Scottish Hospitals Inquiry

Witness Statement of

Professor Michael Stevens

Personal Details

1. My name is Professor Michael Stevens. I am retired but continue to hold the role of Emeritus Professor of Paediatric Oncology at the University of Bristol. My last clinical role prior to retirement was as a Consultant Paediatric Oncologist at the University Hospitals Bristol NHS Foundation Trust and the Bristol Royal Hospital for Children.

2. I have prepared this statement on the basis that reader has read the Case Note Review Overview Report, March 2021 ("the Overview Report") **[A33448007 – Case Note Review Overview Report (March 2021) - Bundle 6, Page 975]** and will refer sections with that report within this statement. I am one of the authors of the Overview Report and will adopt it as part of my evidence to the Inquiry.

3. Once we had completed our draft report NHS GGC provided us with a lengthy document as a response to our final draft. We took this very seriously and we complied a document titled, "*Case Note Review Team Rebuttal of GGC Consultation Response*" **[A43237045 – CNR Team Rebuttal of NHSGGC Consultation Response - Bundle 25, Page 157]**. We took each of their points in turn and where necessary queried or challenged what they said. We did make some changes to our draft document based on the response to some of the things they said but we did not change the overall conclusions, it was simply changes in order to clarify matters. This document was not shared with anyone apart from our team.

Professional Background

4. I completed my undergraduate degree in medicine at the University of London in 1974 and graduated with an MB BS (with distinction). I went on to obtain the MRCP (UK) in 1976 and accreditation for training in Paediatrics in 1982 and in .

Paediatric Oncology in 1986. I was appointed FRCP by the Royal College of Physicians, London in 1992. In 1997 I was appointed a Fellow of the Royal College of Paediatrics and Child Health and awarded Fellowship of the Royal College of Radiologists in 2003.

5. Paediatric Oncology is broadly divided into professionals who look after children with blood diseases including leukaemia and those that look after children with what are understood colloquially as solid tumours. I had a special expertise and interest in the management of children with soft tissue sarcoma.

6. Despite the majority of my career focusing on children with solid tumours I gained comprehensive clinical experience across the board and have experience of looking after children across the breath of children's cancers across my clinical practice. When I first became a consultant in the UK, I undertook the care of children with leukaemia for several years but as my degree of specialism grew, I stopped taking primary responsibility for leukaemia. It should be noted that when undertaking weekend or night work or when on call for ward supervision I was responsible for all patients, so I was involved consistently with patients with leukaemia despite not being primarily responsible for their treatment.

7. I have been involved in a number of significant reviews which are detailed in my professional CV which is appended to this statement. I have also undertaken a number of reviews about patient management for the NHS, both within my own hospital and others. I have experience of the detailed investigation of individual patient management as well as far larger scale reviews of service delivery policy and the practical implementation of health service strategy both in the UK and abroad.

Appointment to the Case Notes Review Expert Panel

8. On 11th December 2019 I received a telephone call from the Chief Medical Officer for Scotland, Catherine Calderwood. She explained, in brief, the emerging situation at NHS Greater Glasgow & Clyde Health Board (NHS GGC) and the recent establishment of an Oversight Board by the Director-General of Health and Social Care consequential on the escalation of the health board to Level 4

of the NHS Scotland Performance Management Framework. She explained that one of the work streams within the role of the Oversight Board was likely to be a need for a review of cases. No more specific information was provided at this point. She stated that she had received a recommendation that I would be a suitable person to undertake that review and that they wished someone from outside Scotland to undertake the role. I said at that stage that potentially I would be interested but there was clearly more to discuss.

9. Around a week after my initial conversation with Catherine Calderwood I was invited to a telephone conference involving Fiona McQueen, who was the Chief Nursing officer at the time and the chair of the Oversight Board, together with Philip Raines who was a civil servant in the Chief Nurse's office. Professor Marion Bain was also in attendance and her role was to have executive oversight of the Case Note Review together with Catherine Calderwood. They provided me with more detail of what the Case Note Review was hoped to achieve, I was asked questions and asked to submit a CV. I was asked to ensure that I was available for meetings in mid-January 2020 and subsequently travelled to Edinburgh for a succession of meetings.

10. The last meeting was with Fiona McQueen and Jeane Freeman who was Cabinet Secretary for Health at the time when I was offered the role. This was the only time that I met Ms Freeman. I did have a few meetings with Fiona McQueen, but she left her role before the end of this process and a new Chief Nursing Officer was appointed. We had very little direct contact with anyone other than Marion Bain and Phil Raines. We were aware that others were being kept up to date with the progress of our work.

11. I had been provided the names of Professor Mark Wilcox and Gaynor Evans and told that the three of us would be working together. I did not know either of them prior to meeting them on 24th February 2020, and I was not involved in either of their appointments. I was relieved that people had been identified with their skills because it was apparent to me from the outset that this review could not be conducted by a clinician alone and that input was necessary from microbiology and infection control expertise. I was not asked for advice or suggestions on recruitment, and I would not have been able to give any on the roles that Mark

and Gaynor took up.

Composition of the CNR Team

12. There were four component parts to the CNR Team. First, there was the Expert Panel which comprised of myself, Mark and Gaynor.

13. The second team was led by Pat O'Connor and included Professor Peter Davey from the University of Dundee. We initially referred to them as the "ground team". They would extract information from all the clinical records for us and provide information about patients who had infections. Pat O'Connor and Professor Peter Davey were absolutely central to the search for relevant clinical information and passing it on to us.

14. Pat O'Connor and Peter Davey were initially appointed to implement work on the Paediatric Trigger Tool ("PTT") referred to in Figure 3.2: Case Note Review Process Map and elsewhere in the report. The term 'ground team' was used only at the very beginning of our work and related to their responsibility for accessing clinical records 'on the ground'. We subsequently referred to them as the PTT team. When first considering how our work would be carried out (prior to the constraints imposed by the Covid pandemic), I had assumed that I would have played a greater role in evaluating data from clinical records at source by visiting Glasgow. In the event, this was not possible and we therefore relied on the PTT team (so called as they were responsible for this component of the Case Note Review) to collect the data we needed from clinical records. The work of the PTT (and of the HPS) team is further described in 3.6 and, more specifically, in 3.6.2.

15. The third team were from Health Protection Scotland (HPS) who subsequently changed their name to ARHAI. This mainly comprised of Fiona Murdoch, Lead Healthcare Scientist, and Jane McNish, a nurse epidemiologist. Their role was the management of other aspects of clinical information.

16. Where the Overview Report describes the work of the Clinical Team at 2.6.2 it is referring to both the ground/PTT team and the team from HPS. Pat O'Connor's team extracted clinical information from case notes and patient records and then

Fiona Murdoch's team collated and presented to us the microbiological data relating to the patients in the cohort. The HPS team also took on other tasks such as generating information about where in the hospital the patients had been nursed so we could ascertain which ward patients were in at the time of their infections or immediately prior thereto. The HPS team were also responsible for helping us manage the information received from NHS GGC about environmental sampling..

17. The fourth team was the secretariat or support team as described at 2.6.3 of the Overview Report. This was made up of Marie Brown and Emma McKay. They organised our meetings, managed our workplan, documents and records, and undertook other administrative tasks.

18. The core project team consisted of all members of the Case Note Review teams with Professor Bain and other members of the Oversight Board. It met about once a month and there would usually be around 8 or 9 individuals in attendance. It was from these meetings that information was passed back to the Oversight Board. During these meetings we would discuss supplementary verbal and written reports and provide feedback. We did not keep formal minutes, but an action log was kept where we noted outcomes or progress reports.

19. The Case Note Review was one of the various components of the work of the Oversight Board. Professor Marion Bain, in addition to chairing our core project team meetings, also held responsibility for the oversight of the governance aspect of the ongoing management of infection in NHS GGC. Professor Bain was our direct line of report. She did in the early days have a lot of dealings with NHS GGC as she used to go and work in the organisation regularly. This meant she was in a good position to guide us in understanding where the information we required could be sought. Phil Raines on the other hand, was our 'go to' person at the outset of our work and provided day to day advice about our review in relation to the wider work of the Oversight Board.

20. We were essentially responsible to Professor Bain for progress of the review and if there were significant decisions to be made. For example, in the Summer of 2020 we were unable to sustain meetings because Gaynor and Mark had

significant responsibilities for COVID management in England. We therefore required to stop our work for a short period. The decision as to that was taken together with Professor Bain.

21. I visited the Royal Hospital for Children in Glasgow in early February 2020 and met with some of the members of the Infection Prevention and Control team. I then attended a meeting with Pat O'Connor in Edinburgh. The panel met for the first time on 24th February 2020. Unfortunately, Covid struck shortly after, and we needed to move all our work online. I think we managed this very successfully.

The Risk of Infections to Children with Malignant Disease

22. The generality of the Case Note Review was about infections in children with malignant disease, i.e. Leukaemia and cancer. There were also a small number of children included in our review who had non-malignant but serious blood disorders, for example Aplastic anaemia and severe Haemophilia. There is typically a higher risk of serious infection for those having bone marrow transplantation but in reality, that risk confronts all children being treated with chemotherapy.

23. A large component of the work of an oncologist is supportive care. In cancer care you use potentially lethal doses of drugs and then salvage the patient from the side effects of those drugs in the hope that they will have a damaging effect on the disease. Supportive care is generally the management of infection, nutrition and transfusion requirements. It comprises of a huge component of the work. All oncologists and haematologists have a day-to-day responsibility for the management of children with infections. There are certain types of unusual or persistent infections for which you would seek advice from microbiologists and in some cases infectious disease colleagues. The management of infection is part of the bread and butter of our role, and we have protocols in place for the management of infection.

The Terms of Reference

24. The Terms of Reference ("TOR") of the Case Note Review are set out in chapter 2 of the Overview Report. They were not set by the Expert Panel. They were determined at an early stage in the project. Although I was involved in discussions about aspects of the approach to be taken by the Case Note Review from January 2020, draft TOR for our work were provided prior to the first meeting of the Panel (held in Edinburgh on 24 February 2020) by Phil Raines. The TOR as reproduced within chapter 2 of the Overview Report are substantially unchanged from that draft. I recall that we discussed them and made some suggestions and they were accepted at that meeting. I do not think we (the CNR Panel) significantly influenced them. We may have refined the language to a degree but did not fundamentally alter them.

25. A key question asked in the Terms of Reference (set out in Section 2.2 of the Overview Report) was the extent to which it is possible to associate the infections identified in patients within the CNR with the environment of the RHC and QEUH. I have been asked about the word "associate" and what it means in this context. I can confirm that this does not mean the same as "caused by". "Association is not causation" is a key mantra in epidemiology. Merely because something happens at the same time or alongside something else does not necessarily mean that one causes the other. It can raise the question that there might be a link. We were tasked with searching for evidence that might link an infection in a patient with a source of that same infection somewhere in the hospital environment. Our investigation was set up to attempt to see whether we could draw inferences between the identification of a bacteria in a child with the identification of the same bacteria somewhere in the hospital environment.

26. Due to the nature of the infections we were dealing with, we focused on the water supply and the wet parts of the hospital. We did not investigate the air supply because the nature of the transmission of infections identified for the CNR was not airborne, and we were much more interested in water supply, drains, sinks and surfaces. We were interested in where water went and how areas were being cleaned. If water is contaminated and then it is used to clean a surface, then bacteria are potentially on that surface.

