Witness Name: Stephen Dealler

Statement No.: WITN7065001

Exhibits: WITN7065002 - WITN7065035

Dated: 14th May 2022

INFECTED BLOOD INQUIRY

WRITTEN STATEMENT OF STEPHEN DEALLER

I provide this statement in response to a request under Rule 9 Request of the Inquiry Rules 2006 dated 10th November 2021

Section 1: Introduction

1. Please set out your full name, address, date of birth.

1. I am Stephen Francis Dealler, date of birth GRO-C/55 and living at GRO-C GRO-C, mobile number GRO-C and using Stephen.dealler@GRO-C. I will say as follows: Information being given to Blood Transfusion Service (personal interaction with Chief of laboratory at Colindale), the inability of testing blood for diagnosing BSE or any increase in CJD in the population, the lecture given to the Blood Transfusion Service, the meeting between myself and the Haemophilia Society who at the time were in South London, Interaction with Prof J.R.Pattison, (Chairman) Professor of Medical Microbiology and Dean of the University College, London, Medical School in which I made this clear that we could not know the risk from blood yet

(1993). Personal 2 representations with the Spongiform Encephalopathy Committee in which I tried to make it clear that assuming BSE to not be infective to humans was unwise (1992 and 1993 possibly). No doubt there are plenty more but I cannot be sure and have no documents because of my multiple movements in household and computer systems. I am happy to attempt to give information to the inquiry and keep it safe and not tell others. However much of what I am saying has either been published or has been told widely to others, often the press at the time. As a scientist at heart all information that I give is true and is neither aimed at anyone else nor felt by myself to be hidden from others except as what the Inquiry has received.

2. Please see a copy of my CV [WITN7065002]. I have worked in 47 hospitals in 9 countries, published over 200 scientific documents, 2 books, and have the medical equivalent of PhD.

3. I am a Consultant Medical Microbiologist (retired) who was working with Professor Richard Lacey in the department of Medical Microbiology at Leeds University. I provided him with information that showed the risks from BSE to humans could not be certain and hence should not be certain as zero either as was being almost assumed by SEAC. As such American researchers were treating the bovine tissues as infective whereas in the UK we were still eating them. With Professor Lacey I was the researcher that found large amounts of Listeria neoformans in ready meals in supermarkets, the poor action of microwaves in killing food bacteria in these meals, and was involved in the finding of Salmonella in eggs. All this data was taken by the Professor, who was looked on by officialdom as causing a major harm by interacting mainly with the media to get the information put about. It must be said that I am not like that and try to act in a standard scientific way and interpret data as any doctors should. My association with Lacey may have been involved with my simply not being believed concerning problems with blood transfusion.

2. ***Please set out your employment history including the various roles and responsibilities that you have held throughout your career, as well as the dates.***

4. My employment history is in my CV which is enclosed [WITN7065002]. It also shows my membership of societies.

3. ***Please set out your membership, past or present, of any committees, associations, parties, societies or groups relevant to the Inquiry's Terms of Reference, including the dates of your membership and the nature of your involvement.***

5. Many of these are present in the CV but I also set up the Spongiform Encephalopathy Research Charity (now defunct) in around 1993. In 1996 (approx.) I set up Microsens Biotechnologies for the private research into BSE and all other TSEs aimed at commercial products. I have never been part of national committees (I have never been invited to do so) but have given evidence to the BSE Inquiry as you can see in your document BSEI0000002. I was permitted 1 year unpaid leave to carry out research at the Public Health Service in Leeds to merely advise them as to how to progress in research into BSE/CJD and investigate potential chemicals to prevent its infectivity. Please see the letter to Dr. Tompkins [WITN7065003].

4. The Inquiry is aware that you provided evidence to the BSE Inquiry in 1998. (BSEI0000002) Please review these documents. Do they remain true and accurate as far as you are concerned? If there are matters contained in these statements that you do not consider to be true and accurate, please explain what they are and why they are no longer true and accurate.

6. The data given to BSEI0000002 remains true as far as I can remember. Many factors are missing, particularly concerning the development of 2 drugs that could be used quickly and would be expected to slow down the progression of prion disease (published from work in mice) [WITN7065004]. The method to create a PCR test for detecting potentially harmful prions in the blood was being created with Microsens Biotechnologies, and with the University of Dublin Veterinary Department, and with one of the Stockholm Universities [WITN7065005]. This was around 2004. The attempts to get it tested and working were difficult and although Stuart Wilson at Microsens tried hard he and myself had great difficulty getting samples or getting results back from the CJD Surveillance Unit and MAFF (Ministry for Agriculture, Forestry & Fisheries), (see NHBT0086239). In NHBT0040825 you can see that Microsens Biotechnologies had managed to get samples and tested them using a highly sensitive system in which pentosan polysulphate (PPS) was attached to a plastic tube to allow it to remove any PrPsc format of prion protein. The normal protein would be washed off and then a strong ELISA was carried out using an antibody that claimed to be against the prion form of the protein. The lack of interest in the UK was more the cause of information going abroad.

5. Could you please confirm whether you have provided evidence to, or have been involved in, any other investigations, criminal or civil litigation in relation to human immunodeficiency virus ("HIV") and/or hepatitis B virus ("HBV") and/or hepatitis C virus ("HCV") infections and/or variant Creutzfeldt-Jakob disease ("vCJD") in blood and/or blood products. Please provide details of your involvement and copies of any statements or reports which you provided.

7. You can see my published work on all these diseases in my CV [WITN7065002]. My work on HIV was through the development of plant based treatment. In relation to Hepatitis B, it was being able to find it in certain conditions, Hepatitis C was similar and some work was carried out in Sudan. My vCJD work has been largely involved with interacting personally with researchers throughout the globe, and the large amount of published literature. Other works on CJD have been through BSE, statistics, viewpoints, and my

long term attempts to get action taken by the DoH. I should say that the researchers and veterinary advisors for MAFF were often perfectly reasonable, but one went to see the Chief Executive and told him that he could not go on misleading the population. At that point the door was closed, and the Chief Exec told him that no word must be said under Whitehall rules or he would be jailed. He quit the next day. He contacted me to tell me of this problem.

