have given us. It has been very helpful to us this morning. We are very grateful.

Memorandum by the Meat Hygiene Service

MEAT HYGIENE SERVICE ENFORCEMENT OF SPECIFIED BOVINE OFFAL/MATERIAL CONTROLS (T3 BSE)

MHS staff operate in accordance with its Operations Manual. This is a comprehensive document which sets down guidance issued by the Agriculture Departments on the application of the various legislative requirements. It is accessible to MHS staff in plants. There are two volumes concerning both operations and legislation.

The Operations Manual was revised to take account of the requirements of the Specified Bovine Offal (SBO) Order 1995 and again in November and December 1995. The SBO legislation has been issued to be put in all Legislation Manuals.

Since November 1995 the MHS Chief Executive has written three times to each member of the MHS plant inspection teams to stress the importance of proper control in respectof the SBO and later the SBM Order.

The Chief Executive has also held regular teleconferences with the MHS's six Regional Managers ensuring that they were up to date with any revisions to SBO/SBM control requirements. Regional Managers are in close touch with plant-based staff either directly or through their Area Resource Managers and/or Principal Official Veterinary Surgeons to ensure that plant-based staff are updated continually.

In addition the MHS Head of Operations has written at regular intervals to Regional Managers, Principal Official Veterinary Surgeons (POVSs), Official Veterinary Surgeons (OVSs), Area Resource Managers as well as all staff based at meat plants with detailed instructions for implementation of SBO/SBM controls, where

necessary reinforcing these instructions, advising them of any tightening up of inspection requirements, requiring MHS staff to ensure plant operators were fully aware of SBO/SBM controls and always emphasising the importance of full compliance with the SBO/SBM controls.

The MHS developed its own action plan to reinforce SBO control requirements after the results of the State Veterinary Service (SVS) audit of slaughterhouses in September 1995 showed approximately 50 per cent non-compliance with controls. (It should be noted that the SVS identify unsatisfactory visits against a number of failure points. Some of these represent breaches of Regulations others are not strictly non-compliance and properly fall into the category of good SBM practice.) The MHS plan included the circulation of further instructions to MHS staff; sending of letters to (i) members of the MHS Industry Forum, (ii) individual plants; daily recording of satisfactory SBO compliance in the Plant Day Book; referral of non-compliance plants to the MAFF's Investigations Branch and the introduction of a programme of management checks on its own staff. Increased resources were provided to ensure compliance. 10 further full time equivalent Meat Inspectors were employed in a number of the largest plants to supervise the dressing of carcases. 100 additional hours per week were devoted by OVSs and POVSs to conduct extra checks on carcases in chillers and to double check that plants were correctly carrying out controls. Extra time was spent by Meat Inspectors to check that SBO had been properly separated from the carcase (at least 30 seconds per carcase), that it was kept separate from meat and from other unfit material, that it was stained correctly, that it was disposed of in accordance with the SBO Order and that the occupier kept proper records.

During the Autumn of 1995 the MHS ran a series of one day training courses for all meat inspection staff throughout the country specifically dealing with SBO controls. At a training course for ARMS in March and a Regional Managers meeting in April staff were instructed on the importance of ensuring 100 per cent compliance with SBM requirements. Further training on SBM controls was provided to POVSs in April 1996 in the light of the recent situation. POVSs are subsequently operating refresher training courses for all staff at the meat plants for which they are responsible. To assist this training exercise, the MHS has drawn up a guidance pack on SBM controls which is being circulated to all members of staff working in plants. The pack is supplemented by a comprehensive set of slides. A video is also in production.

A special piece of equipment has been supplied to staff to assist them in ensuring that all traces of spinal cord have been removed by plant staff from beef carcases.

The MHS has discussed in detail with MAFF Legal the procedures for referring cases of non-compliance by plants to Investigations Branch. To date 10 plants have been passed to Investigations Branch. One plant is currently facing prosecution, one is with the Procurator Fiscal in Scotland, six cases are still under investigation and two have not been pursued. It became evident that investigations could not be undertaken if the carcase containing spinal cord had been healthmarked by an MHS official. In these cases the MHS implemented its own disciplinary procedures.

22 MHS officials have been subject to full disciplinary investigation as a result of their failure to ensure 100 per cent compliance with SBO controls. These have been conducted in accordance with MHS agreed disciplinary procedures and employment law requirements. Following investigation one officer has been dismissed; 12 have been issued with formal disciplinary warnings and advised that further issues of non-compliance are liable to lead to their dismissal and no formal disciplinary action has been taken against nine. However the latter have been issued with a written caution advising them of the need for 100 per cent compliance and further advised of the disciplinary consequences of any further non-compliance. Two contract OVSs have been issued with formal warnings. In addition, the services of a number of contract OVSs have been terminated following their failure to comply with procedures. Before MHS staff re-enter plants they are provided with re-training by their Area Resource Managers and/or plant OVS. Representations have also been made to plant operators where appropriate to improve plant conditions and inspection facilities, eg lighting.

Two MHS Meat Inspectors are currently under disciplinary investigation.

At the beginning of April 1996 the MHS employed 509 red meat inspectors on a full time basis. In addition it utilised 62 red meat inspectors on fixed term contracts of 100 weeks duration. There was also a pool of 203 red meat staff used on a casual basis. The meat inspection staff are supervised by OVSs. The MHS directly emloys 39 OVSs and contracts out work to 110 full time equivalent vets to supplement them. Three vets are used on a casual basis.

To ensure 100 per cent compliance with SBO/SBM controls additional funding of £50.4 million has been made available to the MHS for the recruitment of additional meat inspectors and OVSs.

To ensure the rigorous enforcement of the SBO/SBM controls and particularly the application of SEAC's recommendation for constant supervision during the dressing of beef carcases, an additional two Meat Inspectors are operating in full throughput plants and additional Meat Inspector hours have been provided in low throughput plants to supervise beef dressing to ensure that all traces of SBM are removed. An extra 500 OVS hours/day are also being used to provide additional OVS supervision.

A comprehensive effort has been made by the MHS to ensure that all staff qualified in meat inspection can be identified and recruited to reinforce MHS Inspection staff. Steps taken to date include mail drops and recruitment in various relevant trade journals. The MHS anticipate recruiting up to 300 additional inspectors/technicians to ensure the effective enforcement of SBM controls and thereby 100 per cent compliance with the Order by plant operators.

