3. The early years, 1986–88

This is the first of a number of chapters which tell, in summary form, the story detailed in Volumes 3 to 9 and 11.

Identification of a new disease in cattle

The epidemic of BSE may have started with a single diseased cow. Why should that cow have developed BSE? It is possible that the disease developed spontaneously as a consequence of a genetic mutation. It is possible (though we believe less likely) that a mutant strain of the scrapie agent transmitted to one or more cows. There are other possibilities. No one will ever know.

When was the first case? The epidemiologists, with their skills in back calculation, suggest in the 1970s. Where was it? Again no one can say, though epidemiologists would point to the early concentration of cases in the West Country as suggesting that BSE may well have come from there.

Did the first case get ill on the farm and end up in the knacker’s yard, or was it sent to be slaughtered for human food – perhaps before the signs of the disease were even showing? We cannot know. What we can deduce is that, by one route or another, the animal’s head, together with other unwanted offal, was sent to the renderers. The parts carrying the BSE infection contaminated the batch of meat and bone meal (MBM) produced from the rendering. That MBM was sold to a food compounder and mixed into cattle feed, contaminating that feed. That feed may have infected many cows and some of these, by a similar series of events, infected many more. Thus, like a chain letter, the spread of the disease was almost exponential.

The disease spread wide, and it spread at first unnoticed. It spread wide because MBM may travel long distances from renderers to the feedmill and the cattle feed produced by the mill may be widely distributed. The calves which eat the feed may end their lives far from the farms on which they were born.

It spread at first unnoticed because most infected cattle were slaughtered before showing clinical signs of the disease. When clinical signs did appear, they were similar to those of some other diseases of cattle. Only histopathology of the brain could reveal the existence of the new disease. Before that could happen the carcass had to be sent by a vet to one of the regional State Veterinary Investigation Centres (VICs), and from there the brain had to be sent to the Pathology Department of MAFF’s Central Veterinary Laboratory (CVL) at Weybridge. Most cattle infected with BSE went for slaughter before the clinical signs developed (‘subclinical cases’). Where a single cow fell ill, the farmer was unlikely to want a post-mortem examination and, for some reason, not yet clear, BSE tended to strike down single cows in a herd.

The first brain from a cow with what we now know as BSE reached the CVL in September 1985. It came from a herd in Pitsham Farm in Sussex where unusually
a number of cattle had been struck down with symptoms that we now recognise as
typical of BSE. The CVL pathologist identified the condition of the brain as
spongiform encephalopathy, but concluded that this, and a kidney condition from
which the animal had also suffered, was probably caused by toxicity of some
description.\textsuperscript{10}

172 At the end of 1986 pathologists at the CVL identified the possibility that cattle
had developed a spongiform encephalopathy that was transmissible in the same way
as scrapie was in sheep. This followed the submission of brain samples from a herd
in Kent and another from near Bristol. Mr Raymond Bradley, head of the Pathology
Department, remarked in a note to colleagues:

\begin{quote}
If the disease turned out to be bovine scrapie it would have severe
repercussions to the export trade and possibly also for humans.
\end{quote}

173 One witness described meeting Mr William Rees, the Chief Veterinary Officer
(CVO), who had just heard the news, with ‘steam coming out of his ears’.\textsuperscript{11}

174 The CVL pathologists identified the emergence of a new disease, which they
considered might be a bovine form of scrapie, as soon as could reasonably have
been expected. They are to be congratulated – particularly Mr Gerald Wells and
Dr Martin Jeffrey, who carried out the initial histopathology.

Restraints on information

175 CVL staff thought that they might have identified a bovine form of scrapie,
but they were not sure. The experts in this field were the members of the
Neuropathogenesis Unit (NPU) in Edinburgh. If the CVL had consulted them at this
stage, the NPU would have confirmed that there were very strong indications that
this was indeed a new Transmissible Spongiform Encephalopathy (TSE). In the
event the CVL did not seek the collaboration of the NPU until June 1987, and Mr
Wells did not get confirmation from the NPU of his diagnosis until the end of July.
Having regard to the importance of this matter, we think that Dr William Watson,
the Director of the CVL, should have sought the assistance of the NPU from the
outset.

176 It was important that MAFF should discover not merely the nature of the
problem, but also its scale. If private vets and members of the VI (Veterinary
Investigation) Service around the country were told of what the CVL had found and
asked to look out for cattle with similar signs, reporting of cases, which might
otherwise go unremarked, would be encouraged. Unfortunately, in the first half of
1987 there was a policy that one Senior Veterinary Investigation Officer described
as ‘a total suppression of all information on the subject’. This was encouraged by
an understandable anxiety on the part of Mr Wells that MAFF should not go public
until the CVL was sufficiently sure of its ground to advance a scientifically
responsible claim to have discovered a new disease. In March 1987 a proposed
publication about BSE in Vision, a VI Service newsletter, did not proceed. The

\textsuperscript{10} Vol. 3, paras 1.7–1.33
\textsuperscript{11} Vol. 3, paras 1.34–1.40
decision was Dr Watson’s, who should not have permitted Mr Wells’s concern to prevail over the desirability of effective surveillance.