Section 2. Knowledge of Risk of vCJD transmission via blood transfusions and blood products

The Inquiry seeks to gain an understanding as to how knowledge of risk of vCJD developed over time within the UK Government, Blood Services, Haemophilia Centres and other NHS organisations and the adequacy of their response.

8. At the time there was little information available except from animal testing [WITN7065006; WITN7065007]. What was clear was that the amount of infective material transferred was important in the incubation period and the possibility of disease transfer at all, and that this could not be assumed to be zero. What was known by 1995 was that we really did not know the number of people that should be considered as infected or even adequately exposed [WITN7065008; WITN7065009] but that it would be perfectly reasonable to assume that a unit of blood but not a needle prick may represent a risk. See the lecture given to the BTS (the specific lecture referred to couldn't be sourced, but this article speaks to similar contents of the lecture) [WITN7065010].

6. Where and in what circumstances did you first become aware of the risk of transmission of vCJD via blood and blood products?

9. I became aware that BSE may transfer as a different strain and not that of either BSE or Scrapie in 1988 simply by reading the large international literature. I had become aware of the risk of vCJD almost as soon as it was announced by

Knight and Ironside in 1995. The reason was that the variant could quite easily be a different strain of the disease, and as such act in a similar way to the prion disease of scrapie or in hamsters. I had already approached experts before vCJD appeared and discussed this. They made it clear that it should not be assumed to be the same as scrapie (and so have little effect on humans). I heard that a possible new form of CJD case had appeared in about March 1994 probably after someone from the media rang me. Noticeably only the UK researchers and particularly with the MAFF treated BSE infected tissues as little risk to humans. All others that I knew of assumed that it was a risk and carried out laboratory research in cabinets (called Category 3).

7. *How did your knowledge of the risk of transmission of vCJD via blood and blood products develop over time?*

10. My interaction with international groups gave me further knowledge showing me that I could not expect the disease to be destroyed by blood being kept cold, or there to be extraction procedures for blood. The American group in Montana were worried about it at that time. Again large amounts of work had been done in animals with other TSEs. The incubation period depends on the normal lifespan of an animal species (say 50%), it would depend on the dose that was given. One infectious unit (IU) was the minimal amount when injected into the brain that would cause the disease transfer. The number of IU needing to transfer it by injection into the blood might be 100. But the number of IU in a ml of blood might be small. The major problem would be that by injection into a mouse you simply cannot inject enough blood to test the animal. Even if you injected 0.1ml of blood that might only contain 10 IU, and if 1000 IU was needed to transfer the disease then it would not transfer (it is not as if 1 in 10 of the mice getting the injection would die it would be none at all). In humans we give 500ml of blood commonly as an infusion in health care and that might mean that 50,000 IU was being transferred but 0.1ml in a mouse test would only receive one 5,000th of that i.e. 10 IU. What this meant was that it was not possible to test human blood in an animal while the infective dose could not be

known. While there was no test for blood for CJD there would be no way of offering blood as a method of treatment for people that seriously required it. At that time, the release of this information would cause multiple deaths in people afraid of getting the disease from blood when the calculated risk simply could not be known. As we all know from watching the Coronavirus-19 outbreak, no drug is permitted to be used unless the advantages are seen to be greater than the risks, and drugs that are untested (in this case blood). It could not be accepted for human use except with specific considerations (we have seen this clearly with Covid-19). I was not surprised when many countries' equivalent committees did not permit blood transfusion from people that had been in the UK during the BSE epidemic and hence may have eaten infective cattle tissue. If it was considered necessary to progress ethically with the risk from blood being unmeasurable and yet human blood itself being often urgently needed for human life then certain aspects must be progressed. Potential prophylactics, and treatment for CJD must be investigated, and diagnostic systems other than by using animals must be researched. These would allow blood to be used pharmacologically, and yet minimising the risk to the disease if it was found that it was transferred by blood transfusion. See question 10 below

8. ***An account of your understanding of the relative risks of vCJD infection from the use of domestically sourced blood and blood products and the use of commercially supplied blood products.***

11. I don't think that I had any data on either the domestically or foreign sourced blood except newspaper data. At the time in 1993 it was clear that Hepatitis C outbreaks and HIV were being discovered in recipients of blood factors and I tried to discuss the possibility that BSE may also be a risk [WITN7065011]. The BSE infection might be concentrated in certain blood products but again this was not clear at that time.

12. I met with the head of the Haemophilia Society. I had been trying to show him that the calculated chance of BSE transferring to humans might be low but may not be and if it was, then the risk from multiple regular injections of extracts of UK human blood may represent a risk. The work with various extracts had not been carried out at that time. The head of the Haemophilia Society was comparing the problem to something like the risk of Hepatitis C or AIDS infection. The problem with those was that because blood transfusions initially were not tested (although this was still true even after testing became possible for a period) and they were seeing those diseases appear in haemophiliacs. He was anxious that exactly the same thing would happen with CJD so I had to calm him down and talk about calculated risks. But I was worried as well and that is why the later research did take place on CJD infectivity in blood products.

9. In a letter dated 6 January 1997, Dr. Robinson noted that you proposed to set up an independent meeting to discuss the risks of CJD via blood and blood products, please see: NHBT0004586_001. To the best of your knowledge please outline the following:

(a) Some background to this document, are you aware of the papers that were being referred to in this letter? If so, please set out what the discussion was. Was it the document at NHBT0004586_003? Did this proposed meeting take place? If so, what was unearthed from this meeting and who was in attendance?

