In The Matter Of:

BSE Inquiry

Day 5
March 17, 1998

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17th March 1998
DAY 5

THE BSE INQUIRY

The Committee

THE CHAIRMAN
(SIR NICHOLAS PHILLIPS)

PROFESSOR MALCOLM FERGUSON-SMITH

MRS JUNE BRIDGEMAN

Representation

MR STUART CATCHPOLE - Counsel for the Ministry of Agriculture, Fisheries and Food
MS SARAH MOORE - Counsel for the Department of Health
MR DAVID BODY - Solicitor for the nvCJD families
MR STUART ISAACS QC - Counsel for the National Farmers Union
MR PAUL WALKER - Counsel for The BSE Inquiry

(Transcription of Smith Bernal International
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Tuesday, 17th March 1996
(9.30 am)

MR WALKER: Sir, this morning Professor Lacey is here, and he has provided the Committee with a written statement in advance of his evidence, and it is a statement which I think has five annexes. Is that right, Professor Lacey?

A: True.

Q: Before we start our discussion, I would just like to remind all concerned that today we are seeking only to establish and review the course of events, because that is the purpose of Phase 1 of the Inquiry. I think, towards the end of this year, we will have interviewed the many other scientists and industry and consumer representatives, families and professional bodies and officials and former ministers who have been involved, and we will reach, at the beginning of the next year, Phase 2. That is the stage we have called "Clarification, conflicts of evidence and potential criticisms".

The procedure is that at the beginning of Phase 2, the Committee will consider potential criticisms, and if there are any, for example, that we wish you to answer, Professor Lacey, or anybody else, then the secretariat would write to you, and we would set out those matters,

and that would all be dealt with at that time.

Today, I am just going to be asking you questions about what you did at the time, and what you said at the time, and your reasons at the time. I might also ask you whether you think you could have approached something differently, and if you had, what the effect would have been. But neither I nor members of the Committee today will be making any criticisms of you at all, either explicit or implicit, in our questions, and that is not what we are seeking to do in Phase 1.

So when I ask a question, or if the Committee asks a question, if you think that involves some suggestion of criticism, or any suggestion that you "should" have done something differently, as opposed to "could" have done it differently, I would like you to tell us. Then we can stop and make sure we are sticking to the purpose of today, which is just to establish and the review the facts.

A: Thank you very much. I am pleased to be here, and I will do my best to help, and I will try not to be paranoid.

Q: I have been trying the same thing, probably for different reasons. Well, today, as I said, I am going to be asking you about the things you said and did in the period covered by the Committee's terms of reference. I expect you know that the Committee has been asked to look at the period from the emergence of BSE up to 20th March 1996. Now, that is a cut-off date in the terms of reference, 20th March 1996. I can assure you that the Committee knows about criticisms that you have made subsequently, and the criticisms you have made since 20th March 1996, as well as the criticisms that you made up to that time, will all be reviewed by the Committee for the purposes of Phase 2.

Today, though, what I am seeking to ensure, when I talk to you, is that the Committee has a thorough understanding of what you said and did in the period up to 20th March 1996 and your reasoning. So today, unless I tell you otherwise, I will be asking you not to use hindsight. I will be asking you to try to cast your mind back to events as they happened, and to recall what was in your mind at the time when you said and did things up to 20th March.

Well, I would like to begin, then, with your statement. One of the interesting things I found in your statement, when you set out your professional history, was that quite early on you were involved with child health.

A: That is true.

Q: I think you were awarded a prize for your work in

relation to child health?

A: Yes.

Q: And did the work in child health lead you on to looking at aspects of food and the food chain?

A: The year's work I did in diseases of children, first at the London Hospital and then in Eastbourne, it was clearly a major problem then and still is, infections and nutrition. Those are two central issues, particularly with babies. Yes, it did leave a lasting impression. I do not think I had the right sort of personality to be a consultant paediatrician. I think I was more interested in research. So, in a way, that did influence me to move on to pathology and then microbiology. Then, because of the rise of food-related illnesses in the mid-80s, into Food Microbiology.

Q: One of the things I have been interested to explore with scientists and medics that come to talk to us is the extent to which, before the BSE epidemic, they knew about spongiform encephalopathies. Is it something that, in your training, or in your early years as a doctor, you came across?

A: We were told as a student about "slow viruses", as they were called then, which meant a curious group of diseases, presumably either directly infectious or related to an infection. Indeed, there was some...
[1] evidence that some of the complications of measles was [2] part of this group. It was strange – there was [3] contradiction, it was poorly understood, but we were not [4] taught about them. They were either called "atypical [5] viruses" or "slow viruses." I did not realise at the [6] time, talking about the 1960s, the real importance. [7] We knew about scrapie. I have been talking about [8] scrapie at the time. It seemed to me to be [9] essentially a disease of sheep, full stop. But it was [10] only when I became a professional microbiologist that [11] the details began to impinge with the knowledge of the [12] extraordinary durability of the agent that causes CJD. [13] It was then, as microbiologists, being [14] responsible, for example, for prevention of infection in [15] hospital patients, we had to have policies for [16] sterilising instruments, using instruments. Every [17] single different hospital has to have its own written [18] policies in order to avoid all different types of [19] infection. Then still we have a problem about how we go [20] about with invasive surgery or using graft organs from [21] somebody who has or might have CJD. It was then, [22] really, from the late 60s and early 70s that my [23] knowledge of the detailed information became clearer. [24] Q: Thank you, that is very helpful. The other aspect that [25] I was interested to consider briefly with you was that I [26] know you became a member of the Veterinary Products [27] Committee in 1986.

[1] You put them on certain drugs and they become more [2] vulnerable to the effects of an infection. [3] I was concerned that the use of BST on cattle, [4] that might be incubating BSE, could aggravate the [5] problem. I was also worried about the idea of making [6] holes through the skin of cattle, with the potential of [7] blood product to ooze out and therefore spread. If the [8] worst happened, there was also potential – it should [9] not happen in theory, but if, say, the same needle and [10] syringe were used on more than one animal, there was the [11] potential for BSE to be transferred in that direction.


[14] Q: Thank you. In a moment or two, we will come on to the [15] Southwood Working Party report. I think there was some [16] discussion there about what medicine and veterinary [17] products, so it may be you may want to return to that [18] theme. I am not sure. Just before we do, I wanted to know [19] turn to paragraph 5 of your statement. As I understand [20] that, the sequence of events is something like this, you [21] are working on the book, "Safe Shopping, Safe Cooking, [22] Safe Eating", which we have. We have an extract from it [23] at annex 2 to your statement. When you were working on [24] that, the material relating to BSE was really quite [25] recent, the Southwood Working Party had only reported it
BSE Inquiry

Page 11

[1] something you have written. The interval between
[2] writing it and it appearing in print, and the proofs,
[3] you start worrying about what you actually said. Also,
[4] people started asking me what did I think about BSE?
[5] There is nothing worse than seeing something in print
[6] that you actually begin to realise you have actually got
[7] it wrong. I wrote what I was led to believe. I think
[8] it was an honest mistake at the time. During 1989, I
[9] went back to the original data on which the advice was
[10] given, and the more I thought about it, the more I
[11] became concerned that we were not dealing with cattle
[13] Q: Right. Now, I think, in paragraph 6, you mention that
[14] the first document you went to look at was the article
[15] by Wells and others in the Veterinary Record in October
[16] 1987. Then you turned to the report of the Southwood
[17] Committee. Is there anything in particular you would
[18] like to take us to on the Veterinary Record article, or
[19] can I take you to the Southwood Committee?
[20] A: I would like to mention one conclusion on Wells'
[21] article, and that is that he said that this is a new
[22] disease related to -
[23] Q: We had better show it to you. Can we have the bundle
[24] J/VR. It is at tab 121 in bundle J/VR.
[25] A: What is the number?

Page 10

[1] Q: Tab 121. I think the first document behind divider 121
[4] Q: That is the article by Wells and others which appeared
[5] in the Veterinary Record in 1987. It begins by saying:
[6] "In routine diagnostic submissions to their
[7] laboratories, the authors have recently recognised an
[8] encephalopathy associated with a novel clinical syndrome
[9] in cattle from dairy herds in widely separated
[12] Q: Is that the point you draw attention to?
[13] A: It was new. It had not been described before, although
[14] even at that time there had been claims of a
[15] staggering-type illness in cattle which I had heard of.
[16] It had not been documented. This was the first
[17] documentation that I was aware of.
[18] Q: Thank you. I wanted to take you next to the Southwood
[19] report. Perhaps we could have that. It is IBD/2. In
[20] the IBD volume, it is at tab 2.
[22] Q: And this is the report that you turned to look at?
[24] Q: Now, in your statement, at paragraph 7, you note that

Page 9

[1] the bulk of the report described BSE as a "transmissible
[2] spongiform encephalopathy", and you say that would mean
[3] it is:
[4] "... a disease due to an infectious agent with the
[5] inevitability of the potential to spread to further
[6] members of the same species or indeed to other mammalian
[8] "Then you go on to say that is to be contrasted
[9] with the report's conclusions, which "considered that
[10] cattle were a 'dead-end host' for the infection". And
[11] you read that to mean that it was non-infectious.
[12] A: Well, they interpreted it in their conclusions to mean
[13] that. Could I make a general comment about this report
[14] as I saw it at the time?
[16] A: I understand that the Chief Medical Officer asked Sir
[17] Richard Southwood, Professor of Zoology at Oxford, to
[18] convene a working party. That I find one of the most
[19] strangest actions ever taken, because the terms of
[20] reference of this was to identify any possible risk to
[21] human health. I would have thought that those
[22] responsible for preventing and controlling infections in
[23] people should have been at the centre of this,
[24] particularly as it was the Chief Medical Officer who is

Page 12

[1] The strange - people who were not involved,
[2] including the Chairman - I am certain these were all
[3] people of integrity and excellence in their own field -
[4] but there was no one involved with public health, there
[5] were no medical microbiologists. There were no real
[6] people who had experience with spongiform diseases. It
[7] seemed to be very strange that the membership did not
[8] seem to be appropriate for the task in hand.
[9] Q: Thank you. I would like just to talk to you a little
[10] about this question of a "dead-end host". It is
[11] something I talked about to Professor Southwood and the
[12] other members of his committee earlier in the week.
[13] When we were talking - this is the transcript at Day 3,
[14] at page 133 - I asked Professor Southwood to explain
[15] what he meant by the expression "dead-end host". I will
[16] just read out what he replied, because I think it is
[17] quite interesting. He replied to me, at line 9, on page
[18] 133, this is Richard Southwood:
[19] "That was a term that was applied to mink, when in
[20] that particular case it passes into the female, to the
[21] adult mink, but did not appear to pass to the kits."
[22] In that context, he was talking about whether it
[23] went from one generation to the next. He went on to
[24] say:
[25] "I would agree with you; it is a slightly
Q: Thank you. That is very interesting, because Professor Southwood has written to us since he gave evidence, and he has seen what you have said in your statement. He has said to us: "Well, our report said that it was likely", that was their view, it was likely that the cattle would prove to be a dead-end host. He went on to say, "Well, if Professor Lacey had stated that he differed from us in his assessment of the likelihoods, then I (this is Professor Southwood) would regard that as a tenable position from which to continue a valid debate on such scientific evidence as was available."