27. For avoidance of doubt, the term ‘environment’ in the Overview Report refers to all surroundings to which a patient may be exposed. This may be the hospital, the home or other places in between. However, in the context of our work, given the nature of the infections and the questions of association we were asked to explore, the use of the term environment was almost always synonymous with the hospital surroundings and, because of the predilection of Gram negative bacteria for wet places, we focused on aspects of the environment relating to water (its provision, use, drainage). Water may also be present in air systems, such as air conditioning systems but distribution of Gram-negative bacteria by air flow is not a recognised risk in contrast with, for example, certain fungal infections which we were not asked to investigate. My colleagues Mark Wilcox and Gaynor Evans would be much better placed to provide a more detailed commentary on this point.

28. The most certain scenario would be to find a bacterium in a patient which we could characterise in a way that when you compared it with the same bacteria found in a water supply you could say it was the same infection. Without such certainty, if a patient was shown to be in physical proximity to an identified source of infection, you would be closer to suggesting a causation. This still involves an element of assumption regarding the environment and the child’s exposure to the environment. As our report sets out there was a lot of uncertainty and one of the reasons for this is because the environment was not being surveyed in a particularly frequent manner, and testing was not being undertaken that would allow us adequately to compare the nature of some of the bacteria that were identified in patients with the bacteria found in the environment.

29. Testing to obtain definitive proof would require a regime of testing that was able to demonstrate the presence of precisely the same bacteria in the environment of the patient as found in the patient, with a plausible means of their interaction. This would require prospective and extensive screening of all parts of the hospital environment to which the patient was exposed including, for example, areas like radiology departments, operating theatres as well as ward areas. Confirmation of bacterial similarity would require genome testing of paired samples. This is generally something that is not achievable at scale and might only be

implemented if there were concerns of the nature identified at GGC.

30. As time went on, we increasingly believed that there were sufficient grounds to be suspicious of the environment that a more robust scrutiny could have been set up to try and identify more precisely the link between infections and the environment. Steps were taken by NHS GGC to improve the water supply and sterilise the environment so presumably they believed that the environment was in some way responsible for the infections, but the data collected by them did not help to define that.

31. For avoidance of doubt, 'linked to' in the context used in our classification of infections (described in section 3.6.5 and reported in section 5.6) reflected the level of certainty about the likelihood of a hospital source for the infection. This is discussed in section 3.6.6.

32. When considering issues of potential association, it is useful to reflect on where you see a sequence of infections occurring in different children over a relatively short period of time (a cluster). This would suggest that there is something potentially in the environment- why else would half a dozen children get this particular kind of infection at the same time if there was not some kind of heightened environmental risk? The background to all this is complicated further by the fact that these children are more vulnerable to infection regardless of how risky the environment could have been. Bacteria will not always cause an infection where they are found in healthy people, they are naturally found in the body, especially the gut, but natural defences limit the likelihood of infection except when immunity is challenged.

33. NHS GGC and HPS had, as well as ourselves, managed to identify patterns of infection, and the follow up question to that is how significant those patterns were. We took the view that some of the patterns of infection were more significant than others. I believe NHS GGC in the past had been reassured that they were not outside the ordinary.

34. We provided information regarding patterns of infection in section 4.3 and illustrated by some of the Examples given in chapter 8 (particularly Examples 8.2

and 8.3) before being discussed in section 10.2 where we stated “By 2018, we suggest that simple observation should have identified a disturbing pattern characterised by the occurrence of bacteraemia caused by some very unusual microorganisms and apparent clusters of some of those more commonly encountered”. One example of an unusual pattern of infection by a bacterium not uncommonly seen in children being treated for cancer would be that of Klebsiella (described in section 4.3.3) where 8 of 9 episodes of infection identified in 2016 occurred within a 6-month period and 8 of 9 episodes identified in 2017 occurred in a 5 month period. I have worked as a consultant in two UK paediatric oncology units larger than NHS GGC where I think that the frequency and clustering of Klebsiella infection would have been considered unusual. Similar data for Enterobacter and Stenotrophomonas, other relatively common bacteria in this population, are discussed in sections 4.3.1 and 4.3.2 and would, I believe, raise similar caution.

35. An example of the occurrence of less usual infection would be Mycobacterium chelonae which favours water / water systems. Although infection in immunocompromised patients is more likely, the identification of 3 cases within the Case Note Review would raise concern.

36. Our approach to these patterns of infections was very broad. We were interested in gathering information about bacterial contamination of any aspect of the environment, although a lot of the focus was on water sampling. There are many other sources of environmental testing that can be undertaken. It is true that if you sample areas that are dirty, such as drains, you may find information that cannot be interpreted successfully. For example, if you are looking at the management of intravenous catheters in children. Those caring for them should wash their hands and therefore it is important to go into detail and discuss audits of cleanliness in terms of clinical practice and environmental cleaning as you require to address every possibility of how a bacteria could get from one part of the environment into a patient. The patient’s environment obviously includes outside the hospital and on occasion we required to consider whether or not infections arose from their own environment. We did identify one patient where we considered that the infection had not arisen within the hospital at all.

37. If it was established there was contamination of water, it would raise a concern about the bacteria in the water supply spreading to other parts of the environment. Again, though it is important to recognise that bacteria are very often present in the human body so contamination could come from toilet use and such like and not necessarily contaminated water supply.

38. It was difficult to determine which particular aspect of the environment was the likely source where we considered there was a likely link with an infection in a patient. As indicated, we focused our attention on aspects of the environment relating to water (its provision, use, drainage) but the data available from surveillance at such sites was very limited.

39. We believed there were circumstances where certain bacteria may have more likely arisen endogenously (i.e. within the patient him/herself). For example, where we saw clear evidence of diarrhoea or other indications of gut damage in the presence of a bacterial infection, we considered the possibility that the source was endogenous via gut translocation rather than via contamination of the external environment.

Possible Ways Infections Could Occur

40. We tried to think of all possible ways in which infection could occur, be disseminated, or even prevented. As discussed in part 5.2 of the Overview Report one of the areas we looked at was issues surrounding maintenance, either undertaken routinely or because equipment or system needed to be repaired. This is because such activity may disrupt the environment and potentially disseminate contaminated sources. Issues such as blocked sinks or pooling of water from blocked sinks would be routinely reported to maintenance to fix. One of the frustrations we encountered when looking at maintenance logs was, for example, that a plumber would be called to a specific ward to deal with a blocked sink but there would be no record kept about which sink it was. When it came to trying to link those kinds of interventions to individual patient infections it became impossible. We would know which ward and sometimes which bed a patient was in, but we would not know if they had been in the room where a sink

had been manipulated by plumbers that day or the day before. The information was not useful in this respect.

41. We found very few examples of where "work undertaken in close temporal and physical relationship to the care environment of a patient could be linked to the occurrence of a specific infection or to potential outbreaks" (Overview Report, section 5.2 page 60). We could make general observations for example where we know that plumbers were called to a specific ward several times in a month. We might not know why they were called so it was not very useful information, but it might suggest that there was some issue occurring. Despite that, there might not have been any infection of interest that month so no clear pattern of association. If the maintenance records had been more precise, we would have had a better chance of establishing association.

42. One of the commonest type of infections in central lines do not involve the type of bacteria we were concerned with in the review. The most common kind of infections in central line are gram-positive bacterial infections, staphylococcus particularly. These bacteria sit typically on the skin and are present in everyday life. They only become a problem under certain circumstances, and they are particularly difficult when a central line is fitted. With this in mind, we applauded the work done by the Quality Improvement Group to push down the incidence of central line infection, but the reality is, as important as that work was, it was unlikely to have any significant impact on the occurrence of gram-negative environmental bacterial infection.

43. In Chapter 9 we outlined evidence of good practice and I believe we acknowledged the work the Quality Improvement Group had done. We also indicated that we found evidence of very good documentation of central line care in the nursing records.

44. When it comes to gram-negative environmental bacterial infections, although central line care is very important to minimise the risk of contamination, it does not itself address the source of such bacteria in the environment.

Identification of Cases for Inclusion in the Review

45. This is discussed in some detail in section 3.2 of the Overview Report. The work relating to the identification of cases by HPS was largely completed prior to the first panel meeting. We were presented with a rationale for the identification of cases. HPS had looked at a number of different sources of information because there were different datasets within NHS GGC. They completed work to avoid duplication and to be as inclusive as possible to obtain the fullest possible dataset for us to look at.

46. We did talk about the datasets and the information provided to us. We were confident that all the infections were appropriately included but we do not know if there were other infections that were not in the dataset but occurred within the period of our review. We had to accept that there was a sufficiently rigorous selection of the data, but I had no reason to doubt that the dataset that was presented for us to work on was in any way inadequate.

47. The HPS report was presented in 2019 – **[A33448012** - **HPS Review of NHSGG&C paediatric haemato-oncology data October 2019 – Bundle 7, Page 214]** - before we were involved and is discussed more fully at paragraph 8.2.3 of the Overview Report. The generation of the cohort of patients was identified prior to the first meeting of the panel. We were presented with the cohort and that is what we worked from.

48. At our first meeting it was agreed that the dataset was appropriate. There were representatives from HPS there. I believe Lesley Shepherd was representing infection control.

49. Prior to the first meeting of the Panel on 24.2.20 at which the cohort of cases to be included in our review was agreed, I had participated in a couple of other discussions which included consideration of the make up of the cohort. I have notes of meetings in late January and early February at which Marion Bain was present but I have neither noted nor remember her taking a particular position. It is my recollection that HPS took the lead on this and an email received from Phil Raines on 5.2.20 (to which Marion Bain was also copied in) stated *"I will find out from HPS how it is defining the sample and provide the full detail as soon as I*

get it. My suggestion is to make that available for full discussion – and final agreement – when we all meet on Monday, 24 February".

50. We all agreed the time period, which was straightforward. The hospital opened in June 2015 and the dataset went back on one month before to ensure that if a child were transferred with an infection, it would be able to be traced back to the previous hospital. The dataset ended in December 2019 which was shortly before we started our work.

51. We did reflect on whether other cohorts or bacteria should have been included. For example, very early in our discussions we talked about fungal infections, typically aspergillus fungal infections as these are environmentally linked and do occur in haematology-oncology patients. We discussed it and pulled out some data looking at the early instance of fungal infection, but we concluded two things. Firstly that it was not likely that we would be able to ascertain that there was a link to inappropriate environmental contamination, and secondly that we were not convinced that the pattern of infections from the preliminary data we had access to was of sufficient concern. It does however appear more frequently in environments where there has been recent building work, and it is of a particular risk for immunocompromised patients. We did therefore challenge ourselves whether there were additional infections that we ought to have been considering.

52. We reflected more than once on the question whether if the environment of the hospital was in some way a risk factor for patients, should our work not have also included a scrutiny of infections in other parts of the hospital. There were two reasons for that, firstly because haematology and oncology patients were mainly managed in one area, although obviously visited other areas of the hospital and secondly as we continued our work, we heard the odd reference to concerns about Gram-negative infections occurring in other areas, specifically the NICU and PICU. We were not asked to extend our work and we did not interrogate those data. I do suppose that if you wanted to look at the environmental health of the whole hospital, a much bigger project could have been set out, but it was not what we were asked to undertake.

53. There may have been mention of a wider review in my early discussions about

the project (i.e. before the CNR) but it was pretty clear from the outset the focus was to be on haemato-oncology patients.

