The BSE Inquiry / Statement No 113
Dr Harash Narang (scheduled to give evidence 13th July 1998)

THE BSE INQUIRY

Submission by Dr Harash K Narang

1. INTRODUCTION -

a) My background, qualifications and scientific views

1.1 I have the following qualifications

a. Bachelor of Science - Botany Chemistry and Zoology (Hons-Punjab University 1961)

b. Master of Science - Zoology, Rajasthan University 1963

c. Doctor of Philosophy - Zoology (Parasitology) Newcastle upon Tyne University 1969

d. Diploma in Electron microscopy 1973

e. MRC Path 1981

f. FRC Path 1991

A brief CV is attached to the end of this submission.

1.2 As a research scientist, I have devoted some 30 years of my professional career to the study of those diseases classified as Spongiform Encephalopathies ("SE's") in both animals and humans.

1.3 Since 1969, I have been actively working with these diseases having examined the first brain from a CJD victim in 1970. I gained first hand experience of these diseases occurring naturally in humans and animals and experimentally induced in many animal species.

1.4 I have published many research papers in scientific journals describing in depth these diseases and their origins, transmission, pathogenesis and also the nature of the infective agent. I set out below a list of my scientific papers relating to SE's. I have also presented numerous papers to national and international scientific meetings and have published two books referred to later in this submission. I have also written numerous scientific articles on similar and related topics which are not included in the list below.

Reference	**Date**	**Paper**	**Authors**
J/N/240/106	July 1972	Scrapie Agent and Neurones Nature 1972 24:106-107	H K Narang B Shenton P P Giorgi E J Field
J/NES/17/347	May 1972	An Electron Microscopic Study of Scrapie in the Rat: Further Observations on "Inclusion Bodies" and Virus-like Particles J.Neurol.Sci.1972.17:347 364	E J Field H K Narang
J/RUS/14/108	1973	Virus-like Particles in Natural Scrapie of the Sheep. Res.Vet.Sci.1973,14,108-110	H K Narang
J/AN/28/317	October 1973	An Electron Microscopic Study of Natural Scrapie Sheep Brain : Further Observations on Virus-like Particles and Paramyxovirus-like Tubules Acta.Neuropath.(Berl.)28,317-329(1974)	H K Narang
J/AN/28/37	November 1973	Ruthenium Red and Lanthanum Nitrate As A Possible Trace and Negative Stain for Scrapie "Particles"? Acta.Neuropath.(Berl) 28,37-43 (1974)	H K Narang
J/NEB/4/349	May 1974	An Electron Microscopic Study of the Scrapie Mouse and Rat : Further Observations on Virus Like Particles with Ruthenium Red and Lanthanum Nitrate As Possible Trace and Negative Stain. Neurobiology (1974) 4,349-363.	H K Narang
J/AN/32/163	July 1974	Virus Like Particles in Creutzfeldt Jacob	H K Narang

		Biopsy Material. Acta.Neuropath (Berl) 32,163-168 (1975)	
J/NAN/6/23	May 1979	Further Observations on Particulate Structures in Scrapie Affected Brain. Neuropathology and Applied Neurobiology 1980, 6,23-28.	H K Narang R L Chandler H S Anger
J/MD/4/64	1987	Spongiform Encephalopathies - PHLS Microbiology Digest 4(3)	H K Narang
J/PSEBM/184/375	1987	Scrapie, An Unconventional Virus: The Current Views. Proceedings of the Society for Experimental Biology and Medicine 184,375-388 (1987)	H K Narang
J/PSEBM/184/504	1987	Abnormal Tubulovesicular Particles in Brains of Hamsters with Scrapie. Proceedings of the Society for Experimental Biology and Medicine 184,504-509 (1987)	H K Narang David M Asher Kitty L Pomeroy D Carleton Gajdusek
J/VRS/9/293	October 1987	A Chronological Study of Experimental Scrapie in Mice. Virus Research 9(1988) 293-306	H K Narang
J/PNAS/84/7730	June 1987	Tubulofilaments in Negatively Stained Scrapie-Infected Brains : Relationship to Scrapie Associated Fibrils. Proc.Natl.Acad.Sci.USA.Vol 84 PP7730-7734 November 1987.	H K Narang David M Asher D Carleton Gajdusek
J/PNAS/85/3575	December 1987	Evidence that DNA is Present in Abnormal Tubulofilamentous Structures found in Scrapie. Proc.Natl.Acad.Sci.USE.Vol 85 PP3575-3579, May 1988	H K Narang David M Asher D Carleton Gajdusek
J/L/335/663	March 1990	Diagnosis of Creutzfeldt Jacob Disease by Eletronmicroscopy. The Lancet Vol 335	H K Narang R H Perry
J/MOBI/216/469	May 1990	Detection of Single Stranded DNA in Scrapie Infected Brain by Electron microscopy. J.Mol.Biol.(1990) 216, 469-473	H K Narang
J/INTV/32/185	March 1990	Evidence of ssDNA in Tubulofilamentous Particles: Their Relationship to Scrapie	H K Narang

		Associated Fibrils. Intervirology 1991:32:185-192	
J/INTV/32/316	January 1990	Increased Multimeric Mitochondrial DNA in the Brain of Scrapie Infected Hamsters Intervirology 1991:32:316-324	H K Narang Neil S Millar David M Asher D Carleton Gajdusek
J/REVI/143/381	June 1992	Relationship of Protease-Resistant Protein, Scrapie-Associated Fibrils and Tubulofilamentous Particles to the Agent of Spongiform Encephalopathies. Res.Virol.1992,143,381-386	H K Narang
J/REVI/143/387	June 1992	Scrapie Associated Tubulofilamentous Particles in Human Creutzfeldt Jacob Disease. Res.Virol.1992,143,387-395	H K Narang
J/INTV/34/105	May 1992	Scrapie Associated Tubulofilamentous Particles in Scrapie Hamsters. Inter Virology 1992:34:105-111	H K Narang
J/INTV/36/1	July 1992	Evidence that Scrapie Associated Tubulofilamentous Particles contain a single stranded DNA. Inter.Virol.1993:36:1-10	H K Narang
J/REVI/144/375	April 1993	Molecular Cloning of Single Stranded DNA Purified from Scrapie Infected Hamster Brain. Res.Virol.1993,144,375-387	H K Narang
J/PNAS/724/314	June 1994	Evidence that Homologous ssDNA is Present in Scrapie, Creutzfeldt Jacob Disease and Bovine Spongiform Encephalopathy. Slow Infections of the Central Nervous System Vol.724 of Annals of the New York Academy of Sciences June 6th 1994.	H K Narang
J/PSEBM/212/208	1996	The Nature of the Scrapie Agent: The Virus Theory. P.S.E.B.M. 1996 Vol.212.	H K Narang
J/PSEBM/211/306	1996	Origin and Implications of Bovine Spongiform Encephalopathy.	H K Narang

1.5 My hypothesis is that the infective agent is a virus with a single stranded DNA and with simple structure which is resistant to inactivation by heat and chemical procedures. This is the basis and theme of all my subsequent papers and is fundamentally different from the PrP hypothesis. However since the emergence of BSE virtually all research funds have been devoted to the PrP hypothesis and virtually no funding has been given to scientists working on a hypothesis of their own. From 1990 I was asked by my employer, PHLS to make grant applications for all of my SE related work. However, on a number of occasions after 1990 I completed grant applications which the PHLS did not countersign on one pretext or another - see for example **[M37/0.00/1.1 – 1.10], [M37/92/12.24/1.1]**. **and [M37/92/12.29/1.1 – 1.16]**

1.6 I proposed that a non-host single stranded DNA, a structural component of tubulofilamentous particles, encodes for a "chaperone" or an "accessory protein". Termed "nemo corrupta". The chaperone cleaves and binds normal host 33-35 kDa PrP to form 27-30 kDa PrP, the protease - resistant protein (PrP) (i.e. monomers into homodimers), the scrapie- associated fibrils (SAF). As the 27-30 kDa PrP molecules are added into the chain the homodimers change to homomultimers and morphological assembly of SAF takes place while the non-host ssDNA wraps around SAF, and after acquiring an outer protein coat forms the tubulofilamentous particles termed nemavirus (NVP) (Narang, 1992). With increasing incubation time, more ssDNA is synthesized, which codes for more of the peptide (chaperone) which in turn interacts with normal host 33-35 kDa PrP to form more SAF and thus disrupting the normal up keep of the cell membrane. The PrP molecules on the cell surface may act as receptor sites for the agent and absence of such sites would prevent entry of the scrapie agent into the cell, thus making the animals resistant to infection. This phenomenon is not unique to the scrapie agent. In 1995 Prusiner also acknowledged this phenomenon of post translation modification involving a chaperone in conversion of normal PrP into

abnormal PrP-sc (see my book "The Link" p.126). Prof John Collinge has used this hypothesis to suggest in a number of scientific seminars that blocking this mechanism could be used to develop treatment.

1.7 NVP were first described by me in 1972 in a publication in "Nature" **[J/N/240/106]**. NVP similar to those seen in natural and experimental scrapie of sheep and CJD have also been observed in BSE brains (Liberski 1990, Narang 1994) and have been independently confirmed by others in scrapie infected hamsters, CJD, BSE and natural infected scrapie sheep. (Liberski 1990: Liberski et.al. 1988, Gibson et.al. 1989).

b) Development of Touch Impression Technique

1.8 In the light of the above a definitive diagnosis of SE's can be made by showing the presence of NVP and SAF. A simple grid-touch negative-staining method by electron microscopy (EM) has been developed to demonstrate both NVP and SAF. Full details of the work were published in 1987 (Narang, Asher and Gajdusek, Tubulofilaments in Negatively Stained Scrapie Infected Brains: Relationship to Scrapie-associated Fibrils. Proc Natl. Acad. Sci. USA-8 4:7730-7734, 1987 **[J/PNAS/84/7730]**). Subsequently many more papers were published describing in detail the simple technique using CJD human brains. (See Papers **J/L/335/663; J/INTV/32/18; J/REVI/143/387; J/INTV/36/1; M37/93/9.14/**)

1.9 In a blind study 18 hamsters were divided into 2 groups of 9 each. Group 1 hamsters were infected with the scrapie agent and group 2 were not infected and acted as controls. One hamster from each group was killed on a rota as follows: 3, 5, 7, 10, 14, 18, 21, 24, and 28 days. Examination of grids prepared from both left and right side of the brain revealed NVP and SAF in the right side of scrapie infected hamster brains from 10 days post inoculation, and both the NVP and SAF were observed from 18 days from post inoculation from both sides of the brain. No NVP/SAF were seen in normal control hamsters.

