Witness Name: Professor Charles Richard Morris Hay
Statement No.: WITN3289193
Exhibits: WITN3289194-WITN3289195
Dated: 31/7/2023

INFECTED BLOOD INQUIRY

WRITTEN STATEMENT OF PROFESSOR CHARLES RICHARD MORRIS HAY

I provide this statement in response to a request under Rule 9 of the Inquiry Rules 2006 dated 27 March 2023, in relation to the criticisms of Witness W7214.

I, Professor Charles Richard Morris Hay, will say as follows: -

Section 1: Introduction

1. Professor Charles Richard Morris Hay MBChB MD FRCP FRCPath

   Consultant Haematologist Manchester Royal Infirmary since December 1994.
   Director Manchester Adults Haemophilia Comprehensive Care Centre since December 1994
   Professor of Haemostasis and Thrombosis.
   Senior Lecturer in Haematology Liverpool University and Director Liverpool Haemophilia Centre, Royal Liverpool Hospital 1987-1994.
   Director UK National Haemophilia Database since 2002.
   Member UK Haemophilia Centre Doctors Organisation (UKHCDO) Regional Committee from 1987 and then Advisory Committee since 2007 (when the committee's name changed).
   Vice Chairman UKHCDO 1997 to 2005.
   Chairman UKHCDO 2005-11.
   I have already provided a copy of my Curriculum Vitae to the Inquiry.

Section 2: Responses to criticism of Witness W7214

2. The Manchester Haemophilia Comprehensive Care Centre (Adults) is based in Manchester Royal Infirmary. This was the third largest haemophilia Centre in the United Kingdom. It is now the second largest with >2500 patients with bleeding disorders registered. When I arrived in December 1994, I was the only consultant specialising in adult Thrombosis and Haemostasis in the North-West Region, assisted by a part-time clinical assistant, Dr Monica Bolton. We now have four consultants with this specialism. In 1994, we had three Haemophilia Nurses, one of whom also did counselling and went into the community. There were no clinical research staff. There were no joint clinics and no formal liaison with any other supporting specialism or profession allied to medicine, such as physiotherapy. All the follow-up clinics were conducted in the Haemophilia Centre without any junior staff support. There was no internal training rotation for junior staff, so they spent all their time treating leukaemia. I was on call 1:1 365 days a year except when away or on holiday.

3. In the first year, I introduced an internal training rotation for junior staff so that we had a registrar attached to thrombosis and haemostasis most of the time. I introduced weekly multidisciplinary meetings and arranged for Physiotherapy input for our patients. I rapidly established joint clinics for Orthopaedics and subsequently joint HIV clinics and joint obstetric clinics and later joint adolescent clinics with the paediatric service. Liaison with Hepatology was close throughout this period but not formalised around a clinic.

4. Between 1994 and about 2008 we had only a single hepatologist. We always had a close relationship with Hepatology for obvious reasons but when asked for an opinion on any of our patients they would give it and would then send the patient back to Haematology for further management. We were expected to conduct our own antiviral therapy for hepatitis C and given that we had a cohort of about 150 patients infected with hepatitis C, we became very experienced in managing this. This was a common practice pattern across the UK. In the late noughties, however, there was an expansion in Hepatology locally and across the country such that at the time of Witness W7214's transplant, we had acquired three Consultant Hepatologists, Drs Prince, Greer and Cochar. From 2013/14 a new generation of interferon-free anti HCV treatments were introduced, which were far more effective in eradicating HCV

genotype 1 and which were far better tolerated. From this point onwards, hepatology assumed responsibility for antiretroviral therapy, and we collaborated closely with them to eradicate hepatitis C from the remainder of our cohort who had failed to respond or who had been intolerant of interferon-based antiviral regimens. We have now eradicated hepatitis C from all but a small handful of patients who refuse treatment.

5. This statement is made in response to that of Witness W7214 dated 20 October 2022 and particularly in relation with his unwarranted criticisms of his exemplary medical management. He confirms he has not referred to his hospital records. His account is factually incorrect in many important respects, including descriptions of scans that were never in fact undertaken, consultations that never took place, and conversations that never happened.

6. For this reason, I have included all relevant correspondence relating to Witness W7214 between 2004 and 2015 as exhibit WITN3289194.

