Witness Name:	Howard Thomas
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INFECTED BLOOD INQUIRY

WRITTEN STATEMENT OF PROFESSOR HOWARD THOMAS

I provide this statement in response to a request under Rule 9 of the Inquiry Rules 2006 dated 30 November 2020.

I, Professor Howard Thomas, will say as follows:

Section 1: Introduction

1. My name is Professor Howard Thomas, BSc, PhD, FRCP, FRCPath, FMedSci, DOB born GRO-C 1945. My address is known to the Inquiry.

2. I am currently an Emeritus Professor of Hepatology, in the Department of Medicine at Imperial College, London.

   2(a) from 1974 I was a Lecturer, then Senior Lecturer, Wellcome Senior Research Fellow and then held a personal chair of medicine at the Royal Free Hospital before taking up, in 1987, the departmental chair of medicine at St Mary's Hospital Medical School. Following the formation of Imperial College Medical School, I became head of hepatology and gastroenterology, retiring in 2011.

   2(b) At the Royal Free, I was initially an honorary senior registrar under Professor Dame Sheila Sherlock and then was an honorary consultant. As an academic with consultant status, I had responsibilities for teaching, research and patient care.

2(c) bibliography. See Imperial College staff web site under H C Thomas.

3. At various times while at Royal Free Hospital and then St Mary's, I was on the Council of the Royal College of Physicians and of the British Society of Gastroenterology and had been the President of the British and European Societies for the Study of the Liver. I received the Life-Time Achievement Awards of these Societies.

I am Vice President of the British Liver Trust, Chairman of the board of Trustees of the Liver Research Trust and Chairman and Chief Scientist of Riotech Pharma, an Imperial spinout biotech company developing antiviral compounds: currently collaborating with Porton Down and the Institute of Biodefence Level 4 Unit in Galveston to develop these anti-virals against covid 19.

From 1989 until 2010 I was a member and then chairman of the Government's Advisory Group on Hepatitis (providing advice to DH on the control of hepatitis in the community, excluding transfusion transmitted hepatitis), Chairman of the Hepatitis C Strategy Steering Group preparing the English Hepatitis C Action Plan, a member of the JCVI subgroup on hepatitis B vaccination, a member of the DH Infected Healthcare Personnel Committee, a member of the DH Committee on New and Emerging Pathogens Committee 2003-2009 and a member of the Working Party on Transfusion Associated Hepatitis (Meetings in November 1986 onwards). During these meetings discussions took place as to whether the UK should use ALT/AST and anti-HBc screening as surrogates to exclude infected blood as an interim measure prior to the agent of NANB being identified. My view was that these tests should be either introduced, as in the USA and Germany, or evaluated in a prospective clinical trial. I was also in favour of introducing the first-generation Anti-HCV in blood screening 1989

4. I provided evidence to the Archer Inquiry.

I provided evidence on the biology, natural history and pathogenesis of hepatitis C to the Penrose Inquiry on Infected Blood (circa 2003).

I undertook the 5-year review of the Australian Action Plan for Hepatitis C for the Australian Government (2002-3).

I chaired the group developing the English HCV Action Plan.

I was a member of the German Advisory Board on Competence in Viral Hepatitis (2003-2010)

Member of Pan-London Commissioning Group representing liver services 2004-6

I was Chair of the NICE Hepatitis B Guidelines Development Group (2009-2012).

From 2011 to 2018 I was a Trustee of the Caxton Trust, and a trustee and then company director of the Skipton Fund which then became a limited company. During this time, I reviewed applications for payments when requested to do so by Nick Fish an employee of the Skipton Trust/Company: these cases required clinical knowledge and were mainly focused on whether it was probable that the HCV infection had been acquired from blood transfusion and whether the patient had progressed to cirrhosis which would trigger a stage 2 payment. Initially I did this alone and then as the load increased was assisted by Professor Geoff Dusheiko and then Dr Janice Main until the work transferred to Newcastle in c2017/8.

5.

5(a) In my early days at the Royal Free (circa 1975-80) I prepared monoclonal antibodies to hepatitis B surface antigen and hepatitis B core antigen with Professor Janossy in Immunology and, with colleagues from Haemophilia, looked at the value of these in removing hepatitis B virus from coagulation factors being produced by the NHS Blood Fractionation Group at Elstree. These antibodies were also patented and licenced to industry by NRDC and BTG for use in blood donor screening. I offered the anti-HBc monoclonal (Water JA……..Thomas HC; J Med Virology 1986, 19, 79-86. Identification of

a dominant epitope of the nucleocapsid of HBV, RLIT0000498) to Dr McClelland of the Scottish Blood Transfusion Unit in order to reduce the cost of a proposed prospective study to look at the value of ALT/AST and anti-HBc screening as surrogates for identification and exclusion of high risk donors as discussed at the Working Party on Transfusion Associated Hepatitis November 1986 (NHBT0000023_007) (see later at paragraph 38 where I discuss the role of ALT/anti-HBc tests as surrogate assays for excluding potentially NANB containing blood donations). I also described an episode of NANB hepatitis after an infusion of IV gammaglobulin into agammaglobulinaemis patient; this was then discussed and investigated with Richard Lane's group at Elstree Fractionation centre.

5(b) I did general medical on-call take and had clinical responsibility for the patients admitted on my 'Take Day' and also for patients with all types of clinical liver disease. As a clinical academic unit all the clinical staff went on clinical rounds with Dame Sheila Sherlock and were invited to comment on their management of each case after it had been presented by the ward registrar. On Wednesday afternoon a few cases were identified for further discussion and visiting professors were also present. I was particularly interested in viral hepatitis. I was awarded an MRC grant to study NANB hepatitis in the community and also in a post-transfusion setting. Dr May Bamber's salary was paid from this grant. We published several papers on this work.

6. In 1987 I was appointed to the Departmental Chair of Medicine at St Mary's Medical School.

6(a) see list of publications

6(b) see 5 above and 7 below.

Section 2: Your research about hepatitis

7. I did research on the pathogenesis and treatment of viral hepatitis B and C.

I was awarded the Hans Popper International Award for distinction in Liver Disease 1989 and Ivanovsky Medal of the Russian Academy of Medical Sciences 1997 when I

- identified the molecular basis of hepatitis B vaccine escape variants;
- identified the molecular basis of the HBe negative HBV associated with some cases of fulminant hepatitis.

I contributed to development of interferon, lamivudine and tenofovir treatment for CHB in multicentre international trials

I looked at the natural history of CHC.

I helped develop interferon and ribavirin treatment C.

I looked at incidence of NANB hepatitis in haemophilia patients by review of clinical notes and developed tests to determine whether there was correlation of imaging systems (US, CT and MRI scans) and blood tests (pro-collagen peptide levels) with liver biopsies that had been done during routine clinical care, This was in collaboration with the Haemophilia Unit staff;

(i) There was no established treatment for CHC so trials were done to find effective treatment; was it possible to determine whether an individual had progressive fibrosing liver disease needing anti-viral treatment without resorting to liver biopsy?

(ii) Yes. IFN treatment was of value in a proportion of patients and imaging with blood tests were helpful in determining severity.

(iii) IFN was the standard of care until the orally administered drugs became available. Fibro-scanning and imaging displaced the need for biopsy in assessing haemophilia patients.

