Witness Name: Dr Kate Soldan
Statement No.: WITN7088001
Exhibits: WITN7088002-3
Dated: 19 May 2022

INFECTED BLOOD INQUIRY

WRITTEN STATEMENT OF DR KATE SOLDAN

I provide this statement in response to a request under Rule 9 of the Inquiry Rules 2006 dated 22nd October 2021.

I, Kate Soldan, will say as follows: -

Section 1: Introduction

1.1. My full name is Katherine (Kate) Soldan MA, MPhil, PhD, of work address: UK Health Security Agency ("UKHSA"), 61 Colindale Avenue, Colindale, London, NW9 5EQ, DOB: GRO-C I have a BA in Natural Sciences & Biological Anthropology from Cambridge University (1990), a MPhil in Epidemiology from Cambridge University (1994) and a PhD in Epidemiology from the University of London (2001).

1.2. My post-graduate employment history is detailed in the table below:

Dates	**Position & employer** (Main duties/responsibilities in brief)

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Jan 06-date	**UK Health Security Agency (UKHSA), formerly Public Health England (PHE)** [The UKHSA replaced PHE on 1 October 2021. PHE had replaced the Health Protection Agency (HPA) on 1 April 2013.] **Consultant Scientist - Epidemiologist.** Blood Safety, Hepatitis, STI and HIV Division, National Infection Service, Colindale, London, **Britain**. *Section Head – Intervention Evidence and Evaluation (team of 8), Lead for evaluation activities that inform: the National HPV Vaccination Programme; the National Chlamydia Screening Programme (NCSP), and additional programmes as appropriate.* NB. On leave Mar'15-Feb'16: part-time since return.
Oct-04-Jan- 06	**HPA Epidemiologist-Scientist (Consultant level)**, Locum position. HPA, Colindale, London, **Britain**. *Head of CJD Section (team of 8) & Scientific Secretary to the CJD Incidents Panel. Grading assessor for transition of staff to Agenda for Change terms and conditions.*
Jan-04-Aug-04	**WHO STOP Data-Manager**, WHO/Centre for Disease Control (CDC), Atlanta. *Assisting the Expanded Programme on Immunisation (EPI) Units in Maseru, **Lesotho** (Jan-Apr) and Lusaka, **Zambia** (May-August) to improve surveillance of vaccine- preventable diseases in childhood (particularly polio and measles) and of coverage of childhood and maternal immunisations.*
Oct-02-Nov-03	**WHO Post-doctoral fellow**, Unit of Environmental Epidemiology, WHO International Centre for Research on Cancer (IARC), Lyon, **France**. *Coordination and management of an EC funded multi-centre case-control study to investigate genetic susceptibility to alcohol-related head and neck cancers (ARCAGE).*
Dec-94-Oct-02[1]	**Public Health Laboratory Service (PHLS)/NHSBT Senior scientist (Epidemiology)**, Transfusion-transmissible infections. Immunisation Department of the PHLS Communicable Disease Surveillance Centre (CDSC) and The National Blood Service (NBS), Colindale, London, **Britain**. *Establishing enhanced surveillance system for Transfusion-transmissible infections (TTIs) in blood donors and transfusion recipients (and tissue donors) and conducting related risk assessments and epidemiological studies. During this job I also studied part-time at London School of Hygiene & Tropical Medicine (LSHMT) and submitted my PhD thesis, "The epidemiology of infections in blood donors and assessment of the risk of transfusion transmitted infections", awarded in 2001.* [1] ***I continued as a part-time consultant to the NBS until 2004.***
Sep-94-Nov-94	**WHO Researcher/epidemiologist**, (awarded training fellowship). WHO International Agency for Research on Cancer, Lyon, **France**. *Analysis of reported incidence of Kaposis sarcoma to European population-based cancer registries to investigate the impact of the AIDS epidemic and explore the hypothesis of an infectious causative agent.*
Aug-92-Aug-93	**MRC Researcher**, National AIDS Programme of the South African Medical Research Council, Natal, **South Africa**. *Evaluation of the pilot year of the KwaZulu home-based care programme for HIV/AIDS patients in five KwaZulu Health Districts. Analysis and write-up of a study of barriers to HIV prevention amongst sex workers at truck-stops.*

Jan-92- Jul- 92	**University of Durban Field-worker**, Centre for the Advancement of Science & Mathematics Education, University of Natal, Durban, **South Africa**. *Piloting a service to facilitate professional development of science teachers in six KwaZulu high schools and establishing processes for monitoring this new service.*
Sep-90- Jan-92	**PHLS - Clinical Scientist**, HIV/STI Department of the PHLS CDSC, Colindale, London, **Britain**. *Management of the Unlinked Anonymous HIV Prevalence Monitoring Programme's survey of HIV infection among pregnant women in England and Wales.*

1.3. My membership of professional bodies is detailed in the table below:

2021-	Associate of the Faculty of Public Health (FPH) and a member of the FPH CPD scheme.
2021-	Member of the European Public Health Association (EUPHA).
2008- date	British Association for Sexual Health & HIV (BASHH) Member of HPV Special Interest Group (SIG)
2001- 2011	State Registered Clinical Scientist (HPC)
1996- 2006	Member of the British Blood Transfusion Society.