54. The statement in 4.3.5 about the frequency of infection was prefaced with *"whilst it is not possible to state this with certainty"*. The HPS Review of NHSGGC paediatric haemato-oncology data 2019 report **[A33448012 – HPS Review of NHSGG&C paediatric haemato-oncology data October 2019 – Bundle 7, Page 214]** discusses some of the challenges in demonstrating changes in frequency of infection over periods of time and recommends the use of aggregated data for all bacterial infections within the subgroup of interest to provide a more reliable indicator. Nevertheless, we looked at the relative frequency of individual types of bacteria within our review in relation to those most likely to be seen. As stated, Klebsiella and Pseudomonas are likely to be the most common yet the number of Enterobacter and Stenotrophomonas infections both exceeded that of Pseudomonas and, for Enterobacter, were close to the number of Klebsiella (see Table 4.2 in our report). These are small groups of data and comparisons of this type must always be considered with caution, but this nevertheless influenced our opinion.

The Use of Control Measures to Understand Infections

55. A control measure as referred in paragraph 3.3.1 of the Overview Report is anything you might do to potentially minimise the impact of an environmental risk. For example, NHS GGC chlorinated the water supply at a point in time, which is a control measure. The Health Board also instigated enhanced cleaning regimes which could also be considered a control measure. Control measures are a useful data source in an epidemiological study, for example if there is a change in the incidence of infections following on the implementation of a control measure.

56. When considering a timeline if you see a control measure that has been introduced and the infection numbers drop, it could potentially help you to conclude that the infections were more likely to be linked to the environment. For example, if there was a run of infections in a particular area of a hospital and an

aggressive programme of augmented cleaning was introduced and thereafter the frequency of infections dropped, it could be justifiable to say that there was something in the environment that was suboptimal but that had been overcome. It could be justified to say that there was a relationship between the infections previously identified and the state of the environment. It becomes more difficult when attempting to assess direct impact on patients and it is also very difficult when considering sporadic and relatively infrequent nature of these infections. The influence would be quite hard to pick up in observations. The use of 'direct impact' here is synonymous with defining the burden of the infection on the patient.

The Burden on the Patients

57. At the outset of our work, it had not occurred to me how important it was for us to be able to say something about the burden of these infections because at the beginning the focus was on that these infections had occurred and the question was, were they caused by the environment? As the work progressed, we began to realise that we had something important to say about the impact of these infections on individual patients. I took the attitude that if the infections did not matter, then why were we doing the work? In reality the infections do matter, and with our data we are able to point out the real impact on patients, whether or not the infection was caused by the environment.

Methodology

58. Our methodology is set out in Chapter 3. I suppose I took a significant lead in defining the methodology. It was a little iterative. The process therefore evolved slightly but they were essentially team decisions. I took the lead on how we were going to handle the integration of the clinical and epidemiological data. Fiona Murdoch at HPS presented the way in which she was going to provide us with her information, but we agreed on it and worked together. It was a bespoke approach and as there were different channels of information available to us, we needed to design our own approach in terms of data collection and its synthesis.

Certain elements such as the PTT and the National Framework for Adverse Events in Scotland were taken as previously defined.

59. No other methodologies were considered. I was not aware, at the time or now, of an established methodology we could have used to undertake an assessment of this nature, considering each infection in every patient. The start point was the need to extract relevant clinical and microbiological information for each episode in order to establish a clinical timeline against which we could interrogate a) whatever data were available about how the infection episode fitted within the pattern of other infections in the same group of patients and, b) whether there was useful data from the hospital environment at that time.

60. The demonstration of a causal relationship between an infection and the presence of the same bacteria in the hospital environment requires (as I discuss later) robust time/person/place data supported where possible by genomic typing.

61. The work required to obtain such data prospectively in all cases would be substantial. This is why monitoring infection rates and responding to an unusual pattern (in terms of frequency and type of infection) provides an opportunity to introduce data collection of this type when concerns are raised.

Case Definition

62. At section 3.2.2 of the Overview Report we explained the defined protocol by which blood cultures are identified for inclusion in the study. This was because the difficulty is that if a patient is infected with an organism that shows up in a blood culture, repeated blood cultures may be taken and therefore there may be sequential positive blood culture results over a period of time. The first part of the case definition was that there would be a 14-day period in which you do not recount the same infection. For example, if you had a positive blood culture and then eight days later you were tested and found to have the same blood culture, it would not be counted as a second infection. In that case an assumption would be made that it was linked to the first infection. This tries to avoid a situation where incidents are overestimated. This is an area where both Mark Wilcox and

Gaynor Evans will have an expertise.

63. “Hospital-associated infection is a positive blood culture in a patient who’s been hospitalized for 48 hours, but a healthcare-associated infection is a positive blood culture in a patient within 48 hours of admission, but who has nevertheless had contact with healthcare in the previous 30 days.” Using the same initials for both scenarios may potentially cause problems. More importantly, there is a potential that you are looking at slightly different groups of infection; one that is acquired in a patient who has already been in hospital for at least 48 hours, and one in a patient who has come into hospital, and whilst they may have had contact with the hospital as an outpatient, say, in the past month, they weren’t necessarily acquiring the infection whilst they were in the hospital. This does raise questions about the origin of the infection.

The Use of the Paediatric Trigger Tool

64. The Paediatric Trigger Tool (PTT) is explained in section 2.4.3 of the Overview and the purpose of a trigger tool in general is explained in the first paragraph of section 3.4.2 of the Overview Report. The use of the PTT in the CNR was established prior to my appointment. I think it was the decision of the Deputy Chief Nursing Officer, Diane Murray and she had a particular interest in it. I think she felt that the application of the PTT would illuminate what was happening at GGC.

65. The requirement to include the PTT in our review was made clear to me at the outset and before the CNR panel had met for the first time. I had not previously been involved in its use in my clinical career although I was aware of it as a tool for measuring adverse events in health care settings. I was unclear what its use would bring to our work and did not feel it would directly address the key terms of reference set for the CNR. This was discussed in some of my initial meetings and again at the first meeting of the Panel on 24.2.20. It seemed to me that the PTT was to be used to assess aspects of the quality of the care provided at RHC and I had acquiesced to this. However, I sought to ensure that when clinical data was collected from the patient records for the PTT, the data I had defined as

required for our clinical timeline for each patient would be collected at the same time. In the event, this worked well, especially as we came to rely heavily on Pat O'Connor and Peter Davey for the abstraction of all relevant data from clinical records and to return to these, often on multiple occasions, to answer our queries. If they had not fulfilled this role, we would have had to identify an alternative team to do this.

66. The PTT did not trigger the collection of patients into the cohort – as indicated in this question, this was undertaken by the microbiological definition reached by HPS. Little if any of the data collected specifically for the PTT was of direct use in helping us reach conclusions about any link between infections and the hospital environment.

67. Previously I had no personal experience of utilising the PTT. I initially did not understand what it would bring to achieving our terms of reference and I would say that I did initially push back on its use but I rapidly realised that the information that was required to be extracted from the case notes for the PTT actually allowed for all the information that I wanted for our data synthesis of these patients to be collected at the same time.

68. As time went by, I began to realise that the PTT had a value of its own. Pat O'Connor and Peter Davey wrote a separate detailed report about their findings, which was submitted to the Oversight Board with the expectation that it would be published at the same time as the CNR. This did not happen and I was unable to ascertain why or at what level this decision had been taken. I remain unclear whether this report has yet been made available within NHS Scotland but whilst it did not address the issues of causation in relation to infection and the environment, the findings offer useful insights into good practice in care and record keeping at NHS GGC, and into areas where improvement might be made. On that basis we decided to include the analysis of Adverse Effects experienced by the children, made possible by the work of the PTT, in our chapter on the impact of infection. My initial reservation about use of the PTT was that some of the clinical features were being scored as part of the trigger tool were potentially irrelevant to our patient population and others were so frequent that they were uninformative. I can explain my initial concerns by reference to the Paediatric

Trigger Tool Score Sheet in Appendix C of the Overview Report (at page 132). The first column is a list of possible triggers. The second allows whether the trigger has occurred to be recorded whether the incident has occurred during the period under consideration. The third column records whether or not an Adverse Event (AE) occurred, and fourth column provides for the assessment of the severity of the AE. Returning to my initial concerns trigger PG5 is 'Cranial Imaging' which is presumably a CT or MRI scan of a head or brain. If a child has a brain tumour, then multiple cranial images will be taken because it is not only essential for diagnosis but also for monitoring response to treatment. I did not think that scoring whether or not a cranial image had taken place represented an adverse effect because it did not. It was a required part of treatment. Similarly, on the next page (page 133) there is a trigger PM6 "Anti-emetic given". This is a medication used to stop being feeling or being sick. It is well known that chemotherapy can make you feel sick, so we routinely give antiemetics to patients receiving it. Children would very frequently be given an antiemetic injection prior to and for a day or two after the administration of routine chemotherapy. Also, PL2 'Transfusion' – the use of transfusion of blood, red blood cells or platelets is an integral part of the supportive care of children receiving chemotherapy. I thought it was uninformative. Some of the other things are manifestations of children who are very sick, for example PL5 refers to abnormal shifts in the serum level of sodium but these children are being monitored for these things on a routine basis. I think the intention of using the PTT was to look across a range of different cases. For example, most hospitals will take a random sample a month prior to admission and applying the PTT they will pick out patterns and be able to pick out things such as sodium levels not being correctly monitored and then respond to them. It is a tool for improving practice.

69. As our investigation and processes evolved, Pat O'Connor and Peter Davey, started analysing the data and comparing it to the data from the reporting of adverse events in Datix with NHS GGC. I recognised in this the value for NHS GGC in improving their recognition of adverse events and their use of that information to improve care.

70. The clinicians in the RHC were very exercised about the use of the PTT at the very outset and I was challenged about its use. I think I first met with them on, 3 February 2020, before the panel had even assembled. I do not have a note of individual names of who attended that meeting but there were seven or eight clinicians, predominately senior medical staff from the Oncology team in attendance. It was difficult for me at that point to justify its use because I had not used it myself before and could not rely on firsthand experience. I think they felt it was an attempt to identify that their treatment was suboptimal but in fact if you read our conclusions, we felt that the treatment that the patients received and the documentation of medical notes and the collaboration between the clinicians and the microbiologists in the management of patients was very good. We did however think there were learning points about the reporting of adverse events. The work could have been done without the PTT but it did add an extra dimension and I felt that by the use of it we offered NHS GGC helpful insight in that they probably were not using their own adverse event reporting system as adequately as they might and that there were other ways in which they could look at their adverse event data. I do not believe we were unduly critical of them in any way.

71. We did have ongoing meetings with the clinicians themselves and there is a timeline that starts on page 129 of the report where it shows when the meetings with clinicians took place. They had a meeting with the CMO on 3 March 2020 where I believe they articulated concerns about our work. They were really concerned about what we were going to do and felt that their own clinical practice was in the firing line. The timeline dates the other meetings I had with them throughout the year. I am hoping the issue is settled in terms of the clinicians, I could not share any information with them until the report was produced. It was not until they had the opportunity to read the report that they could reassure themselves. My sense was by the end of the process that they were more grateful for the review than they thought they would have been.

72. In summary regarding the PTT, at the beginning I was challenging about the use of it but then I became agnostic and by the end I believed it actually did add an additional dimension of value to the work of the review. I do not think it illuminated the enunciation of our core terms of reference which was whether

there was an environmental risk to the cause of infection. It did however look at potential impacts on patients and the work that Pat O'Connor and Peter Davey did allowed us to produce the chapter 6 on the burden on the patients.

The Expert Panel Review Process

73. Expert Panel Review Process is the heart of the CNR. We created a systematic approach to the capture, scrutiny and summation of the data available in every case. We sought to achieve and record, in a structured way, a consensus view as set out in the Conclusions record sheet in Appendix D (page 136) and described in section 3.6 of our report.

