1. Introduction

1 In December 1986 a new animal disease was discovered by the State Veterinary Service. It quickly became known as Bovine Spongiform Encephalopathy or BSE. It caused irreversible ‘spongy’ changes to the brains of cattle and was invariably fatal. The public called it ‘mad cow disease’.

2 For ten years the Government told the people:
   - there is no evidence that BSE can be transmitted to humans;
   - it is most unlikely that BSE poses any risk to humans; and
   - it is safe to eat beef.

3 Then, on 20 March 1996, Mr Stephen Dorrell, the Secretary of State for Health, stood up in Parliament and announced that ten young people had contracted a new variant of the harrowing, and invariably fatal, Creutzfeldt-Jakob disease – vCJD – and that it was probable that they had caught BSE. Further cases of vCJD were to follow. By September 2000 there had been over 80\(^1\) cases and the frequency with which they were being reported seemed to be growing.

4 For nearly three years we have been examining all that is known about the history of BSE and vCJD and looking at how these diseases were handled by the Government and by others in the period between December 1986 and 20 March 1996. This Report sets out what we have found.

5 In 1986 the United Kingdom had a worldwide reputation for competence and efficiency in animal health and welfare matters, and in the handling of outbreaks of serious animal diseases. Its skilled veterinarians and scientists, with the State Veterinary Service and veterinary laboratories in the forefront, operated established processes to identify, contain and eradicate animal diseases. They worked closely with farmers, veterinarians in private practice, public health professionals and the relevant industrial sectors. They raised awareness, gave advice, and recommended statutory regulation where appropriate and compensation if need be. The process required well-established communication between advisers and practitioners, effective systems of animal surveillance and information-gathering, programmes of research, and detailed shared understanding of the links between animal and human health in all its aspects, including the food chain.

6 The UK also had highly regarded public health processes of long standing to handle outbreaks of human disease. These included surveillance, preventive action, such as immunisation and advice, and treatment. The health of the nation was at the heart of the remit of the Health Ministers and the professional responsibility of the four Chief Medical Officers, one for each part of the UK, who advised the Government.

7 What went wrong after the new fatal degenerative brain disease of cattle, BSE, emerged in 1986? Why did the announcement in 1996 that humans had probably

\(^1\) Including probable cases who were still alive
been struck down by this particular brain disease find the guardians of public health and the world at large so shocked, and apparently unprepared, and leave the public so disillusioned? Our remit does not extend to the frantic diplomatic activity and other events after that date, but the consequences are still bearing heavily on the British economy and have inflicted tragedy on some families and left blighting uncertainty and fear hanging over many more.

8 The full extent and effects of the human disease will not be discernible for many years to come. Baffling questions include the unusual nature of Transmissible Spongiform Encephalopathies (TSEs), the reasons why specific people have become prey to the human version of BSE, and the extent to which others, particularly those exposed to the agent in the 1980s, may yet develop it. These difficult and still unresolved questions have hampered and bedevilled the whole course of events. What we do know is that as of September 2000, shortly before publication of this Report, over 80 victims of vCJD, most of them young, had had their lives destroyed and their families’ happiness and hopes had been irreparably damaged.

9 BSE has been a peculiarly British disaster. Almost all the victims of vCJD have been in the United Kingdom. Only four other human victims of vCJD have been diagnosed elsewhere. 2 Over 170,000 cattle have been diagnosed with BSE here compared with fewer than 1,500 abroad, mostly it would appear traceable to British-sourced animals or infected feed at the beginning of the British epidemic. So far, over 4.7 million British cattle have had to be slaughtered, and their carcasses burned or buried as potentially dangerous waste. 3 A thriving high-quality cattle and meat export industry has been wiped out. The livelihood of thousands of farmers and businesses has been damaged. Even at this tail-end of the animal epidemic there were still over 2,000 cases of BSE notified in 1999 and cases continue to be reported as we write.

10 Small wonder that people want to know why it happened and whether it was handled wisely and well. In particular:

- What was the cause of BSE emerging and spreading country-wide? Was it as a result of intensive modern farming practices? Was it a result of inadequate regulation or lowered standards? Why is it so overwhelmingly the UK that has been afflicted?
- Seventy-four victims, mostly young people, have died of a new variant of CJD. Is it certain that they contracted this dreadful disease as a result of some form of connection with BSE? If so, why was it that they were struck down?
- Was the emergence of BSE and its threat to human health effectively handled by those whose responsibility it was to do so?
- Did individuals respond as they should have done, having regard to the state of knowledge at the time?
- Was the truth about the nature of BSE and the threat it posed concealed from the public? Has there been a cover-up?
- Did we make proper use of our scientists?

---

2 This represents two confirmed and one probable case in France and one confirmed case in the Republic of Ireland.

Source: CJD Surveillance Unit, 20 September 2000

3 Figures up to 30 June 2000. Source: MAFF
INTRODUCTION

- Did our health and welfare services adequately cater for the special needs of those who contracted vCJD and their families?
- What lessons does the catastrophic course of events hold for public policy and the way we do things in the future?

11 These questions have been very much in our minds throughout this Inquiry, as we have explored exactly what happened day by day during the ten years that led up to the announcement of 20 March 1996 that BSE had probably generated a new and fatal human disease. Some questions, such as the numbers who are likely to succumb to the human disease, we are not in a position to answer. Our remit is to report to Ministers on the course of events and the adequacy of the responses to them in the light of knowledge at the time. We have sought to do so thoroughly and fairly. We have reviewed not only the years since BSE first emerged, but the events that led up to it. We have read a large number of scientific publications. We have sifted 3,000 files of documents, and have studied 1,200 statements and many contributions from the public, whom we have sought to keep fully up to date with every stage of our proceedings. We have listened to 138 days of public oral evidence from 333 witnesses.

12 A recurring theme in the BSE story – a point we look at in detail later in our Report – has been growing public suspicion and dissatisfaction that important information was not being shared and discussed openly so that people were denied proper choices in matters that deeply affected them and their families. One of our goals in settling the conduct of our Inquiry was to make our investigations as open as good practice and modern technology could ensure, with any significant material we received made freely available to all. Witnesses’ statements and transcripts of our hearings have been made available free of charge to all with access to the Internet. Hundreds of fuller dossiers of assembled factual material have throughout been available in more conventional form for those who wish to inspect them at our offices. We have placed in the public domain a unique corpus of official documents, and we have sought to throw light on a range of normally internal public policy processes. Our aim has been to be as thorough, open and fair as we could possibly be. Annex 1 to this volume describes the procedures we adopted for this purpose.

13 We have welcomed the spirit of cooperation we have been shown by the previous and current administrations and many other organisations in opening their archives to us. As some of our witnesses pointed out, they too are consumers of animal products and they too have children and grandchildren whom they cherish. We have made heavy demands for information on many witnesses and the voluntary response has been remarkable.