(iv) Our findings were accurate.

(v) These studies were a collaboration with the haemophilia unit who were in day to day control. The study was then undertaken

retrospectively by review of the case notes where biochemical and biopsy data were recorded.

(vi) Approval was obtained from the hospital ethics committee for treatment trials.

(vii) The work was funded by MRC and Action Research

(viii) Treatment trials involved an oral or written explanation and then oral or written consent. Numbers were in single to double figures but less than three figures and nearly always multicentre.

8,9,10 At this time retrospective studies using information from case notes required approval by the hospital ethics committee but patient consent for case note review was not required. If a publication resulted, then the patient's identity was protected.

If prospective studies were done patient consent would be required and again approval by the hospital ethics committee. In the latter case patient consent would be needed either verbally or written and would be acquired by the physician taking care of the patient usually a haemophilia staff member.

Residual serum after completion of clinically required tests such as HBsAg testing, might be used for, for example, evaluation of putative NANB assays (see later).

11. No

Section 3: Knowledge of and response to, risk

General

12. Hepatitis after blood transfusion was quite common (2-3%) in the UK and perhaps 10% in USA.

13. Most of my information came from the literature and Sheila Sherlock's textbook.

14. We were involved in determining cause, severity and treating patients while prevalence was the concern of the public health doctors.

15. Initially NANB hepatitis was thought to be a benign form of hepatitis characterised by chronic persistent hepatitis which was believed to be non-progressive (Sherlocks Textbook 7th edition; see Penrose Inquiry).

Response to risk

16. Yes. This was particularly the concern of the STD academics and Blood Bank doctors and was mentioned in the Hepatitis C Action Plan. Since retiring I have started public lectures in Corfe Castle on 'Looking after your liver' and other problems (prostate, cardiac....).

17. The risk benefit equation was very much in favour of haemophilia patients being treated with factor VIII concentrates in that this approach reduced the incidence of painful crippling joint problems occurring after intra-articular haemorrhage. So much so the patients requested prophylactic use of the concentrates that allowed the young patients to lead a virtually normal life. In addition, at this time the abnormal transaminase levels were thought to be due to CPH or CLH which were less severe than CAH in that they did not progress to cirrhosis. Furthermore, our data from serum pro-collagen peptide concentrations indicated that levels of collagen synthesis were relatively low, indicating a low risk of cirrhosis.

18. The haemophilia unit staff were very concerned about the significance of the transaminase abnormalities which is why they asked for collaboration from Dame Sheila Sherlock's liver unit of which I was a member.

Section 4: Treatment of Patients at the Royal Free Hospital

19. We treated haemophilia patients with interferon, pegylated interferon and later ribavirin and pegylated interferon by subcutaneous injection having already established that these treatment regimens were partially effective in non-haemophilic patients.

20.

20(a) as stated above, members of the liver unit, particularly myself, were asked to advise on the prognostic significance of an ALT rise in terms of development of cirrhosis. Initially the only way of determining the level of fibrosis was by liver biopsy but latterly pro-collagen peptide levels, fibro-scanning, ultrasound, CT and MRI were shown by comparison with liver biopsy to correlate with degree of fibrosis. This was a retrospective anonymised study to find a non-invasive way of determining fibrosis levels in the liver, the serum specimens were used to measure pro-collagen peptide levels which were correlated to fibrosis levels in liver biopsies obtained during clinical episodes from 1978-1983 and with CT scans done during routine clinical care, (Miller et al; J Clin Path 1988; 41:1039-1043 - available in the exhibit of my first statement at WITN3824004). This study was agreed by the RFH ethics committee.

20(b) Professor Kernoff wanted to establish joint outpatient clinics in which the haemophilia and liver units could help each other decide on best management. I don't know the date that these started but all attendances and opinions were recorded in the haemophilia units case notes.

20(c) no, rather we ran a joint clinic when a consultant or registrar member of the liver unit attended the haemophilia unit.

21. Professor Kernoff had developed a system which minimised exposure of each individual to factor VIII batches. Whenever possible FFP, or cryoprecipitate were used depending on the severity of any bleeding episode and whether the patient already had abnormal LFTs. More details on these issues could be obtained from the haemophilia unit consultants.

21(a) I do not recall the dates.

21(b) Early on it was apparent the patients receiving FFP or cryo-precipitate had a lower prevalence of abnormal LFTs than those receiving concentrates but whether the incidence was the same after NHS and commercial concentrates was unknown. I thought the system

minimising exposure to large numbers of unit of plasma was sensible until we had a clear view of the significance of the elevated transaminase levels. It was very well organised by Professor Kernoff and his colleagues.

21(c) he did not know the significance of elevated ALT levels hence he asked the Liver Unit to help.

22. Yes I believe serum samples were kept: I don't know what Professor Kernoff's intentions were and what the patients were told.

As mentioned earlier I kept residual serum left after testing for the existing hepatitis viruses (HAV, HBV, CMV, and EBV etc) because only by excluding involvement of known viruses could one diagnose NA, NB, NC, NE hepatitis. These sera were eventually used to determine whether candidate NANB assays identified this group of sera indicating that they might be identifying a viral antigen and be part of the virus causing the abnormal transaminases in the haemophilia patients. We evaluated one radio-immunoassay which looked encouraging but was subsequently found to not be of viral origin (Luo et al).

Some sera were sent to my laboratory for improving existing clinically indicated tests (HBsAg assays) and were kept for up to 2 years, determined by the capacity of our deep freeze. My laboratory was developing monoclonal antibodies to the component antigens of HAV and HBV to create better diagnostic assays. We had found that some sera which were negative for HBsAg in the routine Abbott assay were positive in our monoclonal antibody assay which raised the possibility that haemophilia associated NANB might be a variant of HBV which was only detected by the highest sensitivity monoclonal antibody based tests. We subsequently excluded this possibility by molecular sequencing.

Our antibodies were licensed by BTG to several companies and the assays are used for blood screening and routine diagnostics.

The prospective study of 30 patients recording the incidence of abnormal LFTs and persistent abnormalities after infusion of cryoprecipitate, NHS factor VIII and IX or USA factor VIII concentrate were observations recorded during clinical episodes to either stop bleeding or as prophylaxis prior to surgery (High risk of NANB after first exposure to volunteer or commercial factor 8: effect of prophylactic immune serum globulin Brit J Haem 1985 - available at PRSE0003439). This was done because abnormal liver tests had already been reported in USA and it was important to know in each case whether the abnormality had been there before treatment in which case it would not be due to PT-NANB hepatitis but some other cause such as alcohol ingestion or autoimmune problems. This information provided useful information for the care of the individual and, by later analysis, to determine which treatments were most safe. A further 29 patients were studied retrospectively regarding prevalence of persistent abnormalities. In a small subgroup the value of preventing the development of the hepatitis using prophylactic immune serum globulin was determined. The study was supervised by the Haemophilia Unit and Professor Kernoff states in the paper that the study was approved by the RFH Ethics Committee and all patients gave verbal consent. I am not able to provide any further information on this concerning the identity of the patients. My contribution as a hepatologist, was to provide advice on management of the liver abnormalities if they became persistent: at this time interferons were available and were beginning to be used to treat viral hepatitis in the USA and by our group and others in the UK. Jacyna M .....Thomas HC; BMJ: volume 298 14th Jan 1989 page80. Randomised controlled trial of interferon alpha in chronic NANB hepatitis (RLIT0000495).