1.10 Further work on this test was nevertheless halted by MAFF (See para 2.51) although I was and remain convinced that this simple method can provide a rapid means of diagnosis of BSE with very little handling and risk of exposure. In about 1990 when I suggested that I could collect brain materials from local abattoirs I was told by Ray Bradley that it would be illegal for me to do this and the only way of obtaining such material was through MAFF. I was therefore not able even to carry on work on this test using private funding which had been offered to me.

References

Narang, H.K. (1991). Evidence of ssDNA in tubulofilamentous particles: Their relationship to scrapie-associated fibrils. Intervirology 3 2, 185-192. **[J/INTV/32/18]**

Narang, H.K, Asher, D.M. Gajdusek, D.C. (1987). Tubulofilaments in negatively stained scrapie-infected brains: relationship to scrapie-associated fibrils. Proc. Natl. Acad. Sci. USA 84, 7730-7734. **[J/PNAS/84/7730]**

Narang, H.K, Asher, D.M. Gajdusek, D.C. (1988). Evidence that DNA is present in abnormal tubulofilamentous structures found in scrapie. Proc. Natl. Acad. Sci. USA 8 5 3575-3579. **[J/PNAS/85/3575]**

Narang, H.K, Perry, R.H. (1990). Diagnosis of Creutzfeldt-Jacob disease by electron microscopy. Lancet 3 3 5, 663-664. **[J/L/335/663]**

c) Urine Test

1.11 Mice inoculation studies have revealed that CJD blood has 1,000 units of infectivity per ml. I have developed a simple method to concentrate the agent from CJD patients' urine and I have by EM demonstrated the presence of both NVF and SAF similar to those seen in brain samples of CJD victims. I have used this CJD urine test on a number of live subjects who have subsequently died and

were confirmed to have had CJD by the Surveillance Unit in Edinburgh (For example Victims 12, 14, 15, 16,19 and 23). Shortly after I was asked by the parents of victim 12 to carry out a urine test on victim 12 the victim's relatives were contacted by Dr Duke of MRC in October 1995. Dr Duke told the relatives that the test had not been approved and suggested to the relatives that they could take a legal action against me for ethical reasons which he would support. They did not agree with this suggestion and I duly carried out the test. I used the urine test on Victim 16 (see below). His parents approached me after they had been told repeatedly by doctors that he was too young to be suffering from CJD and that his symptoms were not those of CJD. In January 1996 the urine test confirmed that he had CJD. Following this his mother insisted upon the post mortem which her doctors were advising was not necessary because they were unlikely to learn anything. Five weeks after Victim 16's death the post mortem confirmed CJD. In the light of this experience it must be expected that other young individuals who have died of CJD have not been subject to a post mortem and are therefore not included in the definite, probable or possible statistics. After I carried out the first urine test in 1995 on Victim 12 I was told by Dr Will of the Surveillance Unit that more urine specimens from CJD victims would be made available to me. Eventually in 1997 MRC funded a project in Leeds University to verify the urine test.

However many of the urine specimens which we have been receiving from the Surveillance Unit are either not fresh or have been sub-optimal in other ways (in two cases the tubes have been empty). In one instance a specimen letter was dated the 21st July 1997 although a handwritten note in the letter stated that the specimen was collected on 29th July 1997 at 17.30 hours. The post office stamp was 23rd July. The letter was delivered on the 25th July.

d) Western Blotting

1.12 I have developed an alternative method to electron microscopy, western blotting

to detect CJD in humans. The science involved in this technique is well understood. This method is more user friendly and is also cheaper. Hundreds of specimens can be tested within one working day and this process would be very useful for the purpose of testing blood donors. Since March 1997 I have been funded by MRC to carry out work using EM techniques. Initially they told me at a meeting with MAFF that they would also purchase western blotting equipment to develop the urine test. However, they are now very reluctant to allow me to develop western blotting at the same time. Mr Ken Bell agreed to purchase western blotting equipment when my request for funding to MRC, MAFF and Department of Health was not progressed. The only way to understand these problems is to visit the laboratory.

1.13 Had any of these various tests been funded or supported in any way by Government bodies it would have been possible to set up a slaughter house test for diagnosing subclinical BSE in cattle that had not yet shown the symptoms of the disease, but which were entering the national diet. In this way the disease could have been eradicated as I explained on many occasions at the time (see paras 2.12, 2.13, 2.30, 2.31, 2.35 and 2.40). The whole culling policy, which in my view is fundamentally flawed, would also have been avoided. Far from assisting and furthering my researches, however, my employers and other bodies have consistently interfered with and hindered my researches to the extent that such progress as has in fact occurred has been solely due to the generosity of a private businessman, Mr Ken Bell. I will deal in my chronology with the obstacles which have been put in the way of my researches.

1.14 Details of my researches and papers are set out at paragraph 1.14 and in my book " The Link". (ISBN-O-9530764-0-7).

e) Scrapie

1.15 There are over twenty different strains of scrapie in sheep. However, based upon

clinical signs there are two main types, which have been observed - see, for example:

Pattison IH and Millson GC - 1961

Scrapie produced experimentally in goats with special reference to the clinical syndrome. Journal of Comparative Pathology 71:2 (April). 101-108. and

Pattison IH and Millson GC – 1961 **[J/CP/71/101]**

Experimental transmission of scrapie to goats and sheep by the oral route. Journal of Comparative Pathology 71:2 (April) 171-176 **[J/CP/71/171]**

I have classified these types of scrapie as Type 1 and Type 2 ("The Link" p3)

Type 1 Scrapie

This type causes sheep to lose their wool and is the common type. When cattle are experimentally inoculated with type 1 scrapie some will develop a clinical disease. This disease is not clinically similar to BSE. However, Spongiform type changes are seen in the brain. On the other hand if cattle are fed with scrapie brain no equivalent clinical disease or lesions have been observed and the animals remain healthy.

Type 2 Scrapie

This type is different. It was the rarer form of scrapie. It shows up in sheep as trembling ataxia. Clinically it is the same in its major symptoms as BSE and Kuru. It also has the same major symptoms as "new strain" CJD.

1.16 In the UK one breed of sheep (black faced sheep) is resistant to type 1 scrapie. (Foster et al. Studies on restricted transmission of scrapie in sheep and BSE in goats using embryo transfer European Commission Agriculture Transmissible Spongiform Encephalopathies 14-15 September 1993 p229 and p 231). However, if this resistant breed of sheep is injected or fed with BSE brain tissue it will develop type 2 scrapie. (Narang - Origin and Implications of Bovine Spongiform Encephalopathy - Proceedings of the Society for Experimental Biology and Medicine - 1996 - Vol. 211 - pp.306 to 322 **[J/PSEBM/211/306]** and Fraser et

al - Transmission to mice, sheep and goats and Bioassay of Bovine Tissues - European Commission Agriculture Transmissible Spongiform Encephalopathies 14-15 September 1993 p 145 and p155). On the other hand since we know that infection with one strain of scrapie blocks other strains, (Bruce & Dickinson 1987. J Gen Vird 68,78-89) [J/GV/68/79] if a sheep infected with type 1 scrapie is injected or fed with BSE brain tissue it will not develop type 2 scrapie.

1.17 Type 2 scrapie is the disease agent which is most likely to have infected cattle and humans, but MAFF have not undertaken any experiments to determine whether or not type 2 scrapie is the real source of BSE. I consider that cattle developed BSE from eating feed containing cattle remains from cattle which had been given feed contaminated with type 2 scrapie. This may explain why USA does not have a recognised BSE problem despite having used similar rendering processes. It is probable that USA does not have type 2 scrapie in sheep, because there is no mention of it in USA literature.

1.18 Following the meat and bonemeal ban in relation to cattle in July 1988 sheep have been extensively fed with BSE contaminated meat and bonemeal. This has caused type 2 scrapie in sheep including sheep breeds which were resistant to type 1 scrapie (see Fraser et al above). Type 2 scrapie is therefore changing from being a minor strain to a major strain. I have spoken in 1995 to a farmer who had lost all of his sheep from type 2 scrapie in about 1994. He had fed his sheep with meat and bonemeal. MAFF have been aware of the fact that BSE transmits to all breeds of sheep since at least 1993 (See Transmissible Spongiform Encephalopathies - European Commission Agriculture Brussels 14-15 September 1993 p.155).

Organophosphates

1.19 There is a theory that the widespread use of organophosphates might have been responsible for BSE. This theory could have easily been tested in laboratory

animals by exposing some animals to different concentrations of organophosphates at different intervals and then injecting some with BSE but not others and then comparing these animals with animals (both BSE infected and not BSE infected) which have not been exposed to organophosphates.

1.20 BSE cases have appeared on some organic farms where the animals have not been fed with MBM including the farm owned by Jeff Nichols. This led to the belief that organophosphates might be responsible for BSE. However, I have discussed this phenomenon with three organic farmers and I visited several organic farms during 1994. I established that the cows on the farm had been exposed to and had eaten poultry manure, which is widely used on organic farms. I have personally witnessed cows eating poultry manure from a heap of manure waiting to be spread on an organic farm. It is also an established practice to add bird droppings into some cattle feed. Since MAFF allowed poultry to be fed on meat and bone meal until 1996, the poultry droppings would contain large amounts of the undigested agent.

2. HISTORY OF EVENTS

1977 to 1985

2.1 In 1977 whilst I was working with MRC, Dr Codd then Deputy Director of the Public Health Laboratory Service (PHLS) asked me to join the PHLS on a permanent staff basis. He said that I would be very useful to the service because of my general virologist background skills. An interview was arranged for me to go and see the Director of the service, Sir Robert Williams. At that interview I told Sir Robert that I would like to continue working on slow virus research and herpes encephalitis. Sir Robert told me that as long as my research did not interfere with my routine clinical diagnostic work, I had the freedom to pursue my research interests as long as they did not cause any strain on PHLS budget. As far as I am aware this freedom of research and, indeed, the encouragement of research is still an essential part of the PHLS.

2.2 I moved from MRC and started work as a Senior Microbiologist with PHLS at Newcastle General Hospital in 1977.

2.3 In 1982 I was appointed a Principal Microbiologist. I was appointed a top grade Microbiologist in 1984. I remained a top grade Microbiologist from that date to the date of my dismissal which was stated to be on the grounds of redundancy. A copy of my job specification is at **[M37/82/0.00/1.1-1.6]**

2.4 During my time with the PHLS I have pursued extensive exploratory general virological researches, in particular in the areas relating to slow viruses, herpes, encephalitis and diarrhoea. This was in addition to my researches into SE's which I always viewed as being a crucially important part of the function of the PHLS. The significance of SE's to national health is, in my view, self evident. Before BSE came to the public attention PHLS fully supported my researches in the field of SE (see for example Dr Smith's letter to Dr Bartlett of PHLS Communicable Disease Surveillance Centre dated 22nd November 1989). **[M3789/11.22/1.1]**

2.5 However, after BSE surfaced the views on SE's which I had formed as a result of my researches did not coincide with the established view. Prior to my dismissal from PHLS on the grounds of redundancy, my researches effectively ground to a halt for several years because I was the subject of disciplinary proceedings. These disciplinary procedures lasted for several years.

