

PATIENT INTEREST CORE PARTICIPANTS - SUBMISSIONS FOR THE STATISTICS TOPIC

By way of introduction, we would like to emphasise that, in our view, we consider the statistical analysis which the Inquiry has tried to undertake to be an extremely important part of it fulfilling its remit. The reason for this is that an accurate analysis of the statistical material available to the Inquiry is important for an understanding to be reached as to:

- a) The scale and devastating effects of the blood contamination disaster in Scotland. We consider it imperative that the Inquiry is able to furnish those with the responsibility of caring for the victims of the disaster (and indeed those responsible for preventing a re-occurrence of similar such disasters) with an accurate impression of the numbers of victims involved and the populations in which those victims might be traced, supported and treated;
- b) The causes of and reasons for infection in different populations in Scotland. A thorough statistical analysis enables the proper appreciation of the likely timing of the infections and the infection routes of those infected which, in turn, enables a greater focus to be achieved on what measures might have been taken to avoid such infections;
- c) The accuracy and validity of the assertions of those responsible for the care of those infected. A thorough statistical analysis enables the theoretical assertions made by those responsible for the administration of blood and blood products to be tested against empirical reality; and
- d) The context within which each individual patient was infected. Many patients and families affected by the disaster experience a sense of isolation. Through clarification of the numbers, places and methods of infection, infected patients and bereaved families can gain a greater understanding of the place which they occupy within the disaster.

The way in which the evidence on this topic has been collated and analysed has not been without difficulty. Much of the evidence heard in connection with this topic at the original oral hearings has been superseded or supplemented. The evidence relating to patients infected with hepatitis C as a result of blood transfusions received in Scotland (spoken to in the oral hearings by Dr Jack Gillon and Professor David Goldberg) has been superseded by the additional epidemiological analysis carried out by Professor Goldberg and his team and the correspondence between him and the Inquiry on this important topic. The evidence relating to infections amongst the bleeding disorder community

(spoken to in the oral hearings by Dr Charles Hay, Professor Christopher Ludlam and Dr Campbell Tait) has been superseded by the additional material provided to the Inquiry by the UKHCDO.¹ None of the new material has been subject to analysis at the oral hearings. In light of the state of the evidence and the importance of the Inquiry being in a position to give as thorough an answer as possible to the issues related to statistics which we have formulated below, we would respectfully invite the Inquiry to consider either recalling or calling certain additional witnesses to give evidence on this topic. In our view, these should include (a) Professor David Goldberg (to speak to the epidemiological analysis undertaken by him since he last gave evidence) (b) Dr Kate Soldan (to speak to the methodology applied in her epidemiological analysis of the numbers infected with Hepatitis C from blood transfusions in Scotland) (c) Dr Gillon and/or Dr McClelland (to speak to the evidence they have provided to Professor Goldberg and its influence on the assumptions which underpin his epidemiological analysis) and (d) Dr Charles Hay (to speak to the newly produced UKHCDO statistical material). The analysis below attempts to propose answers to the issues which were submitted by us to the Inquiry in light of the current state of the evidence.

HIV infections amongst people with bleeding disorders

1. How many people with bleeding disorders were infected with HIV by blood products in Scotland?

Background

As is recognised in the preliminary report, there are a number of figures from different sources as to the total number of HIV infections in Scotland amongst the population of those with bleeding disorders.² Initial figures provided to the Inquiry suggested that the number of Scottish infections in this population might be either 87 (HPS) or 72 (UKHCDO). As is also recognised in the preliminary report, there is a need to reconcile these figures.³ It would appear that the figure of 87 represents the cumulative total number of HIV infected persons who have been resident in Scotland who are

¹ PEN.019.0927

² PR, paras 3.60 to 3.61 and footnote

³ PR, paras 3.60 to 3.61 and footnote

believed to have been infected by treatment with coagulation factors.⁴ Even in December 1989 (a figure unlikely to have been affected as much by migration as more recent estimates based on residence rather than place of infection) a response to a parliamentary question indicated that there were 76 haemophiliacs who had been infected with HIV in Scotland.⁵

As is discussed in more detail below, the position has moved on since the time when the preliminary report was compiled as (a) certain haemophilia directors have given oral evidence on this topic and (b) the UKHCDO as an organisation has provided updated material on the numbers of patients who it claims were infected with HIV as a result of exposure to blood products in Scotland.⁶ Given that there is no suggestion that the total number in this group is likely to be more than one hundred individuals, an accurate statement of the number infected within this population should be able to be arrived at.

The evidence of the Scottish haemophilia directors

The material compiled by and the oral evidence given by the Scottish haemophilia directors regarding the numbers of patients so infected within Scotland, whilst useful in certain respects does not, in our view, give a complete picture of the numbers so infected. The information which has been provided to the Inquiry by these directors was taken from the database of the UKHCDO. The directors then applied a certain methodology to that information in order to try to derive a total number of infections likely to have occurred in each of the 6 Scottish haemophilia centres, resulting in a total number of infections for Scotland as a whole. Two of the centre directors wrote to the Inquiry to indicate that they did not think that, on the UKHCDO information provided to them, there had been any infections of patients under the treatment of their centres (namely Inverness⁷ and Dundee⁸). An analysis has been provided to the Inquiry for each of the other 4 haemophilia centres in Scotland of the number of patients thought likely to have been infected in each centre. The analysis includes details of the treatment received by each of those patients and the methodology adopted by the appropriate current haemophilia centre director in each centre in the compilation of

⁴ Scottish Centre for Infection and Environmental Health. HIV infection and AIDS: quarterly report to 31 December 2000. *SCIEH Weekly Report* 2001; 35(3): 18-26

⁵ SGF.001.1246 (21 December 1989)

⁶ PEN.019.0927

⁷ PEN.001.0235

⁸ PEN.001.0234

the information in respect of that centre. These documents came from Edinburgh⁹, Glasgow Royal Infirmary¹⁰, Yorkhill¹¹ and Aberdeen¹².

Professor Ludlam gave evidence to the Inquiry on this material and suggested that the total number of infections with HIV of people with bleeding disorders in Scotland was around 59.¹³ As indicated above, a further analysis presented to the Inquiry by the UKHCDO as an organisation has suggested that the likely number of infections of bleeding disorder patients with HIV in Scotland is likely to be between 68 and 70.¹⁴ Our own researches with the Macfarlane trust indicate that 67 individuals in Scotland with bleeding disorders who were infected with HIV as a result of their exposure to infected blood products have received payments from the trust. Given the criteria which require to be satisfied before a payment from the trust will be made (including the fact that qualifying patients require to be registered with the trust by their haemophilia clinician), this would tend to suggest that the figures provided by the haemophilia directors (and indeed the number of payments from the trust for that matter) are likely to represent an underestimate of the likely number of infections in this community in Scotland. Further, the information provided to the Inquiry by Health Protection Scotland suggested that their records indicated that there were 76 patients with haemophilia who were assumed to have been infected by their receipt of contaminated blood products in Scotland.¹⁵

In our view the methodology used in the compilation of the estimates spoken to by the haemophilia directors (in oral evidence by Professor Ludlam and Dr Tait) is flawed in a number of respects, with the result that the figures shown by these sources are likely to be an underestimate of the likely total number of HIV infections in this population in Scotland. We have the following observations to make in connection with the way in which the numbers have been arrived at:

- In the first place, the information contained within the UKHCDO database is unlikely to be completely accurate. The information spoken to by the haemophilia clinicians (even Professor Ludlam who would have had first-hand experience of many of the patients about whom he was speaking) all came from the UKHCDO database.¹⁶ We consider it to be rather unusual that Professor Ludlam seemed to derive his understanding of the numbers infected

⁹ PEN.012.0159 (spreadsheet) and PEN.012.0153 (compiled by Professor Christopher Ludlam)

¹⁰ PEN.012.0158 (spreadsheet) and PEN.012.0152 (compiled by Dr Campbell Tait)

¹¹ PEN.012.0160 (spreadsheet) and PEN.012.0155 (compiled by Dr Chalmers)

¹² PEN.013.0009 (spreadsheet) and PEN.012.0156 (compiled by Dr Henry Watson)

¹³ Transcript for 30/03/11 (day 14); 57 (16) to 58 (3) (Professor Ludlam)

¹⁴ PEN.019.0927 @ 0961

¹⁵ PEN.012.0151

¹⁶ Transcript for 30/03/11 (day 14); 10 (25) to 11 (8) (Professor Ludlam)

in Edinburgh from the database when one would have expected him, as centre director in Edinburgh throughout the period of both infection and diagnosis with HIV to be able to speak to the numbers infected more directly. To this extent, all of the evidence heard depended on the accuracy and comprehensiveness of the UKHCDO records.

- Further, Dr Hay suggested that details of the products received by each individual patient were historically not provided to the UKHCDO by the clinicians. Other information (including the total quantities of products used in a centre) was traditionally provided but patient specific information about product usage was not, according to Dr Hay, provided until around 5 years ago.¹⁷ This information was heavily relied upon in the calculation of the likely place, timing and method of infection of individual patients. It was also accepted that the data particularly from the west of Scotland may not have been as reliable as one might have hoped.¹⁸

Further, it was accepted by Dr Hay that patients at the milder end of the spectrum (in particular sufferers from von Willebrand's disease) may not have had treatment at one of the recognised centres and may therefore not be registered within the system.¹⁹ The data was deemed to be more reliable for the severer patients who would be more likely to be registered with and receive treatment from a recognised centre which would report certain data to the UKHCDO. This does not rule out the possibility of patients having received treatment in Scotland outwith the recognised centres and therefore not having been included in the UKHCDO records at all. Dr Hay appeared to accept that there may well have been patients who were managed outwith specialist centres on whom the UKHCDO would have no data, particularly in the west of Scotland.²⁰ This would be more likely to give rise to the statistical material missing an infection with hepatitis C than HIV (see below) but given that even blood transfusions transmitted HIV, missing data regarding the infections of milder patients cannot be ruled out. That milder patients treated with plasma derived products (and hence at risk of having been infected) may have escaped the analysis of the UKHCDO was accepted by Dr Hay in his evidence.²¹

In his evidence, Dr Hay also confirmed that the database maintained by the UKHCDO was a named database and that they required to comply with data protection legislation (from

¹⁷ Transcript for 18/03/11 (day 8); 17 (11) to 18 (2) (Dr Hay)

¹⁸ Transcript for 18/03/11 (day 8); 10 (9) to 11 (12) (Dr Hay)

¹⁹ Transcript for 18/03/11 (day 8); 22 (19) to 23 (9) (Dr Hay)

²⁰ Transcript for 18/03/2011 (day 8); 54 (1 to 6) (Dr Hay)

²¹ Transcript for 18/03/2011 (day 8); 41 (6 to 18) (Dr Hay)

1968) which meant that they required the patents' permission for information to be kept within their records.²² We would suggest that this may have resulted in patients having opted out of their data being included within the UKHCDO system.

We would submit that all of these factors are likely to render the figures spoken to from the UKHCDO database as likely to be minimum figures, given the fact that they all create a risk of patients or patients' data not being included in the analysis presented to the Inquiry as a complete analysis. Further, as regards the analysis conducted by the Scottish haemophilia directors:

- The way in which infections have been allocated by the haemophilia directors as between Scottish infections and infections likely to have been acquired elsewhere in the UK is flawed. The Inquiry is focused on infections which occurred in Scotland. There is no current inquiry into the infections which are deemed to have occurred elsewhere. No responsibility is being taken for infections of patients who have received treatment in Scotland but have not been allocated as a Scottish infection. The methodology applied in the determination of the likely place of infection by the haemophilia directors relies on the proposition that a patient is likely to have become infected (when he has received treatment in Scotland and elsewhere) in the place where he received the majority of his treatment prior to infection.²³ In our view, this approach is flawed. The timing of the treatment requires to be taken into consideration as there are clearly time periods within which it is far more likely that an individual would have been exposed to an infected product than at other times. None of the material available seems to suggest that anyone became infected in Scotland before 1980. This is due to the fact that the virus was simply not present in the products at that time. The likelihood of a haemophilia A patient being infected from a factor concentrate in Scotland after December 1984 is small given the fact that Scottish factor VIII concentrate was heat treated so as to inactivate HIV by that time. Therefore, a focus on the source of the majority of the treatment over a patient's entire lifetime (prior to infection) may well give an inaccurate impression of the likely place of infection. Further, it appeared to be the position of Dr Tait that the assumption applied to the place of likely infection based on the location of the majority treatment received by the patient prior to the infection was not, in fact, based on

²² Transcript for 18/03/11 (day 8); 18 (6 to 11) (Dr Hay)

²³ See (for example) the methodology statement relating to the patients infected with HIV in Glasgow - PEN.012.0152 @ para 4

the majority of treatment but rather the majority of years of treatment as individual data about the quantity of product received in any one place was not available.²⁴

- Further, the calculations carried out by the Scottish haemophilia directors appear to have been carried out in a way which does not allow further scrutiny of their methodology. Where patients have, for whatever reason, been discounted from consideration as a Scottish HIV infection, they have fallen outwith the system and no details have been provided as to their treatment histories.

In our view, the analysis which has been carried out and the conclusions which have been presented to the Inquiry by the Scottish haemophilia directors are unreliable on this basis. However, given that this is the only detailed information with which we have been presented as available to the Inquiry in this regard (in particular relating to the treatment histories of the 59 patients who they deemed to have been infected in Scotland), it is necessary to take some cognisance of it for the purposes of the important analysis which the Inquiry requires to undertake of the scale of and the reasons for the blood contamination disaster in Scotland. That analysis is undertaken below on the basis of the information which we have but must, at all times, be considered to be subject to the limitations detailed above.

The updated UKHCDO analysis conducted by Dr Hay

In the analysis presented by the UKHCDO in its updated paper on statistics, the approach which has been taken appears to have been more sophisticated. The starting point for this analysis appears to have been individuals who were reported to the UKHCDO by a Scottish centre as having been infected with HIV (73 in total).²⁵ Dr Hay had spoken (in connection with the figures initially presented to the Inquiry by the UKHCDO on this subject) to the fact that the data provided by the UKHCDO appeared to provide the data as to how many people with bleeding disorders who suffered from HIV were managed in Scottish centres, rather than how many people were infected in Scottish centres.²⁶ The records relating to the 73 individuals have then been subjected to analysis of those patients (11 in total) who also received treatment outside Scotland and who may, therefore, have actually been infected outside Scotland though the report of their infection came from a Scottish centre.²⁷ The

²⁴ Transcript for 30/04/11 (day 14); 97 (21) to 98 (5) (Dr Tait)

²⁵ PEN.019.0927 @ 0961

²⁶ Transcript for 18/03/2011 (day 8); 26 (10 to 12) (Dr Hay)

²⁷ PEN.019.0927 @ 0961 - 0965 and tables 4 and 6

analysis of these 11 individuals has shown that five are likely to have been infected in Scotland.²⁸ Of the 11 analysed, there are 3 for whom it is hard to tell the place of infection. All of these have been classed as non-Scottish infections in the report. The analysis appears to have been conducted on the balance of probabilities. As far as patient 2 is concerned, he has been excluded on the basis of his infection prior to November 1984 and the absence of treatment records for the period 1980 to 1984. There is an indication that he was resident overseas. It is not clear when but it is assumed this was over this period. He had been treated only occasionally in London prior to 1980. In our view, the available evidence (omitting speculation as to the period which may have been spent abroad for which no records exist) suggests that the treatment received in Edinburgh in 1984 was the source of his infection. Patient 10 is also excluded. This patient received treatment in both Glasgow and Manchester in the year of likely infection (1985). The earlier the treatment, the more likely it seems, in our view, that it was responsible for the infection as processes are likely to have improved during 1985 for the exclusion (by screening or testing) of donors likely to be positive. It therefore seems more likely that this patient was infected in Glasgow than in Manchester. Patient 11 is also excluded. We are of the view that the infection of this patient is likely to have occurred in Inverness where he received most of his treatment between 1982 and 1984 when most infections appear to have occurred in Scotland. This would mean that 8 out of 11 should be deemed to be Scottish infections. This brings to total on this analysis to 70 infections.

