Witness Name: Dr Francis Toolis

Statement No.: WITN3426001

Exhibits: None

Dated: 11 November 2020

INFECTED BLOOD INQUIRY

WRITTEN STATEMENT OF DR FRANCIS TOOLIS

I provide this statement in response to a request under Rule 9 of the Inquiry Rules 2006 dated 14 June 2019.

I, Dr Francis Toolis, will say as follows: -

Section 1: Introduction

1. My name is Francis Toolis, date of birth GRO-C 1946.

2. Qualifications:
   - MB.ChB (1972), University of Edinburgh
   - M.R.C.P. (UK): Member of the Royal Colleges of Physicians of the United Kingdom (1975)
   - M.R.C.Path: Member of the Royal College of Pathologists (1979)
   - Fellow of the Royal Colleges of Physicians of Edinburgh (1986), Glasgow (1998) and London (2000)
   - Fellow of the Royal College of Pathologists (1991)
   - MBA Health Care Management (1998), University of Stirling

   I was a Consultant Haematologist at Dumfries & Galloway Royal Infirmary (DGRI) from 1.11.1981 until 27.1.2006 when I retired in good standing.

3. I have never been a member of any committee or group relevant to the Infected Blood Inquiry.

Section 2: Responses to criticism of Mr Griffiths

4. I am grateful to the Inquiry for affording me the opportunity to comment on the criticisms made of my care by Mr Griffiths. The Inquiry's letter dated 14th June 2019 posed four questions to me. I will answer them in order, but also two further '*unposed*' questions arising from Mr Griffiths' verbal testimony.

5. This statement was prepared without access to the patient's medical records, permission not being granted until some considerable time later. I was therefore reliant solely on recall of events that occurred some 20 – 40 years ago. When preparing my statement, I had initially thought that access to the relevant medical records might be of assistance specifically in my response to Question 3 posed to me by the Inquiry, namely whether there had been a delay in informing him of his HCV status. By the time, however, that permission for access was granted, I had already come to the conclusion that any further information that might or might not be in the available medical records would not materially affect my statement. My reasoning for this conclusion is given in my response to Question 3. I reserve the right, however, to revisit my decision, should future circumstances warrant it.

Background

6. Before I address Mr Griffiths' criticisms, it may assist the Inquiry to be made aware of the framework within which his haematological care took place at DGRI. I have also sought to explain, as best I can, the scientific and medical thinking and developments occurring at the time period under question, for the better understanding of those unfamiliar with such. I ask the Inquiry's forbearance.

I was not 'his' Consultant

7. Mr Griffiths refers to me as his consultant, giving an impression of exclusivity of professional relationship between us. He stated that he moved to GRO-C in 1971, with the inference in his statement that I was then the haematologist supervising medical management of his haemophilia. In 1971, however, I was a final year medical student in Edinburgh. For the first ten years of his residence in GRO-C, the management of his haemophilia would have been undertaken by a team of two other haematologists then in post in DGRI. On the retiral of one of them, I succeeded in post in November 1981. At that time, Haematology in Britain was changing, moving from a solely laboratory-based specialty to a hybrid of both diagnostic laboratory and clinic/ ward-based clinical service with direct care of patients with haematological disorders. The nature of division of the workload between my laboratory-based colleague and myself would of necessity have left the majority of Mr Griffiths' cryoprecipitate and Factor VIII concentrate administrations to my colleague, whilst any routine (presumably annual) Blood Clinic follow-up would have been mainly by myself. I do, however, clearly recollect undertaking one administration to the patient of cryoprecipitate, my favoured modality of therapy at that time.

8. With the retiral of my colleague in 1990, and replacement by first one and later a second 'hybrid' haematologist, care of all our patients was shared jointly between us. Additionally, I am surprised that he made no reference to follow-up at a recognised Haemophilia Centre (Glasgow in his case), as it was and is my understanding that such is standard practice with all haemophiliacs, even those living at some distance from their nearest Centre.

9. I do not explain this to somehow shift or share any perceived 'blame' onto other doctors, but solely to explain the context in which Mr Griffiths' clinical care was delivered.

Mild or Moderate/Severe Haemophilia?

10. The frequency with which he said he attended Dumfries for treatment (5 – 10 times a

year) is surprising, given his diagnosis of Mild Haemophilia, based on a Factor VIII level of more than 5% of normal. This categorisation certainly fitted with his initial presentation, not with a spontaneous bleed or after minimal trauma in infancy or early childhood seen in Severe and Moderate Haemophilia, but rather with 'provoked' excess bleeding after dental extractions when he was seven years old. His clinical history of frequent bleeds, however, is more in keeping with Moderate/Severe Haemophilia. He did state that he enjoyed playing sports in early youth, sustaining haemarthoses (bleeds into his joints) in the process, and was forbidden to play contact sports from age 13 onwards. It would appear from his testimony, however, that he continued to suffer frequent bleeds thereafter.

Question 1: Did I mislead him over his being treated by my Department with imported blood products? No.