13. Dr Robinson was a reasonable person and generally I was no longer seen to be speaking rubbish. Looking at DHSC0004090_004: As such, when others said they were already doing the work (looking for prophylactics, treatment, and tests for blood) I was probably very relieved. When you are right in the middle of the thick of things and noticing that risks were not being considered elsewhere by officials but they were by scientists I did consider that rational discussions should appear [WITN7065011]. However, Dr Robinson must have seen me as a risk to the NHBT and so would demand that I was ignored. For a short time, I did consider a separate meeting on this but it never happened. The only involvement

I took was to be invited to give a talk to the BTS probably in about 1999 [WITN7065011]. Notably, the talk which was packed with people at NHBT at Colindale finished, and Dr Robinson appeared to take no notice of the data that I had shown which was all scientific and fairly shocking. I was trying not to be a 'loose cannon' when dealing with them directly. The meeting went on and that was it. At a separate talk (I was told there would be no projector!) but by this time the people from Whitehall had been friendly, helpful and might take notice. This was probably in around 1999. Directly before the Phillips (BSE) Inquiry a lot of the documents from MAFF arrived at the Department of Health at Richmond House and left outside in the middle of the night before the BSE Inquiry, when it was realised that the DoH were going to demand them. I knew the scientists that were working on BSE at MAFF and they were reasonable but doing their best to look at things from the farmers perspective. At the following announcement of the Official Inquiry under Lord Phillips much of the data became clear. I was called to give evidence but said little about blood transfusion because I was not asked to much degree.

14. One major factor in BSE was to work out at what age the cattle became infected and whether this was passed down from cow to calf and I suggested that groups should discuss this. In the end I did the maths myself and published the answer (see figure 2) [WITN7065012]. I could find no sign of infection taking place after 7 months. Almost certainly cows may not have infected calves but the food given to the calf previously to the ban had been infective, and this stopped almost as soon as MAFF directed it; an excellent and impressive action. Similarly, I asked that academic groups should get together and try to work out what was happening in vCJD and what could we do to predict a long term effect of the disease. As far as I know this was simply taken up by others and I only went to a meeting in York. At the time some of the best and most effective scientists in the world on this type of disease were working in the UK. Document NHBT0004586_003 seems to be from 1996 when the blood transfer data was not known except in other species and neither was Paul Brown's work showing the distribution of the disease in blood, along with the infectivity of various blood components. At that time many scientists in the field were worried that the UK human population may give themselves more infections than were needed via

blood, tissues or blood components that might be transferred. The message was written in Times New Roman and so I presume I was probably the author (because TNR was automatically used by me). It was suggesting that a group could be asked to discuss the problems. However I do not think that this group got together in any way and it was really just me sending out a list of worries that should be considered by everyone. I notice that pentosan polysulphate for prophylaxis is included as the toxicity is largely known, but no question concerning looking for methods of treatment is mentioned.

(b) What was the rationale for your belief that there were risks associated with CJD and blood?

15. The model was that we gave ourselves such a large dose of blood in a single transfusion. It does make a difference whether you are a mouse or a camel as the number of IU in blood would be expected to be similar in blood per ml, nor would it make any difference about the dose that was needed to transfer the disease. So, you could test a human genetic prion protein (PrPc) (genome) mouse with blood injection into the brain but you just could not give enough intravenously to find out if the blood was infective easily (DHSC0032421_066). Transfer between species was discussed in an article from the USA in 1999 where the scientific injections had taken place (WITN7034017). Much research concerning the presence and transfer of Transmissible Spongiform Encephalopathies (TSEs) in blood has taken place by animal injection. Please see the following references in relation to this: [WITN7065010; WITN7034017; WITN7065015; PHEN0000612; WITN7065017; WITN7065018; DHSC0006331_002; WITN7065020; RLIT0000668; RLIT0000713; WITN7065023; WITN7065024].

10. In an email chain from 18 March 2004, you suggested that, "the most useful way to produce academic results is to have an interaction between departments" and proposed to produce academic results regarding "age [year] of birth and death of CJD". To your knowledge, did such an interaction take place? If so,

please outline the results, please see: DHSC0004090_002. You may also wish to refer to DHSC0004090_004. What was (or would have been) the benefit of this?

16. I had a look at the emails (shown in DHSC0004090_002 and DHSC0004090_004 or it would make sense if I had). It became clear that they were not going to happen and I might be looked on as polite now but still a potential threat to their academic safety

11. At paragraph 94 of your statement to the BSE inquiry, you stated that you were "providing information to SEAC and the DH and have been involved with the risk analysis procedures of blood transfusion". Please outline your involvement and how your information was used by these organisations (You may wish to refer to: BSEI0000002).

17. I gave two talks to SEAC; one to experts from the Dept of Fisheries and Food (with Professor Lacey) and a second one organised through the BMA in a room at Tavistock Square. Of course, there was no data about vCJD as it had not appeared at that time. So, it was difficult to take animal model data, calculated data and species data and use this to get SEAC to listen at all. The Dept of Health rarely seemed to answer calls from me and in the end, I found out that they had in some way been told by Whitehall that this was an agricultural and not a human health issue. When in the end it became clear that the DoH would be fully involved in the investigation from Parliament and the major one, I spoke about it with the DoH and they simply told me that they had received the documents from the researchers for the DFF at Richmond House in the middle of the night.

18. The Public Health Service was particularly unhelpful and did not want me to do any work on this at all. The man in charge of this in Leeds made it quite clear. He told the human resources committee (in Burnley) on my being offered a

consultant post in Burnley that I was grossly incompetent clinically, and when I drove a member of that committee to the station, he warned me just how awful this interview had been. I had in fact been working in Burnley as the Consultant locum for 6 months with no problems so I was offered the job. However, after vCJD appeared the DoH did appear to realise that I was a useful source of information. Kenneth Calman (Chief Medical Officer at the Department of Health) and Dr Jeremy Metters (often on Heath Steering Groups) invited me down to Richmond House with a Public Health doctor in York with whom I had been trying to organise statistics about BSE and CJD with (Dr W.J. Patterson). Officials had made things very difficult for him prior to vCJD. However, after vCJD appeared we were invited to Richmond House and were taken to see these major advisors to the Minister of Health (Stephen Dorrell, MP). They apologised greatly to us and explained why their action against us had happened and we forgave them and I gave evidence to the House of Commons Select Committee on Health on the subject. At that meeting the ministerial advisor and myself were agreeing on our information for that committee. Calman quit his post soon after the meeting. You should be able to see my statement to BSEI0000002 as valid. It did not surprise me that Calman and the DoH had been to some degree told to keep clear of BSE as it was a MAFF problem.

