3. Epidemiology

Introduction

3.1 In this chapter we follow the course of the investigation into the cause of BSE carried out by the CVL Epidemiology Department. We see how Mr Wilesmith, its head, identified feed as the probable source of BSE infection. More particularly, he identified meat and bone meal (MBM) derived from rendering offal from scrapie-infected sheep as the likely infectious ingredient in the feed.

3.2 In the latter part of the chapter we discuss the conclusions reached by Mr Wilesmith as to why BSE emerged when it did, and explain why, with the benefit of hindsight, we have formed the view that these conclusions were erroneous.

Initial investigations and conclusions

3.3 In May 1987 the CVL had a small Epidemiology Department headed by Mr John Wilesmith. Mr Wilesmith was the only qualified epidemiologist at the CVL, having taken, after his veterinary training, a postgraduate degree in medical statistics and epidemiology at the London School of Hygiene and Tropical Medicine. His department consisted of two veterinary research officers (VROs), a mathematician/statistician, a systems analyst, five scientific officers skilled in computer programming, and five clerical staff. The department conducted research on diseases of national, economic and public health importance and on novel diseases of animals.

3.4 Mr Wilesmith knew nothing of BSE until Dr Watson, the Director of the CVL, visited his office in late May 1987 and told him about the newly identified disease in cattle. Dr Watson asked him to investigate its epidemiology.

3.5 On 3 June 1987 Mr Wells gave Mr Wilesmith a more detailed briefing. This included a description of the clinical symptoms and the basic histological features of the disease. He told Mr Wilesmith that histopathological examination had confirmed six cases on four farms in three different areas, and that clinical histories suggested that there had been earlier cases in 1985. The possibility that the disease was similar to scrapie was discussed and Mr Wells said it was possible that the cases had a toxicological cause.

3.6 Mr Wilesmith designed a questionnaire for use on farm visits with the object of eliciting, in the case of each casualty, any information that might have a bearing on the cause of the disease. This included exposure of individual diseased animals to various possible sources of infection such as feed, vaccines and other disease prevention methods, herbicides and pesticides, and contact with sheep. The
pedigree of each animal was explored in order to see whether the disease might have a genetic source.287

3.7 A week later, on 9 and 10 June, Mr Wells held discussions with Dr Kimberlin of the NPU in Edinburgh about a future research programme for BSE. Dr Kimberlin gave advice on epidemiology, recommending investigations into herd structure, age and management practices and the influence of these on the recognition of clinical signs; a common use of bulls; connections with sheep; common denominators of a nutritional, infectious or genetic nature; and, in particular, sources of animal protein in feedstuffs. Mr Wells passed this advice on to Mr Wilesmith.288

3.8 When Mr Wilesmith gave evidence, he said that Dr Kimberlin’s reference to animal protein was probably aimed at the possibility of scrapie having infected cattle feed via the rendering process.289 He added that this was a possibility that they all had in mind at the time, but that he, Mr Wilesmith, was more interested at that point in the possibility of a contaminated vaccine.290

3.9 Armed with the questionnaire, Mr Wilesmith began to visit farms on which confirmed or suspected cases of BSE had been reported. The number of these increased rapidly as the investigation progressed. Mr Wilesmith was looking for some common novel factor to explain the outbreak of the disease. At first this led him to discount the possibility that a component of the feedstuffs was involved in the aetiology.291 The widespread use of organophosphates and synthetic pyrethroids as insecticides appeared to have potentially greater significance. By the end of the year Mr Wilesmith had, however, discounted these for two reasons. Histological examinations conducted by Mr Wells had demonstrated that the pathological changes associated with BSE differed from those induced by toxicity. Perhaps more significantly, no common factor could be identified in terms of the use of vaccines, hormones, organophosphorous fly sprays, synthetic pyrethroid sprays or anthelmintics.292

3.10 On 30 July 1987 Dr Watson noted that Mr Wilesmith was under considerable pressure as he did his best to obtain detailed information from all affected herds by personal visits. On instructions from Mr Rees, Mr Cranwell was seconded from Starcross VIC in Exeter to assist him in making farm visits and recording data.293 During August 1987 the Epidemiology Department began to develop a computer database to record and analyse the data.294

3.11 On 26 August, by which time 15 farms had been visited, Mr Wilesmith recorded that the potential role of feedstuffs in the aetiology was uncertain: ‘Lamb meat and bone is evidently used in commercial dairy rations, but this is not a recent introduction.’ Other ingredients under consideration were dried cassava, tapioca and maize gluten meal.295

3.12 On 27 August Mr Bradley and Mr Wilesmith made a joint report to Dr Watson which proposed the tentative hypothesis that ‘BSE is caused by an unconventional

287 S91 Wilesmith Annex 1
288 YB87/6.17/2.1–2.5
289 T35 p. 84
290 T35 p. 90
291 YB87/7.23/1.1
292 Veterinary Record, vol. 122, 25 June 1988, p. 641. Anthelmintics (or antihelminthics) are substances used to treat parasitic worms
293 YB87/7.30/2.1
294 S91 Wilesmith para. 26
295 YB87/8.26/1.1
transmissible agent coupled with unknown genetic factors’. They recommended that transmission studies in mice, rats, cattle and possibly mink should be started immediately to test this hypothesis. However, they advised that it would be premature to eliminate other potential causes of the disease.  

The identification of feed as a possible cause of infection

3.13 During June, July and August of 1987 Mr Wilesmith accumulated data on the pedigree of animals in affected herds. He examined the possibility of a genetic factor in the incidence of BSE with Dr Vernan Wijeratne, at that time a veterinary geneticist in the CVL’s Animal Production Department. While autosomal dominant inheritance could be confidently excluded, Dr Wijeratne thought that BSE might be caused by an autosomal recessive gene. Data on the sources of semen used for artificial insemination were also collected. On 10 March 1988, Mr Wilesmith reported his conclusion that the data did not support an autosomal recessive mode of inheritance, which indicated that BSE was not solely a genetic disease.