Our task

14 Our Terms of Reference require us:

To establish and review the history of the emergence and identification of BSE and variant CJD in the United Kingdom, and of the action taken in response to it up to 20 March 1996; to reach conclusions on the adequacy of that response, taking into account the state of knowledge at the time; and to
Establishing and reviewing the history of the emergence of BSE and vCJD requires us to consider what occurred and why. Ascertaining what occurred is not straightforward, for we believe that the initial emergence of BSE was neither recorded nor appreciated, and the aid of the epidemiologist is needed to try to reconstruct what happened. Ascertaining why BSE and vCJD occurred is even more difficult. Many scientists around the world have been conducting research which bears on these questions. We have reviewed the results of this research to see what, at the time of writing our Report, can be said with a reasonable degree of confidence about the causes of BSE and vCJD. Many questions remain unanswered, but we believe that a number of widely held beliefs can be shown to be misconceptions.

Next we are required to establish and review the history of the response to the emergence of BSE and vCJD up to 20 March 1996. That was the day on which the Government announced the identification of a new variant of CJD and the conclusion that the cases were probably linked to exposure to BSE.

Establishing the response to the emergence of vCJD involves focusing on the few months leading up to 20 March 1996, during which the emergence of the disease was identified. In contrast, considering the action taken in response to the emergence of BSE has been a massive exercise. That action spanned a period of nearly ten years, starting in December 1986, when the emergence of a new disease in cattle was first suspected. The action involved the five Government Departments to which this Report is addressed, and on occasion other Departments, the Prime Minister and Cabinet. It involved local authorities throughout the United Kingdom charged with enforcing Regulations introduced to deal with BSE. It involved many other public bodies. It involved the rendering industry, the animal feed industry, the food industry, the pharmaceutical industry, and, of course, the farming industry. It involved the media. It involved the consumer and it involved the public.

When we speak of the consumer, we do not refer simply to those who ate beef. Products derived from the cow enter the food chain in a variety of guises. Tallow, the fat that is extracted by the rendering process, and gelatine, derived from the skin and bones of cattle, are used in a wide variety of foodstuffs. But the public was involved not merely as consumers of food. Bovine tissues and fluids are used in, or in the production of, medicinal products swallowed, injected or inoculated. They are used in the manufacture of surgical devices. They are incorporated in cosmetics. The emergence of BSE put in question the safety of each of these products. It also raised questions about the handling of waste derived from the manufacture of these products or directly from carcasses.

Not only have we been required to establish the action taken in response to the emergence of BSE, we have been asked to reach conclusions on the adequacy of the response, taking into account the state of knowledge at the time.
On the last day of the hearings we made the following observations about this part of our task:

The mechanisms by which policy decisions in Government are taken are complex. The important decisions involve preparation of information and advice to submit to a Minister, preparation that often involves a number of different officials. It is easy with hindsight to assert that an assumption should not have been made, or that a decision was inadequate, misguided or dilatory, or that there was a culpable failure to take action that the situation required. Public opinion, as events unfolded and reached crisis point, has made many such value judgements. Hardly a day goes by today without BSE being referred to in the media as epitomising maladministration, usually by the use of an epithet such as ‘the BSE scandal’. We believe that we have been asked to consider the adequacy of the response to BSE so that these accusations, insofar as they relate to the period with which we are concerned, can receive a fair and dispassionate consideration.

As we shall shortly explain, in the years with which we are concerned, most of those responsible for responding to the challenge posed by BSE emerge with credit. But we have found that a number of aspects of the response to BSE were inadequate. There are lessons to be learned from the events of those years. We stress that identifying those lessons is more important than examining whether individuals should be criticised. Nevertheless, any description of inadequacies is bound to lead people to ask whether individuals are to be criticised. We have given anxious consideration to that question.

A finding that an action constituted an inadequate response to BSE does not necessarily mean that those responsible for the action should be criticised. An action may not have been adequate because it did not satisfactorily deal with things that were known about a problem at the time. But it would not be right to criticise an individual unless, given the knowledge of that particular individual, he or she should have acted differently.

We have approached our task on the premise that it ought to be possible to identify those with responsibility for the policy decisions, the actions to implement policy and the public communications that together made up the response to BSE.

In practice we have found allocation of individual responsibility difficult. In part this has been due to the passage of time, which has rendered individual recollection of material facts at least unreliable and frequently non-existent. In part this has been due to the complexity of the administrative processes. The willingness of those concerned to give us unrestricted access to internal papers, and to disclose these to the public, has enabled us and the media and the public to gain an insight into those processes which we believe to be unprecedented.

Our Inquiry has led us to consider in depth:

- the relationship between Ministers and officials;
- the relationship between Government Departments;
- the relationship between administrators and professionals within Departments;
• the relationship between public authorities and expert advisers; and
• the relationship between central and local government.

26 These relationships formed the structure within which major and minor decisions of policy came to be taken and implemented.

27 When considering individual responsibility we have had to bear in mind this structure. We have had to bear in mind the way in which the public administrative system works. Many decisions are the product of a team effort to which individuals have made different contributions. A faulty decision may be the result of an error of judgement in assessing the available scientific and other data, or it may have resulted from an individual failure or failures in the provision of data, or the provision of expert advice in relation to it.

28 We have had to bear in mind the constraints on advisers and decision-makers: constraints of law, constraints of resources, constraints of established government policy; and constraints of the legitimate interests of the agricultural and other industries as well as those of the consumer. The background volumes of our Report (which, as we explain below, have been prepared by Inquiry staff) contain information about these constraints.

29 We describe in Annex 1 to this volume the procedures that we adopted to ensure that this Inquiry was thorough, open and fair. These included particular procedures adopted in Phase 2 of the Inquiry for those areas which we considered might give rise to criticisms of individuals. Fairness demanded that individuals be given notice of any potential criticisms. Such a course had its costs. Those notified of potential criticisms, and the lawyers advising them, naturally devoted and diverted their efforts to attempting to meet the criticisms. This tended to focus attention on the areas to which the potential criticisms related, albeit that these were not necessarily the most important areas of the Inquiry, and thus to unbalance the process.

30 In considering the adequacy of the action of individuals we have kept in the forefront of our minds the dangers of hindsight. We have had regard to all the surrounding circumstances which have often explained and excused action which at first blush seemed open to criticism. We have had well in mind that in any situation there is likely to be a range of responses from the inspired to the unimaginative, all of which fall within the compass of a reasonable response. Only where, having regard to all the relevant circumstances, we have concluded that the response of an individual fell below the standard to be expected of a person holding his or her position, have we indicated that the individual was at fault. We have done so in clear language, stating that the individual ‘should’ or ‘should not’ have acted in a particular way. Where we have not made an express criticism, none should be implied. So as to avoid any misunderstanding, a list of individual criticisms can be found in Annex 2 to this volume, with cross-references to locations in the Report where the matter is discussed.