Following liaison with the Department of Health a joint letter was sent to all Chief Environmental Health Officers and Chief Executives of Local Authorities requesting their co-operation in assisting the MHS enforce controls by encouraging the secondment of Environmental Health Officers.

It is apparent that while substantial efforts to recruit staff have been and continue to be made, the number of personnel qualified in meat inspection nationally may be insufficient to enforce all additional controls. The MHS has therefore developed the concept of "Meat Technician" to provide back up support to Meat Inspectors and OVSs and give additional public assurance that SBM controls are comprehensively enforced. These individuals will be required to hold appropriate relevant qualifications, eg from the Institute of Meat or have practical experience in the meat trade. Their main responsibilities will be to assist Meat Inspectors in ensuring that SBM has been removed from carcases, carrying out dentition checks and helping to monitor and ensure documentation is accurate and complete. A training syllabus, academic and practical, has been drawn up in conjunction with the Royal College of Veterinary Surgeons, University College, Salford and the Royal Society of Health. POVSs will assess the suitability of trainees using a specification drawn up by the University. The Royal Society of Health will undertake final assessment and certification of suitable candidates.

It is not the intention of the MHS to replace Meat Inspectors with Meat Technicians. Meat Inspectors are required to undergo 400 hours of theoretical and 200 hours of practical training. This usually takes place over a period of 1–2 years. They will not be authorised under the Fresh Meat Regulations to inspect or health mark any meat. The Minister has given his approval to this initiative to recruit and train staff to carry out specific tasks to ensure the effective enforcement of SBM controls. The MHS envisages taking on between 50–75 technicians.

May 1996

Memorandum by Professor Tim Lang, Centre for Food Policy, Thames Valley University

BSE and UK food policy: the policy lessons to be learned (T4 BSE 9)

Summary and Recommendations

BSE has provided an object lesson in how not to manage risk. Eight years into the crisis, too much attention has been paid to too narrow a conception of science and not enough attention has been paid to failings of public policy with regard to consumers, decision-making at MAFF and information flow to the public. A chasm was opened up in public confidence by the early 1990s, which subsequent events have only widened. This submission argues that improvements in public confidence in beef will only be generated once longer-term reforms are put in place. Proposals are given to this effect.

A number of conclusions can be drawn from the above:

i. It is wrong to look to science to provide answers to social problems. As critics of science throughout the twentieth century have argued, all science can do is make judgements on base of best evidence. Science reflects, but does not transcend, its social context. Ministerial reliance upon science not only misunderstood the nature of science in general but made unwarranted statements about the certainty of current understanding about BSE in particular.

ii. Unless consumers, or their representatives, are involved in judging risk, their behaviour is likely to be unmanageable. Indeed, there are strong grounds for arguing that even in low risk situations, heightened awareness that there is a risk can lead to risk avoidance behaviour or, as with cigarette smoking, into elaborate rationalisations of chosen patterns of behaviour.

iii. Government has been hoist by its narrow conception of research into consumers. It has not apparently drawn upon work it has itself funded. Representatives of consumer, environmental health and social science should be represented on SEAC, and other relevant committees. BSE shows that a narrow view of science will not provide sufficiently broad base of advice from which government can act sensibly.

iv. There should be a national debate about the role, and case for reform of, the Ministry of Agriculture, Fisheries and Food.

v. There should be a review of government's approach to consumer information. Consideration should be given to setting up a Consumer Information Unit, but only if it was based upon a policy of listening to, and incorporating consumer concerns. The tone, as well as the substance, of information should be inclusive rather than patronising.

vi. No reform process would be complete without a commitment to change direction in national food and farming policy, away from further intensification, and towards a more extensive, sustainable system of production. This has been argued for on environmental grounds, now also on public health and consumer confidence grounds.

vii. The immediate task of reducing the incidence of BSE should take note of the Agriculture Committee's advice in its 1990 report, when it argued that government should go beyond what the scientists say "whether for political, commercial or other reasons". As consumers now know, the government ignored that advice too. No resolution is possible without following that earlier advice.

viii. Behind this sorry saga is a wider, moral truth. Food should not be for ever treated as the output of an industrial process. Nature is not to be raided and pillaged. This is a crisis of industrialised food and farming. Diseased bits of dead sheep were fed to herbivores which humans then ate. Labelling should include more information about not just the content of the food, but of the process which generated it.

ix. There should be an independent inquiry into the feedstuff industry, and the rendering industry in particular. Taxpayers' support for the renderers should be conditional upon reform.

1. Introduction: Juggling competing interests

MAFF has had the difficult task of juggling competing interests over BSE. These interests included: defence of the meat industry, public health, consumer confidence, its own reputation, cost, making decisions in a difficult scientific context and, last but not least, government ideology (particularly on supporting industry).

We know that this juggling act has failed. As a result, MAFF has entered the annals of food and public policy history as being the first ministry to help devastate an industry it is in existence to promote. The Select Committee need to decide why this happened. My own judgement is that MAFF and the Government placed an unwarranted reliance upon an evolving scientific understanding of BSE, and that when drastic action such as a culling programme in the early days was appropriate, backed off.

Action was not taken, despite a covert recommendation from the Agriculture Committee in 1990, among others, to do so, out of a short-sighted belief that that would be an unecessarily harsh punishment on the meat industries. Paragraphs 43 and 44 of the 1990 Agriculture Committee report should still be implemented. These argued that the Government should go beyond what the scientists say "whether for political, commercial or other reasons".[1]

As a consequence, certain sections of the food industries have been hammered even harder than they would have been from 1989 on, and the government is now in the embarrassing position of trying to provide financial and other support to a considerable extent—with or without European backing. Annual culling and compensation costs from now on of up to £0·5 billion are no mean bill to pay for a policy mistake.

Government has argued consistently (see section 4 on risk below) that the foundation for its policy is science. This is not the whole picture. Although knowledge about spongiform encephalopathies is by no means new (see, for example, Gajdusek's comprehensive review in 1985, before the UK BSE crisis emerged[2]), it is evolving fast and despite large sums of research money is still not, in the words of one writer in the recent British Medical Journal series of articles, "as robust as once thought."[3]

The Government underestimated the significance of three factors in the policy arena which became critical. These were:

(i) the role of consumers and information in the marketplace.

(ii) the contradictions in MAFF's role in food policy.

