1. The identification of a new disease in cattle

Introduction

1.1 BSE posed novel and serious problems in relation to both animal and human health. As with other new and serious animal diseases, it was important that its emergence should be identified as soon as possible. In this chapter we consider the system that existed for the identification of new animal diseases and the manner in which it led to the identification of BSE. We examine whether there was any inadequacy of response on the part of those involved in operating the surveillance system.

Veterinary Investigation Centres and the Central Veterinary Laboratory

1.2 In 1985 the State Veterinary Service for Great Britain (SVS) comprised the Veterinary Investigation Service (VI Service), the Veterinary Field Service (VFS) and the Central Veterinary Laboratory (CVL). Initially, a newly identified animal disease was the responsibility of the VI Service, though once a disease had been made notifiable its management became the responsibility of the VFS.1

1.3 Within the VI Service were 22 Veterinary Investigation Centres (VICs), spread throughout England and Wales. In Scotland, the Scottish Agricultural Colleges undertook the role of the VI Service. Northern Ireland had its own veterinary service and support facilities.2 The role of the VI Service was to carry out disease surveillance and to provide a consultancy facility for veterinary surgeons in private practice. Members of the VI Service reported to an Assistant Chief Veterinary Officer, who was directly responsible to the Chief Veterinary Officer (CVO) at MAFF in Tolworth (Surrey), rather than the Director of the CVL.

1.4 The CVL’s role was to act as a central facility for the whole of the UK. It provided diagnosis and consulting on animal diseases, specialist and technical expertise for practising farm vets,3 and a source of information for policy makers (though it had no primary responsibility for policy-related decisions itself, or for operational control of disease).4

1.5 The VI Service and the CVL provided a two-stage consultancy service to private vets. VICs would send selected specimens to the CVL for specialised diagnosis within one of its departments: Bacteriology, Biochemistry, Parasitology and Pathology.5 At this time, Mr Ray Bradley, who from June 1987 also coordinated

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1 S93 Cawthorne Annex 1 paras 4–5; T36 pp. 89–92
2 For further information see vol. 9: Wales, Scotland and Northern Ireland
3 S65 Wells para. 9
4 S70 Watson para. 9
5 S70 Watson para. 7
BSE research, headed the Pathology Department. Within that department the Consultant Pathology Unit (CPU), headed by Mr Gerald Wells, provided a consultancy service for the VI Service. It was staffed by six CVL pathologists, from various sections of the Pathology Department, on a rota basis.  

The passive surveillance system

1.6 The Ministry of Agriculture, Fisheries and Food (MAFF) relied for the identification of new diseases in animals on what has been described as ‘the passive surveillance system’. A farmer, troubled by an animal showing symptoms of disease, would call upon the services of a private veterinary surgeon. The surgeon, if unable to resolve the problem, would seek assistance from the local VIC. The VIC would, where appropriate, seek assistance from the CVL. Both the staff of the VI Service, which comprised the VICS, and CVL staff were part of the State Veterinary Service, which in turn was part of MAFF. Important information was passed quickly from the VI Service and CVL up the management chain to the Chief Veterinary Officer and his group at their headquarters in Tolworth. In this way, any new condition was likely, in due course, to come to the attention of the State Veterinary Service (SVS) through the CVL. The structure of MAFF and its constituent parts, including the SVS and the CVL, at this time is illustrated in Annex 1 in vol. 15: Government and Public Administration.

The earliest confirmed case

1.7 The earliest case of BSE to be confirmed by the CPU was a cow owned by Mr Peter Stent of Pitsham Farm in Sussex (Cow 142). Brain samples from Cow 142 were first examined in September 1985. It was not, however, until June 1987 that Mr Gerald Wells, on review of the archives of the Pathology Department, confirmed this as a case of BSE.

1.8 The pathologist who carried out the original examination of the brain of Cow 142 from Pitsham Farm was Ms Carol Richardson. She told the Inquiry that she identified pathological changes in the brain as ‘scrapie in a cow’ and that colleagues at the CPU confirmed her diagnosis. This evidence conflicts with that given by other members of the Pathology Department, who said that BSE was first identified as a likely transmissible spongiform encephalopathy in cattle at the end of 1986. Ms Richardson’s evidence has led to allegations in the media that MAFF became aware of the emergence of BSE in September 1985 and concealed this fact for over a year. The resolution of this issue calls for a detailed analysis of the evidence.