23.

The study established that the risk of PT-NANB was the same after NHS (volunteer donors) and US (paid donor) preparations both of which were produced from around 1500-5,000 donations. Immune globulin appeared to be of possible value.

24.

24(a) Prior to these biopsy results CPH and CLH were thought to predominate in NANB hepatitis and to indicate a benign prognosis (see Dame Sheila Sherlocks book cited in Penrose). As a result of our study and others this view changed as was reflected in the subsequent volumes of Sherlock's book which noted that 20% went on to cirrhosis with a risk of liver failure and carcinoma. This group with progressive fibrosis was identified as those with CAH. We subsequently undertook comparative studies of procollagen 3 peptides with these biopsy observations to see if these were predictive of CAH and progressive fibrosis.

24(b) after this study when around 40% had CAH which in the context of CHB, progressed to significant fibrosis and cirrhosis.

24(c) the liver biopsies were in the main done by the registrars or senior registrars (lecturers) on the liver unit as part of the specialist service. Some were done under ultrasound imaging by the staff of the radiology unit at the RFH. The biopsies were done for standard clinical reasons to assess the prognostic severity of the liver disease, in particular whether CAH was present which puts the patient at risk of cirrhosis and then primary liver cancer (HCC). Verbal or written consent was always obtained and the risk of haemorrhage and how the clotting tests would be corrected prior to biopsy, was explained. This was standard clinical practise for all chronic liver disease patients. The biopsies were continually reviewed once our retrospective study and the published Sheffield study had shown that some patients had CAH with fibrosis and some (c20%) had actually progressed to cirrhosis. Once cirrhosis is present in any type of liver disease, yearly ultrasound scans are undertaken because of the risk of nodules developing into hepatocellular carcinoma. Once cirrhosis is present further monitoring is done by ultrasound CT or MRI and no further biopsies are indicated unless to identify whether a nodule seen on imaging was malignant. Early detection of HCC is treated by liver resection or liver transplant (that also corrects the haemophilia) but

once the HCC gets larger and at multiple sites no treatment is possible. These were standard investigations in patients with persistent transaminase elevation to assess the degree of liver fibrosis. Now serial fibroscan monitoring would be done.

24(d) Professor Kernoff minimised the use of concentrates (NHS or commercial) particularly in young patients who did not already have elevated transaminases, preferring FFP or cryoprecipitate. Once patients had elevated ALT this was no longer so important. It was clear that the risks were similar irrespective of whether NHS or commercial concentrates were used (see earlier).

25.

25(a) as far as I know. These patients would come to the joint clinic when viral suppressive therapy would be discussed. Initially this was with interferon and subsequently with lamivudine or tenofovir.

25(b) patients would be told it is a chronic infection which cannot be cured but can be suppressed for life thereby preventing the risk of cirrhosis and liver cancer. They would also be told that there existed a vaccine to prevent sexual and neonatal transmission.

25(c) they would be informed in the haemophilia unit or in the joint clinic.

25(d) they would be told that it is a chronic infection and between 40% and 80% can be cured depending on the viral genotype. More recently it has been shown to be 100% curable with 4 weeks therapy with small antiviral drugs.

25(e) this would usually be dealt with by the haemophilia centre staff before the patients were referred to the joint clinic.

26. I was not directly involved except when I heard about this data in the joint clinic. This was a collaboration between the haemophilia unit and the immunology unit under Professor Janossy. The suggestion was that T4/T8 rations were indicative of AIDS. I pointed out that my published studies in other

liver diseases indicated that these ratios were altered in CHB, NANB, EBV or after immunisation to HLA proteins during impure concentrates and were not therefore necessarily indicative of AIDS. Independently the immunology department were examining T4/T8 ratios in haemophilia patients without my involvement.

At this meeting someone also raised the issue of whether anti-HBs which was prepared from gay patients plasma and used to protect various groups of patients, including neonates born to CHB mothers, against HBV infection, was safe (i.e. would it transmit AIDS etc which was known to be occurring in the gay community in USA). I had shown and published with the Bureau of Biologics (Director Bob Gerety) that murine monoclonal anti-HBs neutralised HBV in chimpanzee experiments and could therefore be used as an alternative to anti-HBs prepared from gay donor plasma to protect neonates. These antibodies were also used to control the antigenicity of and immune response to, most HBV vaccines.

The T4/T8 ratios were used to monitor progress of HIV and whether they were done was determined by the haemophilia /unit staff.

27. Infectivity would be discussed as far as HBV is concerned because there was a vaccine. The issue with NANB was unclear until the virus was cloned and NAT became available in 1990 allowing quantification of the virus in patients' blood. After these tests were available infectivity could be determined by the level of viraemia and it became evident that HCV did not readily transmit sexually or neonatally from mother to infant. Once this was known this issue of infectivity would have been and was discussed by the haemophilia or hepatology staff.

28. This was discussed as far as HBV is concerned but not initially regarding NANB (HCV). See Q27. I was not involved in advice relating to HIV.

29. You need to discuss issues around HIV with the haemophilia staff. I had little involvement at a clinical or research level with HIV issues but some patients, of course, had both HCV and HIV infection: in these patients the liver disease progressed more rapidly.

30.

30(a) CHB was managed, as far as I can remember, via the joint clinic;

30(b) interferon initially and then lamivudine and then tenofovir were sequentially the NICE standards of care. The later drugs were orally administered and so were much better for treating patients with coagulopathy; interferon, which had to be given by injection, was more problematical. Because of the special needs of patients with coagulopathy they were not involved in clinical trials, but retrospective studies of patients treated on an individual basis were collected together and reviewed. This was considered as part of their routine care and if published to disseminate best care, would be anonymised.

30(c) the liver unit nurses went into considerable detail for interferon treatment because there were significant side effects particularly fever and malaise. In addition, depression was a problem, so we entered into a clinical collaboration with the psychiatrists; this often involved giving anti-depressants;

30(d) because these oral drugs only suppressed HBV rather than stimulating the immune system to control the virus as was the case with interferon, lifelong follow up was indicated and was supervised in the joint clinic as far as I can remember.

31.

31(a) care for NANB didn't really start until 1990 when nucleic acid testing (NAT) for quantitating viraemia, became available. This care was managed on a daily basis by staff of the Haemophilia Unit with intermittent advice from the Liver Unit who were treating non-haemophilic CHC. By 1990 we had done a randomised trial of interferon in non-haemophilic NANB, subsequently shown to be CHC cases by being anti-HCV positive (Jacyna M et al BMJ; 1989, RLIT0000495 as referred to in paragraph 22 above). When this trial showed interferon to be beneficial, we started to consider using

interferon in CHC in haemophilia patients. This was delivered by the Haemophilia Unit staff because the interferon injections were given subcutaneously after an injection of cryoprecipitate or factor VIII to minimise the risk of local bleeding, which ever was being used as their routine coagulopathy care.

31(b) initial treatment was with interferon, PEG interferon, then PEG-IFN with ribavirin and finally direct antivirals which most recently can cure up to 100% of patients with one month of Rx.

31(c) provided by anti-viral unit nurses.