(iii) the nature and perception of risk.

Of each of these, hard questions need to be asked as to whether the Government was given advice. If it was, what was it? If it wasn't, why not?

2. Information and the Consumer

BSE was a terrible shock to consumers and farmers alike, but it has had the effect of bringing them closer together. Consumers who had been worried about modern farming practices, on occasions accusing them of adulterating food.[4] being excessively subsidised.[5] and polluting the land,[6] began to have sympathy for them.

Consumer confidence the key

Polls suggests that support for farmers would be high if farming was to be conducted in more ecologically sustainable and socially acceptable directions. In one poll, 71 per cent said there need to be more control in the methods farmers use; 82 per cent said they favoured an increase in organic farming; 79 per cent said taxpayers' support should only go to farmers who farm in ways that do not harm the environment; 83 per cent saying taxpayers' support should only go to farmers who do not harm animal welfare.[7] Farmers, who had at times seen consumers as necessary but ill-informed evils, found themselves to have been fraudulently treated as consumers of the feedstuffs industry. Both parties have been rendered victims by contaminated material being fed to cattle.

BSE has changed the food policy landscape within consumers think, act and eat. The disease has entered everyday language, featuring in jokes and references for years. The current situation, therefore, may have come as a surprise to people elsewhere in the world, but not to British consumers. There have been countless TV programmes and newspapers articles on the subject since news first broke.

People, in my experience, are generally sober and sensible about the subject, although the scale of the epidemic is clearly considerable. Approximately 160,000 cows have suffered from the disease which now affects 34.4 per cent of British farms[8]—53.3 per cent of dairy farms, but only 14.7 of beef suckler herds.[9]

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[1] Agriculture Committee (1990). Bovine Spongiform Encephalopathy (BSE). Fifth Report London: HMSO. paras 43-44.

[2] Gajdusek, D Carleton (1985). Subacute Spongiform Encephalopathies Caused by Unconventional Viruses, in K Maramorosch and J J McKelvey (1985). Subviral Pathogens of Plants and Animals: Viroids and Prions. Orlando: Academic Press, pp 483–544.

[3] Roberts, R. W. (1995). Furrowed brow over mad cow, British Medical Journal, 311, 1419-1420.

[4] London Food Commission (1988). Food Adulteration, London: Unwin Hyman.

[5] National Consumers Council (1988). Consumers and the Common Agricultural Policy. London: HMSO.

[6] See, for example, Maynard, R (1991). Off the treadmill. London: Friends of the Earth.

[7] NOP poll (February 1992) for: SAFE alliance (1992). What future? London SAFE Alliance.

[8] MAFF (1995). Bovine Spongiform Encephalopathy: A Progress Report. London: Ministry of Agriculture, Fisheries and Food. November p 4.

[9] Gore, Sheila (1995). More happenstance: Creutzfeldt-Jakob disease in farmers and young adults, British Medical Journal, 311, 1416-1418.

Although from an early stage, Government informed consumers that it knew what the disease's cause was—infected feed—scientists were simultaneously asking many, more uncertain questions (see the list of research suggestions in the 1989 Tyrrell Report[10]). From the very beginning of the BSE saga therefore, some ambivalence was built into the nature of information on the subject—certainty alongside uncertainty. Industrial and government interests have tended to favour certainty, while consumers to a greater extent asked the "what if" questions more overtly. Science, being a process, not a static state of knowledge, has always suggested less certainty than everyone desired.[11]

Low confidence in Government over food

It should also be recognised that, in such crisis situations, Government is no longer the only source of information in the public arena. One academic study of the food scandals of 1988–90 (in which BSE was part) found that non-government figures were as significant, if not more so, in media appearances, than government ministers.[12]

Public confidence is a key factor in crises, yet the Government has seriously misjudged and mishandled it, exacerbating consumer volatility and engendering entirely understandable behaviour which went against Government's stated intentions.

The BSE crisis emerged at the end of a decade in which the Government has already been severely dented with regard to its handling of food policy: salmonella, listeria, microwave oven safety, food irradiation, additives. The list seemed long. In July 1989, at the height of the food scandals—with BSE only recently in the public arena—the Consumers Association conducted a poll. Three out of four people agreed with the statement "the government has failed to protect consumers from unsafe food".[13] Seven years later, a similar poll found attitudes hardening, in that 73 per cent of consumers felt that "the Government knew there was a risk and tried to hide it."[14]

Food Labelling: process as well as content

In economic theory, market efficiency depends upon there being many consumers offered goods and services by many producers, and upon there being open and sound information between them. In practice, even food labelling has become a battleground of interests, rather than an effective tool for market efficiency.[15] Food labelling is limited to the list of contents (in decreasing order by weight) and, lately, nutrition information. Even the latter is contentious in that it is given in the form least wanted, understood or liked by consumers, according to research funded by MAFF.[16]

More effort and space in accorded to brand information than process or content labelling; hence the considerable public distaste when it transpired that so many consumer products contain beef products (cosmetics, pharmaceuticals, as well as non-meat foods).[17] Having been told by MAFF that everything consumers need is on food labels,[18] public anger and irritation was understandable when it transpired that it was not.

Over recent years, there has been considerable resistance—led by industry and without apparent strong resistance from MAFF—to giving full disclosure of process information, now being so stongly called for by the public to enable them to avoid products with beef by-products in them. The Food Advisory Committee, for instance, argued strongly against pesticide information in 1991, giving the extraordinary argument that if consumers were worried about pesticide residues, they could always buy organic produce.[19] Such arguments missed the case for positive declaration—upon which market efficiency relies—and they also ignore the precedent set by the E-numbering system for additive declarations, introduced by the European Commission in the early 1980s.

MAFF also consistently argued against labelling of irradiated foods from 1985, but did, to its credit, bow

[10] Consultative Committee on Research into Spongiform Encephalopathies (1989). Interim Report. London: Ministry of Agriculture, Fisheries and Food and Department of Health. June. pp 5–18.

[11] see, for example, Dealler, S (1996). Lethal Legacy. London: Bloomsbury.

[12] Miller, D and Reilly, J (1994). Making an Issue of Food Safety: The Media, Pressure Groups and the Public Sphere in Maurer, D and Sobal, J (eds). Eating Agendas: Food and Nutrition as Social Problems. New York: Aldine de Gruyter. pp 303–336.