1.9 On 22 December 1984 Mr David Bee, a local private veterinarian, was called to examine Cow 133, owned by Mr Peter Stent of Pitsham Farm in Sussex. She had an arched back and had lost weight. Mr Bee visited the farm on several further occasions over the following months, and continued to see animals showing unusual

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6 S65 Wells para. 7; see Annex 1 in vol.15: Government and Public Administration
7 S331D Little para. 16
8 YB84/12.22/1.1; S6 Bee para. 10
symptoms.\textsuperscript{9} Cow 133 developed a head tremor and a lack of coordination before dying on 11 February 1985.\textsuperscript{10} Mr Bee sought assistance from Mr J M Watkin-Jones, a veterinarian at the Winchester VIC.

1.10 By the end of April 1985 five more cows had died on the farm. Mr Bee and Mr Watkin-Jones coined the phrase ‘Stent Farm Syndrome’ to describe these cases.\textsuperscript{11} A number of samples of body tissue were submitted to the CVL for pathological analysis. Various possible ailments were identified, but despite a wide range of tests no definite diagnosis proved possible. It was suggested that Mr Stent submit a live affected cow for slaughter and post-mortem.\textsuperscript{12}

1.11 Later in the summer of 1985, Mr Stent had two live cases of this apparent ‘syndrome’. The first of these cows was sent for slaughter on 1 August, with the head and other tissues being sent on to the local VIC. Unfortunately, the brain of this animal had no diagnostic value as it had been slaughtered by being shot in the head.\textsuperscript{13} The second animal, Cow 142, was sent live to the CVL on 2 September for euthanasia and a post-mortem examination.\textsuperscript{14}

1.12 Carol Richardson, the pathologist on duty at the CVL, received the samples of brain, spinal cord and kidney of Cow 142 on 10 September, and examined them on 13 September.\textsuperscript{15}

1.13 Ms Richardson recorded her observations on a Case Card (Form DL99). The case history recorded that Cow 142 was said to be typical of seven cows on the Stent farm over the previous five months, which were described as ‘nervous’.\textsuperscript{16} She transcribed her comments from the Case Card onto a Pathology Report dated 19 September 1985, addressed to Mr Watkin-Jones. They were as follows:

PATHOLOGY REPORT

Gross observations: Received in formalin a well preserved bovine brain, 2 pieces of spinal cord and pieces of kidney all grossly normal.

Microscopic observations:

\textit{Cerebrum} – mild multifocal (4 foci) non-suppurative perivascular infiltration and focal gliosis.

Thalamus – NVL

Cerebellum – NVL

\textit{Corpora quadrigemina} – mild neuropil vacuolation

\textit{Medulla} – moderate neuronal and neuropil vacuolation of the reticular formation
Spinal cord – mild neuropil vacuolation of the lumbar dorsal horns.

Kidney – chronic mild/moderate non-suppurative interstitial reaction with tubular regeneration and fibrosis.

Also a mild peracute multifocal tubular necrosis with focal hyaline droplet change.

DIAGNOSIS: 1. Moderate spongiform encephalopathy – acute

2. Mild renal nephrosis – peracute

REMARKS: These acute changes suggest a toxicity of some description. The non-suppurative reactions are far more chronic, mild and non-specific.\(^\text{17}\)

1.14 Ms Richardson’s Pathology Report was sent to Mr Watkin-Jones at the Winchester VIC on 19 September 1985, who forwarded the report to Mr Bee, Mr Stent’s vet, with this comment:

I enclose a histological report carried out by my colleague Carol Richardson. I have discussed her findings with her at some length and she comments that the pathological changes found would be consistent with bacterial toxin.\(^\text{18}\)

1.15 As to this comment, Mr Bee remarked in his statement to us:

I recall my disbelief at this statement at the time. I believed the problem had been associated with fungal contamination of feed and mycotoxin production. On 4/10/85 we received a laboratory report stating that a fungal toxin called citrinin had been found in the feed. In any case, by this time, new cases had ceased to develop. I imagined that the problem had run its course.\(^\text{19}\)

1.16 When giving evidence, Ms Richardson was categoric that she did not discuss Cow 142 with Mr Watkin-Jones.\(^\text{20}\) Nor would she have ascribed the pathology that she saw to bacterial toxins. She would have expected bacterial toxins to result in neuronal necrosis.\(^\text{21}\) In her written statement to the Inquiry Ms Richardson said:

