Scottish Hospitals Inquiry
Witness Statement of
Professor Mark Wilcox

Professional Background

1 I am Professor Mark Wilcox. I am Professor of Medical Microbiology at the University of Leeds where I hold the Sir Edward Brotherton Chair of Bacteriology and a consultant microbiologist at Leeds Teaching Hospitals NHS Trust. Since 2020 I have held the post of National Clinical Director Antimicrobial Resistance and Infection Prevention & Control at NHS England. I currently work three days a week. Two of those three days are at NHS England with the remaining day split between my university role where I run a research team focusing on healthcare associated infection and my role at the Leeds Teaching Hospitals Trust which is a research post. I am the lead for Clostridioides difficile infection for what was Public Health England and is now the UK Health Security Agency. My research interests are multiple aspects of Clostridium difficile infection, diagnostics, antibiotic resistance and the gut microbiome, staphylococcal infection, and the clinical development of new antimicrobial agents.

2 Between 1981 and 1986 I attended Nottingham University Medical School, where I gained a Bachelor of Medicine with First Class Honours Class in 1984 and a Bachelor of Medicine and Bachelor Surgery with Honours in 1986. I also have post-graduate qualifications, gaining Doctor of Medicine (Microbiology) in 1990 and FRCPath (Microbiology) in 1992. In 2024, I was awarded ab OBE for services to Infection Prevention and Control, primarily during the COVID-19 pandemic.

3 I have held multiple senior posts within Microbiology, both in Leeds and nationally, including:

1993-95	Consultant Microbiologist, Addenbrookes' Hospital Cambridge.
1995	Senior Lecturer & Consultant Microbiologist, Department of Microbiology, University of Leeds & Leeds Teaching Hospitals Trust (LTHT).
2001-16	Head of Microbiology for LTHT.
2003-08	Director of Infection Prevention & Control/Lead Infection Control Doctor for LTHT.
2004	Professor of Medical Microbiology (University of Leeds).
2005-16	Head of Unified Clinical Microbiology Service for Leeds, including former Leeds HPA diagnostic services.
2006-12	Clinical Director of Pathology for LTHT.
2007	Established and led *C. difficile* Ribotyping Network for England (CDRN, including N. Ireland).
2008	Lead on *C. difficile* for Health Protection Agency (HPA) in England (now Public Health England).
2011	Expert Advisor on Healthcare Associated Infection to the Department of Health (England).
2016	Head of Microbiology Research & Development, LTHT.
2017	Medical Advisor to National Infection Prevention & Control Lead (NHS Improvement), England.
2018	Infection Lead of the Leeds NIHR Diagnostic Technologies Medical Technology and In Vitro Diagnostic Co-operative at LTHT.
2022	Lead of Antimicrobial Resistance and Infection theme of Leeds NIHR Biomedical Research Centre.

4 My main area of interest from a clinical and academic perspective is infection prevention and control and healthcare associated infections, which is the discipline of trying to minimise infection risk for patients within a healthcare setting. Within infection prevention and control clinicians can hold various levels of responsibility, such as an Infection Control Doctor and Director of Infection Prevention and Control, and I have held the full range of such roles

during my career. Prior to the pandemic I was the medical advisor to the Chief Nurse in England and that metamorphosised into becoming National Clinical Director for Infection Prevention and Control and Antimicrobial Resistance. During the pandemic, Sir Patrick Vallance invited me to chair one of the subgroups of Scientific Advisory Group for Emergencies (SAGE) on minimising the risk of infection and acquisition of COVID in hospital; this role meant that I joined SAGE as well. I was a member of SAGE from May 2020 until its dissolution.

5 I have attached at Annex B a resume outlining my National and International responsibilities where I have worked or taken the lead on a number of committees and advisory boards over the years.

6 I have never worked for NHS Greater Glasgow and Clyde (NHS GGC), as a consultant, and this statement relates to my contribution as part of the Case Note Review (CNR) team, which was established in January 2020 to examine individual cases of infection at the Queen Elizabeth University Hospital (QEUH). As part of the CNR Expert Panel, I examined the case notes of those children and young people in the paediatric haemato-oncology service in the RHC and the QEUH from 2015 to 2019 who had a bacteraemia caused by a Gram-negative environmental microorganism (and selected other bacteria, as identified in laboratory tests).

7 I have prepared this statement on the basis that reader has read the Case Note Review Overview Report, March 2021 (“the Overview Report”) 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.