19. My position throughout all this, I think you can see, is the viewpoint of a doctor (putting the patient first, making sure that you do no harm and that treatment is safe).

20. You will see the risk analysis for BSE that I could make in the associated lecture for the National BTS which took place at Colindale [WITN7065010].

21. I did suggest to others that a prospective risk analysis for humans might be possible using the rising curve of cases of vCJD. But that could only really be guessed at when looking too far into the future. There could be a system of using a curve that would be expected for vCJD by looking at the amounts of disease eaten at

different periods in life. All of these would be found to be wide of the mark because of the long incubation of the disease. The potential risk of being infused with an infective dose would be death to the recipient. All I can say about this is that I gave information to SEAC but I never relied on them to take action. That is why you could see me going via the BTS but it felt that I was throwing horrible data into the fire. I felt that they really did not want to hear this.

12. A memorandum sent on 23 April 1997 states that a report drafted by you was due to be published about CJD and blood. Was this produced? If so, please outline its content. If you have a copy, please provide it to the Inquiry. (Please see: DHSC0006429_074).

22. I cannot remember anything about the publication from DHSC0006429_074. The ones involved are enclosed. Many publications were being turned down however. One apparently disappeared from my computer (I think). I did publish one in 1996 on BSE and blood [WITN7065011]. What Ailsa Wight was announcing was that they were getting ready to be able to answer questions from anything that came from me. This was not necessary in that I was not expecting to be spreading frightening documents for blood recipients through the media. One thing that I had learnt from Professor Lacey was that if officialdom simply did not consider the science when answering questions then the only way to force them to do so was through the media. I had a lot of contacts in the media but did not expect to use them. In a way I did feel that things were progressing and a BSE Inquiry was coming.

13. At point 7.1 of the meeting minutes of the Advisory Committee for Microbiological Safety of Tissues and Blood for Transplantation, 1997, it is reported that you:

(a) ***Suggested "we were about to face an explosion of CJD infection transmitted through blood/blood products." ;***

23. I would have been saying that we simply did not know at this time but we might be 'about to face an explosion of CJD infection due to ….. (there was at the time the sheer worry that was facing all in the field. I would definitely have put a part showing that 'we could not be sure but we might be about to face an explosion…'). So that quote of mine would only have been a partial quote.

24. I am a medical doctor and I have always known that if you do not know the risks of a product it may be dangerous to use it on large numbers of the population. This is still true as you can see with the answers we found for Covid-19 (see the JCVI for instance). We must always warn the public of risks even if we are not precisely sure of what they will be. With BSE being transferred into humans, a fatal disease, with no treatment, no method of early diagnosis could transfer. In my article with Professor Kent [WITN7065008] showing that by the time that food restrictions were brought in in the UK (1988) we would have already eaten (fully) large numbers of infected cattle. SEAC was correct in advising those regulations but even then it might be reasonable that infective parts of cattle were still in the diet. So the risk to humans was somewhere between almost 'zero and 100% of the population' [WITN7065008]. The NBTS must have been extremely worried but did not come over as so. After 1994 and the rise of nvCJD (called at that time the new variant CJD) the worry was indeed anxious as we did not know how many would die of the disease but it wasn't going to be zero.

(b) *Felt you "had a duty to warn the public if their safety was at risk";*

25. To give an example: lung cancer and cardiovascular disease (both fatal with no treatment at the time) are associated quite closely with smoking. When this was shown statistically and in laboratories shown by science to be valid in humans then some countries (like the UK) took action to decrease smoking and warn the population. The Government did not deny the findings. Others (e.g. the USA) did nothing. Was it medically acceptable to not warn people of the risks? With BSE we knew that TSEs did cross species barriers. Was it acceptable to ignore this? I believe now it has infected over 20 species and all but one in which it was tested, surprisingly the hamster.

26. At the time it was not clear where in blood products the involving activity might lie and research involving animals may take several years. In 1997 I had been working through Microsens Biotechnology on several potential systems to remove infectivity from brain tissue that was being tested in tissue cultures and using a pentosan polysulphate (PPS) extraction test. It was not clear how this would progress. So did I think it my responsibility to allow the information to the public as a doctor working on the subject? Yes I did; but unlike Lacey I would always try to get the public warned by Government statements and action and others were quiet too [WITN7065024].

(c) Wanted "to discuss with the BTS various suggestions for stopping inappropriate use of plasma."

27. It was well known in medicine that blood as a pharmaceutical was often given too often and when it was not needed. Plasma also should have had very specific usage learned in medical school. When it became clear that there could be a risk to the recipient from plasma then again I felt that the recipient should know that the risk was even unknown at this point (1994). Patients will assume safety of blood products unless they are told and also doctors will. My approach to the Haemophilia Society was really because they had put up with Hepatitis C and HIV in the products given, when those products could have been tested (at a cost and probably not in the UK).

Please refer to NHBT0006016 and provide answers to the following:

(a) How and to whom did you communicate these fears to?

28. I see that it is claimed that I had contacted Peter Flanagan, John Barbara and Kate Soldan. I really do not remember this at all and have no documents about it. It had been put out at some time to ask Peter Flanagan to make me feel more useful….and the tendency was for them to do so. If I said that I was worried that

there was a tendency to tell everyone that 'adequate action', advised by the committee, was being taken. Then if I looked into it and found that their advice was in fact unethical or medically unacceptable, what could I have done? An idea of a second committee (as we saw with Covid public health advice) might have been useful to them. There would never have been an attempt on my behalf to get what I wanted done as I said it but rather a reasonable scientific group. I cannot see a TSE expert among the members listed in NHBT0006016. As far as I know no small group took place.

29. What I can probably tell you is that I interacted with the head of the laboratory for testing blood at the BTS in Colindale in around 1996. I made it clear to him that I did not think that blood could be tested but did not know what else could be done. It was probably asking him to be involved in research. He told me that he had warned advisory officials about a coming problem with Hep B, Hep C and HIV and action was always taken far too late, at great cost to patients and the Government.