A: I was not asked, and already the failure of action was committed. Now, scientists write comments knowing how politicians will respond to them, and it must have been perfectly clear that having used the word "probable", and having used the phrase "dead-end host", that it raised the possibility or probability that there was no need to take measures to control an infection in cattle. The measures which could and should have been taken, but for this statement were obvious measures associated with a serious infection in a food animal, which are proper records, quarantine of herds, cession of breeding, and planned slaughter and replacement.

Q: I would like to come on to those matters because you raise them. I think, in some comments about the Tyrrell...
BSE Inquiry

[1] quote. If I could take the last one first, the
[2] recycling of diseased animal protein. This too is
[3] something I talked to Sir Richard Southwood about. You
[4] may recall, in the report of the working party, they
[5] referred to the Royal Commission on Environmental
[6] Pollution which had discussed the recycling of waste
[8] to a question from me, that what had been said by the
[9] Royal Commission in 1979 was a warning about these
[10] dangers. Would you regard that as correct?
[12] Q: That there had been such warnings?
[14] Q: The second thing I wanted to ask you was what you said
[15] there, the possibility that it could kill 5 per cent of
[16] the population within a generation. I was wondering how
[17] you had reached that figure, 5 per cent?
[18] A: The only comparative data was from the Fore tribe in
[19] Kuru, and of course, having done an oral interview to a
[20] journalist, I cannot actually be absolutely certain
[21] exactly what I said. Usually words, sentences, get
[22] compressed, with respect. The potential for a large
[23] number of human victims comes from a study by Gajdusek
[24] and colleagues on the Fore tribe. Kuru, we knew at this
[25] time, did have some resemblances to BSE. Out of the

[1] fact, in June of 1989, so there was a long delay before
[2] it was published. I think it is right, it is not, that
[3] that report was concerned solely with what research
[4] should be done and trying to assess some priorities for
[5] it?
[6] A: That was the formal terms of reference at the beginning.
[8] A: But they were intermittently and increasingly asked to
[9] comment on events as they occurred.
[10] Q: Absolutely. If I could just pause there for a moment
[11] with you. What happened was that they asked for advice
[12] as to what they should do now that they had submitted
[13] their interim report setting out research priorities.
[14] The Government - I will put this as neutrally as I
[15] can - seem to have envisaged a further role for them,
[16] and eventually we saw them described as the "Spongiform
[17] Encephalopathy Advisory Committee". I am not quite
[18] clear when that metamorphosis came about. We will hear
[19] more about that next week.
[20] A: I would like to comment on the detail of the paragraph
[21] that includes monitoring. I can find it in my book, if
[22] you would -
[23] Q: We have it at IBD/4. So we had probably better look at
[24] it there in that bundle that you have. I think you were
[25] looking at IBD/2 just now. Is that an IBD bundle or is

[1] tribe, about 1 per cent of the population died each year
[2] from Kuru, 1 per cent each year. Of course, the people
[3] at risk, the adult women, it was nearer 2 per cent.
[4] So there is a potential for a substantial
[5] proportion of their population to go down were they
[6] exposed to the infectious agent through eating it. But
[7] I think this general article I have submitted because it
[8] shows the gradual dawning in my mind that something was
[9] wrong. I was still, I think, being persuaded that BSE
[10] was largely due to feedstuffs, and perhaps it was from
[11] sheep, and I was persuaded that there could be no danger
[12] to us. But my worries were increasing. I think this
[14] Q: Thank you. Now then, in paragraph 10 of your statement,
[15] you go on to talk about the recommendations of the
[16] Tyrrell Committee. The expression "Tyrrell Committee"
[17] is one which is a little bit ambiguous in our context
[18] because, as I understand it, one of Southwood's
[19] recommendations was that there should be an expert
[20] committee to look at research. It was Dr Tyrrell who
[21] was eventually asked to head that committee. They
[22] produced an interim report, which is the report I think
[23] that you are referring to in the first sentence of
[24] paragraph 10 of your statement, the one that was
[25] published in January 1990. It had been produced, in

[1] that the Veterinary Record? That is the Veterinary
[2] Record. Perhaps we could put that away?
[3] A: This particular paragraph goes beyond the remit.
[4] Already, in the first report, the Committee were taking
[6] Q: I think it is the bundle you have open, just underneath
[7] the article at tab 4. I think you have found the
[8] interim report. Then, at page 7, there are some
[9] research questions related to BSE.
[10] A: Yes, I think it is the conclusions is the one that this
[12] Q: Thank you. This is paragraph A2.
[14] Q: Which is headed "Human", and then goes on:
[15] "Many extensive epidemiological studies around the
[16] world have contributed to the current consensus view
[17] that scrapie is not causally linked with CJD. It is
[18] urgent that the same reassurance can be given about the
[19] lack of effect of BSE on human health. The best way of
[20] doing this is to monitor all UK cases of CJD over the
[21] next two decades. This UK cohort of CJD cases will be
[22] available for the testing of any future hypotheses. The
[23] cost is low, the priority very high."
[24] A: It is the second sentence that concerned me. Within a
[25] report assessing research priorities, it seems to me
not confirmed. It is still voluntary, and that, I think, is a very serious omission.

SIR NICHOLAS PHILLIPS: Yes, thank you.

MR WALKER: I would now like to move to paragraph 11 in your statement. This is the telephone interview, when you were asked to give an interview to a man who was rehearsing as a part-time radio presenter.

A: Yes, this is absolutely true, this.

Q: Now, can you recall who it was who asked you to do that?

A: It was one of the London radios. I thought it was probably Capital Radio. I thought I was doing him a favour by talking to him over the phone, to help the chap learn his expertise as a radio interviewer. I had heard of Andrew Neil as the editor of The Sunday Times, but I did quite see how the editor of The Sunday Times could be rehearsing as a part-time radio presenter.

Q: One of the things that you have told us you said was that the numbers of cattle confirmed as having BSE were still rising, and that that implied that BSE must be spreading between cattle. Can you tell the Committee why you thought that that implication was there?

A: The official line of ministry spokesmen was still that BSE came from sheep scrapie. Now, sheep scrapie has been endemic or enzootic for some centuries, at least two centuries in most European countries. I had no data, no reason to think - I had not seen any information that there had been an increase in sheep scrapie in recent years. It seemed to be fairly plateaued, maintained by vertical and horizontal transfer. If BSE had been coming from sheep scrapie with an incubation period of, say, five years, we are talking about sheep remains from the early to mid 1980s causing BSE.

Q: If that was occurring exclusively, then I could see no reason why the numbers of BSE cases should be rapidly rising. I still do not see any. At the time, it was the rise of BSE numbers, even before the compensation was raised in early 1990, was already heading for well above the Southwood Committee’s prediction, which was the assumption that it was from sheep scrapie. I knew that the TSFs could be spread by diverse routes. I knew it could be oral, from animal to animal, vertically and horizontally, and via the environment. I was also getting increasingly worried about the extent of involvement of other mammals in particular - this was just after the first cat had been identified with a scrapie-like condition.
Page 25

1. diagnosed as succumbing from a TSE - because there were
2. not any adequate records in most herds, because they had
3. allowed cattle movements, because they had not stopped
4. any breeding, then my estimate was that about 6 million
5. cattle, about half, that could well be exposed to BSE.
6. and no one could tell which were infected and which were
7. not, and it is still not possible to.
8. That meant that the only logical thing to do,
9. which is what has happened in other countries, and was
10. happening in other countries such as Ireland at the
11. time, the Irish authorities destroyed the whole herd if
12. they had one index case. That was already established
13. policy. Therefore, it seems an outrageous statement to
14. make when it comes as a bombshell, but I thought this
15. through, and in fact I have absolutely no regrets or
16. withdrawals of that proposal.