8 My recollection is that I was first contacted about the Case Note Review in January 2020. I cannot now recall if the emails were preceded by phone calls. I was contacted via email and asked if I would be willing to join a panel of independent experts for a case note review and provide infection and prevention control advice, specifically medical microbiology aspects of advice. I am not sure of the process for my selection to the Expert Panel and I was

not interviewed. I was given the contact names of some individuals who I could talk to and get an idea of what was involved, as I did not know anything about NHS GGC and what had gone on. I spoke to a couple of individuals by phone or over email, with Lesley Shepherd being one of them, to understand what was involved, because it was obviously an unusual scenario, but I was not aware of there being a formal process of me being vetted. It never felt like that at any stage.

9 The Expert Panel of the CNR comprised me, Professor Mike Stevens (panel lead) and Gaynor Evans. Within the CNR team there was also a core clinical team. They were Professor Peter Davey, who was involved in acquiring data and discussing aspects of our findings and Professor Marion Bain, a director of infection and prevention control, who was the link between the panel and NHS GGC. There were other individuals contributing, but the Expert Panel was just the three of us.

10 The respective roles of the Expert Panel and various support teams are described in the CNR Terms of Reference. The Expert Panel was responsible for a) agreeing, within the scope of these Terms of Reference, the definitions used to select patients for the review, the scope and direction of the data collection and the methodological tools required; b) overseeing and interpreting the analysis of data obtained and developing the Final Report (and, in discussion with Professor Bain, the provision of any agreed interim reporting);c) progress reporting to relevant audiences, including the RHC/QEUH staff; and d) providing reporting to individual patients and families. It should be noted that despite the existence of support teams, the conclusions in the CNR report are those of the expert panel.

11 The Clinical Team was responsible for: a) undertaking the data collection, storage and submission of case note review material to the Expert Panel; b) resolving data/sampling issues with Professor Bain, the Support Team and the Expert Panel; and c) supporting the analysis and reporting of the Case Note Review through the Expert Panel.

12 The Support Team was responsible for: a) resolving practicalities and resourcing issues with Professor Bain, Professor Stevens and Dr O'Connor; b) undertaking key communication and engagement functions with Professor Bain; c) developing and maintaining the Review workplan; e) providing secretariat and related functions to the Expert Panel, and f) ensuring submission of Final Report to the Cabinet Secretary and publication.

13 There was also a specialised PTT (Paediatric Trigger Tool) Team. The intention of using an adapted Paediatric Trigger Tool in the study of NHS GGC is not to determine preventable or non-preventable harm but to create opportunities to learn from the triggers and adverse events identified. It forms only part of the overarching case review process, and it is anticipated the information from the PTT will underpin the epidemiological and clinical outcome review and the contextual organisational data and reports. The PTT methodology will examine harm in the processes of healthcare in the group of patients selected for case note review and its objectives are to contribute to the overall aim of the case note review by identifying all triggers and adverse events in the cohort of patients identified by the epidemiological review using an adapted PTT; and by describing the rate and severity of harm occurring in hospitalised children in the cohort group.

14 Professor Bain was the Expert Panel's link to NHS GGC. Her role is also set out in the Terms of Reference for the CNR: 'As Executive Lead for infection prevention and control within NHS GGS, as appointed by Professor McQueen, Professor Bain will have oversight of the project as a whole. She will be responsible for its progress and reporting to Professor McQueen, including advice – provided by the Expert Panel and other members of the team below – for any necessary change in key elements of these Terms of Reference.' She was also responsible for communications with patients/families, along with Professor Craig White.

15 The Oversight Board provided professional oversight, holding the core team to account for progress and delivery, together with identifying risk and resolving any problems with sampling or data. The Board was helpful in addressing some of the obstacles we encountered in obtaining data/information. The Oversight Board did not influence the conclusions of the CNR team. Instead, it oversaw our progress in obtaining and reviewing the data necessary to make our assessments and draw our conclusions. The Expert Panel was responsible for providing a Final Report to Professor Bain and the Oversight Board.

CNR and Terms of Reference (ToR)

16 Prior to the commencement of the CNR the panel was provided with an outline of the case criteria and case definitions. The outline case criteria and definitions were sent to us by Philip Raines or Jim Dryden (QEUH Support Unit, Scottish Government), and were set out in an 'Epidemiological Protocol' (February 2020 v0.1; Health Protection Scotland & NHS National Services Scotland). We made some very minor modifications to tighten those up, so they were logical and that they were explained in the report with nothing else to add. We had access to other reports that outlined some other epidemiology of wider bacteraemia, such as Gram-positive organisms. That, for me, underscored the sense in focusing on the so-called environmental Gram-negative bacteraemia. By "so-called" environmental Gram-negative bacteria I mean GNBs which can be found in or recovered from various sites in the environment and can survive well outside animal or human habitats.