[13] poll for Association for Consumer Research, conducted by Research Surveys of Great Britain, July 1989, quoted in Consumers' Association (1991), Consumers and Food Policy. London: Consumers' Association. p.2.

[14] ICM poll for The Guardian, in Linton, M and Bates, S (1996). "Public suspects beef cover-up", The Guardian, 3 April, p.1.

[15] Lang, T (1995). The contradictions of British food labelling policy. Information Design Journal, 8, 1, 9-17.

[16] Black, A and Rayner, M (1992). Just Read the Label. London: HMSO.

[17] Lobstein, T (1996). Beef watch. Food Magazine, 32, January, pp 6-7.

[18] see "Look and the label" Food Sense leaflet series. London: MAFF.

[19] FAC (1991). Food Advisory Committee Report on its Review of Food Labelling and Advertising 1990. FdAC/REP/10. London: HMSO.

to pressure in the 1990s when the process was finally legalised. On genetically engineered foods, the Polkinghorne Report did recommend some labelling, but only for transgenic food products where there might be either a health or ethical concern.[20] A recent review of surveys of consumer attitudes on biotechnology shown that consumers want clear and comprehensive labelling about process as well as content.[21] Consumers have so far not got this. This should be remedied using new technologies such as consumer display systems, now available.

The obfuscation and lack of information with regard to BSE is therefore not accidental but happens with regard to other sensitive issues in food. It can be reasonably asserted that this failure is systematic. It happens too regularly for it to be random. It took nearly two decades of medical argument and evidence before even the current inadequate nutrition labelling was introduced. MAFF can with some justice respond that it is not helped by having to negotiate on such matters with other European member states, but the point still stands, that the UK government reflex is to withhold rather than disclose a wide range of information. MAFF has made some progress, but only when there has been concerted action and campaigning from outside, particularly from Non Governmental Organisations. This culture in MAFF needs to be altered, if public confidence is to be won back. Labelling is not the only information failure, however.

Other sources of consumer information

Besides on-food information, the traditional Parliamentary model of information flow is something like this; scientists inform ministers and MPs; parliamentary debate occurs and the media disseminate it in a kind of "trickle down". This model does not fit well in today's world where the food consumer is barraged with other sources of information. The food sector invests an annual sum of approximately £0.6 billion on advertising[22] and is also beginning to move into direct marketing (supermarket "loyalty cards" being but one example of this trend). From the consumer's perspective, politicians' pronouncements on matters relating to food safety are therefore only one among many sources. Although US-style news management–tight, exclusive and task-focused—might work for political news, it is highly inappropriate in consumer affairs.

When consumer confidence is at issue, actions speak louder than words. When Perrier had a problem in February 1990 with benzene contamination at its bottling plant, it withdrew its product world-wide, announced what it was doing and, although it has never regained its market position (and was taken over by Nestlé), minimised the damage, cleaned up the source of contamination (a failure of filtration at the plant to remove the naturally occurring benzene).[23] There was no obfuscation about there being "no risk" coupled with more cautious messages that "more research is needed". The emphasis was upon action to meet consumers' concerns. "No risk" meant removal of the product and cleaning up the source—a classic public health strategy, and not what happened with BSE. There the source remained in flow, but there was an attempt to reassure consumers by assuring them the contamination bits were chancelled away. Whatever its merits or failings scientifically, this was poor consumer psychology.

The Consumer Panel and Meeting of Chairs of Consumer Organisations

Learning from other food scandals in 1988-90, MAFF set up its Consumer Panel as another forum for communication. Its value must now be called into doubt. The Panel has often prognosticated on BSE, and free from complicated scientific jargon though these deliberations have been, they have merely been reprises of existing positions. No new light has been cast. The point of relevance now is that the Panel has been completely marginal in Government handling of consumers in this BSE crisis. One could be forgiven for arguing that it is a sop when the heat is off MAFF, and irrelevant when it is.

Although members of the Panel and the quarterly meeting of the Minister with Chairs of Consumer Groups were asked to a meeting early on in the crisis, neither body was at the heart of government decision-making or thinking in the immediate aftermath of the BSE crisis. It was not until 14 days after the storm blew that there was a full meeting of the widest number of consumer NGOs with a junior Minister, on the invitation of MAFF's head of Consumer Division. What is the point of such information mechanisms if they are "bolt on extras" rather than at the heart of information flow?

Underestimation of consumer power

The government approached BSE consistently from a narrow perspective. But its fatal mistake was to underestimate consumer knowledge and power. This was ironic, as the present government has been particularly forceful in extolling the virtues of consumer sovereignty. It has not a leg to stand on when consumers take them at their word and withdraw their favours from the marketplace.

[20] Polkinghorne. J (1994). Report of the Committee on the Ethics of Genetic Modification and Food Use, London: HMSO.
[21] Genetics Forum (1996). Pulling off the confidence trick? Splice of Life, 2, 6, pp 6-7.
[22] MEAL figures.
[23] BBC Radio 4 (1996). Shelf Lives. May 7.

For some good to come out of this crisis, there has to be a genuine, rather than cosmetic, change in government thinking with regard to consumers.

The consumer movement has also begun to learn the lessons. Food products, like all objects of consumption, can be highly charged emotionally. It is never surprising, therefore, if when a crisis occurs, consumers perceive themselves as victims.[24] The consumer movement has always been particularly active and popular at times of change in industrial production.[25]

Trust is a fragile beast. The BSE incident has accelerated scepticism about the food industry. Beef sales were gently declining before the current period and have plummeted since.[26] Prior to recent months, the price of beef was only maintained by the rising export of British beef; exports compensating for declining a home market. The hectoring tone adopted by government on occasions has been counter-productive.

3. MAFF's role in food policy: sponsorship versus regulation

The second factor which the Government failed to appreciate is the structure of MAFF and the state of food policy in Britain.