Section 2: Doctoral Thesis

2.1. In my PhD thesis, "The Epidemiology of Infections in Blood Donors and Assessment of the Risk of Transfusion Transmitted Infections" [WITN7088002], I describe methods for establishing a system for the surveillance of infections in blood donors. This was work initiated by, and jointly run by, the NBS and PHLS. This involved collection of information from all Regional Transfusion Centres (RTCs). In order to discuss and understand the information available at RTCs and the best way regularly to collect and return data, I visited all the RTCs in England and Wales during the first half of 1995 to see how their data was managed and to talk to the staff who would participate in this surveillance system. I spoke by phone to colleagues at RTCs in Northern Ireland, the republic of Ireland, the Channel Islands and the Isle of Man. The surveillance systems that were then established created three centralised databases (Donation Testing Surveillance, Infected Donor Surveillance and Post-Transfusion Infection Surveillance) at the National Blood Authority (NBA).

2.2. The three infection surveillance systems (Donation Testing Surveillance, Infected Donor Surveillance and Post-Transfusion Infection Surveillance) were introduced to blood centres in England, Wales, Northern Ireland, the Republic of Ireland, the Channel Islands and the Isle of Man on 1st October 1995. The Scottish Blood Transfusion Service (SNBTS) established a similar system for surveillance of donation testing in April 1995, and provided collated data, in a format comparable to the NBA/PHLS-CDSC surveillance data, to the surveillance centre monthly (as set out on pages 74-77 of my PhD thesis, "The Epidemiology of Infections in Blood Donors and Assessment of the Risk of Transfusion Transmitted Infections" [WITN7088002]). I do not know why surveillance databases were not established prior to the above except that I note that the NBA was only established in 1993 and I was employed to conduct work to establish enhanced surveillance of transfusion of transmissible infections.

2.3. Information submitted to the surveillance databases (for Donation Testing, Infected Donors, and Post-Transfusion Infections) from RCTs were submitted to "The Medical Director (Infection Surveillance)" of the National Blood Authority (Dr Angela Robinson). I managed these data with the Medical Director's delegated authority/permission to do so. These surveillance databases were held by the NBA and are – to my knowledge – now held by NHSBT. I never held a copy of these databases.

2.4. As I recall from my visits to RTCs (in early 1995), there were some variations in record keeping between different RTCs: this was partly why visiting them was necessary in order to design reporting forms and a system in which they could all participate and which would provide the right (and same) data. This variation was not surprising or of concern. In my PhD I described the information available at blood centres, based on my visits, including the variations that needed to be understood and accommodated in the design of a surveillance system in Section 3.1.1, pages 65-68.

2.5. My PhD included:

2.5.1. (Chapter 3) data from the Donation Testing database, the Infected Donor database and the Post-transfusion Infections database for October 1995 to September 1999;

2.5.2 (Chapter 4) data from a survey of HCV seroconversions in blood donors for 1993-1995; data from a review of laboratory reports of acute HBV infections associated with blood transfusion for 1991-1997; and

2.5.3. (Chapter 5) estimates of the risk of transfusion transmitted infections for 1993-1998.

Chapter 5 used data from 1993, partly because the required input data were available (via the work in Chapters 3 and 4) only from 1993, and partly because the aim was to inform current practice for which pre-1993 data would have been increasingly less relevant. The end date for this analysis, of the end of 1998, was due to the analysis being conducted when data up to the end of 1998 were available.

2.6. I do not recall any RTC holding information about HCV infections in blood donors prior to the start of anti-HCV testing in 1991. I do not recall any RTC holding information about non-A, non-B hepatitis infections in blood donors prior to the start of anti-HCV testing in 1991.

2.7. I do not recall any details about the recording of information about blood donations at RTCs that are not described in my PhD thesis. My PhD thesis also describes the data obtained from PHLS Surveillance systems about infections reported as diagnosed amongst blood donors and amongst blood recipients. My PhD was focused on surveillance and epidemiology, not on microbiological testing and diagnoses, and was based on available testing results: no additional testing (on archived or newly collected sera) was therefore conducted.

2.8. In my opinion, based on my years of experience since, the record keeping and archiving/preserving of sera samples from blood donations collected by the NBS (and constituent RTCs) at the time I worked on my PhD werethoughtful and thorough. As I recall, sera samples were kept from every donation for a decided length of time, based on considerations of possible future uses as well as considerations of sample quality (and degradation over time) and appropriate priority use of storage capacity. I do not recall the details of these retention periods: NHSBT may be best placed to assist with any enquiry about this.