17 I have been asked by the Inquiry if the CNR team considered the possibility of taking account of positive blood cultures from other parts of the hospital, which might have been relevant to a more extensive pattern of concern outwith the Schiehallion unit. We discussed this, but our remit concerned this particular group of vulnerable individuals. To my knowledge, there were no similar potential signals of an environmental source elsewhere. Given the nature of these individuals, they are hyper susceptible. The air we breathe has

fungal spores in it, and that is why some bread will turn green eventually, because the fungal spores land on the bread. The fungal spores that we breathe in cause us no harm whatsoever; however, someone with a very depleted immunity, such as bone marrow transplant patients, who breathe in exactly the same air as us can develop a fatal infection. So, they amplify, if you like, bacteria that in this case may well cause no harm in other individuals. To look for potential environmental sources of infection, it was logical to understand the patterns and timings of the clusters of bacteraemias and the types of bacteria involved.

18 I have been asked to explain how we reached the conclusion that there were no potential signals of an environmental source elsewhere. This was my assumption and was formulated from what we were told initially and the focus on bacteraemias caused by Gram-negative environmental bacteria. We were not, to the best of my recollection, given assurances regarding the absence of unusual potentially hospital associated infections outside the Schiehallion Unit patient cohort. I do not recall being made aware of ‘a series of up to six Cryptococcus cases found in patients in the hospital between 2018 and 2020’. Had this occurred, I would have considered such a series to be unrelated, in terms of specific source(s), to the bacteraemias caused by Gram-negative environmental bacteria. This is because the habitats and mode of transmission of Cryptococcus differ from those associated with Gram-negative environmental bacteria.

19 Our remit was limited to Schiehallion patients. Immunocompromised patients are more susceptible to developing infections caused by certain bacteria (including Gram-negative environmental bacteria). Thus, our remit to focus on these patients and these bacteria was logical. We did briefly discuss the merits of expanding beyond these, but we stuck to our original brief.

20 I have been asked by the Inquiry if I could provide a layman’s explanation of the case definition as outlined at Section 3.2.2 in the Overview Report. The focus here was on potentially environmental Gram-negative bacteria, which are split into two broad groups based on a simple staining technique. They

either retain the stain, in which case they are Gram-positive, or they lose the stain when challenged, in which case they are Gram-negative, Gram being the surname of the bacteriologist who devised this technique. The reason bacteria are either Gram-positive or Gram-negative is because of the fundamental difference in the outer coating of the bacteria. That outer coating and the other characteristics of the bacteria fit in with this Gram stain property. If the bacteria are Gram-negative, they are more likely to thrive in wet, moist conditions as opposed to Gram-positive bacteria, which will survive happily in dry, arid conditions. So, the CNR was focusing on Gram-negatives, which needed to have been recovered from blood cultures. They are detected by a conventional technique where you heat the blood, you incubate at 37 degrees centigrade and see whether you manage to amplify the small numbers of bacteria in a blood sample to larger numbers, which create a positive signal in that blood culture. We wanted a deduplication process. If someone tested positive today and then tomorrow and maybe in 10 days' time, you would count that really as one episode. So, it is a persistent bacteraemia, and it is one episode rather than counting that as three.

21 We used a 14-day period to deduplicate the records for the CNR, which means that any bacteraemia occurring within 14 days were counted as one episode. Outside of that, if I get bug X today, and then in four weeks' time bug X again, that is two different bacteraemia, because you should have really cleared that bug from your blood. If it returns, it could be the same episode, but it also equally could be you that could have reacquired it. That is the basis on which we were selecting cases, Gram-negative potential environmental source bacteria in blood cultures causing so-called bacteraemia deduplicated. We set some exceptions; the most obvious ones were that post-mortem samples were excluded. They are not commonly taken, and they can be misleading. We did not want contaminations or samples from other sources such as faecal samples.