3.14 On 10 November 1987 Mr Wilesmith attended a meeting at Tolworth chaired by Mr Rees, the CVO. He informed the meeting that 32 cases had now been confirmed on 29 farms and that a further 96 suspect cases on 50 farms had been identified. Although 54 farms had only one case, one farm had had 11 cases. In one area in Devon there were 14 herds in close proximity with either clinically confirmed or suspect cases of BSE. The note of the meeting records that:

> The animal feed aspects were being looked at – ovine offal can be incorporated in dairy feed – UKASTA (Judith Nelson) has been advised of the work undertaken on feed.

3.15 Judith Nelson has no recollection of being informed of a suspected link between feed and BSE at this time, nor do UKASTA’s documents record that she was so informed. UKASTA’s evidence is that they did not learn of the suspected link until March 1988.

3.16 On 27 November 1987 Dr Watson asked Mr Bradley to provide answers to a number of questions. One of these, relating to BSE, asked: ‘Should we approach the cattle feed industry?’ Replying in writing the same day, Mr Bradley answered:

> Not yet. We must first identify the right questions to ask and we have more data to collect yet. We also need time to think. If we approach too early concealment of information is likely. The direct question is more likely to extract answers or identify areas to probe further rather than asking ‘what changes, additions, sources have you put in feed since 1981?’
3.17 Over three months were to elapse before a direct approach seeking information and assistance was made to the feed industry.

Covert investigation of the feed theory

3.18 On 15 December 1987 another meeting was held at Tolworth, chaired by Mr Rees, at which Mr Wilesmith was present. The note of the meeting records:

Studies appear to show that there may not be a genetic link in relation to this condition. However, there was some evidence that feed (meat and bone meal including material from sheep) might be a factor. 303

3.19 This significantly understated the position. Mr Wilesmith told us that by this stage he was sufficiently confident that BSE was associated with a feed-borne source and that there was no longer any need to canvass some of the other possibilities in the questionnaires that were addressed to affected farms. Commercial concentrates, either as finished rations – such as pelleted calf feed and dairy cow cake – or as protein supplements used in home-mixed rations, had been fed at some time to all the cases for which accurate records were available. 304

Mr Wilesmith concluded that the source of infection was most likely to be meat and bone meal (MBM), although other animal-derived products such as blood and tallow could not be excluded at that stage. 305

3.20 Mr Rees told us that by 15 December 1987 they were fairly sure that MBM was indeed the cause. 306 The note of the meeting goes on to record that it would be useful to have more information from the feed industry and that Mr Keith Meldrum (at that point Director of the Veterinary Field Service) would arrange an ‘off-the-record’ meeting with his contacts.

3.21 Mr Wilesmith prepared a report covering the period up to the end of December 1987: 370 clinically suspected cases had been reported on 237 farms. Of these, 132 cases on 113 farms had been histopathologically confirmed. Completed questionnaires had been received for 90 of these herds. The source of the commercial dairy concentrates used was recorded for 60 of those herds. No single compounder was common to all herds. Mr Wilesmith commented:

Although epidemiological findings support the introduction of a transmissible agent via cattle feedstuffs, it is not obvious, at the present time, how and why this has occurred. The most likely vehicular constituent of cattle feedstuffs is meat and bone meal or other animal derived products such as tallow or blood. Meat and bone meal has been used in cattle feedstuffs for some time usually at an inclusion rate of 3–5%. The analyses of data, in progress, arising from surveys of the composition of cattle feedstuffs will provide evidence of changes in this inclusion rate. At present it is not possible to determine whether the exposure of cattle to a foodstuff-borne agent was for only a short period or is continuing. The studies in progress will assist in this respect. 307

303 YB87/12.15/5.2
304 Veterinary Record, vol 123, 17 December 1988, p. 643
305 S91 Wilesmith para. 32
306 T54 p. 91
307 YB87/12.31/5.8
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A footnote to the report emphasised that:

The abstract should not be entered on the database. There must be no possibility that the feedstuffs industry becomes aware of the current hypothesis. 308

3.22 We were puzzled by the reticence shown by MAFF officials in making confidential enquiries rather than a direct approach to UKASTA for assistance. Mr Rees in his written statement to the Inquiry said:

The investigations into the origin of BSE infection were concentrating on contaminated meat and bone meal (‘MBM’) and tallow in feedstuffs as being the possible vehicle of infection. In order to find out more about the use of MBM and tallow, the co-operation of the feedstuffs industry was needed. Advisers to the feedstuffs industry were very reluctant to assist at first, because of their need to keep the commercial confidence of the various companies they worked for. However, in these early stages we did manage to have some very informal discussions with a few advisers who we knew on a more personal basis, to determine the extent of the use of MBM in commercial feedstuffs, and the distribution of MBM. These were really very informal chats as no one wanted to meet formally with MAFF at this stage. I was hoping I would eventually be able to persuade the industry to help MAFF more formally in the future. 309

3.23 When we pursued this with Mr Rees in oral evidence, it transpired that any reluctance on the part of the feed industry to assist MAFF was more apprehended than real. Mr Rees told us of a single conversation that he had, on a personal and confidential basis, with an adviser to a feed manufacturer. While willing to assist, the adviser was not able to give very sound information as to the content of animal feed four or five years in the past, which was when Mr Wilesmith had calculated most instances of infection must have taken place (see paragraph 3.28 below). 310

3.24 We asked Mr Meldrum why it had been agreed that he would meet his contacts on an ‘off-the-record’ basis. He explained that Mr Wilesmith’s attribution of the cause of BSE to meat and bone meal was only a preliminary view:

When you are dealing with a very early investigation or some very early reports, it is better to keep the investigation to yourself and release the information in a proper and reasoned and planned way when it becomes substantive. 311

3.25 Mr Meldrum told us of a detailed discussion that he had with Mr Paul Foxcroft of Prosper De Mulder (the leading renderer in England and Wales) about current rendering practices. 312 It may be that he did not disclose the reason for his interest, or it may be that the conversation took place a little later in the story. Either way, Mr Foxcroft informed us that he was absolutely certain that he was not aware of any suggestion of a link between BSE and animal feed until March 1988. 313