The goal of "efficient" farming

The BSE crisis has exposed a conflict in MAFF between its role in promoting an industry and regulating it. Ever since Tom Williams' Agriculture Act 1947, the imperative within MAFF has been to promote "efficient" farming. The lessons of World Wars 1 and 2 had been learned. These were that, firstly, the policy of reliance upon an empire or colonies to provide cheap food was now risky and, secondly, Government has a duty to ensure safe and secure food supplies for all its population.[27, 28, 29, 30] The 1947 Act symbolised a change of policy and ushered in half a century of application of industrial practices to food and farming. Labour was shed from the land, replaced by the "liquid hoe" and sophisticated machinery. Efficiency was judged in financial and gross output terms.[31]

By conventional terms, this revolution on and off the land has been very successful. Yet, 20 years ago, doubts about the efficacy of this industrialisation policy began to emerge. The financial, human and ecological costs began to mount up: loss of biodiversity,[32] pollution of water supplies,[33] rise of cardiovascular diseases and food-related cancers,[34] unnecessarily high use of motorways and transport,[35] unequal effects on consumers.[36, 37] Despite growing evidence of public concern, MAFF's policies have continued with only minor adjustment. Conflicts over the definition of "the public interest" emerged into the public arena with increasing regularity and ferocity in the 1980s, beginning with the ill-concealed row in 1982 over the report from the National Advisory Committee on Nutrition Education (NACNE).[38]

A public perception of MAFF's role grew that, far from being an impartial pursuit of the national interest, it was to articulate a thinly disguised but deeply ingrained reflex to defend the interests of industry—farmers and food manufacturers.[39] It would be unfair to accuse one political party of hold a prerogative over this policy; it was shared, albeit more enthusiastically by one than the other.[40] The vision of food progress through industrialised efficiency has been common to all governments since the 1947 Act. It is this vision that has now become the subject of considerable public scepticism, and which lies at the heart of the public reaction to BSE and distrust of Government discussed in the previous section. This distrust is now itself a factor in

[24] Gabriel, Y and Lang, T (1995). The unmanageable Consumer. London: Sage.
[25] Tiemstra, J (1992). Theories of regulation and the history of consumerism. International Journal of Social Economics, 19. 6, 3-27.
[26] Irish Food Board survey, quoted in Southey, C (1996). "German beef consumption falls by half". Financial Times, April 23. p 10.
[27] Beveridge, W (1928). British Food Control. Oxford: Oxford.
[28] Hammond, R J (1951). Food: The Growth of Policy. London: HMSO.
[29] Tracy, M (1982). Agriculture in Western Europe. London: Granada.
[30] Le Gros Clark, F and Titmuss, R M (1939). Our Food Problem. Harmondsworth: Penguin.
[31] Clunies-Ross, T and Hildyard, N (1992). The politics of industrial agriculture. London: Earthscan.
[32] Jenkins, R (1992). Bringing Rio Home; Biodiversity in our Food and Farming. London: SAFE Alliance.
[33] Lees, A and McVeigh, K (1988). An Investigation of Pesticide Pollution in Drinking Water. London: Friends of the Earth.
[34] World Health Organisation (1990). Diet, Nutrition and the Prevention of Chronic Disease. Technical Series, No. 797, Geneva: World Health Organisation.
[35] Raven, H and Lang, T, with Dumonteil, C (1995). Off our Trolleys? food retailing and hypermarket economy. London: Institute for Public Policy Research.
[36] Leather, S (1992). In National Consumer Council, Your Food, Whose Choice? London: HMSO.
[37] Department of Health (1996). Low income, food, nutrition and health: strategies for improvement. London: Department of Health.
[38] Walker, C and Cannon, G (1984). The Food Scandal. London: Century.
[39] Mills, M (1992). The Politics of Dietary Change. Aldershot: Dartmouth.
[40] Cannon, G (1987). The Politics of Food. London: Century.

undermining the capacity of government to manage the crisis. The public can see, if politicians have not, that narrow definitions of market efficiency led to the recycling of dead, diseased animals and their feeding to herbivores. This was cost-cutting at its most ridiculous.

Reform of MAFF

A review of food and farming policy is long overdue, as is a review of its delivery mechanisms. A longer paper arguing this case and specifically reviewing some of the key mechanisms of Government food policy, is under preparation by the present author and colleagues and will be published shortly.[41] A number of suggestions are made with regard to changes in the Ministry.

Their five options for reforming MAFF:

(i) Close it down and divide its responsibilities to other Ministries (notably the Departments of Health, Trade and Industry, and Environment).

(ii) Transfer responsibility for food quality and consumer protection to another body (such as the Department of Health or a Food Standards Agency, based on the US, Swedish or Australian models), leaving the Ministry with an industry promotion remit.

(iii) Transfer responsibility for agri-industrial promotion to the Department of Trade and Industry, and create a new Ministry of Food.

(iv) Retain MAFF but radically reform it.

(v) Leave it as it is.

The final option, in our view, is undesirable. National debate about these options is overdue and should be conducted on the basis of stated policy objectives. Reform of the Ministry's structure could easily be cosmetic—as happened with the creation of the Food Safety Directorate within MAFF. Initially welcome though that was, recent events have shown that unless wider policy as well as the Ministry's internal culture is changed, consumer confidence will be hard to re-build.

Public confidence will not quickly recover without such action. There are already worrying signs of the BSE crisis being subsumed into a wider, more ideological discourse concerning Britain's place in Europe. While that is understandable, it will do little to resolve the difficulty of regaining public confidence in a food product, public policy and government. Euroscepticism may or may not be in order, but it will not make people purchase or consume beef.

Intensification and exports: open and closed farming systems

British exports have been seriously affected, and government policy's reliance on trade liberalisation have been brought up short. There are already serious doubts about the Food from Britain philosophy or exporting more, to compensate for rising food imports. This has measurably harmful environmental effects and doubtful health or economic value.[42] This ideological package will lead to ever bigger farms, more intensification and a fragmented rural culture dominated by agribusiness with its eye on world markets rather than on feeding people with a health-enhancing diet.[43]

The lessons of BSE for food policy point in a different direction. We need more closed systems of farming—farms which produce food from start to finish, which grow their own feedstuffs if possible, which produce food for local markets, and do not just buy in inputs, process them a little, and sell them on. That direction for food production has led us to the absurd situation where cows now need a "passport" to ensure consumer confidence. Such certification is desirable, but is open to fraud unless tightly monitored. In general, we need shorter, not longer trading routes to build up trust between primary producer and end consumer. Hypermarket power is driving the food economy in precisely the opposite direction.[44]

Consumers are also calling for less intensive exploitation of farm animals.[45] More research needs to be done to establish the types of farms BSE appeared on, and their feeding and stocking regimes. BSE has never appeared, for example, on any organic farms, except for two cases where the calves were brought in. Yet MAFF has been extremely reluctant to entertain any policy conclusions.