Infections and Association

22 I have been asked by the Inquiry what is meant by “association” when looking at infections and causation. In the Overview Report we did not define, using our criteria, any definite relationships between the environment and bacteraemia in children, but a relatively large number of probables, the highly probables, highly possibles and so on. The term “association” is qualified by those grounds of probability, and, in infection prevention and control terms, the key here is any clustering in time and place. The more clusters of cases that you get that appear similar, or the more cases in a cluster that appear similar, in this case caused by the same bug, in time over a number of years, maybe a few weeks or months, and in the same place, then the greater the likelihood that the association is real and that there is potentially a common cause. We were looking to see whether there was a pattern of infection that could be associated with the environment, rather than establishing whether infection was caused by the environment. Our aim was to determine a timeline for each of the cases; characterise the cases in terms of time, place and person; and – where possible- to describe the cases in the context of environmental risks and incidents.

Infections and the Environment

23 I have been asked by the Inquiry if the CNR team were looking to establish whether an infection was caused by the environment, or whether there was a pattern of infection that could be associated with the environment. I have been involved in numerous roles, at both local and national level, attempting to establish links and associations and causality in the chain of infection, and looking at both of these areas. My initial approach will be to look for evidence of, let us say, clustering in time and place; these are the cardinal features, which suggest an association. Then one looks for causality, because of course if there is causality, it could be in one of two directions. If you find a bug in a sink, and you find a bug in a patient, you may conclude that they are the same bug; however, you need to establish did the patient put the bug in the sink, or did the bug come from the sink? So, the likelihood that it is sink to

patient increases the greater the size of the cluster in time and place sharing that association.

24 The organisms that we were looking at are generally water-associated or can be water-associated. It does not mean you only find them in water. You can also find them inside people, particularly as part of the bacteria forming the gut, the gut microbiome, or gut bacteria. In the CNR report, we refer to endogenous infections, which are bugs already in or on a person that then go somewhere else within that person and cause an infection - in this case, into the bloodstream. By contrast exogenous infections is where a person may acquire the same or similar bugs from other sources. It was those exogenous possibilities that we were particularly focused on, while not ignoring the chance that they could be endogenous infection as well. The chances of a cluster of cases all caused by the same or similar bug occurring in time and place endogenously decreases, and you start to think, “Well, hold on, it’s more likely to be exogenous,” and we refer to that time and again within the CNR findings.

25 There are many potential sources of bacteria or viruses in the environment, the air we breathe, things we touch, and water sources are probably three of the most common. The hands of patients and/or healthcare workers, or visitors and friends, can act as links between those potential sources or “reservoirs” of infections. These organisms would not normally be thought of as airborne organisms. They MAY splatter from droplets, but they are not airborne in the way that a respiratory virus such as COVID-19 or influenza is transmitted. As part of the review, we focused on primarily water sources or intermediary surfaces. In this context, the “environment” here refers to the physical, biological, and chemical factors external to the patients.

26 I have been asked by the Inquiry how the supporting teams approached the analysis of causation, including identifying causal criteria. The extended team did not play a key part there. The key decision-making around causality was between the three of us in the panel. It did not mean that they could not provide input by identifying information that we might not be aware of or

adding context via records that we had not seen. The actual decisions of something being possible, likely or this is highly probable, was the responsibility of the three of us. In terms of the definitions themselves, they contributed to those definitions, but the final decision about the acceptability and robustness of the definitions was that of the Panel.

Use of the Paediatric Trigger Tool (PTT)

27 As highlighted in the Overview Report at section 3.4.2 a trigger tool is a method for identifying adverse events (AE). In adults, the rate of detection of AE with a trigger tool is typically ten-fold greater than the rate detected through spontaneous reporting systems. Similar results have been reported with paediatric trigger tools in general wards and neonatal intensive care units.

28 In 2014, the UK PTT was developed with the support of clinicians in nine hospitals across the UK in order to detect AE in paediatric care provided in district general hospitals, acute teaching hospitals and specialist paediatric centres. The PTT was used for the CNR not to determine preventable or non-preventable harm, but to create opportunities to learn from the various AE identified.

29 I have been asked by the Inquiry the significance of the paediatric trigger tool, which is referred to multiple times within the CNR report. I think it is a confusing concept, and I had never come across it prior to this review. It is a system simply for determining whether there has been an actual or potential adverse event and seeks to establish what has been the impact of that event. It is a tool that was devised around the care of children specifically, so whilst I have treated children throughout my career, it has not been a particular focus of mine, which is probably why I have not come across it.

30 There are other ways of assessing whether there has been an adverse event, such as for example in Datix reporting. The Datix system is a widely used system in the NHS regardless of which country you are in and whether the people affected are young or old. It was suggested from the outset that the PTT could be a useful way of assessing adverse events. There is a component in the report, where we talk about the impact of these bacteraemia seen as adverse events. The tool was useful there, but we could have used other tools. I recall some discussion early on about concerns being raised by NHS GGC over the use of the paediatric trigger tool. I cannot remember the basis for those concerns; however, I do not think it really matters whether we used the PTT or some other tools/methods because what we were doing was trying to ascribe a likelihood to the environment being the source of a bacteraemia.