2.6 In the early years of my work for the PHLS my research into slow viruses was one of my major interests. I would undertake a lot of this work in my own time and I would also spend a significant part of my time at PHLS in researching slow viruses. Everyone at PHLS was aware of my activities and, until the latter years, supported my activities. I think that they were generally accepted as being important and of benefit to PHLS generally.

2.7 I was awarded a Fellowship in 1985 by the Royal College of Pathologists to work as a visiting scientist with Dr C Gajdusek in his laboratories at the National Institute of Health in USA ("NIH"). This work related to the nature of the scrapie agent. Whilst working at NIH we confirmed the first three CJD cases in young patients who were treated with human growth hormone. Use of human growth hormone was banned immediately in the USA. Dr Gajdusek wanted me to come back to work at NIH and requested in his letter to the PHLS on 15th May 1985 that I should be released to continue to do collaborative work with him. **[M37/85/5.15/1.1 - 1.2]**

2.8 Dr Gajdusek never received a reply to his letter. On my return from the USA in July 1985 I went to see Dr Whitehead. He told me that he had not responded to Dr Gajdusek because it is difficult to say "no" to such a person in writing, but the PHLS needed me in this country.

1986 to 1989

2.9 Between 1970 and 1987 I examined the brains of all CJD victims in the North East region (which included Leeds, Sheffield, Carlisle and Berwick) There were only 7 victims during this period.

In 1988 I saw a change in pattern. The number of cases increased considerably. In 1988 there were 4 victims and the number of victims continued at this level in later years, even though the geographical area which I was covering had reduced (I only covered cases North of Middlesbrough).

In addition we observed that there was a change in the distribution of lesions in the brains, in particular affecting the cerebellum. There was also a change in clinical presentation.

All of these factors concerned me considerably and I classified the cases as "atypical".

2.10 As atypical cases surfaced from 1988 onwards I started work on experiments with CJD brains using transmission studies in animals. I injected hamsters and mice with brain tissue from CJD victims with a view to studying the variations in the incubation period and lesions in different strains of mice which would reveal the source and origin of the agent (see p313 of paper 28 referred to above). I had my own animal house facilities at PHLS. By 1990 these experiments were well advanced and were giving clear preliminary indications that they were CJD cases infected with the BSE strain. In one case the animals were becoming ill and other experiments were ongoing when Dr Lightfoot of PHLS ordered me to destroy the animals. When I refused to do so he had them destroyed in 1990. He made the animal house attendant redundant and soon after he rented the animal house to MRC although it had been designed and built for my experimental requirements. Had these experiments been completed in their entirety and had the preliminary indications been confirmed we would have been in no doubt about the link between BSE and CJD and many lives could have been saved. Had we known of the link in 1990 then the passing of infected material into the food chain would have been prevented earlier, and sloppy practices in abattoirs and butchers would not have been acceptable and would not have occurred. In addition farmers and MAFF would have taken extra care to ensure that infected animals were not sold for human consumption (As it was farmers, for example Jeff Nichols in 1994 were told by MAFF that animals which were just developing clinical signs of BSE could "make it" through the abattoir). These very experiments were started by MAFF in 1996 after a discussion which took place in the British Neuropathological Society, which I refer to below.

2.11 As I mention above at this time I already had a diagnostic test for BSE which I had developed for scrapie in 1985 and 1986 and published in 1987. I was invited to AFRC and MRC Neuropathogenisis Unit, Edinburgh in September 1987 to demonstrate the test. Preparation and examination was carried out on six randomised scrapie and normal brains and correct results were recorded on all six

brains within two and a half hours from start to finish. MAFF were aware of this test because I presented the test to the 74th Meeting of the British Neuropathological Society on the 14th and 15th January 1988. **[M37/88/1.14/1.1 - 1.16]**

2.12 In August 1988, I wrote offering the test to Dr Watson at CVL pointing out to CVL that the test could be done in under a hour. I received no reply from CVL although it seems that Dr Watson wrote to me in America. By this time I was back in the U.K. and did not receive his letter. I was not contacted by MAFF for another 11 months. They then invited me to prove my claims, and I went to the CVL at Weighbridge in August 1989. When I arrived at CVL I met initially with Ray Bradley (who was MAFF BSE co-ordinator. **[M37/92/12.1/1.1]**) and Dr Watson. Then, following a view of the laboratory I presented a seminar to some 50 or 60 people. In the seminar I explained the whole background of scrapie and CJD. At that time very little was known about BSE. I showed them NVP and told them about the touch technique. I explained how the test worked and I also explained its importance in identifying animals with BSE which were not clinically ill. I stressed that the test would help to identify affected herds which could be isolated and selectively culled. Whilst I was at CVL a call came from Eire to say that Eire had identified their first case of BSE.

2.13 I suggested that since it was well established that scrapie is passed on from one generation to another, although the mechanism is unknown, the only way we could eradicate BSE would be to approach the problem in the way that the Australians had approached scrapie. I told them that in the 1950's a flock of sheep arrived in Australia from the UK and developed scrapie. The disease was new to the country. Within a year the numbers of affected animals had increased and it was realised that the disease had been imported from the UK. The Australians therefore decided to kill each and every contact and their progeny. By this method Australia successfully prevented scrapie from establishing itself in that country.

2.14 During a subsequent visit to CVL in October or November 1989 I was given three cattle brains and whilst I was there I applied the test correctly in relation to all three. I remember that when I asked if funding would be available for developing the test Dr Wells of the CVL told me "there is only one cake - if you have it there will be none left for us" so I said "the cake is big enough, I only want a little. There will be plenty left for you". (I only needed a grant of £10,000 over 2 years).

2.15 On many occasions in 1988 and subsequently I proposed at least random postmortem testing of cattle (see paragraph 1.13), but random BSE testing was then judged to be of low priority because MAFF considered that a visual examination of animals, noting the overt symptoms of clinical cases before slaughter, was adequate.

2.16 If random testing had been started when I suggested it we would have been able to discover the true extent of the disease and it could have been eradicated. In a study undertaken for "World in Action" in 1995 World in Action obtained 30 cattle heads from abattoirs in the Midlands. I tested 28 brains and established that 8 of the cattle tested which must have appeared healthy at slaughter, actually had BSE. This BSE would be detected by my test even though they were sub-clinical, symptom free cases. To date, MAFF has no such test of its own and vacuoles are not seen in sub-clinical cases until they develop the clinical symptoms.

2.17 Subsequently nothing happened in relation to my test until November 1989 when I was invited to apply for a MAFF grant to evaluate my test. On the 22nd December 1989 I made an application to MAFF for a grant of £10,000 over 2 years to fund the evaluation of the touch test. **[M37/89/12.22/1.1 - 1.5]**

1990

2.18 In January 1990 I gave a seminar to the British Neuropathological Society entitled

"High Incidence of CJD in the Northern Region" **[M37/90/1.10/1.1 - 1.7]**. Before the meeting I was required to give to PHLS an outline of what I was going to say. I was asked if there would be press present at the meeting and I was told by Dr Smith to co-operate fully with Dr Will. Dr Lightfoot suggested that I should not attend the seminar.

2.19 The seminar was attended by several hundred people, including Dr Will. I had a hostile reception when I expressed my fears that there was a link between BSE and CJD.

2.20 I told the audience that the clinical pattern of the Northern CJD cases which I had seen was very different from those we were accustomed to seeing, with significant

2.23 I had a meeting with my local Director, Dr N F Lightfoot and then with Dr J Smith, Director of PHLS, Head Office, London.

2.24 I expressed my grave concerns to Dr Lightfoot and to Dr Smith. I told them that I thought that BSE had crossed to humans and that we had to be vigilant to monitor this. I said that we should be examining normal human brains reaching post mortem and also brains from patients with atypical neurological symptoms. I said that we should also set up animal experiments to establish whether atypical cases were transmissible. I was told by Dr Smith that I should not under any circumstances talk to the press about my views on BSE/CJD. I was told that I should refer all enquiries to the PHLS press officer. He also told me that all scientific papers must be cleared by PHLS before submission.

2.25 Thereafter PHLS and Dr Lightfoot in particular tried to impose strict rules on public comment **[M37/90/2.8/1.1]**. Often handwritten statements were given to me by Dr Lightfoot to read to the press. I refused to read these because I did not agree with them. Dr Lightfoot would tell me that he would speak to the press. On 21 May 1990 Dr David Clark, raised this question during a Bovine Spongiform Encephalopathy debate **[M7 Tab 9 page 87]**

2.26 Meanwhile Dr Robert Perry of the Newcastle General Hospital, who was a colleague of mine, recorded four cases of CJD in the Northern region health area. Normally he would have expected two cases in any one year. I analysed the brains and identified that two were not typical CJD. They showed a typical SE accompanied by "focal neurofibrillar tangles formation" in the cortex and cerebellum. I classified these CJD cases as "atypical". The clinical symptoms resembled BSE. Dr Perry and I published our findings in the Lancet in March 1990, but the PHLS and the Lancet removed from the publication all references to BSE. At the conclusion of the paper we had suggested that our concern was that BSE had crossed and that atypical cases of CJD were not being identified.

We therefore proposed a large survey of brain tissues of apparent victims of other neurological diseases to establish the full extent of CJD in the population at the beginning of the BSE epidemic. This proposal was excluded from the final paper.

2.27 On the 26th January 1990, Dr Codd wrote to Dr Smith recommending that PHLS should sponsor research into CJD with particular reference to improving diagnostic methods – **[M37/90/1.26/1.1]**. He recommended me to undertake this work. Dr Codd wrote to Dr Smith that although research in this area was not "in the mainstream of PHLS interest and responsibility I have always considered this work to be worth encouraging provided that it did not interfere significantly with Dr Narang's other duties". This proposal was not taken any further.

2.28 In the early part of May 1990 I was approached by a local business man Mr Ken Bell, who owns a substantial food business. Mr Bell had read about my researches and about the fact that MAFF had refused a grant. He told me that he was prepared to provide substantial financial support to enable my researches to continue. Mr Bell confirmed that he was prepared to give £20,000 for developing the diagnostic test and for cloning and sequencing work in order to enable my work to proceed. Mr Bell was known to PHLS because PHLS were undertaking private work for him at the time. Dr Lightfoot assured Mr Bell that PHLS would assist me in every way possible in relation to SE work.

2.29 I was told at the beginning of 1990 that the reason that my grant application had been turned down was that there was limited funding. Within a matter of days the Minister announced that some £10 million additional funds were being made available for BSE research. The following day I telephoned MAFF (Mr McGovern) asking for further details of these funds but was told that the money had already been spoken for.