74. This was an approach created for the CNR - I know of no other similar investigation that has used the same process, but it has similarities to the approach that may be used in a Root Cause Analysis.

75. Figure 3.6: Case Note Review Process Map can be found at part 3.6 of the Overview Report and shows how we looked at each of the 120 episodes of infection. Each of these episodes was looked at and the starting point was essential the extraction of basic information. A key stage before Full Panel Meetings was Data Synthesis. Appendix D (page 134) shows the Data Synthesis Template which on we identified things that we thought were important to know about. These were a series of data items and the first stage was to try and pull out the information that helped populate that data synthesis template. A separate Data Synthesis Template was completed for every infection in each patient.

76. The HPS team provided us with information. For example, dataset item 19 was date of admission and 23 would be date or dates of previous attendance, 25 ward and bed location. This was provided for each of the patients. We were told where the patient had been admitted from and we were shown which beds the patients went to. A visual tracker was established using software which allowed us to see how the patients moved around the hospital.

77. The PTT team who were working on the clinical records pulled out information to provide us with more of a narrative of care. They would indicate things like "Child

attended the day unit with a history of fever for 12 hours" or "Was referred from Fife with a fever". They would describe something about the child's condition, their temperature, where the infection was thought to be and when antibiotics were initiated. The HPS team had access to antibiotic prescribing data too so from those two sources we created a stream of information which then had to be integrated. This was essentially my task - I think I was the most suited as it required clinical knowledge of patient care.

78. For every infection episode I extracted information and populated the data synthesis template. I also created a corresponding timeline to ensure there was a chronology to events.

79. We created a data synthesis and clinical timeline for every episode. I used to meet with Pat O'Connor, Fiona Murdoch, Jane McNeish, Marie Brown and Emma Mackay each week. We would clarify points and I would ask them questions about things I did not understand. I would ask them to go back to the clinical record or clarify something such as where a patient was nursed or when antibiotics were given or stopped.

80. By a process of iteration, it took a bit of time, but we were able to create a report which we thought was sufficient comprehensive to take to a full panel meeting. At that meeting Gaynor, Mark and I would sit down and look at the reports and discuss what we thought was going on, on the basis of that information.

81. The next step in the process involved completion of the second part of the Data Synthesis Template – Summary. This can be found on page 135 of the Overview Report. This was very much expanded when populated with information. We would summarise the clinical situation. We would look to see, for example, whether the patient had evidence of damage to the bowel because that is a possible way in which patients might acquire infections of this kind. We would then look at the Tableau timeline. Tableau is the description of the software we used, essentially it is a timeline from which we could see if there were any clustering of other patients with similar infections. We started adding information from other hospital systems including what responses were from microbiology and the Infection Control team. We looked at the ICNet and Telepath data bases

and at relevant IMT and PAG minutes to see what the response of the organisation had been at the time. We also looked at environmental information - for example where water samples were taken and where swabs of surfaces were taken – and at maintenance and building activity and at cleaning records. We interrogated the Datix system, which is about adverse event reporting, and finally sought to identify whether there was any other relevant information available. Parents also had the opportunity to send us information, and some did.

82. If we identified any clustering, we would then ask ourselves what this might mean. We would look at the information provided by the Infection Control nurses and by the microbiologists to see whether they had made any observations, particularly at observations recorded in the Incident Management Team minutes and the Problem Assessment Group minutes. We would assess whether these gave us an insight into whether there were concerns about environmental hazards that could point us in that direction. We would then look at the evidence of any surveillance cultures. We did make the point that there really was not very good, useful data but there was some. We would look to see if there was anything around that information that would help us. We have already covered the issue about maintenance activity and how difficult that was, but we nevertheless looked at all those things for each patient and tried to draw them together.

83. In reference to second paragraph of Overview Report – Para 3.6.6; this suggests that particular weight was given where a cluster of episodes (understood to be a number occurring within a short period of time) was found to have occurred involving the same bacterium and different patients. The presence of a cluster of similar infections affected our assessment of the probability of an environmental cause

84. Taking all the information together , we then populated the Summary component of the Data Synthesis Template. We asked ourselves all these questions and then tried to answer whether it is possible to link the infection episode with the environment. We then moved to the Conclusion pro forma which can be found on page 136 of the Overview Report. It was not possible to do that with clarity at

one sitting for most patients, we had multiple review meetings, sometimes up to six. We would keep bringing patients back to the panel meetings until we were satisfied that we had as much information as was available and that we had achieved maximum benefit from all the information available.

85. As the process matured, we got better at being able to challenge information, we would ask for things to be double checked or ask for further data items. We got more sophisticated not only in terms of integrating information but also challenging.

86. The whole case note review process was about creating a data synthesis in a clinical timeline and integrating it with all the other sources of information in the red box on the left in figure 3.2 and the having a discussion, sometimes on many occasions, to achieve a final outcome. We did not have any patient identifiers; all the work was done without any knowledge of who the patient was.

Categorising Likelihood of an Environmental Source for an Infection

87. In section 3.6.6 we discussed how we categorised likelihood of an environmental source for an infection. We decided to categorise episodes into one of four levels of likelihood that the hospital environment was a source of a bacteraemia: Unrelated, Possible, Probable or Definite. This approach is discussed further in Chapter 5, section 5.6. In some cases, we thought we might be unable to determine likelihood because of inadequate or conflicting data. The allocation of these descriptors inevitably represented a position taken along a continuum of certainty and, for the two largest groups (Possible and Probable) we attempted to refine our position by further extending our categorisation into Weak Possible, Possible, Strong Possible, Probable and Strong Probable groupings. We did not feel we were able to distinguish between Probable and Weak Probable.

88. In regard to the categories or terms used in Para 3.6.5 and 3.6.6, there is no distinction in meaning between “Confirmed” and “Definite”. Although we had used ‘Confirmed’ on the Data Synthesis template, as we were finalising our assessment via the process of the second review we undertook for all patients,

we chose to use the term ‘Definite’. I don't think any great significance derives from this but I recognise there is a potential for confusion. Although we had initially used 5* descriptors in our final outcome reports (section 3.6.5) as the project progressed, we came to see that this was insufficiently granular. The report describes the sub-categorisation of ‘Possible’ and ‘Probable’ to create the final list of 8* categories used in section 5.6 and Table 5.3. [* means that there are 5 or 8 categories if one includes ‘Unable to Determine’]

89. I do not think it would be fair to say that our final conclusion was circumstantial. We had a lot of different sources of information. One particular piece of information did not allow us to be absolutely certain in our conclusions. What we have tried to describe is how our judgements were made after considering all the different kinds of information available to us. We looked at all the circumstances that were capable of being connected to a particular episode of infection to come to our conclusions. We worked on the basis that the more the various pieces of data available pointed to a particular conclusion, the stronger the conclusion.

90. In Section 3.6.6 at the top of page 44 there is a paragraph that sets out reasons why we thought some cases were not related to the hospital environment. We then went on to talk about some of the factors that helped us point more towards reaching a conclusion that they might be related to the hospital environment. We referred to some of the difficulties about the frequency with which some of the environmental testing was done and how difficult it was to link to it. It is important to remember that association does not infer causation. I recall one of the criticisms made by GGC in their critique of our draft report in which, in reference to section 3.6.6 of the report, they wrote to us as follows:

> *“A key omission for context; there is no reference to published literature on the methodology utilised by the panel given that causality is assessed using the Bradford-Hill criteria (J Roy Soc Med 1965:58:295-300) as any observed association may in fact be due to the effects of one or more of the following: chance (random error) ; bias (systematic error) ; or confounding”*

and they offered reference to a teaching resource (the significance of which we were uncertain). We reviewed this section of the draft report in response to

GGC's comments but felt that our text had adequately set out the caveats that applied to our conclusions. The only significant addition to the text in its final form was the addition (at the end of paragraph 7 of section 3.6.6) of the sentence starting *"Given our remit, we focused on potential hospital sources of infection…. This* goes on to acknowledge the possibility of infection acquired outside the hospital environment.

91. I have been asked to explain how we assessed infection arising outside the hospital environment. Clearly it is difficult to be absolutely certain in making this distinction but we relied on our assessment of the type of infection and the strength (or otherwise) of the opportunity to acquire infection in hospital. For example, one patient who had a chronic blood disease, but unlikely to have been significantly immunocompromised and largely only ever treated as an outpatient, had three episodes of infection with an unusual bacterium (Elizabethkingia). This is widely found in the natural environment and although it has sometimes been described in relation to hospital acquired infection, its finding in that context would be unusual. We felt the clinical circumstances were much more likely to link to infection acquired at home – indeed this was the conclusion of the clinical team caring for the patient at the time.

92. I would not say that there was differential weighting to factors because the exercise involved considering all available factors in any situation. Our conclusions were not driven by the number of factors. Clustering is quite important, but we did not give any formal weighting to one factor or another. We truly attempted to integrate our knowledge of the patient, our knowledge of the behaviour of the individual bacteria and the environment. Some of those elements were incomplete but we used what we had.

93. It was recognised that the possibility of outside environmental sources could not be very easily addressed unless we received some specific information.

94. An 'outside environmental source' means the possibility that the identified bacterium was acquired somewhere outside GGC. In most cases this would have been the patient's home environment but for some patients, care was delivered in other healthcare settings about which we had no information.

95. Root Cause Analysis is essentially what we were doing. We were tracking the evolution of each infection and then trying to fit around it all the possible influences. That is essentially what the approach involves; you say something has happened then you consider all the possible influences on what it has happened and what the outcome was. One of our recommendations to NHS GGC was that this approach should be used by them, and it came as a surprise to us that they did not have a more systematic approach to Root Cause analysis. They did tell us about it, but it was only introduced in late 2019 which was the end of the period of our review. There was very little information from Root Cause Analysis available for us to draw on. If it had been used at a much earlier stage, then it may have provided us with more pertinent information and, more importantly, I think it would have systematised their response to the issue.

96. Root Cause Analysis is not unique to healthcare settings and may be delivered in different ways, but the fundamental approach is to consider an adverse situation / event and look at all possible factors that may have contributed to its occurrence and its outcome. By way of example, I attach [see Appendix] a template I had used previously at my own hospital. This is not the only framework one can use, and I assume GGC had their own, but the value of an RCA framework is that it offers a consistent and systematic approach to investigation and requires the identification of recommendations and actions for the future. We refer to GGC's own policies with regard to the use of RCA in section 3.4.1 and, in section 8.2.2.2, we discuss how we identified RCA utilised in two of the patients included in our review and comment on the development of the template used. I regret I am unable to retrieve this from my records.

The Standard Infection Prevention and Control Assessment

97. On page 44 at 3.6.6 we explain that standard infection prevention and control assessment reflected that "In routine practice, such a conclusion (that the environment was likely the source) would be made until or unless it was possible to confidently arrive at an alternative hypothesis for the cause or source of infection". This means that if one found an infection to be of a potential environmental origin, one would make the assumption that it came from the

environment unless it was possible to demonstrate that there were adequate infection prevention control measures in place and/or there was an alternative reason. Starting from a position of concern about the environment you then should ask whether everything that could usefully be done to prevent infection had been done and if so, had it been done properly. If you conclude that an environment had been managed properly in terms of infection control, then you would reduce your suspicion that the infection arose from the environment.