7. Witness W7214 is one GRO-C with Haemophilia B Leyden. This is caused by a mutation in the promoter region of the Factor 9 gene. This presents as severe or moderately severe Haemophilia B, and not mild Haemophilia as Witness W7214 states in paragraph 9 of his statement. GRO-C GRO-C, he was tested and diagnosed at birth. This condition is unusual in that the defect is hormone-dependent, so that during and following puberty the Factor 9 level increases and in some affected individuals it may normalise. In Witness W7214's case it came up to 44%, which is considered slightly subnormal in many labs.

8. In the late 1970's he required a dental extraction and was given Factor 9 concentrate to avoid prolonged bleeding and sadly, almost certainly, contracted Hepatitis C from that treatment. In general, Witness W7214 had quite a mild bleeding phenotype and required very little haemostatic treatment.

9. On 20 April 1993, Witness W7214's GP wrote to the Haemophilia Centre mentioning his Hepatitis immunisation and the fact that he had not been tested for HIV. Consequently, Witness W7214 was seen by my predecessor Dr G Lucas, on 16 June 1993 and was tested for the Haemophilia B Leyden mutation and, although it is not mentioned in the letter, also tested for HCV. Dr Monica Bolton wrote to the GP on 25 October 1993 informing the GP that Witness W7214 had Hepatitis C and was at risk

of, amongst other things, cirrhosis and hepatocellular carcinoma. The letter was not copied to the patient, and it is not clear to me from the medical records when Witness W7214 was informed.

10. I took up post at Manchester Royal Infirmary in December 2004 and assumed the care of Witness W7214 at that point. I first met him and his wife on 7 February 1996, when I discussed his liver function tests, Hepatitis C and its treatment and treatment side effects at considerable length, contrary to the account in his statement. I also discussed GRO-C. They seemed keen to try Interferon, and so I initiated the mechanism for the health authority to pay for this and arranged to review the couple again three months later.

11. I reviewed the couple again on 16 May 1996. GRO-C GRO-C Witness W7214 was still keen to try Interferon and so Interferon monotherapy, which was the standard of care at the time, was commenced (in 1996 and not 1999 as stated in paragraph 27 of Witness W7214). Unfortunately, he tolerated this badly. It made him significantly depressed and his psoriasis worsened. His liver function tests did not normalise and so treatment was abandoned after 4.5 months. I reviewed him again after he stopped treatment, and we had a long discussion about the prognosis, the general advice in relation to alcohol, (although he drank very moderately), and the importance of keeping his liver disease under review

12. The early results of Interferon monotherapy were generally poor, and Witness W7214 had genotype 1b, a genotype known to be relatively resistant to treatments available before 2013/14, when Sofosbovir and other similar drugs were introduced.

13. We offered Witness W7214 Interferon three times a week combined with Ribavarin in 1998. This was the state-of-the-art treatment at that time. He and his wife were counselled over the course of several consultations about the treatment side effects in advance, because these were often severe and the behavioural side effects such as depression, fatigue and bad temper were very common and could cause significant relationship problems. The severity of the symptoms also frequently caused problems with work, and some patients became unable to work for the duration of their treatment.

14. Witness W7214 tolerated the Interferon and Ribivarin treatment very badly. He suffered fairly severe flu-like symptoms. He became suicidally depressed and very bad tempered, both of which are recognised direct treatment side effects. He also

suffered severely with fatigue. His depression persisted for a while after he came to the end of his six months treatment. Usually, the depression dissipates quickly after treatment finishes but may persist in those particularly severely affected, especially in those with a previous history of depression.

15. At the end of the six months treatment, he was Hepatitis C PCR negative but sadly, at 3 months he had relapsed. This is a bitter disappointment for any patient and would have been a huge blow to him. The treatment is a massive struggle over months, and patients obviously have a great deal invested in a successful result. Almost all that relapse, do so in the first three months after treatment finishes, when they are often still fragile psychologically. Relapse is very difficult for the patients to come to terms with.

16. After this, Witness W7214 failed to keep several appointments and became lost to follow up for a couple of years. When he re-emerged, we monitored his liver disease according to guidelines with regular liver ultrasound and liver function tests. In the interval since his previous course of treatment, the available treatment had advanced with the introduction of Peginterferon.