Further, this report itself recognises that there may be individuals whose test may have been done in a centre outside Scotland (and who therefore would not be included in the base figure of 73) who may have been infected in Scotland as a result of having received treatment there. 23 such patients whose positive test was first reported by an English centre have been analysed and none were deemed to have been likely to have been infected in Scotland (at least none have been added to the total figure of 68 given in the report).²⁹ Patient 21 received the majority of his treatment between 1980 and 1983 in Edinburgh. It therefore seems likely that he was infected there, in our view. Further, there are 16 other patients who could have been infected in Scotland given that the dates of their first positive tests post-date treatment received in Scotland.

On the basis of this analysis and subject to the limitations outlined above, we would propose that a figure of 71 be the most likely minimum number of infections with HIV in the bleeding disorder community in Scotland. This figure could, of course, be higher in the event that other possible

²⁸ PEN.019.0927 @ 0962 - 0963

²⁹ PEN.019.0927 @ 0965

infections are included in the final total. Further, there are limitations on the information available to the UKHCDO and also the analysis has not given any consideration to the possibility that there could be individuals who were infected in Scotland and whose infection has been discovered somewhere other than England. Further, we note from the UKHCDO tables that 488 tests appear to have been carried out on Scottish patients.³⁰ In 1985 there were 690 bleeding disorder patients registered with Scottish haemophilia centres.³¹ This would suggest that not even all of those registered who may have been infected with HIV have been tested. Therefore, the figure of 71 should, in our view, be considered to be a minimum number of the number of patients infected with bleeding disorders infected with HIV by their exposure to blood products in Scotland.

In our view, it appears very difficult to understand why it is that there is such a significant discrepancy between the numbers presented in the first place by the Scottish haemophilia directors and the material later presented by the UKHCDO of which the directors are all members. We would invite the Inquiry to look into this more fully. It should also be remembered that the more detailed analysis of the 59 individual patients considered to have been infected by treatment received in Scotland by the haemophilia directors has not been replicated for other patients whom they did not include (for some reason) in their analysis. Further papers, such as the one prepared by Dr Cuthbertson on the number of patients likely to have been infected by domestically produced products³², have been prepared on the basis of this limited initial analysis. Therefore, such evidence requires to be considered with caution as it does not analyse, in our submission, the full cohort of those infected with HIV in the bleeding disorder community.

2. What proportion of the total number of people with bleeding disorders in Scotland were so infected before the introduction of factor concentrates heat treated so as to inactivate HIV?

The likely infection routes of the patients with bleeding disorders infected in Scotland and the likely timing of those infections is addressed more fully in our submission on the B2 topic, in particular in response to issue 4 on our list.³³ If one takes the figure produced by the UKHCDO of the number of bleeding disorder patients registered in Scotland in 1985 (690³⁴), the minimum number of 71 which

³⁰ PEN.019.0927 @ 0967

³¹ PEN.019.0927 @ 0957

³² PEN.012.1633

³³ PEN.019.0476 from 0478

³⁴ PEN.019.0927 @ 0957

we have presented above represents 10.29% of the entire bleeding disorder community registered with a Scottish centre at that time.

The tabulated figures provided by the UKHCDO suggest that there were 690 registered patients with bleeding disorders in Scotland in 1985 (by which time most of the infections with HIV had occurred).³⁵ It is interesting to note that there have only been 488 patients tested in Scotland.³⁶ This may give rise to the possibility that there are infected patients who have never been tested within the haemophilia system and whose infection may have been discovered by other medical means. This would, of course, have the effect of further rendering the total number of likely infections an underestimate. However, if one uses the minimum figure of 71 infections from the assessment above, this represents 10.29% of the number of patients registered in Scotland in 1985 or 14.44% of the patients first tested here.

3. What proportion of people with haemophilia so infected fall into the categories of mild, moderate and severe haemophiliacs?

Subject to the limitations expressed above about the accuracy of the data and the analysis conducted on it by the haemophilia directors, the material which is available would suggest that no mild haemophiliacs were infected with HIV in Scotland and only four moderate patients were so infected (two at Yorkhill and two at the GRI).³⁷ The chief distinguishing characteristic amongst the different severity classes would, of course, have been the quantity of products to which each patient would have been exposed. The more severe the condition, the greater the amount of product to which the patient would be likely to have been exposed. The statistical material would, therefore, tend to suggest that the greater the exposure to potentially harmful products, the greater the likelihood one had of becoming infected with HIV.

4. What proportion of people with haemophilia so infected is comprised of those suffering from haemophilia A and those suffering from haemophilia B?

Subject to the limitations expressed above about the accuracy of the data and the analysis conducted on it by the haemophilia directors, the material which is available would suggest that only

³⁵ PEN.019.0927 @ 0961 - 0957

³⁶ PEN.019.0927 @ 0961 - 0967

³⁷ Transcript for 18/03/2011 (day 8); 32 (9 to 25) (Dr Hay)

two haemophilia B sufferers were infected with HIV in Scotland (see our submission on the B2 topic, in particular our response to issues 4³⁸ and 17³⁹).

5. When, where and how is it likely that the patients with bleeding disorders in this group were infected with HIV?

As regards the likely place of infection, for the limited numbers analysed by the haemophilia directors, it seems that their tables are likely to give a more accurate picture of the place of infection as between the haemophilia centres in Scotland. This would appear to be due to the fact that the bulk of the figures in the UKHCDO analysis come from the research done and reproduced in table 3.⁴⁰ This data (though of interest in calculating total numbers) is perhaps less accurate when it comes to dividing the infections amongst the Scottish centres. For example, there is a clear discrepancy between the infection figures for Yorkhill and Glasgow Royal Infirmary in the general UKHCDO tables and the numbers presented by the haemophilia directors for the infections at those two centres. We assume that this is because the UKHCDO figures in table 3 are likely to result in more reports being made from the GRI of individuals who were probably infected as children at Yorkhill at an earlier date.

As regards the timing of the infections, Dr Hay conceded the possibility that patients could have been infected earlier than the available material might suggest based on the fact that there might not be archived samples for infections going back to the 1970s or even the early 1980s in some centres.⁴¹ He suggested that archive samples would be more likely to be available for the first half of the 1980s for the Edinburgh centre than for other centres in Scotland.⁴² It was explained by Professor Ludlam that the collection of samples in Edinburgh started in the 1970s "when we were interested in looking at hepatitis B infection and its transmission in haemophilia".⁴³ Though it is useful for present purposes that certain of these samples are available to enable a historical analysis to be undertaken of the likely timing of infections of patients, it is far from clear as to why these samples were stored in Edinburgh and not elsewhere, how such a regional variation could have been allowed to develop in this regard and the extent to which patients' consent was sought for the retention of samples for these purposes. This appears to be a more pressing issue when one

³⁸ PEN.019.0476 from 0478

³⁹ PEN.019.0476 from 0514

⁴⁰ PEN.019.0927 @ 0966 - 0967

⁴¹ Transcript for 18/03/2011 (day 8); 36 (17 to 22) (Dr Hay)

⁴² Transcript for 18/03/2011 (day 8); 37 (2 to 8) (Dr Hay)

⁴³ Transcript for 30/03/11 (day 14); 18 (11 to 25) (Professor Ludlam)

considers that the samples held for bleeding disorder patients appear to have been kept for numerous purposes, in various places and over a long period of time.⁴⁴

As regards all of these matters but in particular the method of infection, we would refer to the submission which we have presented to the Inquiry in connection with the B2 topic, in particular in response to issue number 4 on our list.⁴⁵

6. How many people with bleeding disorders in Scotland who were infected with HIV by blood products were also so infected with Hepatitis C?

In our submission, it is highly likely that all patients with bleeding disorders who were infected with HIV were also infected with hepatitis C. This assertion is based on the fact that the greater prevalence of hepatitis C in the Scottish blood donor population meant that if a patient with a bleeding disorder contracted HIV from a blood product, it is almost certain that that patient would have contracted hepatitis C as well. That virtually all of the HIV patients also contracted hepatitis C from blood products in Scotland was accepted in evidence by Dr Campbell Tait.⁴⁶ It was suggested by Dr Hay that all HIV infected patients would also have been exposed to HCV but that such patients may have cleared the hepatitis C virus.⁴⁷ We would argue that HIV-infected haemophilia patients have a much lower chance of clearing the hepatitis C virus than otherwise healthy patients who have only been exposed once to a single subtype of hepatitis C. In the first place, we refer to the submission made below concerning the low likelihood that multiply exposed haemophiliac patients would clear the hepatitis C virus. The statistical information presented to the Inquiry would suggest that almost all of the HIV infected patients were severe patients who would have had such multiple exposures. Secondly, given the immuno-suppressant qualities of HIV, it seems likely that such patients would not fall within the category of those who clear the hepatitis C virus.⁴⁸ Therefore, it is highly likely that patients with bleeding disorders who contracted HIV as a result of their exposure to blood products will have been co-infected. In our view, the issue of co-infection and its likely impact upon the prospects of clearing the hepatitis C virus or responding well to treatment for hepatitis C is

⁴⁴ Transcript for 30/03/2011 (day 14); 18 (16 to 20), 26 (12 to 15) 31 (16) to 32 (24) and 34 (2 to 14) (Professor Ludlam) - the purposes for which and the places in which these samples were kept are elaborated upon in these passages which include reference to collection "for virological assessment principally in relation to hepatitis B in the 1970s", "when blood was being taken for other purposes to check their haemoglobin or their blood chemistry", in virology...parallel to samples in haematology" and for clotting tests" "we also stored a serum sample" and "duplicate samples...to guard against the loss of potentially valuable samples"

⁴⁵ PEN.019.0476 from 0478

⁴⁶ Transcript for 30/03/11 (day 14); 132 (7 to 11) (Dr Tait)

⁴⁷ Transcript for 18/03/2011 (day 8); 46 (13 to 16) (Dr Hay)

⁴⁸ Transcript for 18/03/2011 (day 8); 47 (21) to 48 (18) (Dr Hay)

not well understood. Therefore, we would suggest that government-funded research into this important category of patients be recommended in order that their position and likely treatment and support needs be understood more fully.

The Inquiry has heard evidence about the particular problems of co-infection, including the worsening of symptoms of hepatitis C due to the immuno-suppressant characteristics of HIV infection and the difficulties which can be experienced when receiving treatment for both infections simultaneously. The statistical material would suggest that it would be erroneous to consider the evidence of the effects of HIV infection independently from the effects of hepatitis C infection as all of those with bleeding disorders infected with HIV in Scotland were likely to have been co-infected.

Hepatitis C infections amongst people with bleeding disorders

7. How many people with bleeding disorders were infected with Hepatitis C by blood products in Scotland?

Evidence from Health Protection Scotland

Professor Goldberg provided the Inquiry with a statement regarding the methodology adopted within Health Protection Scotland to calculate the number of individuals with bleeding disorders likely to have been infected with hepatitis C through their use of blood products in Scotland.⁴⁹ HPS was aware of 351 patients with bleeding disorders who were infected with hepatitis C and for whom there was no information that factor concentrates had been received outside Scotland. Therefore, this figure does not take any account of those who may have been treated outside Scotland but whose infection may have originated here. Further, it is clear that this analysis was based on the number of confirmed infections within this group. It serves as little more than a starting point for the Inquiry's analysis. It is noteworthy, in our view that the position of Health Protection Scotland as regards identifying and recording the likely route of infection appears to be that it is of little significance to them as (a) their priority lies in the prevention of further transmissions and there are likely to be few such transmissions by blood or blood products in the future and (b) the fact that those infected by blood or blood products represent only a small part of the total number of infections with hepatitis C in Scotland.⁵⁰ Further, we note that evidence of this type which focuses on

⁴⁹ PEN.001.0206

⁵⁰ Transcript for 16/03/2011 (day 6); 111 (18) to 112 (7) (Professor Goldberg)

the number of confirmed infections tends to underestimate the numbers infected. It is, in our view, important that numbers such as this be clearly understood in the context in which they were arrived at. They have the potential to give a misleading impression of the total numbers infected. We would wish to stress that we consider it important that the standard of proof applied to the establishment of figures such as this is an important general issue for those who suffer from HIV or hepatitis C. There requires, in our submission, to be a clear, fair and consistent standard applied to the establishment of whether individuals are infected and whether they became infected by their exposure to blood or blood products in Scotland. Figures which require absolute certainty that an individual is infected and that he was infected by blood or a blood product are both unfair and lead to an underestimate of the total likely number of infections by these routes in Scotland.

Evidence presented to the Inquiry from the UKHCDO

Evidence was also made available to the Inquiry from the UKHCDO regarding the methodology adopted by that organisation in trying to arrive at a total figure for infections within this group.⁵¹ The group provided a spreadsheet which detailed the treatment histories of the individuals who the UKHCDO had thought had been infected with hepatitis C in Scotland.⁵² This spreadsheet was based on information held within the UKHCDO database and included the treatment histories of the apparently infected individuals (insofar as they were available to the UKHCDO). Clearly the accuracy of the treatment histories was dependent on the accuracy and completeness of the treatment information provided to the UKHCDO by the local Scottish haemophilia centres about treatment received for bleeding disorders within Scotland (see above).

Evidence on the methodology which had been adopted by the UKHCDO in the compilation of this material was given by Dr Charles Hay and Dr Campbell Tait. Dr Hay accepted that epidemiological evidence was now to the effect that individuals treated in the UK (including Scotland) would have been likely to have been infected with hepatitis C on their first exposure to a concentrate, had they not been infected by being exposed to large amounts of cryoprecipitate before receiving a concentrate for the first time (which would have infected them before their first receipt of the concentrate). According to him, this approach was epidemiologically sound irrespective of whether the patient had received commercial or domestically produced concentrate. He did not, in his

⁵¹ PEN.013.0016

⁵² This was originally provided under reference PEN.001.0062 and then an update was provided entitled " Scot HCV Full Final Spreadsheet"

analysis, distinguish between factor VIII or factor IX concentrate in this regard.⁵³ It is clear that the material available to the UKHCDO may have limited its ability to provide the Inquiry with a comprehensive assessment of the numbers of patients likely to have been infected with hepatitis C (see the submission on HIV infections amongst the bleeding disorder community above). Further, it was pointed out by Dr Hay that there was an ongoing hepatitis C lookback exercise within the UKHCDO which had not yet been completed. The very existence of this exercise and the fact that it had not been completed by the time the UKHCDO material was presented to the Inquiry suggests that that material cannot be taken to be a comprehensive assessment of the numbers of bleeding disorders patients infected with hepatitis C in Scotland.⁵⁴ We wish to suggest that this exercise must be completed without delay and funded so that it can be completed as comprehensively and accurately as possible.

A figure of 410 patients was spoken to by Dr Hay as being the number of patients whom they knew to have been exposed to concentrate in Scotland. He regarded that to be a conservative number.⁵⁵ He accepted that this number would exclude those who may be infected in the community or those who received treatment locally outside the mainstream UKHCDO treatment system, whom the UKHCDO was still trying to trace.⁵⁶ It seems more likely that individuals infected with hepatitis C could have escaped detection by the UKHCDO system than those infected with HIV given the greater prevalence of that virus within the donor population in Scotland and hence the greater likelihood of infection, even if exposed to small amounts of treatment, particularly with factor concentrates. Hepatitis C is often a silent killer, destroying the liver over a period of decades, but remaining undiagnosed.

In the additional material presented to the Inquiry by the UKHCDO, this figure (corrected for double counting) had been adjusted to 447 of whom 193 (43.18%) were dead.⁵⁷ Dr Hay continued to consider this to be an underestimate based on those who continued to be excluded from the data based on the fact that they had been treated outwith the mainstream system.⁵⁸ It is interesting to note, in our view, that if one takes the figure of patients who were registered with Scottish centres for treatment in 1985, there were 690 such patients registered.⁵⁹ This figure has been corrected for double counting. The position appears to have been that it was the milder patients who were both

⁵³ Transcript for 18/03/2011 (day 8); 60 (23) to 61 (11) (Dr Hay)

⁵⁴ Transcript for 18/03/2011 (day 8); 26 (10 to 12) (Dr Hay)

⁵⁵ Transcript for 18/03/2011 (day 8); 60 (6 to 8) (Dr Hay)

⁵⁶ Transcript for 18/03/2011 (day 8); 26 (10 to 12) (Dr Hay)

⁵⁷ PEN.019.0927 @ 0984

⁵⁸ PEN.019.0927 @ 0983

⁵⁹ PEN.019.0927 @ 0957

most likely not to have been registered with a centre and also most likely to have not been exposed to treatment, including treatment with concentrates, outwith the centre. The discrepancy between the estimated number of hepatitis C infections in Scotland in the bleeding disorder community and the total number of patients registered in Scotland would tend, in our view, to suggest that the actual number of Scottish infections in this community is likely to be higher than the UKHCDO have estimated, based on their apparently valid assumption that infection would result from first infusion of a concentrate.