31 Within the CNR report we did say, “regardless of whether there was a likelihood, what was the impact?” with particular interest where we thought there was an environmental source. If you follow that train of thought, the utility of the paediatric trigger tool is not core to our purpose. It is secondary to that; however, I am not saying it did not have some value.

Methodology

32 I have been asked by the Inquiry how the methodology evolved during the CNR, and whether the decision was a done collectively or did someone take the lead. It was very much a collective decision. Gaynor and I have more relevant closer skill sets than Mike did as he was a lifelong paediatrician with an interest in infection, whereas Gaynor and I are infection specialists. Our relative skills were quite complementary in terms of defining what we believe was a case, defining the terms of our investigation and what we felt was reasonable or unreasonable to attempt to do and what we did not think was reasonable to attempt. I do not recall there being any fundamental disagreement or challenge of what we decided.

33 There is no set methodology that one must use to investigate a putative series of transmission events, but there are a certain series of core principles. I have already alluded to time and place, cluster analysis. I could go on about the detailed typing and fingerprinting attempts, and the drawbacks with those or the surveillance in general about the robustness of the investigation tools that we used. I do not mean paediatric trigger tool; I mean the actual incident management team meetings and processes and so on. There are different ways of investigating an outbreak or a series of outbreaks transmission events, but the core principles remain the same. We were confident that we used those core principles appropriately.

34 I have been asked by the Inquiry if the overall analysis is based on circumstantial evidence, defined as a combination of circumstances that are more likely or not to point to an infection episode having been caused by the built environment. In the absence of definitive proof- which in this case would be provided by bacterial typing or fingerprinting methods that matched, as close to certainty as possible, a bug in water with a bug in patient's blood - we were left with making decisions based on grounds of probability. We were using facts on where bacteria were found, how frequently they were found both in patients and in environmental samples, and then making judgments based on how complete investigations were. From my lay understanding of the term "circumstantial," I think I would agree with that, but underlying the decisions we made about the balance of probabilities was a considerable degree of expertise in the likelihood of association and indeed causality.

35 Whole genome sequencing is one of multiple methods that can be used to fingerprint a microbe, in this case according to the make-up of its nucleic acid (DNA or RNA). Typing methods, in addition, assign a type (e.g. name or number) to the output of the fingerprinting method. If it were simply looking at 84 patients and 112/118 bloodstream infection/ bacteraemia episodes all in isolation, it would be extremely difficult to have any confidence in saying what the source of that individual bacteraemia was. If, however, you note that there have been eight bacteraemias caused by an organism with the same species name, characterised to species level, within a handful of months, then either

side of that handful of months no more such bacteraemias, and these patients are all situated and managed in the same place, same ward, then the grounds of probability are very much in favour that there is a common source there. So, yes, it can be circumstantial if we then say that we believe, for other reasons as well, that the environment is a likely source. However, those circumstances that underscore the circumstantial decision are quite powerful in epidemiological terms. A fundamental part of what we do when we investigate transmission events or potential transmission events is the power of the epidemiology, and how supportive it is.

36 If we go back to the pandemic, you would ascertain whether you are a contact and you get pinged, and you are advised to isolate. If you then become COVID-19 positive within a few days, the inference is that it is related to that ping, but obviously that may not have been the case. That is a more circumstantial, less robust association than what we were looking at here, i.e. well-defined time and place associations. So, it is the degree of circumstantial evidence we are discussing here. The more infections of the same type, caused by related microbes, occurring in the same place, and linked in time, the greater the chance that those infections are linked to each other and that the environment could be the source of infection.

37 I have been asked if there was any weighting applied to particular circumstances. I would say probably not, as if we had weighted one line of evidence, we would have to define that and refer to it in our reports. It was almost like ticking boxes of probability. We neither defined nor weighted them, and this approach is standard practice in an epidemiological investigation such as ours.

