IN THE INFECTED BLOOD INQUIRY

WITNESS STATEMENT OF ROBERT JAMES

Introduction

1. I am a 52-year old man with severe haemophilia A. My full name is Robert Magnus Lee James and I live at GRO-C. I was born in 1966 in GRO-C in the Midlands. My diagnosis with severe haemophilia A was made on Christmas Day 1968 at Birmingham Children's hospital, according to my mother. [1]

2. There was no known history of haemophilia in my family and my own diagnosis was an unexpected event for my parents. They informed me that I was diagnosed at Birmingham Children's hospital and thereafter treated at the haemophilia centre in Coventry. My mother remembers the haemophilia diagnosis coming after my tongue would not stop bleeding. This was on or about Christmas Day 1968. I was subsequently diagnosed with HIV and hepatitis C (HCV).

3. I am very familiar with the medical literature, having been a long time treatment activist in the HIV sector, and I currently sit on the guideline writing committee for HIV and viral co-infection for BHIVA and the Clinical Advisory Group for the Haemophilia Society.[2] I am a lecturer in social work at Sussex University having done a PhD in law at Birkbeck College, University of London. I was also one of the team that collected the life histories of people with haemophilia and their families, interviewing some participants and analysing the stories.

4. On the general issues, I have focussed on haemophilia, replacement clotting factor products and the support provided to people with haemophilia and HIV through the Trusts. These are areas that I am very familiar with. For reasons

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[1] See transcript of (C1202/03) David and Sue James within the "Haemophilia Life History Project'" p5 for my father's memory and separately p59 for my mother's although one felt it was an injury to my lip and the other to my tongue.

[2] The British HIV Association (BHIVA) are the NICE approved clinical guideline writers for HIV medicine. I have been on the Haemophilia Society CAG since July 2012.

of length, I have not commented a great deal on the legal cases that I have come across. In any event, law falls within the expertise of the inquiry staff. I am aware many others will make mention of their dealings with the Haemophilia Society and so I have not concentrated on them to a great degree, although they are obviously highly relevant to the supporting of those with haemophilia.[3] I have made mention of contaminants in whole blood transfusions, and potentially plasma transfusions in places. But it is important to note that it is not an area that I have read much about and so whatever information provided in this statement on the contamination of the whole blood supply system should, at best, be considered partial.

5. This statement is divided into two parts. Part 1 gives my perspective on the general medical history and sets the context for how these surrounding issues likely affected me. I have commented on what could have been expected of specialist haemophilia clinicians, and of those operating at higher levels, at various times as to the (potential) contamination of blood products, and the blood supply, within England. Part 2 gives my personal narrative and seeks to set out in some detail, my own experience of haemophilia, how I came to be diagnosed and my experience after being infected by contaminated blood products.

PART 1

Identification of risk

6. There are a number of relevant periods so far as different pathogens and suspected contaminants are concerned. My life has been touched by each of these periods. They are as follows:

    a) Post-war awareness of 'serum hepatitis', more commonly described in the medical literature as 'post-transfusion hepatitis'. This awareness occurred at about the time I was born and refers to the

[3] I was also employed for 6 months by the Haemophilia Society in 1999 to organise a series of meetings for people with haemophilia and HCV to provide them with information about the tests, natural history and treatments for the conditions.

belief that a hepatitis-causing virus was present within a detectable number of blood transfusion recipients, and blood product recipients.

b) The beginning of AIDS in the early 1980s, resulting in my own diagnosis with HIV in April 1985.

c) The hepatitis C risk era, the isolation of this virus and generation of a test and my own diagnosis in 1991.

d) The era of variant CJD: and of my being informed of my 'at risk' status having used British made blood products during the late 1990s.

7. This is the order in which infections would have occurred, rather than the order in which they were necessarily diagnosed, in people with haemophilia. As the test for hepatitis C only appeared in the early 1990s, it can appear as though hepatitis C only later infected blood products. However, in reality, hepatitis C would have been in blood products (and previously some blood transfusions) during the 1970s, prior to HIV first appearing in blood products in about 1980.

8. In my own situation, this means it is likely I would have been infected with hepatitis C in the mid-1970s when I first started using factor concentrates, if not previously with cryo-precipitate. But my own diagnosis with hepatitis C was not until much later, in 1991. This infection order is proven both by the studies of stored blood samples (e.g. at the Royal Free hospital) and by logical reasoning and inference, given the odds of blood donor incidence of hepatitis C, and the batch pool size in the making of factor VIII. Every pathogen identified as an issue for the Inquiry has had its name changed at least once during the relevant history.[4]

9. In this statement, where I claim that something was known, or should have been known by haemophilia centre directors, I will base my assertion from sources in important journals that I would expect were available to most clinicians. (Indeed, not only for those working in haemophilia,[5]) but also from the

[4] There is a short glossary of the names, identifying changes where relevant at the end of this statement.
[5] The journals are Science, Nature, the BMJ, the Lancet, and the New England Medical Journal. In other areas I have identified important papers such as the discovery or isolation of virus or a history piece from other journals such as Blood, JAMA, etc, although some of these are also highly relevant

expressed memories of haemophilia clinicians. Christine Lee, who co-founded the journal *Haemophilia*, noted in her interview for the RCP[6] that before publication of this journal, the papers about haemophilia were spread across a wide range of journals, making it much harder both for clinicians to read all relevant papers but also to identify what articles would have been generally available to clinicians working in the 1970s and 1980s.

The transfusion associated hepatitis risk era

10. This era is important because blood and blood products were known to transmit unknown viruses in the period before HIV. Actions in this time could have changed the number of infections with HIV and hepatitis amongst people with haemophilia.

11. The awareness of liver inflammation after blood transfusions had been suspected for many years.[7] In 1950, the World Health Organisation noted the epidemic and the problems of "conveyance of serum hepatitis by blood transfusions and... human blood derivatives".[8] Publication of details of identification of the first hepatitis virus transmitted through blood came in 1965 and this pathogen is now known as hepatitis B.[9] It was known to be transmissible through blood and plasma products with the first identification of this in 1965.[10] What had actually been identified was a protein on the surface of the virus, known as a viral antigen, now known as the Hepatitis B surface antigen or (HBsAG).[11] It is important to understand that the test for hepatitis B

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and important journals for a haematologist. Some references from other sources have been used to background information commonly known at the time or from later historical perspectives.

[6] Available here: https://rcp.soutron.net/Portal/Default/en-GB/RecordView/Index/62755 ; see transcript p45. She also noted that if an article was not published in the *BMJ*, *Lancet*, or *New England Medical Journal*, it "could kind've get lost" (p45).

[7] Alter, H and Klein, H (2008) "The hazards of blood transfusion in historical perspective", *Blood*. 2008 Oct 1; 112(7): 2617–2626. doi: 10.1182/blood-2008-07-077370 Open access here https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2962447/

[8] Fourth report of the Committee on Programme, adopted at seventh plenary meeting, 25 May 1950 see http://apps.who.int/iris/handle/10665/86234 The note is here http://apps.who.int/iris/bitstream/handle/10665/86234/WHA3.30_eng.pdf?sequence=1&isAllowed=y

[9] Blumberg BS, Alter HJ, Visnich S. 'A "new" antigen in leukemia sera'. JAMA. 1965;191:541–546.

[10] Blumberg BS, Alter HJ, Visnich S. 'A "new" antigen in leukemia sera'. JAMA. 1965;191:541–546.

[11] This was initially named the "red antigen", later the 'Australian Antigen' and finally the hepatitis B surface antigen or HBsAg. The work involved comparison of people with transfusion associated liver disease who were otherwise unconnected and included the blood of an Aboriginal Australian (hence the name Australian antigen).

at this time was a test for a part of a virus rather than a test for the virus. This point has relevance for later aspects of this story because a "a small proportion of individuals"[12] with chronic Hepatitis B do not have this antigen, and a test for a different hepatitis B antigen, the core antigen, had greater potential later on as a proxy marker for non-A non-B hepatitis and/or HIV. This also highlights that one of the continuing themes of risk reduction in blood products rather than risk elimination.

12. The name given to the form of hepatitis B in people who are infectious but test negative for the surface antigen is 'occult hepatitis B' and was only identified some years later.[13] It has been shown that people with occult HBV can transmit the virus through blood products and so anyone with this rare form of chronic hepatitis B could not have been excluded through testing for HBsAG. Using this surface antigen test would therefore significantly reduced the spread of hepatitis B through blood products and transfusions but would not have completely eliminated it. How much the reduction in risk and therefore how many people became infected with hepatitis B using only blood that was HbsAg negative would be dependent on other factors such as the batch pool size and how many different people used each batch of the clotting factor.

13. It is also noteworthy that the place this hepatitis virus was being looked for was in the blood of, among a few other people, "a multiply transfused hemophiliac patient from Brooklyn".[14] This makes clear the expectation that in the early 1960s there was a hepatitis causing agent transmitted through blood and was more likely to be found in those who had received multiple blood transfusions, which at that time included people with haemophilia.

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[12] Nishikawa H, Osaki Y. Clinical Significance of Occult Hepatitis B Infection in Progression of Liver Disease and Carcinogenesis. *J Cancer* 2013; 4(6):473-480. doi:10.7150/jca.6609. Available from http://www.jcancer.org/v04p0473.htm

[13] McMahon B (1998) Chronic carriers of hepatitis B virus who clear hepatitis B surface antigen: Are they really "off the hook"? 28 (1), 265-26. Available at https://aasldpubs.onlinelibrary.wiley.com/doi/epdf/10.1002/hep.510280135

[14] Ibid n7 , Alter, H and Klein, H (2008)

14. In 1972, Alter et al's published research[15] on the impact of the proxy markers for post-transfusion hepatitis being used in the US. This research found that some companies were already using HBsAg as a proxy marker. They calculated that if they had only used HBsAG negative blood, it would have reduced the rate of "post transfusion hepatitis" by 25%. This study also looked at the impact of using blood from paid donors compared to voluntary ones and this found that excluding commercial (paid) donors would have reduced the rate of hepatitis by 70%. Transmissions of hepatitis did occur when using blood from volunteer donors. But there were significantly fewer transmissions of hepatitis, both hepatitis B and non-A non-B hepatitis, than occurred using the blood from paid donors. This study suggests that for whole blood transfusions, the British system of all volunteer donors would be a lower risk option than using blood from paid blood donors. How this risk would then have translated in numbers of people being treated with blood products becoming infected would depend on pool size and the number of people using each batch made. However it does say strongly that of the raw material being used to make the clotting factor products, the blood, was less likely to be infectious under a volunteer donor system.

15. Testing for HBsAg[16] was certainly in use by the English blood transfusion service by 1977[17] and in use by the Scotland Blood Transfusion Service[18] before 1978. This would have had the impact of significantly reducing the risks of HBV after it was introduced, but with the complication of occult HBV it could not have eliminated the risk. The use of this test may also have come with the hope of reducing non-A non-B hepatitis in the blood supply with the test acting

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[15] Alter HJ, Holland PV, Purcell RH, et al. Postransfusion hepatitis after exclusion of the commercial and hepatitis B antigen positive donor. Ann Intern Med. 1972;77:691–699. ALT and AST are called SGPT and SGOT in the article. See page 696 for the calculations of risk reduction. The definition of 'hepatitis' in the paper was repeatedly raised liver function tests including ALT and AST although these were called SGOT and SGPT in the paper.