Prior to the updated UKHCDO statistical material being submitted to the Inquiry, the methodology document originally produced by the UKHCDO⁶⁰ was spoken to in evidence by Dr Campbell Tait.⁶¹ The starting point for the methodology adopted by the UKHCDO was the compilation of a list of all patients who had (according to the UKHCDO records) received treatment in Scotland between 1970 and 1989. In this list it was assumed that patients who had received factor concentrate treatment prior to 1989 had become infected with hepatitis C. Further, it was assumed that all patients who received treatment with cryoprecipitate prior to 1989 would also have been infected (unless they had tested negative for infection) on the basis that "it was known that patients treated with cryoprecipitate also commonly became infected with HCV" (no evidence cited).⁶² It was assumed that no patients would have been infected after 1989.⁶³ It is not at all clear why this starting point was used since the assumption that a patient treated with cryoprecipitate only (or fresh frozen plasma for that matter) would have become infected is, in our submission, epidemiologically unsound. The likelihood of infection via this route alone would depend on the quantity of such treatment which each patient had received. Further, given that cryoprecipitate (and FFP) was not heat treated, the list excludes any patients who received treatment with cryoprecipitate in sufficient quantities to infect them after 1989 but before the introduction of routine anti-HCV testing in September 1991. Further, if this exercise was a genuine attempt to arrive at the total number of infections in the bleeding disorder community in Scotland, we would suggest that there is equally no legitimate basis for assuming that none occurred prior to 1970.

The number of patients treated or registered in Scotland between 1970 and 1989 (according to the UKHCDO) was 715. Assumptions about infection were necessary on the basis that the UKHCDO did not have information about whether these individuals had or had not tested positive for HCV

⁶⁰ PEN.013.0016

⁶¹ Transcript for 30/03/2011 (day 14); From 75 (Dr Tait)

⁶² PEN.013.0016

⁶³ PEN.013.0016 @ para 1

infection.⁶⁴

A further assumption was applied that every patient on the list was infected in the place where he first received treatment, whatever that treatment was.⁶⁵ Given that some of the patients on the list had received their first treatment outside Scotland, this reduced the potential number of Scottish infections to 544. The epidemiological assumption that a patient was infected by his first treatment if that treatment was not with a factor concentrate is, in our view, unsound. Being manufactured from only a small number of donations, cryoprecipitate was considerably less likely to transmit hepatitis C infection on first infusion than factor concentrate. This may have resulted in patients who received treatment outside Scotland with either cryoprecipitate or FFP having been deducted from the Scottish list without good cause if they were subsequently treated with factor concentrates in Scotland. A further 76 were deducted from the list on the basis that UKHCDO information suggested that they had tested negative for HCV on PCR testing. No consideration appeared to have been given to (a) the possibility that PCR testing of the blood would not be a completely reliable guide as to whether a person's liver had been damaged by hepatitis C infection or (b) the timing of the test, important as it would have meant that people who tested negative but who had been infected and had responded to treatment may have been excluded. A further 8 were deducted on the basis that they had not received treatment with plasma derived products. That 15 patients were identified by Scottish haemophilia centres and did not appear on the UKHCDO list makes it clear that the raw data from which the directors were working from UKHCDO was not complete.⁶⁶ A total of 475 patients was arrived at by this method.⁶⁷ A further 16 appear to have been discounted either based on information that they had, in fact, tested negative for HCV infection (8) or that they had received extensive treatment outside the UK prior to infection (8), resulting in a final total of 459.⁶⁸ 314 of these patients have tested HCV positive and so this must be considered as the absolute minimum number of infections in this group based on the UKHCDO data.⁶⁹ It is noted that the number of individuals included within this total who have received cryoprecipitate therapy only is likely to be overstated (and who have not been tested as many of those excluded from the initial list who had tested negative will have been included in this group).⁷⁰ However, when one considers the increased usage of concentrate therapy in Scotland in the 1980s (considered below) we would estimate that there are unlikely to be very many individuals registered with or treated by a

⁶⁴ PEN.013.0016 @ para 1

⁶⁵ PEN.013.0016 @ para 3

⁶⁶ PEN.013.0016 @ 0017, para 6

⁶⁷ PEN.013.0016 @ 0017, para 7

⁶⁸ PEN.013.0016 @ 0017, para 8

⁶⁹ Transcript for 30/04/11 (day 14); 83 (5 to 18) (Dr Tait)

⁷⁰ PEN.013.0016 @ 0018, para 12

haemophilia centre in Scotland over this period who would not have received at least one concentrate treatment.

Conclusion

In her report of the Ross Committee published in March 2003, Dr Kate Soldan had estimated that around 500 individuals with bleeding disorders had been infected with hepatitis C as a result of their exposure to infected blood products in Scotland.⁷¹

In conclusion, we would suggest that the numbers infected in this community are likely to be nearer the number of patients registered with Scottish centres (690 in 1985 and 778 in 1990⁷²). We would suggest that it is likely that a number between the current estimates from the UKHCDO (447 and 459) and these numbers of registered patients is likely to represent the number of infections with hepatitis C of patients with bleeding disorders in Scotland. Given (a) that Scottish concentrates continued to be administered to Scottish patients which were not virally inactivated for hepatitis C until April 1997 (the number of registered patients had risen to 778 by 1990⁷³) (b) the fact that by 1985 certain infected patients may already have died and (c) the number of infections may include patients not registered with a centre who actually received treatment in Scotland, the UKHCDO numbers must be regarded as minimum numbers. Given this, we would obviously refute the suggestion of the UKHCDO directors that their number of 459 presented to the Inquiry was likely to represent the maximum number of infections in Scotland.⁷⁴

8. What proportion of the total number of people with bleeding disorders in Scotland were so infected before the introduction of factor concentrates heat treated so as to inactivate Hepatitis C?

On the basis of the epidemiological assumptions made by the UKHCDO in the compilation of the statistical material which they have presented to the Inquiry (as explored in more detail above) we would suggest that, certainly amongst the moderately and severely affected patients whose treatment would have been likely to have involved concentrate therapy prior to April 1987, the vast

⁷¹ Report of the Expert Group on Financial and other Support (March 2003) @ paragraph 4.8 - <http://www.scotland.gov.uk/Resource/Doc/47034/0024918.pdf> (including the material to be found in SGH.005.7203)

⁷² PEN.019.0927 @ 0957/0958

⁷³ PEN.019.0927 @ 0958

⁷⁴ PEN.013.0016 @ 0018, para 12

majority of patients with bleeding disorders in Scotland treated prior to that date will have been infected with hepatitis C. Many mild patients will also have been infected. In what is a close community, we submit that this disease has had a devastating and all consuming effect. Further, given that haemophilia is a hereditary disease, this high rate of infection has also affected many families who have required to come to terms with the infection of multiple members already affected by haemophilia.

9. What proportion of people with haemophilia so infected fall into the categories of mild, moderate and severe haemophiliacs?

As is submitted above, the prevalence of hepatitis C in the Scottish donor population meant that infections with the virus came from all parts of the bleeding disorder community. All patients treated with factor concentrates in Scotland are very likely to have been infected. As far as the Scottish factor VIII concentrate used prior to April 1987 was concerned (heated to 68 degrees for 24 hours) the PFC did not receive regular reports of apparent infections as it was assumed that most patients, if not all patients, who received concentrate prior to 1987 became infected with NANB hepatitis.⁷⁵

10. What proportion of people with haemophilia so infected is comprised of those suffering from haemophilia A and those suffering from haemophilia B?

Professor Goldberg presented some data from HPS regarding the 351 blood disorder patients whom he had designated as infected in Scotland. Given that he did not have data regarding the type of bleeding disorder for 240 of them, the data he presented is, in our view, of little value.⁷⁶

Dr Hay of the UKHCDO presented data relating to 447 patients who, it was assumed, had been infected due to their exposure to concentrate therapy in Scotland. Above, we have argued that this is likely to be an underestimate of the total number of patients infected with the disease in Scotland. However, of the 447 on whom information has been provided by the UKHCDO, 339 (75.84%) suffered from haemophilia A, 81 from haemophilia B (18.12%) and 26 from von Willebrand disease (5.82%) (one had a temporary coagulation disorder). It is interesting to note the low numbers of infections amongst the von Willebrand community. These are the patients who are most likely to have escaped the analysis of the UKHCDO. As Dr Hay pointed out in his analysis, the disease was

⁷⁵ Transcript for 7/12/11 (day 74); 61 (8 to 16) (Dr Perry)

⁷⁶ PEN.001.0206 @ 0207

likely to have been under-diagnosed at the material time.⁷⁷ This may have had the effect of it not being treated (and so minimising the number of infections in that community). Equally, however, it may have meant that it was not generally treated within a recognised centre but elsewhere with the result that infections within this community may have occurred which are not recognised within the UKHCDO system.

11. When, where and how is it likely that the patients with bleeding disorders in this group were infected with Hepatitis C?

The analysis conducted by the UKHCDO and spoken to in evidence by Dr Tait included allocation of the 475 patients which the exercise uncovered amongst the Scottish centres based on (a) the place of first treatment (which as we have argued above includes the false assumption of infection if the first treatment with otherwise than with a concentrate) and (b) by arbitrary allocation to a centre in the event that treatment was received at more than one centre in the first year of treatment.⁷⁸ Given the assumption that patients will have become infected with hepatitis C on first infusion of a factor concentrate the place of infection of the infected bleeding disorder patients will be likely to follow the population distribution of this group, given that most patients would tend to receive treatment locally (or at least at their nearest haemophilia centre). The figure provided by the UKHCDO for infections per centre come to a total of 600 infections.⁷⁹ This does not appear to have been corrected for double counting (see the total of 447 quoted earlier).⁸⁰ An allocation of the figure corrected for double counting would require to be undertaken and this figure compared with the distribution of patients being treated at the various centres over the relevant period during which infections occurred. This would enable any unduly high infection rate in any one centre to be detected.

As far as the timing of infection is concerned, the assumption of infection on first exposure to a concentrate would tend to suggest that some infections may have occurred long into the past, or at least at the time when larger pooled concentrates came into regular usage in Scotland. As noted above, it would seem legitimate to assume (as the PFC did) that first exposure to a concentrate would have been the most likely infection route in this community.

⁷⁷ Transcript for 18/03/11 (day 8); 22 (19) to 23 (9) (Dr Hay)

⁷⁸ PEN.013.0016 @ 0017, para 7

⁷⁹ PEN.019.0927 @ 0985

⁸⁰ PEN.019.0927 @ 0984

12. The prevalence of the different genotypes of Hepatitis C amongst those with bleeding disorders infected by blood products in Scotland

In this regard, we would refer to the assessment which we have already submitted to the Inquiry in the C3A section⁸¹ and, in particular, to the evidence of Professor Thomas regarding the fact that infections amongst the haemophilia community have tended to be with genotype 1 hepatitis C to a greater extent than is evidenced in the infected population at large.⁸² We would suggest that further government-funded research into the impact of multiple exposures to the hepatitis C virus on the likelihood of responding successfully to treatment be recommended.

HIV infections amongst the recipients of blood transfusions

13. How many people were infected with HIV through blood transfusions in Scotland?

Information available as to the number of individuals who are likely to have been infected with HIV as a result of a blood transfusion received in Scotland comes partly from the results of the HIV lookback exercise which was spoken to in evidence by Dr Jack Gillon (which identified 10 such infections) and reports from clinicians of possible blood transfusion related infections (which discovered a further 8 such infections).⁸³ In his evidence, Dr Gillon clarified that one of the 18 had in fact come to his attention from the Health Protection Scotland database about which SNBTS had previously known nothing.⁸⁴

The limitations of the lookback exercise as a means of identifying the total number of patients likely to have been infected by a blood transfusion received in Scotland is addressed below in connection with the hepatitis C lookback exercise. Similar limitations can be identified in the HIV lookback exercise which has been used as the primary means of ascertaining the number of HIV infections through blood transfusion. However, as Dr Gillon observed in his evidence the HIV lookback exercise is probably inherently more likely to have identified a more accurate number of infected persons than the HCV lookback. This, as he pointed out, was because of the fact that HIV had only been in

⁸¹ PEN.019.0657 @ 0677 - 0678

⁸² Transcript for 11/10/11 (day 52); 49 (4 to 6) (Professor Thomas)

⁸³ PEN.001.0038

⁸⁴ Transcript for 16/03/2011 (day 6); 59 (14 to 16)

the population for relatively few years when compared with HCV which had been around for longer, with the result that once a positive donor had been identified it was only his donations in the last few years which could have been infective and not necessarily those going back many years (as was the case with hepatitis C).⁸⁵ This would result in fewer recipients having to be traced and tested, making the process more likely to identify all infected recipients. Further, very much fewer HIV positive donors were identified and so efforts could be concentrated more fully on the identification of potentially infected recipients as there would be fewer of those given the lower number of positive donations. The lower prevalence of the HIV virus in the population (including the blood donor population) would also be likely to result in fewer transmissions than with hepatitis C. However, the greater likelihood of sexual transmission of HIV means that it is more probable that there would be secondary transmission of the disease though there would be likely to be fewer infections in blood transfusion recipients.

The discovery of blood transfusion as the source of infection by way of reports from a treating clinician is also an unreliable way of ascertaining a complete picture, as it is reliant on the treating clinician considering the possibility that might be the infection route, knowing how and taking the trouble to report it. This is considered in some more detail in connection with Dr Gillon's evidence on hepatitis C caused by blood transfusion below. It is of interest to note that Dr Gillon gave evidence to the effect that the identification of the blood transfusion infected patients by means of reporting by clinicians was an unreliable method of discovering infections caused by this route as there was no legal obligation on the clinician to report HIV either to the SNBTS or indeed to Health Protection Scotland as HIV was not a reportable disease.⁸⁶ There has never even been any agreed policy or an administrative requirement for possible cases of transfusion transmitted infection to be reported by clinicians to SNBTS.⁸⁷ It seems to us that in the interests of disease management and control it should be a legal requirement that all cases of infectious diseases should be reported to HPS and that all those cases of diseases which are transmissible through blood and blood products should also be required to be reported to the SNBTS. In our view, the numbers reported by Dr Gillon can only really be taken as a minimum number of HIV infections likely to have been caused by infective blood transfusions in Scotland.

An Infection Surveillance Report provided to the Inquiry by the National Microbiology Reference Unit includes data on the number of anti-HIV positive (repeat reactive) blood donations collected in

⁸⁵ Transcript for 16/03/11 (day 6); 18 (6 to 24) (Dr Gillon)

⁸⁶ Transcript for 16/03/11 (day 6); 63 (20) to 64 (1) (Dr Gillon)

⁸⁷ PEN.013.1557

Scotland between the introduction of testing here in October 1985 and 12 July 2010.⁸⁸ The data produced shows that in tests undertaken on blood collected between the introduction of testing and the end of 1985, the total number of positive donations per 100,000 was 5.94 (a total number of 4 positive donations in that 3 month period), though the report suggests that the total number of donations figure is only an estimate and so this may be inaccurate. For 1986, this figure had fallen to 4.34 positive donations per 100,000 donations taken (a total number of 14 for that year). For 1987, this figure had fallen to 3.32 positive donations per 100,000 donations taken (a total number of 10 for that year). These figures give some insight into the numbers of positive donations which would have entered the transfusion system in the event that testing had not been implemented. One can assume that before HIV screening measures were implemented in 1983/84 the number of positive donations in Scotland would have been likely to have been higher than the rates indicated here. However, this gives an indication of the number of HIV positive donations which would have been entering the system prior to the introduction of routine anti-HIV testing and despite screening measures.

14. When, where and how is it likely that the patients in this group were infected with HIV?

Dr Gillon supplied the Inquiry with information about the likely dates of infection of 15 of the 18 patients he had identified as having been infected with HIV through a Scottish blood transfusion.⁸⁹ His analysis of the available data suggested that the earliest known transmission was in August 1983 (patient 1), the latest in August 1986.⁹⁰ It is worthy of note that for the 3 patients for whom a date could not be ascertained with any precision, one (patient 2) may have been infected as early as 1981.⁹¹ The data indicates that HIV had certainly entered the Scottish donor pool some months before August 1983 when the blood which was transfused in August 1983 was collected and that it had perhaps entered it as early as 1981. The procedures being used to screen high risk donors in 1983 and 1984 failed to prevent the infections as a result of transfusions over this period identified in the Gillon report.