177 Events after March 1987 demonstrated a policy of restricting dissemination of information about BSE. The principal reason for this was concern about ‘the possible effect on exports and the political implications’ should news get out that a possible TSE in cattle had been discovered in Britain. Publication to the VI Service of information about BSE eventually took place in June. This was not in Vision, which was circulated to Veterinary Investigation Officers (VIOs) not only in England and Wales, but also in Scotland. Instead a circular letter was sent to Senior VIOs in England and Wales, describing the clinical signs and the pathology and calling for notification of similar cases to a Senior Veterinary Officer at the State Veterinary Service headquarters at Tolworth, Surrey. It directed that VI staff should not consult Research Institutes or University Departments, or publish anything about BSE or discuss it at meetings without clearance. A proposed letter by a VIO to the Veterinary Record describing the clinical signs and the pathology of BSE was refused permission for submission to the journal.

178 Primary responsibility for this policy lay with Mr Rees, the CVO, but it received support from his subordinates, Dr Watson and Dr Bernard Williams, the head of the VI Service. We can see why there were concerns that reports of a possible TSE in cattle might harm the industry and, in particular, the export market. But this did not justify suppression of information needed if disease surveillance was to operate effectively. Dr Watson and Dr Williams should have urged the merits of publication and Mr Rees should have permitted it.

179 An article by Mr Wells for the Veterinary Record, which compared the pathology of BSE and scrapie, was embargoed and it was made plain that comparisons with scrapie were not acceptable. This line was taken at the instigation of Mr Rees. He should have permitted publication of the article and he should have permitted comparisons with scrapie.

180 Had there been a policy of openness rather than secrecy, this would have resulted in a higher rate of referral of cases to MAFF in the earlier part of 1987. This, in turn, might have led to a better appreciation of the growing scale of the problem and hence to remedial measures being taken sooner than they were.

181 In the second half of 1987, restraints on publication of information about BSE were progressively relaxed. Articles about BSE were submitted to the Veterinary Record and the disease was the subject of discussion at a number of agricultural trade meetings. In October articles about the disease appeared in the farming and national press. The number of cases reported increased rapidly. At the end of May there had been 6 identified cases and 13 suspected cases. By the beginning of September there were 66 suspect cases, of which 8 were histopathologically confirmed. By the end of October the figures were 120 and 29, and by the end of the year 370 suspects, of which 132 were confirmed.
What was the cause of BSE?

182 The CVL had only one qualified epidemiologist in 1987, Mr John Wilesmith, who headed a small Epidemiology Department. He knew nothing of BSE until late in May, when he was asked by Dr Watson to investigate its epidemiology. There were then 6 confirmed cases on 4 farms, but as we have seen the numbers were about to escalate.

183 Mr Wilesmith prepared a questionnaire, rolled up his sleeves and set off in person to visit farms on which BSE suspects had been reported. Soon Mr M Cranwell had to be seconded from Starcross VIC in Exeter to assist him. By this time, unknown to Mr Wilesmith, thousands of cattle had been infected by recycling of earlier cases and were incubating the disease. Mr Wilesmith assumed, quite naturally, that each new case was an index case (that is, arising as a fresh incident) and that there was some common factor causing all of them. The search was on for that common factor. Vaccines, hormones and organophosphates were considered but ruled out: the disease had been found in cattle exposed to none of these.

184 From the outset feed was a runner. In August Mr Wilesmith noted that lamb MBM was used in commercial dairy rations, but added that it was not a recent introduction. This was a major conundrum. If feed was the cause, what novel ingredient or feature had suddenly started to make the feed infective?

185 Mr Wilesmith carried out calculations which indicated that the exposure of the cattle population to the BSE agent was likely to have begun in the winter of 1981–82. Had anything occurred at about this time to explain the disease?

186 Further investigations were put in hand to explore, with the help of the feed and rendering industries, why it might be that cattle feed had suddenly started infecting cattle. By the end of April 1988 Mr Wilesmith had reached no conclusion on this. He had, however, concluded that feed was the source of infection and that the source of infection in the feed was MBM made from sheep affected by scrapie. He set out these conclusions in a report, recommending a temporary ban on the inclusion of MBM in cattle and sheep feedstuffs, while further enquiries were made.

187 Mr Wilesmith and his colleagues are to be congratulated on the rapid identification of cattle feed as the cause of the cases of BSE that were being reported, and on the advice of a ban on feeding MBM to cattle and sheep. As we shall see, this advice was promptly implemented and cut off most of the source of infection, turning an escalating disease into one that would peak and decline.