The container is not the contents

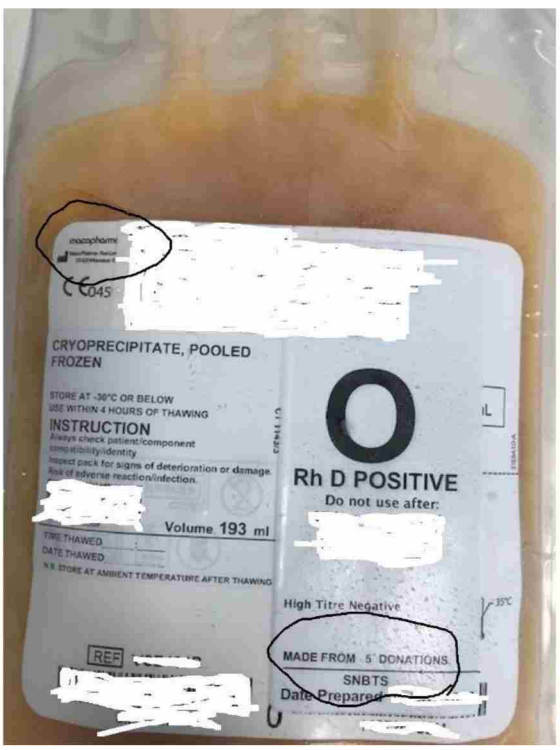
11. As an initial observation, I am concerned by his use of the word 'dismissed' to describe my response to the concern he raised that he was being given anti-haemophilia factor imported from the United States and Australia. I would never have dismissed a patient's anxiety: that would have gone against the holistic approach to Medicine I sought to follow throughout my career. When a patient asserts with confidence that he has been receiving foreign Factor VIII, it strains belief that his claim should be 'dismissed'. The logical action would have been to check the labels of the Cryoprecipitate and Factor VIII for myself and contact my Regional Blood Transfusion Centre for reassurance or otherwise before coming back to Mr Griffiths with their response.

12. That is exactly what happened. His assertion of a foreign source for what we were giving him was so disconcerting that I clearly remember checking the contents of our Blood Bank myself when Mr Griffiths first raised his concern with me. I spoke to my colleague of the time about our Department's practice before I took up my post at the end of 1981, and also sought and was given confirmation as to the Scottish blood donor source of our blood products from colleagues in our Regional Blood Transfusion Centre. In the late 1990s, SNBTS moved to imported plasma (sourced from Austria and Australia, I think) in response to 'Mad Cow disease' and the fear of transmission of variant Creutzfeldt-Jacob Disease (vCJD) by plasma derived from the Scottish population. Prior to that time, my understanding is that no haemophiliac under my care was ever given imported blood products.

13. Mr Griffiths asserts that I 'assured him that Scotland never imported blood products.' I would not have been in a position to give such an assurance. What would have been said is that all the blood products he received from my Department had come from the Scottish National Blood Transfusion Service (SNBTS), never from (foreign) commercial companies, and that the SNBTS sourced all its blood products from donors living in Scotland. Mr Griffiths, by his own testimony, was on more than one occasion given the same reassurance of Scottish source by my staff, who would have supplied from our Blood Bank the cryoprecipitate and Factor VIII given to him – and they, more than anyone else, would have known the labelling details of each and every blood product sent to us by our Regional Blood Transfusion Centre in Glasgow. The Glasgow Centre was our sole supplier of blood and blood products. We at no time purchased from any commercial source. Despite these reassurances of an exclusively Scottish origin, the patient states that he saw foreign manufacturers'

labels on the blood products given to him. I quote from his verbal testimony: *'I had seen the labels on the sachets as I was being infused, labels of laboratories in Australia, labels of laboratories in America, where the material had obviously originated.'* Subsequent questioning at his verbal testimony confirmed that these 'sachets' were bags of cryoprecipitate, with labels from Australia and Baxter of America.

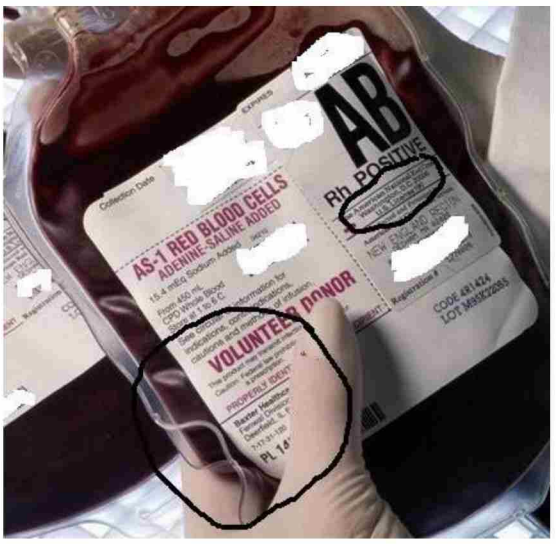
14. When first raising his concerns, the patient could not have mentioned the name 'Baxter', the manufacturer he states seeing on some of the Factor VIII products given to him. If he had done so, that name, Baxter Healthcare, would have been recognised instantly by myself or any of my Blood Bank staff as a major American multinational manufacturer of the disposables of blood donation and transfusion, specifically here of blood bags. I am unfamiliar with any similar Australian Company, perhaps a Baxter subsidiary. It is a perfectly understandable interpretation to see a manufacturer's name on a container and conclude that the contents also derive from that manufacturer. Such a misinterpretation, by a Dumfries nurse, also appears to have occurred in the 1970s, in another case ('Mr X') heard at the Inquiry's sitting in Edinburgh, where she had jocularly informed a patient that his blood transfusion '*had come from America*'. Two images to illustrate my point follow.



15. Having been unable to find an image of a Baxter Healthcare Cryoprecipitate bag, I have attached instead an image of one manufactured by Macopharma, a French multinational.

    This bag contains SNBTS-sourced cryoprecipitate and has had all bar-coding deleted to preserve donor confidentiality. Cryoprecipitate is a 'low-tech' derivative of whole blood, obtained by collecting the blood donation into the primary bag of a triple bag

closed- system, centrifuging the blood to separate the plasma from the red cells, siphoning off the plasma into the secondary bag, freezing the content to form 'Fresh Frozen Plasma' and then partially thawing out to siphon off the liquid 'plasma supernatant' into the tertiary bag before refreezing the still solid residue, i.e. Cryoprecipitate, in the secondary bag.