Q: That is the infected herd proposal?
A: Any herd that might have an infected animal should be
destroyed. It is draconian. The cost of disposal are
enormous, but at least I wanted to get the debate going
because we had not got any measures.
SIR NICHOLAS PHILLIPS: Could I just ask you about the cat,
because when you were giving evidence to the Agriculture
Committee, you commented that if cats were found to be
at risk of catching FSE from cattle, then this would
Page 26

confirm that BSE has a different infective spectrum from
scrapie, a very serious finding. When you made that
comment, was that before any case of feline
encephalopathy had been found?
A: It was after the feline case occurred in early May. I
knew of it before, but I think the media got hold of it
about a week before this time. It was very important
that I have discussed the matter with several vets who
looked at cat's brains, and they had never seen a
spongiform disease, and I believe that. You are
absolutely right, sir.
SIR NICHOLAS PHILLIPS: It was that case that sparked the
comment?
A: Yes.
MR WALKER: You mention, in paragraph 11 of your statement,
that your discussions with Andrew Neil led to a
journalist from The Sunday Times telephoning you, and on
13th May, the paper carried a front-page headline:
"Leading food scientist calls for slaughter of 6
million cows."
We have that article, and perhaps some loose
copies can be distributed.
Now, the second paragraph reads:
Professor Richard Lacey, a former Government
health adviser, said people should not eat beef until
Page 27

half the herds in Britain, each of which had at least
one infected cow, had been destroyed and beef had been
proved safe to eat again."
Does that accurately reflect what you were saying
to The Sunday Times?
A: It is precisely what I was saying, and it is virtually
what has happened, except they have not been destroyed.
Half the animals over 30 months have been destroyed, slaug
they have been rendered and are still awaiting
destruction, burning and so on. But that is right.
That was proposing six years before what they have has
been done in practice.
Q: Now, I also wanted just to take you to page 2 of this
little clip from The Sunday Times of 13th May, because
another aspect of the history which the Committee will
be concerned with is concern among Local Education
Authorities. That is described in the left-hand column
on the second page.
A: Yes.
Q: And that begins:
"The roast beef of old England will be absent from
the menu when the 1,120 pupils of Pocklington Woldgate
School in Humberside sit down to their lunches this
week.
"All meals made from British beef have been banned
Page 28

from the school..."
I was interested to see that was reported on the
same day. Was that something you had played any part
in? Had you been involved in discussions with Local
Education Authorities prior to 13th May?
A: I gather that Hull took the decision in April 1990. I
had not had any discussion with them at all. I have had
discussions with them subsequently. At that time, it
was their independent and individual response.
Q: Thank you. That is very helpful. Now, if I could then
turn on to the House of Commons Agriculture Committee,
in which you deal with in paragraphs 13 to 15 of your
statement. We have this, in that IBD bundle, the one to
your left?
A: I know it, yes.
Q: You have brought your own copy?
A: Yes.
Q: The report is what I would like to take you to first.
paragraph 33 of...
A: Let me just get this. Can you identify the page
number?
Q: It is in tab 7. I think that paragraph 33 is at page
(xiv), using the top left-hand numbering, towards the
front of the - you need to go right back to the
beginning of that section. I think we will see some
Roman numbering at the top, which is where one has
the ...

A: Yes.

Q: And that –

A: Well, not Roman.

Q: No? Keep on going then.

A: My evidence starts ...

Q: I wanted to start with what the Committee said, if I
could, if that is all right? You need to go back, right

towards the beginning of that section. They have Roman

numbering for the actual report.

A: Yes, I have that.

Q: I was interested by the first sentence that:

"Witnesses taking a more pessimistic view seem to
do so mainly on the basis that one should prepare for
the worst possible eventuality."

I want to ask you whether you regard yourself as
somebody who believes one should prepare for the worst
possible eventuality?

A: Ideally, yes. I think, as a matter of principle we need
to – if we do not prepare for what may go wrong, then
we will be taken by surprise. Certainly, as far as
infectious disease goes, we have to have strict
controls, because we know that they will not always be
adhered to. We may need to be seen to be going over the
top in order to achieve what we actually want.

Therefore, we may need to be seen to be taking
unnecessary controls to be certain that we have at least
moderate controls in place, because of the nature of
human nature, the nature of factors – and other
factors. That is the attitude that professional
microbiologists take, certainly so far as safety goes.

Q: Thank you. Now, in paragraph 14 of your statement, you
refer to what was said by the Committee in paragraphs 73
to 74.

A: What page is that?

Q: That is page (xxi). I am sure that you will remember
those paragraphs.

A: Yes.

Q: If I could just ask you to look at paragraph 14 of your
statement, where you were talking about that?

A: Yes.

Q: At paragraph 14 of your statement, you said that in
particular, your comment:

"If our worst fears are realised, we could
virtually lose a generation of people."

That comment was based on the well-documented
instances of almost 100 per cent of all mink succumbing
to spongiform encephalopathy following the eating of
contaminated feed.
PROFESSOR FERGUSON-SMITH: Can I just comment at this point? You refer to the papers by Hartsough and Burger, and we have already looked at these papers. This is on transmissible spongiform encephalopathy?

A: Yes.

PROFESSOR FERGUSON-SMITH: In that paper, they stress at great length — this was in 1965 — that the mink were fed on beef material. How did you take that into account?

A: Unfortunately, it was not properly documented. They had stated in the paper, as you actually say, that it was bovine abattoir material.

PROFESSOR FERGUSON-SMITH: And a form of cattle?

A: Yes, however there were various rumors about the types of TSE in the States at the time. However, we could not find anything actually published. The crucial thing was that we were happy that there was a TSE. It was transmissible spongiform encephalopathy.

PROFESSOR FERGUSON-SMITH: I wondered if that paper suggested that the origin of the agent might have been bovine and not sheep?

A: The dawn — as I said to counsel — it has been a slow process of the evidence building up against sheep towards cattle. You are quite right, this was a step in that direction.
BSE Inquiry

Day
March 17, 1997

[1] We were not familiar with at the time of writing the
[2] article. I take exception to many of the claims in this
[3] rebuttal. I would like to deal with the mouse
[4] experiments now, then, as you raised it, because it is
[6] Q: I think it is entirely fair that you should have the
[7] chance to deal with that. Just before you do so, I
[8] wanted you to refer to the fact that your reply to
[9] Mr. Taylor appears on the subsequent pages. Again, it is
[10] not a very clear copy. I am sorry about that. There is
[11] quite a lengthy reply which the editor published at the
[12] same time as publishing Mr. Taylor's letter. You go
[13] through the different factual matters and make comments
[14] on those.
[15] Then you go on also to make some general points,
[16] and I do not know whether you want me to read out parts
[17] of this, or whether you would be content just to deal
[18] with it orally in your evidence to the Committee now?
[19] A: I cannot actually read this, and I have not seen it for
[20] some time. I gather Dr. Dealer is going to be talking
[21] to you in future.
[22] Q: Yes, he is.
[23] A: Can I suggest a procedure? I do not know if it is
[24] possible that perhaps Dr. Dealer is given notice, reads
[25] this and then responds on our behalf?

[11] Most of the animals challenged with the BSE agent
[12] at this time, and subsequently, were a breed of mouse
[13] known as "R3", which is singularly non-vulnerable to
[14] BSE. They say that it is vulnerable, but it is only
[15] vulnerable when brain material from cattle and spinal
[16] cords with the disease were injected. And the amount of
[17] infectivity is assessed very crudely by making a slurry
[18] of brain material and then diluting it 1 in 10, 1 in 100
[19] and so on. The amount of infectivity is described as a
[20] titre, which is the lowest amount that causes the
[21] disease. With the TSEs as a whole, you get
[22] about a thousand million, 10 to the power of 9,
[23] infectious units per gram of the slurry. With these
[24] experiments, transferring BSE to mice, you typically
[25] get only about 10 to the power of 4 infectious units per
[26] gram. So only the brain, spinal cord and optic nerve
[27] have been found to have any infectivity because the
[28] animal challenged is relatively non-vulnerable.
[29] This is the main disagreement that I have had with
[30] the authorities over the claims that there is no
[31] infectivity in various cattle organs. It is because
[32] there appears to be no infectivity because the
[33] animal is insufficiently vulnerable. There
[34] is also the factor of the timing because although the
[35] brain, the clinical illness, is obviously going to have

[1] SIR NICHOLAS PHILLIPS: That is a very good idea, because I
cannot read this either.
[2] MR WALKER: That would be very helpful. I do apologise for
[3] the fact that we were not able, in the time, to get a
[4] better copy for you this morning.
[5] A: I would like to talk about the mouse experiments now.
[7] A: Unfortunately, the only certain way of identifying
infectivity with TSEs is animal challenge. Whenever any
animal challenge experiment is done, there are two main
variables, one is the inoculum and the amount in the
donor material, what you have there, and the other
variable is the vulnerability of the animal that is
being challenged. Again, the vulnerability of the
challenged animal, the main variable is the genetic
make-up and also its life span, because if we are
talking about diseases that take many years to develop,
and the animal succumbs from natural diseases before it
has had the opportunity to develop the disease, then the
result would be negative. For that reason, many of the
animal challenge experiments go for more than one
generation. A mouse is challenged, then just before it
dies, or when it dies naturally, then the material is
challenged to yet another mouse. Therefore it tends to
build up.

Page 37

Page 38

Page 39

Page 40

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[1] A lot of infectivity before that terminal illness the
[2] infectivity is found in other organs and may well
[3] decline in association with the brain infectivity. So
[4] they may be done at the wrong time. So a negative
[5] animal challenge experiment with TSE never can be used
[6] on its own to prove there is no infectivity there.
[7] Have I explained that? Because it is a very
[8] important issue.
[9] SIR NICHOLAS PHILLIPS: Yes, and I think it is not the same
[10] point, am I right, as the point made at page 263 in the
[11] right-hand column of your paper where you say:
[12] "The low sensitivity of the test technique used by
[13] some researchers may mean these are underestimate... of
[14] the infectivity of the tissues.*
[15] That is a different point?
[16] A: That is the same point. I have used rather different
[17] language, but that is the point.
[18] SIR NICHOLAS PHILLIPS: That is the point you are making.
[19] MR WALKER: Perhaps it is useful just to summarise what you
[20] covered in this paper. You had an introduction with a
[21] survey, really, and then you went on to talk about BSE
[22] clinical and statistical aspects; you talked about the
[23] history of BSE over a number of pages. Then you went
[24] on, at page 250, to talk about epidemiology of TSE and
[25] other animals. Then, on the following page, you looked