It is worthy of note that two of the infections were deemed to be due to transfusions which occurred in August 1986 (patients 11 and 18). Given that routine anti-HIV screening took place from October 1985, this indicates that the screening process which was implemented at that time was not

⁸⁸ PEN.001.0053 @ 0056

⁸⁹ PEN.001.0038 @ 0038 - 0039

⁹⁰ PEN.001.0038 @ 0042

⁹¹ PEN.001.0038 @ 0042

foolproof in preventing HIV infection by way of blood transfusion even as late as 10 months after routine screening was implemented. Dr Gillon reported that 8 of these cases were caused by blood collected in Edinburgh, 7 by blood collected in Glasgow, 3 by blood collected in Dundee.⁹² Further, one infection was caused as a result of a transfusion received in September 1985 (patient 16). This infection might not have occurred had routine anti-HIV screening of blood taken place earlier in 1985. We refer to our submission in the B4 section in connection with this.

The limitations on the reporting and detection mechanisms for these patients mean that the information available in connection with them can only really be taken as a sample of the total potential number of individuals infected with HIV. These individuals cannot necessarily be taken as representative of this group as a whole. The reliability of the information about timing of infection and the place the infective blood was collected must be deemed to be limited in light of this fact.

15. The desirability of an HIV Lookback exercise

We would refer to the comments which we already made in this regard above and in our submission to the Inquiry on the B5(b) topic⁹³, as well as the submissions we have made above.

Hepatitis C infections amongst the recipients of blood transfusions

16. How many people were infected with Hepatitis C through blood transfusions in Scotland?

The importance of the issue and the approach necessary to establish the infection rate in this population

In our view, it is of considerable importance that the Inquiry is able to arrive at a conclusive view as to the number of people likely to have been infected with hepatitis C as a result of receiving a blood transfusion in Scotland. There are a number of reasons for this. In the first place, it appears to be accepted by all those who have commented that this is likely to be the largest population in Scotland of those infected with the viruses with which the Inquiry is concerned through blood or blood products. The population of blood transfusion recipients is unlike the community of those with

⁹² PEN.001.0038 @ 0039

⁹³ PEN.019.0571 @ 0580

bleeding disorders who usually contracted infections through their use of blood products as that latter community is relatively small, traceable and subject to regular blood analysis. Secondly, there is the nature of the disease itself. It has a long incubation period and the symptoms may not manifest themselves for some time after an individual has contracted the disease. These circumstances give rise to problems of detection. It is therefore possible that potentially large numbers of individuals have been infected who are not aware that they are infected or how they became infected and have not received treatment. This is a significant public health issue. Thirdly, as is noted in our submissions in connection with the C2 and C4 topics, it is necessary to have a starting point as to (a) the total number of infections likely to have been caused by this route and (b) the likely timing of these infections, in order to draw conclusions about the significance of failures to implement testing regimes designed to halt the spread of infection predominantly by this route in the latter half of the 1980s into the early part of the 1990s.

This is an issue which appears to have caused some considerable difficulty for the Inquiry. Efforts have been made to try to understand the likely infection rates amongst this population. It does appear that the nature of the disease and the limitations on efforts made to uncover actual infection rates through this route may have resulted in an inaccurate impression having been reached by some as to the numbers affected. It does appear that epidemiological analysis is necessary in order to be able to estimate accurately the full extent of infection in this population. The accuracy and reliability of the available epidemiological material is therefore considered below.

SNBTS data

The Inquiry received a report⁹⁴ and heard oral evidence from Dr Jack Gillon of the SNBTS⁹⁵ on his efforts to try to arrive at a figure for those likely to have been infected with hepatitis C as a result of a blood transfusion received in Scotland. He identified 4 groups of people who were definitely infected and were likely to have been infected by a blood transfusion in Scotland. He did not think it likely that there was any overlap between the groups.⁹⁶ In the first place, Dr Gillon had identified 59 blood donors who had tested positive on giving blood whose only risk factor for the source of their infection was having received a blood transfusion themselves.⁹⁷ It was confirmed by Dr Gillon in his evidence that this category included those who had tested positive on giving a donation (867 in

⁹⁴ PEN.001.0043

⁹⁵ Transcript for 16/03/11 (day 6); 1 to 88 (Dr Gillon)

⁹⁶ Transcript for 16/03/11 (day 6); 40 (11 to 14) (Dr Gillon)

⁹⁷ PEN.001.0043

total) who had identified blood transfusion as the only risk factor for them.⁹⁸ This is clearly unreliable as (a) it depends on reporting from the individual and no further examination (b) it does not mean that those who were positive but who had another risk factors were infected by the other risk factor and not the transfusion and (c) it takes no account of where the person even alleges to have had their transfusion which they have claimed was their only risk factor.⁹⁹ These individuals are just individuals who happen to have presented as blood donors in Scotland. Identifying potential patients by looking at donors is clearly a very limited way of accessing the patients who may or may not decide to be blood donors themselves.

Dr Gillon also relied upon data of confirmed positives which had been linked to blood transfusions as a result of reporting to the SNBTS by clinicians treating patients with symptoms of hepatitis C. Investigations had identified 28 individuals by this route.¹⁰⁰ He had attempted to arrive at a number by cross referencing his figures with figures from other sources, such as Health Protection Scotland. Despite this, he accepted that the numbers which he proposed were likely to be a restricted representation of the likely total in reality due to the fact that identification of an infected individual would depend on it occurring to a clinician presented with a patient showing the signs of hepatitis C infection that it might have been transmitted by a blood transfusion and going to the trouble of reporting that possibility to the blood transfusion services.¹⁰¹ He also pointed out that Health Protection Scotland were more likely to receive reports of infection as they were able to put in place systems which made it automatic that they would receive notification of every confirmed positive test. The reporting was thus more likely than the reporting system relied upon by the SNBTS which was much more dependent on the clinician making a deduction and then a report.¹⁰²

Further, he gave evidence as to the way in which reports of possible transfusion transmitted infection had been investigated both before the conclusion of the lookback exercise in 1998 and since then. He identified that there were 58 such reports which had been made and that there were difficulties in identifying whether transfusion was the most likely cause.¹⁰³

Dr Gillon also identified confirmed positive patients likely to have been infected by blood transfusions in Scotland by referring to the data compiled as a result of the HCV lookback exercise. A

⁹⁸ Transcript for 16/03/11 (day 6); 37 (17 to 20) (Dr Gillon)

⁹⁹ Transcript for 16/03/11 (day 6); 73 (24) to 74 (3) (Dr Gillon)

¹⁰⁰ PEN.001.0043 @ 0044

¹⁰¹ Transcript for 16/03/11 (day 6); 9 (3 to 18) (Dr Gillon)

¹⁰² Transcript for 16/03/11 (day 6); 9 (19) to 10 (14) (Dr Gillon)

¹⁰³ Transcript for 16/03/11 (day 6); 33 (18) to 36 (17) (Dr Gillon)

UK wide lookback exercise was undertaken from April 1995 and was deemed practically complete in 1998. 133 individuals were identified as definitely having been infected with hepatitis C by a blood transfusion in Scotland via this process.¹⁰⁴ In fact, further evidence available to the Inquiry demonstrates the limitations of this process in providing a definitive answer to the number of blood transfusion related infections. The full results of the lookback exercise demonstrate that, in fact, 880 patients had been identified as having been exposed to the virus by having received a blood component from a repeat donor found to have been infected when his repeat donation was tested.¹⁰⁵ Only 70 of these recipients were tested and found to be negative. The position of the others is either that they were tested and found positive (the figure of 133 given by Dr Gillon) or they were not tested either because they were dead (536) or not traced (78). In any event, the 880 only represents the recipients of a proportion (1,356 out of 2,026 - 66.9%) of the components made from blood donated by donors found to be positive from the lookback process. Clearly the number of potentially infected individuals even identified via this limited process is potentially much greater than the figure of 133 given by Dr Gillon. The lookback exercise was generally commendable as means of tracing individuals infected by blood transfusions who would not otherwise be traced and who could receive counselling and treatment for their infection. Further, it provides a useful amount of "hard data" relating to infections amongst a small proportion of the community infected via this route. However, it represents, in our view, an inadequate means of identifying a total number those who are likely to have been infected as a result of receiving blood transfusions in Scotland.

The lookback exercise is of limited use in finding a definitive answer to this issue as it only starts with repeat donors who come back and are therefore able to be tested and their previous donations tested. It would not identify any infections which occurred as a result of the donation of a donor who either (a) did not return to give blood again or (b) for whom the records of his previous donations were not adequate to identify any or all of the recipients (either as a result of faulty record keeping relating to the timing of any previous donations or relating to the identification of recipients of those donations). We refer to our submission on this issue in the C5(b) section, in particular on (a) the delays occasioned in the introduction of a hepatitis C lookback exercise in Scotland, which resulted in the process being less likely to identify as many positive recipients of blood as an earlier process may have done¹⁰⁶ and (b) the limitations an exercise based only around

¹⁰⁴ PEN.001.0043 @ 0044

¹⁰⁵ PEN.002.0801 @ 0803 - report by Andy Kerr to the Health Committee of the Scottish Parliament on the HCV lookback exercise conducted by SNBTS dated 31 January 2006 - @ 0804

¹⁰⁶ PEN.019.0742 @ PEN.019.0748 to PEN.019.0751

repeat donors.¹⁰⁷ We also refer to the evidence of Dr Alexander on the limitations of the lookback, in particular the decreasing likelihood of positive donors returning to donate at the material time.¹⁰⁸

Dr Gillon presented evidence as to the likely dates of transmission for 103 of the 133 infected patients identified via the lookback and for those identified via a clinician report.¹⁰⁹ The earliest transmission accepted as definite was in 1977, and the last in March 1991. As far as the earliest date of transmission revealed via this process is concerned, the process could never have been an accurate representation of the likely earliest date of transmission via this route. Dr Gillon gave evidence to the effect that it was thought that hepatitis C was an ancient virus and that blood transfusion had really started at around the time of the Second World War.¹¹⁰ The proposition that the lookback data demonstrates that the earliest infection in Scotland was in 1977 is inaccurate, given the likelihood that the virus existed before that time and the likelihood (discussed above) that the further back one goes in time, the less likely it is that the infected recipient will still be alive to be able to be identified, either due to death, problems in tracing the individual due to moving etc or due to the lack of accurate records to enable the recipients of the infected blood to be traced.¹¹¹

As was accepted by Dr Gillon in his evidence, the delay in the implementation of the lookback process means that it is more likely that it would not have identified the total number of those infected by this route, in part because the longer one waits, the more likely it is that infected patients would have died (and would not therefore be traced) either from hepatitis C or some other cause.¹¹² We would refer to the submissions which we have made about the delays in implementing the lookback process in our C5(b) submission.¹¹³ These delays have rendered the comprehensiveness of the results even more questionable than they would otherwise have been.

Finally Dr Gillon identified a further 18 individuals who were definitely positive for hepatitis C who had been identified by the west of Scotland renal unit when they started testing their patients in 1991.

¹⁰⁷ PEN.019.0742 @ PEN.019.0751 to PEN.019.0752

¹⁰⁸ PEN.019.0742 @ PEN.019.0751 to PEN.019.0752; and Transcript for 17/01/12 (Day 85); 125 (2 to 18) (Dr Alexander)

¹⁰⁹ PEN.001.0043 @ 0045 - 0046

¹¹⁰ Transcript for 16/03/11 (day 6); 19 (23) to 20 (20) (Dr Gillon)

¹¹¹ Transcript for 16/03/11 (day 6); 23 (3) to 24 (8) (Dr Gillon)

¹¹² Transcript for 16/03/11 (day 6); 22 (7 to 12) (Dr Gillon)

¹¹³ PEN.019.0742 @ PEN.019.0748 to PEN.019.0751

As was accepted by Dr Gillon in his oral evidence, the deficiencies of the methods of blood related infection identification which he had used resulted in the number of blood transfusion recipients identified through that process as having been so infected only being able to be regarded as a minimum number of individuals so infected.¹¹⁴ This, in our view, means that his evidence is merely the starting point for a thorough consideration of this important matter.

The epidemiological analysis of the scale of infection in this population

Against this background, the Inquiry procured a report¹¹⁵ and heard evidence from the epidemiologist Professor David Goldberg on this issue.¹¹⁶ In light of the oral evidence which he gave, further lines of inquiry were pursued with him.¹¹⁷ The conclusions reached by Professor Goldberg in the material which he has presented to the Inquiry on this issue would appear to amount to the following:

- The total number of reports made to HPS and recorded on their hepatitis C database (established in 1996) of individuals infected with hepatitis C possibly as a result of blood transfusions in Scotland (excluding those reported with a known history of injected drug use and those who had received blood transfusions in England) is 304.¹¹⁸
- Professor Goldberg & Ors provided a report to the Inquiry which attempted to conduct a more epidemiological analysis of the numbers of individuals likely to have been infected by blood transfusions in Scotland. A report in this regard was provided in October 2011.¹¹⁹ This paper estimated the likely number of infections by way of lower, mid and upper estimates, which are 1183, 1532 and 1978 respectively.¹²⁰

¹¹⁴ Transcript for 16/03/11 (day 6); 77 (15 to 20) (Dr Gillon)

¹¹⁵ PEN.013.0014

¹¹⁶ Transcript for 16/03/11 (day 6); from 95 (Professor Goldberg)

¹¹⁷ From the material available to us it would appear that the Inquiry wrote to Professor Goldberg seeking his further input and he replied with an updated report in October 2011 (PEN.018.1561), a further letter was sent to him on 14 February 2012 (PEN.019.0896) which prompted a letter which was sent to the CLO on 28 February 2012 (PEN.019.0896) and a further report dated 1 March 2012 (PEN.019.0899). A further request was sent to Professor Goldberg on 23 March 2012 (PEN.019.0914) enclosing comments made by Professor Oliver James on the analysis which had been presented by Professor Goldberg (PEN.019.0916) which prompted a final response from Professor Goldberg dated 29 May 2012 (PEN.019.0922)

¹¹⁸ PEN.013.0014

¹¹⁹ PEN.018.1561

¹²⁰ PEN.018.1561 @ 1563

Whilst recognising the need for an expert epidemiological view on these matters, we have found the evidence presented by Professor Goldberg to be unsatisfactory as a means of arriving at any conclusions on this and related matters. The reasons for this are as follows:

- a) Despite having been asked to give an expert epidemiological view on the numbers infected with hepatitis C as a result of blood transfusions in Scotland, in his initial report to the Inquiry on this issue Professor Goldberg simply detailed the number of positives reported to HPS who may have acquired the infection by means of a blood transfusion in Scotland (304). He accepted in evidence that he had not conducted an epidemiological analysis.¹²¹ It was further explained by him that the exercise of gathering information within HPS was primarily for the purpose of prevention of future infection and not for other purposes of public health with the result (as far as the gathering of information of hepatitis C caused by blood transfusion) that little attention appears to have been paid to the numbers infected by blood or blood transfusions due to the perceived low risk of future infection by these routes.¹²²
- b) Even for the numbers he provided initially, Professor Goldberg confirmed that he could not say that these 304 were more than possible infections as all this was based on was a report coming to him on a form which was not subject to any further examination by him or anybody else of the accuracy of the information contained within it.¹²³

As far as the October 2011 report¹²⁴ is concerned:

- a) For some reason the methodology used by the Goldberg group was initially not produced to the Inquiry with the report which was drafted by HPS in October 2011.¹²⁵ Further, as was indicated by Professor Goldberg to the Inquiry in his letter of 28 February 2012, the epidemiological analysis which he and others at HPS had carried out in conjunction with Drs Gillon and McClelland from SNBTS was "not subjected to rigorous quality assurance (peer review) because of time constraints."¹²⁶

¹²¹ Transcript for 16/03/11 (day 6); 95 (2 to 15) (Professor Goldberg)

¹²² Transcript for 16/03/11 (day 6); 111 (18) to 112 (7) (Professor Goldberg)

¹²³ Transcript for 16/03/11 (day 6); 106 (15 to 24) and 108 (23) to 109 (8) (Professor Goldberg) and PEN.001.0206 @ 0212

¹²⁴ PEN.018.1561

¹²⁵ PEN.018.1561 @ para 4.1

¹²⁶ PEN.019.0896 @ 0898

- b) The starting point for the analysis of the likely prevalence of HCV in the donor population is the study compiled by Crawford & Ors from 1991/92 when anti-HCV testing came into being. This gives a starting rate of 0.088% HCV prevalence. That prevalence rate is then subjected to a number of reductions in order to try to predict the likely prevalence rate for the period during which the analysis is being conducted.¹²⁷ The legitimacy of the reductions is considered further below. However, the starting prevalence is also questionable. It comes from a limited study at a particular point in time. Professor Thomas expressed the view that between 1970 and 1990 the prevalence of HCV in the UK blood donating general community was around 0.5%.¹²⁸ He accepted that the levels were found to be lower than that in blood tested in the first 6 months to a year after anti-HCV screening was introduced. Professor Thomas explained that the figure he had been using was derived from a paper by Minor¹²⁹ (whom he thought would be privy to the accurate figures) which reported "a frequency of 0.4% consistent with previously reported figures" in the plasma from UK donors used to make factor concentrates.¹³⁰ The application of these higher (though vouched) prevalence rates as the starting point for the Goldberg (or indeed the Soldan) analysis would have a significant effect on the calculation of the total number of likely infections. The resultant number would be significantly higher if this alternative starting point were used.
- c) Below we analyse the approach adopted by the Soldan group in reaching an estimate as to the likely numbers infected in this group in the UK and in Scotland. That group appears, as a starting point in the calculation of the likely number of infected donations, to have used the HCV prevalence rate detected in England and Scotland respectively at a time when routine testing was introduced in 1991/92. The Goldberg analysis attempts to factor in the fact that the prevalence rate would have been likely to have decreased over the period under examination. Rightly, in our view, he appears to wish to question the legitimacy of the 1991/92 rate being used as a basis for the estimation of infection at earlier dates. As is pointed out in the October 2011 report, the increasing measures implemented to exclude high risk donors from donating is likely to have had some effect in lowering the prevalence rate in the donor population in Scotland over the relevant period.