31 Consistently with this approach, when considering the actions of Government Ministers, we have not adopted the traditional convention whereby Ministers are held accountable for the actions of those in their Department, regardless of their personal level of involvement. As with other individuals, we have only criticised a Minister where we have concluded that, in all the circumstances, his or her response
fell below the standard to be expected of that Minister in the light of his or her knowledge at the time.

32 This is not to say that we have proceeded on the basis that a Minister should never be criticised for following advice from officials. The fact that a Minister has followed this course cannot preclude the conclusion that he or she should have acted differently. It is, however, an important factor when considering whether a Minister should be criticised.

33 There are some instances where we have found the response inadequate, but have not identified failings on the part of specific individuals. These are usually cases where we have felt that, having regard to the constraints on our time and resources, an attempt to identify individual responsibility could not be justified. In all such instances we would emphasise that it would be wrong and unfair to infer fault on the part of any individual.

The structure of the Report

34 Almost every aspect of the BSE story takes us into territory that may well be unfamiliar to the average reader of this Report. Anyone who wishes to follow the story fully will need to understand:

- the involvement of government in UK agriculture during and after the Second World War;
- the influence of the Common Agricultural Policy on agricultural production;
- the digestive system of the cow;
- intensive feeding methods designed to boost milk production;
- feed compounding;
- rendering;
- slaughterhouse techniques;
- the administrative structure of the Government Departments and local authorities involved;
- the powers available to government to regulate and enforce;
- the use made by government of advisory committees;
- basic human and animal biology;
- genetics; and
- current scientific knowledge in relation to the nature of Transmissible Spongiform Encephalopathies (TSEs).

35 These topics form the background to ten years of activity in response to the emergence of BSE. We must review that activity in context. A key consideration in an exercise as far-ranging and complex as this Inquiry is how best to present and make widely available the significant material and findings we have assembled.
36 We are conscious that while some will wish to follow, in detail, our examination of the BSE story, or some specific parts of it, most will not have the time or the energy for such an exercise. The majority will wish to read, in simple language, a summary account of the emergence of BSE and how it was handled, with particular reference to its implications for human health. More particularly, the majority will be looking to us to answer, as best we can, a number of questions about BSE, vCJD and the conduct of government in relation to them over the period with which we are concerned. This volume aims to meet those wishes of the majority.

37 The emergence of BSE called for responses of different kinds and in relation to different areas of activity. In this volume we propose to follow a topic-based approach. At the outset we shall explain the nature of Transmissible Spongiform Encephalopathies and examine the assumption which lies at the root of this Inquiry: that the variant of the human disease CJD is a consequence of the emergence of BSE. We conclude this chapter by setting out the BSE story in a nutshell. In the next chapter we have included sections about the industries which feature in the BSE story; how government was set up to handle an issue like BSE; and handling risk. We aim in that chapter to give much of the background that will enable the reader to follow the story in the rest of this volume.

38 Chapters 3 to 6 contain a narrative of a part of the BSE story which, for the most part, has been in the public eye:

- the emergence of BSE;
- the theories as to its cause;
- the measures taken to try to eradicate it;
- the concerns that humans might be able to catch BSE and worries about the safety of beef;
- the official reassurances about the risk to humans and the safety of beef; and
- the dreadful discovery that BSE had probably been transmitted to humans after all.

39 In Chapters 7 to 9 we turn to parts of the story of which the public was generally not aware at the time. As a result of recent media coverage, the subject matter of Chapter 7 – steps taken to address the possibility that BSE might have infected medicines, vaccines and cosmetics that used bovine products as ingredients or in the manufacturing process – has now become public. But Chapters 8, 9 and 10 deal with the less familiar topics of guidance given to occupational groups which may have been at risk from handling potentially infected tissues at work; the consideration given to tracing all the uses of bovine tissue and thus all possible pathways along which infection may have been transmitted; and the impact of BSE on pollution and waste control. In Chapter 11, we summarise our main findings about the part played by the Territorial Departments, as they then were, in Wales, Scotland and Northern Ireland.

40 In Chapter 12 we set out the conclusions we have been able to draw about the scientific response to BSE, dealing with some important questions, such as the origin of the BSE agent.
41 We conclude with two chapters which fulfil what we believe to be the essence of our remit, that is, to understand why things happened in the way they did and to suggest how lessons may be learned from the BSE story for the benefit of those facing similarly difficult situations in future.

42 In summarising our findings and conclusions in a manner and at a length which we hope will make them accessible to all, we have had to paint with a broad brush and to leave untouched some parts of the gigantic canvas. The picture is painted in greater detail in the remaining 15 volumes, starting with Volume 2, which contains an analysis of the scientific evidence. Volumes 3 to 9 contain a detailed description and analysis of the events which are summarised in this volume.

43 Volume 10, which is a background volume, describes the impact of BSE on the economy and looks at how international trade was affected. Before BSE emerged, the majority of exports from the UK, of both live cattle and beef, went to the European Union (EU). After BSE emerged, these exports were subjected to restrictions that were imposed under European law. They did, however, benefit from the protection of the Single European Market, which made it unlawful for individual members of the EU to impose more stringent requirements on UK exports. Our Terms of Reference require us to consider the response to the emergence of BSE in the UK. We have not traced the deliberations that took place in Europe – in which representations of the UK played a key role – which determined the extent of the restrictions consequent upon BSE that were placed on our trade with the EU.

44 So far as the export of live cattle was concerned, the EU response was to restrict this to cattle of a BSE-free provenance which, after 1990, were aged less than six months. So far as beef was concerned, exports were restricted to beef on the bone of a BSE-free provenance, or beef off the bone from which all obvious nervous and lymphatic tissue had been trimmed. From December 1994 there were exemptions in respect of beef from younger cattle.

45 Statistics of exports of cattle and beef during the period with which our Inquiry is concerned are set out in Chapter 5 of Volume 10. They make interesting reading. Despite the EU restrictions, our exports of live cattle to the EU climbed steadily between 1988 and 1994, dropping only slightly in 1995. Outside the EU, sales of live cattle slumped to negligible proportions after 1989. The value of exports of beef on and off the bone to the EU climbed by 1995 to well over double their value in 1987. Outside the EU, sales of beef off the bone slumped between 1986 and 1993, before recovering to close to previous levels. Sales of beef on the bone reduced to negligible proportions after 1987.