16. This second image is of a unit of blood collected from a volunteer donor by the American Red Cross, in a blood bag manufactured by Baxter Healthcare.

17. Neither myself nor my Blood Bank staff, confident that Factor VIII products were derived from Scottish plasma, would have realised, as we clearly did not, that the blood bag manufacturer had been the cause of Mr Griffiths' concern. A blood bag was a blood bag: it was its contents upon which we focussed.

18. In relation to freeze-dried Factor VIII concentrate, I cannot offer a correspondingly similar explanation, as I do not recall ever noting the manufacturer of the containers in which it came. It was always an SNBTS product that I used, derived from the Protein Fractionation Centre (PFC) at Liberton (Edinburgh), which began making Factor VIII concentrate about 1975, corresponding to the time at which Mr Griffiths stated that he began receiving this product, its primary advantages over cryoprecipitate being a reduction in allergic-type reactions and its suitability for home and even prophylactic (preventive) treatment. PFC would have sourced its containers for Factor VIII and other blood products from commercial sources, which might very well have been based abroad. To the best of my knowledge and belief, the plasma SNBTS used as its source of Factor VIII supplied to my Department came solely from Scottish donors at the period in question. If such was not always the case, I was never made aware of this.

19. The situation regarding sourcing of Factor VIII was different in England, where Mr Griffiths spent the first 20 years of his life undergoing frequent administrations, and perhaps again later if he required treatment when returning there for holidays or business. England, as I understand it, was not self-sufficient in blood supply and some at least of its Haemophilia Centres imported Factor VIII from abroad, principally the United States.

20. In the end, however, the concept that Scottish blood donations were 'safe' proved an illusion in the absence of reliable laboratory tests able to identify carriers of blood-transmitted viral disease, as evidenced firstly by Hepatitis B (not identifiable serologically until the mid-1960s, despite subsequent studies showing that it has infected humans for millennia[1]), HIV (for which there was no serological test until 1984/5) and HCV (which was not identified until 1989). In the 1990s, 'Mad Cow Disease' and vCJD, for which there was no serological test until 2014 or thereabouts, forced the import of plasma from countries whose populations had not been exposed to potentially vCJD- contaminated beef.

Question 2: Did I inform him of his HCV infection 'inappropriately' by telephone? No.

21. At the beginning of paragraph 6 of Mr Griffiths' written submission, he states that he was first informed of this in a letter from me but, in paragraph 8, states that '*giving me this information* [that I had hepatitis C] *over the telephone was highly inappropriate*.'

22. As for the phone call itself, my thought process would have been to offer him the opportunity for a follow-up telephone call to discuss this finding, now known to him from my letter ['*If you have any concerns in the meantime, please do not hesitate to give me a ring*']. The alternative would have been leaving him with inevitable delay and increased anxiety to await the outpatient appointment I had requested for him with the Infectious Diseases specialist, Dr Gwyneth Jones (not Gillian, as he stated). Worst of all, in my view, would have been sending an initial uninformative letter giving him an unexpected appointment to attend the Blood Clinic outpatient clinic, raising worry as to the reason for the early recall. That would have created the potential for a phone call by him to ask the reason for his early recall.

23. Breaking bad news to patients is never easy, but I have always tried to keep the period of uncertainty to minimum. The offer of a follow-up telephone call under Mr Griffiths' particular circumstances at this point would have been in keeping with such thinking, a personal practice feasible in a hospital the size of DGRI, but which would not have been possible in the large Teaching Hospitals where I trained. I regret that this kindness was not as supportive as it had been intended to be.

Question 3: Did I delay telling him of his Hepatitis-C infection? No.

There was no 'Coalition of Secrecy'

24. Regarding Mr Griffiths' concern at the seeming delay in informing him of his hepatitis C infection, I cannot make a comprehensive response in the absence of his medical records, but doubt in any case whether such access would materially affect my response, for reasons I will explain later. What I can state is that his HCV-antibody positivity was not deliberately kept from him when it was first confirmed. I reject the suggestion that there was any 'coalition of secrecy', the phrase he used in his verbal submission. It is my belief that he was informed before 1999.

25. To explain that, we need to go back to 1977 when, by his verbal testimony, he was tested for Hepatitis B (HBV) and presumably found to be negative, although he speaks to not having been told the result. In the late 1970s or '1980-ish', he was informed that he needed an anti-HBV vaccination. That would be about the time a

safe HBV vaccine was developed to protect HBV-negative patients. Although these dates were before I came to DGRI, the advisability of this vaccination would have been explained by the haematologists then in post. It is hard to conceive a situation in which a vaccination was given without explanation by the haematologist or question by the patient.

26. Accordingly, I believe Mr Griffiths was informed of his HBV test result. Good news may have less of an impact and be forgotten. This was, after all, forty years ago.