188 Mr Wilesmith had, however, made some tentative conclusions which were to prove erroneous. He concluded that the cases being reported were all index cases. He concluded that the common source of infection was scrapie-infected feed which would result in the incidence of BSE rising sharply over a short period of time before maintaining a constant incidence. In a paper published at the end of 1988 he identified a number of factors which might explain why cattle feed had become infective around 1981–82. These included an increase in the amount of scrapie-infected sheep going for rendering and changes in the rendering process which had reduced the temperature applied. In the following year he refined these ideas and
decided that particular significance attached to one specific change in the rendering process. The use of solvent to extract tallow had been widely abandoned at just about the right time to explain the outbreak of the disease. This process might well have played an essential role in inactivating the scrapie agent. When Mr Wilesmith learned of this change he commented that it was ‘too good to be true’. In that, he was correct.

Mr Wilesmith’s tentative conclusions were reasonable on the data available to him at the time, but they were wide of the mark, as he was in due course to acknowledge. The cause of infection of the cases being reported was not the scrapie agent in the feed, but the BSE agent itself. The cases were not first generation cases, but the consequence of recycling of BSE. Far from the incidence of BSE infection being likely to prove constant, it had been escalating year on year and was, in 1988, infecting cattle at a rate that probably exceeded 10,000 cases a month.

Changes in rendering processes may have had some effect on inactivation of the BSE agent, but they were not decisive or even significant.

Mr Wilesmith’s tentative conclusions were widely accepted. They led to misconceptions, some of which have survived to the present day. We will deal with them shortly. They receive detailed consideration in Volumes 2 and 3.

The scrapie theory

The conclusion that BSE had been transmitted from scrapie-infected sheep was generally accepted. It was a reassuring conclusion. Sheep affected by scrapie had been eaten by humans for 200 years or more, without apparent ill effect. It was likely that scrapie in cattle would prove similarly innocuous. Although, as the years passed, evidence mounted that discredited the scrapie theory, this was never made clear to the public and most people are still under the impression that cattle caught BSE from scrapie-infected feed.

The conclusion that rendering changes had permitted the BSE agent to survive unscathed, whereas previously it had been inactivated, is also still widely accepted. There are two variations on this theme:

i. Some accuse the Government of having recklessly relaxed the Regulations governing rendering, or of having failed to impose a sufficiently rigorous regulatory regime.

ii. Some accuse the rendering industry of having put the safety of their product at risk by cutting corners in order to cut costs.

Neither of these accusations is valid. There was no relaxation by the Government of rendering standards. Up to 1981 the rendering industry was largely unregulated. In 1981 Regulations were introduced that set minimum standards for the product of renderers, to be checked by regular sampling. The Regulations were strengthened in 1989. A more complex alternative involving the licensing of rendering plants was not pursued, but this would not have addressed the problem of BSE and the proposed criteria for the grant of licences would not have prevented it. That problem was not foreseen, nor was it reasonably foreseeable.

12 See Volumes 13 and 14
By the same token the changes made by the rendering industry to their processes did not, overall, make them more vulnerable to BSE. Neither the old nor the new processes would have inactivated the BSE agent. No rendering process has yet been devised which can guarantee to do so, though infectivity is reduced.

The theory that the rate of infection would have reached a plateau led to the conclusion in 1989 that the scale of the problem could be related to the rate at which cases were being reported. The Southwood Working Party on Bovine Spongiform Encephalopathy reported that year on the basis that the effect of recycling could be ‘minimal and undetectable’, in which case 350 to 400 cases a month could be expected. In early 1993 cases were being reported at a rate of around 1,000 a week.\(^\text{13}\)

These misconceptions involve no criticism of Mr Wilesmith. They demonstrate that in 1987 and 1988 lack of data made it impossible to appreciate the nature and extent of the disaster that had already occurred.

The ruminant feed ban

While Mr Wilesmith was exploring why cattle were succumbing to BSE, consideration was also being given to the implications that the disease might have for humans. Before turning to that part of the story, let us follow the reaction to Mr Wilesmith’s advice that the practice of including animal protein in cattle feed should be subjected to a temporary ban.

If Mr Wilesmith’s conclusions were tentative, Mr Rees, the CVO, thought that the picture was clear. In a submission to Mr John MacGregor, the Minister of Agriculture, Fisheries and Food, he advised that he was:

\[
\ldots \text{satisfied from the information produced by the investigating teams that} \\
\text{the source of the transmissible agent which has caused BSE is through meat} \\
\text{and bone meal derived from sheep material in which the rendering process} \\
\text{has failed to inactivate the scrapie agent. Affected sheep material is} \\
\text{continuing to be processed and it must be assumed therefore that cattle} \\
\text{continue to be exposed to infection.}^{14}
\]

He advised that the feed industry should be asked to agree a voluntary withdrawal of MBM from ruminant feed, but that if they refused, a mandatory ban should be imposed.