38 Regarding the possibility of outside environmental sources as described in section 3.6.3 of the Overview Report, we had a detailed timeline for each patient; their date of admission, their date of discharge, their date of re-admission, because very often there would be multiple admissions. We sought to establish when the bacteraemia developed in relation to the person entering the potential risk environment. If you develop something such as

bacteraemia or diarrhoea on day one, two or three, it could well be related to what happened before you entered the hospital. If it is beyond that and this keeps on happening with these bacteraemias, as it did, then that increases the likelihood that it has something to do with being in hospital. That does not necessarily mean that the source is definitely the environment of the hospital, because it could be something that is done to the patient. They could have received a medicine that makes their immune system more depleted, which can happen, and it could be that which increases their risk of endogenous infection. Then there is the clustering, which I referred to earlier, which asks the question, “Why would you get a run of endogenous infections with the same bug? That does not seem plausible.”

39 I have been asked by the Inquiry to identify the recognised methods for demonstrating a causal relationship to a standard scientific certainty and whether there are methods that can be used to demonstrate a definite relationship or to definitely exclude a relationship. There are different levels. You can start off with simply saying, “This is a Gram-negative bacterium,” and you get a series of Gram-negative bacteria, and you want to know, “Are they related?” The next thing you would do is name and speciate those organisms, for example, Stenotrophomonas maltophilia. So, we are now dealing with eight bacteraemias all caused by Stenotrophomonas maltophilia. The next question is, “Are those the same Stenotrophomonas maltophilia or very closely related, or are they different?” Bacteria multiply very quickly and so you can get changes in the DNA as you move from this bacterium to its progeny and to the progeny of those progeny. All of this is happening every 20 minutes, with the bacteria multiplying, so there are limits. There is a drift, and that needs to be considered when you apply a typing or fingerprinting method to, in this case, Stenotrophomonas maltophilia to work out if tomorrow’s Stenotrophomonas maltophilia or, indeed, the ones three weeks away related to the ones three weeks previously.

Whole Genome Sequencing

40 There are many different typing and fingerprinting methods, but the more robust methods are based on looking at the DNA in those bacteria. The ultimate fingerprinting method that we have at our disposal is called, “whole genome sequencing,” where you basically read the whole genetic code in a bacterium, and then you compare that genetic code with genetic code of other bacteria. Again, you have to set limits because to expect the genetic codes to be identical is too stringent and depending on the bacterium and experience of looking at bacteria, they clearly are related because you culture them yourselves and look how much they drift or you put them into an animal and see how much they drift in genetic code. That is the ultimate technique, and that ultimate technique was applied post-hoc, not real time, to my understanding, but post-hoc to some of the bloodstream isolates from some of these individuals.

41 We deal with this in the CNR report at section 8.3.1. No-one appears to have said, “Let’s type everything we’ve got, all the isolates causing all these bacteraemias, and type all the isolates that were recovered from environmental sources.” The difference between typing and fingerprinting is semantic; for all intents and purposes the words can be used interchangeably. There was no systematic use of typing, either in real time or after the event. Of even greater concern, and this is detailed in the report, the sampling of the potential environmental sources was not systematic. If you have not sampled the water in a repeated way, you cannot confidently exclude the water as the source. If there is an ad hoc process to sampling the water, not only when you do it, but what you look for when you have got that water sample, then it is perhaps not surprising that we were unable to produce definitive matches of bacteria in blood with bacteria in a water source.

42 We were provided with reports of the sequencing that was done in 2021, but this was only at a late stage in our investigation. We had more than one meeting to discuss the post-hoc whole genome sequencing results that had been obtained. We highlighted that these were incomplete, and the answer was, “Well, these are all the ones that were available.”

43 There is a routine practice for when you grow bacteria from critical samples and blood cultures, which is that you store all the positive bacteria recovered, not least so that one can go back and retrieve them if there is a subsequent need. In this case we are only talking five years or so down the line, so I would expect, had there been a systematic storage process in place, which there should have been, that all the bacteria would still be in that store five years down the line. It would appear not, from the fact that not everything that one would have liked to have been typed was typed.

44 Let’s say you type five bacteria, and you cannot find a match in three of those five, but the two that are missing could have matched to any one of those three that you do have information about, and both of them could match to one of those three, or one of them could match to one of them. So, you are then left with, “Okay, you have done some typing; not found any matches, but you have not excluded by any means the possibility of a link.” That is just with the isolates that you have got from the blood cultures. If you have not got any isolates that you can include in that typing exercise from water samples, then we are not saying there can only be one Stenotrophomonas in water that would cause Stenotrophomonas whenever it occurred in a patient. There could be 20 different Stenotrophomonas species in the water, of which only three ever get into patients, so it is complex. Because of the incompleteness of all those levels then it is not surprising, sadly, that we were unable to produce case examples that match the definition of definite. The level of certainty with which WGS can rule in or rule out matches between human and environmental isolates depends on how comprehensive/extensive are the collections of available isolates.