(b) What was the response you received?

30. I cannot remember any response as I can't remember any attempt at trying to form a group. In fact, I knew that many researchers saw themselves as depending on Government finance or pressure from MAFF (MAFF had gone out of its way not to carry out much of the research required for blood products as this would have been the immediate research to start on this subject). SEAC did not seem to stand against MAFF as many research centres in Veterinary Science had been closed under Mrs Thatcher and during BSE. Privately many scientists encouraged me to make the noise that would force the research to continue but themselves they could not speak out. This was a situation in which a committee from its decisions advises a minister that does not know enough to argue against its findings.

(c) What response did you receive from the BTS (Blood Transfusion Service)?

31. From the BTS I got no documents that I still have.

(d) Were your sentiments shared by your peers or were you met with disagreement?

32. My peers were generally on my side in this. Certainly Lacey but many others abroad agreed. The major one was in Berlin that I visited and discussed the problem with (Diringer).

Any other comments?

33. The NHBT0006016 document itself shows from section 7.23 Dr Robinson had 'taken advice and believed that the present state of knowledge was legally and ethically acceptable not to inform recipients….'. Well, I would like to see that advice and how it was put together. While we could not know how many people had received blood from cases of vCJD that had appeared and were apparently not testing any blood (no test) then should we simply remain quiet? That would be not in the format of any other pharmaceutical that we use in medicine. Risks must always be told to recipients. I cannot believe that Dr Robinson had somehow considered that blood and blood products were outside the ethical position of medicines. Just because you don't know the level of risk does not mean you must hide it. Looking at 7.25 (in the document above) and the idea of deferring decision on CJD blood products does remind me of that concerning Hep C and HIV and kicking the can down the road.

14. *On 20 Jan 1997, Dr Angela Robinson wrote to Dr Jeremy Metters discussing the activities of your independent group (NHBT0004584).*

a. *What led you to form an independent group and who was part of that group?*

34. Angela's letter to Dr Metters (NHBT0004584) probably did not understand my 'having formed a small company with a group in London called Microsens Biotechnology initially. It got going simply to be able to test animals for TSE including BSE (it was used mainly in N. American deer in the end). It used a compound called Pentosan (poly sulphonated poly-xylose) (PPS), an anti-inflammatory that also acted to some degree like heparin. It could be given pharmaceutically orally but reached only very low levels in blood and could not enter the CSF. In the laboratory not only would pentosan stop infectivity in cell cultures (also making them sterile of prions) but it could be used to latch onto PrPsc (the prion form of the prion protein) protein and be used for a rapid test. I would need the permissions of the people in the company at the time in order to give you their names. Robinson seemed to feel as if I was pushing things. In fact I was still so shaken up by the whole process of BSE that people were telling me how normal I must feel now! I found her to be a helpful person who was interested in helping. Pentosan had been found to be active in preventing the disease in mice (work from the Edinburgh Research group into scrapie under Christine Farquar), it could be given directly into the brain through a tube and for a period it was the main treatment for vCJD to be advised (see work by John Collinge) [WITN7065025; WITN7065026; WITN7065027]. I had organised the first patient to be treated at the Children's Neurosurgery unit in Liverpool [WITN7065026]. For a short period pentosan was considered the only effective drug until my two drugs put for testing in mice (see d below) took over [WITN7065025; WITN7065026 and WITN7065027].

b. ***What was discussed at the independent meetings you held? Please provide the meeting minutes if you have them.***

35. Independent meetings like the ones suggested were only had with the company of which I was a director with the data on BSE and CJD. There was nothing more than the data from others already published. There were no minutes.

c. ***Please provide any response you wish to, to the allegations made about you by Dr Robinson.***

36. This is accusing me of being a 'loose cannon'. Presumably just not doing what she wanted me to do and having more information than her about the risks involved. It is also accusing me of 'bent on going on a scare mongering exercise'. As I have said I am an ethical doctor and must always tell the patients the truth and carry out action that is in their interests. In a way I might accuse her of doing quite the opposite: hiding information, not informing patients, not carrying out research to prevent disease, and ignoring European and US actions. A media-involved -exercise would always have been a last resort when nothing else was left. As it is I was working on 4 potential treatments: Pentosan given directly into the CSF, orally given drugs: Trimipramine, Fluphenazine [WITN7065004 and WITN7065028], Sirius Red (data never published) and methylene blue. We were also working on a DNA associated diagnostic technique in which the sample was put in a cell on a plate which had already been coated with pentosan, it could then be washed and the PrPsc format prions would remain but there might be some PrPc. Into the cell then was added two antibodies carrying different DNA fragments were added and again washed. Then a DNA fragment would be added directly and would only react with that from the antibodies if close enough to both forms. Then a PCR would run which would go over from one antibody DNA to the other antibody DNA and include the little piece of DNA between [WITN7065005]. This would allow a highly sensitive and specific test. It was being organised with the Veterinary Dept at Dublin University and one in Stockholm. PPS was found to be extremely good and would render cell cultures infection free of prion disease (Microsens) hence it could act as a prophylactic being given with blood as long as much of its pharmacokinetics was known in humans [WITN7065027; WITN7065029; WITN7065030]. So, reading Dr Robinson's letter, it simply looked as if she did not understand my position.

37. Since then both trimipramine, and fluphenazine have shown major action in mice in PrP disease (also a prion-type disease) in genetically modified mice as has methylene blue. This is also true of chlorpromazine [WITN7065028].

15. ***Please see AMAR0012128.***

a. *Do you recall corresponding with Dr Gascoine in writing regarding the infectivity of BSE in blood transfusions?*

38. I don't recall AMAR0012128. I have a feeling that it may just have not been forwarded to me. Of course, many people would be worried if I mentioned blood transfusion and the transfer of a prion disease and he may have been one of them. As it was, I had to just give up with the work that took place with the Leeds Public Health Laboratory under Dr Eglin, and was very pleased if anyone could carry my baton further.

b. *If so, please outline the content of your written correspondence with Dr Gascoine.*

39. I will have sent him a complete list of publications and possibilities for BPL. At the time I had given up my job as the Consultant Medical Microbiologist at Royal Lancaster Infirmary and was working at numerous hospitals around the country as locums to replace any pension that I was going to need.