2.30 I explained to Mr Ray Bradley at MAFF (MAFF's BSE research co-ordinator) that

the touch test which I had developed in the USA would not only help to remove affected cattle from the human food chain, but would also show the percentage of animals affected and which farms they came from. If BSE affected farms could be identified, these animals and those affected farms could be isolated. Cattle from those farms should not be used for breeding purposes and this would help in eradicating the disease.

2.31 However MAFF did not want to know. Mr Bradley told me in January 1990 that BSE was like scrapie in sheep. He said that there was no risk to humans. It was a dead-end disease as far as cattle were concerned. He told me that my test was very sensitive and that the Minister was fully aware that affected cattle were going through the abattoirs. He told me that my test was too sensitive and that the Minister did not want my rubber stamp merely to prove that they were affected. When I suggested that we might do the study privately for our own knowledge to find out what percentage of animals were infected if any, he told me that results eventually would become public knowledge and this would cause a big headache for MAFF and the Government.

2.32 In early 1990 there was a meeting between PHLS, the Department of Health and MAFF. At this meeting as I understand matters, the PHLS were told that BSE was not a human disease and that PHLS should be therefore not be seen to be involved with BSE. (I know that there was a meeting because Dr Smith had asked me in February 1990 for SE research references so that he could prepare for the meeting).

2.33 On 27th March 1990 a meeting which I had organised in September 1989 took place at Newcastle General Hospital. The meeting took the form of an up-date course on SE including BSE and HIV related CNS infections. Dr Lightfoot wanted me to cancel this meeting. He said that unnecessary alarm would be caused if I talked about a possible link between BSE and CJD. I wrote to Dr Smith on 23rd February 1990 pointing out that the meeting had been arranged

prior to the then current media interest, but offering to cancel the meeting if this would serve a useful purpose. **[M37/90/2.23/1.1]**. However, since the meeting had already been arranged, it duly went ahead.

2.34 Over 100 people attended. I told them about the nature of the agent which causes BSE and why it had crossed over from sheep to cows. I described the varying incubation period which was decreasing from 6 to 10 years in the first passage to 4 to 5 years in the second passage and then 2 to 3 years in the third passage. The period would then stay constant if MBM continued to be fed. However, if MBM was not fed this period would increase to 4 to 5 years. I said that the ruminant feed ban was not sufficient because farmers still had access to MBM. I said that there should be a complete ban of MBM and that stocks should be removed from farmers and the farmers compensated.

2.35 I outlined my proposals for removing asymptomatic animals from the food chain by use of my test and for the introduction of a culling policy specifically targeting farms shown to be producing animals with BSE.

2.36 In my second lecture I dealt with the epidemiology of CJD and described the further atypical cases which had appeared in the Northern region and their significance in relation to BSE. I drew a contrast between the fact that people had not been affected with scrapie and the significant risk that people would be affected by BSE because it is a different strain with unique properties.

2.37 On a date which I can not identify, but which was shortly after the meeting in March 1990, Sue Lawley of BBC Radio wished to interview me in connection with the BSE situation. On the morning of transmission she spoke several times with Dr Lightfoot, who eventually agreed that I should be interviewed. Before the interview took place Dr Lightfoot met with me in the presence of Dr Codd. He said that the programme was "high powered" and that Margaret Thatcher regularly listened to it. He said that if I put "one foot wrong" she would have my

"head on a plate". He said that I should make it clear that my views were unproven. When Sue Lawley interviewed me on the telephone Dr Lightfoot listened in on the conversation. The BBC realised that this was happening and Sue Lawley invited me to the BBC studios to conduct the interview. Dr Lightfoot required Dr Codd to accompany me.

2.38 In June 1990 the House of Commons Agriculture Committee invited a range of scientific experts to present their views of the situation and how best to proceed. They sent two written invitations addressed to me through PHLS, but I did not

6. A study was needed to examine human brains from all suspect cases along with other neurological diseases using the sensitive test I had developed.

7. There were grounds for postulating that humans might possibly acquire S.E. through ingestion of contaminated tissue. It would be unwise to add potentially contaminated foods into the human food chain.

2.41 The memorandum submitted by PHLS to the Agriculture Committee on 11th June 1990 **[IBD 7 p 190]** stated that whilst PHLS were keeping a "watching brief" on the situation the PHLS were not undertaking any programmes of work in the area. The PHLS stated that my opinion on the nature of the agent and on tests for its detection were my personal views and not those of the PHLS.

2.42 None of my recommendations were followed.

2.43 Instead I attended a formal review of my work, chaired by Professor H Smith and attended by Dr J Smith, the director of PHLS, on 23rd October 1990. This meeting was also attended by Mr Bostock, a MAFF representative and Professor J Edwardson an MRC representative. I attach **[M37/90/10.23/1.1 - 1.39]** a copy of the PHLS internal minutes of this meeting which were referred to in the subsequent hearing in the Industrial Tribunal of my claim for unfair dismissal. It is apparent from these minutes that Professor H. Smith and his colleagues took a very negative attitude but could not be certain whether or not my theories were valid. Professor Edwardson said (at page 9) that Dr Perry and I would be a "recipe for mayhem" if I were to be given the go ahead to work with CJD. They therefore took the view that it was important that my test should be independently validated. As Professor H. Smith comments at the bottom of page 36 of the minute "The first thing to be established is whether Dr Narang sees things that other people do not see. If this is true, then developments here are long term". Dr Lightfoot did not want me to continue working in his laboratory.

2.44 On page 38 of this minute Dr J Smith stated that the decision whether to let me use money from support grants was "political".

2.45 Also on page 38 of this minute it is recorded that Professor H Smith said (after I had left the room) "The other side is the smear thing. Try and set up blind trial by independent people. Colindale is told and Colindale tries it out. Dr Bostock said he will raise this in Edinburgh on 24th October."

1991

2.46 Between the 22nd September 1990 and the 8th January 1991 I was given 10 brain samples by MAFF to be examined to establish whether they were contaminated with BSE. The delay was caused by MAFF's difficulties in finding ten control brains **[M37/90/11.27/1.1 - 1.3]**. The results of these tests were returned to MAFF by me on the day I received the specimens. Several months passed and the people who were interested in my researches were becoming increasingly concerned that MAFF had still not published the results of their tests on the 10 samples of brain. On the 13th March 1991, David Clark M.P. Shadow Minster for Food and Agriculture wrote to Dr Lightfoot asking why there was a delay. Shortly after this I received a report from MAFF confirming that my tests had positively identified the presence or absence of BSE in 8 out of 10 cattle brains. The test had not identified the presence of BSE in 2 of the brains **[M37/91/3.15/1.1 - 1.4]**. Although the score was not 100%, I viewed the results as encouraging. It has to be remembered that the test involved examining potentially infected material in order to identify whether or not Nemavirus/SAF were present. It is possible for the examiner to miss the presence, although the chances of this will be reduced by further refinement of the test and/or by identifying more precisely the areas of the brain to be examined. (The number and distribution of NVP vary from one area of the brain to another). I had no control over the supply of the material and only very small quantities of material were supplied. If additional samples had been provided from each brain, it is likely that

the two additional affected brains would have been positively identified.

2.47 Of great significance is the fact that none of the negative samples were identified as positive.

2.48 In addition I was extremely concerned as to the validity of the supply of material. When I received the samples from MAFF, they came with a form stating the number of the specimens and the date of the sample. The dates on these forms do not agree in some cases with the dates shown in MAFF's final report dated 15th March 1991 (see in particular samples 3 and 4) **[M37/91/3.15/1.1 - 1.4]**. These discrepancies suggested to me that there had been a definite mix-up of specimens in the MAFF laboratory. The laboratories may not have been examining the same specimens.

2.49 Whatever the position, the results of the MAFF test should have been seen as the necessary justification for more work to be done to refine the test further.

2.50 However, on 20th March 1991, I received a letter from Mr Bradley of the CVL indicating that in view of these results he considered that we should consider the study concluded. **[M37/91/3.20/1.1]**

2.51 Thereafter my work was substantially disrupted for several years as a result of 2 sets of disciplinary proceedings brought against me by PHLS which lasted for a considerable period of time.

2.52 The first disciplinary proceedings commenced with a letter from Dr Mary Cooke dated 12th June 1991 and related to allegations that I had been involved in breaches of safety rules and regulations arising out of the service of prohibition notices by the Health and Safety Executive on 25th April 1991. I have always disputed these allegations and was supported by Dr Codd with whom I had shared

suspended pending disciplinary interview and my experimental work was stopped for "reasons of safety" (although the HSE subsequently confirmed that my work required no notification to HSE – **[M37/94/1.26/1.1 - 1.2]**)

2.53 This treatment of me by the PHLS is in stark contrast with the reaction of other reputable bodies to the service of such prohibition notices. As the Guardian reported on 16th August 1991, at that time similar notices were also served on the Department of Clinical Veterinary Medicine at Cambridge University and on two laboratories at St Bartholomew's Hospital in London. The other institutions recommenced their activities after compliance with the HSE notices. The PHLS, however, terminated my experiments and destroyed all my materials. **[M37/91/8.16/1.1]**

2.54 The internal disciplinary proceedings took a considerable time to resolve and in the meantime I had to undertaken most of my work abroad. I received a formal written warning on 4th November 1991. During the course of the disciplinary proceedings my Counsel requested access to my perishable clone material so that I could take it to the USA to complete my work. This request was refused and Dr Lightfoot incinerated those valuable materials. Had I been allowed to complete those experiments they might have established the link between BSE and CJD at this early stage of the epidemic.

1992

2.55 Following the termination of the first disciplinary proceedings I continued with my work at the PHLS although at this time Dr Lightfoot introduced a rule preventing work from being carried out outside the hours of 9 to 5, Monday to Friday, without his special permission. Most of my SE work has always been undertaken outside these hours. I also continued with my own researches in relation to BSE related diseases. For a long time I have followed up cases of deaths which appeared to be related to CJD in order to establish a factual background to give perspective to my researches. This work did not involve any laboratory work or

infectious materials. I continued with this work in my spare time whilst undertaking routine laboratory work at PHLS. I was invited to meet with many farmers and families of CJD victims most of whom were desperate for advice and information when for the most part their medical advisers were not in a position to identify the illness. Many families wrote to me and I contacted others through medical colleagues. A summary of many of my factual researches appear later in this paper and details are contained in my book "Death on the Menu". (ISBN-0-97809530764-1-5). During this period publication of two of my papers on SE was delayed by PHLS) **M37/92/6.23/1.1; M37/92/6.29/1.1; M37/92/8.3/1.1; M37/92/8.15/1.1; M37/92/8.17/1.1; M37/92/8.18/1.1; M37/92/8.21/1.1**

2.56 However on 29th July 1992 I received a letter from the PHLS HQ Secretary to the Board, Mr Saunders, complaining about the methods of my investigations into the family background of individuals who had recently died of CJD. In the light of the allegations contained in that letter I was suspended from the PHLS pending an investigation. I was instructed not to engage in any work, including any individual or collaborative work in relation to SE or CJD. In order to continue my researches therefore I had to work in the USA in my own time, funded by Mr Bell. PHLS did not allow me to do this in PHLS time, although I was suspended.