Although I had never seen this type of lesion before in a cow I had frequently seen the combination of neuronal and neuropil vacuolation with this distribution in Scrapie. To me, this was Scrapie in a cow.\(^\text{22}\)

1.17 She went on to explain her diagnosis:

From the history of the case on the Stent farm, it seemed as if the clinical course of the disease was fairly rapid in that metabolic disorders of short duration and heavy metal toxicities were being considered on the farm. Therefore, it seemed likely that the cause(s) of the spongiform changes were a result of an acute clinical disease (rather than a chronic illness) and in the
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absence of a more likely aetiology, toxicity seemed to be the most appropriate category that fitted both symptoms and findings.

I dismissed the possibility that a bacterial toxaemia had caused the spongiform change; in my limited experience of ruminant neuropathology, toxaemia was likely to produce frank neuronal necrosis rather than degenerative vacuolation (cf. Clostridial toxaemia).23

1.18 Ms Richardson added that she sought a second opinion from a ruminant neuropathologist colleague, Dr Martin Jeffrey, before writing her report on 19 September, leaving the brain sections, her findings, and a request for a further examination, on Dr Jeffrey’s bench. ‘I was eager to hear his opinion and immediately after lunch went to collect the slides. Martin had left a note on which was written “Bovine scrapie”.’ She met Dr Jeffrey as she left the room. Dr Jeffrey told her this was the first case that he had seen, but that Mr Wells, Head of the CPU, had examined two cases, and was expecting another two cases.24

1.19 When giving oral evidence to the Inquiry, Dr Jeffrey said that he had no recollection of the discussion of the samples that Ms Richardson left for him. It was not uncommon for a pathologist to leave samples for a colleague to look at, but he did not recall this discussion or the leaving of a note stating ‘Bovine scrapie’. Further, he did not remember mentioning that Mr Wells had two earlier cases of a similar condition. He added that there had been searches of CVL archives, and that these cases did not exist.25 He thought that had these two cases been mentioned to him, with the prospect of two more, then it would have stuck in his memory: ‘One is a novelty, two is a coincidence, three is a problem.’26

1.20 In his written statement to the Inquiry, Mr Wells told us that he was attending a meeting in Cheshire at the time Ms Richardson examined the samples. He said that she had left the samples and a copy of her Pathology Report for him to examine on his return. It was common practice for an examining pathologist to leave sections for a colleague to express a view on.27

1.21 Mr Wells said that on his review of Ms Richardson’s Report:

I agreed with her overall observations and that such observations were not artefactual i.e. caused as a result of post-mortem changes or in the preparation of sections. My conclusion was that the brain lesions observed in this case could not in my experience be attributed to a specific disease, but a speculative comment was made that they could possibly be the result of chronic bacteraemia or an endotoxaemia (the production of poisons in the blood due to infection).28

1.22 Mr Wells made a manuscript note on the foot of Ms Richardson’s Report:

... lesions probable sequel to chronic bacteraemia or endotoxaemia.29

23 S69 Richardson paras. 17–18
24 S69 Richardson para. 13
25 T25 pp. 42–3
26 T25 p. 44
27 S65 Wells para. 15
28 S65 Wells para. 16
29 YB85/9.18/2.1
1.23 He was, however, uncertain whether he made that note on the occasion of his first review of her Report. This was because he reviewed her Report on two further occasions. The second review was on 26 September 1985 when, at Mr Watkin-Jones’s request, he reviewed the four casualties on the Stent farm whose samples had been submitted to the CVL. On that occasion he made a note: ‘No conclusion drawn regarding common aetiology.’ The third review was in June 1987, as part of the review of brain sections in the CVL archives, when he concluded that Cow 142 had been suffering from BSE.

1.24 The manuscript note referred to above was but one of a number of annotations made by Mr Wells to the Report, and he could not recollect precisely when each was made.

1.25 Mr Wells had no recollection of any reference being made to bovine scrapie. He commented that had Ms Richardson felt strongly that the observations she had made were of scrapie in cattle, he would have expected her to come back to him to discuss the matter subsequently, or to take the matter further herself. For her part Ms Richardson said that she had not sought a second opinion from Mr Wells. She had sought a second opinion from Dr Jeffrey. She had simply left the sections for Mr Wells to look at as she thought he might be interested, having already had two similar cases himself.