308 YB87/12.31/5.10
309 S126 Rees para. 36
310 T54 p. 79
311 T69 p. 43
312 T69 p. 48; S184 Meldrum para. 40
313 T60 p. 6
3.26 Early in January 1988 Mr Wilesmith and Mr Meldrum had a meeting with Dr Laurson-Jones, a veterinarian who had worked for BOCM Silcock (a feedstuff manufacturer), and Mr Mike Stranks of the Bristol regional office of ADAS. Each confirmed that significant amounts of UK-produced MBM were included from time to time in compound rations of both calves and adult cattle.314

3.27 On 17 December 1987 Mr Wilesmith had asked Mr Gallehawk, of the Statistical (Agricultural Commodities) Division of MAFF, for information on the composition of commercial cattle feed over the previous ten years. The data that he supplied showed that there had been no increase, or new use of MBM, in cattle feed between 1979 and 1987.315 This information gave no indication of any new factor to explain the emergence of BSE at the end of this period. An important question in this context was the date at which cattle were first subjected to infection. This was a matter to which Mr Wilesmith had been giving consideration.

3.28 By the end of 1987 there had been a sufficient number of victims of BSE to enable Mr Wilesmith to carry out what he described as ‘relatively simple simulation studies’ based upon the age-specific incidences of BSE. He assumed that the incubation period had a log normal distribution, in line with common incubation periods for infectious diseases. Applying this assumption, the simulation suggested that effective exposure of the cattle population was likely to have begun in the winter of 1981–82 and continued until the end of 1984 at least, and that the risk of effective exposure was considerably greater for calves than adults. Calves were either exposed to more of the infectious agent or more susceptible to the exposure, or a combination of both.316

3.29 The results of these studies led Mr Wilesmith to look for some change or changes of exposure in or about 1981. The data received from Mr Gallehawk appeared to rule out any change in diet at this time,317 which led him to consider whether any of the processes in the animal feed industry had changed at the relevant time.

3.30 As to these, Mr Gallagher of the Starcross VIC had been making enquiries about processing at a large renderers in Exeter, which formed part of the Prosper De Mulder Group. He reported that the plant received a large intake of sheep material from both south-west England and South Wales from which an average weekly production of 200 tonnes of MBM was extracted. The temperatures reached during the processing would not destroy the scrapie agent. Distribution from the plant was widespread, although a large bulk was taken up by compounders in the South West.318

3.31 Mr Gallagher ended his report with the observation that, clearly, a central initiative to the industry was the next step.319 Mr Wilesmith had reached the same conclusion. In a note to Dr Watson dated 14 February 1988 Mr Bradley stated that Mr Wilesmith now had enough data to go to the industry with specific questions to answer about feed. He added: ‘Clearly this needs delicate handling.’320

314 S91 Wilesmith para. 38; S184 Meldrum para. 40
315 S91 Wilesmith paras 31 and 38
316 S91 Wilesmith para. 34
317 S91 Wilesmith para. 38
318 YB88/2.15/1.1
319 YB88/2.15/1.2
320 YB88/2.14/1.1
3.32 Meanwhile, at the end of January 1988 Mr Wilesmith received some information which tended to confirm his hypothesis that MBM was the source of infection of BSE. In July 1986 a nyala at Marwell Zoological Park in Hampshire had succumbed to a scrapie-like spongiform encephalopathy. In June 1987 a gemsbok succumbed to a similar condition in the same zoo. Investigation showed that between March 1986 and March 1987 the pelleted antelope diet in use, manufactured by Bockley Mills, contained a meat-based product supplied by Prosper De Mulder in Exeter.321

3.33 MAFF officials set out the position with a degree of caution in a submission sent by Mr Andrews (MAFF Permanent Secretary) to the Minister, Mr John MacGregor, on 24 February 1988. This stated:

Although it is not possible to draw conclusions from the work carried out so far a plausible hypothesis is that BSE may be caused by an unconventional transmissible agent which could have been introduced into affected animals through feedingstuffs derived from the carcasses of animals such as sheep which are known to be susceptible to encephalopathies.322

3.34 Mr Andrews added this comment in a covering minute:

There are a number of reasons for thinking the disease may have originated from some change in feeding practice, possibly as a result of the incorporation of animal residues in feedingstuffs. This line of inquiry is being explored and I am sure that we need to pursue these investigations urgently in order to decide whether some action should be taken to modify the ingredients in animal feedingstuffs.323

3.35 On 26 February 1988 Mr Wilesmith produced a report on his investigations to date. This explained that the only relevant factor common to all BSE cases appeared to be that commercial concentrates were fed to the adults in all affected herds, although no one compounder was common to all herds. In respect of calf feeding, some form of concentrate was the regime in all but one farm. He concluded:

The finding of scrapie-associated fibrils (SAFs) in a proportion of histopathologically confirmed cases is undoubtedly significant in terms of the aetiology; SAFs have only been observed in encephalopathies associated with transmissible agents. The absence of any evidence from the epidemiological investigation of other sources of such an agent together with the occurrence of a common source epidemic strongly suggests the introduction of an infectious agent via commercial cattle feedstuffs.324

The feed industry consulted

3.36 On 29 February 1988 Mr Wilesmith provided his veterinary colleagues with a summary of his findings in relation to the composition of cattle feed. He noted that Prosper De Mulder’s Exeter plant was the last to cease solvent extraction of tallow

321 S91 Wilesmith para. 37; YB88/1.27/1.1
322 YB88/2.18/7.2
323 YB88/2.24/1.1
324 YB88/2.26/5.3
in 1984. The change had come about because the value of tallow had dropped, so that this method of extraction was no longer economical, and in any event compounders were producing feed with a higher fat content. No change in the average rate of inclusion of MBM in dairy rations had occurred during the 1980s. Meanwhile, Prosper De Mulder had increased its market share of industry to the extent that, by 1985, it handled 50 per cent of all animal by-products. (See vol. 13: *Industry Processes and Controls* for a description of the rendering industry.)

3.37 Mr Wilesmith had been informed that, as a result of renderers establishing links with knacker’s yards throughout the country, more sheep carcasses were entering their rendering plants. Information on the details of the rendering processes in the various plants was not available. Mr Wilesmith concluded:

I would suggest that we have obtained as much information about the composition of rations that can be obtained from existing data and through surreptitious enquiries.