2.57 During the course of the second disciplinary proceedings a number of highly important matters relating to BSE and CJD came to the attention of PHLS and, although they were matters of major scientific and public health concern, they were either overlooked or ignored and were not relayed to SEAC or to anybody else. I was told that I must not publish any of the information.

2.58 For example the PHLS has disclosed to me the notes of an interview between Dr Perry, consultant neuropathologist at the Institute of Pathology, Newcastle General Hospital and Dr N Lightfoot which took place on 1st October 1992 **[M37/92/10.1/1.1]**. Dr Lightfoot received the following information:-

" Dr Perry freely reported to me that there had been four cases of CJD in the Northern Region this year (1992) and that he would only expect one or two cases per year. He added that interestingly in two of these cases the histological appearance of the lesions was unusual in that they involved the cerebellum and this was the appearance that is seen in BSE in cows".

2.59

post mortem would have been important because there is no recorded husband and wife case of CJD.

v. In the light of the above it was therefore essential that epidemiological studies should take into account the secondary occupations of victims.

2.61 The disciplinary proceedings lasted for an inordinate length of time. I was not able to work during this period. On 3rd October 1992 I wrote to Dr Cooke because I was concerned that my continuing suspension had completely brought my research activities to an end. I pointed out to Dr Cooke that there were facilities available at Newcastle and funds available from Mr Bell and also that if it was not possible for me to work at Newcastle I was quite willing to work elsewhere and I had offers from Professor John Oxford at the London Hospital and from Dr Henry Wisniewski who were interested in my researches. In particular Professor Oxford was happy for me to work at the London Hospital and Ken Bell had confirmed that he would be prepared to meet the running costs of any experiments and paid £7,000. I never received a substantive response to this letter.

2.62 A disciplinary interview took place on 22nd December 1992.

1993 - 1994

2.63 Following the disciplinary hearing which was abandoned part heard after two hours and never reconvened, very little happened in the disciplinary proceedings apart from some exchanges of correspondence between my solicitors and the PHLS. Finally I received a "final written warning" under cover of a letter dated 13th August 1993. The PHLS proposed that I should be released from PHLS duties and that I should work in London with Professor Oxford with a view to validating my work on ssDNA under a joint grant from MAFF and AFRC. This grant was awarded following pressure on my behalf by Dale Campbell Savours MP. In the meantime I appealed against the final written warning.

2.64 On 14th September 1993 I attended a seminar of experts arranged by the

European Commission in Brussels. At that seminar J W Wilesmith from MAFF **[M9 Tab 27]**. reported that over 3,000 calves BAB from BSE affected animals had died from BSE in the UK and estimated that some 50,000 would die in the future. However, it was not until some 3 years later that MAFF accepted that vertical transmission takes place. His explanation that the deaths were the result of continued use of MBM were ludicrous in light of the numbers involved.

2.65 In the same seminar

A. J A Costelloe reported that Eire had controlled BSE by culling all herds in which a single case of BSE had occurred.

B. M Dawson et al reported that chickens exposed to the BSE agent developed a neurological disease with atypical pathology.

C. M Jeffrey et al **[J/MN/8/105]** reported that histological confirmation of BSE in clinically diagnosed cases was decreasing year by year. He had no explanation for this phenomenon. When I questioned him he was not willing to investigate this issue any further.

2.66 My appeal had not been dealt with by the 4th November 1994 when the PHLS wrote to me to say that my contract of employment was going to be terminated on the grounds of redundancy with effect from 11th November 1994. Since my appeal against the disciplinary proceedings was by now academic I withdrew the appeal and challenged my dismissal on the grounds of redundancy in the Industrial Tribunal.

2.67 Shortly prior to my dismissal on the grounds of redundancy my solicitors had written to the PHLS solicitors on my instructions on 12th October 1994. They asked the PHLS to clarify the policy position of PHLS in relation to identifying the causes of CJD and in relation to the public health implications of the consumption by humans of meat from animals which may have suffered from BSE. My solicitors asked the PHLS to confirm whether the policy position of PHLS was

that such research is of low grade importance. Following the notice confirming my dismissal on the grounds of redundancy this request for clarification of the PHLS position was repeated but was not answered.

1995

2.68 On 5th January 1995 I presented a paper entitled "An Inquiry into Possible Modes of Transmission of Creutzfeldt Jacob Disease Virus" to the British Neuropathological Society **[M37/95/1.5/1.1]**. In this lecture I reported that epidemiological studies of CJD had revealed three types of CJD cases - sporadic, familial and iatrogenic. I outlined the clinical and pathological differences in these groups and explained that a new atypical group of cases has arisen among the sporadic cases. I explained that there were three conjectural modes of transmission namely zoonotic transmission of scrapie or bovine spongiform encephalopathy, activation of a latent endogenous virus infection or person to person transmission. My conclusion was that conventional vertical or horizontal transmission was the more likely route of the origin of the agent of CJD.

2.69 On 31st March 1995 I attended and spoke at a seminar arranged by the Public Health Trust entitled "This BSE Business" **[M9 Tab 23]**. This seminar was attended by, amongst others, Ray Bradley, Jeff Almond and Keith Meldrum. During the course of my talk I outlined the potential problems in the use of meat and bonemeal for feeding to poultry, pigs and fish and I warned at least pigs and probably the other animals would develop the disease. I asked the chairman, Jeff Almond, why bones which were rejected by the E.C. as being dangerous were being incorporated in MBM in this country. Ray Bradley confirmed that the bones were being used in MBM which was being fed to pigs, poultry and fish but refused to answer my other question. In this same meeting Keith Meldrum was asked if he could action the location of burial sites of BSE cattle, but he refused to do so.

2.70 At about this time at a meeting with Mr Amman a farmer in Kent I was informed when discussing how BSE had affected his herd that he had regularly found large

bones buried in feed grain supplied to him. On enquiry he discovered that the same container lorries were being used to transport both feed grain and bones. He also told me that he frequently identified progeny of BSE cattle to MAFF vets, but MAFF were not prepared to accept his evidence of vertical transmission. He is very critical of his dealings with MAFF and their reluctance on one occasion to accept that one of his cows had BSE.

2.71 The hearing of my claim for unfair dismissal took place in the Newcastle upon Tyne Industrial Tribunal on 6 days during the course of June, July and August 1995. The following matters became known to me during the course of the Tribunal hearing and are duly recorded in the Tribunal's judgment.

a. The Department of Health had decided to reduce the central funding of PHLS for the financial years 1994/95 and the two subsequent years and as a result the PHLS had to look for 2% savings in cost on a yearly basis commencing on the financial year of April 1994.

b.

[M37/94/3.10/1.1]; [M37/94/3.24/1.1 - 1.2]; [M37/94/4.5/1.1]; [M37/94/4.11/1.1]; [M37/94/4.22/1.1]; [M37/94/8.2/1.1]; [M37/94/8.18/1.1 - 1.2]; [M37/94/9.22/1.1]; [M37/94/9.00/1.1 - 1.13].

2.72 The confusion over the PHLS' position in relation to its involvement in BSE research became even more profound when on 27th March 1996 Steven Dorrel said in evidence to a Parliamentary Select Committee "in the case of Dr Narang, ... his employment with the PHLS has been brought to an end. The reason why his employment with the PHLS has been brought to an end has been stated many times in the media. That is because he was conducting a private research effort and not conducting himself as an employee of the PHLS in accordance with his contract of employment".

2.73 After giving this evidence to the Committee Mr Dorrel subsequently corrected his evidence and the following note was added to the Parliamentary transcript "note by witness: in fact, contrary to reports in the media, Dr Narang was made redundant. In addition he was conducting a private research effort and not conducting himself as an employee of the PHLS in accordance with his contract of employment". **[M37/96/3.26/1.1]**

1996

2.74 On 12th January 1996 I presented a paper entitled "Genetic Variation of the Host PrP Gene is not a Factor in the Occurrence of Bovine Spongiform Encephalopathy" to the British Neuropathological Society **[M37/96/1.12/1.1 - 1.28]**. I explained that genetic variations seen in different humans do not mean that some will have some natural protection and will not get infected. No incidence difference was found among bovine spongiform encephalopathy affected and healthy cattle between the two groups in frequencies of these variant PrP alleles. Transmission of BSE experimentally to mice and to sheep both to "positive" (susceptible) and "negative" (resistant) lines strongly suggest that BSE is not preferentially influenced by certain PrP genotypes of host. These

experiments demonstrate that cattle have selected a major new strain of the scrapie agent which appears to be more virulent compared to an unselected strain found in scrapie sheep. Transmission experiments have implicated the same strain of BSE agent in the occurrence of SE's in domestic cats, tiger and goat and pigs infected with BSE. I presented evidence that there was a link between BSE and CJD and this could have easily been checked by injecting different breeds of mice. (As I mention above I started similar transmission experiments in 1988 which were destroyed by Dr Lightfoot of PHLS. No experiments have therefore been undertaken using human brains to demonstrate the direct link of BSE with CJD). In my lecture I asked why this should be the case. The only reasons which occurred to me were that we do not want to know the results or have been told not to carry out the experiments or consider humans to be less important than animals. I received no substantive answer.

2.75 During the meeting Dr James Ironside during discussion said that he did not think that BSE had any link with the new CJD cases. He said that it was too difficult to differentiate because now everything was mixed up. I told him that I still have some old brain tissue from the USA which can be used for comparison against new cases in the UK. **[M37/96/1.12/1.1 - 1.28]**

2.76 It was not until March 1996 that Mr Steven Dorrel announced in the House of Commons that a "new strain" of atypical CJD had been identified. To me this was not a new strain. It had been there all of the time.

2.77 In March 1996 I gave evidence to the Agricultural & Health Committee and also wrote an article for the Parliamentary review in which I outlined the way forward. Again I identified the urgent need for a quick, cheap and reliable test to diagnose the presence of BSE and CJD. **[M37/96/5.00/1.1 - 1.2]**

2.78 Only now have the experiments I was trying to do back in 1990 been re-started, using brain material from some of the recently announced "new strain" CJD cases.