¹²⁷ PEN.019.0899 @ 0902

¹²⁸ Transcript for 11/10/11 (day 52); 78 (21 to 23) under reference to his report (Professor Thomas)

¹²⁹ SGF.001.1380

¹³⁰ Transcript for 11/10/11 (day 52); 113 (Professor Thomas)

In particular, the Goldberg report attempts to factor in the likely impact of measures taken by SNBTS to reduce the number of HCV positive blood donations entering the system, in particular (a) the deferral of blood donors at high risk for HIV infection from 1984 and (b) the introduction of anti-HCV screening in 1991.¹³¹ Given that the period under examination is 1970 to 1991, the latter of these seems totally irrelevant. As far as the impact of the former is concerned, the analysis conducted by the Goldberg team appears to be based on an unattributed, unsupported assertion that prevalence of HCV in the donor population reduced "constantly by 66% during 1984 to 1991".¹³² The meaning of that assertion is not clear. Given that the donor deferral efforts introduced in the mid 1980s were predominantly designed to minimise the risks of HIV and not HCV transmission and the fact that efforts were focussed on the deferral of MSMs, it seems hard to imagine how such a considerable, though indirect, reduction in the number of positive donations could have been achieved. An immediate 66% reduction has been applied with no consideration of the fact that the policy was introduced inconsistently throughout Scotland and would, in any event, have taken time to have any indirect effect on the number of HCV positive donations. There is no attempt to explain how this figure has been arrived at other than to say that it is derived from "limited local data and [unidentified] expert opinion". This constitutes assumption 3 in the methodology document provided to the Inquiry in March 2012¹³³ and, as can be seen from that document, this assumption has had a significant effect in reducing the number of infections which this group think have occurred in this population.¹³⁴ In light of the significance of this unexplained statistic in the overall Goldberg analysis, the final figures cannot be accorded any great weight in the Inquiry's analysis of this issue.

- d) It is assumed in the analysis that 25% of infected individuals clear the virus within 6 months of infection and thus, though they were exposed to the virus (and thus would test antibody positive) they would not be able to infect others if they donated blood (assumption 2). As is discussed further below, there appear to be a number of figures which have been quoted as possible clearance rates for the virus. 25% comes from the higher end of these figures. The source of this figure does not appear to be a particular local study but instead the "Global Burden of Hepatitis C Working Group 2004".¹³⁵ The clearance rate applied is therefore questionable. Further, it also appears questionable to have assumed that all of those who

¹³¹ PEN.018.1561 @ para 3

¹³² PEN.019.0896 @ response to question 2

¹³³ PEN.019.0899 @ 0900

¹³⁴ PEN.019.0899 @ 0902

¹³⁵ PEN.019.0899 @ 0902

clear the virus do not have the potential to be infectious, at last for a period before the virus has cleared. As can be seen from the methodology document, this has a significant effect on reducing the prevalence rate of those who are antibody positive to those donors who may have been infectious.¹³⁶

- e) The Goldberg analysis assumes the average number of blood components made per donation.¹³⁷ This is based on "limited local data and expert opinion".¹³⁸ The Soldan analysis uses hard data from the lookback process to arrive at this figure (1.6 units). We are unsure why this could not have been done here. The same appears to be the case with the assumption made about the probability of a blood component being transfused (see below). Indeed, unlike the Soldan analysis, the Goldberg paper appears to make no use whatsoever of the only hard data which does exist, ie that which emanates from the lookback process.
- f) The Goldberg analysis assumes the likelihood of a blood component being transfused as 56%. This is based on "limited local data and expert opinion".¹³⁹ Given the assumption that each HCV contaminated unit was transfused to a different person (assumption 7)¹⁴⁰ this would have had the effect of reducing the final estimated number of infections by around half. This cannot, in our view, be a proper scientific basis upon which to make such a calculation. Without further explanation, we cannot accept that so many units of blood were being discarded at a time when blood donations were rare and valued. The Soldan paper uses a figure of 66% transfusion.
- g) The calculation of the probability of a transfused blood component being infected with HCV is dealt with at paragraph 4.3.1 of the October 2011 report.¹⁴¹ This seems to have been influenced to a large extent by data available relating to the number of injecting drug users in the general population. Assumption 4 in the methodology document confirms that it has been assumed for the purposes of the calculation that the prevalence of injecting drug users (derived from a report by Hutchinson) is used to calculate the likely prevalence rate amongst the donor population historically. As there were fewer intravenous drug users in the general population in the 1970s, it is assumed that the number of HCV antibody positive donors

¹³⁶ PEN.019.0899 @ 0902

¹³⁷ PEN.018.1561 @ para 4.3

¹³⁸ Assumption 5 @ PEN.019.0899 @ 0900

¹³⁹ Assumption 6 @ PEN.019.0899 @ 0900

¹⁴⁰ PEN.019.0899 @ 0900

¹⁴¹ PEN.018.1561 @ 1562

would be proportionately less. It is not clear why information about the number of injecting drug users in the general population should be taken to reflect the likely infectivity of blood donated in Scotland where there would appear to be other risk factors for transmission of HCV and no necessary correlation between the numbers of injecting drug users in the general population and in the donor population. This can hardly be taken to be representative of the likely number of such injecting drug users in the donor population, as that community is likely to be more socially responsible and thus contain a lower prevalence than in the general population. Further, no consideration appears to have been given to the fact that, even if there were a proportionate rise (as the Goldberg analysis contemplates) in the number of positive donors based on the size of the injecting drug user population, the donor exclusion measures used to stop them donating blood was also considerably less. In our view, these two competing factors may well have cancelled each other out, in particular given that until 1983/84 blood was collected from a population in Scotland (prisoners) which would be likely to have had a far higher than average intravenous drug using population. The impact of the injecting drug user statistics in the general population is, in our submission, given undue weight in the Goldberg analysis. This has the effect of considerably and unjustifiably reducing the likely prevalence rates and, consequently, the predicted number of infections.

- h) The October 2011 paper does not attempt to show how the final figures for the upper, mid and lower ends of the scale have been reached nor does it even try to define what these terms mean.¹⁴²

- i) We note the discrepancy between the numbers in the Goldberg analysis and those calculated by Professor James in his methodology applying, in the first place, (a) the number of payments to living patients infected by blood transfusions alive post 2003 (405/607) and (b) the numbers of those infected by this route who are still alive according to a Danish study published in August 2011.¹⁴³ We note (a) that this gives a figure of 2,670 likely to have been infected by contaminated blood in Scotland and (b) the fact that Professor Goldberg has dismissed this as a reliable indicator of the likely numbers of infections not due to the application of the Danish mortality data to the Scottish position but due to the reliability of the Skipton data.¹⁴⁴ In our view, the Skipton figure of 405 is likely to constitute an

¹⁴² PEN.018.1561 @ 1562 - 1563

¹⁴³ PEN.019.0916

¹⁴⁴ PEN.019.0922

underestimate of the likely number of living patients post 2003 who were infected by a blood transfusion. This is predominantly due to (a) the strictness with which the Skipton criteria are applied (in particular in cases where patients do not have sufficient medical records to support their claim) (b) the fact that there are likely to be significant numbers of individuals who may not know that they are infected with hepatitis C or indeed may not know that they acquired their infection via this route with the result that they have not considered making an application to the fund and (c) the fact that certain potential applicants may not even know of the existence of the fund. The figure proposed by Professor James (1.74 times the estimated median number of HCV infections by this route in the Goldberg analysis) constitutes a useful check and indicates that the Goldberg figures are likely to be too low. This is also the case with the other figures which Professor James has suggested, all of which are higher than the median estimate of the Goldberg group, one by as much as 2.61 times (4,000).¹⁴⁵ This figure of 4,000 is arrived at by trying to factor into the number of successful Skipton applicants an increase to account for the number who are likely to be alive but have not received a payment for whatever reason, applied to the Danish survival rate as at 2011.

Therefore, we submit that the Goldberg analysis cannot be accepted as a reliable measure of the likely number of infections with hepatitis C in Scotland by blood transfusion. Further, it simply cannot be valid to conclude (as the Goldberg group does) that the implementation of donor exclusion measures (designed primarily to prevent HIV transmission) "prevented thousands of blood transfusion recipients becoming infected" when the reduction in the numbers of likely infections after these measures appears to be based on baseless assumptions.¹⁴⁶ That the other measure taken by the SNBTS to reduce transmission of HCV (the introduction of routine anti-HCV testing) also prevented thousands of infections is also, in our view, an invalid assertion.¹⁴⁷ It is hardly surprising that a testing mechanism for the virus prevented transmission of the virus. The fact that it took until September 1991 to introduce such a mechanism did not prevent but, in fact, caused unnecessary infections of blood transfusion recipients. We refer to our submission in the C4 section in this regard.¹⁴⁸

¹⁴⁵ PEN.019.0916 @ 0917

¹⁴⁶ PEN.018.1561 @ 1564

¹⁴⁷ PEN.018.1561 @ 1564

¹⁴⁸ PEN.019.0712

Alternative methods of analysis

We also note that Dr Kate Soldan, an epidemiologist at the Department of Health's Public Health Laboratory Service Communicable Disease Surveillance Centre, conducted (along with others) a detailed analysis of this question for England.¹⁴⁹ The analysis conducted by here appears to have taken the UK wide HCV lookback as the starting point for the analysis.

As far as the UK part of the analysis of concerned, the way in which the analysis was done is reflected in the article written by Soldan & Ors dated 2002.¹⁵⁰ As noted, the starting point is the English lookback data. There is a recognition at the outset (a) that the lookback only identified a limited number of infected components (with the result that some infected components and their potential to infect were not considered as part of the exercise) and (b) that for one reason or another certain components which were found to have been likely to have been infected based on an analysis of previous donations made by a positive donor were not analysed as part of the lookback process. The analysis undertaken appears to attempt to work from certain prevalence figures shown by the lookback data and extrapolate it to consider the many infected donations likely to have been given whose destination could not be traced by the lookback exercise. The calculations appear to have been complicated to a certain extent by the fact that the data available to the Soldan group did not represent all of the lookback data but only information from 8 blood centres which handled 80% (not all) of the blood components which entered the lookback programme.¹⁵¹

The methodology appears to work in the following steps:

- a) Use existing prevalence figures for the donor population in England (derived from studies done in the first 4 months after routine testing was introduced in 1991) to work out how many positive donations were likely to have been given in England over the relevant period (by applying the prevalence rate to the number of positive donations). This resulted in a prevalence rate of 0.066% being applied to the total number of donations taken made between January 1980 and September 1991.¹⁵²
- b) Work out how many infected components are likely to have been made from these positive donations (based on the fact that one blood donation is likely to be made into a number of

¹⁴⁹ An abstract of that analysis appears to have been produced at SNB.008.2106 and the article itself is at PEN.013.1580

¹⁵⁰ PEN.013.1580

¹⁵¹ PEN.013.1580 @ 1581

¹⁵² PEN.013.1580 @ 1581

- components). This results in a total number of probably infected components. This was calculated by using the observed number of components made from the components made from donations in the lookback programme (a figure of 1.6 components per donation).¹⁵³
- c) Deduct from this the likely number of non-transfused positive components based on data for the proportion of components actually transfused (based on the fact that some will be soiled, lost etc). This will give you a figure for the likely number of transfused infected components.
 - d) A calculation of the number of likely infected recipients needs to be made. This can be deduced from the number of infected components by (a) reducing the number of components in order to reflect the fact recipients will receive a number of components (ie not every recipient gets a single unit of blood) and (b) making a deduction to reflect the fact that not all recipients of an infected component will actually become infected with the virus. This has been done in the Soldan analysis by using the infection rates observed in the lookback exercise and extrapolating them for the total number of likely transfused infected components to give an estimated overall figure of likely infections.

The analysis attempts to take the total number of infected components, work out the observed prevalence of anti-HCV in English blood donors at the start of testing in 1991 (which was assumed to give an indication of likely prevalence of HCV in the English blood donor population in the pre-testing years) and plot the likely number of recipients exposed to infected blood and calculate the number of likely infected recipients from that.¹⁵⁴ The analysis appears to have been carried out to the end of 1995. The results appear to demonstrate that there were less than 14,000 individuals likely to have been infected with hepatitis C as a result of blood transfusions in England in the decade prior to the introduction of routine anti-HCV testing (to 1991). Over 60% of these were expected to have died by the end of 1995.

It appears that this analysis works on the basis that the infected donations detected by means of the lookback exercise and traced through to the point where a recipient could be identified and was tested can be used as a representative basis for the assessment of the number of recipients likely to have been infected who received an infected component. Though the assumption that the infection rate amongst the lookback-identified positive recipients can be applied more generally to the population of those who received all blood components is not a valid one, the advantage of this

¹⁵³ PEN.013.1580 @ 1583

¹⁵⁴ SGH.005.7203

approach is that it does take some cognisance of what limited "hard" data about the relationship between exposure to an infected donation and infection that the lookback was able to provide.

In our view, the approach is to be preferred over the general approach taken by the Goldberg team in seeking to arrive at a figure for the numbers likely to have been infected with hepatitis C as a result of blood transfusions in Scotland.

Dr Soldan also carried out an analysis of the numbers likely to have been infected in Scotland. She gave evidence to the Ross Committee to the effect that 3,498 people received components likely to have been infected with hepatitis C as a result of blood transfusions in Scotland.¹⁵⁵ This analysis appears have taken account of (a) the total number of recipients identified by the Scottish lookback exercise whether dead or alive or declining testing and those who had actually been tested and had been found to be positive (excluding only those who were tested and found to be negative) and (b) the prevalence data for HCV infection amongst Scottish donors. A reduction appears to have been factored in for the likelihood that an infected component was not transfused, though this was based on data available for English transfusion likelihood in the absence of Scottish data. The analysis does not attempt to investigate the possibility that infections were caused by a means other than the infected transfusion which would require a more in depth analysis of the individuals concerned and the possibility of their infections having been caused by other means. As the analysis recognises, many of the infected individuals remain unidentified and so it seems that such an analysis could not have been undertaken at the time when this estimate was prepared.