Mr MacGregor was even more decisive. On 19 May 1988 he determined that there should be a ‘speedy and compulsory ban on sheep meat material in feed for ruminants’. It fell to Mr Alan Lawrence, a Grade 7 official in MAFF’s Animal Health Division, to implement this decision in consultation with departmental lawyers and with the benefit of advice from his administrative and veterinary colleagues. It was decided that the ban should extend to the feeding of ruminant protein to ruminants. In effect the ban was subsequently operated as if it encompassed all animal protein, for no renderers attempted to segregate their raw materials in order to produce non-ruminant MBM. The ban was achieved by an

\(^{13}\) See vol. 4: The Southwood Working Party, 1988–89  
\(^{14}\) YB88/5.6/11.3
Order\textsuperscript{15} signed by Mr MacGregor and Welsh and Scottish Office Ministers on 10–14 June. This made it an offence to sell, supply or use for feeding to ruminating animals any feedstuff in which the offender ‘knew or had reason to suspect’ that any animal protein had been incorporated. The ban was initially only up to the end of 1988, but it was subsequently to be extended, and finally made permanent.

202 This simple Order has been described by one distinguished epidemiologist as:

\begin{quote}
A spectacularly successful control measure . . . one of the notable success stories of global disease control.
\end{quote}

203 It has, today, come close to eradicating an epidemic that, at its height, was of gigantic proportions. Primary credit for this goes to Mr Wilesmith and his Department for their diagnosis of the source of infection, but credit also is due to Mr Rees and Mr MacGregor for their prompt and decisive response. Unhappily, though, the measure was not a total success. There were shortcomings in its implementation. We turn to consider why this was.

204 The question arose in the course of consultation as to when the ban should come into effect. After consulting its members, the UK Agricultural Supply Trade Association (UKASTA) asked for a three-month period of grace to enable the industry to clear from the distribution channels all stocks of ruminant feed that had already been compounded. After taking advice from the veterinarians in MAFF, Mr Lawrence proposed a two-month period of grace. MAFF’s press office advised that a delay as long as this would lead to accusations of risking the further spread of the disease simply to make life easy for the industry. Mr MacGregor, on the advice of Mr Alistair Cruickshank,\textsuperscript{16} compromised and decided that the ban should come into effect on 18 July – five weeks from the date of the Order.

205 We initially questioned the grant of this period of grace, but concluded that our reservations were the result of being wise after the event. Mr Kevin Taylor, one of the MAFF veterinarians involved in the preparation of the ruminant feed ban, explained to us his reasons for viewing a period of grace of as long as two months as perfectly acceptable from a veterinary point of view. On the basis of the information then available it did not seem to him that such a delay was going to make very much difference. The industry had been exposed to infected feed for 380 weeks. A few weeks more would not make a great deal of difference.

206 In June 1988 MAFF officials reasonably expected, on the basis of Mr Wilesmith’s advice, that the rate of infection was likely to have stabilised at about 60 cases a month. Mr Taylor considered that if no period of grace had been granted, farmers and the industry would initially have disregarded the ban. We found force in these points and reached the conclusion that the compromise period of grace decided upon by Mr MacGregor could not be criticised. Had it been appreciated that cattle were being infected at the rate of thousands of cases a week, we have no doubt that a very different approach would have been adopted.

207 Much later it became apparent that infected feed had continued to be fed to cattle on a substantial scale after 18 July. Nearly 12,000 cattle born after the ban (BABs) in 1988 and over 12,000 born in 1989 subsequently developed clinical signs.

\textsuperscript{15} The Bovine Spongiform Encephalopathy Order 1988
\textsuperscript{16} MAFF Under Secretary (Grade 3) responsible for the Animal Health Group
of BSE. A far larger number must have been infected, but slaughtered before signs became apparent. Some of these cases will have resulted from accidental contamination of feed. Some will have resulted from farmers, who had little or no means of knowing whether their feed contained ruminant protein, continuing to use the feed they had in stock. But we are satisfied that some feedmills and feed merchants deliberately continued to sell cattle feed containing animal protein after the ban come into effect.

208 Had the only source of contaminated feed been existing stocks of cattle feed made up before the ban came into effect, the BABs would have come to an end once this had been consumed. In the event, over 5,600 cattle born in 1990, 4,500 born in 1991, 3,000 born in 1992, 2,200 born in 1993 and 1,000 born in 1994 were to go down with the disease. With hindsight, it is clear that most of these infections resulted from cross-contamination of cattle feed with pig and poultry feed, containing infective MBM, in the feedmills. The risk, indeed the certainty, of a degree of cross-contamination when the same production lines are used to produce different batches of feed is, and was in 1988, well established. One reason that has enabled us to conclude that cross-contamination did indeed result in infection of cattle is knowledge that we now have as to the quantity of infectious material that suffices to transmit BSE orally in cattle.