[1] extraordinarily durable the agent is. It is very
[2] important in any attempt to finally get rid of the
[4] SIR NICHOLAS PHILLIPS: Mr Walker, there are one or two
[5] questions of detail that I wanted to ask in relation to
[6] this paper, would this be a convenient moment to do so?
[8] SIR NICHOLAS PHILLIPS: Could we go, first of all, to page
[9] 259? At the bottom of the first column there you refer
[10] to the additional recommendations that were made by the
[12] BSE, questioning some of the activities in abattoirs,
[13] questioning breeding from cattle that may be infected
[14] with BSE and so on. Then at the top of the right page
[15] there is this comment:
[16] "The tight measures to control BSE are expected to
[17] revive the demand for British beef."
[18] This rather suggested to me that you and your
[19] coauthor suggested if these measures were implemented
[20] then there would be a satisfactory control regime?
[21] A: No, sir. We thought that the public would believe there
[22] was a satisfactory control regime.
[23] SIR NICHOLAS PHILLIPS: Yes, I see.
[24] A: That has been a fundamental issue, that we have measures
[25] taken to restore confidence rather than that are

[1] at the effect of post passage on the properties of the
[2] infectious agent. And you produced a table with the
[3] range of animals to which SIs from various animals can
[4] be transmitted. You went on to look at infective tissue
[5] from the TSE-infected animals, and the infected period
[6] of animals with a spongiform encephalopathy. Then you
[7] talked about the destruction of the agent. This is
[8] something you mentioned earlier in your evidence to the
[9] Committee?
[10] Q: How important was that?
[11] A: It is exceedingly important because of the physical
[12] resistance to chemicals, rays, heat. As far as I am
[13] aware, at this time, and still, there is no
[14] straightforward way of eliminating infectivity. And
[15] this is one of the factors that must require that the
[16] elimination of the agent is achieved by animal husbandry
[17] rather than relying on, for example, autoclaving.
[18] Cooking, chemicals, gamma rays. It is of fundamental
[19] importance. The physical resistance of the infectious
[20] agent was the main reason why Stanley Prusiner in 1982,
[21] following on from one or two other people, suggested
[22] that the agent was proteinaceous without any DNA or RNA.
[23] Because DNA and RNA are relatively easy to destroy with
[24] these agents. This has been known for 40 years, how

[1] radical.
[2] SIR NICHOLAS PHILLIPS: Yes, thank you. Could I go to a
[3] quite different matter at page 266? In the left-hand
[4] column you refer to a rather remarkable herd in Surrey
[5] which, in 1989, had experienced 14 cases of BSE, and
[6] then in 1990, if I understand it right, this figure rose
[7] to between 60 and 80. That is correct?
[8] A: That is the information we received from Mr Winter.
[9] SIR NICHOLAS PHILLIPS: He was the owner of the farm, was
[10] he?
[12] SIR NICHOLAS PHILLIPS: This is not any published
[13] information about – from the –
[14] A: No, that is just personal...
[16] A: At this time there had been a number of reports that we
[17] had from farms in which mothers, dams, had apparently
[18] been healthy clinically well but suddenly the whole of
[19] the offspring went down with the disease. And the
[20] interpretation, at that time, was that it was vertical,
[21] the mother was sub-clinically infected, rather like
[22] these mouse experiments you have to do over one
[23] generation, it was then coming out.
[24] SIR NICHOLAS PHILLIPS: Yes. At the time you wrote this
[25] paper not nearly as much was known about BSE as is known
(1) now?
(2) A: No.
(3) SIR NICHOLAS PHILLIPS: And you were drawing conclusions
(4) both as to maternal transmission, which others have
(5) since, or some others certainly, have confirmed to a
(6) degree, and also you were considering lateral
(7) transmission, the possibility of infectivity from
(8) pasture or contact. Having regard to the figures up to
(9) the present date that we have of the diminution of BSE,
(10) have you formed a different or a clearer view about the
(11) risk of lateral transmission?
(12) A: This is a fairly big subject. Do you want to do it
(13) now?
(14) SIR NICHOLAS PHILLIPS: If you can deal with it at least in
(15) summary now by saying "yes" or "no"?
(16) A: Right. I will say the – there is a great deal of
(17) pressure on many people for various reasons to hope and
(18) want BSE to spontaneously disappear. However, in 1990,
(19) and now we had enormous experience – by "we" I mean
(20) scientists as a whole – of sheep scrapie, which was
(21) honest unemotive work showing that sheep scrapie had
(22) been maintained in many parts of the world for
(23) centuries. They were not exposed to unnatural feed,
(24) certainly not initially; and there was evidence of
(25) vertical and horizontal transfer and contamination via

[additional text]
BSE Inquiry

[1] SIR NICHOLAS PHILLIPS: What I was not quite sure about is whether it was being suggested that because there was infection that the gene was in codon, or you happened to find the infection in cells where this particular gene was particularly busy producing this protein?

[2] A: This was a correct quote as it was at the time. I think the understanding of molecular events now is slightly different.


[4] MR WALKER: I wanted just to take you, if I might, on to your conclusion, because after having talked about chemical structure of BSE-infected agents, and then the concentration of infectivity, and the prion protein, and a number of other matters, you then have a section headed: "The risk of BSE to man", and finally a section headed: "Beef as a potential hazard to man".

[5] Looking at this, it seemed to me that what you had done was first to look at the most pessimistic view. I think you say at page 275:

"In discussing the possible effect on man the most pessimistic view has been taken so far."

[6] Now, your most pessimistic view was, and this is jogging back to page 272, right-hand column:

"There is little reason to believe that the agents responsible for TSEs are found actually within or around muscle fibres..."
[1] Promotional angle of the meat industry.

[2] Q: Thank you. Now, I then wanted to take you to paragraph
[3] 19 of your statement, where you refer to the letter to
[4] the Veterinary Record. We have that in the Veterinary
[5] Record bundle which is J/V/R. I think it is on the floor
[6] there. Yes. If that can be passed to you? Our
[8] you have told us in your statement - do we have the


[11] Q: I hope it is there at page 146, the letters page.


[13] Q: The first thing is an article, and I hope that it has
[14] the letters page. It seems to be missing from your
[15] bundle. I will give you mine. That is the letter that
[16] you wrote, and the response from Mr Taylor, is that
[17] right?

[18] A: That is correct.

[19] Q: I think you have told us that you wrote your letter
[20] because of the revelation that the age of the confirmed
[21] cases had dropped.

[22] A: It would appear that there were more three- and
[23] four-year-olds as a proportion than there had been
[24] previously. That gave me concern because, when you get
[25] an established endemic, with a disease passing from one

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Can

generation to the next, then the infectious agent
become
more virulent, and therefore the effects are more
severe. One of those could be that the animals succumb
at a younger age. At this time, I was becoming
increasingly concerned that there was vertical and
horizontal transfer, and I proposed this was happening.
Q: We also have on that page the response from Mr Taylor,
who made some points about figures.
A: Well, yes. Although the animals were from different
sources, they were not exactly the same catchment
territory. What I was pointing out was the percentage
of the ages. It did not really matter where they were
from. He rightly, of course, points out that some
England and Wales and others were from the whole of
Britain. That was not really relevant. Throughout the
dialogue I have had with the ministry vets, they have
always managed to bring in red herrings and not face up
to the real issues, and this is yet another example.
I would also like you to note that the editor of
the journal - again this seems to be standard
practice - was, at the time of publishing my letter, to
allow the apparent neutralisation of its contents by the
ministry vets.
Q: Thank you. Mr Taylor has written to the Committee. He
has said that he agrees with the facts that you set out

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This article begins:

“...A fivefold increase in the number of young cattle
dying from mad cow disease over the past two years is
fuelling concern that a three- and a half-year ban on
feeding infective offal has failed to halt the
epidemic.”

He goes on to quote Kevin Taylor:

“Kevin Taylor, the Government’s assistant chief
veterinarian, said yesterday he was still confident the
offal ban was working. ‘We expect to see the number of
cases begin to fall this year’.”

But Richard Lacey, Professor of Microbiology at
Leeds University and critic of Government policy said
The number of young animals dying should have begun to
fall, not rise, and this strongly suggests that the
offal ban has not stopped the spread of the disease.”

Does that accurately report what you were saying?
A: That is completely accurate. This is now three and a
half years after the cannibalistic feed ban. The offal
ban, the word they were using at the time, was not
strictly correct. It was the cannibalistic feed ban of
1988. With three and a half years on, certainly the
number of three year olds should have been dropping not
rising. I would also like to point out that almost
every year of the BSE issue, the veterinary officers

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[1] have always claimed that the disease is just about to go
[2] away in the next few months. Of course 1993 - we are
[4] would also like to point out that Mr Taylor, I
[5] understand, is now a consultant for the Meat and
[7] Q: Thank you. Just the passage that goes over the page in
[8] this article, there is a paragraph beginning:
[9] "It is also thought that feedstuffs ...", do you
[10] have that, just four lines from the bottom of the first
[11] page, "... contained the highest concentrations of
[12] infection just before the ban, and this could explain
[13] why so many young, vulnerable animals died.
[14] "The 15 born after the ban may also have eaten
[15] contaminated feed held in stock."
[16] Q: Have you any comment on that?
[17] A: I do not think any infectivity has been found in the
[18] feed at all; and I do not think they have any documented
[19] research linking the feed in recent years with BSE.
[20] Indeed, my last paper, I point out there is absolutely
[21] no evidence at all at this time that the feed was
[22] responsible. And -
[23] Q: Just pausing there, to make sure I have it clear in my
[24] own mind, the Southwood Working Party were telling the
[25] Committee last week of their concern that experiments to
[26] detect whether scrapie-infected feed could have caused
[27] BSE were not carried out. Is that the point you have in
[28] mind?
[29] A: No, it is a slightly - there are two points here. One
[30] is that no experiments have been done in this country,
[31] taking sheep scrapie and challenging it to calves to see
[32] if it causes BSE. That is one point. The second point
[33] is that they have never found any infectivity in the
[34] feed, full stop. What is more, the data linking the
[35] exposure to the feed in the recent years for BSE is
[36] zero. I think the feed was important, but not at this
[37] time, and to claim that they are exposed to high amounts
[38] of infectivity just before the feed ban, there is no
[39] evidence to support that.
[40] A: As I have mentioned already today, as far as I can
[41] see, the claim that it was from sheep scrapie, why on
[42] earth should it be suddenly greater just before the feed
[43] ban? It does not make sense.
[44] SIR NICHOLAS PHILLIPS: If there was a recycling element in
[45] increasing the amount of infectivity around, and that
[46] this was responsible for the increasing numbers of BSE,
[47] then you would see the increase continuing up to about
[48] this period, would you not?
[49] A: Well, that, your Honour, is a very big "if". They had
[50] not actually got any data linking the feed to the