Section 5: Treatment of Patients at St Mary's Paddington

32. When I left the Royal Free to go to St Mary's, which did not have a haemophilia unit, I was no longer involved in haemophilia cases with CHC but took care of many non-haemophilia patients with liver disease.

While at St Marys I set up an eight bedded Clinical and Research Unit to treat patients with chronic liver disease. This included chronic hepatitis B and C. The latter excluded those with haemophilia who were cared for in the Haemophilia and Liver Units at the RFH where Professor Geoff Dusheiko took over my clinical responsibilities or at St Thomas's Haemophilia Unit. I had some patients with post-blood/plasma transfusion viral hepatitis (PTH) and infection transmitted by other routes including IVDU. Many patients going through this unit were included in multi-national clinical trials. All patients included in trials gave written consent after being given a written explanation of the nature of the trial.

32(a) my Liver Foundation funded nurses and NHS nurses, under the direction of our senior nurse, Louise Campbell, and the consultant staff of the Clinical Centre, routinely informed patients of the trial protocols and the side effects of the medication being used. All trials compared any new treatment with standard of care treatment.

32(b) interferon, interferon and ribavirin and then pegylated interferon and finally protease and polymerase inhibitors produced by commercial pharma companies. These were usually multicentre randomised controlled trials.

32(c) our hepatitis nurses enrolled patients into trials which involved detailed patient information sheets and written consent as well as institutional ethics review

32(d) long term follow-up by fibroscan was undertaken to document whether fibrosis reversed once the virus was cleared

32(e) these were cared for by the paediatricians

32(f) we had a psychiatrist and social worker working with my unit in the trials unit

32(g) we had considerable difficulty funding the building of the 8 bedded unit and virtually all patients were taken care of by my Foundation funded nurses and research fellows. The Liver Research Foundation received funds from the profits of the Journal of Viral Hepatitis which I founded and with my colleagues edited for 25 years. I negotiated with Blackwells that 20% of the profits of the journal would, instead of coming to me, go to the Foundation so that my colleagues who supported the journal could also benefit. Graham Foster , an international editor and myself also received 5-10k each per year .This arrangement allowed me to put £350,000 to building the Clinical Research Unit on the 10th floor of the QEQM Building at St Mary's; I also received around 100,000 from the Hesketh/Guinness family and friends in return for naming the Unit the Robert Hesketh Research Unit. The nurses were later funded by the NHS and money from trials.

Section 6: Safety of blood products

33. While at RFH I was investigating PT-NANB (now HCV), CHC, and CHB prevention, prognosis and treatment. I also wrote papers on Primary Biliary Cirrhosis, autoimmune CAH and primary liver cell cancer.

I was also working with the immunology and haemophilia units on producing monoclonal antibodies to HBs antigen and HBc antigen initially with the intention of making improved diagnostic assays which we achieved; these antibodies were developed with funding from NRDC and BTG. Substantial Royalties came to the RFH Medical School and the staff also received sums personally, as several companies used them to make assays for use in hospital diagnostic laboratories and for blood screening.

I also investigated the capacity of the anti-HBs monoclonals to neutralise HBV in chimpanzee studies with the Bureau of Biologics in Bethesda USA. Once we had shown that these antibodies neutralised HBV and the epitope to which the antibodies bound it was possible to use them to standardise HBV vaccines and make sure that the evoked response would neutralise the virus. These antibodies were of great value to vaccine manufacturers and to international regulatory bodies. No charge was made for this use, but Royalties were paid for the diagnostic and blood screening uses.

We also investigated whether these antibodies might be used to pull out residual HBV from preparations of coagulation factors and other blood products and whether they might be an alternative to hyperimmune human globulin in stopping neonatal transmission of HBV. They were not used in this area. When working with the MRC Agammaglobulinaemia group at the RFH (Dr Webster et al) we also described an outbreak of NANB in agammaglobulinaemia patients. This resulted in my being invited to consult with The Blood Products Lab at Elstree to help them deal with the problem of viral contamination of blood products in the context of NANB and also HBV transmission during preparation of gammaglobulin.

34.

34(a) Please see above

34(b) None;

34(c) Please see above. Heat inactivation of gammaglobulin was being investigated following our paper on NANB associated with the use of gammaglobulin. This involved studies in chimpanzees.

35. Serum samples during the recovery phase of acute NANB hepatitis were tested for a precipitin reaction against acute phase serum from the same patient. If this appeared it was interpreted that the recovered phase serum contained antibody to a component of the NANB virus present in the acute phase. This recovery serum was then used to extract globulin to coat micro-titre plates to form a radio-immunoassay.

We then determined the prevalence of the antigen identified in the RIA within groups of patients which were known to have a high incidence of NANB. The serum specimen used for these studies were left after routine diagnostic testing for HAV and HBV and were stored in diagnostic groups eg renal disease, STD clinic, haemophilia, CHB, IVDU, hospital staff etc, without knowledge of the name of the patient. This work was done on the serum left after routine clinical service testing during the diagnosis of NANB hepatitis which involved serological exclusion of HAV, HBV, CMV. EBV etc. This RIA test system was encouraging but was subsequently found to be due to a rheumatoid factor and not to be detecting the NANB virus. This work was funded by the MRC and did not involve BPL, companies or DHSS. This work was done by Dr Luo a visiting fellow from China.

36. Please see 35 above.

37. The above RIA (Luo et al) was discussed and ways of determining whether it was detecting NANB were considered. Professor O James offered access to his panel of post-transfusion hepatitis sera and the USA validation panel was also discussed. Funding was considered. Our putative assay was shown not to be detecting the NANB virus but an anti-globulin.

38. The dates appear to be wrong; those on the papers are 1982 and 1984 and not 1987. The issue being addressed is whether we should introduce anti-HBc/ALT surrogate NANB screening of blood donations. My view was that we should, but we might have to do a trial first. In order to reduce the expense of a hypothetical trial, I offered to provide my monoclonal antibodies to HBc rather than the trial group haying to buy them; my antibodies had been licenced to Roche and were being used commercially. This offer was made to Dr McClelland at the meeting because he was willing to do such a study. I gave him the telephone number of my colleague Dr (now Professor) Peter Karayiannis to liaise (Penrose Inquiry text has copy of Dr McClelland's notes of meeting bearing PK phone number. I'm not sure what happened subsequently but the trial did not come about.

On 3rd January 1984 Dr Lane wrote to convene a meeting because Lever, Webster and I had reported cases of NANB after IV immunoglobulin in the Lancet and Dr Lane of Elstree wanted to discuss the implications for his plasma fractionation program.

The anti-HBc and other monoclonals were owned by NRDC who had funded the work; they were dealing with the commercial arrangements on behalf of the Royal Free

39. Having not introduced anti-HBc/ALT screening in 1980s (see 38) I thought the first anti-HCV assay should be introduced on ethical and economic groups in 1990 as argued in my paper in Medical Virology 1991 vol 1 p 67-71 (Brown and Thomas); I cannot add any more.

Section 7: UKHCDO

40. See above 38 and 39

Section 8: Pharmaceutical companies and medical research / trials

41. I was involved in advising MSD on whether they should include HBc antigen as well as HBs antigen in their vaccine. I had shown that whereas anti-HBs neutralised the virus, injection of anti-HBc caused chronic infection

(experiments in chimpanzees with Dr Bob Gerety of Bureau of Biologics in Bethesda USA (see Thomas,,,,,and Gerety).