209 An experiment carried out by the NPU has demonstrated that ½ gram of homogenised brain from BSE-infected cattle is sufficient to transmit the disease orally across the species barrier to sheep and goats. Another experiment carried out by the CVL has demonstrated that 1 gram of such material can transmit the disease orally to cattle.\(^\text{17}\)

210 The results of these experiments were not available when the ruminant feed ban was introduced. What consideration was given at that stage to the amount of material that might infect? What consideration was given to the question of whether cross-contamination might pose a risk of infection? UKASTA witnesses spoke of receiving repeated reassurances from MAFF right up to 1994 that a large amount of contaminated feed would be necessary to infect a cow.

211 We found no specific evidence of when or by whom such assurances were given. A number of MAFF administrators spoke of their understanding that a large amount of infective material was needed to infect. Some of the professionals – Dr Watson, Mr Kevin Taylor, Mr Bradley – told us that they had no idea what the minimum quantity would be. There was general surprise, when the result of the attack rate experiment was made known, that as little as 1 gram had sufficed to infect. Although there is no record of Mr Keith Meldrum\(^\text{18}\) reassuring UKASTA that there was no need to worry about cross-contamination, he is recorded as telling representatives of the cattle industry in June 1988 that feedmills presented at worst a low contamination risk and would not be investigated. He advised at the same meeting that MBM could safely be used as fertiliser because the dose that might be received by grazing cattle would almost certainly be too low to cause disease.

212 Was there any valid basis upon which Mr Meldrum could have concluded in 1988 that cross-contamination in the feedmill would not involve sufficient quantities of infective material to give rise to transmission? We have concluded that

\(^{17}\) See vol. 2: Science
\(^{18}\) Mr Meldrum succeeded Mr Rees as Chief Veterinary Officer in June 1988
there was not. Mr Wilesmith told us that he had concluded that a very small amount of infective material would suffice to infect. This he deduced from the small inclusion rate of MBM in calf rations. He believed that his view should have been widely shared by administrators at MAFF. Those who designed the experiments at the NPU and CVL, to which we have referred above, envisaged the possibility that $\frac{1}{2}$ or 1 gram would suffice to infect. Had the question of the amount of material needed to infect been explored at the time of the imposition of the ruminant feed ban with those best placed to advise, the conclusion should have been reached that this amount might be very small.

213 Mr Meldrum told us that if he or any other MAFF or industry representative had known at the time that the infective dose was so low as to lead to cross-contamination problems, the issue would have been pursued. As it was, the existence of a danger from cross-contamination was not considered to exist at the time.

214 We have concluded that at the time that the ruminant feed ban was imposed, there was a lack of rigorous thought about its implementation. One person who should have given more thought to this was Mr Meldrum. He had knowledge of how feedmills operated, and of the problem of cross-contamination between batches. He assumed this would not matter but did not have adequate grounds for that assumption. A failure to attach significance to the possibility of infection through cross-contamination in feed was understandable when the apparent rate of infection was only about 60 cases a month. However, in the course of September 1988, 435 cases of BSE were reported in Great Britain. Once this was apparent, Mr Meldrum should have ensured that proper consideration was given to this matter. This should have led to guidance being given to both the feedmills and to those farmers who mixed their own feed, on the need to take precautions to minimise cross-contamination.19

215 Mr Meldrum is a man of great energy and industry. He had only just taken up the reins of the CVO. His national and international duties were onerous. These are considerations which should temper any criticism of his oversight on this occasion.

216 Failure to appreciate that cross-contamination mattered carried with it a failure to appreciate the importance of a test that would detect cross-contamination. When the ruminant feed ban was introduced, there was no test which would detect animal protein in compound feed, let alone ruminant protein. Without such a test the Order was unenforceable. Steps were put in hand to develop, in-house, the ELISA technique so as to produce a test that would identify ruminant protein in feed. This was not treated as a matter of priority. Deliberate breach of the ban was not considered likely and accidental cross-contamination was not considered to be cause for concern. Development of the ELISA test followed a leisurely course and did not approach achievement until the end of the period with which our Inquiry is concerned.20

Exports

217 The United Kingdom exported very little compound feed, but did export significant quantities of MBM. This was exported initially to Europe to

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18 For detailed discussion see vol. 3: The Early Years, 1986–88, paras 4.117–4.171
manufacturers of concentrates who re-exported their products to the Middle East or North Africa. Some have suggested that the United Kingdom should have imposed a ban on the export of MBM when the ruminant feed ban was introduced to try to make sure that foreign countries did not infect their cattle with BSE. This would have been difficult. Renderers were still permitted to sell MBM to British purchasers for incorporation in pig and poultry feed. Most MBM that was exported was used for the same purpose. An attempt to prohibit exports would have been likely to be challenged in the Courts. It could be argued convincingly that foreign importers could be adequately protected by warnings that MBM should not be fed to cattle.