[16] Hepatitis B surface Antigen - proteins on the surface of the HBV virus. It has since been found this is not the most accurate test for HBV infection with a condition known as 'occult hepatitis B', covered in paragraph 12 of my statement. See also https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3082745/

[17] It is mentioned in Levine PH, McVerry BA, Attock B, Dormandy KN. Health of the intensively treated hemophiliac, with special reference to abnormal liver chemistries and splenomegaly. *Blood* 1977;50:1-9.

[18] C. J. Burrell, S. H. Black, and D. M. Ramsay (1978) Antibody to hepatitis B surface antigen in haemophiliacs on long-term therapy with Scottish factor VIII, Journal of Clinical Pathology, 1978, 31, 309-312

as a proxy marker, although I have seen no explicit statement of this. In the 1980s, an HBV antigen test was specifically proposed as a proxy marker for non-A non-B hepatitis. It was not the surface antigen, but instead, the core antigen. (see below paragraph 24 for details of this).

16. There were outbreaks of HBV amongst recipients of factor concentrates from the very first usage of them in this country.[19] This included some of the participants in the haemophilia and HIV life history project who described infection with hepatitis B from their blood products.[20] The risk of transmission of HBV from large pool blood products was therefore known. There were also strong indications of another hepatitis causing agent that was assumed to be a virus from the very beginning. The article in the Lancet on the outbreak of hepatitis in Bournemouth also notes that measures were being taken at this time by the National Blood Transfusion Service "to reduce the incidence of transfusion hepatitis"[21] which included using single donations or small pools of plasma. There appeared to be an understanding, then that the plasma pool size itself might be important in terms of risk of transfusion hepatitis. The Lancet article also notes that "transfusion hepatitis is a known hazard with large-pool products prepared from volunteer UK donors"[22]. This shows that in the mid-1970s steps were being taken to prevent or at least reduce the transmission of non-A non-B hepatitis through blood and some blood or plasma products by the adoption of a number of approaches in both the UK and US.

17. Donor pool size remained a concern in the 1980s. There is an un-credited BMJ editorial on post-transfusion hepatitis[23] in 1981 which highlights this. It does not, however, come to any conclusion about a suitable pool size. (although it notes two studies with differing results.[24]) Neither of the two studies described,

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[19] E.g. Craske J, Dilling N, Stern D. An outbreak of hepatitis associated with intravenous injection of factor-VIII concentrate. Lancet 1975;ii:221-3

[20] See Haemophilia and HIV Life History Project, C1086/22/01-04 and C1086/12/01-06.

[21] Ibid n20, Craske et al (1975); p222

[22] Ibid n20, Craske et al (1975); The citation for this is personal correspondence with Rosemary Biggs, one of the developers of cryo-precipitate and a major figure in the history of treatment development for people with haemophilia in the UK.

[23] Anon 1981 'Post-transfusion Hepatitis' BMJ, Vol 283 No 6283, pp1-2

[24] The two studies were Stirling ML, Beckett GJ, Percy-Robb IW. Liver function in Edinburgh haemophiliacs: a five-year follow-up. *J Clin Pathol* 1981 ;34 :17-20 and Levine PH, McVerry BA, Attock

contradicted the view that there was a greater risk in large pool products. One of the studies found a significant link between liver abnormalities in recipients and large pool products (factor VIII concentrate) and the other study found a higher incidence in large pool products (factor VIII concentrate) but not significantly higher than in small pool products (cryo-precipitate).[25] The concern about pool size and hepatitis - and later HIV[26] - was repeated in late 1983 by two representatives from Glasgow and West of Scotland Transfusion Service, in a letter proposing the use of cryo-precipitate for people with mild and moderate haemophilia.[27]

18. In 1998, Charles Rizza, who was the chair of the UKHCDO through much of the 1980s, said that in his opinion: "[A]ll new therapies are potentially dangerous, I think, and have to be used with great care". GRO-D

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19. Therefore by the late 1970s it was well established that hepatitis B could be transmitted through blood transfusions and blood products. People who had had multiple transfusions were known to be at high risk of hepatitis B, hence the search for it in the blood of a person with haemophilia. A test was developed and used to identify the hepatitis B surface antigen. This had been introduced in both the English and Scottish Blood Transfusions Services for the purposes of excluding some donors as a means of reducing the risk. By 1975 the English

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B, Dormandy KN. Health of the intensively treated hemophiliac, with special reference to abnormal liver chemistries and splenomegaly. *Blood* 1977;50:1-9.

[25] Studies in n24 Stirling et al (1981) and Levine et al (1977). Both studies compared the risk of liver problems for people using factor concentrates, made from donor pools of about 6,000 litres to people using cryo-precipitate which has a vastly smaller donor pool of the order, I believe, of 10-20 donors. A study on the use of blood products manufactured by the Scottish Blood Transfusion service found a definite link between the use of factor VIII concentrates and liver abnormalities that was not there for those people using cryo-precipitate while a study comparing a US cohort using concentrates with an English cohort using only cryo-precipitate found a higher incidence of liver abnormalities in those using factor VIII concentrates compared to cryo but not significantly higher.

[26] The letter from the Scottish clinicians (see n27) uses the term AIDS rather than HIV because HIV had not been isolated at that time but it is about reducing the risk of the causative agent for AIDS, or HIV.

[27] G S Gabra and R Mitchell (1983) Freeze dried cryoprecipitate and home therapy, J Clin Pathol. 1983 Nov; 36(11): 1321–1322

Blood Transfusion Service had also began to introduce measures to reduce the risk of transfusion hepatitis from blood and plasma transfusions (consistent with the "precautionary principle"). A shift from very small donor pools, less than 10, to a donor pool size of many thousands was understood as exposing the recipient to "a higher risk of contracting transfusion hepatitis".[28] It was unclear if there was a threshold above which this risk did not increase or if the risk continued to escalate as the donor pool size increased further. Commercial factor products from the US using paid blood donors were shown to have a higher risk of transfusion hepatitis than volunteer blood donors but the risk existed in both types of product. Cryo-precipitate was known to transmit transfusion hepatitis but appeared to do so at a lower rate than factor VIII concentrate. There was confusion about what "transfusion hepatitis" was since it appeared to be both hepatitis B and additionally one or more other viruses.

20. The fact that there was another hepatitis causing agent in factor VIII and transfused blood, though, was not in doubt by the late 1970s. In 1975, on top of the Bournemouth infections of HBV, there were also a number of people with haemophilia at the centre with a form of hepatitis that was not HBV and described as "non-B hepatitis."[29] Another paper that year was published in the New England Medical Journal about transfusion associated hepatitis being caused by another agent in blood[30] with further acknowledgment of the presence of "non-B" hepatitis among those receiving commercially produced factor VIII in the UK in 1978.[31] Since this agent was found to be neither HAV nor HBV, it became known as "non-A non-B" hepatitis. However, it was very clear that this was a transmissible agent, that it was transmissible through blood

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[28] Ibid n20; Craske J, Dilling N, Stern D. An outbreak of hepatitis associated with intravenous injection of factor-VIII concentrate. Lancet 1975;ii:221-3. See p221.

[29] Ibid n20; Craske J, Dilling N, Stern D. An outbreak of hepatitis associated with intravenous injection of factor-VIII concentrate. Lancet 1975;ii:221-3

[30] Feinstone SM, Kapikian AZ, Purcell RH, Alter HJ, Holland PV. (1975) 'Transfusion-associated hepatitis not due to viral hepatitis type A or B'. *N Engl J Med.* 1975 Apr 10;292(15):767-70. DOI: 10.1056/NEJM197504102921502

[31] Craske J, Kirk P, Cohen B, Vandervelde EM. (1978) Commercial factor VIII associated hepatitis, 1974-75, in the United Kingdom: a retrospective survey. J Hyg (Lond). 1978 Jun;80(3):327-36.

transfusions and blood products and that it could infect chimpanzees as well as humans.[32]

The non-A non-B hepatitis risk era

21. David Tyrell, who was part of the Biological Products Subcomittee of the Committee of the Safety of Medicine during the period before detection of hepatitis C, stated in the Wellcome witness seminar in 1998: "it was part of the documentation that every ampoule should have a warning that there was a risk of infection".[33] There was greater surveillance of the use of factor concentrate from this period onwards. My father designed a form to record each injection administered at home to provide a record for Taunton Hospital. I later discovered that this form had been slightly amended and was given to other families using the Taunton haemophilia centre to record their own usage of factor VIII. This means that I have records of my injections and the batch numbers of each product from 1977 to 1990, although there are a couple of missing sheets. These are appended to this statement.

22. The diagnosis of non-A non-B was largely through negative testing: hepatitis identified from deranged liver function tests and then a lack of HAV and HBV and other causes of hepatitis. However the results of these liver function tests would go up and down and changes could be caused by other factors. This meant that a single blood test result could not identify a case of non-A non-B. A series of blood test results were required, extending over a period of time. The diagnosis was therefore not a positive identification of a specific disease but a reactive assumption as the only option remaining when others had been excluded.[34] This meant that it was going to be difficult, if not impossible, to accurately identify all donors with non-A non-B hepatitis without a test for the specific causative agent, or agents.

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[32] See Alter HJ, Purcell RH, Holland PV, Popper H. Transmissible agent in "non-A, non-B" hepatitis. *Lancet*. 1978;1:459–463 and Tabor E, Gerety RJ, Drucker JA, et al. Transmission of non-A, non-B hepatitis from man to chimpanzee. *Lancet*. 1978;1:463–465.

[33] This seems to have been variously called the Committee on Safety of Medicines Sub-committee on Biological Products' or the 'CSM Sub-committee on Biologicals'. See Witness seminar 1998, p70 for quote. Wellcome Trust Witness Seminar on 10 Feb 1998; HAEMOPHILIA :RECENT HISTORY OF CLINICAL MANAGEMENT. Available at http://www.histmodbiomed.org/sites/default/files/44826.pdf

[34] See almost any of the papers cited before this as such as Feinstone et al (1975) at n31, Levine (1977) n24, Craske et al (1978) n31, Stirling et al (1981) n24, for their description of non-A non-B hepatitis.

23. By the early 1980s there was growing awareness that testing could result in an inaccurate diagnosis of non-A non-B hepatitis in the case of occult HBV.[35] People with this particular type of the hepatitis B virus do not produce the surface antigen and so appear negative on this test. However, since other HBV specific tests had been developed by this point occult hepatitis B could be identified using other markers. The main impact of this was to indicate that people testing negative to the HBsAg test could not be assumed to be free of hepatitis B. This meant that the test used by blood transfusion services in the early 1980s was a risk reduction measure rather than a risk elimination measure in the case of hepatitis B.

24. There were a number of proposals in 1981 of a proxy marker[36] for non-A non-B hepatitis that could be used by blood transfusion services to reduce the risk and number of infections. These predicted various reductions (29-43%) in the number of infections in recipients of whole blood transfusions but it is difficult to know what reduction, if any, this would have had on the infectivity of large-scale multi-donor blood products. The HBV antigen being used was not the surface antigen described above but the core antigen of HBV, sometimes abbreviated as HBcAg having been identified as a superior proxy marker.[37] Further papers were published in journals on this issue. However, while these journals were sometimes prestigious in their own field, it would be hard to expect a haemophilia clinician to study and so to know of these potentialities (although one would hope that those working within a blood transfusion service would have seen these papers.[38]) These papers also highlight the serious impact of

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[35] It was first reported in 1979, see Tabor E, Hoofnagle JH, Smallwood LA, Drucker JA, Pineda-Tamondong GC, Ni LY, Greenwalt TJ, Barker LF, Gerety RJ. Studies of donors who transmit posttransfusion hepatitis. Transfusion. 1979;19:725–731.