I feel the following questions need to be put to the industry.

(a) What variation in the rendering processes has there been over time to produce MBM and tallow and what variation is there in these processes between plants?

(b) How much MBM and tallow has been produced by the various plants which has been used in cattle rations?

(c) What changes have there been in the inclusion rate of tallow in all cattle feedstuffs?

(d) What changes have taken place in the inclusion of sheep material to produce MBM and tallow?

(e) What are the real possibilities of tracing MBM and tallow from rendering plants to compounders, particularly if there is a significant variation between plants?

3.38 So far as the last question is concerned, a number of people had suggested to Mr Wilesmith that it would be impossible to trace the source of the products used by feed compounders because their practice was to buy through brokers rather than directly from the rendering plants.

3.39 In March 1988 Mr Rees asked Mr Meldrum to head a Task Force, to include Mr Wilesmith, Mr Gallagher and Mr Stranks among others (see paragraph 3.44 below). The Task Force was to visit all the main rendering plants in order to obtain full details of supplies of raw material, processing methods and distribution of the finished product, and any changes which occurred in relation to these in the early or mid-1980s. Mr Rees asked for a report by the end of the month so that further action could be considered.
3.40 On 8 March Mr Meldrum, Mr Wilesmith, Mr Gallagher and Mr Alan Lawrence (of MAFF) met with representatives of UKRA, UKASTA and GAFTA after having held initial meetings with these Associations individually. At the meeting:

The renderers took careful note of what we said, and made no attempt to challenge our tentative conclusions. In response to our request for co-operation in further studies, their Chairman, Mr Field, said he was convinced that nobody in the rendering industry would be so irresponsible as to refuse to co-operate fully. They will therefore be nominating representatives to join with the UKASTA and GAFTA representatives in assisting our task force.

They provided considerable information about the different rendering processes currently in use and about some of the changes in rendering that had occurred over the past decades. Among other things, it was suggested at the meeting that there had been a significant reduction over the past ten years in the harvesting of sheep brains for human consumption, so that over 90 per cent were being left in the heads, which were going for rendering.

3.41 MBM had become a constituent of cattle cake in the late 1960s. Thereafter there had been an increase in its fat content in order to provide a high-energy feed until 1984, when milk quotas were introduced. After this the fat content had been reduced to satisfy the need for cheaper concentrates.

Changes in the production processes of MBM identified

3.42 Brokers were frequently used in the marketing of the products of rendering plants. Eighty per cent of MBM would be distributed within a radius of 100 miles, but the balance would go further afield. The industry representatives agreed to attempt to trace the source of feedstuff components that had been supplied to a number of farms that had experienced BSE.

3.43 On 30 March 1988 Mr Andrews met with Mr Rees, Mr Cruickshank and Mr Bradley. Mr Rees said that it was:

more and more likely that the source of the disease lay in feedingstuffs and, in particular, in a combination of factors related to changes in procedures in the early 1980s. We were still investigating precisely what those procedures were and how they had altered. It was agreed that this work should be pursued vigorously regardless of what else was undertaken. It might be possible, by urgent action, to cut off the source of material for cattle . . . The industry was likely to be willing to follow a code of practice pending further legislation.

329 UKRA = UK Renderers’ Association; UKASTA = UK Agricultural Supply Trade Association; GAFTA = Grain and Feed Trade Association
330 YB88/3.4/4.1
331 YB88/3.8/1.5
332 YB88/4.6/4.1
3.44 The investigations to which Mr Rees referred were being carried out by members of the Task Force led by Mr Peter Smith, a Senior Scientific Officer from the VIC in Preston. He produced an interim report on 28 March 1988 and a further report on 27 April. Mr Smith visited rendering plants with the objective of establishing what changes may have taken place in the feed industry to lead to the current problem. He was accompanied on some of these visits by Mr Meldrum and Mr Wilesmith. He inspected 11 renderers and obtained data on 16 more, out of a total of some 58 plants in the United Kingdom. He reported:

. . . cooking times and temperatures are poorly recorded, inaccurate and in general do not provide the theoretical limits necessary to destroy the scrapie agent which can survive temperatures of 136°C for 18 minutes.

3.45 Mr Smith reported that, since 1980, there had been a change in the nature of the material being processed, for the following reasons:

(a) The sheep population has increased from approximately 22 million to 35 million resulting in more sheep material being processed.

(b) There has been a reduction in the number of operators (200 in 1960; 90 in 1980; 58 in 1988) resulting in more waste material being processed in fewer plants.

(c) Knacker’s yards are thought to have decreased in numbers resulting in more casualty and condemned animals entering the rendering industry.

(d) The practice of skinning sheep heads and harvesting of sheep brains has almost ceased resulting in more whole unsplit heads going for rendering.

(e) There has been an increase in the number of cases of scrapie each year as measured through the VIDA II returns (1,083 extra flocks since 1980). In practice the number of actual cases which are unrecorded will be much higher. These casualty animals or slaughtered carcasses together with subclinical cases will end up in the rendering industry in increasing numbers.

3.46 Mr Smith also found the following changes in the processes used to produce tallow and MBM:

(a) Solvent extraction has almost disappeared: only two operators are thought to exist.

(b) Low temperature renderers have commenced operations.

(c) There has been a drift from batch cooking to continuous rendering in response to the needs for economy of scale to retain profit margins.

333 YB88/3.28/1.1–1.6
334 YB88/4.27/5.1–5.6
335 S45 Smith para. 2
336 YB88/4.27/5.2
337 YB88/4.27/5.2; VIDA = Veterinary Investigation Diagnostic Analysis
(d) Stord Bartz systems have been introduced which seem to operate at lower temperatures than Stork Duke systems.

(e) The continuous rendering systems will probably reach lower temperatures than batch rendering due to continuous movement of material in the cooker and displacement with cold raw material.\textsuperscript{338}

3.47 His conclusions were as follows:

The rendering industry operates with processes which with few exceptions are inadequate to destroy scrapie agent. This together with increasing amounts of sheep material consisting of whole carcasses and heads which undoubtedly contain increasing numbers of scrapie cases may have resulted in contaminated tallow and meat and bone meal reaching the animal feed compound industry.