17. I introduced the possibility of a further attempt at viral inactivation using Peginterferon and Ribivarin for 12 months, when I saw him in August 2004. I arranged to review him with his wife 3 months later to discuss this further. In November 2004, I explained to the couple that the response to this treatment for his genotype was 40%. I also explained that depression would be an inevitable side effect and that we should start antidepressants at the beginning of treatment, since they would take three weeks to work. He seemed keen to give treatment a further trial but, as he says in his statement, had reservations about antidepressants. The couple went away to think about this for three months. I also discussed the small risk HCV transmission with

GRO-C

18. In February 2005, Witness W7214 saw my registrar in clinic. Witness W7214 was keen to start treatment, and started on Peginterferon once a week, with Ribivarin and Fluoxetine (antidepressant). This was the most advanced approach at the time. Arrangements were made for him to receive further counselling from Sr Paula Mohn, and to have blood tests to monitor the treatment, treatment side effects, and response to treatment, every two weeks.

19. Witness W7214 tolerated this treatment much as before, although his depression was thought to be perhaps not quite as severe. In general, the flu-like symptoms were felt to be more severe with this regimen than with standard Interferon. His review in October of 2005 indicates that he had initially tolerated treatment well but had become increasingly fatigued and had been signed off work. Fortunately, his employing bank was very understanding and supportive.

20. At the end of 48 weeks of treatment, in January 2006, he was again PCR negative. We wrote to Lloyds TSB to thank them for the support that they had given Witness W7214 during his treatment. Sadly, on 7 March 2006, two months after finishing treatment he was again PCR positive, which was a very bitter disappointment for him. When I discussed this with him on 10 May 2006, I tried to put this into context, telling him of research that showed that five years after unsuccessful treatment, treated patients were significantly less likely to have cirrhosis than untreated patients. I also discussed his last ultrasound, which was essentially normal. I told him that we would consider him for further treatment when improved treatment became available. However, at that point we had exhausted the therapeutic antiviral regimens then available.

21. I reviewed Witness W7214 again in October 2007. I discussed his ultrasound, which showed mild fatty change. I emphasised the need for only moderate alcohol intake, but he said he did not drink during the week at all. I checked his liver function and arranged a further ultrasound.

22. I reviewed Witness W7214 in October 2008. Again, the ultrasound showed only fatty change. We reviewed the possibility of further treatment but there was nothing new to offer.

23. When I reviewed him on 21 October 2009, he was well and there was no change in his ultrasound.

24. Eight months later, when reviewed with his wife on 6 July 2010, by my registrar Dr Pike, Witness W7214 was complaining of low mood and tiredness. He admitted to suicidal ideation. He felt that he had been slightly depressed since his treatment with Peginterferon 4 years earlier. We asked the GP to see him to deal with this. Ultrasound, liver function tests, and alpha fetoprotein were arranged, and a review interval set for 4 months. He then failed to keep his appointments and was lost to follow up until early 2012.

Period early 2012 until 2015:

25. This is the period covered by paragraphs 68-82 of Witness W7214's statement, and his account of events in the period he identifies as 2012-13. His account of his professional contact with me during this time are completely wrong, as is his account of his first consultation with Dr Prince and the remarks he attributes to Dr Prince, which are explicitly denied by Dr Prince. The correct sequence of events is set out below.

26. Witness W7214 was reviewed by Dr GRO-D my registrar (not me, as stated in para 68 of his statement), on 18 January 2012. She noted that he had some bloating of his abdomen. She booked an ultrasound of the abdomen and checked his liver function tests.

27. The consultation described in paragraph 69 of Witness W7214's statement, did not take place. Although Witness W7214 asserts that I ordered 4 ultrasounds that year, he in fact had only 2 and only one of these was ordered by me.

28. Paragraph 70 of Witness W7214's statement is also incorrect, and the events described did not happen.

29. I next saw Witness W7214 on 21 November 2012, (not early 2013 as stated by Witness W7214 in paragraph 71 of his statement), when he attended with his daughter. GRO-C which I completed Witness W7214's statement, at paragraphs 71-73 appears to conflate two visits with me and, other than the presence of his daughter, is entirely incorrect.