46 Volume 11 looks at the important role in the BSE story played by scientific committees and independent scientists. It forms the basis for a large number of lessons to be learned about the use of expert scientific committees which are set out in the final chapter of this volume.

47 The factual parts of these volumes have been based in large measure on ‘draft factual accounts’, which were collated from the evidence, were published as the

---

4 The European Union (EU) came into existence on 1 November 1993 as a result of the Maastricht Treaty. It incorporated but did not replace the European Community. Throughout the volumes of this Report, the term EU is generally used for consistency’s sake (even if sometimes chronologically incorrect), except where specific reference is made to the functions conferred by the European Community Treaty or to its legal effect.
Inquiry progressed, and have been revised on the basis of comments received and additional evidence. To these we have added, in Volumes 2 to 9 and 11, sections of comment and discussion in which we have considered conflicts of evidence and explained the conclusions that we have drawn from the facts. Readers who want detailed explanations for the findings and conclusions set out in this volume will find them in those volumes. They will also find an abundance of references to source material, which will remain accessible to the public. In this volume we have sought to keep references to a minimum.

Volumes 10 and 12 to 15 contain background material which provides a detailed context in which the BSE story is set. Volume 16 contains relevant reference material. It should be noted that Volumes 10 and 12 to 16 are background volumes which have been prepared by researchers on the Inquiry team under our supervision and guidance. Conclusions of the Committee are not to be found in these volumes.

It has been clear that speedy access to Inquiry material through the Internet has been widely appreciated, and we have therefore cast and referenced our Report and its supporting material in a form immediately transmissible through this medium. We hope that it will thus prove another example of open practice on matters of legitimate public concern.

Transmissible Spongiform Encephalopathies

Our Terms of Reference speak of two diseases: BSE, a disease of cattle; and variant CJD, a human disease. These are varieties from a rare group of diseases known as Transmissible Spongiform Encephalopathies (TSEs). TSEs cause the appearance of microscopic holes in the brain, giving it a sponge-like appearance – hence the term ‘spongiform’. They are invariably fatal and affect both humans and animals. In 1986 a number of TSEs had been identified both in animals – scrapie in sheep and goats, Chronic Wasting Disease (CWD) in wild deer in North America and Transmissible Mink Encephalopathy (TME); and in humans – Creutzfeldt-Jakob Disease (CJD), Gerstmann-Sträussler Syndrome (GSS), kuru and Fatal Familial Insomnia (FFI). Although a signal feature of these diseases is that they are transmissible in the manner described in paragraph 52 below, they can occur, at least in humans and probably in other species, as a result of a genetic mutation that is inherited or, in some cases, that may arise spontaneously.

When BSE was first identified, the nature of the infectious agents causing TSEs was a matter of controversy. It was known that the agents were extremely difficult to inactivate – they could withstand treatments commonly used to disinfect virus-contaminated materials – and that researchers had failed to detect an immune response in hosts to their presence in a variety of experiments. Although these features suggested that TSEs were not caused by conventional viruses, some believed that they must be caused by an unconventional virus. This belief was challenged by those who thought that TSEs were transmitted as a result of a reaction between proteins. This theory has now won general, though not universal, acceptance.

How, under this theory, does transmission of these diseases occur? Let us take BSE as an example. The building blocks of every animal, including the human
animal, are proteins. These are minute particles which have different chemical compositions. BSE involves the deformation of one of these proteins (prion protein) in very large numbers within the brain of the cow, until the brain develops a spongy appearance and is fatally damaged. The same deformation of this protein takes place in other specific tissues in the cow. If some of the deformed proteins of an animal suffering from BSE are introduced into the body of another animal or into a human (‘the host’), they may induce similar proteins that are found in the host to deform in the same way. By a kind of chain reaction, deformation of these proteins may spread to and within the brain of the host, until finally the brain is so damaged that the host is taken ill and dies.

53 The prion protein exists in its normal form in all animals, but its chemical composition is not precisely the same in each. It can even have slight variations in animals of the same species as a result of minor variations of the prion gene. The more similar the prion protein in infected animals to that in the host animal, the easier the transmission of a TSE appears to be. Thus transmission is easiest between animals of the same species. When the animals are of different species, the ‘species barrier’ will sometimes prevent transmission altogether.

54 The obvious way in which deformed protein from an animal incubating a TSE may be introduced into another animal is as food. There are, however, other possibilities. For instance, medical products administered by injection are sometimes derived from animal tissues or fluids. Experiments have shown that it is very much easier to transmit a TSE to an animal by injecting infected tissue directly into the brain than by feeding it to the animal. A minute quantity will suffice for such intracerebral transmission; indeed CJD has sometimes been transmitted on surgical instruments used in neuro-surgery despite their sterilisation.

Transmission to humans

55 The two most worrying questions people ask about BSE are:

- Is it certain that the victims of the variant form of CJD have caught BSE?
- And, if so, how many victims are there likely to be?

56 We shall here summarise our conclusions about the link between BSE and vCJD, which are the subject of more detailed coverage in vol. 2: Science and in vol. 8: Variant CJD.

57 The unusual clinical features and novel pathology of the early cases of CJD in young people suggested this was a new variant of the disease. Much experimental work has been done to investigate whether there is a link between this new variant of CJD and BSE, and we believe there is now sufficient evidence to be confident that vCJD is caused by the transmission of BSE to humans. In outline, the main evidence, in addition to the temporal and geographical association of the two diseases, which leads us to reach this conclusion is as follows:

---

5 Professor Stanley Prusiner, who coined the term ‘prion protein’ and who was awarded a Nobel prize for his work in this field, assisted us with a presentation of the prion theory in Phase 1 of the Inquiry.
i. in strain-typing studies in both mice and primates the disease patterns (incubation period and disease pathology) of BSE, vCJD, feline spongiform encephalopathy (FSE) and TSEs of exotic ruminants were shown to be extremely similar, while differing from those of scrapie and sporadic CJD;

ii. patterns known as glycosylation patterns, produced by analysing samples of brain using a technique called western blotting, are the same for BSE and vCJD. The patterns for BSE and vCJD are different from those for other TSEs such as sporadic CJD and iatrogenic CJD; and

iii. in transgenic mice in which the mouse prion gene has been replaced by the bovine prion gene, inoculation with tissue derived from BSE-infected cattle produces the same disease pattern and incubation period as inoculation with tissue derived from patients with vCJD.

58 It is not possible to say whether BSE was transmitted to humans through consumption of beef or beef products, or by some other means; nor is it possible to say when individual infection occurred. There are a number of other unanswered questions:

• Why does vCJD affect young people? Possible explanations meriting further investigation include: the possible disproportionate consumption by young people of beefburgers, some of which contained high-risk material; higher incidence of infections such as tonsillitis or gastroenteritis in children than adults, giving rise to transmission through broken skin or mucous membranes; infection through gum lesions associated with eruption of teeth; and transmission via childhood vaccines prepared in cultures containing bovine constituents.