Scrapie contaminated material has probably always entered and survived the rendering process but only in small amounts prior to 1980. With the change to continuous rendering large volumes of contaminated products have been introduced to the food chain.

It is not possible to say what the critical limit is for agent relative to final feed concentration necessary to cause infection. Equally it is not possible to say with certainty which plants may be responsible for the present problem but it would seem likely to be one operating in the South West or South East due to the usual practice of distributing finished product mainly to the local area. These geographical areas are those which are prominent in the number of cases of BSE. Further dissemination of finished product will obviously occur but only on a limited scale.\textsuperscript{339}

Feed identified as a common factor

3.48 On 8 April 1988 Mr Rees chaired a meeting of MAFF officials and representatives from UKASTA, GAFTA and UKRA. The industry representatives were brought up to date with the results of the Task Force investigations. They were told that the detection of SAFs in the brains of affected cattle had pointed to BSE being a scrapie-like condition which had crossed from sheep to cattle. Feed had been identified as a common factor and the most likely cause of the disease. An increase in the amount of scrapie-infected material rendered down to MBM and incorporated in cattle feed was a suspected culprit. The UKASTA representatives again offered to help in attempting to trace the sources of feed supplied to affected herds.\textsuperscript{340}

3.49 On 13 April Mr Wilesmith sent to Miss Nelson, of UKASTA, details of the feedstuffs that had been used in the case of 50 affected herds, together with the compounder or supplier, in the hope that it would prove possible to identify the source and inclusion rates for at least a proportion of these feedstuffs.\textsuperscript{341}

\textsuperscript{338} YB88/4.27/5.2
\textsuperscript{339} YB88/4.27/5.3
\textsuperscript{340} YB88/4.8/5.1–5.3
\textsuperscript{341} YB88/4.13/4.1
3.50 Mr Wilesmith subsequently discovered that in the case of 20 of the herds the suppliers had been Cooperatives who were members of the Federation of Agricultural Cooperatives (FAC) and not of UKASTA.342 So far as 12 of the herds were concerned, Dr Brian Cooke of Dalgety Agriculture Ltd, the supplier of the feed, was optimistic that the company’s mill records would enable the sources of the feedstuffs to be identified. Requests for similar assistance were made to the other three companies which, with Dalgety, were on the UKASTA scientific committee – BOCM Silcock Ltd, J Bibby Agricultural Ltd and Pauls Agriculture Ltd.343

3.51 On 14 April 1988 the Minister of Agriculture, Mr John MacGregor, held a meeting at which two other MAFF Ministers, Mr John Gummer and Mr Donald Thompson, were present, together with Mr Andrews, Mr Edward Smith, Mr Meldrum, Mr Wilesmith and other MAFF officials. Mr Meldrum reported on the current state of the feedstuffs investigation, saying that while it was too early for any definitive results, it was evident that in the early 1980s a change in production processes had occurred. They hoped to have a further report in the next two or three weeks.

**MBM the likely factor**

3.52 The rendering process produces MBM and tallow. Mr Wilesmith was concerned to discover the relative risks of tallow and MBM which contained the scrapie agent. On 5 April 1988 he wrote to Dr Hope at the NPU seeking his advice on this point and enclosing the reports that had been prepared by Mr Peter Smith in relation to the rendering processes.344

3.53 Dr Hope replied on 27 April expressing the view that no guarantee could be made that scrapie infectivity was absent from any grade of waste. He suggested that laboratory experiments could provide guidance on the relative risks of tallow and MBM containing BSE/scrapie infectivity.345 Mr Wilesmith subsequently discussed this matter with Dr Kimberlin, who had just left the NPU to set up as a private consultant. Dr Kimberlin expressed the view that scrapie-like agents would be more likely to partition with the MBM fraction because of their ‘membrane adherence’ properties.346

3.54 On 26 April Mr Meldrum asked Mr Wilesmith to produce a report within the next two days to enable Tolworth to prepare a final paper for submission to Whitehall. He added: ‘I apologise for the rush but we do need to come to some firm conclusions by the end of this week.’347 Mr Wilesmith replied the following day, saying that he could not meet this deadline, and summarising the state of play as follows:

In brief we have no conclusions to explain why BSE should have occurred from the findings of the various investigations on the feedstuffs industry thus far.348

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342 YB88/4.15/1.1–1.2
343 YB88/4.18/1.1; YB88/4.18/2.1
344 YB88/4.3/1.1
345 YB88/4.27/1.1–1.2
346 S91 Wilesmith para. 48
347 YB88/4.26/2.1
He went on to explain that investigations were in hand aimed at collecting data from compounders on sources of ingredients. In the circumstances he could only produce a progress report for Whitehall. 349

Mr Wilesmith told us:

In the course of May 1988 I received information which had been requested from feed suppliers on the composition of rations. This information indicated that all affected animals had consumed feed containing MBM. I reported this to Mr Rees, the CVO. This accumulated evidence of MBM as the vehicle of infection was considered sufficient by Mr Rees to submit an appropriate Ministerial submission on 6 May, 1988. 350

On 3 May 1988 Mr Wilesmith produced the report. This summarised the epidemiological investigations that had been completed and were in progress and suggested possible actions for control of BSE. Mr Wilesmith advised:

The epidemiological picture is typical of an extended common source epidemic in which the incidence increases sharply over a short period of time and then maintains at a constant incidence. 351

Mr Wilesmith set out the findings reported by Mr Peter Smith (see paragraphs 3.44 – 3.47). He explained that enquiries of feed compounders were in hand in an attempt to identify the source of MBM and tallow used in the feedstuffs of some 40 affected herds. He added that it was the present opinion of experts on the scrapie agent that MBM was more likely to be the vehicle of the agent than tallow. 353

By way of possible action to control BSE, Mr Wilesmith recommended a temporary ban on the inclusion of MBM in cattle and sheep feedstuffs, and added:

It would be helpful in investigating this disease if this ban were made public; epidemiological studies (case control study) could proceed more rapidly. 354

This comment reflected the fact that so far discussions with UKRA, GAFTA and UKASTA had been on a confidential basis, and that their members (other than
those involved in the discussions) had not been informed of the fact that animal feed was the prime BSE suspect.