"Would be sevenfold longer."
I think that is a typographical error.
A: That is right it should be "several".
Q: So that the sentence reads:
"This suggests that the incubation period in man,
assuming vulnerability, would be sevenfold longer, e.g.
12 to 50 years."
A: That is correct.
Q: I think we ought to see it. We have here loose copies
of both the parliamentary question and the
correspondence that it led to. If I could ask that both
of those be handed round? What you have told us in your
statement is that the parliamentary question was one
Mr Hinchcliffe asked on your behalf.
A: Correct.
Q: We have it as here from Hansard in the "Written Answers"
for 12th June. The written answer was by Mr Soames:
"I shall write to the honourable member shortly."
So the next document that we have is a letter,
which appears to be lost in our copy. We are told in
your book it was dated 25th June, so I have worked on
that basis. The reply from Nicholas Soames says:
"In your parliamentary question of 12 June, you
asked for details of BSE transmission studies which had
produced a spongiform encephalopathy in the experimental
animal.
[11] "As promised in my reply, I am writing with the
[12] information you requested which is described in the
[14] "Copies of this letter and the table are being
[16] Before we come on to the table, I would just like
[17] to deal one with one aspect of this, which is that you
[18] expressed some surprise that it was not actually
[19] published in Hansard. Mr Soames has written to us also
[20] expressing some surprise if that was the case. He has
[21] said that he will try to look into it to find out
[22] whether it was indeed published in Hansard. No doubt,
[23] if it was, he will come back and tell us. He has
[24] written to tell us that he anticipated that it would
[25] have been.
[17] A: Well, I have checked on Hansard and I could not find it.
[18] Q: No. Neither have we been able to find it.
[19] A: And I find it unbelievable that a formal written
[20] question and answer was not in Hansard. The only
[21] interpretation I have is that this was a deliberate
[22] attempt to make this information not available to other
[23] people.
[24] Q: Well -
[25] A: As you know, I have published that.

[1] Q: Indeed. You have no reason to think it is incorrect in
[2] saying that copies have been placed in the Library of
[3] the House, so that it was there in the library of the
[4] House?
[5] A: I do not know that. I have not got access. I do not
[6] think that is used very much, is it?
[8] A: I think, for people who want to know what is going on,
[9] they refer to Hansard, I think. May I suggest that
[10] Mr Soames is asked specifically to account for his
[12] Q: Thank you. Now, about the table, you have told us in
[13] your statement that of most significance for the
[14] potential threat to man were the findings in the pigs
[15] and in the marmoset monkeys. You also went on:
[16] "Also of relevance to potential human infection is
[17] the incubation period of the disease in the short-lived
[18] primate, the marmoset ...", which has an incubation
[19] period of four months.
[21] Q: Sorry, four years, 48 months.
[22] A: Yes.
[23] Q: And that leads to the last sentence, where we just
[24] corrected the typographical error.
[25] A: That is right. In general, with these infections.

[1] experimentally the incubation period is directly related
[2] to - is inversely related to the dose. Therefore, the
[3] higher the dose, the shorter the incubation period. It
[4] is also [ ] directly related to the natural life span of
[5] the species. This is both experimentally and under
[6] natural conditions. If you take - rodent incubation
[7] period can be six months to a year, mink perhaps 18
[8] months, sheep three and a half years, cattle four and a
[9] half to five years, and, obviously, man would be very
[10] much longer. And the various factors that separate the
[11] marmoset experiments and man is that the natural life
[12] span of the marmoset is about a third of us. The
[13] material was very concentrated brain material and was
[14] injected directly.
[15] That is why, on that sort of assumption, the sort
[16] of range of incubation period I have suggested is 12 to
[17] 50 years, which is compatible with all the new variant
[18] CJD cases have been over 18 or more at death.
[19] Q: Thank you. Now, in paragraph 22 of your statement, we
[21] A: May I just make one or more comment about this table?
[22] Q: Yes of course?
[23] A: It confirms that mice are not very vulnerable, so BSE
[24] has a tropism that tends to favour higher animals.
[25] Q: Yes, then come on to 1993. In paragraph 22 of your

[1] statement, you tell us that you asked Dr Tyrrell for a
[2] meeting because you were dismayed about the lack of
[3] action taken to control BSE. How did you make that
[4] request? Were you speaking to him on the telephone or
[5] did you meet him or what?
[6] A: I wrote to Dr Tyrrell at the Royal Society with various
[7] concerns about lack of action, suggesting that we met.
[8] This took about three months to set up. I did not know
[9] if he was going to bring Mr Bradley and Mr Wilesmith with
[10] him, and he did not know I was going to bring Stephen
[11] Dealer. So the five of us met at the National
[13] Q: Just pausing there. I do not think I have seen the
[14] letter that you wrote to Dr Tyrrell. Perhaps, in due
[15] course, we could ask you for a copy of that? Would that
[16] be all right?
[17] A: I will try to find it.
[18] Q: Thank you. In relation to setting up the meeting,
[19] Dr Tyrrell has told us that:
[20] "It was not easy to arrange a mutually convenient
[21] time and place, and we [ ] think that is Dr Tyrrell and
[22] others on the SEAC side thought the meeting would be
[23] more successful if it were private, small and not part
[24] of a formal SEAC meeting."
[25] Do you recollect him saying that?
A: Yes, I thought it was private and informal.
Q: Yes. In fact there was a record prepared. What
Dr Tyrrell has told us about that is:
"We told Professor Lacey there would be no formal
minute, a document that would have been available within
departments. However a record or note was prepared as
an aide memoire for the three of us, and as a report to
one or two senior MAFF staff that a meeting had
occurred."
A: That is clearly what happened, yes.
Q: Thank you. Now, I think you have expressed a wish
to take the Committee through this note of the meeting.
Perhaps we could ask people to turn to the annex to your
statement, annex 5, so that everybody has it in front of
them.
A: Could I say that I had not seen these minutes until
February 26th, following a television programme.
Q: February 26th of this year?
A: This year. And that, according to the last page, they
had been agreed between Dr Tyrrell, Mr Wilesmith and
Mr Bradley. Now, if there is a scientific meeting and
there are minutes, it is absolutely automatic that the
contributors are offered the opportunity of saying
whether or not these are accurate as far as they
recall. Occasionally, there are disputes, but if any

![Page 65](image1)

![Page 66](image2)

![Page 67](image3)

![Page 68](image4)
And the cattle bones are the main source of gelatin, yet people were still eating bone products in soups and stocks and beef on the bone. There was clearly a danger. And gelatin is a very fragile protein; it will not be destroyed by the manufacturing process. As is stated, I made this point and it was ignored. When finally when the bone ban came in December, because it was so late, it was ridiculed.

Q: We have received some comments from some of those who were present at the meeting. I would just like to read out to you something that Mr. Bradley has said:

"There were several points at issue between us, but what we considered unsound views were vigorously upheld by Professor Lacey and Dr. Dealer. I disagree that there was no scientific dialogue. I concede that the dialogue could have been better...", says Mr. Bradley. "...particularly on some points like maternal transmission, but the entrenched views held permitted little helpful discussion. Perhaps a subsequent meeting after digestion of the first would have enabled a better dialogue to develop."

Would you agree with that?

A: Yes, I would. I was very happy to talk and discuss. I think if someone’s views disagree with your views, at least what you think your views are or what your views are claimed to be, then you can accuse the other person of having entrenched views. But at this time, a number of young animals, now five years after the BSE ban was such that they had to admit vertical transfer.

Q: Thank you. He also adds:

"I disagree we were not prepared to consider their points. We were well aware of many of them from other sources, for example the validity of the mouse bio assay, but we live in the real world with sound scientific arguments for continuing to use this useful and practical model."

That was Mr. Bradley's comment on that.

A: Well, I do not think that is a reasonable response. This was raised numerous times in Parliament by David Hitchcliffe at this time about why there was a failure to use the obvious target animal, which is calves. For example, testing the safety of milk, instead of injecting a tiny drop into a mouse brain, it would have been much better to make a call from a BSE-free herd with BSE-infected milk. The question of who had the entrenched views, I put it to you that it is not me or Dr. Dealer, but that it is the Ministry vets.

Q: I ought also to tell you that we have had some comments from Mr. Wilesmith. He says that he did not know in advance of the meeting precisely what the agenda was to be or what epidemiological information he would be required to give, so he went prepared with a selection which he thought would interest you, and he provided all data and information requested directly at the time or via Dr. Tyrrell.

A: I can recall Mr. Wilesmith was exceedingly nervous at this meeting. He was shaking, he was frightened of something. When he produced some of the data, which I think the report says that I did not believe - it was the ages of the BSE cases over the last few months because the great majority were recorded precisely, yet I knew that in many farms they had not got adequate records. This is one of the recommendations I made. Indeed, the reason why only a tiny number of herds in Northern Ireland were lifted from the beef ban yesterday was because of the total failure in general of proper records.

Although I am not a trained vet, I have enough knowledge to know that it is exceedingly difficult in the abattoir to know whether a cow is three years, 11 months or four years, one month. It is because of this lack of records which makes me very doubtful about some of the data I was shown.

Q: Thank you. There is one other point made by Mr. Wilesmith that I should mention now. He says that:

"Professor Lacey and Dr. Dealer did not suggest any additional epidemiological research within my domain and responsibility. It is not correct to say that I had or have a pre-determined role, nor a closed mind."

He says that this is demonstrated by some of his publications. He goes on:

"To the best of my recollection, Professor Lacey did not make any practical suggestions as to further epidemiological research that should be undertaken at the Central Veterinary Laboratory."

A: I think the proper documentation of cattle ages, births, locations, was absolutely fundamental to everything, and I made that point very clearly.

Q: Thank you.