Permission for HIV testing was requested

27. In 1985, Mr Griffiths was tested for HIV, soon after the emergence of transfusion-transmitted HIV, the group commonly referred to as the Edinburgh Haemophiliac Cohort, and the development of a blood test to detect HIV. As it happens, I had volunteered to be the designated HIV/AIDS Physician for DGRI, in the light of my experience in the medical management of immunosuppressed patients on treatment for acute leukaemia. At that time, treatment for AIDS was essentially 'fire-fighting', dealing with the atypical and often severe infections to which AIDS patients were prone, rather than being able to do much about the underlying viral infection itself. As part of my effort to acquire the necessary skills, I read up on the disease, attended conferences and training courses, and visited St Mary's Hospital and the Middlesex Hospital in London, both by then recognised leading centres in Britain for the management of AIDS. I was also fortunate enough, while on holiday in the United States, to visit San Francisco General Hospital, acknowledged at that time as world leader in HIV/AIDS management. One subject that had repeatedly arisen, engendered by patients' fear of stigmatisation, was the ethics of testing and the mandatory careful consent process preceding it, all under strict confidence. Accordingly, I was very aware of the sensitive importance of seeking informed consent to test for HIV and again of informing the patient of the result, whether good or bad. I have clear memories of discussing the result with him: it was noteworthy because HIV testing was seldom indicated or undertaken by me in the course of my clinical workload. Even the very few HIV/AIDS patients I cared for were late-stage AIDS returnees from the United States, their care soon to be handed over to the specialist units which had developed in Edinburgh and Glasgow. I followed exactly the same procedure (explanation, consent and inform) with HBV and HCV, regarding their testing and confidentiality in the same way as I would have with HIV.

Advice was given on NANB (HCV)

28. I also recall alluding, although probably not by name, to Non-A Non-B Hepatitis (NANB), first identified in the mid-1970s and the precursor nomenclature of what would turn out to be HCV. It was postulated at the time to be a group of blood-borne viruses manifesting as a usually asymptomatic post-transfusion abnormality in liver function tests, sometimes transient, sometimes persistent. Given that there was no easy way to distinguish NANB from other causes of deranged liver function tests (LFTs), the medical thinking then was that this was a relatively benign condition for which, in any case, no treatment was available or warranted so long as the patient remained clinically well. Even if some of those patients with persistently abnormal LFTs should progress eventually to cirrhosis, this would not occur until decades later and treatment would be largely supportive. As I remember it, my advice was something along the lines of: '*There might be other viruses lurking out there somewhere, but nothing like HIV or HBV. Even so, the advice is to clean up for*

yourself any cuts or bleeding you might happen to get. Don't let others do it for you.' There was no evidence to suggest that NANB could be transmitted sexually: hence no mention of the use of condoms. Overall, my tone would have been reassuring. This conversation happened nearly 35 years ago and memories fade. I only remember this one because it was so different from the usual consultations in my clinical practice.

29. If Mr Griffiths' LFTS were significantly deranged in 1985, the tenor of my words might have been different. I stress significantly because haemophiliacs, going back to the pre-HIV era of treatment exclusively with low-pool cryoprecipitate, often had mild derangement of liver function tests (LFTS), thought to be a chemical or immunological reaction to 'foreign' proteins.

The 1990 Tests

30. Turning now to the microbiology report of 1st November 1990, I am confused about this on two counts – the tests for Hepatitis A (HAV) and HCV: not the results, rather the tests. HAV is not a blood-borne disease and I cannot think of a reason why I might have tested for it. Additionally, the positive HCV antibody test was read out in evidence as if it were on a separate 'add-on' report form. The diseases that I would have requested tests for were HIV and Hepatitis B (what was meant by 'hepatitis' in my letter dated 2nd November). These, I have no doubt, I would have specifically mentioned to Mr Griffiths in seeking consent. Although SNBTS from 1984/85 had been screening out high-risk potential donors and those serologically positive for HIV and Hepatitis B (HBV), and Mr Griffiths had been vaccinated against HBV, the true test of success was a serologically negative recipient. The presence of Hepatitis A in the lab report suggests to me that the originating request form, giving the Clinical Details as 'Haemophilia', might had asked only for 'hepatitis screen', being understood by me to mean HBV in light of the diagnosis given, and by the Microbiology Laboratory staff as Hepatitis A, B and C.

31. There was no test to detect HCV until 1989, when the development of what was subsequently described as a 'first-generation' enzyme immunoassay (EIA or ELISA) was published. Given the inevitable delay in developing, manufacturing, disseminating and validating this first test out with a research laboratory and into the clinical diagnostic world, it is improbable that I would have requested or knowingly had available to me in 1990 a test for HCV from DGRI's Microbiology laboratory and its Virology reference laboratory (this test was not performed in DGRI but sent off to a specialist laboratory). SNBTS was not in a position to deploy HCV testing of all blood donors until September 1991 (*Penrose Inquiry, Chapter 31*). The practice of DGRI Microbiology was to issue reports as soon as they came back from the Reference Laboratory, and the likelihood is that the three results (four if HIV is included) did not return as a single report. The HCV test, being new, may very well have been run only as batch testing when a sufficient number of request samples had arrived. Hence my mention of 'add-on,' with the HCV result coming back after the others. In that case, although very unlikely, it is possible that this supplementary report was filed in the case-notes without coming to my attention. Whether or not this was the case, the result was very clearly noted at his 1991 attendance when the HCV Antibody test was repeated.

32. An inevitable consequence of knowing that HIV and HBV were tested for in 1990 (and he would have been informed, as I explained previously) would be wanting to know

the results. It seems to me most probable that, rather than waiting until the next routine Blood Clinic appointment, he was informed of the (negative) HIV and HBV results, presumably by telephone, being as it was good news. Excluding the HCV result for now, good news may have less of an impact and be forgotten.

Was the 1990 result valid?

33. The question then becomes: was Mr Griffiths also informed of this HCV test result? The simple answer is: I do not know. One possibility mentioned above was mis-filing. I think, however, that the most likely explanation is that he was not informed at this point because this was a new test without any track record and could show only past exposure to HCV, not ongoing infection. With all screening tests (and, indeed, procedures such as radiological screening for breast or bowel cancer), it is important to realise that few, if any, are completely reliable, relating to sensitivity (how many patients with a particular disease does the test fail to detect) and specificity (how many people without the disease does the test wrongly label as positive).