[36] These were raised ALT scores or a specific hepatitis B antigen, the surface antibody or the core antigen. The logic for each was that a raised ALT score identified that a person's liver was managing some kind of inflammation and the core antibody test identified someone who had previously had one hepatitis virus and was therefore likely to have had a higher likelihood of having another hepatitis virus. See refs in n37 and n38.

[37] Koziol DE, Holland PV, Alling DW, et al. Antibody to hepatitis B core antigen as a paradoxical marker for non-A, non-B hepatitis agents in donated blood. *Ann Intern Med.* 1986;104:488–495

[38] Aach RD, Szmuness W, Mosley JW, Hollinger FB, Kahn RA, Stevens CE, Edwards VM, Werch J. 'Serum alanine aminotransferase of donors in relation to the risk of non-A, non-B hepatitis in recipients: the transfusion-transmitted viruses study' *N Engl J Med.* 1981 Apr 23;304(17):989-94 DOI: 10.1056/NEJM198104233041701. See also Alter HJ, Purcell RH, Holland PV, Alling DW, Koziol DE.

non-A non-B hepatitis and the need for a proxy marker in the absence of test for the specific virus. These papers also make it clear that the causative agent for non-A non-B hepatitis was considered to be a virus and the search for it was in the field of virology and there was no suggestion that it was any other type of pathogen. The use of chimpanzees to test the safety of factor VIII for being free from hepatitis B and non-A non-B hepatitis was also a known possibility, albeit one that was thought not possible on a major manufacturing basis.[39]

25. This demonstrates a number of matters relevant to blood transfusion services and haemophilia clinicians, prior to the infection of people with HIV through factor concentrates. It was acknowledged that there was an unknown, or unidentified, virus in blood products. For the English blood transfusion service there had been the earlier adoption of the "precautionary principle" (whether so described at the time or not) to reduce the risk of viral hepatitis through blood transfusions but little evidence of any further use of this principle in factor VIII concentrate prescribing.[40] Factor VIII concentrate was used because of the ease it offered and greater control over haemophilia management.[41] There had been discussions in the medical literature about the use of proxy markers for non-A non-B hepatitis to reduce transmissions. It was not about eliminating transmissions of non-A non-B but reducing the numbers. It had also been shown that there was the potential use of chimpanzees to test the safety of factor concentrate products for non-A non-B hepatitis and therefore also for the removal of this in the future heat-treated concentrates.

'The relationship of donor transaminase (ALT) to recipient hepatitis: Impact on blood transfusion services' *JAMA*. 1981;246:630–634. and Koziol et al 1986, n37.

[39] Letter from Arthur Bloom to all Haemophilia Centre Directors, 11th January 1982.

[40] E.g. Use of cryo-precipitate, using products only from volunteer blood donors rather than US commercial factor VIII products.

[41] E.g. As Craske et al (1975) puts it "Factor VIII activity is much greater in freeze-dried concentrates, which can be given by syringe and needle instead of intravenous drip, are stable and have a much more predictable action than cryo-precipitate. They do not produce pyrexia and urticaria which occasionally occur with cryo-precipitate. They have made home treatment more practicable, and major operations on haemophiliac patients much easier." Ibid n20 for the full reference. N.B. The use of an intravenous drip for cryo only ever happened while I was an in-patient and because the injecting of a medicine into a vein required a doctor so a drip was just a practical way of giving medicines that allowed doctors to see other patients while the infusion happened. Cryo was always 'given by syringe and needle' to me by my parents or a haematologist at the haemophilia centre so this 'advantage' seems misplaced to me.

26. Haemophilia clinicians knew by 1980 that there was at least one agent that was universally assumed to be a virus that caused liver problems in recipients of factor concentrates. There was some evidence that factor concentrates, which were inevitably made from large pools of donors including volunteer UK donors, were riskier in terms of viral transmission than cryo-precipitate and even the evidence that did not find a significant difference in risk between large and small pool products did not contradict that finding. This large pool and small pool distinction seems to have been well accepted by some eminent clinicians in haemophilia. In writing to his colleagues about the then new heat-treated concentrates in 1982, Professor A Bloom states that the clearest way to assess how much the risk of viral hepatitis is reduced is through a study using these products in "patients needing treatment who have not previously exposed to large pool concentrates".[42] It is also noticeable that he says that the best way to test these new heat treated products as being truly "hepatitis-safe" was in these patients, those either not exposed to any blood products or only to small pool products such as cryo-prepcipitate.

27. While it is quite hard to expect a haematologist to have read a journal specific to another speciality, by 1980[43] it appeared to be clear to gastroenterologists, that non-A non-B hepatitis was a serious condition potentially leading to cirrhosis amongst those diagnosed with it. This does not seem to be the perception among those consultants treating people with haemophilia in the UK or the US. A 1983 review of the use of clotting factor concentrates by a US haemophilia consultant, Aledort,[44] mentions hepatitis only once and that is in relation to international political pressure rather than as a concern for a clinician. In a letter in 2007, Aledort makes the assertion that he wrongly believed non-A non-B hepatitis was a mild or transitory condition and that a new agent in the blood supply was unimaginable. Aledort writes "At the time, the accompanying exposure to hepatitis B and non-A non-B (C) appeared to be an acceptable risk. Only in retrospect have we recognized that the risks are far greater than we

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[42] Letter from Arthur Bloom to all Haemophilia Centre Directors, 11th January 1982.
[43] E.g. Koretz RL, Stone O, Gitnick GL (1980) 'The long-term course of non-A, non-B post-transfusion hepatitis' *Gastroenterology*. 1980 Nov;79(5 Pt 1):893-8.
[44] Aledort, L.M., Goodnight, S.H., Rao, A.V. et al. La Ricerca Clin. Lab. (1983) 13: 33. https://doi.org/10.1007/BF02904743

could predict. No one could have anticipated an additional risk, HIV, which entered the blood supply at the end of the 1970s".[45] The letter came in response to an Italian haemophilia clinicians' remembrances in the previous issue that says much the same thing.[46]

28. Throughout the 1970s and 1980s, papers looking at haemophilia treatment highlight the risk of non-A non-B in factor concentrates. This knowledge, however, does not appear to have become common amongst people with haemophilia or their families as demonstrated by the lack of awareness among those who were interviewed for the life history projects about viral hepatitis prior to infections. It is certainly possible that these risks were explained but if so it appears not to have been done in a manner that was memorable.

29. Reading a paper in the BMJ by Peter Jones from September 1985, [47] after it was known that many people had been infected with the AIDS virus, it is apparent that there had been a significant change in view as to the level of infection of non-A non-B. He certainly no longer saw this as a *risk* of infection, but a certainty.

30. "Several thousand donations are required per batch of concentrate. Thus each patient using a concentrate is exposed to material from many donors every time he injects himself, and viral contamination of the source plasma through individual contaminated donors has become a well-recognised hazard in the management of haemophilia. All patients using multi-donor material can be expected to have been repeatedly subject to hepatitis B, hepatitis non-A non-B and the human parvovirus."[48] A similar finding was seen by Charlie Hay in a

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[45] Aledort, LM *J Thromb Haemost* 2004; 2: 521.
[46] Mannucci PM. AIDS, hepatitis and hemophilia in the 1980s: memoirs from an insider. *J Thromb Haemost* 2003; 1: 2065–9.
[47] Jones et al (1985) AIDS and haemophilia: morbidity and mortality in a well defined population. J BMJ, 09/14/1985, Vol.291(6497), pp.695-699. Note this comes up as 'morbidity and morality' in the BMJ database, presumably a typo rather than a comment on views about AIDS at the time.
[48] Jones et al AIDS and haemophilia: morbidity and mortality in a well defined population *Lancet* 14 September 1985 **291**; 695-9

paper from 1985 [49] whose title expresses it: "Progressive liver disease in haemophilia: an understated problem". [50]

31. It would also seem relevant that any statement about the seriousness of non-A non-B to patients should have been made by a specialist in liver conditions or after consultation with gastroenterologists/hepatologists unless a haemophilia clinician was well versed in this literature. Any description of non-A non-B hepatitis as a 'mild disease' after this time would, at best, be a very thin description, failing to acknowledge that at least a minority of people with it would progress to cirrhosis even if it remained less serious in the majority of people with it.

The AIDS risk era

32. As is now well-known, the first indication of a new severe immune disfunction syndrome was noted as a result of an unusually high demand for pentamidine to treat *pneumocystis carinii* pneumonia in 5 gay men in Los Angeles hospitals.[51] At the time, this was an extremely unusual condition in the US. This phenomenon was published in the now famous issue of Morbidity and Mortality Weekly Report (MMWR) of June 5th 1981.[52] The newly named AIDS syndrome was then identified in people with haemophilia in the US in two further MMWR reports in July and December 1982[53] with factor concentrates suggested as the potential route of transmission.

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[49] Hay et al, 'Progressive liver disease in haemophilia: an understated problem?', Lancet, 1985;i:1495-98

[50] This article is odd in critiquing the view that chronic persistent hepatitis is "benign and non-progressive" but the reference to that view is a 1979 paper and there were a lot of papers about non-A non-Bin the meantime. According to a quick pubmed search there were 445 published papers that used the phrase "non-A non-B hepatitis" during this time and a further 70 with "non-B hepatitis". 101 of these 445 were in what they describe as 'core clinical journals' with 13 in the Lancet alone so the view of benign and non-progressive may already have changed.

[51] Now known as *pneumocystis jiroveci*

[52] CDC. Pneumocystis pneumonia---Los Angeles. MMWR 1981;30:250--2. Available on line here https://www.cdc.gov/mmwr/preview/mmwrhtml/june_5.htm

[53] CDC (1982) Epidemiologic Notes and Reports Pneumocystis carinii Pneumonia among Persons with Hemophilia A, *MMWR* July 16, 1982 / 31(27);365-7 and CDC (1982) Update on Acquired Immune Deficiency Syndrome (AIDS) among Patients with Hemophilia A, *MMWR*, December 10, 1982 / 31(48);644-6,652.

33. It is unlikely that UK haemophilia consultants would be reading internal US reports; but they may have heard of them through contacts with US colleagues. A report about AIDS and haemophilia was published in the *New England Medical Journal* in January 1983[54] and this would have been available to British medics. In September 1983, the news of the first death of a British person with haemophilia and AIDS at the Cardiff centre was communicated to haemophilia society members in 'Haemofact 2'[55] and published in the *Lancet* in November 1983.[56] According to the *Lancet* paper, AIDS was suspected by the clinicians in May 1983 and the man met the CDC diagnosis of AIDS in August, dying a few days later.

34. In addition, there were the similarities between the signs of immune systems dysfunction among people diagnosed with AIDS and people with haemophilia that had not shown symptoms of AIDS. This was the reversal of the CD4-CD8[57] ratios consistently found in people with AIDS (e.g. Anon 1983a[58]). Before the end of 1983, this ratio reversal was also seen disproportionately in "at risk" gay men (Kornfeld et al 1982[59]), compared to men not perceived as at risk; and in haemophilia cohorts in Cleveland, Milwaukee and Washington in the US and in Newcastle (UK) (Menitove et al 1983, Lederman et al 1983, Luban et al 1983, Jones 1983[60]). The Lancet report about the first AIDS case and death in a person with haemophilia in Cardiff also found this person had this reversed CD4-CD8 ratio.