3.62 Mr Wilesmith’s report, added to other information which had been assembled, gave Mr Rees a solid basis for putting forward proposals for action. On 6 May he sent a submission to the Minister which had been agreed with Mr Cruickshank. This submission stated:

The Chief Veterinary Officer is satisfied from the information produced by the investigating teams that the source of the transmissible agent which has caused BSE is through meat and bone meal derived from sheep material in which the rendering process has failed to inactivate the scrapie agent. Affected sheep material is continuing to be processed and it must be assumed therefore that cattle continue to be exposed to infection.355

Discussion

3.63 This completes our summary of the epidemiological investigations up to the time that the decision was taken to recommend a ruminant feed ban. Eighteen months had elapsed since the initial identification of BSE and 11 months since Mr Wilesmith’s investigations began. Over 11 further years have now passed. While there is a general consensus among the scientists that MBM was the vector responsible for the spread of the BSE epidemic, it is remarkable that controversy still exists about the origin of the BSE agent.

3.64 We consider that the origin of the BSE agent remains uncertain. With hindsight, the most likely possibility is that it is a novel TSE agent arising in either sheep or cattle. This is discussed further in vol. 2: Science. We have, however, in that volume felt it right to explain our reasons for concluding that Mr Wilesmith was correct in identifying animal feed, and more particularly the MBM component of that feed, as the vector for the epidemic of BSE. At this point we pause to consider, as part of our duty to comment on the adequacy of the response to BSE, whether Mr Wilesmith’s investigation in the period up to the beginning of May 1988 was carried out with due speed and diligence.

Was there delay in identifying feed as the cause of infection?

3.65 Should Mr Wilesmith have been involved earlier? He told us that an epidemiologist likes to be involved at the outset of the emergence of a new disease, and to have an input into any circulars sent out seeking information about its incidence.356 When the decision was taken to ask Mr Wilesmith to assist in investigation, six cases had already been confirmed by histopathology, involving four different farms. A further 13 cases were suspected on these farms on the basis of clinical criteria. Reports of a similar clinical syndrome occurring on other farms had been received by some VICs.357
3.66 We do not consider that Mr Wilesmith’s colleagues at the CVL can be criticised for not seeking his assistance at an earlier stage. The resources of the Epidemiology Department were limited and could not be directed to every suspected new animal disease. It was not until the stage was reached at which a number of suspect cases had been identified that the skills of the epidemiologist had a significant contribution to make. Where we have been critical is in respect of the delay in reaching that stage, which resulted from the restrictions on dissemination of information about BSE in the early days (see Chapter 2).

3.67 The next matter that we have considered is whether the identification of MBM as a front-runner in causing BSE could not and should not have been made more swiftly by seeking the assistance of the rendering and animal feed trade associations and their members at the outset. We have drawn attention to the emphasis that was placed on the need for informal approaches and the preservation of confidentiality about the theory that feed was the cause of BSE. We note Mr Bradley’s comment ‘if we approach too early concealment of information is likely’ (see paragraph 3.16 above). In so far as MAFF’s reticence was attributable to a fear that an open approach might result in a refusal to provide the information sought, we have some sympathy with this. Indeed, we find that the extent of cooperation provided by UKRA and UKASTA and the leading members of those associations, while commendable, was surprising. The theory MAFF was seeking to explore was that feed compounders and renderers had been marketing products that were so unfit for their purpose that they were infecting the cattle to which they were fed. The minutes of the meeting between MAFF, GAFTA, UKASTA and UKRA on 8 April 1988 end:

UKASTA offered help in tracing the source of feed supplied to affected herds.

It was emphasised that this exercise was not intended to find a ‘culprit’ but to pinpoint any fault in the system and jointly seek a remedy. There would be no public statement until the proposed sampling operation was complete and results assessed.

UKASTA asked what the legal position would be if suspect plants were identified. They understood that there could be no product liability because of the lack of scientific evidence at the time the contamination occurred. However, they would welcome clarification on this point and a statement which would discourage spurious claims from farmers.358

3.68 It seems that no response was made to the request for clarification of the legal position. MAFF was not in a position to provide reassurance. The reality was that, if the link between BSE and animal protein had been established, those who could be shown to have supplied an infective product would have been susceptible to claims for damages by those to whom the product had been sold. We do not believe that those who insured compounders and renderers would have wished their clients to provide information that might have had this result. Much later in the story the CVL’s Epidemiology Department sought to investigate the processes involved in the compounding of commercial feedstuffs in order to identify possible risk factors that allowed the inclusion of ruminant-derived protein in cattle feed. The feed compounders took legal advice and, on the basis of that advice, denied access to this

358 YB88/4.9/5.3
information. A similar reaction might well have been experienced in relation to Mr Wilesmith’s enquiries in 1988, and MAFF had no statutory powers to require information. MAFF officials are not to be criticised for treading gingerly in conducting their investigation.

3.69 Mr Meldrum has, however, informed us that, as far as he was concerned, any fear that an open approach might result in a refusal to provide information sought ‘did not come into the decision to make informal approaches to the rendering and feed industries’. His explanation was: ‘When I was asked about these “off the record” meetings I commented that it was the general agreement of the meeting on 15 December 1987 that they should be “off the record”’, because:

We were still considering the hypothetical, indeed the possibility that meat and bone meal could be the cause of BSE; and whilst those investigations were being taken forward it seemed wise to keep them confidential. . . . When you are dealing with a very early investigation or some very early reports, it was better to keep the investigation to yourself and release the information in a proper and reasoned and planned way when it becomes substantive.

3.70 When asked what the result would have been if, in December 1987, the State Veterinary Service had gone on record in its dealings with the feed industry, disclosed that MBM was suspected as the cause of BSE, and asked for assistance, he replied:

I think in one word confusion, because in December [1987] we were still at a very early stage in the investigations. If we had made that information public in a disorderly way I think it would have caused significant confusion both to the renderers, feed industry and to farmers, not least to the public at large. It would not have been very helpful.

3.71 We have not found the logic of these comments easy to follow and feel that they may be indicative of a general approach to the management of information.