34. Given the uncertainty of this novel test's reliability (confirmed by the later adoption of second and even third generation ELISA tests and alternative techniques), I consider it most likely, in the presence of good health and no significant derangement of his liver function test, that he was not informed of the result, to spare him unnecessary anxiety over a perhaps spurious result requiring confirmation.

The 1991 Test

35. Whatever my reservations about the 1990 test, that performed on 7th November 1991 was unambiguously positive for HCV antibody ('*Positive for anti-hepatitis C virus, confirmed by RIBA-2*'). RIBA or Recombinant ImmunoBlot Assay was a later developed test using a different technique to detect antibody to HCV, hence its use as a confirmatory test. Even so, false positives and negatives occurred with both tests, which have now been superseded by more reliable techniques. These tests showed the presence of antibody against HCV, not evidence of the presence of the virus itself. Detection of the virus, with presumptive evidence of ongoing infection, would come later and, by what I had gathered at the time from colleagues in that field of study, had very much been 'work in progress' throughout the 1990s and early 2000s to achieve reliable and ease-of-use methodology.

36. So, from 1991 onwards at least, it was known that Mr Griffiths had at some time in the past been exposed to Hepatitis C, almost certainly through blood products, most likely freeze-dried Factor VIII, given its multiple-donor origins. Why was he not informed of this until 1999? Human memory is imperfect and prone to error, a topic I will return to in my response to Question 4. I believe he would have been informed, probably not in 1990 but certainly at his Blood Clinic attendance in 1991 when the need for the test would have been explained.

37. Given its very recent discovery, the natural history of infection by HCV was not known in the early 1990s, although some inferences could be drawn from NANB, later shown to be almost entirely due to HCV. Viral hepatitis was a subject falling outside the usual area of expertise of haematologists, and we would have been guided by the general and evolving expert thinking of the time, which was, to the best of my recollection, that HCV infection did not result in long-term carrier status in all such patients, progressed to active hepatitis in only a relatively small minority,

with a smaller proportion still going on to develop cirrhosis and a minority of those developing hepatic cancer, all on a timescale spanning decades. It was also considered, as was established by later experience, that the risk of onwards transmission of HCV was almost solely by blood and rarely by intimate contact, unlike HIV, and posed no significant likelihood of danger to family members in the absence of high- risk behaviour such as needle-sharing. I do not know how and when Mr Griffiths was informed of the 1990/1991 results, but am confident he was, if for no other reason than he needed to be aware of the theoretical or potential risk his blood might pose to others. The news was likely to have been given to him in an optimistic manner for his reassurance – something along the lines of *'we've found out you've had an infection with Hepatitis C virus; we think the chances are good that nothing further will come of it, but we'll be keeping an eye on your blood tests.'* Whether a theoretical risk to a partner would have been mentioned is a moot point, engendering as it might have unnecessary worry and family tensions. I can only suggest that a reassuring and optimistic presentation of the result (as would have been in keeping with what was then known) may have led to it being forgotten by him as the years went by with no further untoward news.

First Do No Harm

38. I cannot disprove the possibility, however improbable in my judgement, that Mr Griffiths might not have been informed of the 1990/1991 results. Again, I go back to the lack of knowledge at the time of the natural history of HCV infection. Generally, the presence of antibody against a virus has meant immunity against that virus, as happens with vaccinations against Measles or such like. Unfortunately, as shown by HIV/AIDs, that is not always the case. The thinking, accordingly, might have been that Mr Griffiths had at some time in the past received a HCV-positive blood product, and might very well have developed true immunity to HCV (as some HCV-infected patients do). If, however, he had become a carrier, with the possibility of ongoing chronic infection, this could not be confirmed until a reliable test to detect the presence of actual and active virus was developed. Even then, until there was an effective therapy, there was nothing that could be done for him except to watch for any significant and/or progressive deterioration in his liver function, when only supportive or palliative therapy could be offered. It was not until the very late 1990s/early 2000s that an effective therapy with (pegylated) Interferon and Ribavirin was developed. If Mr Griffiths had not been informed of his HCV antibody positivity, it would have been an outcome arrived at – I will not call it a decision as I am sure there was no such deliberation – that informing him of a past and perhaps now closed infection episode served no useful purpose and would have resulted only in pointless anxiety.

The missing Liver Function Tests (LFTS)

39. At the verbal submission, there was a sentence read out from Dr Jones' letter of reply to me after Mr Griffiths' first attendance at her that confused me: *'the only previous liver function test dated back to that time [1990] when both AST and ALT were mildly elevated.'* To my knowledge, every patient without exception who attended the Blood Clinic had a Full Blood Count, Urea & Electrolytes (a check on kidney function) and LFTs done routinely. Additionally, Mr Griffiths' HCV status was rechecked in 1991, and it is inconceivable to me that his LFTs were not checked at the same time. That quoted sentence accordingly raises some additional questions in my mind. Was Mr

Griffiths being followed up annually at the DGRI Blood Clinic throughout the 1990s? Were blood tests results misfiled in the case-notes available to Dr Jones' clinic? Was there another volume of case-notes (many haematology patients had multi-volume case-notes)? In any case, the true guide to active therapeutic intervention, such as was available, was not deterioration in laboratory tests but rather in the clinical state of the patient as, for example, the development of jaundice or liver enlargement.