BSE brings into question the main liberalising thrust of food policy in Europe for the last 20 years. Just when barriers to trade are coming down across Europe following the Single European Act 1987, and when humans no longer need passports to get between member states, cows now *de facto* have to have one. BSE

[41] Lang, T. Millstone, E, Rayner, M and Raven, H (1996). Modernising MAFF. London: Centre for Food Policy, Thames Valley University, forthcoming.

[42] Paxton, A (1994). The Food Miles Report. London: SAFE Alliance.

[43] Lang, T (1966). The public health impact of globalisation of food trade. Paper to 7th Public Health Forum. London School of Hygiene and Tropical Medicine. 1-3 April.

[44] Raven, H and Lang, T with Dumonteil, C (1995). Off our Trolleys? London: Institute for Public Policy Research.

[45] NOP poll for SAFE Alliance. SAFE Alliance (1992). What future? London: SAFE Alliance. p 4.

underlines the necessity for barriers, controls and restriction of movement. This is not old fashioned protectionism, but appropriate production of public health and the policy principle of a government ensuring security of food supply. BSE has undermined that.

The feedstuff industry: why compensate the renderers?

It should also be noted that the feedstuffs industry, in general, and the rendering industry, in particular, got off lightly and deserve rigorous scrutiny. If any other industry has wittingly or unwittingly helped devastate an industry, it would be allowed to go bust. In 1993, the Monopolies and Mergers Commission (MMC) had been critical of the largest renderers for being monopolies.[46] Indeed, involvement by the MMC and the Office of Fair Trading can be traced back further, to the time BSE is supposed to have begun.

In 1985, following an enquiry begun in 1982, the MMC judged that one company, Prosper de Mulder had a monopoly (defined as 25 per cent of a market in UK law), with 44 per cent of market share, yet six years later, the same company's market share had risen to 60 per cent and it was investigated for wanting to take over another with 5 per cent market share.[47] In 1993, the MMC found that Prosper de Mulder had "failed to fulfil certain of the undertakings it gave to the DGFT [Director General of Fair Trading] in 1986 following the MMC's earlier animal waste report."[48] Neil Hamilton, MP, Corporate Affairs Minister, asked the DGFT to seek undertakings that Prosper de Mulder would publish a sample of prices and charges on a weekly basis. In 1995, the MMC further censured the company for "discriminatory pricing."

With this track record, on what moral grounds have the renderers been given a taxpayers' hand-out of £110 millions?

4. Science and the perception of risk

Re-building a public interest ethos

The Government's index of consumer confidence in beef is, it seems, to be measured by beef sales, rather than consumer attitudes, values or concerns. Attitudes and values could be dismissed as long as the behaviour was correct and the cash tills kept ringing. This was a mistake and it is also an unnecessarily crude model of how people think about, purchase and consume food.[49] Food businesses have long learned that consumer confidence has to be earned, not anticipated.[50] Government now has to re-learn that same lesson.

Ironically, British Government used to be one of the most sophisticated, world-wide, in this respect, mainly due to its experience of relating with, and managing, consumers through the Ministry of Food in World War 2.[51 52 53 54] That Ministry was closed and merged into MAFF in the mid 1950s, and much of its public orientation and public health perspective lost. It is time to recreate that ethos. One mechanism for re-building this within MAFF could be the Minim system (the internal system of objectives) instituted by the present Government. The annual setting of Minim goals should be publicly debated.

The dangers of excessive reliance upon risk assessment: risk vs trust

BSE has highlighted the centrality of risk in public and individual decision-making. Risk assessment tools are widely used—for example in control of food poisoning risks—and have been widely promoted in the UK, following the Food Safety Act 1990. Excessive reliance on the technique can, however, pose risks of a different sort. Companies and Government can easily turn to risk assessment to try to manage and contain consumer volatility. If used in this way, it sets up a polarity between the expert—"we know best, don't worry your pretty little heads"—and the consumer.[55] Treat people like childen and they behave accordingly, quickly sensing victimhood.[56]

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[46] Monopolies and Mergers Commission (1993). Animal Waste in England and Wales and in Scotland. Cm 2340. London: HMSO. September.

[47] Monopolies and Mergers Commission (1991). Report on Prosper de Mulder Limited and the rendering business of Croda International PLC: a report on the merger situation. Cm 1611. London: HMSO. August.

[48] Department of Trade and Industry (1993). Press Release P/93/563. p 1.

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[56] Gabriel, Y and Lang, T (1995). The Unmanageable Consumer. London: Sage. p 117ff.

There is a burgeioning literature on risk assessment and its application both in "hands on" and wider policy contexts.[57 58 59] Companies specialise in risk control. Globally, in anticipation of the 1994 General Agreement on Tariffs and Trade (GATT), considerable thought has been expended on thinking through its application in trade contexts.[60]

Risk assessment is not without its critics. Some sociologists have argued that society is taking on a new cultural form, the so-called risk society where social institutions can no longer contain the risk that post industrialisation has engendered.[61] Science policy researchers argue that what is at stake is trust in institutions rather than risk itself and that excessive burden is placed on the victim to prove harm. Based on analysis of environmental crises such as Chernobyl,[62] Wynne has argued that the "breakdown of trust is more to do with the inability of expert institutions to frankly admit ignorance, contingency and lack of control when appropriate, than with the supposed growth of risk *per se*."[63] Others suggest that risk assessment is a version of cost benefit analysis and acts as a mechanism of control,[64] substituting diverse, democratically accountable standards for harmonised standards imposed "top down" in the interests of large traders, rather than local small and medium enterprises.[65 66]

Consumers' risk reducing behaviour: MAFF's own research

Despite this wider, theoretical debate about the role of risk assessment, the fact is that considerable research has been conducted on it. In 1993, MAFF was already considering the significiance of consumers' perception of food-related risk.[67] This work was addressing the importance of "assessing a risk tool-kit" for assessing consumers' perceptions "and taking them into account". The tool-kit was to include "improving communications about risk and formulating new policies." What has happened to this?

When choosing foods, studies have shown that even in low risk situations, consumers act to reduce risk.[68] The dimensions of perceived risks include the physical, financial, emotional, social and time. Such studies suggest that the government's reliance upon science to inform consumers about risk was entirely inappropriate. Instead of meeting consumers half-way and addressing their concerns, government advice was perceived as hectoring, irrelevant, inappropriate and insensitive. This failure of management of risk communication is one of the more serious failings to be exposed by the BSE saga.