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[54] Desforges (1983) AIDS and preventative treatment in haemophilia, *New England Medical Journal*, 308 (2), 94-95, Jan 13th

[55] I have a photocopy of this having donated my original copies to the Haemophilia Society after the Archer Inquiry.

[56] Daly and Scott (1983) Fatal Aids In A Haemophiliac In The UK, Lancet, 1190-1191, Nov 19th

[57] CD4 and CD8 cells have a number of names in these publications including T-helper and T-suppressor in MMWR articles; OKT4 and OKT8 in Kornfeld, Luban and Lederman (1982); T4 and T8 in Menitove (1983) see below n60 for full refs. These are different names for the same cells.

[58] Anon (1983a) Acquired Immunodeficiency Syndrome, Lancet, 162-164, Jan 22nd

[59] Kornfeld et al (1982) T lymphocyte subpopulations in homosexual men, Lancet 307 (12) 729-731, Sept.16th

[60] Menitove et al (1983) T-Lymphocyte subpopulations in with classic haemophilia treated with cryoprecitptate and lyophilized concentrate, Lancet 308 (2) 83-85, Jan 13th; Luban et al (1983) Altered Distribution Of T-Lymphocyte Subpopulations In Children And Adolescents With Haemophilia, Lancet 503-505, 308, Mar 5th; Lederman et al (1983) Impaired T cell immunity in patients with classic hemophilia, Lancet 308 (2) 79-83, Jan 13th; Jones, P (1983) Altered Immunology in Haemophilia, Lancet, 120-121, Jan 15th

35. Alongside this had been a diagnosis of AIDS in at least seven people with haemophilia in the US (see para 33 MMWR Dec 1982 and Desforges Jan 1983). Further, there had been the specific linkage of the first AIDS case to the use of commercial factor concentrates, in the context of haemophilia, in the UK (Daly and Scott 1983), followed by a second UK case. Yet none of this evidence was considered sufficient for a senior haemophilia consultant and author of one of the CD4-CD8 ratios reversal in haemophiliacs report to propose changes. There was no recommendation of any changes to the provision of factor VIII concentrates, commercial and/or NHS produced, or greater consideration of cryo in April 1983.[61] Nor was it considered sufficient to change the "watching and waiting" approach adopted at the time, apparently because it did not constitute conclusive proof that the blood products were contaminated. Haemofact 2, a newsletter produced by the Haemophilia Society (which would have reflected the view of their Medical Board) and sent to members in September 1983, repeated this unchanged view, stating that: "THE ADVANTAGES OF TREATMENT FAR OUTWEIGH ANY POSSIBLE RISK."

36. If these features had been understood as a warning signal of contamination of all blood products, or US commercial blood products, a number of actions would have been possible to reduce this risk of further AIDS cases. (a move away from US products or a return to cryo-precipitate, for example). Many people would already have been infected with HIV at this time but at the time haemophilia clinicians believed that everyone without symptoms did not have the causative agent of AIDS. This means a risk reduction intervention at this time would have been understood as preventing new cases of AIDS in people with haemophilia. This is because of the lack of understanding of the disease as having a symptomless phase, prior to an AIDS defining illness appearing. At this time, the notion of latency - that people were already infected with AIDS causing agent but were not expressing symptoms - is not expressed, implied or logically possible in the way the papers published by haemophilia specialists

[61] Anon (1983b) ACQUIRED IMMUNODEFICIENCY IN HAEMOPHILIA, The Lancet April 2nd 1983. Although this editorial is unsigned, Virginia Berridge, states that it was written by Peter Jones, see *AIDS in the UK: the making of policy 1981-1984*, OUP, p39.

were written. The issues of Haemofact, written by haemophilia specialists, also assume this notion. These papers understand the condition as one that appears to cause symptoms soon after infection rather than to be symptomless, or detectable only by its impact on blood test results.

37. Concepts of latency, precursor states and incubation were discussed by the MRC Working Party on AIDS, which included Professor Bloom and Dr R Tedder, in October 1983 according to the minutes of the meeting, see para 42. This does not seem to have led to any change in understanding of the natural history of AIDS. The disregard of the possibility of an apparently symptomless period for a pathogen transmitted in blood products by haemophilia clinicians, now seems unusual, when they would have managed cases of 'transfusion hepatitis'. This was recognised as a chronic condition, although with uncertainty about its severity

38. There is a suggestion in the Penrose Inquiry final report, para 11.139-11.142, that some haemophilia consultants were sceptical that AIDS in haemophilia was caused by a transmissible agent and that the correct cause was the cumulative effect of taking blood products. However, even if this had been the case, the idea could not provide support to promote the continued usage of blood products. Even if there was disagreement among haemophilia clinicians about the exact causes of AIDS in people with haemophilia, everyone saw it as relating to the use of blood products.

39. The words of Peter Jones at the Wellcome witness seminar on haemophilia in 1998 seem to confirm the assumption made by haemophilia clinicians at the time that there was no incubation period with HIV. He felt this was what had led them to believe that AIDS would affect only "1 in a thousand" people with haemophilia and then to publicise that belief. In the Wellcome Trust witness seminar, when comparing vCJD with HIV, he said that vCJD had "a very long incubation period which is what caught us out with 'the one in a thousand' at

the beginning of HIV".[62] It is very hard to read about this belief of a "one in a thousand" risk. The infamous haemofact leaflet, authored by Christine Lee, repeated this assertion, thereby further disseminating that inaccuracy. The incidence at the time of the haemofact leaflet was indeed 1 in a thousand, 3 people with haemophilia having been diagnosed with AIDS (and it was thought there were 3000 people with haemophilia A), but a 4th case would have changed the incidence from 1 in a thousand to 1 in 750; 3 more cases and it would have stood at 1 in 500. At this point, pushing a figure of 1 in a thousand when we had already reached that incidence level logically suggests a belief that there would be no more cases which seems an extraordinary assumption.

40. I can only see this as a form of denial about what was happening: that the "nice" patients being seen by haemophilia doctors just could not have this disease of society's pariahs. No action was taken and no recommendations for action were made beyond the continual restatement in Haemophilia Society literature to continue using factor VIII concentrates.[63] This may not simply have been a reflection of disbelief by clinicians but also "antagonism" and "no leadership whatsoever from central government or the Department of Health"[64] at the time, as described by Peter Jones at the Wellcome witness seminar. At this seminar in 1998, Peter Jones also said that "[W]ith HIV, we realized that we had got a problem after the description of the first cases in 1981". This view does not appear in his published papers covering the period 1981 and 1984, nor in any of the Haemofacts sent out by the Haemophilia Society during this time. This suggests either that he chose not to express these views publicly at the time or that the perspective from 1998 influenced his recollection of the past.

41. This repeated exhortation that not taking Factor posed a greater risk to those of us with haemophilia than AIDS was sadly misplaced and became - and remains - a source of much anger amongst people with haemophilia.[65] The

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[62] Wellcome Trust Witness Seminar on 10 Feb 1998; HAEMOPHILIA :RECENT HISTORY OF CLINICAL MANAGEMENT p75. Available at http://www.histmodbiomed.org/sites/default/files/44826.pdf

[63] See issues of Haemofact from this time and the Haemophilia Society newsletter, The Bulletin

[64] Peter Jones at the Wellcome witness seminar, p65. Available at http://www.histmodbiomed.org/sites/default/files/44826.pdf

[65] Quite a number of the life history interviews talk about this anger.

infection coming through the treatment also induced huge guilt amongst mothers who had injected their sons with the treatment - and the virus – that then went on to kill their sons.[66]

42. As noted in para 37 this concept of a latency period for HIV was considered in the minutes of the MRC Working Party meeting on AIDS in October 1983 with a discussion about the clinical features of AIDS, the problems of blood tests in the "precursor states" for AIDS and the "varying and considerable period of incubation (1 to 4 years)" of the disease. These matters did not appear in the literature provided to people with haemophilia, unless the exhortation to continue taking factor concentrates was predicated on the assumption that we had already been infected and therefore there was no point in protecting us. I would struggle to believe this. It is strange, as noted in the Penrose inquiry,[67] that there is no reference to the work by Francois Barré-Sinoussi[68] at this meeting - especially as the Wellcome witness seminar suggests that there was a strong link between UK and French clinicians working in haemophilia.[69] It would be surprising if the French clinicians in haemophilia were unaware of Francois Barré-Sinoussi and Luc Montaigner's work on AIDS. Further, Arthur Bloom who was at this MRC meeting published the results of a survey of European haemophilia[70] in which he thanked MJ Larrieu, a French haematologist centre director, and simultaneously published a paper with Larrieu on prevalence of von Willebrands disease,[71] suggesting he had good contacts there.

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[66] E.g. see 'HIV in the Family: an oral history of parents, partners and children of those with haemophilia and HIV' interviews C1202/09 p45 and C1202/13 p6.

[67] See the preliminary report at 8.55 available here http://www.penroseinquiry.org.uk/preliminary-report/chapter-8/

[68] Barré-Sinoussi et al (1983) 'Isolation of a T-lymphotropic retrovirus from a patient at risk for acquired immune deficiency syndrome (AIDS)'; Science 1983; 220:868-871

[69] Note the comments by Peter Jones on page 67 and Christine Lee on 68. Available at http://www.histmodbiomed.org/sites/default/files/44826.pdf

[70] Bloom A (19840 ACQUIRED IMMUNODEFICIENCY SYNDROME AND OTHER POSSIBLE IMMUNOLOGICAL DISORDERS IN EUROPEAN HAEMOPHILIACS, *Lancet* June 30, 1984, pp1452-5

[71] Mannucci PM, Bloom AL, Larrieu MJ, Nilsson IM, West RR. (1984) Atherosclerosis and von Willebrand factor: I. Prevalence of severe von Willebrand's disease in western Europe and Israel. *Br J Haematol* 1984; 57: 163–9

43. In July 1983, Lord Glenarthur in the House of Lords stated that the Government was considering the publication of a leaflet identifying the "circumstances in which blood donations should be avoided" because of the concern that "AIDS" could be transmitted through blood and blood products.[72] This suggests a return to a precautionary approach was in the minds of politicians, and presumably the blood transfusions services, by seeking to restrict entry of the virus into the blood supply. There does not appear to have been the same concern about the use of existing non-heat-treated large pool products in patients.[73] The approach to make treatment safer for people with haemophilia was therefore not to change the treatment to a small pool donor product (cryo-preciptate) but to wait until donor deferral measures were introduced by the blood transfusion service and wait for further technological solutions through heat-treating those blood products.

44. Lord Glenarthur also stated that by April 1983 there were only 14 confirmed cases of AIDS in the UK of which at least 2 must have been people with haemophilia as described above. Although these numbers are miniscule, the fact that 14% of the cases concerned people with haemophilia feels, perhaps with the benefit of too much hindsight, another worrying signal. The Italian clinician, Manucci, in his remembrances of this period, says he offered patients a choice to return to cryo-precipitate but that as he felt the chance of AIDS affecting his patients was small and passed this understanding on, "very few elected to discontinue" factor concentrates. Oddly, he repeats the notion that at the time, about 1983, it was well-known that factor concentrates had a negative effect on the immune system without any recognition that these abnormalities were almost certainly due to HIV. The deleterious effect on the immune system of people with haemophilia in the two papers cited[74] are identical to the effects experienced by someone with HIV infection. As Peter Jones pointed out in 1985: "since 1983 it has become increasingly likely that

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[72] See https://hansard.parliament.uk/Lords/1983-07-14/debates/99960c0b-853c-4798-9c0d-b36b916028e1/AidsIncidenceAndControl

[73] Strangely in the 14 July 1983 House of Lords debate, Baroness Masham asks about the risks of transmission through cryo-precipitate although she may have confused this with factor concentrate as she mentions "blood compounds". Lord Glenarthur however had not heard of cryo and so could not answer.