• How many more people will succumb to vCJD? To attempt to answer this question is not required by our Terms of Reference, nor would we feel able to do so. Estimates of the possible size of a vCJD epidemic are made difficult by the many variables associated with the disease. Many important factors in determining the likelihood of BSE transmission to an individual are unknown, such as dose, route of exposure, incubation period, genetic susceptibility and scale of the species barrier between cattle and humans. Nevertheless, several groups of epidemiologists and statisticians have attempted to predict the possible number of cases. Projections have in the past ranged from small numbers to many millions and it is not possible at this stage to reach a firm estimate.

• Is occupation a risk factor in vCJD? Among occupational groups exposed to BSE, to date farmers are the only group to have an excess over the incidence of CJD for the population as a whole. Between 1990 and 1996 four cases of CJD occurred in farmers who were known to have had cases of BSE on their farms. In addition, two farmers’ wives succumbed to CJD. The affected farmers were aged between 54 and 64 and had signs and symptoms typical of sporadic CJD. They did not have glycosylation patterns associated with vCJD. To date, no one has demonstrated a link between these cases and BSE.

6 It is thought that domestic cats caught FSE and exotic ruminants a related TSE through the consumption of BSE-infected food
INTRODUCTION

The story in a nutshell

What happened?

59 This is a summary of the more significant events in the BSE story. In responding to the emergence of BSE, the Ministry of Agriculture, Fisheries and Food (MAFF) and the Department of Health (DH) took the lead. For the most part, Wales, Scotland and Northern Ireland followed that lead. This summary will focus on the action taken by MAFF and DH.

60 A TSE known as scrapie has been endemic in the sheep population of the UK for nearly 200 years. In the later stages of the disease the fabric of the brain is attacked. The pathologist can diagnose the disease by the spongiform appearance of the diseased brain. At the end of 1986 pathologists at the Central Veterinary Laboratory (CVL) identified similar degenerative changes in the brain samples of diseased cattle from two different herds. These were early cases of BSE.

61 By May 1987 this novel disease had been confirmed in four herds. No publicity, even within the State Veterinary Service (SVS), had been given to these early cases and it is likely that others had gone unrecognised and unreported. From May, however, the fact of the existence of a novel disease was gradually disseminated and Mr John Wilesmith, head of the CVL’s Epidemiology Department, was asked to investigate its cause.

62 Over the next six months, as he carried out his task, reported incidents of the disease proliferated. By 15 December 1987 there were 95 confirmed cases on 80 farms. Mr Wilesmith had formed the provisional view that the cause of the outbreak was contaminated meat and bone meal (MBM) that had been incorporated in cattle feed. His confidence in this theory grew stronger early in 1988, and he concluded that the likely contaminant was offal of scrapie-infected sheep, rendered down to make MBM. Enquiries of feed compounders tended to confirm this view.

63 On 18 May 1988 Mr John MacGregor, the Minister of Agriculture, on the advice of Mr William Rees, the Chief Veterinary Officer (CVO), decided on what proved to be the principal step taken to eradicate BSE. A prohibition on feeding ruminant protein to ruminants (‘the ruminant feed ban’) was introduced on 14 June 1988 to take effect on 18 July. This was, at the time, regarded as a measure to protect animal health. The risk that BSE posed to human health had not, however, been ignored.

64 Officials at MAFF had been concerned from the outset at the possibility that BSE might pose a risk to human health. Diseased cattle were going into the human food chain. Scrapie was not transmissible to humans, but there was no certainty that the same would be true of BSE. By 19 February 1988, 264 cases of BSE from 223 farms had been confirmed. On 24 February Mr Derek Andrews, the Permanent Secretary, forwarded a submission to Mr MacGregor. This recommended that BSE should be made a notifiable disease and that a policy of compulsory slaughter with compensation should be introduced. Mr MacGregor had reservations about such a policy and accepted the suggestion that the advice of Sir Donald Acheson, the Chief Medical Officer (CMO), should be sought on the implications that BSE had for human health.
Sir Donald, in turn, recommended that an expert working party should be set up to advise on the implications of BSE. This was done. The Working Party was chaired by Sir Richard Southwood.

Before the first meeting of the Southwood Working Party, and at the same time that the ruminant feed ban was introduced, Mr MacGregor, on the advice of his officials, introduced a requirement for compulsory notification of all cases of BSE.

On 21 June 1988 the Southwood Working Party made interim recommendations that included the compulsory slaughter of animals showing symptoms of BSE and the setting up of a committee to advise on research. The Government accepted these recommendations and, on 8 August 1988, an Order came into force making slaughter of BSE suspects compulsory. Compensation of 50 per cent of the sound value of the animal was paid if, on post-mortem, it was shown to have had BSE and 100 per cent if it did not. Although made under the Animal Health Act 1981, the primary object of this measure was to take sick animals out of the human food chain.

By 13 January 1989, 2,296 cases of BSE had been confirmed on 1,742 farms.

The Southwood Report was submitted to Ministers on 9 February 1989. This endorsed Mr Wilesmith’s conclusion that the source of infection was probably scrapie-infected meat and bone meal. It concluded that it was ‘most unlikely that BSE would have any implications for human health’. It recommended that the Health and Safety Executive (HSE) and the authorities responsible for human and veterinary medicines, which had already been alerted by the Working Party, should take appropriate measures to address possible risks posed by BSE, and advised manufacturers of baby foods not to include in their products ruminant offal including thymus, which, from what was known about scrapie, would be most likely to be infective. Sir Richard Southwood clarified later in February that this offal did not include liver or kidney.

The Working Party concluded that the risk posed by BSE-infected animals which had not yet developed clinical signs did not justify any further measures to protect human food. The Government accepted this, and on publication of the Southwood Report announced that secondary legislation would make it illegal to sell baby food containing the types of offal identified by the Report. MAFF Ministers, however, had concerns which, after discussion with officials and with DH and after wide consultation, led, on 13 November 1989, to the introduction of a ban on the use for human consumption of Specified Bovine Offals (SBO), namely those tissues in cattle considered most likely to be infective. This became known as ‘the human SBO ban’. Tissues from cattle aged under six months were exempt from the ban on the basis that scrapie infectivity had not been found in lambs of this age.