Samples from human cases of new strain CJD have been inoculated into mice. These experiments will provide the first direct proof of a link between BSE and CJD. SEAC has stated that this is the most important experiment ever to be carried out on BSE and CJD. If this is the case why was the experiment started so late? It did not start until April 1996. Yet Steven Churchill died almost a year earlier. I had diagnosed in his case an "atypical strain of CJD". Why was the test not initiated then? If it had been the results would have been available a year earlier. Perhaps more to the point I was doing similar experiments almost 6 years earlier back in 1988. As I explained above although initial signs from one human case clearly demonstrated a link Dr Lightfoot the director of PHLS in Newcastle ordered that the animals should be destroyed before the results came in from other CJD cases. (see para 2.10)

2.79 There are many other people who have died of the new atypical strain of CJD but were never neuropathologically examined for this purpose. For example in September 1996 Victim 19 (see case studies), aged 59, died after having been ill for 18 months. Around 1991 she had had a blood transfusion following an ulcerated gullet. The brain pathology was similar to that seen in the "new variant" CJD and identical to that observed in CJD patients who had died after receiving growth hormone.

2.80 The validity of my test still remains at issue. I was told that I would be given a chance to have the test validated. To that end I approached MAFF in March of 1996. Unfortunately, in the time since then validation of the test still has not been completed. However, if either the touch technique or urine test had been properly validated at the time when they were developed BSE could have been eradicated, as I explain in paragraph 1.13. There is no reason why the two tests, and also the western blotting test, could not be validated swiftly and cheaply if I were to be provided with the necessary infected and control specimens and proper laboratory facilities. To date this has never happened.

2.81 I understand that MAFF gave to Electorphoretics Plc, a company run by Sir Michael Grylls an exclusive right to develop a live test for BSE in 1995. I do not consider that it is in the public interest for research on a matter of such public concern to be restricted in this way.

2.82 In all this time, in contrast as I have explained above, I have had to beg and borrow laboratory facilities, often having to use "the back door" and to presume on professional acquaintances and on the generosity of private supporters.

2.83 In my view the above history demonstrates that the handling of the BSE crisis was influenced by the preferences and prejudices of civil servants whose main concern was to protect the well being of the farming industry rather than to tackle the problem on a scientific basis and to protect their own position. From the start of the crisis the situation was approached not as a matter of science but as a matter of policy with Government bodies viewing research as some kind of convenience store where the hypotheses that suited were selected. As a result many obvious experiments have never been done simply because the results may have been inconvenient. (see paragraph 4).

3. SUMMARY OF INDIVIDUAL CASE STUDIES

3.1 The document "BSE Timeline" records Stephen Churchill's death on 21 May 1995. He is described as the "first known victim of nvCJD". This is not correct. The work which lead to my victimisation by the PHLS related to case studies of individual CJD victims and their families. I set out below a summary of the main findings of some of these case studies, full details of which are included in my book "Death on the Menu" but which have been ignored by all Government agencies.

3.2 All of the people listed below demonstrated the clinical signs of atypical CJD. The main difference between the old classic CJD and "new" strain CJD is that the classic disease starts with dementia while the new disease starts with balancing

problems and difficulties in walking. Most of the known cases of this new form follow a set pattern; initial psychological problems and depression, followed by physical instability, ataxia, coma and death. Brain pathology shows a gross vacuolation of the cerebellum.

3.3 Classical CJD predominantly affected the middle aged and elderly with only a few exceptions, chiefly the so called iatrogenic cases where infection is transmitted accidentally during surgical procedures. Although the relatives of all of the people mentioned below have given me permission to publish the details of my researches together with names, in the hope that such publication might lead to greater scientific and public awareness of the currently unrecognised widespread nature of this dreadful disease, in this submission I will refer to the victims by reference numbers. Full details can be made available if necessary and are already in the public domain.

I have identified with an asterisk those victims who I believe are not included in official figures of nvCJD.

Victim 1 *	Victim 1 died in 1984 after an illness of 3 months. Her first symptoms were that she became somewhat forgetful and then began to stumble and have trouble walking. Initially diagnosed by her GP as having had a nervous breakdown, she was later referred to hospital as her condition worsened. There she was finally diagnosed as suffering from CJD. Her Clinical symptoms were consistent with new strain CJD. Victim 1's family recalls that before her death a man came from a hospital and took a full history of Victim 1. He was very interested in the fact that Victim 1 had at one time lived on a farm in Canada. After that he wanted to know if she had ever been to Papua New Guinea and explained that his question related to the disease Kuru. He was also interested in the fact that she had lived on a farm and wanted to know whether she had been in contact with sheep. Her favourite meal was calves brains on toast.
Victim 2 *	Victim 2 died in 1989. The first sign of trouble was when Victim 2 complained about problems with his eyes. He became frightened and the fear grew so intense that he could not put one foot in front of the other. He

	forgot who his relatives were. Victim 2 was in his 50's when he died. There was no post mortem but when Victim 2 was in hospital a brain biopsy was done. Using my touch technique I confirmed that he had CJD. His clinical symptoms were consistent with new strain CJD.

Victim 3
*

*	looked after her during her 6 months illness told me about their frustrations during the course of the investigation of her illness. Victim 7 herself wrote in her diary "they are going to test me for mad cow disease". Her clinical symptoms were consistent with new strain CJD.
Victim 8 *	Victim 8 died suffering from CJD in 1994 in her 40's. She started experiencing "funny feelings" and while walking would bump into people. She had balancing problems and developed jerky movements in her legs. Her clinical symptoms were consistent with new strain CJD.
Victim 9	In his late teens Victim 9 died from new variant CJD in 1995. The Lancet Medical Journal published his case in October 1995 and this immediately rang alarm bells. Sporadic CJD usually strikes people far older and Victim 9 had had no links with known sources of the disease, such as infected growth hormone. Victim 9 had clinical symptoms consistent with new strain CJD. I talked to Victim 9's family before his death. They had great concern about the way their son was being treated and insisted that I should be present at their his post mortem. A day after the post mortem I confirmed to Victim 9's parents that his death was due to CJD. Victim 9 had originally been seen by psychotherapists and psychiatrists and was originally diagnosed as suffering from clinical depression. They were then told by a neurologist that Victim 9 was suffering from a progressive degenerative disease of the brain. He was then subjected to multiple tests at a special unit in London. Eventually his parents were told that he might have CJD. They were asked if Victim 9 had had any blood transfusions or growth hormone treatment.
Victim 10 *	Victim 10 was in his 50's when he died in 1995 after being ill for three months. Following an unrelated eye accident his partner became aware that his behaviour was changing and within weeks she noticed that he was having difficulty holding a cigarette to his mouth because of a tremble in his hand. His driving became erratic and he was never able to return to work. His clinical symptoms were consistent with new strain CJD but no post mortem was performed. Victim 10 was seen by a doctor from Edinburgh who took a blood sample. He told the family that Victim 10 had CJD. He told the family that this was "the same as mad cow disease".
Victim 11	Victim 11 died of CJD in her 20's in 1995. Her partner told me of the enormous problems he had in obtaining a diagnosis of CJD. Victim 11's clinical symptoms were wholly consistent with new strain CJD. Her partner was told by Dr Zeidler from the CJD unit that Victim 11 had CJD. He told him that there was no connection with beef. He said that CJD had been around for centuries.

	Her partner complained to me that Victim 11 was included in the 10 cases of new strain CJD referred to in the announcement in the House of Commons in March 1996. He told me that Martin Zeidler telephoned at 8.30pm on that day and he was very upset that Mr Zeidler had not told him previously that Victim 11 was suffering from the new strain CJD. He put to Zeidler that Zeidler "must have known that Victim 11 was one of the 10" and Zeidler answered that he did. He feels that he was grossly misled.
Victim 12 *	Victim 12 died in 1995 from CJD aged in her 30's. Victim 12 was diagnosed, while still alive, as having CJD after her family asked me to conduct my urine test. She died shortly after I confirmed that she had CJD. One of Victim 12's family wrote a series of letter to Government Ministers, including the Prime Minister John Major and the opposition leader, campaigning for recognition of my urine test for CJD. Her clinical symptoms were consistent with new strain CJD. The surveillance unit identified a PrP mutation.
Victim 13	Victim 13 died aged in his 30's in 1995 from new strain CJD. He had wasted away for 8 months prior to his death. He had been a fit man who rarely needed to see the doctor. He would cycle as well as being a keen basketball player. An inquest found that Victim 13 died of new variant CJD.
Victim 14 *	Victim 14 who had also worked in an occupation that involved the handling of meat for a number of years, died from CJD in his 50's in 1996. As a hobby he often brought home beasts' heads, removed the horns and carved them. Victim 14's partner contacted me and asked me to test Victim 14 using the urine test for CJD. The test was performed a month before his death and was found to be positive for CJD. His clinical symptoms were consistent with new strain CJD. The post mortem confirmed CJD.
Victim 15	Victim 15 died in 1996 in her 30's. The cause of her death, established by post mortem, was bronchial pneumonia brought on by new variant CJD. The first signs of a developing illness were first noticed by her family in 1987 although the significance was not realised at the time. These initial signs included a complete change in her personality. Victim 15 was referred to a hospital for tests for diabetes and thyroid deficiencies.
Victim 16	Victim 16 showed the first symptoms of CJD in his late teens. He died, in his early 20's in 1996. Six months later a coroner determined that it was "more likely than not" that his death had been caused by eating contaminated beef before 1990. Victim 16 was a vegetarian from the age of 16 but his favourite food as a youngster had been beef burgers. The consultant neuropathologist of told the inquest "most neurologists would link CJD to the BSE epidemic, but would probably not say so in public". After repeatedly being told by doctors that Victim 16 was too young to be

	suffering CJD his parents asked me to perform a urine test. In January 1996 I confirmed that Victim 16 had CJD before he died. This confirmation prompted a full post mortem. The acknowledgment following the post mortem that Victim 16 died of the new strain of CJD sparked a new urgency in official investigations into the link. If I had not tested Victim 16 the authorities might not had admitted the existence of the new atypical strain of CJD. With Victim 16's case the Government began to accept for the first time that BSE was the likely source of infection. In doing so, they were confirming the validity of what I had been publicly claiming for several years in the face of continuing and regular official opposition and discouragement.
Victim 17	Victim 17 was in her 40's when she died from CJD in 1996. Her symptoms started with depression, balancing and walking difficulties and clinical symptoms consistent with new strain CJD. Her partner told me that medical staff blamed him for causing depression. After her death she was confirmed as one of the first 10 cases of new strain CJD. Victim 17 was a blood donor. Her partner was told by Dr Martin Zeidler from the CJD surveillance unit that the cause of her illness was a rogue gene. He said that in 99.9% cases there are cases on the other side of the victims' family with Alzheimer's Disease. Following Mr Stephen Dorrel's announcement Dr Zeidler came back to her partner to confirm that Victim 17 was one of the 10 cases referred to in the announcement.
Victim 18 and Victim 19 *	Victim 18 and Victim 19 were twins. They both died of CJD. Victim 18 died first in her 50's in 1989. She woke up one morning with her shoulder hurting. Her doctor diagnosed a trapped nerve in her shoulder. Within days she developed weakness in her legs and found it difficult to walk. She was treated for depression. Huntington's Chorea was suspected. After an EEG test, her family was told that she had CJD. Her clinical symptoms were consistent with new strain CJD. Victim 18 had been a blood donor from 1981. In 1996 Victim 19 died after an 18 month illness aged in her 50's. Around 1991 she had had a blood transfusion following an ulcerated gullet. Starting with a slight tremor in her hands the early symptoms of Parkinson's Disease gradually progressed until Victim 19 was staggering and losing her balance. She started falling over just as Victim 18 had done, reminding the family once again of the same symptoms. I was asked to carry out a urine test. This was done and Victim 19 was found positive for CJD. The family discussed the importance of a post mortem with me and asked me to witness the post mortem and carry out other independent tests on her brain. Examination of Victim 19's brain revealed extensive vacuolation of the cerebellum and numerous PrP positive plaques demonstrating that new strain