45 I have been asked by the Inquiry if I found it surprising that complete typing was not done, and that level of sampling was not what I expected. Let me deal with the sampling first because that, perhaps for me, is the most surprising. There was a lot of noise in real time within NHS GGC about the environment, and especially on the quality of the water. There were lots about reports and concerns, incident meetings, lots of interventions that were put in place by NHS GGC to attempt to mitigate water-associated risks. All those together say that in real time, there were question marks, at the very least, around water quality. With that you need to then ask why there was not a robust systematic investigation process, surveillance process, sampling process in place to assure or describe what was happening with respect to water. Not least because you put in these expensive system-wide interventions, and unless you are sampling systematically before and after the intervention, you cannot know how effective that intervention has been.

46 For example, one of the interventions was the chlorination of the water system by the addition of chlorine dioxide. If you are simply going to wait to see whether there are more or less infections in patients, then that is a very blunt tool to do that. I would have expected to see a systematic sampling process, sampling key parts of the system rigorously in a set schema, and how you then process those samples. In other words, “What did you look for in those samples?” Did you simply count the total number of bacteria, or did you simply look for “bug X,” and you were not bothered in bugs A to Z apart from bug X? This appeared to be happening with very large numbers of samples, and I believe we saw all the records that were available.

47 The lack of use in real time of definitive fingerprinting methods and whole genome sequencing, is less surprising, because it is an evolving methodology, and was evolving through the time in which these events were going on. The ability to use whole genome sequencing through much of that decade was largely down to reference laboratory capacity rather than being used routinely in each and every hospital. NHS GGC, just like many of the laboratories, were referring bacteria to a reference laboratory (Colindale). The Colindale laboratory is part of UK Health Security Agency (formerly Public

Health England). It provides reference (expert) laboratory services, in particular to identify and type or fingerprint microbes.

48 Where Colindale did have that capability, which perhaps was not as robust and discriminatory as whole genome sequencing but not far off, NHS GGC were doing it in an incomplete fashion. The laboratory was not referring all the isolates and, of course, if you are not sampling systematically, then you are using a needle-in-a-haystack sampling approach. The chances of finding matches are diminished. So, I was less surprised about that, but still surprised that there was not a greater attempt to make these matches. As soon as you make just one match, which is not easy to do this because of all the difficulties of finding bacteria in water sources, but if you did find a match, then that is absolute proof that there is a problem, and you have to focus everything on it and consider closing units and moving, and that is what happened. There was concern about this, but the concern about the putative source of these bacteria was not matched, to my mind, by the robustness of the investigation of these putative sources.

49 Water environmental microbiology is technically demanding. It is key to have a systematic sampling scheme in place in order to increase the chances of identifying contaminating bacteria, which may only be found sporadically (from time to time) in a water source(s). Such a scheme should include the collection of appropriately collected (to minimise the chance of contamination during collection) water samples in adequate volumes, taken from sites that are truly representative of the water system under investigation. Also, when processing the water samples, there are a variety of microbiological methods that can be employed, some of which aim to count overall numbers of bacteria present, and others that can target/identify whether specific bacteria of interest are present.

50 It is always difficult when you look back because we were not there in real time. The best thing we had to go on, having realized that this was what happened, was the IMT minutes. Oddly, they did not highlight this as an issue. Had I sat in one of those IMTs, I would have been banging the table, saying, “We need to do it this way. The sampling techniques need to be like this, and

then we need to type everything that moves." I am not saying there was never any reference to the need to match bacteria, but the drum was not banging very loudly.

Categorising the likelihood of an environmental source for an infection

51 In considering the likelihood of the hospital environment as the source of each bacteraemia, we took into account all available patient, clinical, infection prevention and control, microbiology, local investigations (including Datix and IMTs where available) and hospital environmental data- everything which was provided to us. This is described in section 3.6 of the Overview Report. The standard epidemiological way of determining causality of, and potential links between infections is according to 'time, place and person' information.

52 I have been asked by the Inquiry how the approach of determining causation, based on whether a causal link was more likely than not, map onto the descriptors of the likelihood adopted by the expert panel. A" strong possible' and above represents a greater than 50 per cent likelihood, and we categorised the 'strong possibles' alongside the 'probables,' of whatever strength. On the balance of probabilities, to my understanding, it means more than 50 per cent.

Datasets

53 I have been asked by the Inquiry if I can explain what the various processes and datasets relevant to HAI reporting are supposed to do and how they link together to help identify infection risk and link.