3.72 While, with hindsight, we are inclined to think that a direct approach to UKASTA in November or December might have saved some time, we are not disposed to criticise the information-gathering technique adopted by Mr Meldrum and his colleagues. It resulted in Mr Wilesmith’s obtaining the information that he was seeking and making a correct diagnosis as to how it was that cattle were contracting BSE.

3.73 On 9 and 10 September 1991 a Visiting Group consisting of two distinguished epidemiologists, Professor Roger Morris and Professor R A Robinson, conducted a review of the Epidemiology Department of the CVL. In their report, they described the Department’s investigation into the cause of BSE, of which we have so far only considered the first phase, as ‘a careful and valid epidemiological approach to identifying the vehicle of transmission of BSE, ie meat and bone meal contaminated with a scrapie-like agent’. They gave high commendation to the work of Mr Wilesmith and his team:

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359 S184H Meldrum para. 3
360 S184H Meldrum para. 3; T69 pp. 40–8
361 T69 p. 50
362 M36 tab 4 p. 9
We believe the staff of the Epidemiology Department should be recognised as having moved quickly and effectively to define the nature and course of the epidemic, and to identify the mechanism of transmission in the absence of almost all the diagnostic aids which would normally be called on in such an investigation. This problem ranks among the major disease puzzles of the century, and has been solved by application of epidemiological techniques far faster and more definitively than virtually any other equivalent problem in human or veterinary medicine. It will prove to be a landmark example which will be used for many years to demonstrate how epidemiological methods can be applied to unknown diseases.

It is important that Department staff take pride in their achievement in this area, because enormous progress was achieved with minimal resources as the epidemic unfolded.363

3.74 When giving evidence to us, Mr Wilesmith described his achievement in more modest terms:

It really was by exclusion of all those other things quite simply that we were left with – [it] had to be the meat and bone meal . . . It was not anything more clever than that, I just excluded all those other things.364

3.75 Mr Wilesmith accurately describes the process that led him to conclude that MBM was the source of infection with BSE. We would, however, echo the commendation of the Visiting Group. It took Mr Wilesmith about six months to identify the vector of the BSE epidemic, by a process of elimination, and gain sufficient confidence in the identification to recommend appropriate remedial action. In May 1988 his conclusions were still tentative to a degree. His belief was, and it was a logical belief, that the common source of the outbreak of BSE must be some novel element affecting the nature or the quality of some of the MBM that was being incorporated into cattle feed. What that element was remained unclear and, until it was identified, further epidemiological research was needed. In the meantime the achievement of the modest Epidemiology Department of the CVL in identifying the probable source of infection in the space of six months constituted an impressive response to the challenge of BSE.

Why was the feed infectious?

3.76 Mr Wilesmith had concluded that the cases of BSE that were breaking out all over the country could not have been caused by a single point source of infection. He thought that each case was an index case and that there was a common source of infection. The probable source was scrapie-contaminated MBM. Scrapie-infected sheep must, however, have been a constituent of the MBM which went into cattle feed for decades. Why had it only started to transmit to cattle in about 1982? And why was this transmission only occurring in Great Britain when scrapie-infected sheep were undoubtedly being rendered down and incorporated in cattle feed in other parts of the world?

3.77 Mr Wilesmith suspected that the answer might lie in the rendering process. In July 1988, with the help of Mr Martin Atkinson, who was then Deputy Regional
Veterinary Officer for Midland and Western Region, he developed a questionnaire for a proposed survey of all rendering plants. This sought information from the rendering plant about methods and, in particular, about the raw materials and time/temperature of the cooking process on a year-by-year basis, between 1980 and 1987. As an afterthought, questions about the use of solvent extraction were added (see vol. 13: *Industry Processes and Controls* for a description of solvent extraction).

3.78 The survey was carried out by three Veterinary Officers, Mr David Mouat, Mr Andrew Proud and Mr Paddy Grant, who gave oral evidence to the Inquiry. Their voluminous Survey Reports are in evidence.

3.79 In a paper published in the *Veterinary Record* on 17 December 1988 Mr Wilesmith advanced the following comments on the cause of BSE:

> There was no clear or single explanation why, in 1982, cattle apparently became first exposed to a transmissible agent sufficiently to result in clinical disease. A number of factors have been identified which when combined are undoubtedly significant in the occurrence of this epidemiological phenomenon. These include: a dramatic increase in the sheep population in Great Britain which commenced in 1980 and has continued (MAFF 1988); a probable increase in the prevalence of scrapie infected flocks (J. W. Wilesmith unpublished data); the greater inclusion of sheep heads in material for rendering; the greater inclusion of casualty and condemned sheep in material for rendering as a result of the reduction in the number of knacker’s yards; the introduction of continuous rendering processes during the 1970s and 1980s which may have resulted in the rendering of animal material at a lower temperature and, or, a shorter time than previously and the decline in the practice of using hydrocarbon solvents and terminal heat treatment for fat extraction since the mid 1970s (MMC 1985).

> These factors provide a possible explanation for a change in the exposure of cattle to sheep-derived protein and the scrapie agent.

3.80 When, however, he came to analyse the results of the rendering survey in January 1989, he concluded that the change from batch rendering to continuous rendering processes could not have been a relevant factor. The change had not taken place during the relevant period and the potential for heat penetration and thus an inactivating effect on the scrapie-like agent was probably greater under continuous rendering than under the batch processing systems. The cessation of the use of solvent extraction seemed, however, a more likely explanation for the development of infectivity in MBM in around 1981–82.

