Witness Name: Professor Howard Thomas
Statement No.: WITN3824001
Exhibits: WITN3824002-004
Dated: 29 October 2019

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 16 October 2019.

I, Professor Howard Thomas, will say as follows: -

Section 1: Introduction

1. The statement is based on my memory and on my review of the documents that have been forwarded to me with the request for a statement, dated 16 October 2019. I have not had access to any other medical records or clinical records.

2. I am Professor Howard Thomas BSc, PhD, FRCP, FRCPath, FMedSci, of GRO-C GRO-C Dorset GRO-C born GRO-C 1945.

3. I am currently an Emeritus Professor of Hepatology, in the Department of Medicine at Imperial College, London. 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. At the Royal Free, I was initially an honorary senior registrar under Professor Sheila Sherlock and then was an honorary consultant. As

an academic with consultant status, I had responsibilities for teaching, research and patient care.

4. At various times while at 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.

5. I am Vice President of the British Liver Trust, and Chairman of the board of Trustees of the Liver Research Trust.

6. 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.

7. 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 Dangerous Pathogens Committee and a member of the Working Party on Transfusion Associated Hepatitis (single Meeting in 1986). During this meeting 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, or evaluated in a prospective clinical trial.

8. In 1991, I was asked to write a paper giving the argument in favour of the immediate introduction of anti-HCV screening of blood donations: this was in the form of a debate with the argument against the immediate

introduction being made by a member of the blood transfusion service (Brown and Thomas; Reviews in Medical Virology 1991: 1: 67-71).

9. I provided evidence on the biology, natural history and pathogenesis of hepatitis C to the Penrose Inquiry on Infected Blood (circa 2005).

10. I undertook the 5-year review of the Australian Action Plan for Hepatitis C for the Australian Government.

11. 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. Initially I did this alone and then as the load increased was assisted by Professor Geoff Dusheiko and Dr Janice Main.

12. 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 GRO-C in 2019 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 2: Responses to criticism of W1002

13. The paper 'Short Incubation NANB transmitted by factor V111 in patients with congenital coagulation disorders' by Bamber, Murray, Arborgh#, Scheuer#, Kernoff#, Thomas, Sherlock# (# deceased); Gut 1981:22,854-859 was a retrospective anonymised study of 10 patients presenting with clinical problems from 1975-1979 and sent for publication in April 1981. Studies up until that time had suggested sporadic (Bamber et al; Gut 1983: 24: 561-4) and transfusion

associated NANB (Hruby and Schauf JAMA1978; 240: 1355-7; Craske et al Lancet 1975; 2: 221-3) were mild and self- limiting diseases but in 1978 we became aware that patients were presenting to our clinical liver service with significant jaundice and with persistently abnormal liver tests, raising the possibility that NANB was not a benign problem but caused chronic liver disease. Six of these patients underwent liver biopsy, after verbal or written consent, in order to determine the type of liver disease that was present. The possibilities included obesity and alcohol related steatosis with or without superimposed PT-NANB, atypical intrahepatic hepatitis B, or autoimmune disease. Clinically we also needed to establish the prognosis: patients with CPH and CLH were thought to have benign disease whereas CAH progressed to cirrhosis and would require treatment either with interferon (we and others had started studies of interferon in CHB and CHC at this time and shown promising results (Jacyna et al 1989; BMJ: 268: 80-2) or steroids for autoimmune CAH or behavioural modification for steatosis either due to alcohol excess or obesity. The names of the patients involved in this study are not available to me.

14. The study that Kathleen Stewart cited at paragraph 26 of her witness statement as "page 474 of the study" (WITN1002013), was another study, which was again a retrospective anonymised study recording the incidence of abnormal LFTs after NHS and US factor 8 were infused 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). This was supervised by the Haemophilia Unit and Dr Kernoff states 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. The study established that the risk of NANB was the same after NHS (volunteer donors) and US (paid donors) preparations both of which were produced from around 1500-5,000 donations.

15. The lab specimens mentioned in exhibit 14 were blood specimens stored so that if candidate assays were found, these sera could be used to validate the assays as being related to NANB or when the NANB virus was discovered (subsequently called hepatitis C in 1989) then

the time and involvement of any agent in an individual case could be established. These specimens were subsequently used for this purpose in 1989-1990.

16. During a retrospective anonymised study to find a non-invasive way of determining fibrosis levels in the liver, the serum specimens were also 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).

Section 3: Other Issues

17. There are no other issues in relation to which I consider I have evidence which will be relevant to the Inquiry's investigation of the matters set out in its Terms of Reference.

Statement of Truth

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

Signed GRO-C

Dated 29/10/2019

Table of exhibits:

Date	Notes/ Description	Exhibit number
1981	'Short Incubation NANB transmitted by factor V111 in patients with congenital coagulation disorders' by Bamber, Murray, Arborgh, Scheuer, Kernoff, Thomas, Sherlock	002
1985	'High risk of NANB after first exposure to volunteer or commercial factor 8: effect of prophylactic immune serum globulin' by Brit J Haem 1985	003
1988	'Non-invasive investigation of liver disease in haemophilic patients' by Miller et al; J Clin Path 1988; 41:1039-1043	004