Approach to Causation

98. In terms of our approach to causation, this represented a gradient of increasing certainty. It was because of this that we decided to group the Strong Possible, Probable and Strong Probable groups together within the category of 'most likely'. Table 5.6 shows that by doing so we included only 4 cases of Strong Possible (and 3 cases of Strong Probable). By restricting ourselves to the inclusion of only those that were identified as Probable or Strong Probable, our conclusion would have been that 28.2% of the whole group fell into the 'most likely' group rather than the 31.6% we gave in our report. I suggest that this does not represent a significant difference in our overall message.

99. When looking at recognised methods for demonstrating a causal relationship there are statistical methods you could apply. Looking at the issue very simply, if you have two populations of patients, and one had been exposed to an abnormal water supply but the other had not, and then you look at the frequency of an event – say an infection in both populations – you could do a statistical test to prove that the number of infections was greater in one population than the other. On completion of the statistical test, it would allow you to come up with a statement that on the balance of statistical probability, the exposure of this population to this abnormal water supply meant that they got infections while other people did not. The difficulty there is that you have to be sure that there are no confounding variables. You also have to have enough patients in the analysis because it cannot be done reliably with small numbers of patients. I think in our setting it was very difficult to use statistical information to give any

kind of meaningful conclusion. I do not know if there is a method available to establish a causal relationship in such a setting. The process of causal inference is complex, and arriving at an inference of causal or non-causal nature of an association is a subjective process. It comes down to judgment. If we had been able to apply a test and say that the link is definitely caused by the environment it would have been a much easier piece of work.

100. It is perfectly possible to undertake a statistical comparison between two groups when those groups are clearly defined and are otherwise comparable in terms of possible factors which may distinguish them. In the case of our work, we had no comparative population. It will be interesting to see a comparative study with another group of patients from elsewhere but the validity of any conclusion reached will need to be assessed. It may also be relevant to point out that staff at GGC had themselves suggested a link between infections and the environment – see, for example, the text of our Example 8.4 on page 90 of the report.

The Support and Literature for the Approach Taken by the CNR.

101. One of the criticisms that was levelled at us by NHS GGC in response to our draft report was in respect of lines 828-829 of the draft which related to paragraph 3.6.6 of the Review. According to NHS GGC: "A key omission for context is no reference to published literature on methodology used by the panel, given that causality is assessed using the Bradford Hill Criteria". We responded by saying that we believed that it was implicit in our approach.

102. There is a whole science behind infection prevention control and environmental infections. Time, place, person approach is standard practice if you are asked to investigate an outbreak of infection.

103. Bradford Hill was a very distinguished occupational physician and statistician in the 1960s. He published a piece of work which talked about the criteria that you might use to try to associate a factor (not necessarily an environmental factor) with a specific disease. He talked about the strength of association, the consistency of the association, how specific the association was – the temporal

relationship. These remain broad principles but as times have moved on they can and have been challenged.

104. I don't think we are challenging the work of Bradford-Hill but merely pointing out that whilst the principles of his observations about association remain important, they have been challenged. Indeed, this is referred to in the reference provided by GGC quoted and commented on at paragraph 93 of this statement.

105. In saying that, I do think we were doing exactly what he set out. He said that you have to think about all these different things and the web reference that NHS GGC provided us in their letter addressing our report in this section refers to a teaching session on the Bradford Hill criteria. It simply talks about them and says that these were historically important. The conclusion of the link states, "The process of causal inference is complex, and arriving at a tentative inference of a causal or non-causal nature of an association is a subjective process". We recognised that and integrated the information that was made available to us and then made a judgment on it. We have been quite upfront about the fact that the review is our judgment.

106. I hope we have demonstrated how much information we carefully considered and how we used it. It will be for others to judge whether it stands up to scrutiny but clearly NHS GGC did not feel that what we did stood up to their scrutiny. However, I stand by what we did and our conclusions.

Case Note Review Concerns About Use of Systems by GGC

107. Our concerns are captured in Chapter 8. We talk at some length about NHS GGC's management, investigation and reporting of infection episodes. We discuss the availability of data, its quality and our access to information systems in general. We had difficulty with the environmental microbiology and facilities and maintenance work data and the lab information systems. We wrote at length about the problems were and attempt to illustrate it in the review. We were particularly critical of the fragmentation of their approach to the IMT meetings. Each investigation of an infection or a series of infection seemed to stand alone,

and nobody raised the point that the same issue had been discussed a few months ago or asked the question as to why they were not linking back to what had been discussed previously. We also had concerns about bacterial typing.

108. Our concerns about bacterial typing are discussed in section 8.3.1 of our report. We recount challenges with the documentation and detail of results, and the lack of any system to allow results to be aggregated, linked or searched.

109. We had concerns about clinical records, not what the medical or nursing staff wrote but their organisation. It is not a unique problem to NHS GGC, but electronic patient records have evolved in a way which is not always completely intuitive in terms of how you go about finding information, it is not always necessarily chronological.

110. A summary of our approach to/ concern about the clinical records is described in section 8.4 of our report. I can amplify this by referring to the separate report by the PTT team **[A48184781 – A Paediatric Trigger Tool Review of Patients a the Royal Hospital for Children in NHS Greater Glasgow and Clyde – Report by Dr Patricia O'Connor, University of Stirling and Prof. Peter Davey, University of Dundee – March 2021 – Bundle 25, Page 304]** which, in section 4.3, sets out how the medical records were arranged and the challenges of identifying data within them.

111. We deal with the use of SPC charts at 8.2.3 in our report, on p88. We say that we had reservations about the reliance on SPC methodology. It is a perfectly respectable methodology but there are concerns about trying to apply it in situations where there are a relatively small number of incidents. It is a way of looking at trends of occurrence of events over periods of time, and the trend is much more tightly defined if you have lots of things happening. It is just like trying to draw a line through a series of data points, lots of points means you can get a much straighter and convincing line. If the data points are all over the place, it's much more difficult. We said two things, one that you need a baseline for SPC because what you are trying to do is to compare what is happening now with what happened in a previous period. The second thing is that we thought the numbers for this approach were small.

112. We took a simple approach. We just looked at timelines. We stretched out all these infections and asked what was "going on here?". We give an example in a box (Example 8.2 on page 88) looking at a bacteraemia called Klebsiella. Klebsiella is a relatively common infection in these patients, so it is not so unusual. We however pointed out that just under half the infections that were identified in the review occurred in a period from June to November 2016 and then again in 2017. It was a clear clustering and as we have acknowledged a cluster does not tell the whole story, but it does ask the question if the infections should be looked at or taken more seriously. I think NHS GGC were reassured by the SPC analysis and we just think that analysis was probably inappropriate and that they put undue confidence in it.

113. You do not know what a Statistical Process Control (SPC) analysis means unless it is compared to something before. The first time you do an SPC analysis you are asking what is going on but if you want to observe a change then you have to compare what is happening now with what happened before but there was no comparison with what was happening before in this case. We wrote, "it can be argued that the use of data for the instance when the hospital was located in Yorkhill merely swaps one environmental baseline for another". The trouble was it was not the same environment, so the baseline was not really applicable. If you open a new hospital, you do not have a baseline, so you have to use another way of saying "I am not sure this is right".

114. There was an issue about alert lists or the microbiology alert system. ICNet is one of the IT systems that is used to record positive microbiology results. It has the facility by which you can add an alert for a specific bacterium so that, if an infection with that bacterium is identified in the future, the system automatically triggers a notification to the Infection Prevention Control team. We went into a bit of detail about this in our report. This can be found on page 96 in the section numbered 8.4.2. [I regret this was erroneously mis-numbered and should have been numbered 8.3.2]. Our Examples 8.2 (on pages 88 & 89) and 8.7 (on page 97) are also relevant. NHS GGC told us that the alert list had been extended to include a broader group of infections but this did not seem, from the data we examined, to have been the case.

Whole Genome Sequencing as a Tool

115. Bacterial typing data is Professor Mark Wilcox's area, and he will be able to talk to the limitations of bacterial typing. The important thing to understand is that you might have an infection with a bug like Pseudomonas. Its subtype is aeruginosa and there are half a dozen patients who are infected with Pseudomonas aeruginosa – can it be said it is the same infection? At certain level it is the same infection but within pseudomonas aeruginosa there are further subtypes and sub-characteristics. Not all Pseudomonas aeruginosa infections are exactly the same. That is reflected in the genetics typing and looking at genetic characteristics. If you take 100 isolates of Pseudomonas aeruginosa infection and subject them to bacterial typing they would not all be exactly the same. There would be a great deal of overlap and there are variations within the behaviour of Pseudomonas aeruginosa infections which would be reflected in subtle and sometimes not so subtle differences in their genetic makeup. Genotyping is looking at the genetic building components of the bacterium and comparing them.

116. If there is a Pseudomonas aeruginosa found in a water sample and the same is found in the patient, you would be looking for a very close degree of similarity between the two before you could be confident of a match. They would not necessarily be absolutely identical, but they would be close. Bacteria evolve so when it spreads from one person to another, the bacterial itself may subtly change its genetic make-up. There are rules about what you would call a close match and what is not a close match and that again is Mark's area of expertise. The ideal situation is where you find a bacterium in the environment which is a close enough match to that in the patient, you can say it is the same bacterium.

117. The HPS and PTT team were not involved in collecting the bacterial typing data. Bacterial typing is laboratory information and came from the microbiology labs. The PTT and HPS teams provided us with more conventional data about admission dates, antibiotic information and operation and culture dates and such like. We established right at the beginning a pipeline for getting all the

information, but it was only relatively late in the process that we were able to get information about genotyping from NHS GGC.

118. NHS GGC criticised us in in their response to our draft report for ignoring the value or diminishing the value of the work completed on genotyping. The problem we had is that most of the patients we reviewed did not have genotyping done. NHS GGC provided us with a lot of information about genotypes from other infections that were not included in our review. This is discussed at the bottom of Page 95, going into 96 of the Overview Report. To be fair, genotyping of bacterial infections is not standard practice and it would not be done for every infection because it is expensive and time-consuming. I think our position would be that, had it been recognised there was a problem, a systematic approach to evaluating infections using genotyping could have been implemented at a much earlier point. I think it would be fair to describe the typing data we received as patchy both in terms of samples both from patients and the environment.

119. The point is that whilst it would be unreasonable for bacterial genotyping to be applied in a widespread and non-specific manner, once it became clear that there was a problem, a programme of genotype testing could have been introduced selectively to ensure that specimens from the patients affected were compared both with each other (where bacteria were the same) and with those obtained from (augmented) surveillance of the environment (where positive findings were identified).

120. The limits of sensitivity are discussed on page 100 in respect of the lack of complete data about the location of patients within the hospital. Whole Genome Sequencing is a way of looking at all the genetic information with bacteria, so essentially, it is a genetic fingerprint of the bacteria. It identifies what I have mentioned already that first of all that the genetic code within bacteria can evolve and mutate so that things change. As the bacteria reproduces itself and spreads, it evolves and mutates.

121. There are degrees of difference. Rules are applied differently by different people in relation to how closely similar bugs need to be for you to believe that they are essentially the same bug. There is a measure of genetic difference called single

nucleotide polymorphism (SNP). Essentially if you imagine a series of building blocks and each building block is an SNP many people would say that if there is anything more than about a 25-building block difference in the sequence of gene information then you are moving away from this being a similar strain of bug. Again, this is an area for Mark but I would bring your attention the following paragraph towards the foot of paragraph 95 in the Overview Report, "It is likely that bacteria found in environmental locations may exist as multiple types and it may be best to say that whilst a demonstration of a close relationship between a patient specimen and an environmental isolate is strongly indicative of a relationship, the reverse does not necessarily apply". We tried to make the point in the report in a number of places that the absence of a proven connection does not eliminate the possibility that the connection is there.