Interferon trials were undertaken with Roche or Schering Plough. Other antiviral drugs subsequently became available and were evaluated in randomised multicentre trials; all trials were evaluated and given approval by the local hospital-based ethics committee. Consent, usually written, was obtained prior to entry into a trial.

42. Main area in this respect was declaring commercial interests if presenting papers in USA; in this area I went to AASLD in Chicago every year and if presenting would declare interest. Medical school also wanted to know consulting arrangements. I did comply. I cannot recall dates when these rules were introduced. The main focus of med school was to regulate how much private practise was done.

43. I used some factor VIII concentrate left over after treatment of patient (given to me by haemophilia unit) to try to clone NANB; this was unsuccessful. I received no funds for this.

44. Yes, from clinical treatment trials.

45. Yes, via ethics committee. We had an administrator who dealt with all this admin.

Section 9: Involvement with the financial support schemes

46. I was a member of the Caxton Foundation and provided information on medical issues but was not directly involved in evaluating applications except when the applicant appealed a decision when, as far as I can remember, these applications came to the whole committee. As far as I can remember the grant application committee met monthly. As an example, I gave a lecture on NANB/HCV etc to staff and board members of Caxton and Skipton early on (CAXT0000109_113).

47. I subsequently became involved with the Skipton Fund where there was a direct need for someone medically qualified to evaluate applications and, in

particular to determine, usually from non-invasive test data, whether the patient had developed cirrhosis which triggered a stage 2 lump sum payment and regular subsequent payments.

48. These payments changed in value from time to time as determined by the DH.

49. Following the closure of these charities in approximately 2016, I was appointed a member of the Reference Group involved in modifying the system of ex-gratia payments to include additional diseases that triggered automatic stage 2 payments and to identify 'special category' stage 1 patients that had compromised health, particularly cognitive and mood problems. I continued to review applications for stage 1 and 2 payments (EIBSS) for NHS Business until it moved to Newcastle in 2018-9 when Professor Bassendine took over. I also prepared a statement for the Judicial Review relating to the clinical logic behind the ex gratia payments.

Section 10: Establishment of the Skipton Fund and the Caxton Foundation

50. The Skipton Fund was to make ex-gratia payments to those infected with blood or blood products using criteria determined by the department of health and their advisors of whom I was one. I was not involved in the setting up of the Skipton Fund and was involved in assessment of individual cases from 2010 as an unpaid volunteer.

The criteria for stage 1 and stage 2 payments and the amounts involved changed over the life of the Fund.

51. The Caxton Foundation was formed to allow a more individual approach to applicants' needs.

52. see Q51.

53. The Skipton Fund and Caxton Foundation were funded by the NHS and overseen by Mr Peter Stevens in the early years. Once the charities became Ltd Companies, he became the chairman of the boards and we appointed a CEO (Jan Barlow)

54. I do not know whether there was involvement from the Department of Health in setting up the Skipton Fund and Caxton Foundation. There must have been because DoH was a source of funding.

I made no contribution to this process. There was no discussion as to why the government decided to distribute funds through the AHOs rather than directly. I suspect this was related to the same reason that the payments were ex-gratia rather than compensatory.

There was no discussion of whether chronic CHB should be involved at this stage. There was however a question asked at the reference committee by the Haemophilia trust representative that I cannot recall. It should be in the meeting minutes.

I thought the scheme for HIV infected patients was more generous than that for HCV. I was subsequently asked to write a paper on behalf of the Department of Health as part of their response to a judicial review in which this issue was raised. I reviewed the morbidity and mortality of the two patient groups to act as the basis for modifying the HCV payments to be more equivalent to those for HIV. See a draft copy of my statement exhibited at WITN3824009.

55. I was not involved with the MacFarlane trust or the Liaison group with Caxton.

Section 11: Structure and Operation of the Caxton Foundation and Skipton Fund

Appointments of trustees/directors

56. The process for appointment of trustees and eventually directors was handled by the CEO and board chairman Mr Stevens as far as I am aware.

57. see Q56

58. see Q56

59. I was aware that head hunters were involved in the appointments to the Caxton Foundation because it was difficult to get the right people. I suggested that Prof Dusheiko joined me and worked through Mr Stevens. I was appointed

to the Caxton Foundation when Charles Gore resigned: I was already involved in application assessment when asked to join the Skipton board.

60. I do not know whether this was laid down by the department of health.

61. there was no defined term as far as I know.

62. As far as I was concerned, I worked as an unpaid volunteer. When NHS Business took over I was living in Dorset and was not prepared to travel frequently to Newcastle because this would entail 7+ hours travelling each way and an overnight stay and I was already 70. The NHS Business offered my successors payments.

63. I cannot recall precisely whether there was overlap in the trustee/board members of Skipton and MacFarlane. Before my involvement I think this was the case.

Structure of The Caxton Foundation and Skipton Fund

64. All the AHOs shared premises, some staff but had separate offices. I am not aware of any data sharing or confidentiality issues. However, some patients received ex-gratia payments from more than one trust.

65. I do not know why the Caxton Foundation acted as an employer to all the AHOs

66. I am not aware of the relationship between the different AHOs.

67. The senior management of both Skipton and Caxton were shared. I am not aware of any particular difficulties between the management and the board.

Relationship with government

68. Initially both The Skipton and The Caxton were independent. In particular, there was no limit on the funding the government provided to Skipton. All patients meeting the criteria in Q47 were funded. The level of funding provided to individuals was determined by the government.

After NHS Business took over, I was unaware of the relationship.

69. As mentioned in Q68, the size of the payments for stage 1 and stage 2 were determined by the Department of Health but there was no restriction on the numbers of patients receiving these payments. Late on in the life of The Skipton Fund, at the patients request, special payments were made for a subgroup of stage 1 patients who had mental problems including depression and difficulties with concentration. Because I had published papers on "brain fog" and depressive problems I was asked to join the Reference Committee to introduce these modification of the stage 1 payments and to add additional criteria for stage 2 payments. See "SFL Annual Financial accounts 2015" attached for details.

During my involvement with Skipton and Caxton in November 2014, I wrote a letter to the Secretary of State/Minister of health indicating that we were following the patients with pre-cirrhotic stage 1 disease which would progress to irreversible cirrhosis if not treated. This would put them at risk of liver failure and liver cancer. 90 patients every year were in this pre-cirrhotic stage and I felt that it was important, as a result of our "duty of care", that these should be treated with the new antiviral drugs which had not yet been considered by NICE. Please see a copy of this letter exhibited at WITN3824008

70. See letter in WITN3824008

71. I do not know about the contact with DWP and its predecessor but at the Reference Committee an advisor to the DWP was present and provided helpful information. You may get information from the minutes of this committee at which I was present but cannot recall the details of.

72. Department of Health funded both Skipton and Caxton and worked through 2 people: Mr P. R. Stevens (who was chairman of the directors) and the CEO of Skipton when it became a limited company (name of whom I do not know).

Section 12: Funding/finances of The Caxton Foundation and Skipton Fund

The answers to the questions for Q73 – 76 in this section can also be found in the annual reports, an example of which can be found at SKIP0000057_052.