	CJD does not only affect the young under 40.
Victim 20 *	Victim 20 died in his 40's in 1996 after an illness of 11 months. His partner told me about his initial symptoms as having difficulty in walking and balancing. He developed shakes similar to those seen in BSE cattle. His symptoms were attributed to muscular pains in his neck. His mental confusion appeared only later. He had diagnosed himself as suffering from mad cow disease. His partner was told by doctors that he could not have BSE because he was too old. The surveillance unit's findings on post mortem were that "examination of the fixed brain confirms the clinical diagnosis of Creutzfeldt-Jacob Disease in this patient. The histological and immunocytochemical features in this case are distinct from the new variant of CJD identified earlier this year. The appearances in this case are a characteristic of a subset of sporadic CJD cases in which plaque formation occurs, being most evident on PrP immunocytochemistry and spongiform change is most pronounced in the cerebellum and sub-cortical white matter". However my examination of the brain revealed no significant pathological differences between this brain and those brains identified by the surveillance unit as affected by nvCJD. The surveillance unit clarifies this brain as a "subset" of classic CJD.
Victim 21	Victim 21 was healthy until CJD struck. She died in her 30's, in 1996 after being ill for 11 months. For most of her illness doctors and psychiatrists treated her for depression. She would fall while walking and had great difficulty balancing herself. Her clinical symptoms were consistent with new strain CJD. Her family suspected that she was suffering from mad cow disease and doubted medical diagnosis. They demanded that Victim 21 be tested for CJD. Results sent from America the day after she died confirmed that she was suffering from CJD.
Victim 22 *	After being ill for about 10 months Victim 22 died in her late teens. Her clinical symptoms had been those of new strain CJD; depression, balancing difficulties and shaking. She was treated in the same hospital as Victim 16. After a number of tests she was first suspected as suffering from ME and later from a brain tumour. Eventually Victim 22 was diagnosed as suffering from CJD. She had no links with any known source of the disease such as infected growth hormone and there was no history of the disease in her family. Her mother told me of the feeling that she was left unsupported and on her own without anybody to help or counsel the family with the difficulties they faced. The family felt angry and frustrated. Although Victim 22 was clinically diagnosed as suffering from CJD, no advice was given on the importance of a post mortem and therefore none was performed. Victim 22's death is therefore not included in the CJD surveillance statistics as a definite nvCJD

	victim.
Victim 23 *	Victim 23 became ill in his 40's and in 1996 he changed dramatically from being sociable. As his illness developed he staggered increasingly and had difficulty in walking necessitating the transfer of his bed to the ground floor when he could no longer climb the stairs. His clinical symptoms were consistent with new strain CJD, depression, staggering, falling over, having difficulty in walking. Initially he was sensitive to his tendency to stagger and when out used a walking stick to let other people know that he was not drunk. He had been a regular blood donor and the final occasion on which he had donated blood was only some 6 months before the appearance of his symptoms.

Victim 24

Victim 24 died of CJD but has not been classified as a nvCJD victim. She is a nvCJD victim.

Victim 24 became ill in May 1993. Her immediate clinical symptoms included loss of weight, loss of memory, balancing problems, poor eye sight, depression and tiredness and were consistent with nvCJD.

She was admitted to hospital on 6th August 1993 where a brain biopsy was carried out. She and her family were told by the Registrar that she had spongiform encephalopathy. The Registrar was asked to write this down so that the family could remember the words. He wrote "spongieform (sic) encephalopathy".

However Victim 24 then survived for a number of years. Whilst she was alive I wished to carry out a test on her urine, but no arrangements were ever made to allow me to carry out the test.

A CSF (cerebral spinal fluid) test was carried out at the end of October 1996. Victim 24's family were told that this test had a success rate of 83/84. The test was negative for CJD and the conclusion was therefore drawn that Victim 24 did not have CJD in light of the CSF test and the length of her period of survival.

Victim 24 died at the end of 1997. Her next of kin agreed that I should be present at the post mortem as an independent observer and agreed that I should be provided with specimens for independent researches. My tests on her brain tissue have revealed that Victim 24 died suffering from CJD. There were gross spongiform changes in her brain including the cerebellum, which is only seen in cases of nvCJD. She also had PrP positive plaques although the plaques were not typical of nvCJD plaques. However, such variations will be seen in different individuals with different genetic make up. As I understand matters this is now accepted as a possibility by the National CJD Surveillance Unit. This acceptance clearly demonstrates that cases which have not been classified as nvCJD because of their atypical distribution of PrP plaques may

	well have been, as I have always maintained, nvCJD, the explanation for the plaque distribution differences being variations in the host prion protein. Subsequently the CJD Surveillance Unit confirmed that Victim 24 did indeed have CJD.

4. CONCLUSIONS

4.1 In my opinion which I expressed at the times mentioned below the following steps should have been taken or should have been taken at a much earlier date:

a. In 1988 MAFF should have fed MBM to cattle to determine the true incubation period in order to identify the true cause of infection. I said this in my seminar at CVL in August 1989 (paragraph 2.12) and on many subsequent occasions.

b. MAFF should have injected or fed both types of scrapie to cattle to check that scrapie was the source of infection and to identify the strain of scrapie agent responsible. It did not do so. I said this during discussions following my seminar in Brussels on 14 September 1993 **[M37]**.

c. According to the SEAC report of the 20th March 1996 **[YB96/3.20/1.1]** MAFF's infectivity tests are not sufficiently sensitive to determine whether or not red meat and milk contain the BSE agent. Nor does anyone know according to the same SEAC report, what level of infectivity would be harmful to man. As I have recommended on several occasions since 1988, the most sensitive test would be to feed muscle meat and milk from known BSE cases to mink. This is because of all animals, mink are known the be the most susceptible to BSE. If the meat could infect mink, it would do

so in two years or less. However, this experiment has not been carried out.

d. My tests should have been adopted in order to "clean" the national herds and guarantee the return of European confidence in British beef. My test could have been used in two ways (and still can).

i. As a test in slaughter houses to pick up sub-clinical cases. The progeny of these animals could then have been traced back to farms and slaughtered. This would have helped to eradicate BSE from the national herd. Professor Almond in his evidence to the Inquiry has accepted that sub clinical cases will have been processed for human consumption.

ii. The urine test could be used on farms to clean the herds that have animals in them which are incubating BSE and give those herds which yielded no BSE cases a clean bill of health.

I said this on many occasions, including the occasions referred to in paragraphs 2.12, 2.13, 2.30, 2.31, 2.35 and 2.40

e) I should have been allowed to complete the transmission studies referred to in paragraph 2.10 for the reasons set out in that paragraph. Similar studies were started by MAFF in 1996 using only the brains of those CJD victims they considered to be nvCJD. However, there are many other CJD victims who have died of similar clinical symptoms and histopathological features. I considered these cases to be atypical starting from 1988. I strongly believe that these CJD victims died after being infected with BSE contaminated tissues although their ages may be higher than the Surveillance Unit believes relevant (See "Death on the Menu"). This "age limit" is arbitrary and since the relatives of these victims also believe that their loved ones have died after being infected with the BSE agent it is

vital that brains from these cases are used in transmission studies. MAFF are still not testing such atypical cases which would be necessary to give the ultimate answer as to whether these people have been infected from the BSE agent and therefore establish the full extent of CJD cases caused by BSE, which may substantially exceed current Surveillance Unit statistics.

4.2 Given this possibility the Government should have made the development of this test an absolute priority. It should have funded research and facilitated trials. Above all it should have kept an open mind and should have encouraged a wide variety of approaches to the problem, rather than shutting down any line of enquiry which did not conform.

4.3 Instead the government sponsored ill-conceived work which was peer-reviewed by people who have had no experience of working first hand on SE's and have a vested interest in receiving grants themselves. For example Professor Jeffrey Almond was actively involved in peer-review although he had no personal working experience in the area, and received over £1m in research funds.

4.4 As I pointed out to Professor Almond in a public meeting on 31st March 1995 Professor Almond's own researches were far from satisfactory. He endorsed transgenic mice experiments which were highly publicised in January 1996 when it was announced that transgenic mice inoculated with BSE material did not develop the disease. By implication, therefore, the risk to humans was considered remote. Unfortunately this leak was based only on premature results and the final results were some two hundred days away. The final results were not available when the link between BSE and CJD was accepted. However, the experiment had already been used to reassure the public that BSE posed no threat to humans.

4.5 When the results did become available, they proved that mice did develop the clinical disease. Professor Almond is, nevertheless, the trusted advisor of the

Government. In my view which I expressed at the time, Professor Almond's research whilst of possible academic interest was unnecessarily expensive and of no immediate practical benefit because;

a. Transgenic mice are no more susceptible than non-transgenic mice. There are many different breeds of mice which have different incubation periods after inoculation with the same source of the agent. Therefore the creation of another type of mouse merely creates a different incubation period. If all of the breeds develop the disease without the insertion of a foreign gene then the value of transgenic mice experiments is very limited and of academic importance only.

b. Only a handful of mice were used in the experiments, compared to the 55 million people in this country alone who are on trial.

c. Mice have a six hundred day life span, compared to the 60 years plus for humans.

d. It was similar to experiments which had already been carried out in U.S.A. which eventually concluded that PrP itself is not the agent.

4.6 I expressed the following views on many occasions as and when the issues arose in various seminars and public meetings which I attended.

Culling Policy

4.7 MAFF's simple visual examination of live cattle to establish whether or not they are showing BSE symptoms, did not stop sub-clinically infected cattle from entering the food chain. These affected animals killed have not been included in the figures as BSE cases.

4.8 According to MAFF continuing cases of BSE are the consequence of meat and

bone meal produced since the feed ban of 1988 being fed to cattle. If this is correct the implications are appalling. Since 1988, this meat and bone meal was prepared from cattle which were assumed to be healthy and fit for human consumption (apart from the cattle waste that was used to produce meat and bone meal, the animals went for human consumption).