3.81 In a paper published in the *Veterinary Record* on 2 March 1991 Mr Wilesmith included a graph which, he suggested, showed:

> The cessation of hydrocarbon solvent extraction of fat from meat and bone meal is consistent with the temporal estimate of the onset of exposure.
3.82 He went on to explain that the rendering survey indicated that the rendering process had ‘seldom if ever’ produced sufficient heat to deactivate the most heat-resistant strain of scrapie. He continued:

This suggests that cattle may have always been potentially exposed to a scrapie-like agent via meat and bone meal and the occurrence of BSE cannot therefore be explained simply by a change from total inactivation of a scrapie-like agent in meat and bone meal to partial inactivation, but the extent of the partial inactivation may well have been reduced by the move away from solvent extraction. The use of hydrocarbon solvent extraction was an additional, and in the main, terminal process whose removal has not resulted in a change in the basic heat treatment of material which is the application of dry heat, albeit in a fat rich environment, rather than moist heat. The solvent extraction process did, however, include the application of steam to the low fat content meat and bone meal. This component of the solvent extraction process therefore represented not only an additional application of heat, but a more effective heat treatment with respect to the inactivation of the scrapie agent. This process may not have totally eliminated a scrapie-like agent, but could have reduced the titre sufficiently to prevent clinical disease occurring during the normal lifetime of cattle, as the incubation period of the scrapie-like infections is related to the dose of agent received . . .

The cessation of this hydrocarbon solvent extraction process may have increased the contamination of meat and bone meal with a scrapie-like agent in two ways. First, the loss of any additive effects on inactivation by the combination of heat plus solvents and, secondly, the loss of an additional moist heating stage. This represents a biologically plausible hypothesis to explain how cattle became exposed to a scrapie-like agent sufficient to result in clinical disease. The magnitude of the stepwise reduction in the proportion of meat and bone meal produced by solvent extraction represents a major and sudden change in the potential exposure of cattle to a scrapie-like agent and is consistent with the abrupt change in the effective exposure as indicated by the epidemic curve and the predicted time of onset of this exposure in 1981/82.369

3.83 By way of conclusion Mr Wilesmith suggested the following hypothesis:

As suggested previously, the epidemiological data are not consistent with a hypothesis involving the emergence in the sheep population of a novel strain of scrapie which is pathogenic for cattle. Epidemiological results obtained so far are consistent with the hypothesis that the occurrence of BSE in cattle has been the result of an increase in exposure to the scrapie agent, via ruminant derived protein in feedstuffs. This implies that cattle have always been susceptible to the scrapie agent but their exposure has, in the past, been insufficient to result in a detectable incidence of clinical disease. The findings are also consistent with an increase in exposure of cattle, as a result of the change in the rendering process, to a cattle ‘adapted’ strain of a scrapie-like agent which had been present in the cattle population for some time. Again in this case the infecting dose would previously have been

insufficient to result in clinical disease occurring during the normal lifetime of cattle at a detectable incidence.  

3.84 This theory has survived almost to the present day, as one possible explanation for the emergence of BSE. When we first learned of this theory, it seemed to us unlikely that there had been a change, or a combination of changes, in rendering that were peculiar to Great Britain and yet so nearly universal within Great Britain that they permitted an almost simultaneous transmission of scrapie to cattle throughout the country, or, alternatively, the emergence of a widespread disease that had hitherto lain latent in cattle. The theory was, it seemed to us, only viable as the least unlikely explanation for the emergence of an extended common source epidemic. Thus we were anxious to explore whether epidemiological modelling made it possible to postulate that there may have been a single point source for the BSE epidemic. Having investigated this in Phase 2 of the Inquiry hearings, we have concluded that it is indeed a possibility. We have consequently reached the following conclusions as a matter of probability, though not of certainty.

3.85 The source of BSE is not a long-established scrapie agent which would have given rise to an extended common source epidemic. BSE is a novel and unique TSE which probably started as a consequence of a genetic mutation in a single animal and which has been propagated as a consequence of recycling by rendering and incorporation in cattle feed. This would constitute a point source for the epidemic.

3.86 The reasons for these conclusions are developed in detail in vol. 2: Science. Shortly summarised, they are:

i. BSE is a single strain, which differs from any known strain of scrapie.

ii. Scrapie has many strains. It is hard to conceive of near simultaneous transmission to cattle all over the country resulting in a single strain of BSE.

iii. Increases in the British sheep population or in the incidence of scrapie do not impress as a plausible factor. The sheep/scrapie content of individual batches of rendering must always have varied widely. The comments made by Mr Peter Carrigan (Director of a major tripe manufacturer) were cogent.

iv. Use of solvent extraction was far from universal prior to 1980.

v. Tests have shown that solvent extraction has no significant effect on deactivation of the scrapie agent.

3.87 We do not consider that Mr Wilesmith is to be criticised for concluding that the emergence of BSE was attributable to an extended common source or that that source was scrapie. The sophisticated techniques that have demonstrated that BSE could have had a single point source went beyond the expertise of the CVL’s modest Epidemiology Department. Furthermore, we question whether any epidemiology department, in the early days of BSE up to the end of 1989, would have concluded that it might have had a single point source.

3.88 In his statement to the Inquiry Mr Wilesmith referred to a review of the epidemiology – in which he was assisted by Professor Robert Curnow, an applied statistician, and the strain typing results of Dr Moira Bruce – which established that

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370 Veterinary Record, vol.128, 2 March 1991, p. 203
371 T58 pp. 13–14
all BSE cases that were tested had a single strain, including one case that had most likely been infected in the early 1980s. This had led Mr Wilesmith to alter his views on the likely source of infection for cattle during the early years of the clinical epidemic. He explained:

Thus, while I still subscribe to the sheep scrapie hypothesis as the explanation for the origin of BSE (the origin is, of course, the subject of continuing research), it appears that the BSE strain was selected quite rapidly by cattle and that the clinical epidemic of BSE is essentially due to the recycling of infected cattle tissues via MBM.372

3.89 The erroneous conclusion that the cases identified up to 1988 were index cases (ie, first cases in a specified group), probably infected from scrapie as a common source, affected estimates that were made of the future incidence of the disease. Mr Wilesmith felt unable to estimate the effect of the recycling of BSE itself and contemplated the possibility that this might not be significant. Had it been recognised that the cases being identified were the product of recycling BSE, which could well have been in progress for over a decade, the probability of a rapid escalation of clinical cases would surely have been appreciated. As it is, at the time that MAFF introduced a ruminant feed ban, it was believed, on the basis of Mr Wilesmith’s advice, that cases of cattle infected before the ban might well continue at a constant rate (approximately 60 clinical cases a month). In fact, the reality was that the rate at which cattle were being infected was destined to produce clinical cases at a rate of over 3,000 a month.