Uncertainties in the scientific process

And what of government's own chosen area of evidence—the scientific advice much quoted by Ministers over recent years?

The BSE crisis has been in the public arena for eight years. Since the disease became public in 1988, many government reports have been written, parliamentary questions asked, conferences and reviews conducted.[69] The Commons Agriculture Select Committee reviewed the situation in 1990.[70] MAFF has done an admirable job in up-dating the chronology of the disease.[71] Not surprisingly, a positive torrent press coverage was unleashed.

Large sums of taxpayers' money have been expended on an extensive monitoring exercise, and even more

[57] Sapp, S, Harrod, W and Zhao, L (1994). Social construction of consumer risk assessments. Journal of Consumer Studies and Home Economics, 18, pp 97-106.

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[61] Beck, U (1992). Risk Society: towards a new modernity. London: Sage.

[62] Wynne, B (1191). Public Perception and Communication of risks: What do we know? The Journal of NIH Research, 3, pp 65-71.

[63] Wynne, B (1996). Patronising Joe Public. Times Higher Education Supplement. 12 April, p 13.

[64] Adams, J (1996). Risk. London: University College London Press.

[65] see Adams, J (1995). Cost Benefit Analysis: Part of the Problem, Nor the Solution. Oxford; Green College Centre for Environmental Policy and Understanding.

[66] Avery, N, Drake, M and Lang, T (1993). Cracking the Codex. London: National Food Alliance.

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[68] Mitchell, V W and Boustani, P (1992). Consumer risk perceptions in the breakfast cereal market. British Food Journal, 94 (4), pp 17-27.

[69] Department of Health & Ministry of Agriculture, Fisheries and food (1989). Report of the Working Party on Bovine Spongiform Encephalopathy. London: DoH/MAFF. February.

[70] Agriculture Committee (1990). Bovine Spongiform Encephalopaty (BSE). Fifth report. Session 1989-90. London: HMSO.

[71] eg. MAFF (1995). Bovine Spongiform Encephalopathy: A Progress Report. London: Ministry of Agriculture, Fisheries and Food. November.

on the scientific endeavour to understand the aetiology of the disease.[72] This effort has been driven by a concern to contain the epidemic and to prevent the disease jumping species to humans. Throughout the last six to eight years, a stream of reassurance from Ministers and Ministries was issued to the public to the effect that beef was entirely safe.[73] Despite a number of public relations gaffes—notably by Mr Gummer when feeding a hot burger to his daughter—the message was unchanged: British beef is 100 per cent safe. The source of this assurance was said to be the scientists, but this is not the whole picture. From the Southwood Report on, *caveats* have been expressed in print. Even as recently as November 1995, MAFF was admitting to difficulties in "accuracy of clinical diagnosis".[74] Uncertainties were endemic in that a multiplicity of scientific disciplines all added insights.

Politicians, however, were more categorical than they ought to have been. Although Mr Gummer has taken most public flak, his predecessor is not without blame in this respect. John MacGregor, MP, then Minister of Agriculture, made a written reply to a question about Southwood which talks not of certainty, but of risk. The "risk of transmission of BSE to humans appears remote and it is therefore most unlikely that BSE will have any implications for public health."[75]

Mr Gummer, too, placed considerable faith on categorical nature of science, arguing, for instance, against a ban on ruminant protein in pig and poultry feed thus: "Doctors and scientists see no need for that."[76] He reiterated that government policy was based on "the best independent scientific advice available", which is a mite more circumspect than the latter assertions that "beef is 100 per cent safe". By the mid 1990s, government statements had banished any doubts, to a degree that was unwarranted[77] and politically stupid. The Select Committee should ascertain why and how this transition occured.

Management of the 20 March announcement

Although accusations about causes, conspiracies and confusion have dogged BSE since its first appearance,[78] nothing has been of the order or global reach that was unleashed once Stephen Dorrell, MP, the Secretary of State for Health, made the announcement on 20 March 1996 that there was a "possibility" that ten recent cases of Creutzfeldt-Jakob Disease might be due to BSE. The policy context changed dramatically, as did the tone and tenor of the discussion.

It is important to cite Mr Dorrell's exact words. He said that "a previously unrecognised and consistent disease pattern" had emerged, and despite there remaining "no scientific proof that bovine spongiform encephalopathy can be transmitted to man by beef, . . . the Committee has concluded that the most likely explanation at present is that those cases are linked to exposure to BSE before the introduction of the specified bovine offal ban in 1989."[79] Statements from SEAC and the Chief Medical Officer were similarly worded.[80]

The reaction to this announcement was considerable. So much so that the Secretary of State joined with the Sun newspaper in the accusation that the public, not cows, were mad.[81] Yet no assessment of the Government's handling of this situation is worthwhile unless it accepts that public reaction to this momentous event was rational rather than irrational. Ministers might doubt public rationality, but such feelings were best kept private. By its own policy objective—the maintenance of beef sales—it was a disaster. Sales halved, companies withdrew products, a world-wide ban ensued.

Trying to woo consumers back: price and Euro-bashing

Because the Government was eight years into its strategy of maintaining beef while banning Specified Bovine Offal, the only mechanisms left to it were piecemeal. To retailers, the main mechanism was price. The triumphant announcement by, first, J Sainsbury and then Asda, that consumers had flooded back, when induced by low beef prices were predictable. Consumers who wanted to eat beef would take advantage of bargains. The figures collected by the Irish Food Board give the lie to the assertion that confidence was back:

[72] See Consultative Committee on Research into Spongiform Encephalpathies (1989). Interim Report. London: Ministry of Agriculture, Fisheries and Food & Department of Health. June.

[73] eg MAFF (1990). BSE Information Pack, London: Ministry of Agriculture, Fisheries and Food Food Standards Division.

[74] MAFF (1995). Bovine Spongiform Encephalopathy: A Progress Report. London: Ministry of Agriculture, Fisheries and Food. November. p 5.

[75] MAFF (1989). The Government's Response to Southwood Report on BSE. News Release 84/89. London: MAFF, p 1.

[76] MAFF Food Safety Directorate. John Gummer's Statement on BSE. FSD 19/90. London: MAFF, p 2.