Whilst pointing out that the work done by Soldan & Ors had its limitations, Professor Goldberg acknowledged that the work done by Soldan was, in his view, very good and that Dr Soldan was probably the person who knew more about this field than anybody else in the UK.¹⁵⁶ He indicated in a letter written in response to the Inquiry that Dr Soldan remained prepared to speak to her figures, had she been called to give evidence to the Inquiry.¹⁵⁷ We therefore respectfully submit that, given the disparity in the figures outlined above, the Inquiry takes that opportunity at a further oral hearing.

¹⁵⁵ Report of the Expert Group on Financial and other Support (March 2003) @ paragraph 4.8 - <http://www.scotland.gov.uk/Resource/Doc/47034/0024918.pdf> (including the material to be found in SGH.005.7203)

¹⁵⁶ Transcript for 16/03/11 (day 6); 132 (22) to 133 (3) (Professor Goldberg)

¹⁵⁷ PEN.019.0896 @ 0897

It should be remembered, however, that the Soldan analysis relates only to the period from January 1980 to the introduction of routine anti-HCV screening in September 1991 and therefore cannot be taken to represent the total number of infections with hepatitis C from blood transfusions in either Scotland or the UK.

Further, we would contend that use of the figures as to HCV prevalence rates in the donor populations of England and Scotland for the few months after the introduction of testing in 1991 is likely to result in a figure which is far less than the real total number of infections based on the fact that the use of this prevalence rate constantly over the period is unlikely to represent the real prevalence rate of HCV infection in the donor population. As the Goldberg analysis attempts to recognise, the likely prevalence of infection in the Scottish donor population in 1991 is likely to have been different from the level it was at earlier in time. In our view, it was less than it would have been previously. Measures about which the Inquiry has heard evidence, such as the introduction of measures to exclude high risk donors for HIV in 1983/84, the cessation of collecting blood from prisoners in the first half of the 1980s and the introduction of anti-HIV testing in October 1985 are likely to have resulted in less HCV positive blood getting into the system year by year over the decade. Experience of these processes will have been likely to have improved over time, hence lowering the rate to the point observed in 1991/92. Therefore, a higher prevalence rate would require to be applied to the flowchart analysis of the Soldan group for the earlier years of the analysis. This would result in the template producing a greater number of likely infections. Further for the period before the group's reference period (ie before 1980) the prevalence would, in our view, have been likely to have been significantly higher than the 1991/92 rate. Also, as is observed above, Professor Thomas has spoken to a far higher prevalence rate in the donor population in the 1970s and 1980s than was used as a starting point by either the Goldberg or the Soldan groups.

Conclusion

In light of this, we would invite the Inquiry to conclude that the figure proposed by Dr Soldan is likely to represent the most accurate figure for infections in this group at which the Inquiry has been able to arrive at this point in time, for the period with which it is concerned from 1980 to 1991. Her figures are not out of line with many of the figures generated by the alternative methods of calculation proposed by Professor James as valid ways of calculating the likely total number of infections by this route in Scotland which range from 1,800 to 7,150.¹⁵⁸ What is clear, in our view, is

¹⁵⁸ PEN.019.0916

that the far lower figures proposed by the Gillon or Goldberg methods result in a total figure which runs the risk of a considerable under-estimation of the numbers infected through this route. We note that Professor Goldberg reported that Dr Soldan had told him that she would still have been prepared to speak to the figures had she been called to give evidence at the Inquiry.¹⁵⁹

Efforts have clearly been made to understand the way in which UK figures produced by the Soldan group and released by the Department of Health for the period including the 1970s as well have been arrived at. In his evidence, Professor Goldberg was asked by Inquiry Counsel about this, given that the figures in the original Soldan analysis (and in the Scottish analysis carried out for the Ross Committee for that matter) had been prepared in order to arrive at a figure for the likely number of infections over the period from 1980 to 1991. It was pointed out to Professor Goldberg that whereas this figure had come to around 13,500, a further analysis for the period from 1970 onwards appears to have added 10,000 further infections for that decade. Inquiry Counsel queries how this additional figure for the decade of the 1970s had been arrived at.¹⁶⁰ This was followed up by the Inquiry but Professor Goldberg was unable to shed any further light on this, despite having met with Dr Soldan.¹⁶¹ It appears to us that this figure may be derived from the figure produced on the final page of the 2002 Soldan & Ors article.¹⁶² There it is suggested that there may have been a further 10,000 extra HCV infected blood recipients in the 1970s if one were to use the prevalence data from the 1991/92 papers applied to the 1970s transfusion data. Of course, the article itself suggests that that analysis has not been done (as we suggest above) precisely because the prevalence data would not be appropriate for that time period. This merits further investigation with Dr Soldan.

Co-infection

As far as co-infection amongst the population of bleeding disorder patients is concerned, the epidemiological evidence applied to the analysis done above suggests that all patients infected with HIV through blood products are likely to have been infected with hepatitis C as well due to the higher prevalence of HCV in the donor population and the exposure of those patients to pooled plasma products. Co-infection is therefore a very real issue for patients infected within the bleeding disorder community.

¹⁵⁹ PEN.019.0896 @ 0897

¹⁶⁰ Transcript for 16/03/11 (day 6); 139 (2) to 140 (1) (Inquiry Counsel)

¹⁶¹ PEN.019.0896 @ 0897

¹⁶² PEN.013.1580 @ 1584

The position with transfusion transmitted infection appears to be somewhat different. The limitations on the comprehensiveness of the data presented by Dr Gillon as regards patients with transfusion transmitted HIV and hepatitis C are discussed above. Within the limited numbers of which Dr Gillon was aware through his researches, he was not aware of any case of transfusion transmitted co-infection.¹⁶³ However, this appeared to be due to the relatively low number of cases of transfusion transmitted HIV which he had been able to identify and he did accept that such co-infection was actually quite likely.¹⁶⁴

17. When, where and how is it likely that the patients in this group were infected with Hepatitis C?

The question of the likely timing of the infection of patients infected by blood transfusion in Scotland is, to an extent, discussed above under reference to the evidence of Dr Gillon and the results of the lookback exercise.

As far as the place of transfusion of the infected blood is concerned, Dr Gillon provided material in respect of 103 infected patients as to the region in which the infective blood transfusion was taken. This question is potentially of interest as if there were a particular imbalance in the prevalence of infective donations being taken in a particular region when compared with another, it might tend to suggest that the practices of donor selection in the region with the higher prevalence region were of questionable effectiveness and quality. 42 were in Greater Glasgow/WBTS, 24 in Lothian, 21 in Tayside, 10 in Aberdeen and 6 in Inverness. The statistical significance of this is limited given the small number of patients for whom this information is available when compared with the full extent of the likely number of patients infected via this route.

18. The accuracy and adequacy of the efforts made by the government in Scotland to identify patients infected with Hepatitis C through blood transfusions in Scotland and the desirability of further such efforts

As we have already outlined above and in our submission on the C5(b) topic, the efforts made to identify patients infected with hepatitis C by a blood transfusion in Scotland via the lookback process have been insufficient to identify many of the patients so infected.¹⁶⁵ The disparity between the

¹⁶³ Transcript for 16/03/11 (day 6); 77 (21) to 78 (3) (Dr Gillon)

¹⁶⁴ Transcript for 16/03/11 (day 6); 78 (14 to 19) (Dr Gillon)

¹⁶⁵ PEN.019.0742

figures spoken to by Dr Gillon and those estimated by Dr Soldan are indicative that there are likely to be large numbers of people who were infected with the disease by a blood transfusion who have not been identified. It seems likely that many of these people will now have died. This is likely simply due to the passage of time but also due to the fact that those who receive blood transfusions are more likely to die within a few years of the transfusion than the average person given that there would be an underlying and potentially lethal medical reason for the blood transfusion being received in the first place. Dr Gillon suggested that studies had shown that it might be that as many as 50% of all blood transfusion recipients would be dead within a few years of receiving the transfusion anyway.¹⁶⁶ The majority of patients identified as having been exposed to a potentially infective blood component had died by the time the lookback exercise was complete even in 1998 (536 out of 880 or 60.9%).¹⁶⁷

We are aware of a current project in Scotland instigated by the hepatitis C trust to try to identify people infected with hepatitis C by blood transfusion in Scotland who are still alive. Such efforts are likely to be of limited success given (a) the funding constraints on a non-governmental initiative of this nature (b) the potentially high numbers involved and (c) the emphasis on the patients having to take the initiative to report possible symptoms. In our view, there are compelling public health reasons for there to be a government led and funded initiative to trace those still alive who have been infected with hepatitis C via this route.

HIV - the progression of the infection to AIDS

19. What proportion of those infected with HIV through blood or blood products in Scotland go on to develop AIDS?

The Inquiry has heard evidence about the fact that hepatitis C may clear spontaneously or that it may respond to treatment (see below). In HIV, the position appears to be that a significant proportion of those infected through exposure to blood or blood products in Scotland have died from AIDS (see below). The disease has progressed to AIDS in the others who have been infected but who are still alive.

¹⁶⁶ Transcript for 16/03/11 (day 6); 29 (5 to 14) (Dr Gillon)

¹⁶⁷ PEN.002.0803 @ 0804

Hepatitis C - the progression of the disease

20. The proportion of those infected with the Hepatitis C virus through blood or blood products in Scotland who clear the disease spontaneously and the proportion who progress to the various stages of the disease (including the effect of co-infection with HIV on these proportions)

In the first instance, we would refer to our submission in the C3A topic and, in particular, on the evidence from Professor Thomas regarding the fact that patients in the bleeding disorder community infected with hepatitis C from exposure to blood products are more likely to progress to the more severe stages of the disease due to multiple exposures.¹⁶⁸

The various stages of progression in hepatitis C were described by Professor Thomas in his evidence as¹⁶⁹:

- The acute phase of the disease¹⁷⁰
- The chronic phase of the disease
- The development to progressive liver disease and the cirrhotic phase of the disease
- The development of hepatocellular cancer (which with hepatitis C will only be possible for those who have reached the cirrhotic stage of the disease¹⁷¹)

In general terms, he stated that 30% of those who are infected with hepatitis C will only advance to the acute stage and will not develop to the chronic stage at the 6 month period. The remaining 70% of those generally infected with the disease progress to the chronic phase.¹⁷²

In the original material presented to the Inquiry by the UKHCDO it was suggested that the clearance rate from hepatitis C would be in the region of 15% of those who have contracted the disease.¹⁷³ In his oral evidence Dr Hay described this initial analysis as conservative, suggesting that "recent discussions with various experts" had suggested to him that, in fact, 25 to 30% of cases would

¹⁶⁸ PEN.019.0657 @ 0677 - 0678

¹⁶⁹ Transcript for 11 October 2011 (day 52) @ page 8, line 19 - page 9, line 2

¹⁷⁰ The acute phase comes to an end 6 months after the identification of the infection - transcript for 11 October 2011 (day 52) @ page 12, lines 19 to 22

¹⁷¹ Transcript for 11 October 2011 (day 52) @ page 13, lines 10 to 11 (Professor Thomas)

¹⁷² Transcript for 11 October 2011 (day 52) @ page 13, lines 3 to 9 (Professor Thomas)

¹⁷³ Transcript for 18/03/2011 (day 8); 40 (22 to 25) (Dr Hay)

remit.¹⁷⁴ The source of these figures was not specified by Dr Hay on either occasion but later he did suggest that this had come from Professor Brian Gazzard who had been part of the Department of Health Committee which had advised the Secretary of State for Health regarding changes to the Skipton fund.¹⁷⁵ To the extent that this analysis can be taken as accurate in light of this, we would argue that these general figures for remission rates cannot be deemed to apply to the population of individuals with bleeding disorders infected with hepatitis C. Such individuals will have been likely to have been exposed to multiple strains of the virus given their repeated exposure to infected blood through their (usually frequent) use of pooled plasma products making the likelihood of clearance very much less than in the general population. Dr Hay himself accepted that it was extremely common for the more severe (and therefore more frequently treated) patients to have been exposed to multiple genotypes and that one genotype would become dominant over the others.¹⁷⁶

Dr Tait spoke to the fact that certain research was done in 2006/2007 under the guidance of Dr Watson in Aberdeen to research the progression of the disease in patients infected with hepatitis C amongst the bleeding disorder community who were receiving treatment in Scotland.¹⁷⁷ The research into these questions was not repeated for the purposes of the Inquiry.¹⁷⁸ A more detailed exercise was undertaken by the UKHCDO for the Inquiry to identify the likely number of patients infected in Scotland but this did not seek to answer questions about progression of the infection in this group. The 2006/2007 research was conducted on less individuals than the number identified for the inquiry as probably having been infected in Scotland. Also, the 2006/07 research did not focus on patients infected in Scotland but on those who were being treated in Scotland, irrespective of the likely place of their infection. Therefore, this 2006/07 research cannot be taken to be anything more than indicative of the likely progression of the disease in the Scottish-infected patients. Dr Tait summarised the conclusions of the 2006/07 research as having found that the progression rates amongst the patients with bleeding disorders who were studied were broadly the same as progression rates in other populations of individuals infected with hepatitis C.¹⁷⁹ We note that the clearance rate which was observed was 17.7% which appears lower than the rates spoken to by Dr Hay in his evidence (see above).¹⁸⁰ It is interesting to note that this cohort only identified 33 co-infected patients (less than half the number actually co-infected in Scotland, as discussed above). As Dr Tait accepted in his evidence, it was likely that virtually all HIV positive patients would have been

¹⁷⁴ Transcript for 18/03/2011 (day 8); 41 (1 to 4) (Dr Hay)

¹⁷⁵ Transcript for 18/03/2011 (day 8); 64 (14 to 18) (Dr Hay)

¹⁷⁶ Transcript for 18/03/2011 (day 8); 62 (3) to 63 (20) (Dr Hay)

¹⁷⁷ PEN.013.0008

¹⁷⁸ Transcript for 30/04/11 (day 14); 126 (21 to 23) (Dr Tait)

¹⁷⁹ Transcript for 30/04/11 (day 14); 91 (9 to 14) (Dr Tait)

¹⁸⁰ PEN.013.0008

hepatitis C positive.¹⁸¹ Professor Thomas pointed out that higher viraemia in co-infected patients would be likely to cause more rapid progression into the worse stages of the hepatitis C. He noted that given that immuno-suppression is a consequence of infection with HIV, there will be likely to be greater levels of the hepatitis C virus present, meaning that co-infection is likely to cause patients to progress more rapidly through the stages of the disease.¹⁸² He pointed out in evidence that it is likely, therefore, to lead to a higher likelihood of severe disease and death than in mono-infected patients.¹⁸³ Therefore, had the 2006/07 assessment included all co-infected patients, the observed clearance rate would have been likely to have been lower than 17.7%. The observed progression rates to the later stages of hepatitis C are also likely to have been greater. We would recommend that further research requires to be done along these lines into the progression of the disease amongst the patients identified as having probably been infected in Scotland in order to ascertain, primarily for public health reasons, whether the progression in this cohort is indeed the same as the progression in patients infected by other routes.

As addressed in our submission on the C3A topic, multiple exposure to the hepatitis C virus has tended to mean that bleeding disorder patients are more likely to be infected with genotype 1 hepatitis C which has had the result of their infection being less susceptible to treatment.¹⁸⁴ This is borne out by the 2006/07 research conducted by the Scottish haemophilia directors who discovered that two thirds of the bleeding disorder patients in Scotland on which they had the relevant data (178 in total) were infected with genotype 1, resulting in them being less likely to respond to treatment.¹⁸⁵

The Health Protection Agency published a report entitled "Hepatitis C in the UK" on 28 July 2012 to correspond with World Hepatitis Day. The report was compiled in conjunction with Health Protection Scotland.¹⁸⁶ The report provides up to date figures to the effect that there are around 216,000 people who are chronically infected with Hepatitis C in the UK, with around 90% of those being infected with either genotype 1 or 3. The report notes that in the UK as a whole both hospital admissions and deaths from HCV-related end stage liver disease (ESLD) and hepatocellular carcinoma (HCC) are continuing to rise in the UK. Hospital admissions have risen from 612 in 1998 to 1,979 in 2010, while deaths have risen from 98 in 1996 to 323 in 2010. An overall increase in

¹⁸¹ Transcript for 30/04/11 (day 14); 132 (7 to 11) (Dr Tait)

¹⁸² Transcript for 11 October 2011 (day 52) @ page 58, lines 9 to 13 (Professor Thomas)

¹⁸³ Transcript for 12 October 2011 (day 53) @ page 17, lines 9 to 15 (Professor Thomas)

¹⁸⁴ PEN.019.0657 @ 0677

¹⁸⁵ PEN.013.0008

¹⁸⁶ http://www.hpa.org.uk/webc/HPAwebFile/HPAweb_C/1317135237219

registrations for liver transplants with a code of post-hepatitis C cirrhosis has been observed from 45 in 1996 to 101 in 2011.¹⁸⁷ Whilst these figures are national and relate to the entire population of those infected with hepatitis C and not just those who contracted the disease through blood or blood products, they demonstrate the increasing severity of the disease as time progresses and the consequent increasing pressure on medical services for those who require treatment.