Meanwhile, on 27 February 1989, the establishment of a committee chaired by Dr David Tyrrell was announced. The Tyrrell Committee was to advise on research in relation to BSE, thus implementing one of the first recommendations of the Southwood Working Party. This Committee met three times and delivered to the Minister of Agriculture and the Secretary of State for Health what they described as an ‘Interim Report’ on 13 June 1989. This identified the key research questions that needed to be answered and set in an order of priority the research studies needed to answer those questions.
The Report was not published until 9 January 1990. By this time funding had been put in place which enabled the Food Minister, Mr David Maclean, to announce that all projects identified by the Tyrrell Committee as ‘urgent’ or of ‘high priority’ had either been put in train or would start as soon as possible. Experiments to check the belief that BSE was transmissible had been put in hand at an early stage. In September 1988 transmission to mice by intracerebral inoculation of brain tissue had been confirmed. By February 1990 transmission to cattle had been established by the same route and transmission to mice by oral ingestion had been achieved.

Meanwhile, on 28 July 1989, the EU banned the export of UK cattle born before 18 July 1988 and of offspring of affected or suspect females. This was the first of a number of restrictions placed by the EU on the export from the UK of live cattle and (from June 1990) of beef.

By the end of 1989, 10,091 cases of BSE had been confirmed in the UK.

Anxiety had been expressed in many quarters that 50 per cent compensation might be inadequate to procure full compliance with the requirement to notify BSE suspects and, on 14 February 1990, Mr John Gummer, who had succeeded Mr MacGregor as Minister of Agriculture, introduced entitlement to 100 per cent compensation.

On 1 March 1990 the EU restricted exports of live cattle to those aged less than six months. Importing Member States were required to ensure that these were slaughtered before they reached that age. Offspring of whatever age of affected or suspected females continued to be banned from export.

On 3 April it was announced that Dr Tyrrell was to chair a new expert committee – the Spongiform Encephalopathy Advisory Committee (SEAC). The Committee had a wider membership than the Tyrrell Committee and wider terms of reference:

To advise the Ministry of Agriculture, Fisheries and Food and the Department of Health on matters relating to spongiform encephalopathies.

It was government policy in relation to BSE to act on ‘the best scientific advice’. Thereafter the Government was to look to SEAC to provide that advice.

One of the recommendations of the Southwood Working Party had been the need for surveillance of CJD cases in order to detect whether there were any changes in their incidence that might be attributable to BSE. In May 1990 the CJD Surveillance Unit was set up under Dr Robert Will, a consultant neurologist at the Western General Hospital in Edinburgh.

On 10 May 1990 it was announced that a Siamese cat had died of a spongiform encephalopathy – the first known case of feline spongiform encephalopathy (FSE). This resulted in a rash of media comment, speculating that the cat had caught BSE and that humans might be next. Humberside Education Authority had already banned beef from school meals and a number of other Authorities threatened to follow this example. Public statements by the CMO and by Mr Gummer that beef was safe to eat failed wholly to reassure. The House of Commons Agriculture Committee announced an Inquiry into BSE. After receiving evidence from most of
the key players in the BSE story, the Committee reported on 12 July 1990 that, while there were too many unknowns to say anything with absolute certainty, ‘we heard no evidence of any sort to constrain those taking a more balanced view of the risks from eating beef’. The measures taken by the Government ‘should reassure people that eating beef is safe’.

81 On 8 June 1990 the EU Council of Ministers agreed that bone-in beef exported from the UK must come from holdings where BSE had not been confirmed in the previous two years, while boneless beef was required to have obvious nervous and lymphatic tissue removed.

82 Meanwhile, there had been controversy as to whether the SBO that had been banned from human food should be permitted to be fed to animals. Pet food manufacturers had voluntarily ceased to incorporate it in their products. UKASTA, the feed producers’ trade association, had pressed strongly for a ban on including SBO in the material rendered to make MBM for inclusion in pig and poultry feed, and advised their members to exclude it. MAFF officials and Ministers opposed a ban on the ground that it was without any scientific justification. SEAC was about to advise on this question when, early in September, a pig, which had been inoculated with BSE-infected brain tissue, succumbed to the disease. In an emergency meeting SEAC advised that, as a precautionary measure, SBO should not be fed to any animals. MAFF, which had anticipated this possibility, immediately banned the incorporation of SBO or its products in animal feed (‘the animal SBO ban’). Export of feed containing SBO to the EU was also banned. This was followed in July 1991 by a ban on the export of material derived from SBO to third countries.

83 Among the many matters on which SEAC was asked to advise were slaughterhouse practices. There was concern that the removal of brain and spinal cord (both SBO) in slaughterhouses might contaminate meat going for human consumption. There was also concern about the practice of the mechanical recovery of remnants of meat and other tissues adhering to the vertebral column, in that these might include scraps of spinal cord not cleanly removed by slaughterhouse operators. SEAC advised that head meat should be removed before brain, but that no further measures were necessary provided that the rules were properly followed and supervised. This advice was implemented first by guidance and then, in March 1992, by statutory regulation.

84 By the end of 1990, 24,396 cases of BSE had been confirmed in the United Kingdom.

85 One of a number of recommendations of the House of Commons Agriculture Committee was that the Government should ‘establish an expert committee to examine the whole range of animal feeds and advise on how industries which produce them should be regulated’. Some debate ensued as to how to implement this recommendation, but on 6 February 1991 MAFF announced the establishment of an Expert Group on Animal Feedingstuffs chaired by Professor Eric Lamming. It met on 14 occasions over the next year and reported on 15 June 1992. The Group considered the steps taken to prevent the BSE agent being transmitted to animals in feed and concluded that they were satisfactory and adequate. In particular the Group considered whether the practice of feeding animal protein to animals should be
discontinued. It decided that there was no scientific justification for such a step. It did, however, recommend that:

\[
\ldots\text{an independent Animal Feedingstuffs Advisory Committee be established to take an overview of all feedingstuffs issues.}
\]

86 Although the Government initially accepted this recommendation, it subsequently decided not to proceed with it.

87 With compulsory slaughter of sick animals and the human SBO ban to deal with potentially infective tissues in apparently healthy animals incubating BSE, the Government considered that there were in place appropriate measures to deal with the risk that BSE might be transmissible to humans in food. Action was taken to see that medicinal products both for humans and for animals were not sourced from potentially infective bovine tissues. Ruminants were protected by the ruminant feed ban and other animals by the animal SBO ban. No further major measures were considered necessary to protect human or animal health in the period with which we are concerned. In March 1992 SEAC concluded ‘that the measures at present in place provide adequate safeguards for human and animal health’. Several relatively uneventful years were to pass before it became apparent that the measures in place were not achieving all that had been expected of them.

88 Because of BSE’s lengthy incubation period, it was appreciated when introducing the ruminant feed ban that years would pass before it would have a visible effect. What was not known was the rate at which cattle had been infected in the period up to 18 July 1988, when the ruminant feed ban came into force. At the time of the Southwood Report suspected cases of BSE were being reported at the rate of about 400 a month. It was considered that these had been infected with scrapie and that this source would have continued to infect cattle until the ban at about the same rate. Whether, or to what extent, recycling of BSE might have increased the rate of infection was not known.