218 Were adequate warnings given? Mr John Gummer urged before the ruminant feed ban was introduced, when he was the junior Agriculture Minister, that we had a moral duty to warn our neighbours of the danger of feeding MBM to cattle. Under European law this country was obliged to give notice of the ruminant feed ban to all EU members and did so. What of the countries that were not members of the EU? Mr Meldrum told us that he relied on the customary means of communicating with them on the subject of animal diseases. He notified the Office International des Epizooties, which passed the information on to all members in a report of its annual General Session in May 1989. In February 1990 Mr Gummer, by now the Minister of Agriculture, Fisheries and Food, insisted that Mr Meldrum take the further step of writing a letter of warning to Chief Veterinary Officers of all countries which imported MBM from the UK. There is scope for arguing that Mr Meldrum should have done this earlier. We think the argument is academic. The only country outside the EU where it is suspected that cattle were infected with BSE as a result of importing MBM is Switzerland, and it seems that the MBM in question reached Switzerland via Belgium. If this occurred after the ruminant feed ban, both Belgium and Switzerland were aware that ruminant protein was suspected to be the cause of BSE. Accordingly we have seen no need to pursue this issue further.

Human health implications

219 BSE had implications for human health in many different ways. The one of which the public was most aware was the possibility that BSE posed a risk through food. Responsibility for addressing this risk was shared by MAFF and the Department of Health (DH). Mr Meldrum emphasised to us that DH was responsible for assessing risk to human health. He told us that he did his best to avoid making public comments on this matter. He saw MAFF’s role as being risk management, together with the provision of advice to DH on matters that fell within the expertise of the veterinarians.

220 We have not found it easy to draw a distinction between risk evaluation and risk management. Throughout the BSE story, MAFF officials and Ministers appear to us to have proceeded on the footing that it was their responsibility to see that whatever left the slaughterhouse to go into the human food chain was safe to eat. MAFF made the running in considering both what was and what was not safe to go into the food chain, and how what was not safe should be kept out of it. Problems arising over the safety of animal feed, which were unquestionably MAFF’s responsibility, tended to mirror problems of the safety of human food. In relation to the latter, DH was consulted, but not often actively involved in the initial
formulation of policy. Whether DH should have been more involved is a matter that we shall consider.

221 BSE also posed a potential risk to human health as a result of the use of bovine products or by-products in the making of pharmaceuticals and cosmetics. So far as the former were concerned, DH had responsibility for human medicines and MAFF for veterinary medicines. Responsibility for the safety of cosmetics fell to the Department of Trade and Industry (DTI). These areas, and the occupational risks posed by BSE to those who handled cattle, or their products, we consider in separate chapters of this volume.

222 MAFF Ministers were first informed about BSE after the General Election in June 1987. Mr MacGregor was appointed Minister of Agriculture, Fisheries and Food, and Mr Gummer his Minister of State. Mr Donald Thompson retained his post as MAFF Parliamentary Secretary. In a note to him about the disease, Mr Rees commented, ‘There is no evidence that the bovine disease is transmissible to humans,’ a statement that was to be frequently repeated. Mr Thompson met officials on 22 July. The Permanent Secretary, Sir Michael Franklin, observed that the establishment of any risk to human health was the highest priority, and Mr Thompson said that he was particularly concerned about this. In a paper for him, which was subsequently seen by the Minister, Dr Watson advised that there was no reason at all to believe that any risk to human health existed.

223 By the end of July, 46 probable cases of BSE had been identified involving 18 herds. Both Mr Thompson and Sir Michael Franklin had raised concerns about human health. Mr Rees did not share those concerns. He viewed BSE as an animal health not a human health problem. Dr Watson thought it very unlikely that BSE posed a risk to human health.

224 In mid-September Mr Rees prepared a progress report for Ministers. This included a statement that DHSS was aware of the problem. Dr Watson had told Mr Rees that Dr Thomas Little, the Deputy Director of the CVL with responsibility for veterinary medicines, had discussed BSE with DH colleagues at a meeting of a subcommittee of the Committee on Safety of Medicines. Regrettably Mr Rees did not explain to Ministers the limited nature of the communication that had occurred. There had been an informal discussion in the margins of that meeting, but news of BSE had gone no further within DH.

225 By this time there were 73 suspected cases in 36 herds across 11 counties. In a Q&A briefing for the media in October, Mr John Suich, who headed the Animal Health Division, included the following:

Q : Can it be transmitted to humans?

A : There is no evidence that it is transmissible to humans.

226 On 11 November 1987 he repeated this comment in a briefing for Mr Thompson, adding the suggestion that reassurance could be drawn from an analogy with scrapie.