Section 3: 2002 Journal Article

3.1. The 2002 article entitled "The Contribution of Transfusion to HCV Infection in England" (PRSE0000620), which I co-authored, estimates the number of anti- HCV positive donations collected during the 1980s and until September 1991, and the possible number of infected recipients from these untested/unidentified anti-HCV positive donations (see page 588).

3.2. In the discussion, we (the authors), noted the number of infections that may have been transmitted during the 1970s, if the same assumptions were applied. One reason that we did not include the 1970s in the main analysis was that donor selection criteria that aimed to exclude from blood donations people who had injected drugs (amongst others) should have lowered the prevalence of HCV amongst collected blood donations. Less is known about the incidence and prevalence of HCV in the 1970s. I am aware of work to back-calculate the epidemiology of HCV amongst people who inject drugs that has found the prevalence for the pre-1980s period to be similar to the 1980s (Harris et al, JVH 2019) [WITN7088003].

3.3. The estimate of HCV infected blood donations during the 1970s, based simply on prevalence in 1991, is, therefore, likely to be a minimum estimate or an underestimate. I believe the limitations of these assumptions were made clear and would have been well understood by the readers of this journal, and also that they did not affect the main findings and message that should have been taken from this paper, i.e. that transfusion had infected a large group of individuals, but constituted a very small, and declining, proportion of all HCV infections. The findings (and limitations) of this work were not intended/expected to inform donor testing, selection and transfusion practices, but to inform HCV testing in the community and to plan care for HCV-related disease for the general population and for transfusion recipients specifically.

3.4. The Inquiry has asked me to comment on the number of extra HCV infected blood recipients that would be generated by the estimation process in the 2002 article (as above) if the prevalence of HCV in blood donors was instead assumed to be:

a) throughout 1970-79 the estimated rate presented to the MRC in 1974 namely 1% (PRSE0002988), and

b) throughout 1980-91 the rate observed in a 1990 UK study, namely 0.55% (OXUH0000030_002).

This could be calculated by entering the resulting number of infected donations into the subsequent probability steps, however, I do not believe either of these estimates/observations are correctto be used in this way.

(a) The estimated rate presented to the MRC in 1974 namely 1% (PRSE0002988)

3.5. The estimated rate presented to the MRC in 1974 of 1% (PRSE0002988) is of "patients judged to have developed icteric or anicteric post-transfusion viral hepatitis", and is based on 8 patients judged to fall into this category. The report to the MRC goes on to specify that 4 of the 8 patients were hepatitis B antigen positive: HBV (not HCV) would therefore be the putative causative infection for their jaundice. This report pre-dated knowledge of HCV. From my reading of this report, my understanding is that the estimates they produce concern hepatitis that the researchers determine as attributable to HBV, cytomegalovirus (CMV) or Epstein-Barr virus (EBV), as these were the jaundice-associated infections for which they had testing targets and technologies at that time. They provide some information about transfusion recipients included in their study that showed ALT rises (without serological or virological markers of the infections above): I suspect these patients may be the better group from which to derive a putative prevalence of HCV in blood donations at that time. Some of these patients were noted as having a history suggesting their ALT rises may have been alcohol-induced. I am unable to derive an estimate of HCV infected blood donations from this report: the authors (or an appropriate expert to review the data they present) may be better able to comment on which patients they would now classify as potentially HCV infected, and whether an estimate of HCV prevalence in blood donations could be so derived.

(b) the rate observed in a 1990 UK study, namely 0.55% (OXUH0000030_002)

3.6. The rate observed in the 1990 UK study, namely 0.55% (OXUH0000030_002), is of blood donations that "were repeatedly reactive in a commercial assay for

antibodies to the C100 protein of hepatitis C virus", and is based on 6 donations falling into this category. The report goes on to state that only 1 of these 6 transmitted non-A non-B hepatitis and tested positive for HCV RNA by the polymerase chain reaction test applied to all 6 donations. From my reading of this report, my understanding is that this report provided evidence of the likely inaccuracy (and therefore unsuitability) of the assay they used for antibodies to the C100 protein as a screen for HCV infected blood donations. However, the authors (or an appropriate expert to review the data they present) may be better able to comment on whether an estimate of HCV prevalence in blood donations in the late 1980s could be derived from this study.