40. For your information during the period of locums I always continued with research. In Milton Keynes I learned a computer language and wrote out the program to allow people to get personally tested for chlamydia. In Chesterfield I worked on a system of nasal swabs for the diagnosis of Alzheimer's disease (it did not work), and earlier I got two of the compounds for treatment of Alzheimers tested in mice.

c. *Please provide the Inquiry with copies of this written correspondence if you have it.*

41. Please see response to Q16.

16. ***Do you recall corresponding with Dr Gascoine by telephone as he suggests you did in AMAR0012133. Can you recall what was discussed in these phone conversations? Did this inform your knowledge of risk of vCJD transmission via blood/blood products? If so, how?***

42. Dr Gascoine (AMAR0012133) sounds like a reasonable and effective person. I can say that I did not receive things back from him but may simply have sent him the details after meeting him at an international conference on Alzheimer's Disease/CJD. As he said: By this time vCJD has stopped progressing rapidly and to everyone's pleasure and my data on it may have meant little unless a continued use of products for BPL could be found and used.

43. There is one thing in the background: the cases of vCJD started so soon after the BSE epidemic that it is still wondered if there is another vCJD epidemic to come. This would be so if those people with vCJD had a reason to become infected (very high dose, gut problems allowing it to be taken into the blood, eating the tissue when very young etc). By rights transfers from one species to another often pushes up incubation period to 50% of the life expectancy of the recipient. At that rate another epidemic may appear but in 2030. I hope not.

Section 3: Actions and decisions

17. ***Please provide an outline of any proposals, whether accepted or not, that you made to those at the Department of Health or the National Blood Authority (or other organisations) in an effort to protect the blood supply from the risk of vCJD, including but not limited to donor selection and exclusion policies. (You may be assisted by NHBT0063610)***

44. NHBT0063610 does make all this plain. Dr Love made a specific decision to try to have a meeting of people at Skipton House in London.

18. ***Please outline the response that you got to these proposals.***

45. I have no documents of the meeting taking place at this time. We all knew that a risk was there, but we just did not know how to stop it by picking out specific donors. The lecture that I had given made this completely clear. The EU and USA had specific decisions about this which were much easier. I probably did not know that the Department of Health or the National Blood Authority were having a specific meeting (NHBT0063610), they did not contact me for my advice. I tried to contact numerous MPs during this period. The response from the Government had been repeatedly that of MAFF, SEAC and MPs themselves seemed one of the only ways around. This was possibly because I was being contacted by several patients that have been diagnosed with CJD. There was one in Northern Ireland for instance and a doctor from Cheshire. They contacted me not because of diagnosis but because I was a potential source of treatment and information. However, at that time Pentosan did not have adequate data and all I could do was to suggest drugs from a research group in California under Stanley Prusiner in which he tried a wide range of compounds. The two most effective compounds given orally now in animals currently two produced by Microsens one was a tricyclic antidepressive (Trimpramine), and the other a common antipsychotic (Fluphenazine) have now been shown in animals to be active in prion disease in mice probably for a similar reason. They have not been tried in CJD but this would be suggested in animals initially. A single patient with clinical GSS appeared to improve dramatically when given both fluphenazine (see US term flufenazine) and trimipramine.

46. It may have been that I was unclear whether he actually had the disease and offered to use the pentosan test on his blood. As far as I know that never happened and so I do not know what happened. If it did then I could only have explained to the patient that the value of the test would be unclear.

Section 4: Assays and Testing

19. ***In an email to Patricia Hewitt dated 9 June 2004, you mentioned you had contact with a patient who had received implicated blood products from a donor who had died of vCJD. Please outline any other contact you had with patients and what the consensus was with regards to testing and willingness to be tested. (You may wish to refer to: NHBT0017807)***

47. Concerning NHBT0017807, all I can remember is an individual who seems to have claimed to have had a blood transfusion as such and was worried that he might be developing vCJD. Going back to this clearly there was no proven test on blood. Even if a test on him was done it would require controls (probably from abroad), and I do not remember trying to get the academic permission OK for all this although I investigated it. If a test was offered then it could only apply to the individual and it being extremely difficult to interpret. I would have told anyone that. Liz Love said that there would have been a report from the NBS perspective [WITN7065034] and a short review [WITN7065030]. I cannot remember anything further on this.

20. ***The Inquiry understands that you were involved in discussions with the National Blood Service to try and collect blood samples that could be used as controls for a vCJD test for asymptomatic patients. Please set out what the outcome of these discussions were. (You may find NHBT0017804, NHBT0017805, NHBT0017807 and NHBT0017809 of assistance.)***

48. Specifically concerning samples for controls. Concerning NHBT0017804, NHBT0017805, NHBT0017807 and NHBT0017809. Indeed, in order to use UK

controls to assess blood it would be needed to have specific standard groups (which I could suggest) numbers (so make sure of significance), volunteers (not expecting the result from the testing themselves hopefully), Roger Eglin feels that positive as well as negative controls would be needed for technical reasons at least. I am not sure where the 5% positivity rate came from, it was probably a random guess with which to organise the numbers, and if the results were less than 5% it would be able to say so. If you are looking at 100 eggs and expect 5% to be rotten then you must test around 250 eggs in order to give a 5% figure with stats, but if the figure is 2% or less than 1% then this might be clearer. If the figure had been 25% then that would be easily found. The aim of using 5% was to aim at a lowest precise figure but realising that the actual findings could be higher or lower than that and further research carried out to show what the right result actually was.