122. Our main frustration was that it appeared to us that the testing carried out was somewhat hit or miss. We could not work out what the rationale behind the water-testing regime was. Samples were not taken from every tap once a month, for example. I do understand that there are substantial resource implications here but if NHS GGC believed they had a problem, which I am not sure they all did, they could have been much more systematic about applying these techniques.

123. As discussed on pages 95 and 96 of the report, whole genome sequencing was only carried out on three groups of isolates: *Enterobacter* spp., *Stenotrophomonas* spp. and *Cupriavidus* spp. We did not have any other typing data. My understanding is that this methodology is easier to carry out on some organisms than on others. There may be different clinical priorities for doing it under different circumstances. We were particularly interested in Stenotrophomonas for example and 84 genotypes had been done on it. 15 were isolates from patients in our review but there were also 10 other patients and 59 environmental strains. Five of the children in our series were not included and I do not know whether it was done for them and was not successful but an incomplete set of data limits your ability to draw conclusions from it.

124. I recognise that GGC placed a lot of emphasis upon the use of this typing showing that there was not a link with infections but again it comes back to earlier

discussions, merely because they did not demonstrate a link does not mean to say a link was not there, firstly because the environmental sampling was incomplete and secondly because not all the patients were typed.

125. I do not know why NHS GGC limited the isolates to just three groups. There may have been other bacteria that they were working on that were not relevant to our investigation. They may well have been looking at other bacteria for other reasons. They appear to have set greater stock on the work they did than we could understand.

126. GGC told us that they had identified definitively two infections, one *Cupriavidus* and the other was a *Mycobacterium* which was included in our review. Our position was that it was not the end of the story, because there were challenges in sampling did not mean that we could assume there were only two. I think we felt like we were being told by GGC that they had done this work and that there were only two environmentally associated cases of infection and therefore by inference the environment was not much of a problem. Our position was that we did not accept that the environment being a problem could be excluded as a possibility.

127. There were two cases of Cupriavidus (a relatively unusual organism) in the Case Note Review series and both infections occurred within 4 months of each other in separate patients on Ward 2A between September 2017 and January 2018. A subsequent report from HFS technical water investigations confirmed water testing positive for the bacterium from multiple outlets on the ward. Seven samples taken from the environment on Ward 2A were included in the GGC's WGS data on Cupriavidus, as was one sample from a patient. However, the environmental samples were taken between November 2019 and January 2020 (long after the infections in the two patients in our review) and the date of the patient sample (February 2018) did not match the date of the infections in the patients in our review. We therefore did not consider these results informative. Coincidentally, we were aware that there was a link established between a sample from a tap in the Aseptic Dispensing Unit in pharmacy with a Cupriavidus infection in a patient in 2016 but this patient was not part of our review and I do not know if this link was established by WGS.

128. GGC also indicated that they had established a relationship between a patient (included in our review) with Mycoplasma chelonae and samples from the water system. We identified the environment as the source of this patient's infection as 'Highly Probable' and made the following note in our data analysis: "there is close temporal relationship with isolates from water samples in Ward 6A although the IMT concluded that the exposure was with water supply out with Ward 6A. The organism was isolated from a swab at the site of the patient's central line, which had been the location of a sterile abscess arising 3 months earlier within 6 weeks of the line insertion in Theatre 6. Filters had not previously been fitted to taps in theatres at that time".

129. In summary, I felt we were being told by GGC that they had only identified links to the environment in 2 cases and that, on that basis, the likelihood of wider environmental causation was small. Our position remained that the data were limited and that the inadequacy of environmental sampling and the very limited number of samples from patients in the case note review subjected to WGS could not exclude this as a possibility in others.

130. With regard to NHS GGC's critique of our draft report, we made it clear we would review their comments, but we would not be issuing them with a written response. Overall, the submission received from them was a very significant piece of work. The document they sent us was 70 pages long and contained something like 28 embedded documents and, in the timescale that we had put aside for taking on board stakeholders' comments, it was difficult for us to respond to this. We felt under pressure to modify the nature of some of our comments and I did wonder whether the purpose of giving us this much feedback was to overwhelm us. Nevertheless, we took all their comments seriously and I generated a provisional response and shared it with my colleagues before we agreed if and where we were going to make changes to our report.

131. In response to a criticism by NHS GGC in respect of lines 868 onwards in our draft report, stating that species level clustering is not evidence of transmission, we clearly identified the other factors we included in making our assessment and we held it to be indirect evidence of transmission. With specific reference to the issue of bacterial typing technology, we considered that we had commented on

the typing information and the limitations of the data provided and, crucially, how such limitations relate to the sampling of the environment. We do not believe we dismissed the typing data, rather, we critiqued its value as evidence. Essentially we felt GGC were saying that just because you get infections that happen at the same time does not mean to say they are environmentally caused, we believed that the simultaneous/temporally linked incidence of cases of infection is one factor providing indirect evidence of a common environmental source. We believe that 'clustering' of similar infections in different patients provides one piece of evidence that you should consider in determining whether there could be an environmental origin.

Meeting with GGC Following the Draft Report

132. In terms of the letter from Jane Grant **[A35308833 – Letter from Jane Grant to Prof Stevens 1 March 2023 – Bundle 25, Page 151]**, the timeline went that we sent the draft report out to all stakeholders, including NHS GGC on 22 February 2021 and that this letter was generated on 1 March. We agreed to a meeting with them on 4 March and Jane Grant asked for a one-to-one phone call with me the day before the meeting. She wanted, perfectly reasonably, to agree how the meeting was to be handled and we agreed the process beforehand. I did however feel that I was under pressure in that call to adjust our report on the basis of her concerns.

133. We had the meeting and all three of us were present together with a number of individuals from NHS GGC. They made the case that we should change certain things in our report, but we gave no undertaking that we would although we agreed that we would consider what they had said. I received a second letter from Jane Grant on 5 March, I thought this slightly strangely, given we had met the day before. That letter said that our report was going to cause disquiet amongst patients and staff and asked if we could include in our report a statement of reassurance about the current situation, i.e. that we now thought the environment at NHS GGC was safe. I took advice from my colleagues and we agreed that it was not in our terms of reference to assess the current state of

the hospital. Indeed, we were in no position to do so as we had been reviewing retrospective data up to a period almost 18 months before that conversation took place.

134. I feel that there was a concerted effort to get us to see NHS GGC's point of view. They challenged us on certain points and had issued a very comprehensive rebuttal document to our draft report.

135. NHS GGC made the point that they were taking advice from various people throughout. My response was, however, that if you are confronting a problem which is not resolved after following standard advice or policy then you have a responsibility to think about things in a different way. I was not convinced that what they put forward was an adequate explanation. We ourselves came to the conclusion that there was a problem and it seemed to us that they had acted because they also thought there was a problem. In effect NHS GGC had acknowledged the problem: they had shut a ward, chlorinated the water supply and spent an awful lot of money on remediating the environment and yet were not collecting data that we thought they should have realised would have been helpful to monitor the outcome of those actions. Regardless of the advice they were getting, if they believed they had a problem then one would ask "why were they not thinking about the problem in a different way and why did they not adequately monitor the consequences of the changes they had made"

136. Regarding the Oversight Board report, I received a copy but I am not familiar with its content beyond that which related to the Case Note Review. I believe NHS GGC tried to respond to the report by suggesting there was no obvious difference in patterns of infection between GGC and the other paediatric oncology units in Scotland at Aberdeen and Edinburgh. We made a few observations in our report (section 8.2.3) about this, concluding that the October 2019 report from HPS on Paediatric Haemato-Oncology Data did not offer "any message of either reassurance or concern about past events". We also pointed out that the Glasgow unit was significantly larger than the other two. The Aberdeen haematology-oncology service, in particular, is so small that it would be very difficult to see patterns because of the likely frequency of events.

Limitations to the Review

137. I have been asked to what extent our findings are restricted by the limitations in the datasets with which we were provided. Our position is outlined in section 5.5.2 of the Overview Report which relates specifically to the water sampling. In terms of WGS I think the situation was more that NHS GGC seemed to feel that they had the data that essentially established that there was not a problem whereas we did not think that was the case. We certainly found the lack of data about environmental sampling frustrating. It might be asked "would you expect to get this information from other hospitals under routine circumstances?" to which my response would be that these were not routine circumstances and, once they had identified that there was a problem, they should have carried out their investigation of its cause in a much more systematic way.

138. There were other challenges in how the information as presented to us, particularly the information about maintenance work and facilities management. It was extraordinarily difficult to work with these data until they were revised. I know that NHS GGC felt that we did not ask for information until late in the process, but I believe we did ask for it early on, although we asked for more of it later. There was a considerable sense of dissatisfaction throughout the review process about the availability of information from NHS GGC, the way in which information was provided to us, and our ability to use the information given to us. I think if you are struggling to understand information as provided to you, you have to ask the question about whether it has been adequately presented.

139. I think the quality of data avaliable did influence our ability to reach findings. It was not just that data was missing (there is an example of this on page 86, Example 8.1) but it was also that we were not terribly confident about some of the information provided to us.

140. The illustration offered by Example 8.1 is a relatively simple issue, yet one which challenges the adequacy of GGC records. We read in IMT minutes (23 March 2018) that water samples were positive for Stenotrophomonas (the infection under consideration in the patient being discussed) yet although we

subsequently identified a positive water sample in the data provided by GGC, its location was not recorded. How then can we be sure that what was recorded and believed true by the IMT was in fact the case? Our reservations about the adequacy and presentation of data taken from environmental sampling and records of maintenance work undertaken in Wards 2A/2B/6A are discussed further in section 8.1.2 of our report.

141. We felt more confident with the information that we derived from clinical information and the temporal proximity of other infections of the same type. Chapter 5.6 deals with likelihood that infections were linked to the hospital. At the top of page 69 we have stated that the lack of any episodes being classified as definite reflects the tight criteria we believed that we needed to reach that point. We have stated, “Decisions at this level were influenced by the inconsistency with which our own data could be informed by data systematically investigating the microbiological environment, the water system and the likelihood that, by using typing methodologies, different bacterial isolates were linked. Microbiological information alone was insufficient to reach our conclusions, and we also looked carefully at clinically relevant information.”

142. Section 3.6.6 of our report may usefully illuminate what we were trying to articulate on page 69 as part of section 5.6. It shows, I hope, that we defined, at the outset, what we thought our criteria should be in agreeing the likelihood of environmental origin, and that we had given thought to the constraints on environmental sampling and genotyping. In the event, we were limited in what we could gain from such data and had to rely more on our interpretation of clinical circumstances and the clustering of similar infections.

143. In this context an example of clinically relevant information would be that a child who has been in hospital for several weeks and then develops an infection. Under those circumstances you can be very much more confident that you are not looking at the impact of the environment outside the hospital.

144. I think one needs to assess all elements of the ‘environment’ when considering risks of this nature and that even if some or all of the elements on which we commented in sections 5.2-5.5 had been more effective / informative, we would

still have taken a holistic view in delivering the conclusions offered in section 5.6.

145. We were very conscious throughout our work that we needed to avoid the criticism of having seen causation in correlation and, if anything, I think we often erred on the cautious side in reaching our conclusions for individual patients. Perhaps this illustrated by the fact that we felt unable to identify any patients as having a 'Definite' link to the environment, and scored only 3 as having a 'Strong Probable' link?