Discussion

1.26 Conflicts of evidence are bound to occur when witnesses are asked about events that took place some 15 years ago. Contemporary documents provide a valuable aid to solving such conflicts. Having carefully considered all the evidence, we have concluded that Ms Richardson is mistaken in her recollection that, in September 1985, she and her colleagues at the CVL identified the disease affecting Cow 142 as a scrapie-like disease in cattle. While we think that she may well have noticed a pathological similarity to scrapie, we do not believe that she can have concluded that Cow 142 was suffering from a transmissible spongiform encephalopathy.

1.27 The following are our reasons for reaching this conclusion:

i. The suggestion in Ms Richardson’s Pathology Report that the spongiform encephalopathy was an acute condition attributable to ‘a toxicity of some description’ is incompatible with a conclusion that Cow 142 was suffering from a transmissible spongiform encephalopathy (TSE).

ii. It is plain that Ms Richardson had a discussion about Cow 142 with Mr Watkin-Jones in which she commented that the pathological changes were consistent with bacterial toxin. Her memory must be at fault when she says: (a) that the conversation never took place and (b) that she would not have ascribed the

30 T26 p. 61
31 S65 Wells para. 17
32 YB85/9.26/1.1
33 T26 p. 71
34 T26 p. 58
35 T26 p. 74
36 S65 Wells para. 15
37 T28 p. 24
condition to a bacterial toxin. The suggestion that a bacterial toxin was the cause is incompatible with a conclusion that Cow 142 was suffering from a TSE.

iii. The suggestion made by Ms Richardson that a bacterial toxin might be the cause echoes the manuscript note added to the foot of her Report by Mr Wells. We believe that he must have raised the possibility of bacteraemia on his first review and that Ms Richardson accepted this as a possible cause.

iv. We are satisfied that Dr Jeffrey cannot, in September 1985, have identified Cow 142 as ‘bovine scrapie’ and said that Mr Wells had had two such cases and was expecting two more. Had this been the case both he and Mr Wells would surely have remembered this, and records of the cases in question would have been preserved. Neither Dr Jeffrey nor Mr Wells has any such recollection and no such records exist.

1.28 We believe that in the years that have passed, Ms Richardson’s memory may have attributed to September 1985 events that took place at the end of 1986, when early cases of BSE were identified at the CVL.

1.29 We do not consider that Mr Wells, or anyone else who studied the brain sections of Cow 142 in September 1985, is to be criticised for failing to diagnose that the cow was suffering from a TSE. In his statement Mr Wells drew attention to the fact that samples from previous cases had not included brain sections and that the main post-mortem finding in relation to those cases had been that of internal bleeding.

1.30 There were no indications from the tissues provided that it might be desirable to investigate further into the nervous tissues of these, or possible future cases. As Mr Wells said in his statement to the Inquiry:

Taken in isolation and in the light of these factors, the case in September 1985 did not at that time suggest that a new disease had been identified. Vacuolar changes in the brain of that particular animal were not severe and there was previous, and current, evidence of other disease problems . . . It was not, therefore, immediately apparent from the post-mortem histopathological examination of the brain of one animal in this herd that it was the first and unprecedented case of a new disease.

1.31 There could have been a number of causes of the degeneration of the cerebral tissue in Cow 142. By the time that Mr Wells diagnosed the true cause in June 1987, he was much more familiar with the signs of BSE and was specifically searching for them.

1.32 The CVL received samples of nervous tissues and other organs from a further Stent cow on 10 September 1986. The histological examination by Dr S Done, dated 22 September 1986, revealed a mild spongiform change in the medulla (hindbrain).

1.33 At the time this case appears to have made no impact on Mr Wells, if he indeed was told about it. When he came to review it in June 1987 he found that there was

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38 S65 Wells para. 17
39 T26 p. 34
40 S65 Wells para. 17
41 YB86/9.22/1.1–1.2
insufficient information to make a conclusive retrospective diagnosis of BSE. We are left with a strong suspicion that all nine cows which appeared to manifest the ‘Stent Farm Syndrome’ may have been cases of BSE.

The identification of the emergence of BSE

1.34 In April 1985 a private veterinarian, Mr Colin Whitaker, was called to Plurenden Manor Farm in Kent, to look at a Friesian/Holstein cow that was showing symptoms including a change in behaviour, aggression and a lack of coordination. Over the following year Mr Whitaker continued to be called to the farm to treat cattle that had suffered changes in character and behaviour. In particular, the cattle became nervous, aggressive and progressively incoordinated until unable to rise without assistance. Mr Whitaker consulted Veterinary Officer Mr Carl Johnson of the Wye VIC in November 1986.