Skipton Fund

73. Funds were provided by DH and allocated according to a process determined by DH and updated often under pressure from devolved administration schemes and by patient pressure groups. In 2016-17 the sum distributed by Skipton was 41,545,739

74. None

75. Not aware. Contact with Gov and DoH was via CEO and Chair

76. Skipton cumulative awards are in Skipton Board minutes

77. As far as Skipton funding was concerned the sums awarded to stage 1 and stage 2 payments are dealt with in SKIP0000057_052 and were determined by the Department of Health. There was no limit put on the number of patients that could be awarded these payments.

78. This was dealt with by the chairman of directors and the CEO. I do not know whether requests for additional funds were made.

79. Yes, I think so. Between CEO, chairman and the department of health.

80. The chairman and the CEO met with the department of health, but the other members of the board did not (as far as I recall).

81. Not as far as I know.

82. There were no other sources of funding other than the department of health.

Financial management/governance.

83–93: I cannot answer the questions regarding the Caxton Foundation funding. I only contributed to the Audit Committee and attended the board meetings.

Section 13: Identifying beneficiaries for the Caxton Foundation and Skipton Fund

94. CEO

95. We had a list of all stage 1 patients and I believe this was used to undertake a census of whether recipients were satisfied or dissatisfied. This was undertaken by the CEO and reported to the board. It will have been recorded in the meeting minutes to which I assume you have access.

96. See my answer to question 95 above.

97. I do not know whether or not the department of health made other attempts with the devolved administrations to increase awareness of the two charities.

98. I believe the CEO used the list of stage 1 patients (referred to in Q95).

99. I think the attempts made to contact people were reasonable.

Section 14: Eligibility for The Skipton Fund and The Caxton foundation

100. the eligibility criteria did change with time and I was involved in the medical side of this process. This is summarised in SKIP0000057_052 and was again reviewed at the Reference Committee when additional stage 2 criteria were added (MPGN and Cryoglobulinemia) and a special category for mental problems was added for stage 1. I was asked to present the evidence for this because I had shown that the Hep C virus was present in the brain and interferon caused long term effects on mental function. The main manifestations of the infection of the brain were depression and cognitive problems which were difficult to differentiate from mental problems unrelated to Hep C and interferon.

101. The policies of the Skipton Foundation and Caxton Foundation were available online and were summarised on the application forms. I was familiar with the Skipton but not the Caxton Foundation in this respect.

102. Yes. I provided information on new criteria, particular at the Reference Committee meeting.

103. The main change is referred to in SKIP0000057_052.

104. I was not involved.

105. I was not involved.

106. Medical opinion was important for a proportion of the decisions made by Skipton during the awards process. Certain criteria, when fulfilled, were dealt with by Mr Nick Fish (our long-term administrator). An example would be that all haemophiliacs receiving concentrates would automatically qualify for a stage 1 payment. Medical opinion was sought when the potential source of infection was a blood transfusion. I had to decide whether it was probable (greater than 50% likely) that the infection was related to an NHS blood transfusion. This involved checking with the GP and hospital consultant notes to see whether this was recorded. These were difficult decisions. Sometimes we would ask for evidence of a surgical operation i.e. photograph of a scar or surgical survey indicating how frequently blood would be used during the procedure.

The second area where medical opinion was required was whether the patient had cirrhosis as this would trigger a stage 2 payment. Here GP and hospital letters (when available) were examined.

As the work increased, I realised it was important to have someone else involved and asked Prof Dusheiko to join me as a trustee/director and expert clinician. Once he became involved, we would work together on the more difficult cases.

I should also point out that the patients could appeal any decision made and there was a verbal dialogue between Nick Fish and the applicant before the any appeal went ahead. I believe this was appreciated by the patients involved as Nick would suggest evidence the patient might use in their case. This was lost after transfer to Newcastle.

107. At the inception of the stage 1/stage 2 payment process a subgroup developed some criteria for determining the presence of cirrhosis which was the most difficult role of the Skipton medical staff. This was presented in the application form. It was reviewed at the Reference Committee.

108. See answer to Q106.

109. Once the special category subgroup of stage 1 cases was introduced the patients' GP/hospital consultant had to indicate on the application form whether depression or cognitive problems were not related/possibly related/probably related to Hep C infection. By this time, it had become clear that it was impossible to differentiate these HCV related problems from those commonly occurring in the general population and unrelated to HCV. Only if the form said it was not related to HCV would the application be denied, hence the majority of stage 1 received the special category payment.

It also became apparent to me, and I think also to my colleague, that interferon treatment could cause long term problems such as depression and hypothyroidism which did not resolve after viral clearance.

110. Yes, there were differences well known to the department of health and changes were made.

111. Yes, criteria did evolve as experience developed. Both Prof Dusheiko and I continually referred to the literature to see whether there was any new evidence that should be taken into account. Several new causative associations of HCV with new syndromes were incorporated in the application forms. See SKIP0000057_052.

112. Yes, there were concerns about some individual decisions and this resulted in:

- Discussions on the phone with Nick Fish to make the patient aware of additional information that would support their application.
- If finally the application was unsuccessful the patient could go to the appeal panel.

The applicants to Caxton did not like the awards being subjected to means testing

The Skipton applicants wanted a single payment in much the same way as happened in Ireland.

In general patients were concerned about the evidence needed to establish that the PTH was probably (greater than 50 % likely) due to NHS blood and not other means of infection such as IVDU, tattooing etc. All haemophilia patients who had received factor VIII concentrate were automatically accepted as infected by NHS material; this was established by several prospective studies showing that the incidence of abnormal ALTs after concentrate infusion, both in the literature and our own study, was almost 100%. Nick Fish signed off on these cases without clinical input. In cases of blood and plasma transfusion, we had to say that it was more than 50% likely to be due to transfusion. In the absence of GP or hospital case notes this was very difficult. Occasionally we had evidence of a major operation where in virtually all cases blood transfusion would have been necessary e.g. cardiac valve replacement or major trauma resulting in pelvic or femur fractures. These cases usually involved both Professor Dusheiko and myself and involved detailed consideration. In my view this was as objective as possible.

In the absence of case notes it was almost impossible for Professor Dusheiko and myself to provide this level of certainty. In many cases all we could do was to exclude non-transfusion related modes of transmission such as body piercing and IVDU. Sometimes we had evidence of surgery which did not usually require transfusion but infrequently did; this again required clinical judgement.

The appeal committee essentially provided a second opinion; often no additional information was available so their decision might again have been subjective but was final. All patients had the right of appeal and there was no down side to appealing so many gave it a go.

The DoH set up the scheme and initially Nick Fish and the non-medical directors made judgements without medical input. At this time the rate of appeal was understandably high. It is possible that I was asked to help because I was a clinical hepatologist with potentially useful clinical expertise; you need to ask the chairman why I was asked to join the assessment process. I was not aware that the appeal rate was unsatisfactory once I was involved and the rate of successful appeal was around 30%.

The introduction of the SCM payments made evaluating stage 1 cases even more subjective and as a consequence the clinical assessors relied on the GP and consultant opinion of whether the CHC was the cause of the mental problems. Most applicants for SCM were successful.

I think the criteria for stage 2 payments were on much better ground because objective criteria were available.

113. The exclusion of patients who have cleared the virus from ex-gratia payments was initially thought reasonable in that Koch's postulates state that a causative role is supported by the presence of the infective agent with the syndrome and the absence of the infective agent on recovery.