146. I have been asked to what extent was the CNR team's ability to answer the second of the four questions asked of the CNR team in its TOR (as set out on page 25 of the Overview Report to the end of section 2) impaired by the limitations in data retained and provided by NHS GGC in respect of maintenance of the built environment, cleaning and SICP, hand hygiene and environmental microbiological surveillance. I would argue that our work was significantly impacted by the limitations in data retained and provided by NHS GGC in respect of maintenance of the built environment, cleaning and SICP, hand hygiene and environmental microbiological surveillance.

Discussion of our Conclusions

147. In respect to whether it was possible to link each infection episode with the environment of the QEUH/RHC the 'most likely' group was a merging of those infections we had initially assessed as "strong possible," "probable" and "strong probable." It was a subjective judgement and we recognised it as a potential criticism of our work. We had stated (section 3.6.6 of the Overview Report) that we did not feel we were able to distinguish between 'probable' and 'weak probable'. We could have just used our original review scale categories of "unrelated," "possible," "probable," "definite," but we put in those intervening steps as we ultimately felt that the difference between "possible" and "probable" was too big a gap. Our final categorisation involved repeated discussion of each individual case with outcomes finely balanced according to the circumstances identified.

148. There were eight cases that were considered unrelated and that was because we felt the evidence was strong enough to exclude a link. If there had been insufficient evidence, they would have been in the unable to determine box. These were predominantly, if not exclusively, patients who had clear evidence of gut toxicity. Chemotherapy frequently damages the gut; it makes the mouth sore, and it can also damage bowel. Because the bowel is such a big repository of bacteria, once damaged the bowel becomes 'leaky' and bacteria can pass into the bloodstream very easily. So, where we saw children with positive blood cultures who were sick with bowel problems, typically as a result of chemotherapy, then we tended to say, "The likelihood is that this was an intrinsic infection that came from a damaged and leaking bowel." Therefore, under such circumstances we were as confident as we could be that that was not related to the environment.

149. At the end of section 8.4.1 on page 96 we have stated that NHS GGC told us it was possible to link the environment with infection using Whole Genome Sequencing in only two patients (one with *Cupriavidus* spp, who was not included in our review, and the other with *Mycobacterium chelonae*), but we never saw those data and on that basis did not record the *Mycobacterium chelonae* case as a definite but as a Strong Probable.

150. We may have seen the relevant WGS data on Cupriavidus as this formed one of the three groups of bacteria that were studied by GGC but we did not see the data on the patient with Mycobacterium chelonae – specifically, we found no reference to microbiological fingerprinting/genetic sequencing tests on any sample of M. Chelonae in the datasets provided to us by NHS GGC.

151. In expanding my view on the response of GCC to the issues identified within the paediatric haematology oncology patients, I would reflect on what we wrote in the last paragraph of section 10.2. This was an organisation which argued that there wasn't proof of an environmental link to patient infections yet took substantial measures to address the possibility that there was and then failed to establish systems to adequately monitor and collect data to assure the safety of the patients.

152. In relation to clustering we have commented at the end of section 4.3.5 that, "There is evidence for both increased frequency of specific gram-negative bacteraemia and episode clustering in time and place. Neither phenomenon proves that some of the bacteraemia had hospital environment sources, but the observations are consistent with this hypothesis." What this means is that SPC charts are perfectly respectable methodology, but they were we believed in this case probably inappropriately applied and that a much simpler approach, just looking at a timeline and looking visually at the clustering of infections could have been a much more useful tool in terms of suggesting that something was not quite right. We pointed out in paragraph 4.3.5 that some of these bacteria are quite common in this population of patients. However, for infections caused by bacteria such as enterobacterium Stenotrophomonas, they are relatively uncommon and to see those cases clustering in time and in space, because it is the same part of the environment, suggests that something is not quite right. So, we are saying this does not prove they had a hospital environment source, but the observation will be consistent that this was the cause.

153. In section 4.3.5 we are pressing the point that simple analysis of patterns of infection can be as important as what may be considered as more sophisticated methodologies. SPC charts have their place and offer a way of visually interpreting trends that may not be apparent when looking at basic data but they are also less sensitive when sample sizes / numbers of events are small. The HPS report in 2019 **[A33448012 – HPS Review of NHSGG&C paediatric haemato-oncology data October 2019 - Bundle 7, Page 214]** included various SPC charts and made this comment "The SPC charts included in this report describe that there has been instances of variation outside what would normally be expected in this patient population, the latest was a breach in the upper warning limit for Gram-negative blood culture episodes in September 2019. The characterisation of these cases alongside understanding in the context of environmental microbiology is critical to understanding and managing risk". In other words, one has to take a broad view and be sensitive to the possibility of a problem – how much better to prove that there isn't a problem by enhancing surveillance and monitoring further infections than to dismiss the possibility and be found to be wrong.

154. The point was made in the NHS GGC response to our draft report that "Clustering is not evidence of transmission events." And our response was, "No, it isn't, but it suggests it." It suggests it could be; it raises the question. My overriding thesis is how many hints did this team need to believe that something was not quite right, especially when they spent several million pounds on remedying something which they said was not a problem?

155. More typing would have helped but as important would have been the approach to environmental sampling. For example, if you have a cluster of Stenotrophomonas infections and you had systematic water sampling, and you picked up the Stenotrophomonas in the water during that period, then you could use typing to ascertain the closeness of the relationship. But even without the typing, you would have a very good shot at saying, "You have got Stenotrophomonas in the water at a time that you are having what appears to be a cluster of infections. How can you ignore the possibility that one caused the other?"

Meeting Dr Peters and Dr Inkster

156. We did have a meeting with Christine Peters and Teresa Inkster. We deliberately did not meet with them until right at the end of the process. We knew they had a lot to say and a lot of knowledge. We did not however, want it to seem that we had been unduly swayed by their views, until our work was complete. We did not meet them until end of January 2021.

157. In the second paragraph of page 67 we did report that we had been told that some key staff were denied access to water sampling/testing results despite multiple requests". Our source was Dr Peters, I think. She was concerned about the safety of the water system and the testing of water. Even after our report was complete and our work finished, she contacted me because she had been asking for access to data within GGC. She was told that the information was not available to her, and she had to get my permission as the lead for the Case Note Review to access it. This struck me as absolutely bizarre because this was shortly after we finished our work or as we were finishing it. I wrote to Jane Grant

and said I could not understand why access to information was blocked to somebody who works within the health board. So, we knew, first-hand, that access to certain information was restricted in some way and that Christine Peters had been trying to get access to water sample results and had not been allowed to have them.

Conclusion

158. I do not know whether NHS GGC followed any of our recommendations or not. I would have very much liked to have known what the status of their response had been. They did suggest that some of the things we had raised had already been fixed. The disappointment for me was that the Oversight Board appeared to change in function within a fairly rapid period of time after the submission of our report. I do not know whether any of the recommendations we made have been or will be fully implemented and it was not made clear to me who was going to monitor this. This did not seem to be a very robust conclusion to a substantial piece of work – we made over 40 recommendations but have never had a clear understanding of who was going to monitor the response or the implementation of any change.

Declaration

159. I believe that the facts stated in this witness statement are true. I understand that this statement may form part of the evidence before the Inquiry and be published on the Inquiry's website.

160. The witness was provided access to the following Scottish Hospital Inquiry bundles/documents for reference when they completed their questionnaire/ statement (Appendix A).

161. The witness introduced/provided the following documents to the Scottish Hospital Inquiry for reference when they completed their questionnaire/ statement (Appendix B).

Appendix A

A43293438 - Bundle 6 – Miscellaneous Documents

A49585984 - Bundle 25 - Case Note Review Expert Panel, Additional Reports and DMA Canyon

A43909077 - Bundle 7 – Written Reports prepared by Health Protection Scotland (HPS), Health Facilities Scotland (HFS) and Antimicrobial Resistance and Healthcare Associated Infection (ARHAI)

Appendix B

A43030605 – CV Professor Mike Stevens

Professor Michael Stevens

Curriculum Vitae for Scottish Hospitals Inquiry

March 2023

PERSONAL DETAILS

Full Name : Michael Charles Garston STEVENS

Date of Birth : [redacted]

Nationality : British

Contact details : [redacted]
Mobile: [redacted]
Email: [redacted]

UNDERGRADUATE EDUCATION

Undergraduate Studies University of London
St Mary's Hospital Medical School
(1968-1974, with external research year 1972-73)

Qualification: MB BS (with distinctions) (1974)

POST GRADUATE QUALIFICATIONS

MRCP (UK) 1976

Accreditation in Paediatrics (Joint Committee on Higher Medical Training) 1982.

MD, University of London, 1983.

Accreditation in Paediatric Oncology (Joint Committee on Higher Medical Training) 1986.

FRCP (London) 1992

FRCPCH 1997

FRCR 2003

CURRENT POSITION:

Emeritus Professor of Paediatric Oncology, University of Bristol

GMC Registration: [redacted] – Voluntary erasure from Register May 2022

Medical Defence Union Membership: [redacted] – Inactive membership

EMPLOYMENT HISTORY (most recent first)

July 2021	Retired
January 2020 July 2021	Lead, Independent Expert Panel, Queen Elizabeth University Hospital /Royal Hospital for Children, NHS Greater Glasgow and Clyde Case Note Review for QEUH/NHSGGC Oversight Board
September 2001 - September 2021	Consultant Paediatric Oncologist, University Hospitals Bristol NHS Foundation Trust / Bristol Royal Hospital for Children (part time from March 2015)
September 2001 - March 2015	CLIC Professor of Paediatric Oncology, University of Bristol (& then Emeritus Professor)
December 1985 - August 2001	Consultant Paediatric Oncologist Birmingham Children's Hospital NHS Trust
June 1985 - November 1985	Member of Staff, Division of Haematology/Oncology, The Hospital for Sick Children, Toronto, Canada Assistant Professor, Department of Paediatrics, University of Toronto.
September 1984 - June 1985	Terry Fox Clinical Training Fellowship Division of Haematology/Oncology, The Hospital for Sick Children, Toronto, Canada.
January 1984 - September 1984	MRC Clinical Research Fellowship, MRC Laboratories, University of the West Indies, Kingston, Jamaica.
January 1983 - December 1983	Clinical Fellow, Division of Haematology/Oncology, The Hospital for Sick Children, Toronto, Canada.
September 1980 - December 1982	Senior Registrar, Department of Paediatrics, John Radcliffe Hospital, Oxford.
October 1978 - September 1980	MRC Clinical Research Fellowship MRC Laboratories, University of the West Indies, Kingston, Jamaica.
October 1977 - October 1978	Tutor (Honorary Registrar), Department of Child Health University of Manchester.
August 1977 - October 1977	Locum Registrar, Paediatrics, Radcliffe Infirmary, Oxford.
January 1997 - July 1977	Senior House Officer, Neonatal Paediatrics, John Radcliffe Hospital, Oxford.
August 1976 - January 1977	Senior House Officer, General Paediatrics, Radcliffe Infirmary, Oxford.
August 1975 -	Senior House Officer, General Medicine,

July 1976	Radcliffe Infirmary, Oxford.
January 1975 - July 1975	House Surgeon, General and Paediatric Surgery, St Charles Hospital, London, W10.
June 1974 - December 1974	House Physician, General Medicine, St Mary's Hospital, London, W2.

August 1972 - July 1973	Wellcome Trust Undergraduate Research Fellowship, Tropical Metabolism Research Unit, University of the West Indies, Kingston, Jamaica.