1.35 Mr Johnson referred the brains of three animals to the CVL in November and December 1986. He wrote a note to the Pathology Department at the CVL with some background information on the herd. This stated: ‘5 cows destroyed in last 2 years with similar symptoms.’ Mr Wells conducted histopathological examinations of these brains, and discovered that they had a common novel pathology – ‘multifocal spongy transformation of the brain parancyma and a degeneration of neurons, principally large neurons, in the brain stem.’ Mr Wells felt that these ‘features were consistent with the clinical neurological signs observed in the animals’. Compared with most other animal disorders the changes most closely related to scrapie, but there were subtle differences, and it was not possible to rule out other degenerative conditions, or entirely to discount metabolic or toxic causes.

1.36 The CVL also received brain samples from a cow that was referred by Langford VIC, Bristol, on 11 December 1986. Dr Jeffrey conducted the histological examination of these brain samples. His diagnosis stated: ‘Mild brainstem spongiform change with neuronal vacuolation.’

1.37 The initial communication from the Pathology Department to others at the CVL, and thence to MAFF more widely, of the possibility that a TSE had been identified in cattle is largely undocumented. On 19 December 1986, the departmental head, Mr Bradley, sent a minute to his colleagues at the CVL, Dr William Watson (the Director) and Dr Brian Shreeve (Director of Research), informing them of similar cases submitted from the Bristol and the Wye VICs. He commented:

The reasons for the interest are that the lesions observed have similarities to spongiform encephalopathies of other species and in particular scrapie of sheep.
The observations so far made can only lead to a morphological diagnosis. VIOs [Veterinary Investigation Officers] are I understand collecting significant epidemiological data re contact with sheep, whether the animals are genetically related and whether or not they are indigenous.

I would advise keeping an open mind about the aetiology until we have more information. The principal lesions are degenerative and non-specific.

If the disease turned out to be bovine scrapie it would have severe repercussions to the export trade and possibly also for humans if for example it was discovered that humans with spongiform encephalopathies had close association with the cattle. It is for these reasons I have classified this document confidential.

From the little data I have available I would not wish to speculate beyond stating that the sudden occurrence in several adult cows in one herd perhaps points more to a recent exposure to some toxic agent than to a slow infection. Nevertheless an open mind should be kept. At present I would recommend playing it low key because a simple explanation may be forthcoming as a result of current investigations which will allay fears.

You may also find the information valuable for defence of the CVL in a political sense.50

1.38 Although the memory of some witnesses put the relevant date a little earlier, we think that it must have been after this minute that news spread of the possible emergence of a new TSE in cattle. Dr Watson (Director of the CVL) informed Mr William Rees (the Chief Veterinary Officer) by telephone.51 Mr Kevin Taylor (Veterinary Head of MAFF’s Notifiable Diseases Section) then met Mr Rees ‘walking down the passage with steam coming out of his ears’. It seems that the scientists at CVL had demonstrated a degree of self-congratulation in having identified the new disease which struck no chord with Mr Rees, who could see all too well its wider implications.52

1.39 Six months later, when providing Dr Watson with material for a submission to Ministers, Mr Bradley included the observation:

At present BSE is regarded as a major discovery to the cattle industry that will when reported bring deserved prestige to MAFF, ADAS, the CVL and the workers involved.53

1.40 This was perhaps optimistic. Nonetheless the Pathology Department of the CVL deserves credit for identifying the new disease when it did and noting its similarity to scrapie. We will consider in the next chapter why it was that, six months later, Mr Bradley was still looking forward to the date when it would be ‘reported’.
When were the first cases of BSE?