3.7. Based on my understanding of the epidemiology of transfusion-transmitted infections, including HCV, it is my opinion that donor selection and exclusion policies introduced throughout the 1980s would have lowered the prevalence of HCV in blood donations collected by the NBA. Because I used more recent HCV prevalence rates, the estimates I generated of the number of people who may have been infected by blood transfusion between 1980-1991 are likely to have been slightly underestimated whereas the estimate for the 1970s (included only in the discussion) is likely to have been underestimated to a greater extent. The assumed prevalence of HCV in blood donors was one of a large number of assumptions, with limitations, used in this estimation process. The conclusions from this estimation process are, I believe, robust even given the limitations of the assumptions and resulting inaccuracy of the estimates: these limitations were unlikely to affect the overall conclusion that transfusion was a small contribution to the epidemiology of HCV in England.

3.8. Regarding the potential value of surrogate testing, as a scientist, my very broad understanding of the use of surrogate tests to identify an emergent infection, or rather to identify the increased risk of an emergent infection, is that careful assessments need to be made of the consequences of the sensitivity and the specificity of the surrogate marker, amongst the population being considered for surrogate testing, and in light of the benefits and harms that could be experienced as a result of the surrogate testing.

3.9. As far as I can recall, at the time I was working with the NBA, the UK Blood

Transfusion Services had and used access to suitable experts to conduct these assessments and to consider options for employing surrogate testing. My understanding is that these assessments did not identify a suitable surrogate test for testing blood donations. I do not have (or have the relevant expertise to form) an independently derived opinion on the suitability of surrogate tests of NANB hepatitis, ALT and anti-HBc to screen donors before September 1991.

3.10. I am an infectious disease epidemiologist. I do not have the appropriate expertise or education to comment on the notification of infectious diseases and issues that may arise in relation to this. This would be within the expertise of a public health specialist.

3.11. The calculations I did, at the request of Dr McClleland, to estimate the numbers of patients probably infected by blood transfusions in Scotland, including estimates akin to those in the 2002 paper for England, are contained in document SGH0057203, which the Inquiry has on record. There is no other document detailing these estimates. Please note this was for transfusion of blood components not for receipt of blood products.

Section 4: Effectiveness of Lookback

4.1. The 2002 paper referred to above is my (and my co-authors') assessment of the probable contribution of transfusion to HCV infection in England: within this is some necessary estimation of the likely extent of transfusion transmission of HCV that was not detected by the HCV 1995 lookback. The objective of this work was to use the available information to assess the role of transfusion in the epidemiology of HCV and thereby inform HCV testing and planning for care provision. The objective was not to evaluate the lookback exercise. The purpose of the HCV 1995 lookback was stated by the Chief Medical Officer in April 1995 as "to trace, counsel and if necessary treat patients exposed to HCV infection prior to September 1991 by transfusion." Determining the success of the lookback, which is slightly different from the aim of the 2002 paper, would require firstly defining the total or absolute desired result or effect of the lookback. To my understanding, this was never reasonably expected to be identifying *every* transfusion recipient put at risk or identifying *every* infection. There were very many reasons why it was not

possible to identify all transfusion recipients who may have been put at risk, and/or not possible to test them all. From my experience, the HCV 1995 lookback methodology was thorough in pursuing the tracing of components and the offering of counselling (and consequent testing and care) to as many patients who were put at a known increased risk as possible. It is my understanding that the limitations in identifying infected transfusion recipients described for the HCV 1995 lookback, such as access to hospital records, matching identifiers recorded in different places, locating and contacting patients, tend to be present in other lookbacks. The completeness of lookbacks will vary due to different timings and different natural history and past epidemiology of the infection that is the subject of the lookback. I am unable to comment on the efficacy of the vCJD 1998 lookback or any other lookback as I have not been involved in reviews or assessments of these.

Statement of Truth

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

Signed GRO-C

Dated 19/05/2022

INDEX TABLE TO WITNESS STATEMENT OF DR KATE SOLDAN

1	WITN7088002	07/2001	PhD Thesis *"The Epidemiology of Infections in Blood Donors and Assessment of the Risk of Transfusion Transmitted Infections"* by Dr Kate Soldan.
2	WITN7088003	21/01/2019	Journal article, Journal of Viral Hepatitis, "*Monitoring the hepatitis C epidemic in England and evaluating intervention scale-up using routinely collected data*" by R Harris, H Harris, S Mandal, M Ramsay, P Vickerman, M Hickman, D De Angelis.
3	PRSE0002988	21/02/1974	Journal of Hygiene, Cambridge - 'Post-Transfusion Hepatitis in a London Hospital - Results of a Two Year Prospective Study' by the Medical Research Council Working Party, dated 21 February 1974.
4	OXUH0000030_002	16/06/1990	The Lancet, 'Detection of hepatitis C viral sequences in blood donations by 'nested' polymerase chain reaction and predictions of infectivity', J A Garson et al (1990).
5	PRSE0003921	01/07/2002	Report prepared by K Soldan for DBL McClelland titled 'Estimated Number of Individuals Infected by Blood Transfusion in Scotland.' The total number of individuals probably infected with HCV by blood transfusions is said to be 3,498.