SIR NICHOLAS PHILLIPS: When you make that point, were you talking about cattle that show clinical signs of the disease, or all cattle?

A: We are talking about all cattle. That is the scheme that has come in, in the last year, the cattle passport scheme, which really still is not complete, but it is under operation four years too late. I thought this was absolutely fundamental and common sense to define the boundaries of the epidemic or endemic with proper records.

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MR WALKER: I wanted to ask you just a couple of things about the note. The first one I wanted to ask you about was paragraph 8 of the note. Do you have that?

A: Yes.

Q: It is recorded there that you did not support Mr Purdey's organophosphate hypothesis for BSE. Is that right you told them that you did not support that?

A: I did not and I do not.

Q: It is right that you told them?

A: I told them I did not. Do you want me to expand?

Q: It would be useful to know why, at this time, you did not support that theory.

A: The paper produced by Mark Purdey proposing the organophosphorus cause of BSE was published in the Journal of Nutritional Medicine, of which I am on the editorial board, and I refereed it. It was sent to me. Also, incidentally Dr Ebringer's paper on autoimmune cause. Both of those papers, in my capacity as referee and editor, I said know of in these young animals. The criticism of Mark Purdey's paper was that he had shown, rightly, a statistical association between BSE and the use of organophosphorus chemicals - that is right - and he had also pointed out that BSE was very rare in organic beef herds where OPs were not used. That is correct. But the interpretation that OPs caused BSE is wrong for these two reasons: the great majority of organic beef herds, the animals are slaughtered before they are aged two and a half years, there only being 80 confirmed cases in this young animals. Therefore, the age of the animal guards against BSE, not the lack of use of OPs. Of course, with the dairy herds, he had not provided any evidence looking at the association of OPs in other countries and the lack of BSE. In the dairy herds, the apparent association of OPs and BSE was a common factor, the cow. So, true, the cows were having OPs, the cows were suffering from BSE, and also did not explain the epidemiology because OPs had been used for some years. The editor overruled my objections by saying, I suppose with some reason, that everyone had the right to express a hypothesis. But I do not accept it.

Q: Thank you. Now I wanted to come on to paragraph 12 of this note. It says there: Maternal transmission was discussed, and evidence from the epidemic presented to support the MAFF position. Professor Lacey stated his belief (without evidence) that maternal and horizontal transmission was important, especially later in the epidemic. He [that is you] dismissed the evidence that placenta was not detectably infected and thought this was the origin of both the types of transmission he favoured. Could you tell us about your thinking at that time in relation to the placenta?

A: At that time, they had published all their tissue experiments which were injecting this non-vulnerable breed of mouse, reporting there was only infectivity in the brain and the spinal cord. Regardless of how the infection gets into the animal, in the first place, other organs have to be infected in order to arrive at the brain. This again gives a manifestation of the use of the wrong target animal. If they wanted to look at maternal transmission by this means, they should have taken a placenta from BSE dams and challenged it to calves which were BSE-free, to see if that went - that would be the way forward. Not taking bits of placenta and injecting into the non-vulnerable mouse. Also, they seemed unable to even consider the massive evidence that sheep scrapie had been maintained enzootically for centuries, and it has to be vertical and horizontal. The two are necessarily linked because of the access of the placenta to other members of the herd or flock. I am afraid at this point there seems to be a complete mental block on this, and there still is.

Q: Thank you. Then my next point, I think, was paragraph 16 of the note, where you are recorded as considering that specified offals from calves under six months old should be included in the ban.

A: Yes.

Q: Then that you went on to say that there was a weakness in the pathogenesis experiment. Could you explain a little bit about that to the Committee?

A: This is different words for saying that the breed of mouse used is non-vulnerable.

Q: This is the point you made earlier?

A: That is the point I made earlier. Now, interestingly, on June 30th 1994, the thymus and intestines were banned in calves under six months. Do not ask me why the rest were not. It might be rather difficult to get out, but they were not. Everything I suggested here has actually come about.

Q: Thank you. Now, paragraph 17 of the note, there is some discussion there about eyes. I do not know - can you recall what was said about eyes?

A: I think the issue at this time was that the Department of Education had instructed schools and universities not to dissect cattle eyes because of the potential danger of transmitting BSE, yet they were still being consumed. I think it was another anomaly. It is a bit like the bones and gelatin. I certainly raised eyes.
Q: The note records you saying that you would give the name and address of premises where this occurred indirectly to Dr Tyrrell. You were going to use a third party to get the name and address across. Do you remember that?
A: I cannot remember doing it.
Q: Do you remember promising it?
A: No, I cannot. But certainly within the regulations, eyes were allowed to be consumed at this time, but not dissected.

Q: Then there is some discussion about Dr Deallor's paper.
That is followed by an intriguing paragraph, which says:
"If accepted, the publication in its present form, is likely to fuel media response in an adverse way with potential effects on the beef industry. Dr Deallor asked Professor Lacey if he should tell us about 'DISCS'. Professor Lacey said 'no'."
The authors of this report say: "We do not know the significance of this."
Can you enlighten us at all, Professor Lacey?
A: No. I cannot remember. I do not know the word 'DISCS' referred to. I suspect this is a tape recording that has been incorrectly transcribed. There were certainly concerns about Stephen's publication, but

I think that sentence is very important, very important indeed.
Q: That first sentence I read out?
A: The potential effects on the beef industry. I can understand members of the Meat and Livestock Commission or farmers or those with a direct involvement with it being concerned. But these three people, Dr Tyrrell is a medical doctor, consultant virologist, Chairman of the Government Advisory Committee at the time, and two Ministry vets. I would have thought that their primary responsibility was towards public health.
Q: Now, the next thing I wanted to ask you about was the last paragraph, before the heading "Conclusions". Do you see the heading "Conclusions", at the foot of the second last page?
A: Yes.
Q: Just before that, I quote: "Professor Lacey confirmed he had removed a reference to SE in a dog from his paper, but he was not intent on changing much else."
That is the end of my quotation. Do you recall making a reference or telling them about removing a reference to SE in a dog?
A: This refers to a paper that was subsequently published, called "BSE: The Gathering Crisis", in the British Food Journal.

If they had to admit that this discussion had happened, and I had asked for various things, they would then have to respond as to why they had not taken action. Therefore, I accepted the confidentiality that I thought it was, and I was appalled that I had not received a copy of the minutes, yet all sorts of other people had.
As I actually started the meeting, not to send me minutes of it not only is extremely rude but very offensive, and also indicates serious questions about morality.
MR WALKER: In your statement then, you went on to deal with the next relevant event, which was your article in the British Food Journal, which we have here loose.
A: That is right. That is the article I removed the reference to a dog from.
Q: Just before we look at the article in your statement, I think on the third line of paragraph 23, you refer to BSE cows typically succumbing at around age six to seven years. I am not sure whether that has been typed correctly.
A: That was an error, I do not know if it was my fault or Mr McHenry's fault. It should be 4 to 5.
Q: Thank you. Now, above the title of the article, there is a sentence which I suppose described the thrust of it, would that be fair?
controls because they were thought to have BSE but were not confirmed, received the same type of meat and bone meal as those in the positive herds, yet this article claims to have shown that the BSE was caused by the feed. I am amazed how this actually was published. The procedure of refereeing on editorial policy of this journal, I think, requires scrutiny. It is this that enabled the Ministry vets to go on claim it was all due to feed, and that the regulations have been broken.

Q: Thank you. Unless there is anything else in this paragraph you wanted to refer to – SIR NICHOLAS PHILLIPS: Just on that point, in the paper you wrote with Dr Deaker in 1990, you accepted the thesis that the emergence of BSE was attributable to feed. Are you now saying that by 1993 you had changed your view, or simply focusing on the BSE cases?

A: I think all the causes that might have happened have happened. That, if BSE had been due exclusively to the feed, then the number of cases by now, by 1993, should have been plummeting very fast. In fact, 1993 was the highest number of confirmed cases. Obviously, we are looking at issues as occurred at the time. In 1990, I thought the feed was important so did Stephen. By 1993, we were thinking that these other mechanisms of transfer were at least as important. Today, I can see a succession, a combination of means, perhaps feed is a major factor, but vertical and horizontal transfer. Bear in mind that, experimentally, work done by Dr Kimberlin and co in Edinburgh has shown very clearly that it is quite difficult to infect animals experimentally, orally. It is much easier to do it through injection, and it occurs much more readily through blood, and so on. So I think you have to incriminate the feed in order to explain the big geographical area at some point, but it does not mean to say that it is all due to the feed. I think we were looking at seeing data now suggesting that these other routes were important.

MR WALKER: I would like now to take you on to paragraph 25 in your statement. There you refer to discrepancies in figures. We have an electronic printout of the article by James Erlichman in The Guardian on 2nd October 1993. In fact, it is on the earlier loose document I gave you. Do you recall we looked at the second article?