89 It soon became apparent from the numbers of BSE cases reported that the rate of infection had not reached a plateau, but had been increasing rapidly in the years leading up to the ruminant feed ban, and that the reason for this was the effect of recycling the BSE agent in MBM.

90 Thus the Government found it had to deal with many more cases infected before the ban than it had expected. But of even more concern were cases in cattle that had been born after the ban (BABs). The first of these was announced on 27 March 1991.

91 When exploring the possible sources of infection of the BABs, the CVL epidemiologists were able to rule out maternal transmission in most cases. The likely source of infection of the earlier BABs was thought to be ruminant feed in which ruminant protein had been incorporated before the ban and which was in the distribution pipeline, or still unused on farms when the ban came into force. This remained the view of MAFF officials at the beginning of 1994, by which time Mrs Gillian Shephard had succeeded Mr Gummer as Minister of Agriculture. Cross-contamination of ruminant feed by non-ruminant feed in the feedmills was

\footnote{For statistics, see vol. 16: Reference Material}
considered, but discounted after September 1990, when the animal SBO ban should have prevented SBO from being incorporated in any animal feed.

92 In the course of 1994 opinions changed as to the source of infection of BABs. By August the CVL had reached the conclusion that the more recent BABs had been infected by feed which had been contaminated in the feedmill by feed containing ruminant protein. Two factors had led to this conclusion. First, there had been an increasing volume of evidence, some of it cogent, of widespread infringement of the animal SBO ban, so that SBO was contaminating non-ruminant feed. Second, interim results of an experiment, which started in 1992, indicated that a single quantity of as little as 1 gram of infective material – the size of two peppercorns – had sufficed to infect cattle to which this had been fed.

93 MAFF officials approached the problem of the cross-contamination of cattle feed on two fronts. Their primary emphasis was on tightening up the implementation of the animal SBO ban. This was facilitated by the transfer of enforcement functions in slaughterhouses to central government. What had been the responsibility of some hundreds of individual local authorities became the task of a new national Meat Hygiene Service (MHS) from 1 April 1995. A revised statutory scheme was introduced that required SBO to be identified by a distinctive blue dye and kept separate at all times from other material. At the same time plants rendering SBO were required to do so in separate facilities. The consultation process was thorough and lengthy, with the result that the introduction of the new Regulations was not completed until August 1995. Their introduction was combined with a campaign of more rigorous enforcement and monitoring of the Regulations by the MHS and the Veterinary Field Service (VFS).

94 At the same time as tightening up on the implementation of the animal SBO ban, MAFF officials took steps to address cross-contamination in feedmills. So far as these were concerned, effective monitoring of compliance with the ruminant feed ban had been initially impossible for want of any method of testing for the presence of ruminant protein in animal feed. It had been hoped that an ‘ELISA test’ would be perfected within about 12 months, capable of detecting this. In the event, it was not until 1994 that the test was ready for use, and even then its results were not sufficiently reliable to provide evidence that would support a prosecution for breach of the Regulations. The test was, however, employed on a voluntary basis, with cooperation from UKASTA, and resulted in at least some feedmills taking steps to reduce the possibility of cross-contamination.

95 Hindsight confirms that, between 1989 and 1994, the ruminant feed ban had resulted in a steady but substantial year-on-year reduction in the numbers of infections, and that the measures taken in 1994 and 1995 radically accelerated this decline (see Volume 16, Figures 3.2 and 3.34).

96 The years 1994 and 1995 also saw developments in relation to the risks posed by BSE to human health. An interim result of a pathogenesis experiment conducted by the CVL demonstrated infectivity in the distal ileum (small intestine) of a calf within six months of oral infection with BSE. This led MAFF, with the agreement of DH, to extend the human SBO ban to include the intestines and thymus of calves which had died aged over two months.
On 27 July 1994 the European Commission decided that existing restrictions on the export of UK beef should be replaced with two measures. One was a ban on export of bone-in beef except from cattle which had not been on holdings where BSE had been confirmed in the previous six years. The other measure affected beef from cattle which had been on such a holding within that time. This could not be exported unless it was deboned with adherent tissues removed. In December 1994 the Commission amended this decision to exempt from these measures beef from cattle born after 1 January 1992. Subsequently in July 1995 this exemption was replaced with one that exempted beef from cattle less than 30 months of age at slaughter.

In July 1994 Mrs Shephard was succeeded by Mr William Waldegrave, who oversaw the introduction of the MHS. He in turn was succeeded by Mr Douglas Hogg in July 1995. At the direction of Mr Hogg, the MHS set about raising standards of meat inspection, a task that was to prove to require the employment of several hundred additional staff.

More rigorous monitoring of slaughterhouses in 1995 disclosed a number of occasions on which Meat Inspectors had applied the health stamp to a carcass to which fragments of spinal cord remained attached. This led SEAC to recommend a ban on the practice of extracting mechanically recovered meat (MRM) from the spinal column of cattle. MAFF accepted that advice and introduced the ban in December 1995.

In the course of 1995 a number of events served to increase public anxiety that it might be possible to contract CJD as a consequence of eating beef. Cases of CJD were reported in farmers whose herds had had BSE and in several young people – the latter being particularly significant because up until then the disease had almost invariably struck down its victims late in life. A distinguished scientist questioned the safety of beef offal. These events received wide media coverage. The CMO and the Secretary of State for Health each responded with public assurances that it was safe to eat beef.

The first two months of 1996 saw the CJD Surveillance Unit and SEAC concerned at an increasing number of young victims of CJD. On 16 March SEAC advised the Government that a new variant of CJD had been identified in young people and that the most likely explanation was that these were linked to exposure to BSE before the introduction of the SBO ban in 1989. A series of urgent meetings of Ministers and then of the Cabinet ensued, and SEAC’s advice was sought as to further precautionary measures.

On 20 March 1996 the Government announced the likelihood that the recent cases of CJD in young people had resulted from exposure to BSE before 1989 and stated its intention to adopt further precautionary measures in accordance with SEAC’s advice. These were that carcasses from cattle aged over 30 months must be deboned and that the use of MBM in feed for all farm animals would be banned. These measures proved inadequate to reassure the public and, within two weeks, were replaced with a total ban on cattle over the age of 30 months being used for human food or animal feed.

By 20 March 1996 approximately 160,000 cattle affected by BSE had been slaughtered. In addition about 30,000 cattle suspected of BSE, but not confirmed to
have the disease, were slaughtered. These figures can be compared with over 3.3 million cattle slaughtered and destroyed under the Over Thirty Month Scheme in the period from March 1996 to the end of 1999.