Medical letters to GPs do not generally record what was said to patients

40. Access to Mr Griffiths' case-notes might answer these questions. Access specifically to the letters sent from the Blood Clinic to Mr Griffiths' GP might help to resolve whether he had been told of his test result in the early 1990s, but probably not: it would have been left to inference readily understood by another doctor, rather than being positively stated. The primary purpose of Blood Clinic letters was to serve as a medical record of a patient's medical status; written, one might say, by myself to a future myself or colleague at the next Blood Clinic. It was not to record all that passed verbally between patient and doctor which, even if it were feasible, would be in danger of obscuring the relevant medical facts.

41. I offer my response to Question 3 as explanation for Mr Griffiths' perception in 1999 of not having been informed previously of his test results of 1990/1991. For the sake of simplicity, the rest of my statement will read as if he was not informed of his HCV status until 1999, without accepting that such is the case.

Question 4: Did I fail to provide him medical advice on the management and risk of his infection? No.

42. I am surprised by the detail Mr Griffiths recalls of a telephone conversation from 20 years ago. I observe that he had just come back from holiday to be confronted by bad news in a letter and that, as described in his verbal submission, he phoned me immediately. For myself, I have no recollection of this telephone call, my only comment being that the phraseology he ascribes to me was not one I recognise as my normal way of speech.

Patients' retention of information

43. There is a considerable medical, scientific and forensic literature on the fallibility of the human memory. Studies, for example, have shown retention as low as 20% of the information given to patients, and creation of false memories with up to 50% of what is 'remembered' being wrong [2-9]. Retention can be particularly poor when bad news is being given. So often throughout my professional career, I saw patients stop listening once the word 'leukaemia' or 'cancer' was mentioned, despite standard counselling practice being followed. The implications to patients of such Bad News words, whether accurate or not, so often drown out what is being said next. Breaking bad news is an iterative process. It can be a long-term iterative process, too: people mis- remember what was said or done. I do not exclude myself from failure of memory. I can recall very little of the presumably regular routine clinic attendances by and interactions with Mr Griffiths between 1981/82 and 1999: there were many clinic attendances and many interactions with many patients over that period. Those that were routine and uneventful were quickly forgotten. I have memories only of those where something exceptional, something noteworthy happened, and have described these involving Mr Griffiths as I best remember

them.

'Life-threatening'; 'Nothing to worry about.'

44. What had been called Non-A Non-B hepatitis was identified as Hepatitis C in 1989, and the medical world had had 10+ years of experience of its natural history when Mr Griffiths received my letter. Hepatitis C is now widely recognised as perhaps the commonest cause of chronic hepatitis, cirrhosis and liver cancer in the world. I knew this at that time, I knew it could kill eventually but hopefully not for many years, and I knew there were promising treatments available already or soon to come. I have no doubt that that is what I would have told Mr Griffiths, although not in those exact words. I would have also told him that I possessed no specialist expertise in viral hepatitis, but that I had referred him to one for further care. Mr Griffiths was not abandoned without onward care, as implied by paragraph 7. What I would have told him was what I knew of the disease, its prognosis and treatment. I would never have told him there was 'nothing to worry about' in such simplistic terms.

45. Turning now to the specifics of Mr Griffiths' recollection of what passed between us in this telephone conversation, he stated in both his written and verbal testimony that I said [Hepatitis C] '*is not life-threatening. It is only life-shortening.*' My exact words, he claims. I do not accept that I would ever had said such a thing. As Mr Griffiths himself pointed out in verbal testimony, such a statement does not make sense. What I would have explained, given the knowledge at the time of HCV, was that his Hepatitis C infection per se was a chronic, non-fatal condition, but that complications could arise that might eventually prove more serious. I would not have used the word 'life-threatening' on the phone, unless responding to a use of the term by Mr Griffiths himself. As to the suggestion that I told him there was nothing to worry about, again this relates to time frame and, even then, with qualifiers, given my knowledge at that time of his 'greatly deranged' liver function tests and the confirmed presence of ongoing HCV infection (the positive HCV PCR, indicating the presence of virus). In the short term, his HCV infection by itself was not a cause for concern regarding life expectancy. In the longer term, there was the risk of cirrhosis and of hepatic cancer. Consider, however, that he had just been informed he had HCV infection and that he was being referred immediately to an Infectious Diseases specialist (his 'liver specialist'). This might very well be construed as life-threatening and would have required a degree of reassurance. I return to my earlier point about 'bad news' words: once uttered, patients tend to stop attending to the information that follows.

46. As an illustration of my point, Mr Griffiths' account of my telling him that he might experience mild flu-like symptoms is actually a description of what happens with some individuals when *first* infected with HCV. The only point of saying that would have been in relation to a question as to when he was infected (e.g.: 'what would I have noticed at the time'?')

How long had he been infected?

47. In Mr Griffiths' account of the telephone conversation, he asked how long he had had the infection. If such a question was asked at that time – in my professional experience, patients faced with bad news become very focused on the future (how long have I got; how will it affect me; what about my job), not on the past – no accurate answer was possible. There was then, and is now, no way to know when he had first been exposed to HCV. It could have occurred any time between that first

administration of Factor VIII product in Birmingham in 1958 right up to shortly before he was first noted to be HCV antibody positive. The reply he attributes to me in his verbal testimony ('I can't really tell you') captures the core of the answer I would have given him, although I doubt that my answer would be been so brief and unexplained.

How long did I know about his HCV infection?

48. I have difficulty in accepting that such a question would be asked at that time and under the circumstances of receipt of distressing news and onward referral to an Infectious Diseases specialist.