[77] Pattison, W J and Dealler, S (1995). Bovine Spongiform Encephalopathy and the public health. Journal of Public Health Medicine, 17, 3, 261–268.

[78] Lacey, R (1994). Mad Cow Disease: the history of BSE in Britain. St Helier: Cypsela.

[79] Hansard. 20 March 1996, column 375.

[80] Statements by Spongiform Encephalopathy Advisory Committee and by the Chief Medical Officer. London: Department of Health, 20 March (reproduced in MAFF Food Safety Information Bulletin, 72, April 1996, pp 21–23.)

[81] Dorrell, S (1996), interviewed on BBC Radio 4 Today Programme. 26 March.

the picture was, and is, more complex. Like many issues in contemporary consumerism, confidence was fragmenting. Some consumers had never lost it, some had never had it, some had had theirs weakened, and some were still making up their minds. The management of risk perception is itself a highly volatile entity.

Mixed messages about risk from BSE since 1989

Government had for years made what it is now suspected were unwarranted, categorical statements about beef being 100 per cent safe. Yet, for years, slightly different messages were also dispatched in the form of the gradual tightening up of regulations. Excellent chronologies of events have been published by MAFF[82] and Professor Lacey.[83] From the consumers' point of view, messages were mixed. On the one hand, reassurance was being given about safety to an almost absurdly high degree, while, on the other hand, regulations about removal of specified bovine offal were being tightened up, thereby implying that perhaps they should have been tighter earlier. MAFF's own monitoring in 1995 underlined and contributed to this gap. Spot checks on abattoirs found a lamentable degree of failure to implement the Specified Bovine Offal orders. The September check found 48 per cent establishments visited failing, and the October visit found 34 per cent failure.[84] It is anticipated that subsequent spot checks will show a better performance.

In trying to explain the slow response of the disease curve to the ban on specified bovine offal,[85] MAFF has argued that "there has been some continued leakage of BSE infected material into animal feed".[86] The spot checks suggest this is, indeed, so, but it is still unclear whether this explains the 25,000 animals which have gone down with BSE born after the SBO ban. Only the experiment MAFF is conducting into maternal transmission will begin to answer the other leading interpretation.

From the consumers' point of view, it has hardly been reassuring that throughout the BSE saga, MAFF's prognoses that the disease incidence would decline have had to be delayed. This predictive failure suggests that the aetiology is not fully understood—hardly surprising in the face of such a new disease.[87] Pronouncements of certainty should not be made in such circumstances, if they arise again.

MAFF and the government continued to act and make pronouncements to the public as though they were in full control of the facts. To add insult to injury, the SBO spot checks suggest, not just a failure to enforce the SBO ban, but the morality and self-regulation of the feedstuffs industry.

The failure to enforce the SBO order raises questions about the validity of Mr Dorrell's March 20 statement that the 10 CJD cases were linked to exposure in the period up to 1989. Exposure has probably been longer. Since early days, abattoir workers have complained about speed-up on the line, about the pressure to maintain a high through-put of carcasses. What is astonishing is, not that public reaction was so great from 20 March onwards, but why it was not greater from 1995 on.

Handling Europe and the world

When the 20 March announcement was made, the story went world-wide. The speed and scale of the dynamics intensified. The European Commission imposed a world-wide ban on UK beef exports, thereby killing off a prime export market which accounted for around 20 per cent of UK production. This action then engendered a furious counter-reaction from government and its backbenchers which argued, with some success, that this was an example of heavy-handed European bureaucracy; 55 per cent in the ICM poll agreed the EU was "over-reacting and being unfair".[88] In a matter of days, a national public health issue became a site for a full-blown political show-down on a world scale. Beef consumption even fell dramatically in the rest of Europe.

According to figures from the Irish Food Board, consumption was down in the week of 15 April, compared to the period before Mr Dorrell's announcement on 20 March, by 45 per cent in Germany, 40 per cent in France and 30 per cent in Spain and Italy. In the UK, it was down by 36 per cent.[89]

[82] MAFF (1995). Bovine Spongiform Encephalopathy: A Progress Report. London: Ministry of Agriculture, Fisheries and Food. November, appendix 1.

[83] Lacey, (1994). Mad Cow Disease: the history of BSE in Britain. St Helier: Cypsela. pp xii–xv.

[84] MAFF (1995). Bovine Spongiform Encephalopathy: A Progress Report. London: Ministry of Agriculture, Fisheries and Food. November, appendix 3.

[85] Lacey, R (1994). Mad Cow Disease: the history of BSE in Britain. St Helier: Cypsela. p 127-128.

[86] MAFF (1995). Bovine Spongiform Encephalopathy: A Progress Report. London: Ministry of Agriculture, Fisheries and Food. November. p 5.

[87] Dealler, S (1996). Lethal Legacy. London: Bloomsbury.

[88] Linton, M and Bates, S (1996). "Public suspects beef cover-up", The Guardian, 3 April, p 1.

[89] Irish Food Board figures, quoted in Southey, C (1996). "German beef consumption falls by half". Financial Times. 23 April. p 10.

Membership of Government's scientific committees

It could be argued that a better, more sensitive approach to consumers might have mitigated this dire effect. Only quick action, in the early days of BSE (1988–89), could have retained public confidence in government. Instead, the Government displaced focus for action upon a tiny number of scientists. This strategy has now undermined science itself. How has this happened?

Over the last eight years, Government advice on BSE—at least that in the public arena—has been taken from three over-lapping committees: Southwood (a Working Party), Tyrrell (a Consultative Committee)[90] and SEAC (an Advisory Committee). The latter has been the key body and under the chairmanship of Professor John Pattison since October 1995. Until a time coinciding with the appointment of Professor John Pattison, membership of these committees was drawn from too narrow a pool of expertise. In my submission to the 1990 Agriculture Committee inquiry, I argued that this was foolhardy, and that consumer representatives should be drawn into the deliberations.[91] This did not happen.

The failure to include consumer and other representatives on key committees such as SEAC lies at the heart of the failure of Government since 1988. The membership of SEAC should be widened to include consumer, environmental health and social science expertise. Lessons should be learned across Government in this respect.

April 1996

[90] MAFF (1989). Consultative Committee set up to look at research into BSE and other spongiform encephalopathies. News Release 85/89. London: MAFF.

[91] Agriculture Committee (1990). Bovine Spongiform Encephalopathy (BSE). Fifth Report. London: HMSO. Appendix 5.

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