SIGNIFICANT NHS CLINICAL MANAGEMENT & LEADERSHIP POSITIONS

Birmingham Children's Hospital

- Clinical Director, Haematology & Oncology (1990 – 1998).
- Medical Director (1998 – 2000)

University Hospitals Bristol NHS Foundation Trust / Bristol Royal Hospital for Children

- Chair, Paediatric R&D Group (2002 - 2005)
- Lead Clinician, Paediatric Haematology Oncology & BMT (2006 –2008)
- Chair, Advisory Group for Teenage & Young Adult Cancer (2008 – 2009)
- Trust Interim Director of Research & Development (2009 –2010)

NHS South West

- Chair, South West England Children's and Young Persons Cancer Services Group (2002 – 2003, 2005 – 2009)

UK ACADEMIC MANAGEMENT & LEADERSHIP POSITIONS

Birmingham Children's Hospital

- Co-Director, West Midlands Regional Children's Tumour Research Group, (1986 -1999).

University of Bristol

- Director, South West Children's Cancer Research Registry (2001 – 2020)
- Head, Academic Unit of Child Health, Faculty of Medicine and Dentistry (2002 – 2005)
- Director, Institute of Child Life and Health, Faculty of Medicine and Dentistry (2004 – 2008)
- Chair, Medical Education Committee, Faculty of Medicine and Dentistry (2005 –2011)
- Chair, Bristol Cancer Research Strategy group (2008 – 2011)

NATIONAL PROFESSIONAL & ACADEMIC LEADERSHIP POSITIONS

UK Children's Cancer and Leukaemia Group

- Group Chair (1997 – 2000 & 2008-2011).
- Sub Group Chair: Soft Tissue Sarcoma Group (1989 – 1994); Late Effects Group (1993 – 1996); Education and Training Committee (1994 – 1998); Epidemiology and Registry Group (2002 – 2015); Clinical Research Governance Group (2003 - 2005)
- Cancer Research UK appointed lead for CCLG national reconfiguration project (2008-2011)

UK Commission on Human Medicines

- Member, Haematology & Oncology Expert Advisory Group (2006 - 2009)

- Member & Vice Chair, Paediatric Medicines Expert Advisory Group (2007 - 2013)

Medical Research Council

- Member, Leukaemia Clinical Trials Data Monitoring and Ethics Committee (2003 - 2014)
- Chair, UKALL R3 Data Monitoring Committee (International clinical trial for children with relapsed leukaemia), (2003 – 2013)

NHS / NHS England

- Cancer Referral Guidelines Steering Group (1999 – 2002)
- National Cancer Action Team Advisory Group for the Implementation of NICE Guidance on Improving Outcomes for Children and Young People with Cancer (2005 - 2012)
- NHS Information Authority. National Cancer Dataset Steering Group (2000 – 2008)
- National Cancer Intelligence Network: Chair, Children Teenage & Young Person's Clinical Reference Group (2008 – 2013)
- NHS Improvement / National Cancer Survivorship Initiative: Children's & Young People Workstream Steering Group (2010 – 2013)
- NHS Improvement / NHS Improving Quality: National Clinical Adviser CYP Cancer Survivorship & Transition (2012 - 2014)
- NIHR Cancer & Nutrition Collaboration: Executive Committee & Chair CTYA Working Group

INTERNATIONAL PROFESSIONAL & ACADEMIC LEADERSHIP POSITIONS

European Paediatric Soft Tissue Sarcoma Group

- Chair (2000 – 2013)

European Cancer Organisation

- Board Member (2002 – 2008), Treasurer (2003 – 2008)

International Society of Paediatric Oncology

- Chair, Soft Tissue Sarcoma Studies Committee (1993 – 2005)
- European President (2000 - 2003)

UK EXPERT ADVISORY APPOINTMENTS

- Chair, London Paediatric Oncology Review. NHS England (2014 – 2015).
- Chair, Independent Expert Panel for Case Note Review. Queen Elizabeth University Hospital / Royal Hospital for Children, NHS Greater Glasgow and Clyde. January 2020 – June 2021.

INTERNATIONAL EXPERT ADVISORY APPOINTMENTS

External Reviews

- External Reviewer, Department of Paediatric Oncology, Institut Gustave Roussy, France: (1996).
- Report on national provision of children's cancer services in New Zealand. Government of New Zealand: (1999).
- External reviewer, Department of Paediatric Haematology & Oncology, University of Toronto and Hospital for Sick Children, Toronto: (2009)

Academic Appointments

- Jordan University of Science & Technology: External Examiner (2011-2012)

- External assessor for academic promotions at: University of Otago, New Zealand (2009); University of Toronto, Canada (2009); St Jude Children's Research Hospital, USA (2011); University of Florida, USA (2013); Israel Institute of Technology (2014); University of Leeds (2019)

SCIENTIFIC ADVISORY & ACADEMIC STEERING COMMITTEES

United Kingdom

- University of Birmingham, Department of Epidemiology. Steering Group for British Childhood Cancer Survivors Study (BCCSS) (1998 – 2013). Funded by Cancer Research UK.
- University of York, Social Policy Research Unit. Steering Group for project "Care and Support Needs of Children with Cancer and Leukaemia and their Families" (2002 – 2004). Funded by Cancer & Leukaemia in Childhood (CLIC).
- University of York, Epidemiology and Genetics Unit. Oversight Committee for the United Kingdom Children's Cancer Study (UKCCS) (2006 - 2009). Principal funding by the Leukaemia Research Fund
- University of Oxford, Department of Paediatrics. Scientific Advisory Committee for the Childhood Cancer Research Group (CCRG) (2006 – 2013; Chair from 2009).
- NIHR Medicines for Children Research Network, South West, Steering Group (2007 - 2011)
- University of the West of England, School of Psychology. Steering Committee for the Centre for Appearance Research (2008 - 2015)

International

- Cochrane Childhood Cancer Group (Amsterdam, The Netherlands). Member of Editorial Board (2006 – 2015)
- European School of Oncology (Milan, Italy). Scientific Committee. (2007 – 2014)
- Chair, Independent Data Monitoring Committee for Paediatric Hepatic International Tumour Trial (PHITT) (2017 - current)

EDITORIAL & OTHER ACADEMIC ACTIVITIES

Grant reviewer – including:

- Auckland Medical Research Foundation, New Zealand; Cancer Research UK; Cancer Research Trust New Zealand; NIHR Research for Patient Benefit programme; NIHR Senior Research Fellowship Programme; ODAS Foundation, The Netherlands)

Journal Reviewer - including:

- Annals of Oncology; Blood; Cancer; European Journal of Cancer; JAMA Paediatrics; Journal of Clinical Oncology; Lancet; New England Journal of Medicine; Pediatric Blood and Cancer; PLOS One.

Editorial Boards

- Member, Editorial Board, Medical and Pediatric Oncology (now Paediatric Blood & Cancer, 2001 - 2003)
- Paediatric Editor, European Journal of Cancer (2005 - 2010)

RESEARCH SUPERVISION

Post Graduate Degree Supervision :

- 1987 – 1989 Dr D Smith. Nutritional status of children with malignant disease. MD, University of Cambridge (Awarded 1991).

- 1989 - 1990 Ms J Lees. Eating behaviour of children with cancer. MSc (Clinical Psychology), University of Birmingham (Awarded 1991).
- 1990 - 1991 Ms C Evans. Adaptation behaviour of siblings of children with cancer. MSc (Clinical Psychology), University of Birmingham (Awarded 1992).
- 1995 - 1998 Dr H Traunecker. The biology of drug resistance in human sarcoma cell lines following a brief exposure to doxorubicin. PhD, University of Birmingham. (Awarded 1999)
- 1999 – 2001 Dr H Jenkinson. The epidemiology and molecular genetic basis for second malignancy after treatment for cancer in childhood. PhD University of Birmingham. (Awarded 2002).
- 2002 -2006 Dr ML Yeap. Growth and metabolic consequences of treatment for childhood leukaemia. MD, University of Bristol (Awarded 2009)
- 2002 – 2006 Dr A Penn. Health related quality of life after diagnosis of childhood brain tumour. PhD, University of Witwatersrand, Johannesburg, South Africa (Awarded 2013)
- 2007 – 2009 Dr N Davis. Cardiac and metabolic risk factors after bone marrow transplantation for childhood leukaemia. PhD, University of Bristol (Awarded 2013)
- 2010 – 2014 Dr C Wei. Mechanism of Impaired Glucose Tolerance in Survivors of Childhood Leukaemia treated with and without Bone Marrow Transplantation. MD, University of Bristol (Awarded 2014)

External Examiner For Postgraduate Degrees :

- University of Newcastle upon Tyne, 1995. (PhD Examiner).
- University of Leeds, 1997. (MD Examiner).
- University of Liverpool, 1998. (PhD Examiner).
- University of London, 1998 (PhD Examiner)
- University of Amsterdam, 2004 (PhD Examiner)
- University of Birmingham, 2008 (PhD Examiner)
- University of Oxford, 2010 (DPhil Examiner)
- University of Leeds, 2013 (MD Examiner)
- University of Amsterdam, 2015 (MD Examiner)

TEACHING LEADERSHIP

Undergraduate education

- Chair, Medical Education Committee (Oversight of MB ChB Programme: Governance & Standards), University of Bristol (2005 – 2011)

Postgraduate medical education

- Faculty, European School of Oncology / International Society of Paediatric Oncology postgraduate courses (1996 x2, 1997, 2003, 2004)
- Faculty, FECS / AACR / ASCO International Workshop on Methods in Clinical Cancer Research, Switzerland (2002, 2003, 2004)
- Co-Founder & Chair of European School of Oncology / SIOP Europe Master Class in Paediatric Oncology held in: Orta, Italy (2006); Ascona, Switzerland (2008); Castel Gandolfo, Italy (2010); Castel Gandolofo, Italy (2012); and Lujbljana, Slovenia (2014)
- Faculty, University of Amsterdam School of Paediatric Oncology, residential Master Classes (2007 & 2009)

WORK WITH CHARITABLE ORGANISATIONS

Trusteeships

- Society of Parents of Children with Cancer (West Midlands Regional Parents Support Group), (1989 – 2001).
- Trustee, Joshua Gilbert Rhabdomyosarcoma Appeal (national charity for research in rhabdomyosarcoma), (1995 – 1997).
- Trustee, Lisa Thaxter Trust (national charity for childhood cancer support and research), (1996 – 2006).

AWARDS:

1974 – Distinctions in Surgery and in Obstetrics & Gynaecology, Final MB BS, University of London

1981 - Golden Stethoscope Award – annual award from the clinical medical students at Oxford University for their most revered teacher

2001 - NACCPO Award from the National Alliance of Children's Cancer Parents Organisations, for services to children with cancer and their families

2017 – Macmillan Cancer Support – National Innovation Excellence Award

PUBLICATIONS

Book Editorship

Cancer in Children: Clinical Management. Oxford University Press
5th Edition. (Eds. Voute PA, Barrett A, Stevens MCG, Caron HN. (2005)
6th Edition. (Eds. Stevens MCG, Caron HN, Biondi A) (2012)

Book Chapters (n= 12)

Major themes: undergraduate medical education; rhabdomyosarcoma and other soft tissue sarcomas of childhood; late effects of childhood cancer treatment

Peer Reviewed Journal Publications (n >150)

Major themes: children's blood disease and cancer including work on: sickle cell anaemia; soft tissue sarcoma; brain tumours; patterns of cancer survival; late consequences of treatment; nutrition; time to diagnosis