[74] The Lederman 1983 and Menitove 1983 NEJM articles in notes 60

immunological disturbances in haemophilia are caused by the aetiological agent for AIDS rather than any alloantigenic response to denatured protein".[75] What is interesting is not how few but that any of Dr Manucci patients opted to change their treatment.

45. In the Wellcome witness seminar, Peter Jones lamented what he saw as the initial waste of money on AIDS at this time. He said that what was needed was to take every haemophilia centre director and the "the small number of other people working with the early groups affected by HIV"[76] to visit the US to learn about treatment and care for people with AIDS. Given that volunteer Haemophilia Society representatives were publicly expressing their view that they "did not want to share a platform with any other 'At Risk' Group"[77] in 1985, it is unclear whether such an idea would have found favour with the trustees of the society. The attitude of those volunteers illustrates the extent of the prejudice towards gay people and drug users at the time, including by people with haemophilia - a community of people affected by the same stigmatised condition.

The hepatitis C risk era

46. Although hepatitis C was only isolated in 1989[78] and an antibody test patented the same year,[79] the testing had a controversial beginning. The introduction of the HCV test was not immediate and came over the next few years. With an hepatitis C prevalence of 1 in 2000 in blood donors in 1991,[80] and factor VIII

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[75] Jones et al AIDS and haemophilia: morbidity and mortality in a well defined population *Lancet* 14 September 1985 **291**; 695-9

[76] Peter Jones at the Wellcome witness seminar, p66. Available at http://www.histmodbiomed.org/sites/default/files/44826.pdf

[77] UKHCDO meetings as late as October 1985

[78] Qui-Lim Choo, George Kuo, Amy J. Weiner, Lacy R. Overby, Daniel W. Bradley and Michael Houghton (1989) "Isolation of a cDNA Clone Derived from a Blood-Borne Non-A, Non-B Viral Hepatitis Genome" *Science*, Vol. 244, No. 4902 (Apr. 21, 1989), pp. 359-362.

[79] G. Kuo, Q.-L. Choo, H. J. Alter, G. L. Gitnick, A. G. Redeker, R. H. Purcell, T. Miyamura, J. L. Dienstag, M. J. Alter, C. E. Stevens, G. E. Tegtmeier, F. Bonino, M. Colombo, W.-S. Lee, C. Kuo, K. Berger, J. R. Shuster, L. R. Overby, D. W. Bradley and M. Houghton (1989) "An Assay for Circulating Antibodies to a Major Etiologic Virus of Human Non-A, Non-B Hepatitis" *Science*, Vol. 244, No. 4902 (Apr. 21, 1989), pp. 362-364

[80] I have not been able to find the published figure after the introduction of hepatitis C antibody testing in 1991 but the prevalence is cited as 1 in 2000 blood donors in this paper; W L Irving, K R Neal, J C E Underwood, P N Simmonds, V James, on behalf of Trent Regional Hepatitis C Virus Study Group (1994) 'Chronic hepatitis in United Kingdom blood donors infected with hepatitis C' *BMJ* 1994;308:695-6.

concentrates using larger pools than the 2000-6000 litres[81] in 1975 to produce a batch, it became mathematically inevitable that there would be hepatitis C in each batch made during the 1980s.

47. Treatment for this condition remained arduous and often unsuccessful for people with it until the appearance of Direct Acting Anti-virals (DAAs) from about 2014 onwards. The rationing of this treatment by NHS England,[82] despite approval from NICE that the treatments met the cost-effective criteria, required for a technology approval[83] delayed the availability of effective hepatitis C treatment to many people with haemophilia in England. In Scotland and Wales the medicines were available to all.

The vCJD risk era

48. This really begins in the mid-1990s when it became apparent that variant CJD had been transmitted through blood transfusions. This led to the risk assessments by the Department of Health in 2003[84] and 2009[85] to assess if it was likely that people with haemophilia were at risk of contracting this prion through blood products made from the blood of UK donors. The later report came after an autopsy of a person with haemophilia in which the prion was found in their spleen, but not in their brain[86] I received the standard department of health letter regarding my risk status but as far as I can tell it has had no impact on my health. I have been asked about CJD risk whenever I have been

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[81] Factor concentrates generally had a significantly larger donor pool than 2000 per batch. The 1975 Craske paper (ibid n20) on the hepatitis outbreak in people using concentrates describes the commercial concentrates using pools of 2,000 to 6,000 litres, approximately 4,400 to 13,200 pints. As pool sizes increases it reduces the cost of manufacturing but it becomes inevitable at least one, and probably multiple, donors will have hepatitis C. It would only need a single donor to contaminate the whole batch as even a single donor will have millions of copies of the virus circulating in their blood.

[82] See https://www.theguardian.com/society/2016/jul/28/nhs-abandoning-thousands-by-rationing-hepatitis-c-drugs

[83] See here for a list of the Technology Appraisals of various DAA combinations https://www.nice.org.uk/guidance/conditions-and-diseases/infections/hepatitis/products?ProductType=Guidance&Status=Published

[84] DNV (2003): Risk Assessment of exposure to vCJD infectivity in blood and blood products. Final report for Department of Health, February 2003

[85] Bennett, P and Ball, J (2009) vCJD Risk Assessment Calculations for a Patient with Multiple Routes of Exposure, Department of Health, June 2009 BMJ 2009;338:b705

[86] See Eaton, L (2009) Haemophilia patient had variant CJD agent in spleen, BMJ 2009;338:b705 and for a fuller description Peden et al (2010) Variant CJD infection in the spleen of a neurologically asymptomatic UK adult patient with haemophilia, Haemophilia. 2010 Mar;16(2):296-304. doi: 10.1111/j.1365-2516.2009.02181

admitted to hospital for an operation in the last 10 years, but answering yes or no appears to make no difference to the care I have received. The two recent cases of CJD, although appearing to be sporadic CJD rather than variant CJD, are of some concern regarding blood products, even if neither had haemophilia A.[87] It is possible these two recent cases are simply part of the natural distribution of this condition and unrelated to factor concentrates though the 2009 case seems very much related to the taking of factor VIII. The Department of Health's most recent risk assessments still place me in a category in some, but confusingly not all, of the parts of the current transmission risk guidance because I have undoubtedly had a blood component from more than 300 donors.[88]

49. In October 1999 leucodepletion was started for blood donated in the UK. This was the process of removing the white cells from the blood in the belief that it was those cells that would harbour the prion that caused v CJD[89]. This was a return to the adoption of the precautionary principle - it was unknown then whether it would be effective in reducing the risk of transmission. It is quite likely that this had no impact on the risk but it was a measure that was introduced in an effort to reduce risk. It is a shame that it took so many viruses and deaths for this learning to have an effect on blood product manufacturing. However the Safety of Blood, Tissues and Organs (SaBTO) is currently engaging with stakeholders[90] about relaxing some of the recommendations

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[87] Urwin et al (2017) Sporadic Creutzfeldt-Jakob Disease in 2 Plasma Product Recipients, United Kingdom, Emerg Infect Dis. 2017 Jun;23(6). Available at https://wwwnc.cdc.gov/eid/article/23/6/16-1884_article

[88] Minimise transmission risk of CJD and vCJD in healthcare settings - NHS Guidance (2012 – updated 2017) Transmissible Spongiform Encephalopathy Agents: Safe Working and the Prevention of Infection: Annex J Available at https://www.gov.uk/government/publications/guidance-from-the-acdp-tse-risk-management-subgroup-formerly-tse-working-group [accessed 29/08/18]
See section J12 includes "People who have received blood or blood components from 300 or more donors since 1990 (additional information on how this group is defined can be found in the ACDP guidance" but it is none of the questions in J1 that are supposed to be asked.

[89] See CMO (2003) 'Health Check The CMO Annual Report 2003, p30. Available at https://webarchive.nationalarchives.gov.uk/20130105045555/http://www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@en/documents/digitalasset/dh_4086811.pdf

[90] SaBTO (2019) Information sheet for stakeholder engagement - Importation of plasma and use of apheresis platelets as risk reduction measures for variant Creutzfeldt-Jakob Disease, DH and SaBTO (2019) Stakeholder Engagement FAQ, DH. The Haemophilia Society is a stakeholder of this group and as a member of the Clinical Advisory Group I have had sight of it.

about the use of plasma in the light of a further risk assessment of future infections.

PART 2

My personal story

50. I believe I did once receive a blood transfusion as a haemophilia treatment but I have no memory of this as I would have been a baby at the time. I do remember using cryo-precipitate as a treatment for my haemophilia, initially travelling to hospital two or three times a week for it to be administered by a doctor. Later on, my parents were taught to inject me and I started on home treatment with cryo-precipitate, keeping it stored in a freezer. I was allergic to cryo-precipitate but the rash it caused was fairly easily controlled by using an antihistamine, piriton.

51. Preparation of cryo involved warming up frozen plastic bags of yellow and orange gunk into which you would jab with a needle and draw out the contents in a syringe. There was not much in each bag of cryo: 5-10ml, I think, and it would take 7 or 8 bags to fill a 50ml syringe of cryo which was the amount administered when I was a child.

52. In 1976 my parents moved from GRO-C to GRO-C and I moved from the haemophilia centre in Coventry to the one in Gloucester. As this was a small centre, I was also registered with the nearest comprehensive care centre at the Bristol Royal Infirmary. I was only in GRO-C for a year before my parents moved again to Somerset in September 1977 and I registered at the haemophilia centre at Musgrove Park Hospital, Taunton. I have no memory of any serious events relating to haemophilia during my short time at Gloucester Hospital.

53. I had started on the concentrates by the time I was being seen at Musgrove Park Hospital, Taunton.[91] The concentrate, while often taking an age for the

[91] See my own treatment records for injections starting from January 1977

powder to dissolve, was simpler to prepare. It required only the addition of sterile water to it. This water was supplied in a bottle and you stabbed a needle into each bottle and drew up the water into a syringe and then squirted it into the bottle with the powder in it. It was also much more portable as it did not need to be frozen and so could be carried in a cool box and came in small boxes rather than multiple frozen plastic bags. In essence, the factor VIII concentrates were easier to store, only needing to be in a fridge and simpler, if not necessarily quicker, to prepare.

54. I remember that I was informed by my doctors about the risk of hepatitis B in factor VIII. I do not have a date for this but the risk was clear to me by my teenage years, while I was living in Somerset. My memory is that that infections had happened in the past and that although measures had been taken to prevent it (presumably HBsAg testing of donors), the risk should not be assumed to have been eliminated. Hepatitis B was also seen as a disease that, while sometimes serious on infection, the body was able to manage and destroy the virus in the majority of cases, once a person had become infected and cleared the virus they would thereafter become immune. A small number of people, often those infected through vertical transmission, went on to be "chronic carriers" and were able to infect others. I think the product information at the time also stated that it should not be assumed to be free from hepatitis B. (It would be worth the Inquiry ascertaining if the information stated that that referred to hepatitis B or hepatitis viruses in general.)