Section 15: Decisions on substantive applications.

The Process

114. A review of the symptoms and the pathology associated with HCV was commissioned by the department of health from an external consultant (WITN3754072). This resulted in the two new automatic criteria mentioned earlier being accepted and also supported the introduction of the special criteria of cognitive abnormality and affective problems (depression). I think the person who prepared this for DoH was a Dr Brunton. I have no further information. I think the Haemophilia Society were involved at this stage; they wanted the review on symptoms that should be accepted as causatively related to HCV infection.

115. The written policies for the determination of the applications of the Skipton Fund were presented in the application form.

116. See earlier answers

117. Yes, as discussed previously.

118. See annual reports from Skipton of which an example is available at SKIP0000057_052.

119. Reasons for refusing an application were provided to the applicant.

120. Yes, patients could contact Nick Fish and other support staff by telephone.

121. All applications were retrospective in that the infections happened prior to applications being made.

122. Applicants were assisted by GPs, hospital consultants and HCV specialist nurses and received help from Nick Fish via telephone.

123. See annual report example in SKIP0000057_052.

124. I cannot recall the content of the discussion alluded to in the question. In general, once we had seen several cases where a positive outcome had been agreed this became a precedent for the future.

Skipton Fund

125. In order to qualify for stage 1 payment, the applicant must provide evidence of receiving NHS blood or blood products. All haemophilia patients who had received Factor 8 concentrates were assumed to qualify. The eligibility for stage 2 payments is described on the application form and involves AST/ALT ratios, platelet count and any additional information such as scanning or endoscopy. Please see application form.

126. Nick Fish, a Director and increasingly myself and Prof Dusheiko were authorised to sign off stage 1 and stage 2 payments. I do not know what happened when NHS business took over.

127. The medical information on the form had to be signed off by, as far as I can remember, a hepatitis nurse or a consultant hepatologist/gastroenterologist. These individuals had access to the notes.

128. I cannot remember these figures. I think there was a review which looked at this issue. Every year in the annual report the number of patients awarded payments was recorded. The annual report of Skipton referred to above in SKIP0000057_052 should give you an idea of the information usually present. I would suggest that you obtain these annual reports which will provide you with a lot of the information you require. See my answer at paragraph 112

above. It was difficult because most hospital notes were no longer available. Sometimes we found useful data in GP notes.

129. see Q109

Caxton Foundation

130. As far as I was aware, each application was considered on its own merits in the same way as the Skipton Fundoperated. I gather experts from the DWP and board members with social care expertise were on subcommittee assessing applications.

131. I don't have this information but the CEO no doubt has this.

132. I cannot recall the rationale here, it should be in the Caxton Foundation minutes.

133. I was not involved in determining the outcome of applications through this foundation. A subcommittee dealt with this.

134. 134: I don't know why this was reduced in 2014. Please contact the CEO.

135. The Skipton Fund payments were standard. There was no flexibility in this binary system. It is possible that the Caxton Foundation was founded to help individuals with more intense problems but I'm not sure there was any coordination between Skipton and Caxton.

Non-financial support

136. Yes, this support was available and was administered through the office staff. The CEO of Skipton would know the details.

Section 16: Complaints and appeals

137. A patient could request review by the appeals committee. This was a group of 3 people including a medic (Prof Peter Mills), a lawyer and a blood transfusion expert. You should have minutes from these.

137(a) Appellant gave evidence in writing

137(b) I don't know if they could have a representative to advise them but their consultant usually provided written evidence

137(c) I don't know the criteria for a successful appeal, please contact the appeal committee

137(d) The original decision maker was not involved

137(e) Written reasons were given for rejection of an appeal either by the appeal committee chairman or the staff of the original assessment group.

137(f) I can't recall if there were time limits in place.

137(g) The appeal was made by the infected individual rather than the affected.

138. My involvement with this was minimal, you'll need to contact the CEO of The Caxton Foundation.

139. You need to contact the appeal committee concerning the criteria used in their assessments.

140. I do not know how often appeals were successful. As far as I can recall, there were very few appeals. The proportion that were successful will need be obtained from the appeals panel chairman. I think the figure was around 30% of a small number of appeals. The original decisions were relatively well accepted by the applicants.

141. Because we only recorded complaints, we had no idea of the numbers who were satisfied with the outcome of their applications. The CEO therefore organised a survey which you should be able to obtain. I recall that the majority of applicants to the 2 charities were satisfied. It did however become apparent that applicants to Caxton did not like the "means test" component of the process.

142. This information is present on the letter informing the applicant of the outcome of the initial application.

Section 17: Engagement with the beneficiary community

143. I recall from the board meetings of Caxton that this was of concern and the CEO arranged the census referred to in Q141. As far as Skipton was concerned, I believe that the Department of Health reviewed the sums allocated in stage 1 and stage 2 payments. This was why the process was reviewed by the Reference Committee and resulted in an assessment of whether the payments to HCV infected individuals was equitable in comparison to those given to HIV patients. In Addition, the mental symptoms of stage 1 patients were deemed significant and required additional payments resulting in "special category payments".

144. You need to contact the CEO of Caxton.

145. Because the Caxton and Skipton shared a CEO, who dealt with the patient group meetings, integration was good.

Section 18: Relationship with other organisations

146. The Skipton and Caxton had contacts with the Haemophilia Society This was usually through the CEO.

147. For details of the relationship with the Haemophilia Society you'll need to contact the CEO. I had the impression that the Haemophilia Society thought that payments were inadequate, but I don't know the details of this.

148. **GRO-A**

149. I don't know.

150. See Q 149

151. I of course was in contact with the members of my department at St Mary's but had no contact with the haemophilia directors or society. This was because after leaving The Royal Free my focus was mainly in treating other groups of chronic HCV infected individuals.

Section 19: Reform of the Skipton Fund and Caxton Foundation

152. I was involved in the Reference Committee.

153. I thought it was unfair that dependants did not receive support after a patient had died. My main contribution to the Archer inquiry was that widows, widowers and their dependents should be supported. This was subsequently implemented.

154. I am assuming that you are referring to the winding up of the charitable funds/Ltd companies and their work being taken over by NHS Business. Initially we made this work but when the whole service was moved to Newcastle this was impossible for staff with family commitments and retired people like me who were living outside London. Initially when I retired, if Professor Dusheiko was away, and I could not travel to London, Skipton had used registered post to send applications to me in Dorset to make sure patients got a timely response to their application; I reviewed and returned these within 24-48 hours. I think the service deteriorated when the assessment and administration was moved to Newcastle. taking no account of whether existing staff were able to move their families from London to continue the work. In particular Mr Nick Fish, who had worked with the Skipton assessment process since its start, could not transfer and as a consequence his massive experience was lost. Prof Dusheiko, Dr Janice Main and I were also unwilling to travel up and down to Newcastle on a weekly basis when we were all over retirement age and in my case working as a volunteer without payment. We did however offer to help find experts in the Newcastle area, recommending Prof Margaret Bassendine and Prof Quentin Anstee, both distinguished hepatologists living in the vicinity of Newcastle. Prof Bassendine decided to help and I arranged an induction process for her, to provide continuity. At this stage the assessment process had moved from the voluntary unpaid stage to paid employment by NHS Business. The whole environment changed and it soon became apparent that Prof Bassendine needed additional help and assistants were employed after advertisement and interview. At least one additional doctor came from my old department.