The report goes on to state that of the estimated 39,000 people living in Scotland with chronic hepatitis C infection, only approximately half are thought to have been diagnosed by 2011.¹⁸⁸ It is interesting to note, in our view, that the section relating to initiatives aimed at increasing awareness of infection and detection appears to focus predominantly on those who may have been infected amongst the drug infecting population. There is no mention in that section (or in the entire report for that matter) of those who may have contracted the disease through blood transfusions in Scotland.¹⁸⁹

In Scotland, of the estimated 18,000 diagnosed individuals living with chronic hepatitis C in 2011, an estimated 26% attended a specialist centre in 2011. Between 2007/08 and 2011/12 more than 4,000 individuals had been initiated on antiviral therapy in Scotland. Among patients (with either genotype 1, 2 or 3) initiated on pegylated interferon and ribavirin across nine clinics in Scotland during 2000-2007, 58% achieved a sustained viral response (SVR); this ranged from 39% in those with genotype 1 infection to 70% among those with genotype 2 or 3 infection.¹⁹⁰ The report points out that despite action plans and work programmes which have been developed, the morbidity and mortality from HCV-related liver disease is still increasing and therefore there is still much work to be done.¹⁹¹

21. The number of people who have contracted Hepatitis C through blood or blood products in Scotland who are still alive and have not yet suffered symptoms but are likely to do so in the future

The difficulties in establishing the total number of individuals infected with hepatitis C through, in particular, blood transfusions is discussed in detail above and the position as regards mortality is considered below.

¹⁸⁷ ibid @ page 7

¹⁸⁸ ibid @ page 9

¹⁸⁹ ibid @ page 8

¹⁹⁰ ibid @ page 10

¹⁹¹ ibid @ page 11

Mortality statistics

22. How many people who contracted HIV through blood or blood products in Scotland have died? and

23. For how many people who contracted HIV through blood or blood products in Scotland who have died did AIDS make a material contribution to their death?

Dr Gillon indicated in his limited report on the 18 individuals of whom he knew who had been infected with HIV by a Scottish blood transfusion that 15 were known to have died as at 31 December 2010 (83.33%).¹⁹² He did not provide any data on the cause of death amongst this group, which he had done in his similar report on those who had died in the group who had been infected with hepatitis C as a result of transfusion. In evidence, he did confirm, however that data available to HPS suggested that many of these will have died of AIDS.¹⁹³

As far as deaths amongst the bleeding disorder community are concerned, our own researches with the Macfarlane trust indicate that of the 67 infected individuals with bleeding disorders in Scotland in respect of whom payments have been made, 46 have died (68.66%). Of the 76 patients with haemophilia who were assumed to have been infected by their receipt of contaminated blood products in Scotland listed in the information held by Health Protection Scotland, 46 were known to have died (60.53%).¹⁹⁴ Information from this source as to the cause of death depended largely on the accuracy of data on death certificates or local clinicians taking the trouble to contact HPS with information that HIV may have contributed to death.

The material presented by the Scottish haemophilia directors would tend to suggest that there have only been 39 deaths in this community in Scotland. This is made up of 19 (out of 21) from the Edinburgh infections,¹⁹⁵ 8 (out of 21) from the Yorkhill infections¹⁹⁶, 10 (out of 12) from the GRI infections¹⁹⁷ and 2 (out of 3) from the Aberdeen infections¹⁹⁸. This comprises 66% of the total of 59

¹⁹² PEN.001.0038 @ 0040

¹⁹³ Transcript for 16/03/11; 66 (3 to 6) (Dr Gillon)

¹⁹⁴ PEN.012.0151

¹⁹⁵ PEN.012.0153 @ 0154

¹⁹⁶ PEN.012.0155

¹⁹⁷ PEN.012.0158

¹⁹⁸ PEN.013.0009

spoken to by the haemophilia directors. However, if one cross references that figure which are available from the UKHCDO relating to infections with hepatitis C and cause of death, it is interesting to note that there appear to have been either 48 AIDS deaths based on the collated figures (table 9)¹⁹⁹ or 58 AIDS deaths if one analyses the UKHCDO material broken down by Scottish centre (table 10).²⁰⁰ This would be suggestive both of a higher number of deaths than the directors had spoken to and also a higher number of deaths from AIDS.

A varied approach appears to have been taken by the haemophilia directors as to whether these deaths were caused by the HIV infection. Information about cause of death appears to have been derived from a combination of the HPS records and the UKHCDO database. As discussed above, the former appears to be derived solely from the death certificate and the latter is not really intended as a death recording system.²⁰¹ Professor Ludlam suggested that it contained very little information at all.²⁰² Both sources are therefore of limited value for this purpose. Professor Ludlam acknowledged that there would have been certain anxieties amongst family members about having HIV or AIDS listed on the death certificate and so this might not now be viewed as the most reliable source of information about the cause of death in these patients.²⁰³

Further, Professor Ludlam suggested that it would be appropriate to sub-categorise deaths as (a) related to HIV/AIDS (b) HIV contributed (c) probably not related to HIV/AIDS or (d) not due to HIV/AIDS.²⁰⁴ In the first place, this approach seems unnecessarily complicated, in particular in light of the fact that, in our submission, the immuno-suppressant qualities of HIV infection will be likely to have made a material contribution to the death in almost all, if not all cases. Further, this approach is not consistent with the total number figures of deaths from AIDS (58) in the UKHCDO tables, as discussed above.

¹⁹⁹ PEN.019.0927 @ 0986

²⁰⁰ PEN.019.0927 @ 0988 - 0991

²⁰¹ Transcript for 30/03/2011 (day 14); 23 (4) and 28 (8 to 11) (Professor Ludlam)

²⁰² Transcript for 30/03/2011 (day 14); 45 (19 to 24) (Professor Ludlam)

²⁰³ Transcript for 30/03/2011 (day 14); 28 (16) to 29 (1) (Professor Ludlam)

²⁰⁴ PEN.012.0153 @ 0154

- 24. How many people who contracted Hepatitis C through blood or blood products in Scotland have died? and**
- 25. For how many people who contracted Hepatitis C through blood or blood products in Scotland who have died did their Hepatitis C infection make a material contribution to their death?**

General

In Scotland, liver-related deaths among people diagnosed with hepatitis C increased from 44 in 1996 to 133 in 2010, at an average annual increase of 8.9%. In recent years (2007-2010), the average annual increase was 6.4%. By linking records in Scotland's National Hepatitis C Diagnoses Database to the national register of deaths, it is possible to determine that only 609 (50%) of the total 1,222 liver-related deaths during 1996-2010 among people diagnosed with hepatitis C, had any mention of hepatitis C on their death certificate. Among the 133 liver-related deaths in 2010, 96 (72%) had liver disease recorded as the underlying cause of death (alcoholic liver disease was the most prevalent underlying cause in 49), and 37 (28%) had liver disease only as a contributing cause of death; 103 (77%) were male, and 73 (55%) were aged less than 50 years. End Stage Liver Disease-related deaths among people diagnosed with hepatitis C in Scotland increased from 16 in 1996 to 50 in 2010 (Figure 9), at an average annual increase of 9.2%. Of the total 532 ESLD-related deaths during 1996-2010 among people diagnosed with hepatitis C, only 300 (56%) had hepatitis C mentioned on the death certificate.²⁰⁵

Bleeding disorder infections

In the material presented to the Inquiry by the UKHCDO, it was assumed that deaths from liver disease amongst those exposed to hepatitis C as a result of blood product use were contributed to by hepatitis C infection.²⁰⁶ In our view, this is a fair assumption to make. Even of those who do have alcohol intake as a likely contributing factor to their death, their underlying hepatitis C infection will have materially contributed to their death on the basis that they will have been considerably less tolerant to alcohol as a result of their infection than they otherwise would have been. Therefore, in

²⁰⁵ [@ page 18](http://www.hpa.org.uk/webc/HPAwebFile/HPAweb_C/1317135237219)

²⁰⁶ Transcript for 18/03/2011 (day 8); 51 (4 to 21) (Dr Hay)

our view, the approach advocated by Dr Hay to the effect that all liver disease deaths amongst the population of those who were infected with hepatitis C should be deemed to have had hepatitis C as a material contributor to the death was correct.

The material presented to the Inquiry by the UKHCDO on cause of death provided a cause of death for only 84 of the individuals in the bleeding disorder community who were thought to have been infected with hepatitis C and have subsequently died (89 are listed with 5 unknowns).²⁰⁷ This is only a fraction of the total number who are dead from this community, estimated to be 193 of the 447 infected in the UKHCDO tables (43.18%).²⁰⁸ Of this number (84), 21 were reported to have died from liver disease or liver related causes (26.19%) although cancer (which may include hepatocellular cancer) accounts for a further 22 (26.19%). In our view, these statistics are generally unreliable on the basis that many of the listed causes of death will have had hepatitis C as a contributory factor. The liver is a complicated and important organ and hepatitis C infection is therefore likely to have a contributory effect on death in some form even if not listed as a cause of death. This applies equally to the analysis of those infected by blood transfusions below.

Blood transfusion infections

As is noted above, Dr Gillon suggested in evidence that studies had shown that perhaps as many as 50% of all blood transfusion recipients would be dead within a few years of receiving the transfusion anyway.²⁰⁹ The majority of patients identified as having been exposed to a potentially infective blood component had died by the time the lookback exercise was complete even in 1998 (536 out of 880 or 60.9%).²¹⁰ In his limited analysis of those identified by the lookback process as definitely having been infected by a blood transfusion and for whom the relevant information was available, just over half (53 out of 103) had died as at January 2011.²¹¹ Hepatitis C was recorded as having materially contributed to the death in 15.09% of cases (8 out of 53).²¹² It is suggested elsewhere that in fact this figure should be 14 out of 53 which represents 26.4%.²¹³ The implications of this are considered further below.

²⁰⁷ PEN.019.0927 @ 0986 - 0987

²⁰⁸ PEN.019.0984

²⁰⁹ Transcript for 16/03/11 (day 6); 29 (5 to 14) (Dr Gillon)

²¹⁰ PEN.002.0803 @ 0804

²¹¹ PEN.001.0043 @ 0047

²¹² PEN.001.0043 @ 0047

²¹³ Transcript for 16/03/11 (day 6); 113 (7 to 9) (Inquiry Counsel)

In his oral evidence, Professor Goldberg suggested that 85 of the 304 individuals who he thought had been infected by blood transfusions in Scotland were dead as at December 2009 (27.96%).²¹⁴ The source of this information was the General Registers of Scotland. 31 of these deaths had liver disease (including alcoholic liver disease) as the primary or secondary cause of death (36.47% of known deaths). For the 351 bleeding disorder patients thought to have been infected in his statistical analysis, Professor Goldberg reports that 78 were dead as at December 2009 (22.22%). 30 of these deaths had liver disease (including alcoholic liver disease) as the primary or secondary cause of death (38.46% of known deaths).²¹⁵ The fact that alcoholic liver disease is listed as a separate category in the statistics is of interest. Hepatitis C infection would, of course, render an individual far more susceptible to suffering from alcoholic liver disease. Further, we would argue that certification of death from this cause may, in some cases at least, be based on an erroneous assumption regarding the cause of the liver disease.

In her report to the Ross Committee published in March 2003, Dr Kate Soldan had estimated that around 800 of the 3,498 individuals infected with hepatitis C as a result of blood transfusions in Scotland were likely to have been alive at that time.²¹⁶ She estimated that 365 of the 500 individuals with bleeding disorders infected with hepatitis C as a result of their exposure to blood products in Scotland would still have been alive in 2003.

The epidemiological methodology document provided to the Inquiry by the team led by Professor Goldberg at HPS was based on the assumption that the survival rate of those who received HCV contaminated blood components did not differ from the survival rate of those who had received non-contaminated components. We submit that this assumption will have had a significant effect on the conclusions of that group as its effect is to deny entirely that hepatitis C infection will increase the likelihood of an individual dying. That assertion is based on a paper by Harris & Ors (2006)²¹⁷ which showed that all-cause mortality in infected blood transfusion recipients and controls at 16 years post transfusion did not differ significantly.²¹⁸ Given that the signs of hepatitis C will often take many years to manifest themselves, such an analysis at 16 years post transfusion cannot be said to be a reliable guide as to the likely increase in mortality from hepatitis C infection. As is noted in the Goldberg methodology analysis "information about later survival of that cohort was not

²¹⁴ PEN.013.0014 and PEN.013.0024

²¹⁵ PEN.001.0206 @ 0208

²¹⁶ Report of the Expert Group on Financial and other Support (March 2003) @ paragraph 4.8 - <http://www.scotland.gov.uk/Resource/Doc/47034/0024918.pdf> (including the material to be found in SGH.005.7203)

²¹⁷ LIT.001.3898

²¹⁸ PEN.019.0899 @ 0901

available".²¹⁹ Further, the limitations of this analysis include the reliability of the data as to the cause of death which (as is observed elsewhere on this submission) is unreliable when it comes to the recording of deaths caused by hepatitis C.

26. The accuracy of the reporting of Hepatitis C/HIV as a cause of death amongst those infected with Hepatitis C and/or HIV through blood or blood products in Scotland

The Inquiry has heard evidence relating to the inadequacies of the recording of deaths from hepatitis C and HIV as having been caused or indeed contributed to by those diseases.²²⁰ In his analysis of the available mortality data provided by Dr Gillon, only 15.09% or 26.4% were reported as having had hepatitis C infection contribute to their death (see above). Given that all of the patients under consideration were definitely infected with hepatitis C, Dr Gillon considered it surprising that this figure was so low.²²¹ Despite the limited number of cases analysed, this certainly gives an indication that there is a distinct under-reporting of hepatitis C as a cause of death amongst those infected by blood or blood products in Scotland. Professor Goldberg was dismissive of the reliability of death certificates as a means of identifying hepatitis C as a cause of death.²²² He described the number of reported deaths from hepatitis C in Scotland as a "gross underestimate".²²³

Statistics relating to the use of blood products in Scotland

27. How have the amounts of the different types of blood products used in the treatment of people with bleeding disorders in Scotland varied over the Inquiry's reference period across the country?

The Inquiry has access to statistical material provided by the UKHCDO relating to the amount of products used by patients with bleeding disorders in the various haemophilia centres in Scotland. The tables which were included in the preliminary report were prepared by Dr Charles Hay,

²¹⁹ PEN.019.0899 @ 0901

²²⁰ Evidence was heard from two professional witnesses in connection with the death of Mrs O'Hara that hepatitis C was not included on her death certificate but it should have been - see transcript for 10/03/11 (day 19); 78 (14) to 79 (11) and 85 (10 to 19) (Dr Mutimer) and 130 (5 to 8) (Dr Dunn)

²²¹ Transcript for 16/03/11 (day 6); 55 (4 to 6) and 56 (21 to 22) (Dr Gillon)

²²² Transcript for 16/03/11 (day 6); 113 (13 to 19) (Professor Goldberg)

²²³ Transcript for 16/03/11 (day 6); 114 (7) (Professor Goldberg)

chairman of the UKHCDO.²²⁴ A fresh report altering the contents of the tables was produced by the UKHCDO in April 2012.²²⁵ In our submission, these figures provide a useful general starting point for a proper analysis of the policies and ethos behind product selection and use in Scotland at a time before factor concentrates became heat treated so as to inactivate HIV and subsequently hepatitis C. Although the precision of the UKHCDO material might be questionable and hence of only limited use for other purposes (such as the identification of the most likely time and method of infection on the case of a particular individual), these figures show certain general trends which, in our view, are worthy of note. In particular, we would make the following observations:

The divergence in practice as regards product use amongst the various haemophilia centres in Scotland is nothing short of astonishing. The risks of viral transmission from imported commercial factor concentrates were well known, at least in the second half of the 1970s (which can be seen from the material discussed in the 1975 World in Action DVD considered by the Inquiry). Against such a background, it is hard to comprehend that such divergent practices could have developed as between Yorkhill and Edinburgh as regards the use of commercial concentrates.