The first article on that sheet, it is a clip of two pages that looks like this. The first printout there is The Guardian, 2nd October 1993. “Mad cow figures massaged by backdating dates of deaths.” That is by James Erlichman. It begins:
(1) Government claims to have halted the 'mad cow'
(2) epidemic faced a new challenge yesterday after
(3) discrepancies were revealed in its death toll
(4) statistics.
(5) "Some 51,875 cattle died from BSE from 1988 to
(6) 1991, according to figures supplied by the Government
(7) and published in the Lancet, but in a Commons answer
(8) last November, the Government said 48,526 had died in
(9) that period."
(10) The ... there is a quote from you:
(11) "It looks suspiciously as if the Government has
(12) massaged the figures by backdating deaths to earlier
(13) years."
(14) That is the end of the quote. That is said to
(15) come from you as being the person who spotted the
(16) discrepancy. Is that right? Is that what you said?
(17) A: That is absolutely right. Could we just briefly go back
(18) to the meeting I had in June?
(19) Q: This is annex 5?
(20) A: In Warwickshire. Yes, Mr Wilesmith and Dr Tyrrell I
(21) recall telling me that the number of BSE cases was just
(22) about to go down. This was in the summer. They had not
(23) actually got the data, but they alluded to it in their
(24) report. There was an important international veterinary
(25) conference in the autumn of 1993 covering BSE, and I was

(1) amazed to find that the figures for previous years had
(2) dropped — had changed, had increased by several
(3) thousand, about 3,000. Of course, that will distort the
(4) epidemic. If you change the age allocation, the date
(5) allocation, then you will get an appearance that the
(6) disease is tending to fall rather than rise. I have
(7) raised this with Mr Wilesmith, and I have not got an
(8) adequate explanation. At one point, he claimed that
(9) these figures included the Channel Islands and the Isle
(10) of Man. They could not, there was not enough. Now the
(11) figures have subsequently reverted back to where they
(12) were.
(13) Q: Thank you. Did you have any further discussions — you
(14) have mentioned a discussion with Mr Wilesmith — with
(15) Dr Tyrrell, for example, or anybody else about these
(16) figures?
(17) A: No, I published them in my book in 1994, which was read
(18) by all those people without any comments.
(19) Q: Now, in paragraph 26 of your statement, you mentioned
(20) this question about the Channel Islands and the Isle of
(21) Man. I think you have dealt with that in the answer
(22) that you gave earlier.
(23) A: Then the next point you make in your statement,
(24) paragraph 27, deals with a further parliamentary
(25) question by Mr Hinchcliffe asking the Minister "how

(1) procedures for isolating, monitoring and reporting BSE
(2) livestock suspects born after 18th July 1988 differ from
(3) those born prior to that date."
(4) Then you refer to Mr Soames' answer.
(5) Now you say that you have quoted the answer in
(6) your book. We thought the most convenient thing was to
(7) do some photocopies of parts of your book. I hope you
(8) will not mind. I am pretty sure it is not enough to
(9) constitute a breach of copyright. That seems to be the
(10) most convenient way of dealing with it.
(11) If we could hand up some copies of that?
(12) Eventually, I should say, this will go into our
(13) materials bundle series, which is the end series, and we
(14) will notify people of the reference once we have
(15) determined it.
(16) Now you have reproduced the answer at pages 139
(17) to 140. I hope that has come through in our
(18) photocopying exercise. Mr Soames' answer was:
(19) "Suspect cases of BSE reported in cattle born
(20) after 18th July 1988 when the ban on the use of ruminant
(21) protein was introduced, as with all suspect BSE cases,
(22) are compulsorily slaughtered only when a confident
(23) clinical diagnosis of the presence of the disease can be
(24) made. Slaughter may be carried out sooner if necessary
(25) to prevent suffering; this affects compensation

(1) arrangements but not the way in which the case is
(2) handled and laboratory diagnosis carried out.
(3) "Particular care is taken in the clinical
(4) assessment of BSE suspects born after the feed ban
(5) because it was found, at an early stage, that more than
(6) 90 per cent of such cases were negative on laboratory
(7) examination, compared with a negative rate of 15 per
(8) cent for all cattle. In February 1992, Ministry field
(9) staff were therefore instructed that suspects born after
(10) 18th July 1992, the ban should not normally be slaughtered at the first
(11) visit, but that the further visit should be made at
(12) least seven days later, to reassess the progress of the
(13) case. An earlier revisits was to be made if the owner
(14) reported that the animal had deteriorated, and immediate
(15) action was to be taken to protect the welfare of the
(16) suspect animal. This observation period allowed many of
(17) the suspects to return to normal, or an alternative
(18) diagnosis to be made and treatment given by the owner's
(19) veterinarian surgeon. This change has resulted in an
(20) improvement in the negative rate in this group of
(21) animals from 90 per cent in 1991 to 28 per cent in
(22) 1993.
(23) "Animals which are suspected to have BSE do not
(24) have to be isolated ... when calving."
(25) SIR NICHOLAS PHILLIPS: "Except when calving."
MR WALKER: So sorry, "except when calving". You have told the Committee that the effect of these changes in procedure in 1992 was to distort the number of BSE cases. A: That is true, it is bound to. The first point I would like to make, this early 90 per cent negative was obviously because, soon after the feed ban there would be many young animals, and we know you get more negative in young animals. So that is to be expected. If you have any system whereby you put an extra obstruction into making a diagnosis, then you are likely to change, usually adversely, the ability to make the diagnosis. If you reduce the number of false negatives, you are likely to miss cases as well. What stimulated me to ask David Hinchcliffe to get this question asked is that a farmer approached me from near York at about this time with the history that he had had a herd with a dam dying of BSE, and subsequently two of its offspring. One had them been born a year after the feed ban. This particular case to him was clinically absolutely classical BSE. He was visited by the first Ministry vet from Leeds, who said "Yes, that is BSE", clinically put a restriction order on it, and a few days later, the second visitor, according to this protocol, and the second Ministry vet took a cursory look at the animal and said: "It is not BSE because it is born after the feed ban", and said to the farmer: "Have it slaughtered and send it into the food chain if you want to use it." The farmer was ethical and not at all happy about this. His own vet had been called in and diagnosed it suffering from a condition he called ketosis, which means nonspecific chemical change. I saw the animal. It was losing weight, its muscles were twitching, it had lost weight, an awful amount of weight, it could not stand up properly. It was in a terrible state, it is clinical BSE. I have seen several. So the farmer gives the animal to me and, Stephen has it slaughtered, and we have the head taken off and the brain preserved and sections taken. And we sent the sections to three different laboratories, including Weybridge, the Ministry's, and they all confirmed BSE. And that is why I had this question asked because I thought this was a procedure to obstruct the diagnosis of BSE, and this has proved to be so.

Q: I want to make sure I understood, when you say "Stephen", you are referring to Dr Dealer?

A: Dr Dealer, yes.

Q: You and Dr Dealer went to the trouble yourselves of arranging for the animal to be slaughtered and for the head to be preserved?

A: Yes, at this time Stephen was acting as a senior registrar in York.

Q: I think this is the same animal you are talking about in paragraph 29 of your statement.

A: That is right. It is the basis behind paragraph 27. That is why I asked the question because I thought that something funny was going on.

Q: One point to clarify with you, in paragraph 29, you say there that it was MAFF veterinary surgeons who told the farmer that the animal was suffering from ketosis.

A: Both the MAFF veterinary surgeons and his own vet said that. Ketosis is not a diagnosis, it is a manifestation of a variety of diseases.

Q: That had brought me ahead to paragraph 29. Just before leaving paragraph 27, Mr Taylor has written to the secretariat to say something about those instructions that were described in Mr Soames' answer. He said that the purpose of the instructions, this is the way of dealing with these cattle born after the ban: "Was to try to improve the accuracy of clinical diagnosis and ensure that the epidemiological information collected about each suspect was as comprehensive as possible."

A: That would be something that you would agree with,

animal and said: "It is not BSE because it is born after the feed ban", and said to the farmer: "Have it slaughtered and send it into the food chain if you want to use it." The farmer was ethical and not at all happy about this. His own vet had been called in and diagnosed it suffering from a condition he called ketosis, which means nonspecific chemical change. I saw the animal. It was losing weight, its muscles were twitching, it had lost weight, an awful amount of weight, it could not stand up properly. It was in a terrible state, it is clinical BSE. I have seen several. So the farmer gives the animal to me and, Stephen has it slaughtered, and we have the head taken off and the brain preserved and sections taken. And we sent the sections to three different laboratories, including Weybridge, the Ministry's, and they all confirmed BSE. And that is why I had this question asked because I thought this was a procedure to obstruct the diagnosis of BSE, and this has proved to be so.

Q: I want to make sure I understood, when you say "Stephen", you are referring to Dr Dealer?

A: Dr Dealer, yes.

Q: You and Dr Dealer went to the trouble yourselves of arranging for the animal to be slaughtered and the...
(1) described in paragraph 29, in a letter to the Veterinary Record. We have that at page 166 of this photocopy of extracts from your book. It starts at page 166 and goes on through to the foot of page 168. Essentially, I think the key points have been summarised in your statement.

(2) A: Yes. This is the case I referred to that I have mentioned already.

(3) Q: Then, at page 169, you set out the reply from Mr Taylor, which included this paragraph:

(4) "Finally on the question of vertical transmission, neither vertical nor horizontal transmission have been ignored during the course of investigations, and the possibility that either may occur has always been recognised despite the lack of evidence that either actually does so. While there have been 534 confirmed cases that were offspring of BSE positive dams, this in itself does not prove that the transmission was from dam to calf, since interpretation of data has been complicated by the fact that most positive offspring had also been exposed to ruminant protein. A case control study targeting herds containing confirmed cases born after October 31, 1988, is nearing completion, and should shed some light on the extent to which transmission by routes other than food may be taking place."

(5) place. The conclusions of that study will be published in due course."

(6) Q: Now, at the time, not with hindsight, what was your view on this answer that you had received?

(7) A: At the time, I did not believe they had any data to substantiate that the most positive offspring had also been exposed to ruminant protein. Where was the data? What about the data from the negative offspring? They had no data. In any case, as I have said already, they had no evidence at this time that feed – or in the previous years – that feed had been responsible. As far as I was concerned, that was fabrication. How did they know most positive offspring had also been exposed to ruminant protein?

(8) Q: You did not know?

(9) A: I did not know. I accepted that if that – it is just possible that if the ban had been broken slightly after 1988 – I cannot see how there can be "most". What is more, even if there is a small amount, I cannot quite see how this can be responsible for the illness. In any case, they had not established that the meat and bonemeal had been responsible in previous years. I took this to be a lie that they were adopting. Anything to avoid admission that there is vertical and horizontal transfer, because the implication of that was total herd

(10) slaughter. The science was being manipulated to fit, to appeal to what was politically convenient.

(11) Q: Unless you wanted to say anything more about that reply, I wanted to move on to paragraph 31 of your statement.

(12) SIR NICHOLAS PHILLIPS: Perhaps it is right to observe that, in relation to that experiment, data was subsequently published in which they accepted that the experiment indicated that there was maternal transmission, so that would seem to suggest that your worst suspicions were unfounded in that case?

(13) A: My worst.

