Dear Tracey

Re: Professor Goldberg – further requests regarding statistics: 14th February 2012.

The following is Professor Goldberg’s response to the five questions posed by Janet Marsh in her letter, dated 14th February 2012:

Question 1
How were the figures obtained in the table which shows the range from 1183 to 1978 estimated to be infected with HCV? It would be really helpful to us, and the other parties, to have an explanation in layman’s “terms”.

Response to question 1
Appended is a detailed account of the methods used to generate the estimates as above.

Question 2
Can Professor Goldberg confirm the weighting, if any, given to the fact that prisoners were donating blood in Scotland from 1970 to early 1983 in the calculations?

Response to question 2
The model used to generate the estimates of the number of individuals in Scotland who became infected with hepatitis C via blood transfusion between 1970 and 1991 incorporates the factor which accounts for the deferral policy introduced by SNBTS in 1984. This deferral policy involved making potential donors aware that if they belong to “high risk” groups, they should defer from donating their blood. This policy was assumed to have reduced the HCV prevalence in the donor population constantly by 66% during 1984 to 1991 – an assumption based on SNBTS expert opinion. Accordingly, prisoners donating blood during 1970 to early 1983 may have included individuals at higher risk of infection; but such individuals would not have been deferred from donating their blood as a consequence of their risk status.

Question 3
During his oral evidence on 16th March 2011 (page 140) Professor Goldberg was asked if, when he met Dr Soldan (which he said he was going to do the following week), he could ask her about the calculations underpinning the DoH review figures. Did he ask her, and what was her response?
Response to question 3
Professor Goldberg did meet Dr Soldan and did ask her about the figures. She indicated to Professor Goldberg that she had undertaken the work a long time ago and had difficulty remembering much about it. Professor Goldberg did not think it his place to take matters further in this respect. Dr Soldan had prepared some estimates for Scotland in 2002 and Professor Goldberg’s understanding from speaking to her was that she would have been prepared to speak to these estimates as a witness for the Inquiry.

Question 4
We note that the work Dr Soldan undertook for Dr McClelland produced an estimate of 3498 people infected with hepatitis C from blood transfusions in Scotland between 1980 and 31st August 1991. The work Professor Goldberg carried out for us last year produced estimates of those infected between 1970 and 1991, with a mid-estimate figure of 1532. We know that Dr Soldan’s time period is a decade shorter than Professor Goldberg’s: can he provide a detailed, reasoned explanation for this? It would be helpful to have as clear an explanation as possible, as we hope to understand it, and intend to make available to other parties as well.

Response to question 4
It should be understood that the model used by Schnier and Goldberg in 2011 for the Penrose inquiry was very different to that used by for Dr McClelland in 2002. Further, the former model was built following considerable consultation with Dr McClelland and Dr Gillon of SNBTS. The principal differences between the models is that the Soldan one used “lookback data” to inform it’s estimates and did not factor in any variation in HCV antibody prevalence among blood donors during 1980 – 31st August 1991. The Schnier and Goldberg model did not use lookback data to inform its estimates but did use estimates of HCV antibody prevalence among blood donors for each year during 1970 – 1991. It is the estimated HCV prevalence in the donor population which, effectively, drives the risk of a recipient of a donation becoming infected. The greater the chance of a donor being infected the greater the chance of a recipient acquiring the infection. As indicated in the paper submitted to the Penrose inquiry on the 16th September 2011, and again in more detail in the paper appended (see question 1 above), HPS was in the position to estimate the prevalence of HCV antibody among blood donors during 1970 – 1990 because i) it had generated estimates of the incidence of HCV infection among IDUs for work published in 2005 by Hutchinson et al, and ii) it was evident from a variety of sources that approximately 90% of HCV infected individuals in Scotland had acquired their infection directly through injecting drug use and that an appreciable proportion of the remainder had acquired HCV indirectly as a consequence of injecting drug use (e.g. being born to an infected injecting drug user or having unprotected sex with an infected injecting drug user). Schnier and Goldberg, in consultation with McClelland and Gillon, made the assumption that the size of the HCV infected IDU population in Scotland was directly proportional to the HCV infected donor population. Anchoring the HCV antibody prevalence among blood donors using 1991 observed data and having estimates of the size of the HCV infected IDU population permitted the calculation of the estimates of the HCV antibody prevalence among donors for each year during the 1970 – 1990 period.