49. Assuming, however, that Mr Griffiths did indeed ask that question at that time, how would I have responded? Given that this happened 20 years ago, I can only put myself into what I believe would have been my likely mind frame and response. Confident that he had been told years previously of his HCV antibody, I would have been taken aback by his question, a question unlikely to have been posed in a neutral voice (it is not a neutral question). Evidently, if the account of the conversation is accurate, he had forgotten what he had been told in 1990/1991. To challenge a distressed patient, and over the telephone ('but we told you years ago') would have been an unkindness I would not have done. Nevertheless, it would have been a quandary as to how to respond. There would have been a problem, too, a source of misunderstanding between us as to what was meant by 'infection'. In my letter of 2nd December 1999 to Dr Jones, quoted in the verbal testimony, I differentiate between HCV antibody [infected in the past] and HCV PCR [ongoing infection]. In the absence of a previous negative PCR, it could reasonably be inferred that he had been PCR positive for some time, perhaps even persistently from the beginning. How long then had I known (or suspected) in retrospect that he had had an active HCV infection? No definite answer was possible and I would have responded accordingly, but not in the evasive way Mr Griffiths' testimony portrays.

50. As an aside, I want to explain why he was not tested for HCV PCR before 1999, according to the quoted extracts from his medical records. Firstly, there was the availability or not of the PCR test before that time, and I have already touched on the development problems this test seemingly experienced. Secondly, knowing he was PCR-positive would have had no effect on his essentially wait-and-see management, in the absence of any treatment for the HCV infection. Whilst Mr Griffiths' LFTS may have been abnormal in the intervening period since 1990/1991, it would only be when they showed a significant deterioration indicating possible cirrhosis and impending liver failure that specialist advice might be sought. Even then, in the absence of definitive treatment, and without signs of that actual liver failure, little could be offered by specialists. Thirdly, there was no local source of specialist advice until 1998 when Dr Jones was appointed as DGRI's first Infectious Disease Physician. Lastly, treatments for HCV were becoming available about this time, as I have already mentioned earlier in this submission.

Failure to provide medical advice

51. Concerning Mr Griffiths' assertion that I gave him no information (paragraph 7), or misleading information ('nothing to worry about' … not life threatening'), I have already given my response above. The way his testimony was presented implied that I gave him no advice at the time of the phone call in 1999. That may very well be true in the strict sense: the 'advice' I was giving him was to be seen in the very near future

by an Infectious Diseases specialist, who would then give appropriate advice. Giving advice myself at that point was neither necessary nor appropriate.

52. An alternative interpretation of his assertion of no advice given refers to any earlier time, and that is what I assume Mr Griffiths meant: that he should have been given appropriate medical advice about HCV in 1990/1991. I have lifted verbatim the section on Counselling Patients with HCV from the website of the United States Centers for Disease Control and Prevention (CDC), as of 2nd July 2019[10]:

- Patients should be informed about the low but present risk for transmission with sex partners [but see below*].
- Sharing personal items that might have blood on them, such as toothbrushes or razors, can pose a risk to others.
- Cuts and sores on the skin should be covered to keep from spreading infectious blood or secretions.
- Donating blood, organs, tissue, or semen can spread HCV to others.
- HCV is not spread by sneezing, hugging, holding hands, coughing, sharing eating utensils or drinking glasses, or through food or water.

What should HCV-infected persons be advised to do to protect their livers from further harm?

- HCV-positive patients should be advised to avoid alcohol because it can accelerate cirrhosis and end-stage liver disease.
- Viral hepatitis patients should also check with a health professional before taking any new prescription pills, over-the counter drugs (such as non-aspirin pain relievers), or supplements, as these can potentially damage the liver.
- Clinicians may wish to consider vaccinating HCV-positive patients against hepatitis A and hepatitis B even in the absence of liver disease.

*HCV is transmitted primarily through parenteral exposures to infectious blood or body fluids that contain blood. Possible exposures include

- Injection drug use (currently the most common means of HCV transmission in the United States)
- Receipt of donated blood, blood products, and organs (once a common means of transmission but now rare in the United States since blood screening became available in 1992).
- Needlestick injuries in health care settings
- Birth to an HCV-infected mother

Although infrequent, HCV can also be spread through:

- Sex with an HCV-infected person (an inefficient means of transmission, although HIV- infected men who have sex with men [MSM] have increased risk of sexual transmission)
- Sharing personal items contaminated with infectious blood, such as razors or toothbrushes (also inefficient vectors of transmission)
- Other health care procedures that involve invasive procedures, such as injections (usually recognized in the context of outbreaks)

- Unregulated tattooing

53. Bearing in mind that CDC has the advantage of a longer experience and greater knowledge of HCV that was available in the period up to 1999, I maintain that Mr Griffiths *was* given appropriate medical advice, going at least as far back as 1977 when he was tested for HBV, most certainly when he was tested for HIV in 1985, and again in 1990/1991. CDC serves a very different population in 2019 to that of a haemophiliac in GRO-C in the period under consideration. As an instance, it would have never occurred to us to warn Mr Griffiths against sharing toothbrushes or needles, nor would advice against donating blood or other tissue have been necessary as he would not have been accepted, and would have known that. The alcohol issue would have been covered by the advice routinely given to all haematology patients concerning the official safe limits and the advisability of those with evidence of liver dysfunction reducing or stopping alcohol intake completely. The only debatable point concerns sexual transmission, which may not have been discussed, for reasons given earlier in this submission.

Question 5: Did I or others fail to obtain consent for blood tests, a question implied by Counsel's line of questioning in Mr Griffiths' verbal testimony? No.