21 The Department of Health and Social Security (DHSS) split into two separate Departments – DH and DSS – during 1988.
On 4 December Lord Montagu of Beaulieu wrote to Mr MacGregor expressing concern at the fact that cattle with BSE were being slaughtered for human consumption. He suggested that:

Perhaps this is an area where the Ministry should make the disease notifiable and pay compensation at the full value for animals infected.

It seems that this letter served as a catalyst for formal consideration by MAFF officials of whether action should be taken to address the possibility that BSE might be transmissible to humans, though other letters from the public were received to similar effect. Mr Rees chaired a meeting of MAFF officials on 15 December. It was agreed that a paper should be prepared for Ministers setting out the options. On 29 December an article in *The Times*, headed ‘Mystery Disease Strikes at Cattle’, observed that there was no indication of whether the disease was transmissible to humans. By the end of the year, 370 suspect cases had been reported and 132 had been confirmed.

The options to be submitted to Ministers were discussed by, among others, Mr Rees, Mr Cruickshank, Dr Watson, Mr Meldrum, Mr Wilesmith and Mr Lawrence. The submission was perfected by 16 February 1988 and forwarded by Mr Cruickshank to Mr Edward Smith, the Deputy Secretary at MAFF. In his covering minute, Mr Cruickshank remarked:

We do not know where this disease came from, we do not know how it is spread and we do not know whether it can be passed to humans. The last point seems to me the most worrying aspect of the problem. There is no evidence that people can be infected but we cannot say there is no risk.

This was an acute analysis of the position so far as humans were concerned. Mr Cruickshank’s analysis of this aspect of BSE was not to be bettered, or even significantly augmented, by the scientists who were to consider the problem in the months to come.

The submission itself observed that it was uncertain whether the disease was transmissible to humans, and continued:

We could therefore be criticised for allowing affected animals to be sold for human consumption. MAFF are already being asked to advise on whether there is any risk to humans.

The option recommended was a policy of slaughter of affected animals with payment of compensation, the principal advantage of which was to enable the Government to answer criticism about human health implications. The submission took some pains to emphasise that payment of compensation was appropriate as the measure would be taken mainly for public health reasons, not in order to eradicate the disease.

Mr Smith forwarded the submission to the Permanent Secretary, now Mr Derek Andrews, adding that as the policy was in the interests of public health, it would not be appropriate to look to the industry to fund it.
It is remarkable that MAFF officials had prepared this submission, whose recommendation was based essentially on an evaluation of risk to human health, without involving anyone at DH. The expressions of concern in the summer of 1987 by Sir Michael Franklin and Mr Thompson, coupled with the growth of the epidemic, called for joint consideration by MAFF and DH, with assistance from experts in TSEs, as to whether BSE might be transmissible to humans. Had this course been followed, we have little doubt that a joint submission would have been made to both MAFF and DH Ministers to the same effect as that which went forward to Mr MacGregor, but backed by conclusions as to the uncertainty about risk to humans that would have carried more weight than those of MAFF officials alone. It might moreover, as we shall see, have brought together those licensing veterinary and human medicines to consider their shared problems.

We sought explanations for the failure to involve DH from Dr Watson, Mr Cruickshank and Mr Rees. We have summarised their explanations in Volume 3. We find that the true reasons were (i) a belief on the part of some that BSE was an animal and not a human health problem and (ii) a degree of interdepartmental reserve which led Dr Watson, Mr Rees and Mr Cruickshank to conclude that BSE was their problem to be resolved without the need for outside assistance – or interference – from DH. In this, each of them was at fault. The consequence, as we shall show, was a lengthy delay in reaching a decision as to the precautionary action to be taken.

**Mr MacGregor’s reaction**

Mr MacGregor’s previous office had been Chief Secretary to the Treasury. We believe that MAFF officials anticipated that he would have reservations about a policy that involved paying compensation out of public funds to farmers for the slaughter of sick animals. In this they were correct. Mr MacGregor’s initial reaction to the submission was to be ‘very cautious’. He expressed concern that if compensation were paid for slaughtering cattle with BSE, there would be a ‘read across’ to situations where the destruction of diseased crops had been ordered without payment of compensation. Rhizomania, a disease of sugar beet, was an example.

Mr Cruickshank told us that he and his colleagues considered Mr MacGregor’s reaction to the submission to be a peremptory rejection. Sir Derek Andrews demurred at this description, and so would we. Mr MacGregor’s initial reaction to a policy that involved payment of compensation was unfavourable, but he nonetheless agreed that the advice of the Chief Medical Officer (CMO) should be sought. His reaction affected, however, the manner in which the CMO, Sir Donald Acheson, was approached. The intention had been to tell him that MAFF wished to introduce a slaughter and compensation policy and to ask him to advise whether or not BSE posed a risk to humans. Had that approach been adopted, we think it likely that Sir Donald would have endorsed MAFF’s proposed policy. As it was things took a different turn.