(14) SIR NICHOLAS PHILLIPS: Your suspicions that this was all a lie?

(15) A: Well, I think that the lie is that the positive offspring have been exposed to ruminant protein. That is what I did not believe, and still do not believe.

(16) The maternal transfer was published in the summer of 1997, but its preliminary data was in Hansard in 1993, establishing very likely vertical transfer which they knew.

(17) SIR NICHOLAS PHILLIPS: Yes, yes.

(18) MR WALKER: We shall have to look in, when we come to hear evidence later on, to the extent to which there was knowledge of these things at the time. I would like to come on to paragraph 31, where you said that you had been wondering why the Ministry had not tested the hypothesis that sheep scrapie was the cause of BSE experimentally in the UK. You arranged for a parliamentary question to be asked about that, and got the reply that there were no such experiments being undertaken.

(19) Q: Then you go on to say that, yet you learnt that as late as January 1992 that experiments had been taking place on the feeding of calves with infected BSE material.

(20) A: This shows that the availability of calves for experimentation was there, which we knew anyway. The initial hypothesis, going back to 1989, that scrapie was a cause of BSE could and should have been tested experimentally by feeding or by injection. The Americans published work in 1994 showing that sheep scrapie did not cause a spongiform disease in cattle.

(21) Q: Sheep scrapie, when put into feed?

(22) A: Injected, did not cause spongiform. The Americans had shown – this was shown in 1994, published in 1994, by Coyle & Co. I viewed this at the time as a very essential omission, having a hypothesis used to reassure the world that BSE could not cause infections in people because it was caused by sheep scrapie. Not to attempt to test that experimentally is one of the most
[1] incredible lapses in modern science, I just cannot
[3] Q: You contrast that with the willingness to test the
[4] experimental feeding of calves with BSE material?
[5] A: Feeding cows to calves they had the calves available.
[6] There was absolutely no excuse at all.
[8] about that? I have the paper here from Cutler which
[9] you just referred to. Although the Americans did not
[10] find that the pattern of disease was the same as
[11] scrapie, they did find that they had a motoneuronale
[12] disorder. Can you explain just how had you interpreted
[13] that evidence?
[14] A: It was actually very interesting because it is
[15] possible – and I have had a student working on this for
[16] some time – that scrapie is actually a cause of
[17] motoneuronale disease, that is possible, or indeed
[18] multiple sclerosis. That needs to be studied. But the
[19] changes are not spongiform. And I give the Americans
[20] full marks for taking the initiative to do this. It
[21] makes it even more tragic that we did not in this
[22] country.
[23] PROFESSOR FERGUSON-SMITH: But, of course, it was a fairly
[24] limited experiment, was it not, the calves were not kept
[25] for too long?

[1] A: Yes, that is right.
[2] PROFESSOR FERGUSON-SMITH: If they had been kept, they
[3] might have shown –
[4] A: I gather that they are still doing oral challenge
[5] experiments. I do not think, even now, we have started
[6] any of these experiments in this country.
[7] MR WALKER: We will have to look into that too, perhaps.
[8] Dr Martin suggested last week that some had, but I do
[9] not have details of that.
[11] Q: You mention a number of articles in paragraph 32 of your
[12] statement. I was not proposing to ask you particular
[13] questions about the articles because you tell us in your
[14] statement something about them. I wanted to ask you
[15] though about your book, the one we see the cover of on
[16] up on our screen. It was published in November 1994.
[17] The publishers were is Cypsel.
[18] A: I am not sure how that is pronounced.
[19] Q: Is it Cypsel in the Channel Islands?
[21] Q: Can you describe to me or to the Committee what actually
[22] happened after publication. Publication, to my mind,
[23] involves sending it to book shops?
[24] SIR NICHOLAS PHILLIPS: I would be quite interested to know
[25] how you came to get those particular publishers to

[1] vets, who were highly critical of this book, basically
[2] saying I was lying; and subsequently not a single major
[3] book chain stocked it. I purchased 1,000 copies and
[4] gave them away to people I thought ought to see it.
[5] Subsequently, I was speaking to one of the authors
[6] of The Times article who told me, over the telephone,
[7] that he had not actually read the book.
[8] MR WALKER: Was it available for members of the public to
[9] buy?
[10] A: They had to write to the publishers, as they could not
[11] know who the publishers were, it was a question of
[12] writing to the Channel Islands. It was not listed in
[13] any catalogue and was virtually impossible to get. Lots
[14] of people used to phone me up and ask me for it and I
[15] used to give them a free copy.
[16] Q: I was then going to move on to paragraph 34 of your
[17] statement, which deals with the article in the Journal
[18] of Nutritional and Environmental Medicine which I
[19] should say at the moment is not in our journals
[20] bundles, but will find its way there. You have set out
[21] the six commonly presented claims; and you have told the
[22] Committee you analysed each of those claims and found
[23] them to be untenable. Is there anything that you would
[24] like to add in relation to that article? If there is I
[25] can distribute some loose copies now.
control of diseases in animals that have a primary and
adverse effect on farming and farming communities, like
foot and mouth disease, like salmonella pullorum, which
is entirely a chicken salmonella. The disease has been
taken vigorously and sensibly and radically. Diseases
which impinge onto the human population, for example
salmonella Campylobacter and BSE, the main thrust of the
control has been cosmetic to appear to be taking action,
and the main attempt has been to so-called restore
consumer confidence. Thus food poisoning in general
continues to rise. We have had no adequate resolution
of the salmonella in eggs problem. We have had no
adequate resolution of the very high numbers of
Campylobacter from affected animals. So there has been
a major difference.

If BSE had had a major impact on farming, cattle
farming, then more action would have likely been taken.
As the disease largely affects dairy cows towards the
end of their life, the potential effect on human
population has obviously not been taken as the first
priority; the first priority has been towards the
welfare of the animal husbandry.

Q: The last question I have for you is about your dietary
habits. I read with interest paragraph 5 of your
statement:

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A: There has not really been any firm data on this. It would be really nice to know exactly the answer to that question, I do not.

PROFESSOR FERGUSON-SMITH: I am going to be very interested in seeing the book that is to be published in July. I would love to hear a little bit more about the thing that springs to mind is: can you explain the sudden explosion of infection that started? We are led to believe it started mostly in Kent and south-west England. The latter was a rather rapid explosion where it appeared in numerous herds throughout the country. How does that fit with what I have not had time to think about, your recent theory, but how does that fit with that theory?

A: I think that the first point is that the actual identification of the disease encouraged other people to look for it and report it. Therefore, I think in a way, as soon as people had become aware of it in one area than other people looked for it and found it. But if you want me to try to make a unitary hypothesis to explain what was going on, I would suggest that perhaps as long as the 60s or 70s that this particular new infectious agent was entirely sub-clinical, and it was being spread around, fairly inefficiently, by the feed. But then it was being passed from one animal to another. Then suddenly in the mid 1980s, and subsequently, it was then in enough concentration for the clinical effects to be seen for the first time. There is no proof, we cannot have proof of that, but it would explain what was going on.

I would see that the role of the rendering plants and the feed as being the initial, followed by the amplification from direct animal to animal spread. I think with nearly all infectious diseases our understanding is that a truly new disease is extremely rare. HIV is the only other major infection that is truly new over the last few decades, that I can think of. That the most ready spread of any micro-organism is in one particular natural host, and then you get secondary cases subsequently in other hosts, for example salmonella, in which its prime host is the chicken. We are infected secondary. Therefore, I would have thought that when you get a huge incidence of disease like BSE, the primary host has to be cattle.

PROFESSOR FERGUSON-SMITH: You mention somewhere one of your papers that this could be linked to cases of sporadic CJD, and in fact that some of these cases may have occurred as a result of the past eating beef.

You mentioned that as a sporadic there is no evidence of a genetic thing.
[1] Know it is an infectious agent.
[2] In general, if you have an infection that is
[3] sporadic virtually, by definition, you are excluding
[4] human to human transfer. Therefore, you have to ask
[5] where it comes from. There are some environmental
[6] infections like, legionnaires' disease which you
[7] understand about water, but the great majority of
[8] sporadic infections have a point of origin. As
[9] people have excluded sheep, pretty well, as a cause of
[10] spreading CJD, then the only other common animal that is
[11] eaten on a massive scale which is likely to provide the
[12] source are cattle.
[13] Again, this is an area for more research. At
[14] least it can explain it. One of the supports for that
[15] theory is that there is a higher than expected incidence
[16] of sporadic CJD in farmers who had dairy herds
[18] PROFESSOR FERGUSON-SMITH: One of our major concerns is
[19] to
[20] consider new variant CJD. And my understanding is that
[21] a lot of evidence has been produced that the phenotype
[22] is quite different in sporadic. Do you have any comment
[23] in relation to that?
[24] A: I accept that. I accept that BSE and new variant CJD
[25] are one and the same. And that if there has been a
[26] change from sporadic to new variant CJD it is very

[1] substantial, I accept that.
[2] Could I just make one other point about the
[3] expression of CJD or other related diseases? Although
[4] we are emphasising and interested in potential numbers
[5] of new variant CJD, it does not actually establish, with
[6] certainty, that that is the only type of manifestation.
[7] although that is all that has occurred so far. There
[8] are potentials for other types of diseases to come from
[9] either cattle themselves or from other intermediaries
[10] variants. For example, if BSE did infect another
[11] species, for example sheep, then the infection we might
[12] get could well be different from new variant CJD.
[13] PROFESSOR FERGUSON-SMITH: Are you referring to the fact
[14] that BSE might pass back into sheep and come to us in
[15] that route?
[16] A: Yes. If it went to sheep, it may not come to us, we do
[17] not know. But if it did it could come to us in a form
[18] that was different to new variant CJD, we cannot
[20] SIR NICHOLAS PHILLIPS: Just on this topic, in 1990 in your
[21] paper you accepted figures that Dr. Roberts had put
[22] before the standing committee of unreported incidence of
[23] CJD, which are rather startling figures. Since then we
[24] have had the CJD surveillance unit, and one would hope
[25] that clinicians are very well aware of CJD now. Have