55. I have a memory of having jaundice during another Christmas spent in Coventry hospital[92] as a child with a black eye aged 7 or 8. It could potentially have been a sero-conversion illness for hepatitis C although, if so, the knowledge of it appears to have been lost as a result of my parents moving around the country. Otherwise it may be that I have confused this illness with something else while in hospital.[93]

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[92] According to my parents I "spent alternate Christmases in hospital" apparently to avoid seeing my grandmother, transcript of (C1202/03) David and Sue James within the 'Haemophilia Life History Project', p81.

[93] I had a number of allergic reactions to cryo-precipitate and other medications and it is possible this was the cause of jaundice, a fever, and other symptoms while I was in hospital.

56. In 1977 when moving from the comprehensive care centre of Bristol Royal Infirmary to Taunton hospital haemophilia centre, I was described as "negative for Hb s Ag, and has had abnormal liver function tests in the past although no clinical history of hepatitis".[94] The clinical details referring to a later period, when I moved to GRO-C to study at University, do not mention previous abnormalities of liver function tests: merely that I was "Australian Antigen [HBsAg] negative".[95] Frustratingly, the haematologist at Swansea hospital appeared somewhat uncommunicative and there was barely any correspondence between him and Taunton over the four years. When a summary was requested, it was the haemophilia nurse that responded.[96]

57. I do not appear to have had a diagnosis of non-A non-B hepatitis despite evidence of previous liver function test abnormalities at a number of points prior to my diagnosis with hepatitis C. The 1977 introductory letter from Bristol to Taunton haemophilia centre refers to previous abnormal liver function tests and[97] copies of my blood test results between 1979 and 1982 show I had liver function test results above the normal range on multiple occasions.[98] Blood test results are intermittent or illegible in my notes after this point but by 1989 my liver function test results were again beyond the normal range.[99] However, at no point was I referred to a gastroenterologist or hepatologist despite these abnormal liver function test results.

58. During the early 1980s I first became aware of AIDS and through the media of the link with haemophilia products and blood transfusions. I was not a member of the Haemophilia Society before this as my parents had found it an irrelevant

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[94] Letter dated 15th August 1977 from G Scott (Clinical haematologist at BRI) to M Phillips (Taunton).
[95] Letter dated 21 January 1986 from E Thompson (Clinical assistant in haematology, Taunton) to GRO-D (Morriston hospital, Swansea)
[96] Request letter dated 8 June 1987 from Taunton and response dated 7 July 1987. Much of the further correspondence appears to have been with the haemophilia nurses.
[97] See the introductory letter from G Scott (Clinical haematologist at BRI) to M Phillips (Taunton) 15th Aug 1977.
[98] Blood test results Chemistry reports for Somerset Area Pathology Service, between 10 Aug 1979 and 7 Jun 1982, 8 out of the 9 readable scores have the AST above the normal range given (4-15) and varying from 23 to 70. One is in the normal range on 4 Aug 1980.
[99] Chemistry Laboratory Report, 28 Jun 1989, scores it at 65 with a reported normal range of 1-40.

organisation. It was not a bad organisation but it failed to provide anything beyond that which they could discover from the hospital staff and it appeared solely interested in raising money and in not spending it. My initial knowledge about the possibility therefore came from the media rather than the Haemophilia Society. I particularly remember the World in Action and Horizon programmes about AIDS. I re-joined the Society at about this time in order to receive more information about the risk of AIDS.

59. Prior to my joining/re-joining the Haemophilia Society, David Watters had been employed there. During the time of his leadership, he had a major impact on the organisation which I feel was a very positive one in terms of promoting advocacy and the involvement of people with haemophilia. He was also not homophobic, unlike many of the volunteers and members of the society at this time, which was important to be able to work with other HIV organisations.

My diagnoses

60. In April 1985, when I was 18, I was diagnosed with HIV. It appears that I had the blood test done in March or possibly earlier at Taunton hospital[100]. I had arranged an appointment the following day because of some bleeding problem and Dr Liz Thompson informed me that I had been tested and they had found HTLV-III antibodies in my blood and at that point I was the only one in Taunton hospital who had been infected. She said that she did not know what it meant and that it could be that I was infected with the AIDS virus; that I was immune to the AIDS virus or that nothing might happen. By chance, my father had come in with me to the appointment (he must have not been needed at work) and so I did not have to tell my parents since he found out at the same time as me and he told my mother. Some years later, my father told me that she, Dr Thompson, told him she had been up all night with worry about how she was going to tell me.

[100] My own medical notes. I was told on 25th April 1985 according to my own notes and confirmed by a letter sent to my GP in my medical notes.

61. The following day I contacted the Terrence Higgins Trust to ask what it meant being diagnosed with the HTLV-III antibodies. They quickly said it did not mean I was immune to the virus but that it was uncertain how many people with this result would progress to AIDS or how quickly. I remember this phone call because of "the gulp". I have heard this from people diagnosed early in the epidemic as well and it was an audible swallow by the volunteer on the other end of the phone line when I said I had been diagnosed with HTLV-III. There were not many people diagnosed with the virus at this point in the UK and few with haemophilia were comfortable contacting what they saw as a gay organisation such as THT. THT received lots of abusive calls, as well as calls from people worried about AIDS who had not been tested. This meant somewhat paradoxically that it was unusual for its volunteers to speak to someone actually diagnosed with the virus. The number of people diagnosed with HIV by the end of 1985 in England Wales was 2543, of which about 700 were reported as infection through clotting factors.[101]

62. As I was in the middle of my A levels, I concentrated on them at this time, telling a few friends, but not many. However, when I went to Swansea University, I had problems with the Physics department who were scared around my haemophilia. There had been a lot of publicity concerning a young boy with haemophilia and HIV at a school in Hampshire[102]. The other parents had initially removed their children. This demonstrates that the link between haemophilia and AIDS was prominent in the press at this point. The lecturer was uncomfortable about having a person with haemophilia in the department in case he had AIDS. The senior management of the University was very supportive of me, however, and after changing courses, I did a psychology degree instead because the lecturers in that department were not bothered by my haemophilia. Following the initial problems I had when joining, I met a lecturer who was involved in AIDS campaigning, Paul Heritage, and went on to enjoy my time at Swansea. I was generally quite open about having the virus during my time there. My openness was in contrast to the majority of people

[101] Taylor and Mortimer (1989) HIV infection in England and Wales: a changing pattern. Epidem. Inf. (1989), 102, 355-359

[102] See https://www.youtube.com/watch?v=Sv-licbDFsY for a news report at the time

with HIV, including those infected through blood products, or other transmission routes.

63. During my time at Swansea, I contacted Jonathan Cooper[103] at the Haemophilia Society after he had informed my clinician in Taunton that the Society was looking for members to do media work campaigning for HIV compensation and to promote the legal case. In the late 1980s and early 1990s, together with Jonathan, I spoke at a number of conferences including those arranged by the Terrence Higgins Trust and the National AIDS Trust. I also formed a self-help group in Swansea with an ex-heroin user and a gay man that I had met as a volunteer at Swansea Drugs Project and through the local AIDS helpline. On leaving University in 1989 and moving nearer to London, I did media work in the campaign for compensation for HIV infection, appearing in a number of national newspapers[104] and on television.

64. In 1990 or 1991 I visited a friend who was studying at Cardiff University and spent a day in their University library researching hepatitis C among people with haemophilia using their medical journals. It was obvious from my research that everyone with severe haemophilia had this new virus so I assumed I must have it. However, this was still the time when there was no effective HIV treatment and it was seen as always terminal and so I gave it little thought. I was certain I would die from AIDS long before I had liver failure. I was diagnosed with hepatitis C sometime in the early 1990s but I do not remember the year, although it was most likely in 1991.

65. Now that my own treatment is a recombinant form of factor VIII containing no donated human products my risk of future human infections should be negligible.

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[103] He is now working at Doughty Street Chambers.

[104] The Sunday Times 15th October 1989 and the Mail on Sunday 22nd July 1990 are a couple of examples that I have copies of.

Warnings about risk

66. My memory, as set out, above is that I was aware of transmission of hepatitis B through concentrates at the time and that this was a known issue. I first became aware of AIDS when it started making the news in this country, in particular a Horizon programme about it called 'killer in the village'.[105] After that I was aware that AIDS had been diagnosed in people with haemophilia although my memory is that the concern always related to US blood products and at that point I was on NHS produced blood products.[106] The last US blood product I took before my diagnosis was on November 27th 1981.[107]

67. I have no memory of being offered a choice to return to cryo-precipitate, or of my parents being offered this choice, as a risk reduction method at any time before my own diagnosis with HIV (see paragraph 60). I cannot recall any one in the life history projects - either those with haemophilia or the parents, partners, etc - speaking of having this choice, but it may only have been made to people with mild or moderate haemophilia. It would be interesting to see if those people with mild and moderate haemophilia who were later diagnosed with hepatitis C but avoided HIV were treated with cryo-precipitate at any point between 1982 and 1986. This would demonstrate that precautions were being taken. I am not convinced that the offer of a choice or implementation of this risk reduction strategy actually happened.

68. After diagnosis with HIV,[108] I learned much more about the condition and being at a University fairly soon after diagnosis, I had access to a few medical journals in the University library. I also became involved in setting up a local support

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[105] See, https://www.bbc.co.uk/iplayer/episode/p01z2lbp/horizon-19821983-killer-in-the-village

[106] See my treatment records for this period which identify BPL concentrates as my treatment that year.

[107] This was Hemofil made by Baxter. My own treatment records of the period show that I had taken products from multiple manufacturers including US, European and UK made factor VIII up to my HIV diagnosis (Kryobulin, made by Immuno in Vienna, Hemofil made by Baxter in the US and NHS made concentrate in England. After my diagnosis I took Koate produced by Cutter, and products by Alpha as well.

[108] I was informed it was HTLV-III, although this is unfair on the actual discoverer of the virus, Francois Barre-Sinoussi, who together with Luc Montaigner named it LAV. A much more sensible name as it bears little relation to the conditions caused by HTLV-I and HTLV-II. See Barré-Sinoussi et al (1983) 'Isolation of a T-lymphotropic retrovirus from a patient at risk for acquired immune deficiency syndrome (AIDS)'; Science 1983; 220:868-871

group for people with HIV in Swansea and later in larger national HIV organisations and also the Birchgrove group that met in Cardiff.

69. In the early 1990s I became more aware about hepatitis C and, through a friend, I copied a number of articles about hepatitis C and haemophilia from which I learnt that a diagnosis of hepatitis C was inevitable in every person with severe haemophilia. I therefore have almost no memory of the actual diagnosis. It just felt like a confirmation of the obvious and inevitable and it seemed at the time that I was still far more likely to die of AIDS before the hepatitis made me ill so the diagnosis had no impact.

70. In the late 1990s I became aware of the risks of vCJD within blood and consequently the potential for transmission through blood products. I did receive one of the standard letters from the Department of Health about my increased risk related to having taken blood products made from blood donated with in the UK. I have not been informed that I have used treatments that were known to have been made from blood donated by an individual who later developed vCJD but as I moved fairly frequently and changed centres in the important period - including one centre that refused to be part of the UKHCDO database and so did not send treatment or patient details to the UKHCDO –I would have been difficult to notify.