It was unfortunate that Mr MacGregor did not share his officials’ view of the merits of the slaughter and compensation policy. It would not, however, be fair to criticise him for his reservations, for they did not lead him to reject the policy. His
decision to consult the CMO before reaching a final decision fell well within the range of responses that were reasonably open to him.

**Sir Donald Acheson’s advice**

238 Mr Andrews wrote to Sir Donald Acheson on 3 March 1988. He described the nature of BSE. This was the first that Sir Donald had heard of the disease. Mr Andrews then raised the question of whether BSE might be transmissible to humans. He wrote:

> It would be very helpful therefore to have your advice on the view we should take of the possible human health implications and how we should handle questions about the risks to human health.

239 This put the ball of recommending what action should be taken into Sir Donald’s court, and with no warning at all. Sir Donald’s reaction was to call an interdepartmental meeting to consider the matter.

240 Those present at this meeting were not able to form a firm view as to whether or not BSE posed a risk to human health. It was agreed to recommend to Health Ministers that a small group of experts be set up to advise on the human health risks and possible preventive measures. Sir Donald commented that he thought it highly likely that the advice would be that carcasses of affected animals should not go for human consumption.

241 We found this decision disappointing. MAFF officials had formed the view that unless one could be confident that they posed no risk to humans, sick animals should not be permitted to be slaughtered for food. The Southwood Working Party, set up on Sir Donald’s recommendation, was to take the same view immediately it met. This was, we feel, no more than common sense. Referring the matter to an expert Working Party was bound to result in significant delay. A better and more robust response would have been to recommend that the practice of eating diseased cattle should cease at once. We have concluded, however, that it would not be fair to criticise Sir Donald for the course that he took. He was put in an invidious position, being asked for advice without notice on policy that had significant consequences. Those whom he summoned to help him decide on what to do expressed uncertainty. In these circumstances, we find that the decision to recommend that the matter be referred to an expert group fell within the range of reasonable responses open to Sir Donald.

242 Delay did indeed result, however. Over three months were to elapse before the Southwood Working Party was constituted and met for the first time. During this period MAFF came under increasing pressure to take action. On 22 April 1988 a front page article in *Farming News* accused MAFF of seriously underestimating the extent of BSE and referred to disquiet about whether the disease posed a danger to humans. By then there had been 421 cases confirmed in 352 herds.

243 Mr MacGregor continued to set his face against any suggestion that the Government should fund a compulsory slaughter and compensation scheme. He accepted a recommendation that BSE should be made a notifiable disease – a measure designed to give MAFF a better picture of the incidence of the disease and
the power, if necessary, to impose movement controls on animals. BSE was made notifiable in June 1988 by the same Order that introduced the ruminant feed ban. The rate of reporting leapt almost overnight from 60 cases a month to 60 cases a week. The Order required that the heads of all these cases be surrendered to MAFF; the brains were then removed and examined by the CVL. So far as the proposal for compulsory slaughter was concerned, discussions were carried on with the farming industry to explore the possibility of an industry-funded scheme. Industry was told that there was no question of government funding being provided. Industry’s response was that it was for the Government to fund compensation if compulsory slaughter were to be introduced.

244 On 4 June 1988 an article in the British Medical Journal, co-authored by a doctor and a dietician, pointed out that if BSE were transmissible to humans it might be years before infected individuals succumbed. The authors wrongly assumed that animals showing signs of sickness would not enter the food chain, but went on to say that it was ‘naive, uninformed and potentially disastrous’ to assume that animals incubating the disease but not yet showing signs posed no risk to humans.

245 On 20 June the Southwood Working Party met for the first time. They were horrified to learn that animals sick with BSE were being slaughtered for food. The next day Sir Richard Southwood wrote to Mr Andrews recommending that carcasses of BSE-affected animals should be condemned and destroyed. Mr MacGregor’s officials advised him that compulsory slaughter should be introduced and that the Government would have to pay compensation under the Animal Health Act 1981 – they recommended that this should be fixed at 50 per cent of the value of a sound animal. Mr MacGregor wrote to Mr John Major at the Treasury urging, though with reluctance, that payment of compensation at this level be approved.

246 At the same time, Sir Donald Acheson informed Mr David Mellor, the Health Minister, that destruction of the carcasses of clinically affected animals was essential on the grounds of risk to humans. It was on this basis that the consent of the Treasury was given to the payment of compensation. Mr MacGregor had suggested that the cost of this measure would be around £250,000 a year on the basis that cases would continue to be reported at a rate of about 60 a month. He cannot yet have been aware of the increase of the reporting rate consequent upon the notification requirement.

247 The Order providing for compulsory slaughter and destruction of cattle suffering from BSE came into force on 8 August. Nearly six months had gone by since MAFF officials had first recommended this course.