21. ***The Inquiry has seen correspondence between you and Elizabeth Love at the NBS NHBT0040825 in which you express concern that 'we are sitting on our hands watching the Yanks take the best testing system away'. What was the outcome of this correspondence? What action did you consider the NBS should have taken?***

49. Indeed, that would have been true for me. Concerning NHBT0040825 Liz Love is a researcher and I contacted enormous numbers during this period long ago. I had expressed worry for a long period about BSE and now that we had it in humans (or at least must assume so), we were seeing heavy pressure on researchers. The major lab in Edinburgh (VLA) was being closed and people moved elsewhere for instance. Whereas in the USA, particularly under Prusiner's Nobel Prize there was plenty more work going on. I cannot remember the interaction at all. We were having potential clinical problems but funding was getting lower (except in Collinge's lab), meanwhile we had ideas, and exceptional scientists. When I found out in 1991 that no BSE infected tissue was available to UK labs, it had been sent willingly to the Montana Lab, Oregon Lab, Staten Island Lab, and Prusiner's lab (if he asked). At international

conferences I found that USA studies into BSE and vCJD were taking off. For instance, mink had been fed with BSE tissue in Oregon…no problem spreading there! The only animal they could not infect were hamsters. This should all have been done in the UK and scientists felt chained up to some degree. Sitting on our hands might seem a good description.

50. Clearly in the UK the wide attempts at oral transfer of BSE to other animal species using known quantities of BSE infected brain tissue from cattle. I give a long list of research not being done in my statement to the BSE Inquiry (BSEI0000002) look at section 80.

22. *You requested frozen basal brain tissue in your research for a test in 1999. What was the outcome of this research? (Please see: NHBT0086239)*

51. The research into testing tissues in animals could be diluted and a good idea as to the sensitivity of the test found if animals were involved (which they were not). I did indeed try to get some human tissues but failed. We had Category 3 facilities and systems to disinfect prions (as in the USA). The aim initially was to find what documents they would require from me. This if anything would give again a dilution system to give an indication of the sensitivity of the test but little else. In deer the test was very sensitive and quick. I do not have the results of these. What I do know is that infective material made it to the Microsens Biotechnology Lab in N. London and it was used to grow prions in cell culture and that was used to check drugs for action against the prion culture.

23. *Please provide an outline of any further proposals regarding the development of screening or diagnostic tests, whether accepted or not, that were made by you in an effort to protect the blood supply from the risk of vCJD.*

52. All I have are the contacts who do have any results. As far as I know no blood was ever tested usefully. Everyone is hoping that there are few clinical cases of vCJD and if there are then blood testing may become unnecessary. We will not know this for several years so please contact John Collinge concerning this. I organised a group based in Leeds (at the same Public Health Lab where the chief medical Doctor has got at me so hard [WITN7065003[to see if various dyes could be used in either diagnosis or treatment. The laboratory work in Leeds PH took over the work and did it badly. In the end we did find a dye that was working against the prion (Sirius Red). All I can do now is to quote that nothing was published. Dreadful incompetence. If this was the group that Angela Robinson was referring to then she need not have worried.

53. There are some companies that have used methods of testing blood for Alzheimer's disease. There are more appearing and more accurate and specific monoclonal antibodies for the PrPsc format prion protein. With a new disease in humans needed are a test for it, assessment of the test, data on the distribution in the population of the disease and geographical distribution. You are also needing data on transfer between humans, prophylaxis, and treatment for the condition itself. This is made clear in the Inquiry.

Section 5: Scale of Exposure

The Inquiry seeks to gain an understanding as to the number of people who have been exposed to vCJD and the extent to which this can be assessed and quantified.

Please provide the following:

24. ***A summary of any research studies or papers, reports, recommendations, look back exercises and databases which you have contributed to which***

have addressed the prevalence of the transmission of vCJD in blood and blood products.

54. Almost all my work in this respect has been in reviewing work already carried out. I enclose some of these as references [WITN7065008; WITN7065009]. The best ways are to work out the proportion of the human population that might have had clinical disease and then do retrospective and prospective statistical examination of the figures. When working from the numbers of people that ate infected meat it would be very high (towards 100%, the proportion that may have eaten enough infectivity much lower but incalculable). Using animal studies of a series of species could again give a proportion of them that might be expected to have eaten enough. Last time I looked, all species fed BSE caught it if given enough except hamsters. All mink fed it died of the disease. But this still does not give precise ideas.

25. ***The Inquiry understands that you submitted a proposal to the Medical Research Ethics Committee (NHBT0040827_005) for ethical approval for research into the level of prions in different groups of research participants of different ages. This application was unsuccessful (NHBT0017809). Has this piece of research ever been undertaken to your knowledge?***

55. It did not surprise me that I had tried to apply (although I cannot remember doing this) or that it was turned down. It had become clear to all working in this field and that of Alzheimer's Disease that in the UK it was exceptionally difficult to get ethical approval for science of post mortem samples. This would be because the person donating the sample of blood in patients could have symptoms and in which case their permission was ethically considered unacceptable. It might only be possible in asymptomatic people and from there it would be difficult to interpret. An article was published by the BMJ in about 1995 showing this. I have no idea as to whether any of the work has been done and gradually since then things have become easier for researchers to some

degree. It was as if no research was permitted that was not directly for the improvement of the patient that was involved and others in the community were not of significance. By the way, a relative of a patient that was dying of a familial form of CJD called me and I tried to help how I could. I did offer her a test…but this was to check her genetics and nothing to do with prions in order to see if she was at risk. But I am not on the ethical committee. Some of the blood tests for Alzheimer's disease could potentially be modified for use in CJD.

26. ***Your view on the effectiveness of any look back studies, in particular TMER, to trace recipients of vCJD infected blood and blood products.***

56. Using TMER (The Transfusion Medicine Epidemiology Review) now may actually be useful simply for computerised systems.

27. ***Details of any studies which provide a regional comparison of the prevalence of vCJD in the UK.***

57. Regional comparisons, which I presume is what the research Ethics Committee would have been (I expect) retrospective including regional. The distribution of BSE in the UK was well laid out in documents from the researchers at MAFF and finding that the distribution of vCJD was similar could be followed up on that.