UK Self-Sufficiency

71. It is obviously impossible for me to know what impact self-sufficiency would have had in England or the UK as a whole. However, some papers provide indications of the potential effect of self-sufficiency on the rates of HIV and hepatitis C infection. Belgium and the Netherlands were both self-sufficient in factor concentrate production and they had significantly lower rates of HIV infection (5% and 24%). Scotland was also self-sufficient and had an infection rate with HIV of 16%. In England there was a reported 10% rate of infection for

those using the NHS made factor VIII concentrate compared to 60% for those with severe haemophilia overall.[109]

72. The picture is more complex with HCV because the infections most likely occurred from the very start of the use of factor concentrates in the UK. This means that the point at which any self-sufficiency was introduced would be relevant, as would be the fact that it was not possible to test for the causative agent until the 1990s. However, a calculation could be made from the prevalence rates of blood donors for the purposes of estimating the likelihood of batches of factor concentrate being infected. The incidence of 1 in 2000 in English blood donors in 1991 was noted above.[110] The figures cited in the Penrose preliminary report[111] gave the incidence in blood donors in Scotland in 1992 at a lower rate, of about 9 per 100,000. This Scottish figure equates to about 1 in 11,000 donations. Over 12,000 pints of blood were needed to make the early batches of factor VIII,[112] rising to 40,000 pints later. People with severe haemophilia, and in some case mild haemophilia, also had multiple transfusions using different batches of factor VIII concentrate.

The Alliance House organisations

73. There was an initial campaign by the Haemophilia Society for recompense alongside the legal case from 1985. As a response to this campaign and media coverage of the legal developments, the government announced in 1987 that it would give £10 million pounds to the Haemophilia Society for a hardship fund for people with haemophilia who had contracted HIV. This Haemophilia Society decided it did not wish to run this hardship fund within its own organisation and so the Macfarlane Trust (MFT) was set up.[113] The chair of the Haemophilia

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[109] Figures from Rosendaal et al (1988) AIDS and Haemophilia , Haemostasis 18: 73-82 (1988), Melbye et al, (1984) HTLV-III seropositivity in European haemophiliacs exposed to Factor VIII concentrate imported from the USA. Lancet. 1984 Dec 22;2(8417-8418):1444-6 and Zaman et al (1986) Prevalence of antibody to HTLV-III in haemophiliacs in the United Kingdom, BMJ 293 19 July 1986, 175-176.

[110] See paragraph 46 with the figure cited in W L Irving, K R Neal, J C E Underwood, P N Simmonds, V James, on behalf of Trent Regional Hepatitis C Virus Study Group (1994) 'Chronic hepatitis in United Kingdom blood donors infected with hepatitis C' *BMJ* 1994;308:695-6

[111] Preliminary report at 3.112

[112] See Craske et al 1975 at n20.

[113] The description by the Department of Work and Pensions of the MFT is that it was "for the relief of poverty and distress", see paragraph 4.5 here, https://www.gov.uk/government/publications/recovery-

Society, Alan Tanner, became the MFT chair and a board of trustees was chosen including one or two people with haemophilia and HIV although they must have left the board before the end of the year.[114] There were a small numbers of staff in the MFT who administered the grant giving process and a committee that met monthly to approve or reject the applications for one off payments. This charity provided one-off grants for people who were sick and a hardship fund for those in poverty. The one-off grants were based around specific areas such as nursing care, mobility needs, relief of stress and education although the organisation maintained a high level of "discretion" over what they would and would not provide a grant for.[115]

74. I was a registrant of the MFT from the start of the organisation and it was at about this time I became involved in the Haemophilia Society's campaign for recompense for the infections.[116] As a student at the time, I was deemed to be in financial hardship and so I received a regular monthly payment from the beginning. This was the maximum of £20 per week. This was approved in May 1989 but backdated to November 1987.[117] It was necessary to back date payments because it had taken many months for the organisation to be set up. On top of this, I was able to apply for one off grants relating to my health needs. From October 1990, regular monthly payments became standard for all registrants with a minimum rate of £15 per week regardless of their income.[118]

75. From 1989 the MFT staff and trustees held meetings with registrants at haemophilia centres or haemophilia groups[119] and I had contact with Tudur Williams, the original manager of the MFT, and a number of the trustees of the MFT. These meetings were ostensibly to outline what types of things could be

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of-benefits-and-or-lump-sum-payments-and-nhs-charges-technical-guidance/recovery-of-benefits-and-lump-sum-payments-and-nhs-charges-technical-guidance#the-law.

[114] The Trust maintained for many years it was illegal for them to have a person with haemophilia and HIV as a trustee if they received any money from the trust. A 'winter payment' was approved for all registrants in November 1989 so any such trustees would have been asked to step down at that point, see MFT letter, 'Special Winter Payment' 22nd Nov 1989.

[115] E.g. See MFT newsletter 5.

[116] See letter from my clinician at Musgrove Park Hospital dated 4 August 1988.

[117] Letter to me from MFT 2 May 1989

[118] See MFT Newsletter 5

[119] E.g. see MFT newsletter 2

claimed for through the grants process. At most of the meetings I attended a significant amount of anger was directed at the MFT for its slow decision-making processes and for what appeared to be inconsistencies in what they would give grants for and what they would not. The chair of the MFT then, Alan Tanner, was very calm but appeared to understand all the anger and frustration as part of a grieving process arising out of HIV infection. He would not concede that there were problems with the MFTs systems and processes. The relationship between the MFT and some registrants was therefore strained from the beginning.

76. After the formation of the MFT, there were also significant changes at the Haemophilia Society with Jonathan Cooper and the long-time General Secretary[120] David Watters both leaving. There was some kind of restructuring with no replacement to fill Jonathan's position in campaigning around HIV and new titles were adopted for incoming staff. The de facto Chief Executive was Graham Barker with whom I had a polite but difficult relationship resulting from the fact that it felt to me that all the support for those with haemophilia and HIV was now being left to the MFT, with the Haemophilia Society focussing on services for families with newly diagnosed haemophilia with some work on hepatitis C. It was at this time (1988-2004) that Birchgrove was at its most active in terms of conferences, producing newsletter and lobbying the MFT and the Haemophilia Society to better meet the needs of those infected with HIV, their families and widows. Much of this activity is described in the newsletters, series 1 (1994-2000) and series 2 (2001 -2005).

77. There are a number of reports and consultations produced by Birchgrove, sometimes in partnership with the Haemophilia Society or commissioned by the MFT[121] concerning the processes and procedures adopted by the MFT, and a

[120] This was the equivalent of the Chief Executive role.

[121] E.g. Living with Haemophilia and HIV – a discussion document from November 1993, the PAS report preliminary report in 1994 and final report in 1995; Keeping it in the Family – a study on behalf of Birchgrove and the Haemophilia Society in 1995; The Voice of the Registrants' by GRO-A in October 1998. And finally the setting up of the partnership group to allow some level of registrant feedback mechanism to the MFT and finally registrant trustees. The reports are available to the inquiry if needed. There were further strategic reviews commissioned or undertaken by the Trust after this such as in 1998.

gradual acceptance of involvement of registrants within the working of the MFT because of problems with these policies and procedures.

78. On top of the MFT grants 2 ex gratia payments were announced at different times, November 1989 and May 1991. In February 1990 I received the first ex-gratia payment of £20,000 through the MFT .[122] The delay in the processing related to the need for the MFT to set up a completely new trust because the payment of a fixed amount to all was not permitted by the MFT deeds. The Macfarlane Special Payment Trust (MSPT) was therefore set up to ensure we could be legally given this payment.

79. In May 1991 I received the ex-gratia payment that settled the litigation against the government. I received £32,000 in this settlement. Yet again there was a delay between the announcement in December 1990 and the distribution of the monies because it could not be paid through the MFT or the MSPT. This necessitated the setting up of a third trust, the Macfarlane Special Payments (No2) Trust or MSPT2.[123] This problem of setting up a specific trust for those in very specific circumstances - while excluding people in very similar circumstances - has dogged the distribution of funds to people infected with HIV or hepatitis C through blood or blood products. [124]

80. For example the Eileen Trust had to be set up for people infected with HIV through blood transfusions because the trust deed of the MFT excluded them, although the Eileen Trust appeared to have the same rules, grant processes and was run by the same staff team.

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[122] See MFT letter Feb 1990.
[123] See MFT Newsletter and MFT letter dated
[124] A brief look at the current DWP website on benefit recovery gives a list of the number of schemes and funds set up including the MFT, MFT Special Payments Trust, the MFT Special Payments Trust (no. 2), the Eileen Trust, the Skipton Fund, the Caxton Foundation, to get over the legal hurdles of distributing the money, see paragraph 4.5 here, https://www.gov.uk/government/publications/recovery-of-benefits-and-or-lump-sum-payments-and-nhs-charges-technical-guidance/recovery-of-benefits-and-lump-sum-payments-and-nhs-charges-technical-guidance#the-law

81. By 1994, after repeated concerns about their systems and processes being expressed by registrants (including in a discussion paper written by the wife of a registrant following a Birchgrove conference[125]) the MFT commissioned a company to look at its customer service. Public Attitude Surveys Ltd produced a report known as the PAS Report.[126] This was critiqued both at Birchgrove events and in the Birchgrove magazine for its failure to discuss the causes of the tension between MFT and a proportion of the registrants. GRO-A, the Birchgrove Chair at the time, said to me: "it is not about how long it takes them to answer the phone, it is what they say when they answer it".[127].

82. After some years, the MFT produced guidelines for registrants about which single grant applications would be approved and which would not. The MFT also later published periodic newsletters.[128] Over a number of years and possibly because of the problems of approving and refusing single grants, the MFT moved towards a system of regular monthly payments for all, reducing the single grants available over time.

83. See the table below:

Trust/fund	MFT	MFET Ltd	Eileen	Skipton	Caxton
Route of transmission	Blood products only	Blood and blood product	Blood	Blood and blood product	Blood and blood product
For those infected with which virus	HIV only	HIV only	HIV only	Was for those with hepatitis C regardless	hepatitis C but excluding those with

[125] Living with Haemophilia and HIV was written by GRO-A, wife of GRO-A. GRO-A died in 1999 and GRO-A in 2001.
[126] The title is actually Macfarlane Trust Membership Survey – final report June 1995. My copy is available to the inquiry.
[127] Birchgrove Magazine Issue 4 (1994 first series). GRO-A died in 1997.
[128] I have the 2009 edition but there a number before this date. I believe older versions I had may be with materials I donated to the Wellcome Library for them to consider if they wished to add them to their collection. I also have copies of some of the MFT newsletters as well as those referred to in this statement.

				of HIV status	HIV and hepatitis C
Single grants / hardship	Yes	No	Yes	No	Yes
Monthly payments	No unless poor	Yes	No unless poor	Only if very ill (stage 2)	No
Lump sums paid	No	Yes	No	Yes	No

84. The MFT was the main charity for many years and went through four chief executives. The first, Tudur Williams, did not have this title but his successors, Ann Hithersay, Martin Harvey and Jan Barlow, all adopted the title.

85. From about 1999 the MFT started to run its own residential events, initially in partnership with the Haemophilia Society and later on its own. This allowed Birchgrove to wind down this service. One of my fellow CPs later became the person who organised these residential support events for people who used the MFT, including specific events for partners, widows and families as well as registrants. During my time as chair of Birchgrove, we concentrated on completing the monument celebrating the lives of those infected with HIV through blood products at Stratton Wood in Swindon and on the setting up of a website. After our official unveiling of the stone, the wood has become a place of pilgrimage for many of the families affected by HIV.