54. When any patient attends a Blood Clinic, there is an understanding and tacit consent that blood samples will be taken for initial diagnosis and follow-up monitoring. It is impractical to treat every blood sampling as if it required the 'informed consent' of a surgical procedure. Specific permission is not sought to undertake 'routine' blood tests such as a Full Blood Count, Urea & Electrolytes, LFTs, thyroid function, immunoglobulins or whatever tests are indicated by the patient's underlying disease. Accordingly, no-one (including Mr Griffiths) would have been directly asked for permission to take such blood samples. The only occasion when direct verbal permission would be required, and would be sought, was for certain sensitive tests, specifically here HIV, HBV and HCV. On each and every one of the few occasions I had cause to perform any of these tests, I always explained the justification for them and obtained clear verbal consent from the patient. I reject the implication that I did not obtain Mr Griffiths' consent to be tested. My reasoning for this statement has already been explained.

Question 6: Was Mr Griffiths deliberately obstructed from obtaining the laboratory records of his blood products received? No.

55. This is not a question posed to me, but a reading of the transcript of the verbal testimony might lead some to conclude that DGRI was deliberately concealing the records of Mr Griffiths' Factor VIII administrations. I quote from the testimony:
Q. Did you receive or did you ask for, did you receive any explanation as to why there were no or only one record of Factor VIII products?
A. When I asked the hospital why there was no record of the Factor VIII treatments or very little they told me that these records were not kept.
Q. Were you given any explanation as to why those records had not been kept but other records had been?
A. No.

56. I empathise with the difficulty Mr Griffiths describes in extracting relevant information from his medical records, having experienced the same problem at times. This was in a pre- computerisation era when all records were paper-based. Haematology records could be particularly extensive, running sometimes to 5 or 6 bulky volumes. I recall one patient with case-notes occupying 40 feet of shelf space.

57. In any case, DGRI's hospital case-notes would have no record of Mr Griffiths' therapy if he attended the Haematology Department for treatment or to collect Factor VIII for his home therapy. What he received was recorded only in separate Blood Bank files. About 10,000 blood products were issued annually, each generating a hand-written record in large, heavy ledgers of the date, batch or unit number of the blood product, and name and identifying details of the recipient. I assume that Medical Records Department staff facilitating Mr Griffiths' access to his medical records were unaware of these separate records. It would, however, be laborious in the extreme to go through these ledgers line by line back to 1971 to find out every single administration of Factor VIII given to Mr Griffiths. That would be just the first step, these blood-product identifying numbers then going to the Regional Blood Transfusion Centre in Glasgow for them to go through their (paper-based) records and then, in the case of Factor VIII concentrate, sending batch numbers to SNBTS' Protein Fractionation Centre (which was closed down in 2007). Presumably these records still exist somewhere, but they are in effect lost to any practical search short of employing archivists for that purpose.

Conclusion

58. I have responded truthfully to the issues raised by Mr Griffiths. My answers are, to the best of my knowledge and recall, accurate. Where I cannot be certain of what happened, I have stated so and given my considered view of the most likely outcome. Where I can be confident of events, I have also stated so.

Section 3: Other Issues

59. The first issue I wish to raise relates to the procedures of the Inquiry itself. I and, I would hope, every healthcare professional providing clinical care to patients with haemophilia, am deeply sympathetic to their plight. Haemophiliacs like Mr Griffiths were born unfortunate at an unfortunate time when the only treatment was a blood product potentially and in actuality carrying three viruses for which there was neither a screening test nor treatment until too late for some. The era was doubly unfortunate for those dependent on blood products because of the appearance of hitherto unknown HIV/AIDS, the explosive increase in intravenous drug abuse and the growth in international travel, making for ease of spread throughout the world.

60. My motivation for entering medical school was to be a caring doctor to those afflicted by disease. Perhaps naively, I had imagined I would cure everyone. Such, of course, is not possible in many instances, specifically here with haemophilia. At best, I and the many caring and conscientious colleagues I encountered, could treat only the consequences of that incurable disease, namely bleeds, and did so to the best of our ability. The Inquiry's proceedings, both in the several submissions I have read and in the articles featuring in the Media, seem very much about blame, about seeking revenge. If the Inquiry had insisted all submissions were anonymous, I might have thought differently. What Mr Griffiths' assertions and sound-bite testimony has done is to cause unwarranted great distress to myself and my family (his testimony featured almost verbatim in the local press), and to besmirch my good reputation, without hope of redress. The Inquiry may accept the validity of my submission: the wider world has moved on and will not.

61. I have a greater charge to make against the proceedings of the Inquiry, namely permitting itself to be a platform for erroneous assertions that could so easily have been corrected at the time by a simple enquiry. The particular assertion I have in mind,

a confusion between contents and container, is that made by Mr Griffiths (and another witness, 'Mr X') concerning the supposed foreign source of presumptively infected foreign blood and blood products dispensed by DGRI's Blood Bank. That claim is likely to have caused unwarranted alarm amongst others who had received blood transfusions and other blood products at the hospital. If anyone had come to harm through this, the Inquiry must bear the responsibility.

62. I strongly favour those who have been infected by blood being given the opportunity to tell their story, 'to be listened to.' My reason will not, however, be the same as those affected individuals testifying to the Inquiry. Hurtful though it is to read their comments directed at those who were only doing their best for them, it is necessary that they be heard. The anger directed against doctors and the wider NHS strikes me as akin to a grieving process, one influential model of which proposes anger[11] as a stage through which someone must pass before reaching acceptance of their circumstances. The widespread anger clearly being felt by those who, through no-one's fault (difficult though many may find that to accept), have been infected through blood needs to be heard if people are to move on to a kind of peace within their minds. I very much hope that many will find that peace when this Inquiry's work is done.

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Statement of Truth

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

Signed GRO-C

Dated 11 November 2020