104 This brief narrative has concentrated on events that have been most in the public eye. As we explained above, we shall also cover in later chapters of this volume precautionary measures taken in areas which, while important, did not come to the attention of the general public. These include medicines, cosmetics and occupational health.

**Why did it happen?**

105 The Report of an Inquiry such as this inevitably focuses on the areas where things went wrong. It is those areas that government and the public are most anxious to have thoroughly explored. For this reason we think it desirable to give at the outset an overview of why things happened in the way that they did.

106 Why initially a cow or cows developed BSE will probably never be known. Why the early case or cases began a chain of transmission that ended with hundreds of thousands of cattle becoming infected is now clear. It was because of the practice of rendering cattle offal, including brain and spinal cord, to produce animal protein in the form of meat and bone meal (MBM), and including MBM in compound cattle feed. This resulted in the recycling and wide distribution of the BSE agent.

107 Many have expressed the view that it was not surprising that a practice as unnatural as feeding ruminant protein to ruminants should result in a plague such as BSE. Had BSE emerged soon after this practice was introduced, there might have been force in this reaction. However, the practice of feeding MBM to animals in the UK dates back at least to 1926, when it was given statutory recognition in the Fertilisers and Feedingstuffs Act of that year. It is a practice which has also been followed in many other countries. It was recognised that it was important that the rendering process should inactivate conventional pathogens. Experience had not suggested that the practice involved any other risks. In these circumstances we can understand why no one foresaw that the practice of feeding ruminant protein to ruminants might give rise to a disaster such as the BSE epidemic. Accusations have been made both against the Government and against renderers of causing BSE by relaxing rendering standards. As we shall explain, changes in rendering practices and regulatory requirements are unlikely to have made any substantial difference.

108 There were a number of factors that made it inevitable that, whatever measures were taken in response to its emergence, BSE would be a tragic disaster:

- it had an incubation period of five years on average;
- it tended to strike a single cow in a herd;
- it had clinical signs which were similar to those of a number of other diseases in cattle;
- it was impossible to diagnose before clinical signs appeared; and
- it was transmissible to human beings, but with a much longer incubation period than that in cattle.
INTRODUCTION

109 These factors had the following consequences:

- the emergence of the disease may well have gone undetected for ten years or more from the time of the first cases. A farmer would not be likely to send a single casualty for a post-mortem. It was only when, by chance, several cases were experienced on the same farm that the pathology was carried out that disclosed the new disease;

- by the time that BSE was identified as a new disease, as many as 50,000 cattle are likely to have been infected;\(^8\)

- it is also likely that by this time some of the human victims had been infected;

- it was not until nearly ten years after BSE was identified as a new disease in cattle that the first human victims succumbed to the disease, thus showing that, contrary to expectation, it was transmissible to humans.

110 Given the practice of pooling and recycling cattle remains in animal feed, this sequence of events flowed inevitably from the first cases of BSE. It was inevitable that, whatever measures were taken, many thousands of cows would succumb to the disease in the years to come. It was inevitable that if humans were susceptible to the disease, some would be infected with it before its existence was even suspected.

111 The measures that were taken in response to the emergence of BSE greatly reduced the scale of the disaster. The MBM component of feed was diagnosed as the vector responsible for the disease with commendable speed, and the ruminant feed ban was a swift and appropriate response. That ban reduced the rate of infection by 80 per cent overnight and established a diminishing trend which would, ultimately, have resulted in the eradication of the disease. Unhappily, as the cases born after the ban were to demonstrate, there were shortcomings in formulating and carrying out both the ruminant feed ban and the animal SBO ban, which should have provided a second line of defence against infection of cattle feed. These shortcomings had serious consequences. Over 41,000 cattle that developed clinical signs of BSE in the years that followed were infected after the ruminant feed ban came into effect. Many more must have been infected but slaughtered before the signs developed. When the link between BSE and the new variant of CJD became apparent in March 1996, the Government was unable to demonstrate that the source of infection had been completely cut off. Had they been able to do so, some of the drastic measures that followed might have been avoided. The reasons for these shortcomings receive detailed consideration in our Report.

112 There is a popular misconception that the Government did nothing to protect the public against the risk BSE might pose to human health until the likelihood of transmissibility was demonstrated in 1996. It is important to emphasise that the most significant measures to protect human health were taken at a time when the likelihood of transmissibility to humans was considered to be remote. Those were the compulsory slaughter and destruction of sick animals introduced in August 1988 and later, in November 1989, the human SBO ban, which was intended to remove from the human food chain those parts of apparently healthy cattle most likely to be infective if the animals were incubating BSE. At the same time steps were taken to ensure that bovine ingredients of medicines came from BSE-free sources.

\(^8\) S9 Anderson para. 1
113 These were vitally important measures. For a period of nearly ten years continuous consideration was given to addressing the possibility that BSE might be transmissible to humans, although few believed that there was any likelihood of it. This is a matter for commendation.

114 Yet again, however, there were shortcomings: shortcomings which led to delay in introduction of the precautionary measures, and shortcomings in formulating and carrying out the ban. Despite the SBO ban, some potentially infective bovine tissues continued to enter the human food chain. The reasons for these shortcomings also receive detailed consideration in our Report.

115 The other casualty of the BSE story has been the destruction of the credibility of government pronouncements. Those responsible for public pronouncements – or at least some of them – were aware of the possibility that humans might have become infected before the slaughter policy and the SBO ban were introduced. They saw no reason to draw attention to this. They believed that the measures taken had effectively removed the ‘theoretical risk’ of infection. They were concerned that the public should not be misled by scaremongers or the media into believing that it was dangerous to eat beef when this was not the case. Ministers and, on occasion, the Chief Medical Officers, made statements about the safety of beef which were intended to reassure the public. Insofar as these statements were believed, many clearly treated them as assurances that BSE posed no danger to human beings. In the case of some, there was a growing scepticism as the media reported cases of possible human victims of BSE which were then challenged by the Government. When on 20 March 1996 it was announced that cases of new variant CJD were probably attributable to contact with BSE before precautionary Regulations were introduced, the reaction of the public was that they had been misled, and deliberately misled, by the Government.

116 We have examined with care the public pronouncements that were made about the risks posed by BSE, and have concluded that allegations of a government ‘cover-up’ of the risks posed by BSE cannot be substantiated. There were, however, mistakes in the way risk was communicated to the public, and there are lessons to be learned from these.

117 As we go through the story we shall describe in greater detail what happened and how it came to happen in the way it did. We shall consider the response to BSE of the individuals principally concerned in the story. At the end of this volume we shall review what went right and what went wrong, before turning to the lessons to be learned from the BSE story.