86. During the time of Martin Harvey (2003-2010) and under the Chair, Peter Stevens, a mechanism was established in an effort to improve communication and allow some dialogue between interested registrants of the MFT and the management. This was called the "partnership group" and really began because of the continuing perception of problems with the management of the organisations and inconsistency in decision-making about grants. One of our goals was the re-establishment of registrants on the Board. I had made a number of representations in the past about this because registrants were, after

the introduction of the first regular monthly payments, excluded from being trustees. At an early meeting I proposed that I be paid as a trustee the same amount of my monthly payments and not to accept them as MFT payments. Alan Tanner refused this option and asked what skills I had. This resulted in an angry response from my father about my professional qualifications (a degree in psychology and a masters in social work) and the experience of chairing a number of voluntary groups. Later when the Haemophilia Society was able to nominate trustees to the MFT, I proposed to Karin Pappenheim, the then Chief Executive, that a ballot be held amongst registrants and the winner be chosen as the Haemophilia Society trustee. This was again declined. Eventually after many years of lobbying, it was finally accepted that appointing registrants as trustees would not be in breach of charity law, just as we had been arguing for over a decade. A registrant trustee, GRO-A was appointed in 2001.[129]This reflected the typical inertia of the MFT, and reflected the general difficulties in gaining support from the Haemophilia Society in pressing the MFT for changes.

87. A number of notable concerns were dealt with through these forums about communication. Although it did not involve me, the taking of a stake within a registrants' house to avoid them becoming homeless seemed a sensible use of the MFT resources. The MFT would pay the remaining part or a portion of the remaining mortgage for these properties in order to prevent the family from being evicted. However, speaking to one of the widows affected, it seems that the MFT viewed this as an investment and so they held a proportion of the equity in the house. The value of the house had increased significantly. This meant that the equity owned by MFT had increased in value, making a profit for them. The MFT chose to do this rather than provide her with a loan with a fair rate of interest to be repaid on sale of the property which would have allowed her to move.

88. When the payments for hepatitis C were announced in August 2003, the MFT was eventually asked to manage the distribution of its single payments. There was initial concern that people co-infected with HIV and HCV would be excluded

[129] MFT Newsletter 48, Spring 2001

from these payments (as had been one of the original campaign aims of the Haemophilia Society) and negotiations around this are described in the MFT newsletter at the time.[130] Eventually, it was decided following pressure from the MFT on ministers[131] that the hepatitis C payments would be available to everyone infected with hepatitis C through blood or blood products. The Skipton Fund was then housed in the same building as the MFT with the addition of a single new member of staff to process the one or two one-off payments to beneficiaries.[132]

89. I received the Skipton Fund 'Payment No 1' in September 2004 because I had HCV[133] but not 'Payment No 2' as I did not develop cirrhosis of the liver during the time this fund existed. The Skipton Fund was set up as a new fund because the MFT, MSPT, MSPT2 and the Eileen Trust were not able to be used to give money to people with hepatitis C who did not have HIV. Finally the Caxton Foundation was set up in 2011 as a hardship fund for those people with hepatitis C from NHS treatments because the Skipton Fund did not have a hardship element to it. I did not receive any money from the Caxton Foundation as those with HIV were excluded from its hardship fund on the assumption they could access the MFT. The MFT had by this time moved almost exclusively to monthly payments rather than one off grants. Over the lifetime of the MFT I generally avoided applying for one off grants as I disliked the system so intensely and preferred to get the money for such items through employment.

90. An initial issue with the Skipton Fund concerned the exclusion of the HCV payment to widows or bereaved partners whose husbands or partners had died prior to a specific date.[134] Eventually this was changed and I supported a widow[135] in applying for the first payment . Her husband, GRO-A had severe haemophilia A and had died of AIDS in the early 1990s. I believed he would have had hepatitis C. This story appeared to me typical of the way all of these

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[130] MFT Newsletter Winter 2003, p1.
[131] Personal communication from Peter Stevens
[132] Nick Fish
[133] Remittance Advice from Skipton Fund dated 10 Sep 2004
[134] 29th August 2003, see HQ Issue 3, Spring 2004, p2.
[135] I am happy to provide her name to the inquiry but as I am no longer in touch with her I would ask that it remain confidential.

schemes operated. They made it overly difficult to apply for funds from them, rather than supporting and facilitating the distribution of the monies they held in trust for those of us infected and bereaved. GRO-A's widow collected her husband's medical notes from St Thomas' hospital[136] and I went through them for her to save her the sadness that this would cause and because of my knowledge around HCV.[137] GRO-A had never been tested for hepatitis C[138] but did have some raised liver function test results and having received hundreds of batches of factor VIII, it seemed to me inconceivable he would not have been infected with HCV. She and I collected this material together and applied for the payment for her. I also spoke to the Skipton Fund worker in Alliance House, Nick Fish, about the progress of this application. He informed me that because there was no positive test result for hepatitis C he could not automatically approve the payment and so she would be turned down. She then had to appeal to a committee that would review the application and decide whether she was entitled, based on the balance of probability as to hepatitis C infection.

91. The process then involved sending off an application she knew would be refused, itself a dispiriting thing to do, and then having to appeal against the decision to a committee that I was absolute certain would approve the payment. The committee reviewed her application and predictably the payment was approved and she received it. It seemed to me an unhelpful process for a widow to have to go through. As I already had a relationship with the Skipton Fund employee, I could find out what the process was and I could support GRO-A's widow through it. A more timid or uninvolved person may have given up, either because there was no positive result or having applied, decided not to appeal after the initial refusal. A process of allowing the worker, who was by this time well versed in the application process, to automatically forward some of the

[136] The haemophilia centre was extremely helpful in doing this and I believe they did not charge her the usual fee.
[137] I worked at Mainliners, a service for injecting drug users, as their HCV centre manager. Prior to this I had had a short-term post supporting the hepatitis C worker at the Haemophilia Society, Lucy McGrath, organising meetings to explain treatment options to people with haemophilia and HCV.
[138] I believe this was because when the test became available he was already very ill and I can understand the centre seeing little purpose in informing a man about to die that he also had a second potentially fatal condition.

cases he was unable to approve would have been much more supportive of widows.

92. These schemes were finally closed and the "infected blood support schemes" (IBBS) for the four nations were created. These schemes have the benefit of managing everyone infected with viruses through blood products for their country, but with three different payment amounts.[139] I was initially allocated to the English scheme, EIBBS, but after a successful appeal I was allocated to the Welsh scheme, WIBBS, because I had lived in Swansea in 1989. I currently have a lower monthly payment than my equivalents in England who receive a payment for HCV problems that is not available in WIBBS but initially I received more than my equivalents in England. WIBBS recently announced it will introduce an equivalent scheme for those with extra problems to match the English scheme.

The impact of my infections

93. The viruses I have had to live with have had a massive impact on my life. It is difficult to think of any words that are adequate to describe the situation of being diagnosed with a terminal illness that was subject to such extraordinary stigma at the age of 18 and then to survive when so many of my friends - with or without haemophilia - died with AIDS or liver failure. The support of these people helped me cope with my own medical, physical and social needs. My involvement in the HIV and later hepatitis voluntary sectors has been a source of much support and enjoyment for me. Working and socialising with people with HIV, though, meant I got used to the deaths of friends and colleagues on a regular basis because it became a normal part of my life.

94. I have seen and been on the receiving end of bigotry and prejudice sometimes because of my HIV but more often through my association with people infected with HIV through other routes. I have been described as an 'innocent victim' of

[139] E.g. For a person with haemophilia, HIV and hepatitis C Scotland (SIBSS) pays a regular income of £36,000, Wales (WIBSS) pays £24,000 and England (EIBSS) pays £18,000. EIBSS has a separate scheme, the Special Category Mechanism (SCM) which increases this to £36,000 if the person has had problems with their hepatitis C which most of those with HIV and hepatitis C probably receive. NIIBSS follows the EIBSS system but I am unaware if they have SCM or not.

AIDS by people who clearly thought friends of mine infected in other ways deserved to die. I have watched many of those friends die.

95. I became a treatment activist in HIV, educating myself in medical terminology, lobbying pharmaceutical companies and working with other HIV activists to improve treatments and trials as I thought it was the most likely way to stay alive. I also want to be sure when taking treatments that I knew would lessen my quality of life that there was sufficient proof, or at least promise, of some health gain. This continued with hepatitis C, knowing how debilitating interferon is to take and how poor the success rate of it was, I wanted to be sure when I did take it I was giving myself the best possible chance of only taking it once in my life. My medical situation is complex and with so few people living with haemophilia and HIV now that the expertise in it is spread very thinly and there remains the issue of drug interactions with any medications I may be prescribed for other reasons.

96. I have swallowed, injected and inhaled a multitude of disgusting medications over this time and while my current medication is only half a dozen tablets every morning, it has taken a long time to come to this. Throughout this time I have also had to inject myself with factor VIII two or three times a week. In the 1980s I had to complain repeatedly about bizarre and irrational infection control procedures desired by some health care professionals, particularly dentists because of my viral infections.

97. The year spent taking pegylated interferon and ribavirin for hepatitis C was particularly horrible, with multiple side-effects and ultimately ineffective. A second round of treatment with DAAs was much shorter but I was still unable to finish the course because of intolerable side-effects of one of the drugs. The treatment was successful in curing me of hepatitis C though which makes my medical situation somewhat simpler as I no longer need to avoid medications contra-indicated in liver disease.

98. Although I have been employed for the majority of my adult life I have only very rarely been able to work full-time. In the past, this was because of a mixture of

illnesses, necessary surgery and rehabilitation and the collection of hospital appointments with multiple specialists. However in the last ten years it is because I do not have the energy necessary to work full-time.

99. I was involved in the original legal case and lobbying of government for compensation by the Haemophilia Society appearing in the media and at events promoting this cause. I lobbied the MFT for many years to adopt a more caring approach and to involve its registrants in decisions about the use of the money given by government for our benefit. I lobbied the Haemophilia Society for many years about the needs of those infected with HIV and hepatitis C, often working with them and sometimes in opposition. I have not been involved in campaigning, lobbying or media work the last few years as I am tired after 30 years of this. I am pleased that this inquiry has been set up and I hope the outcome will allow me to move on in my life.

Conclusion

100. I hope that my activism and academic research – combined with my personal experience as someone affected – will assist the inquiry's work. My firm view remains that opportunities were missed for early identification of risks from contaminated blood products. Not least among the reasons, was a fear of associating the spectre of AIDS – with all the societal stigma that that carried – with the Haemophilia community. The subsequent treatment and care regimes have ranged from appalling to simply bad for my quality of life – and now, perhaps, tolerable. I welcome inquiry's questions on my personal history. I will also be happy to share any perspectives that may be useful to the wider issues under consideration.

STATEMENT OF TRUTH

I believe the facts stated in this Witness Statement are true.

Signed: GRO-C .....

Dated: 27 March 2019

Glossary of medical terms

Transfusion hepatitis – unknown pathogen that caused hepatitis found in people who had blood and plasma transfusions, later identified as hepatitis B (HBV) and/or hepatitis C (HCV) –

Red antigen, Australian antigen, surface antigen, HBsAg – a protein on the hepatitis B virus

Core antigen, HBcAG – a protein in the hepatitis B virus

Non-B hepatitis - a cause of hepatitis in people that was known not to be hepatitis B and assumed to be another virus and known to be spread through blood transfusions

Non-A non-B hepatitis - a cause of hepatitis in people that was known not to be hepatitis A or hepatitis B, assumed to be another virus and known to be spread through blood transfusions. When that virus was isolated it was called hepatitis C.

Liver function tests - The most common tests now are for ALT and AST, these were previously known as SGPT and SGOT

HTLV-III - the original name for HIV. In France it was called LAV or LAV-1 rather than HTLV-III. It was agreed to replace both names with HIV.