28. ***An outline of the system of recording the cause of death from vCJD infection from blood or blood products in the UK. Please provide your views on the accuracy of information captured about the cause of death and any areas of weakness or failures in this system to investigate, certify or record the cause of death where it was potentially linked to vCJD.***

58. Retrospective post mortem testing may well have been a useful way of doing this but would require the permission of the relatives that remain and no indication by the dying person that they did not want this. In general, this type of testing runs much smoother if the person dying permits it themselves. It would help of course to be able to find out if the person dying of vCJD had received a blood transfusion in the past. The blood donor may well be fully alive and asymptomatic. If a test became possible then this could be offered to them as part of a study perhaps. The documents of this transfusion are already available through the BTS's data and at least has been thorough. Specific areas in the UK became infected with BSE in order. Starting in the South East it seemed to spread to the North and Scotland. People eating beef (particularly children) living in those areas and eating local beef may well have a higher proportion of infected blood tests (if there were any). The major problem is that people in the UK move around the country and experimental controls from abroad may have been useful for statistical value.

Section 6: Other issues

59. Much of what MAFF had been doing seems to have been initially reasonable and necessary. I enclose a list of all the other actions taken by Governments [WITN7065008] to prevent the transfer of the disease. MAFF seemed to be going out of its way to make sure that only its own researchers were working on the subject initially and wanted nobody else to be speaking out. It was at a time when many of the Veterinary Research Stations around the UK were being closed for financial reasons. In the end, for Public Health concerning blood transfusion I felt that if the NBTS was not taking the ethical actions that they should, and other researchers could not, then it was up to me personally to aim at the diagnostics, prophylactics, and would-be treatment that permit blood usage to be as it is. I felt I was treated as valueless. [WITN7065032; WITN7065033]

Table of exhibits:

Reference	Title	Unique ID
1	Dr. Stephen Dealler, Locum Full CV - 2021	**WITN7065002**
2	Letter to Dr Tompkins from Dr. Dealler re: not returning to PHL, Leeds	**WITN7065003**
3	Chung et al (2011) Styryl-Based and Tricyclic Compounds as Potential Anti-Prion Agents	**WITN7065004**
4	PCR system being tested by Microsens for very sensitive testing	**WITN7065005**
5	Infectivity of blood early publications A: Table 1. Attempts to defect infectivity in the blood of animals with scrapie or CJD	**WITN7065006**
6	Infectivity of blood early publications B: Table 2. Attempts to detect infectivity in the blood of humans with CJD	**WITN7065007**
7	Dealler and Kent (1995) BSE: an update on the statistical evidence	**WITN7065008**
8	Dealler (1993) BSE: The potential effect on the Epidemic on the human population	**WITN7065009**
9	Peden et al (2005) Risk of variant CJD by blood transfusion	**WITN7065010**

10	Dealler (1994) A matter for debate: the risk of bovine spongiform encephalopathy to humans posed by blood transfusion in the UK	**WITN7065011**
11	Dealler (2001) Should young UK cattle be considered free of BSE or is it endemic	**WITN7065012**
12	Brown et al (1999) Further studies of blood infectivity in an experimental model of TSE, with an explanation of why blood components do not transmit CJD in humans.	**DHSC0032421_066**
13	Salamat et al (2021) Preclinical transmission of prions by transfusion is influenced by donor genotype and route	**WITN7034017**
14	Brown (1998) Donor pool size and the risk of blood-borne Creutzfeldt-Jakob disease.	**WITN7065015**
15	Andreoletti et al (2012) Highly Efficient Prion Transmission by Blood Transfusion	**PHEN0000612**
16	Hunter (2003) Scrapie and experimental BSE in sheep	**WITN7065017**
17	Note on three separate recent publications are raising concerns about the dangers of the BTS becoming infected with variant CJD	**WITN7065018**
18	Hunter et al (2002) Transmission of prion diseases by blood transfusion	**DHSC0006331_022**
18a	Cervenakova et al (2003) Similar levels of infectivity in the blood of mice infected with human-derived vCJD and GSS strains of TSE	**WITN7065020**
19	Ironside (2006) Variant CJD Risk of transmission by blood transfusion and blood therapy.	**RLIT0000668**

19a	Houston et al (2008) Prion diseases are efficiently transmitted by blood transfusion in sheep.	**RLIT0000713**
20	vCJD: the epidemic that never was? Letter by Stephen Dealler	**WITN7065023**
21	A report on TSE and Transfusion Safety by Subgroup of TSE	**WITN7065024**
22	Todd et al (2000) Intracerebroventricular infusion of pentosan polysulphate in human vCJD	**WITN7065025**
23	Dealler et al (2003) Pentosan polysulphate as a prophylactic and therapeutic agent against prion disease.	**WITN7065026**
24	Pentosan polysulphate potential prophylactic agent against nvCJD by Stephen Dealler. 2002 Submitted to BMJ	**WITN7065027**
25	Stincardini et al (2017) An antipsychotic drug exerts anti-prion effects	**WITN7065028**
26	The pharmacokinetics of pentosan polysulphate (PPS) as a potential prophylactic against transmissible spongiform encephalopathy (TSE) by Stephen Dealler	**WITN7065029**
27	An optimistic future for variant CJD must not be assumed by Stephen Dealler (2002)	**WITN7065030**
27a	Prevention of cross infection in variant Creutzfeldt-Jakob Disease (vCJD) Stephen Dealler (2001) Vol 2, Issue 1, 5-8	**WITN7065031**

28	Bovine spongiform encephalopathy and the public health W. J. Patterson and S. Dealler J. Public Health Medicine (1995) 17, 3, 261-8	**WITN7065032**
28a	Bishop et al (2008) No Major Change in vCJD Agent Strain after Secondary transmission via blood transfusion	**WITN7065033**
29	Technical aspects of the development and validation of tests for vCJD in blood transfusion. Minor, P Vox Sanguinis (2004) 86, 164–1	**WITN7065034**
30	Prion disease; advances in diagnosis and treatment. Dealler S. 2005 Morecambe Bay Medical Journal Jan 1st 276-8	**WITN7065035**

Statement of Truth

I believe that the facts stated in this witness statement are true.

Signed GRO-C

Dated 14.05.2022