4.9 In the Autumn of 1995, in a blind trial filmed for the World In Action T.V. programme, I used my post mortem test to examine brains taken from cattle that were apparently healthy when slaughtered. I found 29% of the brains to be positive for BSE.

4.10 These findings alone highlight the gross short comings of the culling policy. Meat from animals over 30 months old is no longer allowed into the food chain. However, culling animals over 30 months old (ie. those most likely to exhibit obvious symptoms of BSE) merely means that animals under 30 months which are incubating the disease, but not yet showing symptoms will continue to enter the food chain as before.

4.11 MAFF's position is unsupportable. It assumes that sub-clinical animals will not contain enough of the disease agent to infect humans. However, people will continue to eat meat from cattle affected by BSE. Mr Ray Bradley reported to the House of Commons Select Committee (Report page 72 -1990) **[IBD 7]** that "furthermore, the scientific evidence demonstrates conclusively, from studies from natural sheep scrapie, that muscle, the meat, the beef, is not infected or has no detectable infectivity even in clinical cases, never mind sub-clinical cases. So I am very happy about even the clinical cases going through and the meat being eaten, although we do not want it". He also said "the perception at the moment of the public is that there is still an increasing occurrence of clinical BSE. While this is happening I am very confident indeed that the infectivity is falling". This is not correct see p.40 of Public Health Trust conference report **[M9 Tab 23]**.

4.12 It may well be that a mass culling will win back public and European confidence under false pretences. According to MAFF's recent correspondence with me about verifying my test, MAFF cannot provide me with 10 negative urine samples from live cattle in this country, because it cannot guarantee that those samples will be BSE free. MAFF tells me that it is trying to source those samples in New Zealand, which is BSE free. The import of this is obvious: if MAFF cannot provide 10 samples from British cattle and guarantee that those animals are BSE free, how can it maintain that beef from cattle under 30 months old is safe to eat?

4.13 Under the culling policy, thousands of BSE free animals are killed every year. Since MAFF cannot distinguish between infected and BSE free animals, there remains the difficult and dangerous matter of mass disposal. Cattle remains cannot be safely spread on fields, nor can they be buried at the risk of polluting the soil and local water tables.

4.14 Another MAFF misapprehension is that only cows with clinical signs of BSE can contaminate farm land with their dung and urine. This is not the case. If an animal has been eating contaminated feed it will excrete any undigested disease agent from day 1. It does not have to be diseased itself to recycle the disease onto farm land, where it will remain for several years.

Absence of BSE Brain Lesions at Post Mortem

4.15 Vacuoles in neurons aftermath of infection have diagnostic significance and have become a well known diagnostic hallmark of scrapie BSE and CJD. Even in the natural disease vacuoles may be sparse and hard to find.

4.16 Lasmezas et. al. (1997 Science, 275,402-405), in the first passage from cattle to mice demonstrated transmission of the BSE agent to mice in the absence of detectable PrP-sc and Vacuoles. In clinically diagnosed BSE cases born after the feed ban (BAB) histological studies have revealed an increasing number (39 to

57%) of brains having no significant lesions or vacuolation (Jeffrey et.al 1994, In Bradley R et. al, Eds. EEC meeting Brussels September 1993. Proc Cons BSE Sci Vet Comm Comm E C 347-358) In Scotland the number of cases with no significant lesions in cattle BAB has gradually increased from 26% in 1988/89 to 41%. The rising number of cattle showing the typical BSE symptoms with negative histopathological results in cattle BAB, and no alternative diagnosis being established, raises a number of serious concerns. These include the question whether all cases of BSE arise from contaminated feed or whether they represent maternal transmission. Atypical cases may be caused by other strains of the scrapie agent with a different distribution of lesions. This significant number of clinically suspected cases BAB with no characteristic vacuolar pathology, have therefore been presumed BSE free despite the presence of clinical symptoms of BSE. After so much practical experience of BSE symptoms is it likely that vets are now making the wrong diagnosis in nearly 50% of cases? I would submit that it is more likely that following the ban the BSE disease agent has mutated in some way, possibly through the process of maternal transmission, so that the BSE symptoms are apparent but old strain BSE brain lesions are not. Furthermore cattle suspected of BSE are now killed within days of showing clinical symptoms rather than weeks and months as used to happen in the early stages of the disease, where the pathology was therefore more obvious.

4.17 However, these cases are not being thoroughly investigated and it is important that a second line of tests should be used to determine the nature of the disorder. One clue as to these "BSE free" BSE cases may lie in the type of the scrapie strain.

4.18 Likewise, in humans there are now many suspect CJD cases where the post mortem pathology is not typical. My belief is that many of these cases may well be different strains of CJD.

4.19 Although there were scientists who had worked directly with scrapie and CJD before the BSE outbreak, they remain conspicuously absent from those bodies

which have since been advising the Government. Those people who have been advising the Government are all experts in their own field and eminent in other specialities. However they are not expert and eminent in the fields of scrapie and CJD. This may explain why research which should have been done has still not been done since funding was approved or not approved by these people.

BSE In Other Animals

4.20 Since the emergence of BSE, similar SE's have been diagnosed in domestic cats and captive wild animals at several zoological collections in the British Isles (BSE in Great Britain, a progress Report, May 1996).

4.21 Three cases of SE's with an unknown infectious agent have been reported in ostriches (Struthio Camellus) in two zoos in north west Germany (Schoon @ Brunckhorst, 1999, Verh ber Erkeg Zootiere 33:309-314). These birds showed protracted central nervous symptoms with ataxia, disturbances of balance and uncoordinated feeding behaviour. The diet of these birds had included poultry meat meal, some of which came from cattle emergency slaughter cases.

4.22 BSE was declared a statutory notifiable disease by MAFF on 21st June 1988. The feeding of ruminant meat and bone meal (MBM) to ruminants was blamed as the source of infection for cattle. To prevent further transmission of the infective agent, a ban on the use of MBM food stuff was introduced in July 1988. A call was made to extend the ban to pig and poultry feed in case the BSE agent "jumped species". MAFF, however, argued that pigs were not at risk because they are natural scavengers (and have evolved defences against pathogens) and poultry were not at risk because of the enormous zoological divide between cattle and poultry. However, a ban on the use of MBM in all farm animals including pigs and poultry was introduced in August 1996. A notification of Spongiform Encephalopathy was introduced in October 1996 in respect of ungulates, poultry and any other animal.

4.23 MAFF have carried out their own transmission experiments with hens. In these experiments, some of the chickens exposed to the BSE agent showed neurological symptoms. However MAFF have not so far published details of the symptoms seen in chickens. Examination of brains from these chickens did not show the typical pathology seen in other SE's.

4.24 A farmer in Kent in November 1996 noticed that one of his 20 free range hens, the oldest, aged about 30 months was having difficulty entering its den and appeared frightened and tended to lose its balance when excited. Having previously experienced BSE cattle on his farm, he took particular notice of the bird and continued to observe it over the following weeks. It lost weight, its balance deteriorated and characteristic tremors developed which were closely associated with the muscles required for standing. In its attempts to maintain its balance it would claw the ground more than usual and the ataxia progressively developed in the wings and legs, later taking a typical form of paralysis with a clumsy involuntary jerky motion. Violent tremors of the entire body, particularly the legs, became common, sparked off by the slightest provocation. This is similar to that seen in many BSE cases where any excitement may result in posterior ataxia, often with dropping of the pelvis, kicking and a general nervousness. Three other farmers and a bird breeder from the UK are known to have reported having hens with similar symptoms. The bird breeder who has been exhibiting his birds for show purposes for 20 years noticed birds having difficulty getting on to their perch and holding there for any length of time without falling. Even though the bird was eating normally, he noticed a weight loss of more than a pound in a bird the original weight of which was 5 pounds.

4.25 Histological examination of the brain revealed degenerative pathological changes in hens with a minimal vacuolation. The presence of PrP immunostaining of the brain sections revealed PrP-sc positive plaques and this must be regarded as very strong evidence to demonstrate that the hens had been incubating Spongiform

Encephalopathy.

Vaccine

4.26 There are some 20 different strains of the scrapie agents. Infection with one strain blocks the others. (See para 1.16).

4.27 Comparative mink experiments using scrapie and BSE strains demonstrated that mink fed with scrapie brains did not develop the clinical disease and only 10 to 15% of those injected developed the clinical disease. However, all mink fed or injected with BSE brain tissues developed the clinical disease. Similar results appear to have been coming out of cattle experiments. I therefore consider that it is important to feed and inject with BSE brain tissue mink which have been fed on scrapie brains to demonstrate that the scrapie strain would block the up take of the BSE agent. This could be used in cattle to produce a vaccine and protect environmental infection of cattle. If this phenomenon is true for humans, many of us will have eaten scrapie infected tissues and will therefore be naturally protected from BSE which would be a great comfort to know.

Blood Donors

4.28 As a result of my work in USA in 1985 (see para 2.7), in May 1985 I wrote to Dr Whitehead (Director of PHLS) **[M37/85/5.6/1.1]** warning of the consequences of the use of human growth hormone in the UK. At a subsequent discussion with him, on my return to the UK, Dr Whitehead told me that no one knew what the outcome of growth hormone treatment in the UK would be and that he did not think that it was right to do anything at that stage. (In one case in the UK growth hormone treatment was stopped in 1989 only after a patient developed clinical symptoms). Similarly, I had been warning about the use of blood and blood products in blood transfusions which might produce a devastating effect (as in the case of growth hormone treatment) in the next few years.

4.29 Finally, therefore, I would like to utilise this inquiry for the purpose of a plea that all future blood donors should be tested utilising my urine test to prevent further transmission from blood products. It has been well established from Japanese and American studies since 1980 that if animals are injected with blood from human CJD cases they develop a clinical disease. (see the detailed references on p260 of "The Link"). Transmission studies in mice have shown that each millilitre of blood contains 1000 units of infectivity (see p260 "The Link" and detailed references). Recently Dr Rohwer has told me that in animal experiments he has demonstrated that infectivity is present in both cells and plasma at all stages of the incubation period.

4.30 I have identified in my case studies of victims at least 12 cases of CJD victims who have been blood donors. I understand that the brain pathology of CJD victims where the disease was caused by growth hormone treatment is different from the pathology of classical and nvCJD in that there is a different distribution of PrP plaques. In one case where the patient had had a blood transfusion about four years before symptoms appeared, an examination of the brain revealed a distribution of PrP plaques similar to that seen in growth hormone human to human transmission.

4.31 In one study, Esmonde et al (1993) ("The Link" p260) it was found that out of 202 definite and probable cases of CJD, 21 had received a blood transfusion. As a matter of urgency their brains should be re-examined to demonstrate whether they have similar features to those seen in Victim 19. This would demonstrate without delay whether blood transfusions pose a risk to human health. In the light of this it would be prudent for the brains of those people who have died of CJD and who have had recent blood transfusions to be re-examined. All new cases should be properly tested.