155. I do not know what the attitude of the department of health was to the AHOs and whether any attempts to take into account the issues raised in the consultation document were made.

156. Yes, we did raise concerns which were put in writing. My concern regarding the 2016/7 reforms, which I expressed at the Reference Group, was that the mental problems of mild cognitive and depressive problems are very common in the general population and it would be impossible to determine whether any symptoms were related to CHC. This turned out to be the case and virtually all applicants received SCM payments.

Section 20: Winding up of the AHOs and the Devolved Schemes

157. I thought that the Skipton fund and the Caxton fund were well run and that the move to Newcastle and inclusion in the NHS Business unit was a retrograde step. This tendency to central control has of course been repeated in the current pandemic in that unpaid medically qualified volunteers have no role in either the test and trace or now the vaccination programme. I have volunteered for both but have given up now because of the bureaucracy and the need to respond to the IBI.

158. I had very little role and I was against the move.

159. I cannot comment on this since I have had no involvement in the EIBSS.

160. See Q159

161. I have occasionally helped Prof Bassendine when she was working alone on difficult cases.

162. In the reference committee I was the only Hepatologist and was therefore involved in defining the issues related to the special category payments in stage 1. I also reviewed the literature to substantiate the inclusion of two new criteria in triggering stage 2 payments. I was also asked to try to establish equivalence between HCV and HIV payments based on how the wellbeing of patients from each group had been affected. Professor Dusheiko also gave help on treatment.

163. I was involved in the development of the special category mechanism (SCM) because I had published extensively on HCV infection of the brain and the symptoms that this causes. In particular we had cloned an atypical virus from the brain of some patients and had used magnetic resonance spectroscopy to show that the brains of stage 1 patients had changes similar to those seen in HIV. My colleagues, Dr Dan Forton, Prof Simon Taylor-Robinson and Prof Peter Karayiannis at St Mary's and Prof Michael Manns (professor of medicine in Hanover medical school) were involved.

164.

164(a) The difficulties in setting up the SCM stemmed from the high prevalence of depression and "brain fog" in the general population without HCV infection and the impossibility of differentiating these from those caused by HCV.

164(b) I thought that there was consensus at the committee despite the difficulties mentioned above (a)

164(c) These issues were not discussed with me. The funding of the scheme was discussed by the department of health, I presume.

164(d) All of the issues I raised from a medical perspective were included in the new criteria so long as I presented scientific proof.

165.

165(a) I was involved in the department of health's work on whether mental health should be included in the criteria because I had published papers in the area and these had been confirmed by studies in Hanover Germany.

165(b) I do not know the background of the other members of the department of health reference group except I do know a member of the haemophilia society was there and an advisor to the DWP.

165(c) See Q164-a

166. I was involved in devising the criteria for the SCM and additional criteria for stage 2 payments irrespective of whether these infections were acquired from NHS blood or blood products.

167. Most of the assessment was relatively objective as indicated in the application form. However, when the SCM was introduced the evidence were very soft and really we relied on whether the patients' GP or hospital consultant considered this was possible/probable/not related to HCV. We eventually allowed all possible/probable applications because we believed the GP or hospital consultants would know the patients better than us.

168. I cannot comment. You should ask Prof Bassendine and her colleagues.

Section 21: Look back

169. I assume you are referring to the lookback undertaken by the Blood Transfusion Service in which patients who had received infected blood, would be traced. As a hepatologist my role was to advise on what care the patients identified would need should it be found on lookback that they were HCV positive. I cannot provide estimates of patients that were sent to me following this exercise, we did not collate this information. It was relatively few.

The efficacy and consequences of the lookback exercise were limited. The number of patients identified was lower than the 40,000 predicted by Dr Adrien Renton because many patients receiving blood or plasma had cancers and therefore had limited survival.

Most of the organisation of this process was undertaken by the blood transfusion consultants and I was only involved in advising on medical issues such as prognosis of those identified and their management.

The figure of 40,000 cases likely to be identified by the lookback exercise was an estimate: at that time the estimate of the total number of HCV infected individuals was in the order of 150,000 and around 30-40% were believed to have been infected by blood or blood products, the remainder being principally IV drug users.

A second group of 'lookbacks' referring to my work in the AGH (as a member and then chair of the AGH (Advisory Group on Hepatitis) the mandate was to control hepatitis in the community excluding transfusion transmitted infections), when cases of HBV and HCV followed the work of HBV and HCV infected dentists and surgeon, were also undertaken. I was in favour of undertaking 'look-backs' initially to see how frequent this was and subsequently whether we could get dentists and surgeons back to work by either viral suppressive treatment in the case of HBV or curative treatment in the case of HCV. As far as I know, these programmes got several dentists and surgeons back to work without subsequent infections of their patients.

The third area to which you may be referring is whether we should have "lookback" or high-risk population screening. Where I was involved it would have been through the AGH and all of the discussions were minuted and should be available to you.

170. I was advising on the subsequent tests that the lookback cases would need to determine whether they had liver disease.

Some of the previous discussions related to the use of my monoclonal anticore in studies to establish cost effectiveness and efficacy of these antibodies with ALT as surrogate markers of NANB.

I was also asked to prepare a paper on treatment recommendations for the care of those identified in the lookback.

171. As chairman of the AGH I was asked to chair a series of meetings to develop an Action Plan for Hepatitis C in England. The strategy was requested by the department of health as a result of a question asked in the house of commons.

The aims and objectives of the Action Plan and the final recommendations can be found in the published department of health document (SAFT0000066).

The focus was on control of HCV in the general population not related to transfusion issues. In the last few meetings, we were asked to remove some

of the goals that we had set to make sure that implementation was in a timely fashion. This was particularly an issue on how quickly patients should be treated at that time with interferon and ribavirin. This was said to be because this would be dealt with by NICE who required that any treatments already recommended had to be available in a set time frame which was I think of the order of 3 months. I felt that the removal of targets reduced the effectiveness of the strategy; I wanted particularly to focus on improving access to treatment at that time.

To improve access to treatment in other ways I became involved in the pan-London commissioning process. This involved setting up the criteria for a service based around regional networks with recommended personnel in each region. I felt that we did not receive an appropriate priority bearing in mind that at this time deaths from liver diseases were increasing and this was the fourth largest cause of mortality at the time.

172. Only two complaints were made against me and neither were upheld.

One complaint was to the hospital committee suggesting that I had treated someone inappropriately for tuberculosis: this was reviewed by the infectious diseases consultants who agreed that the treatment given was appropriate.

The second complaint was made to the GMC by a nurse who claimed she had not been told that she had Hepatitis C: she had gone to work in the Middle East and claimed that since she did not know of this diagnosis and therefore did not declare it, she could have been placed in jail. Again, this was not upheld in that I had a letter indicating that she had been told of the diagnosis and the implications.

173. I cannot think of any other issues relevant to the Inquiry that have not already been covered.

Statement of Truth

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

Signed GRO-C

Dated: 1/3/2021

Table of exhibits:

Date	Notes/ Description	Exhibit number
01/11/2014	Letter to Secretary of State	WITN3824008
15/12/2017	Draft witness statement for judicial review	WITN3824009