In addition to the above, Schnier and Goldberg used a factor to account for the introduction of blood donor deferral in 1984 – a factor not used by Soldan et al.

Two other factors used by Soldan were modified for Scottish purposes; instead of the number of units generated from one blood donation being 1.6 (Soldan), the Scottish estimate, based on local data and expert opinion, was deemed to be 1.25; the proportion of units transfused was estimated to be 56% and not 66% (Soldan).

In conclusion, incorporating the deferral factor leads to an increase in Schnier and Goldberg’s estimates, compared to those generated by Soldan: in contrast, the use of i) the decline in HCV antibody prevalence among donors going back from 1991 to 1970, ii) a 1.25 is as opposed to a 1.6 factor for units generated from one blood donation and iii) a 56% as opposed to a 66% rate of units transfused, would all lead to lower estimates than those generated by Soldan et al. As indicated above, the main driver of the size of the HCV infected blood transfusion population is the HCV antibody prevalence among donors and so the fact that Soldan applied the same
It is appreciated that the Soldan estimates apply to a shorter period than those for Schnier and Goldberg but it should be acknowledged that the number of transmissions occurring in the 1970s would be much less than the numbers occurring during the 1980s as HCV transmission in Scotland only really began to take off in the late 1970s/1980s.

In the opinion of Professor Goldberg, the Schnier and Goldberg estimates, in view of the above, are more robust than those provided by Soldan in 2002.

Question 5
Council also have a query specifically referring to the Health Protection Scotland website. With regard to the figures for infection with the hepatitis C virus to 30th September 2011, the website states that there have been 30934 diagnoses in Scotland. Of these, 361 have “blood factor” as their risk factor. There is a further column totalling 1662 for “other” risk factors, such as transfusion, sexual contact, tattoo/piercing etc. We would be grateful if Professor Goldberg could provide the Inquiry with a breakdown figure for the number infected through transfusion alone.

Response to question 5
The number possibly infected through transfusion alone is 344. Please refer to the paper submitted by Professor Goldberg to the Penrose inquiry in March 2011 regarding “the processes involved in collecting, and the composition of data on, HIV and hepatitis C held at HPS and relevant to the Penrose Inquiry”. Particular reference should be made to the section on hepatitis C, bullet point four which states “in contrast to HIV, in instances where information indicating “blood transfusion” had been recorded on request forms accompanying a sample for HCV testing, HPS has not sought information to confirm acquisition of infection through this route because of the relatively large numbers involved (though these represent only about 3% of all hepatitis C diagnoses) in the context of a surveillance initiative which is slightly less robust than that of for HIV and one which is not about patient identification but about epidemiology for public health action. The lack of confirmation associated with “blood transfusion” is a weakness in the system; however, it should be noted that Scotland’s hepatitis C diagnoses database is more comprehensive and accurate than that existing for any other country including those elsewhere in the UK and the European Union. Individuals for whom the risk of “blood transfusion” applies, are categorised as such (unless they are other “more likely” risks – namely haemophilia, injecting drug use); accordingly, such individuals, unlike those who have received blood factor or who have injected drugs, are considered by HPS to have possibly (as opposed to likely) acquired their infection through this route (i.e. blood transfusion).”

I hope the responses above satisfies the Penrose Inquiry. With respect to the paper appended and that submitted on September 16th 2011, it should be appreciated that the considerable amount of work undertaken by HPS, in collaboration with SNBTS colleagues, has not been subjected to rigorous quality assurance (peer review) because of time constraints. Please also note that Professor Goldberg indicated, in his September 16th paper, that if the Inquiry required a more detailed account of the process involved in generating the estimates this would be made available. Further, Professor Goldberg is, and has been, extremely willing to discuss the findings in person with the Inquiry team.

Professor David Goldberg 28th February 2012