1.41 There is anecdotal evidence to suggest that there may have been earlier, undiagnosed cases of BSE. Various farmers and veterinarians gave evidence of seeing cows in the early to mid-1980s with similar symptoms to those of BSE.54

1.42 One of the most well-documented cases occurred in Malmesbury, Wiltshire. A veterinarian, Mr Ray Williams, examined at least five cows between October 1983 and May 1985 on the farm of Mr Paul Lysley. He described the initial symptoms as being:

. . . seen a few weeks post calving with hyperexcitability, inco-ordinated gait, with progressive weight loss despite appetite being near normal, including parlour concentrates. Two cases last year became so inco-ordinated that they fell down on several occasions and were eventually slaughtered.55

1.43 In January 1985 Mr Williams saw a further cow from a different unit of the same farm (Cow 36) which showed similar symptoms. He sent Cow 36 to the University of Bristol Veterinary School at Langford. Cow 36 was slaughtered and the partial post-mortem report described the cow as having ‘progressive nervous signs, hyperaesthesia, tremors, mania and hind leg ataxia’.56 Mr Williams sent at least one further cow from Mr Lysley’s farm to the CVL in 1987. This animal was killed in December 1987 and the histological examination of the brain confirmed the presence of BSE.57

1.44 In his written statement, Mr Williams said: ‘In hindsight everyone involved now believes that the cases described above were all classic cases of BSE in adult dairy cows.’58

1.45 Mr Richard Sibley wrote to the Inquiry as Chair of the BSE Group of the British Cattle Veterinary Association (BCVA), which is a specialist division of the British Veterinary Association (BVA). The BCVA has 1,600 members, of whom 1,000 are practising veterinary surgeons working with cattle in farm animal veterinary practices. Mr Sibley’s written submission on behalf of the BCVA had this to say of the ability of the passive surveillance system to detect cases of BSE:

The referral of cases or samples from practitioners to VI Centres has established a rudimentary disease surveillance system, whereby data is collected by the centres and submitted to a central database. However, the data depends entirely on the submissions received from private practices. Not all practices submit samples or refer cases to their local VI Centre. Private laboratories provide a similar and competitive service. Many practices have their own in house facilities . . .

The supposition that practices would refer all cases to their local VI Centre, which would then collect data, is incorrect. Submission of cases and samples was based on a voluntary commercial decision of the attending veterinary surgeon. It may be that the original geographical recorded incidence of this
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disease was influenced by the vigilance and activity of veterinary surgeons in those areas and the relationship they had with their local VI Centre . . .

The surveillance of this disease up to June 1988 and indeed the surveillance of any non-notifiable disease is based upon the vigilance and inquisitiveness of the farmer and attending veterinary surgeon, with the voluntary referral to a VI Centre.59

1.46 Epidemiological evidence given to the Inquiry suggests that it is likely that the first cases of BSE pre-dated by some years – possibly as many as ten or more – the first case of BSE to be diagnosed as such.60 Earlier cases may well have been slaughtered before clinical symptoms developed, but we consider it possible that individual cases showing clinical symptoms were never referred to the SVS. In June 1987 Mr Wells carried out a review of selected brain sections held by the CVL and a small number of veterinary schools.61 The sections were chosen on the basis of the clinical history of the cows from which they had been taken. In this context it is right to observe that Mr Wells only discovered one positive case of BSE – Cow 142.

Discussion

1.47 While we have not found any shortcoming on the part of MAFF in identifying the emergence of BSE, we accept the evidence of Mr Sibley of the BCVA as to the natural limitations of the passive surveillance system. Since BSE emerged there have been radical structural changes within MAFF, including the reduction of the number of VICS and their incorporation into what is now called the Veterinary Laboratories Agency.62

1.48 Surveillance of animal diseases plainly needs to be reconsidered in the light of these structural changes. We understand that a Surveillance Group on Disease and Infections of Animals has been established in the course of 1999, chaired by the Chief Veterinary Officer, with membership from the Agricultural Departments of all four UK countries, the Department of Health, the Veterinary Medicines Directorate, the Joint Food Safety and Standards Group, and the Public Health Laboratory Service. Section 28 of the Food Standards Act 1999 provides for the new Food Standards Agency and the relevant Departments to share information on ‘food-borne zoonoses’ and for coordination of their activities on matters connected with such zoonoses. Diseased animals may pose a risk to humans in a variety of ways, of which food is only one. If surveillance is to be effective, it is vital that:

- any new disease in animals should be identified as soon as possible;
- once identified, that the potential implications for human health of the disease, having regard to all potential routes of transmission, are considered by scientists with appropriate qualifications; and
- where potential risk is identified, appropriate measures are taken to address that risk.63

59 S421 Sibley para. 3
60 T111 p. 56
61 S65 Wells para. 31
62 The structure of MAFF and its constituent parts, including the SVS and CVL, at this time is illustrated in Annex 1 in vol.15: Government and Public Administration
63 For further discussion on this, see Chapter 7 of vol. 2: Science