If you were treated in Aberdeen, you would have been most unlikely to receive commercial products at all. Between 1969 and 1991, commercial products were only used in 1978, 1979 and 1998 and then in relatively small quantities.²²⁶ It would have been very unlikely that any HIV transmissions from commercial products could have occurred there as these products were not used at all in Aberdeen in the early part of the 1980s. One can deduce from this that despite the apparently limited amount of domestic factor VIII concentrate available to Aberdeen until 1982 onwards (more than 3 times as much PFC factor VIII was used in 1983 than had been used in 1980), those responsible for product selection there did not start to rely on commercial producers, instead making up the requirements of patients with the safer but less convenient cryoprecipitate. The position in Dundee shows a similar pattern of usage with no commercial product showing in its records at all until 1988 (Alpha's Profilate) and then in relatively small quantities.²²⁷ Cryoprecipitate also played a prominent part in therapy there until 1982, when PFC factor VIII concentrate appears to have become available in greater quantities. A similar picture emerges from an analysis of the figures from the Inverness centre where commercial factor VIII was only used in 1974.²²⁸

²²⁴ Transcript for 18/03/11 (day 8); 8 (20 to 24) (Dr Hay)

²²⁵ PEN.019.0927

²²⁶ PEN.019.0927 @ 0935 - 0937

²²⁷ PEN.019.0927 @ 0938 - 0940

²²⁸ PEN.019.0927 @ 0954 - 0955

On the other hand, at Yorkhill, commercial concentrates were used in enormous quantities.²²⁹ Between 1977 and 1979 inclusive, the proportion of the factor VIII concentrate used which was of commercial origin was 65.1%. In 1980, it was 80.9%, in 1981 58%, in 1982 48.5%, in 1983 3.18% and in 1984 0.5%. During the period when many of the HIV infections occurred at Yorkhill, there was a disproportionate reliance on commercial factor VIII concentrate. Very little cryoprecipitate was used over that period. It is also interesting to note that whereas the small usage of commercial product in Aberdeen came from different commercial sources (Travenol/Hyland and Alpha), the sole factor VIII product in use at Yorkhill between 1980 and 1984 inclusive (when its use ceased) was Armour's Factorate. The figures for the Glasgow Royal infirmary also show a considerable reliance on commercial factor VIII in the late 1970s and early 1980s, though from a wide range of sources and in considerably smaller proportions of the total quantities of factor VIII concentrate used than at Yorkhill. We would refer to the submissions we have made in the B2 section regarding the startling lack of co-ordination between the approach adopted in certain parts of the country where (a) a reliance on domestic concentrates was considered to be the safer option for patients and (b) cryoprecipitate was used where supply could not keep up with demand and other areas where a completely different approach based at times almost entirely on commercial concentrates was preferred.²³⁰

The product usage in Edinburgh is also interesting.²³¹ No commercial products were used at all during the 1970s and there would appear to have been a heavy reliance on cryoprecipitate in order to deal with the fluctuating availability of domestic factor VIII (more than twice as much PFC factor VIII seems to have been used in 1976 than in 1979, for example). Therefore, the shortfall in domestic factor VIII concentrate appears not to have resulted in resorting to use of commercial products. We are aware that there was a change of directors at the Edinburgh centre in 1980, Dr Ludlam having been appointed to replace Dr Davies. This would seem to have provoked a change in approach in two respects. In the first place, far greater amounts of therapeutic material were used than had been used in the past. Over eight times as much PFC factor VIII was used in 1980 than had been used in 1979 (despite the use for the first time of some commercial factor VIII). Around twice as much cryoprecipitate was used. At the other centres, no such rise was evident. In the same two years the amount of factor VIII used in Aberdeen decreased.²³² In Dundee the usage increased by only 29%.²³³

²²⁹ PEN.019.0927 @ 0951 - 0953

²³⁰ PEN.019.0476

²³¹ PEN.019.0927 @ 0941 - 0945

²³² PEN.019.0927 @ 0935

²³³ PEN.019.0927 @ 0938 - 39

At the Glasgow Royal Infirmary usage of factor VIII rose by 15.5%.²³⁴ At Yorkhill increased usage was around 72%.²³⁵ In Inverness, the amount of factor VII used increased by around 79%.²³⁶

This increased factor VIII usage in Edinburgh when compared with the usage levels in the 1970s continued throughout the first half of the 1980s. The average annual usage of all factor VII between 1976 and 1979 was 317,349 units. The average annual usage between 1980 and 1985 was 1,874,178 (almost 6 times as much). A similar analysis of the average annual amounts of factor VIII concentrate over these years used in the other centres shows increases in the following proportions - Aberdeen (2.39 times), Dundee (3.08 times), GRI (1.77 times), Yorkhill (3.96 times comparing the periods from 1977 to 1979 and 1980 to 1985 inclusive for which records are available) and Inverness (1.69 times). The usage of factor VIII concentrate in Edinburgh therefore rose out of proportion with the other centres by some considerable margin over this period. We note with interest that the centres with the greatest increased usage of concentrates in the first half of the 1980s account for large majority of HIV infections in Scotland. The vastly inconsistent product usage suggests that product selection policies were adopted and treatment programmes instituted by centre directors on a general basis and without regard for the needs of individual patients. This process was allowed to go on without control by or official guidance from government or the NHS, despite the fact that certain of the programmes (in particular those relying on large amounts of commercial products) must have been hugely expensive.

This increased demand in Edinburgh was happening against a background of considerable strain on the PFC to meet the requirements. In 1975, 163 patients were registered to receive treatment in Edinburgh (before correction for double counting).²³⁷ In 1980 there were 203.²³⁸ In 1985 there were 265.²³⁹ The number of patients being treated there had therefore increased by only 1.63 times. We would refer to our submission in the B2 section and suggest that this increased usage was linked to the implementation of home treatment and prophylactic treatment regimes which resulted in patients being exposed to greater quantities of products (in particular factor concentrates) and greater strain being placed on the achievement of national self sufficiency.²⁴⁰

The second phenomenon which one can see in Edinburgh is an unprecedented reliance on

²³⁴ PEN.019.0927 @ 0947

²³⁵ PEN.019.0927 @ 0951

²³⁶ PEN.019.0927 @ 0954

²³⁷ PEN.019.0927 @ 0956

²³⁸ PEN.019.0927 @ 0957

²³⁹ PEN.019.0927 @ 0957

²⁴⁰ PEN.019.0476

commercial concentrate to meet demand where it could not be met out of domestic supplies. As noted above, before 1980, commercial concentrate had not been used in Edinburgh at all. Cryoprecipitate had been relied upon when there was not enough domestically produced factor VIII concentrate. In 1980, Factorate comprised 9.07% of the total usage of factor VIII concentrate in Edinburgh. In 1981, the only year in the first half of the 1980s (other than 1985) when the amount of available domestic factor VIII dropped, commercial factor VII usage rose to 34.37% of the total. In that year the amount of cryoprecipitate fell by almost half.

On the question of the processes involved in selection, procurement and distribution of products for the treatment of patients with bleeding disorders in Scotland in the first half of the 1980s, we would refer to the submission which we have presented to the Inquiry in connection with the B2 topic, in particular in response to issues 13 and 15 on our list²⁴¹ and in connection with the C3A topic, in particular in response to issue 14 on our list.²⁴²

General - the maintenance of public statistical information

28. The relative responsibilities of different bodies in Scotland for the compilation and maintenance of statistical information relating to those infected with HIV and/or Hepatitis C as a result of treatment with blood or blood products in Scotland and its accuracy

Statistical information about infection caused by blood or blood products in Scotland came from the UKHCDO, HPS and the SNBTS.

Much of the information to which the Inquiry had access relating to patients with bleeding disorders has come from the UKHCDO. In his evidence, Professor Ludlam stated that he could not guarantee that the UKHCDO records of the treatment histories of patients were complete.²⁴³

Until recent legislative changes in 2008, the notifiable disease legislation required the reporting of viral hepatitis. HIV has never been a notifiable disease. In his evidence to the Inquiry, Professor Goldberg confirmed that this system resulted in clinicians rarely reporting possible infections with

²⁴¹ PEN.019.0476 from 0509 and 0511 respectively

²⁴² PEN.019.0657 from 0701

²⁴³ Transcript for 04/05/11 (day 19); 58 (4 to 5) (Professor Ludlam)

these diseases to health boards meaning that they were rarely reported to HPS.²⁴⁴ It is noteworthy, in our view, that Professor Goldberg sought to explain that the system of gathering and retaining information about hepatitis C in Scotland was better in Scotland than elsewhere.²⁴⁵ That does not, in our view, lead to the conclusion that the system is a good one. The apparent (a) low likelihood of incidences of hepatitis C or HIV being reported to HPS and (b) lack of investigation of the accuracy of the information reported to it are addressed above.

The evidence given by Dr Gillon about the lack of any requirement (legal or administrative) to report cases of possible transfusion transmitted infection to SNBTS is addressed above. The HPA appears to have maintained a national HCV register for research purpose since 1998. There was no requirement for SNBTS to report cases to them and the two systems appear to have been operating separately.²⁴⁶

The focus of haemophilia clinicians is rightly on treating the patients with bleeding disorders. They have no responsibility for the larger cohort of patients infected by blood transfusion. The focus of Health Protection Scotland in the area of hepatitis C (and indeed HIV) is likely to be on infections caused by other means (such as injecting drug use) which account for the vast majority of such infections in Scotland. In his evidence, Professor Goldberg accepted that the main focus of HPS in this area is on prevention of future infections and so blood transfusion does not play a large part in his organisation's consideration of these diseases.²⁴⁷ There is a need for there to be a greater focus in the identification and treatment of those infected with HIV and/or hepatitis C as a result of their exposure to blood or blood products in Scotland. This, in our submission, stems from the fact that this group have been infected by NHS treatment and the fact that the circumstances of their infection may result in them having distinct care, support and treatment requirements from those infected by other means.

²⁴⁴ Transcript for 16/03/11 (day 6); 103 (11 to 22) (Professor Goldberg)

²⁴⁵ Transcript for 16/03/11 (day 6); 110 (10) to 111 (2) (Professor Goldberg)

²⁴⁶ PEN.013.1557 @ 1559, para 5

²⁴⁷ Transcript for 16/03/11 (day 6); 111 (18) to 112 (14) (Professor Goldberg)

Recommendations for the future

29. What recommendations for the future should the Inquiry make arising out of the material considered in this topic?

In our submission, it is of the utmost importance that the Scottish government and the National Health Service in Scotland have as accurate an estimate of the number of people who were infected with HIV and/or hepatitis C in Scotland as possible. It is important that the scale of the blood contamination disaster in Scotland be comprehended as the basis for planning to cater adequately for the continuing needs of victims, both identified and unidentified, in Scotland.

Further, in terms of its fourth term of reference, the Inquiry has been charged with the investigation of the systems for recording and monitoring the numbers of NHS patients in Scotland treated with blood and blood products, with particular reference to the numbers exposed to risk of infection with the hepatitis C virus and HIV and the numbers contracting either or both such infections as a consequence of such treatment. Therefore, the Inquiry is responsible not only for achieving an estimate of the numbers affected by the blood contamination disaster but for assessing the quality of the systems in place in Scotland for achieving an understanding of the scale of likely infection with infectious diseases such as HIV and hepatitis C. In our submission, the difficulties which the Inquiry has experienced in ascertaining the likely scale and severity of infection of individuals with these diseases is indicative of an ineffective system of disease detection and hence of disease prevention and control.

In this regard, we would recommend the following:

- 1) The Inquiry processes have revealed the difficulties in assessing the numbers infected, particularly those infected with hepatitis C as a result of blood transfusions. The disparity between the numbers who have been identified as actually infected as a result of the lookback procedure and the numbers who are estimated to have become infected as a result of blood transfusions is stark. It is therefore imperative for the purposes of strategic planning from a public health perspective, in our view, that a full NHS-funded project be undertaken to offer all patients who have received blood transfusions in Scotland prior to September 1991 a blood test for hepatitis C infection. This initiative

should be medically led with tests being offered to all those who have received a blood transfusion prior to September 1991. It should also be advertised to the general public on the basis that certain at risk individuals may not have their blood transfusion history recorded in their medical records. Such an offer of a test to investigate the possibility of infection by blood transfusion may also have the effect of encouraging individuals to come forward for testing more generally, given that infection by blood transfusion is likely to be perceived as a more socially acceptable way of having become infected.

The figures which have been released in the recent Health Protection Agency paper (referred to above) would suggest that around half of those who are chronically infected with hepatitis C have not been diagnosed. Given the current emphasis shown in that paper on identifying those who may have become infected through intravenous drug use, we consider that greater public awareness of the risks of having contracted the disease from a blood transfusion is also necessary. In our view, this is imperative in order to give those who have been infected by this route and who may not suspect infection a chance of successful treatment.

- 2) Further, we submit that the Inquiry must recommend that funding be provided to analyse historic blood donor samples in order to identify more accurately individuals who have been infected with HIV or hepatitis C by means of receiving a blood transfusion.
- 3) The Scottish government and the NHS in Scotland are responsible for the development and implementation of strategies for the management of public health in Scotland. In order to assist with this role, we would recommend that it is imperative that there be properly maintained registers in Scotland containing information essential to the running of the NHS in Scotland. In this we would include the need for a well run, independent Scottish Haemophilia Register. The information contained in it must still be input into the national UK-wide database to which access must still be available for Scottish clinicians to enable comparative analysis to be undertaken. However, we feel that if the Scottish data is kept and managed separately it is likely that a greater amount of relevant information on bleeding disorder patients will be able to be stored and that the information will be able to be managed more successfully than appears to be the

position at present where the Scottish data is simply handed over and assimilated into the larger UK-wide system.

- 4) As far as the recording of statistical information relating to transfusion transmitted infection is concerned, we would suggest that it be made a legal requirement that such possible cases be reported to a single agency responsible for the maintenance of an official register of infections with information about the likely source of infection and the progression of the disease in the infected individuals to which HPS and the SNBTS should have access for the fulfilment of their own responsibilities with regard to transfusion transmitted infection.
- 5) The information presented to the Inquiry by the UKHCDO regarding the likely number of infections with hepatitis C amongst the bleeding disorder community in Scotland was based on epidemiological assumptions about the likely infections as well as the timing and place of those infections. It is essential, in our view, that all those within the bleeding disorder community (whether registered with a haemophilia centre or not) should be offered an HCV test in order to confirm the precise extent of hepatitis C infection rate amongst this population in Scotland.
- 6) The evidence given to the inquiry by members of the UKHCDO suggested that the compilation of full and accurate data regarding infections (including deaths from such infections) (a) was subject to an ongoing Hepatitis C lookback exercise within that organisation and (b) was not complete, to the extent that "filling in [the] gaps would require a considerable amount of work and would require review of historical medical records".²⁴⁸ In our view, this is an important exercise which requires the allocation of extra resources to be completed.
- 7) Within this topic (and elsewhere) the Inquiry has heard assumptions which have been made about the progress of hepatitis C amongst patients infected by blood or blood products in Scotland. Assumptions, such as regarding the number of infected patients who will clear the infection spontaneously, appear to be applied universally without any consideration being given to the method of infection. We consider that it would be advantageous from a public health perspective for government funded research to be

²⁴⁸ PEN.013.0016 @ 0017, para 8

undertaken into the question of whether these assumptions are validly applied to multiply exposed bleeding disorder patients for whom the progression of the disease (given this infection route and the likelihood of patients having been multiply exposed to the virus) may well be found to be different to those infected via other routes. The validity of assumptions applied to co-infected patients from the bleeding disorder community equally requires further study.

- 8) Given the apparent unreliability of the reporting of deaths from HIV and/or hepatitis C, we would recommend that government-funded research be undertaken to arrive at a more accurate picture of the number of deaths amongst the populations infected with these viruses by blood or blood products in Scotland which are connected to the infections. Particular research into the numbers who have contracted cancer in these populations is also necessary.

JTD