30. At the consultation of 21 November 2012, Witness W7214 complained of some ankle swelling. I noted, looking at his face from across the desk that he had "paper money skin". Since this may be seen in patients with cirrhosis, I examined him. I found and documented further signs and symptoms of cirrhosis including spider naevi, gynaecomastia, and hepatomegaly. The previous ultrasound report from earlier in 2012 was unavailable, having not been reported, as we confirmed with the X-ray department. I warned him that I suspected cirrhosis and told him that if this was confirmed, he would have to be referred to a hepatologist. He was shocked by this news. I agree it was not ideal to tell him this with his daughter present, but I felt I had to share my suspicions without delay. I also reviewed possible further antiviral

treatment. By that stage triple therapy was being used (Peginterferon, Ribivarin and a protease inhibitor), but the results were poor in previously relapsed patients. Since he had suffered so much with previous treatments, I felt that this was not a realistic option. Dr Prince agreed with this assessment when he saw Witness W7214.

31. I reviewed Witness W7214 on 21 January 2013. His ultrasound confirmed cirrhosis with a normal direction of portal flow and a small amount of ascites, which I discussed with him at length. His platelet count and his albumin had both declined over the previous year suggesting a hepatosynthetic defect. It was clear that he had cirrhosis and some degree of hepatic decompensation. I discussed cirrhosis with him and referred him to Dr Martin Prince, Consultant Hepatologist. Witness W7214 was shocked by my diagnosis and was struggling to come to terms with it. I did not mention the possibility of liver transplantation with him although I did discuss this in the final paragraph of my referral letter to Dr Prince, I also mentioned screening for oesophageal varices.

32. Dr Prince saw Witness W7214 and his wife on 17 March 2013. This consultation is described in paragraphs 74-80 of his statement. The account is refuted by Dr Prince. Witness W7214 had undergone three attempts to eradicate his HCV, on each occasion with the most advanced treatment available. Witness W7214's account, and the remarks attributed to Dr Prince are not reflected in Dr Prince's correspondence.

33. The tests I had arranged in January 2013, including the ultrasound, were available to assist Dr Prince with his evaluation, although Dr Prince had to telephone the ultrasound department to obtain the report. These showed an albumin of 27, platelets of 62, and an elevated bilirubin of 59 indicating mild jaundice. It was clear from the investigations and the history that the deterioration and hepatic decompensation had been of relatively recent onset over the previous year. Dr Prince referred Witness W7214 to Birmingham for consideration for liver transplantation and discussed this with Witness W7214 and his wife at length. He also arranged endoscopy to screen for varices. Referral for consideration of transplantation was not urgent, contrary to Witness W7214s suggestion in paragraph 78 of his statement.

34. Witness W7214 was seen on 9 May 2013 by Dr Elsharkawy, Consultant Hepatologist, in Birmingham. Dr Elsharkawy concluded in the letter back to Dr Prince that Witness W7214 had *"a UKELD of 53, although his albumin is reasonable at 34*

(normal)…. It may be that we deem him a little early for transplantation" (WITN3289195).

35. Witness W7214 was subsequently admitted for assessment, added to the transplant list in August of 2013, and found a donor relatively quickly, undergoing liver transplantation in Birmingham on 30 October 2013. This went very smoothly. His Hepatitis C recurred in the transplant, and he underwent a further successful course of antiviral therapy using Viekirax, Exvirera, and Ribivarin in 2015. The former two drugs had not been available prior to his transplant.

36. Witness W7214 has undoubtedly suffered greatly over the years, partly from the anxiety engendered by his chronic liver disease, the severe side effects of his first three attempts to eradicate the Hepatitis C virus, the symptoms of liver failure, and the anxiety engendered by liver transplantation. However, at least since I took up post, the management of his liver disease has been exemplary. We did everything possible to arrest the progression of his liver disease and, when he had progressed to cirrhosis, we referred him on to hepatology (with the suggestion that transplantation be considered) in a timely manner. Haematologists do not refer patients directly for liver transplantation. They always refer to hepatologists first.

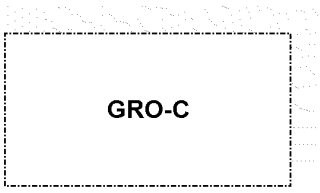
37. Even with retrospect, reviewing Witness W7214's medical records, I cannot see that we missed anything or that we could have done anything differently.

Section 3: Other Issues

38. None

Statement of Truth

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

Signed 

Dated 31/7/2023