54 The CNR report refers to statistical process charts (SPC). Based on observations over a long period of time, you assess a threshold which you believe is the natural baseline occurrence of a certain event, in this case the bacteraemia caused by a particular bug. You look over several years and you

see that, this particular bug, bug X, occurs once a year causing a bloodstream infection. There is a mathematical way of ascribing limits around that threshold of 1 per 12 months. Those limits, looking at the line above and below, is the line above we are interested in, the limit above. Once that is exceeded, that sets an alert that there is a potential issue here. You could not really set a statistical process chart for every possible bug causing every possible infection, as you would have charts coming out of your ears. In reality you use an SPC process in real time for particular bugs of interest.

55 SPC charts are not infallible. As with all tools / techniques to investigate possible (genuine) increases in infection incidence, it is important to examine the raw (time / place / person) data to identify potential clusters/outbreaks. Thus, SPC charts should be seen as an adjunct to rather than the key determinant of investigations of potential increases in the incidence of infection.

56 MRSA and Clostridium difficile were particular bugs of interest in the early 2000s, so we had national targets for these, and organisations will have used statistical process charts to track the occurrence of those bugs. It would be uncommon to use that process for, in this case, Gram-negative bacteria of potential environmental source. Instead, you would use a common-sense approach to thinking, “I would expect to see this organism uncommonly.” When I see eight episodes of Stenotrophomonas maltophilia, for example, occurring within four or six months I would consider these to be clusters. Those are quite powerful analyses, just to say, “Investigation is warranted here.” Investigation is mandated to keep patients safe. We start off with a basic epidemiological look-see to look at that for the evidence of clustering in time and place, and then we spread the net further to look for putative sources.

57 I have been asked by the Inquiry if the CNR panel had concerns over the use of the systems by NHS GGC and the breaching of upper limits. We were not expecting that for each and every bug there would be prescribed limits; however, it is standard practice to review infections in real time and look for potential clustering and potential common sources or common causes. The

panel were not assured that that process was robust enough, either in terms of when it occurred in response to this time-place clustering or how it occurred. We saw several examples of incident management meetings where the actions set in one meeting would not be reviewed in the next meeting, or any written evidence that they would be reviewed at a later date. The whole point of an Incident Management Team is recognising a potential incident and setting actions. The first thing you do at the next IMT meeting is to review those actions and ensure they have been followed through. We found multiple examples where that robustness of process was not followed.

58 I have been asked by the Inquiry whether, where the necessary datasets are available but the reporting from the laboratory does not confirm a relationship, it means that a relationship is excluded, or whether it means no more than a relationship has not been proved to a desired standard of certainty. I would say that it is absolutely the latter. Finding a match is not a simple process at all; you have to try hard in terms of how frequently you are sampling, how assiduously one is looking for bacteria in those samples, and then how robust are the typing methods used. Each one of those can fundamentally flaw the process.

59 I have been asked by the Inquiry if there are recognised methods for excluding a link. I would say that, had there been a systematic process in place for both sampling in time and place and the way those samples were processed and then the way any subsequent bacteria that were recovered were then typed/fingerprinted, that would have been the desired way of doing things. All one could then potentially argue about is, well, “How frequently are you sampling? You are only doing it monthly, I would have preferred to see it weekly or daily,” or whatever. I am stretching the point for effect. Those tests about robustness of frequency, time and place sampling were not, to my mind, met. The methodology about what was looked for, the systematic process, was not met. The typing process was not met; therefore, it did not meet the ideal for each of those three pillars if you like.

NHS GGC Response to CNR

60 I have been asked by the Inquiry for my thoughts on the document which was prepared by GGC in response to our final draft of the Overview Report. We took this very seriously and we complied a document titled, "*Case Note Review Team Rebuttal of GGC Consultation Response*". My overall impression is that I felt it was defensive rather than constructive. There seemed to me to be an attempt to highlight the lack of definitive proof that we had been able to highlight about the environment as a source of infections, as opposed to accepting the weight of evidence and the balance of probabilities about such sources. Admittedly you would expect an organisation faced with a critical report to defend itself, but I have been in multiple situations where one issues a report and then there is a rebuttal, and I felt it was more defensive than I would have thought necessary. It did not accept the weight of evidence that we produced highlighting both the likely associations with the environment and the deficiencies we had highlighted in process about investigating the bacteraemias.

61 It felt as if the NHS GGC's prime aim was to defend as opposed to learn. I have spent four decades working in the NHS and the culture has changed markedly from when I first started working in the NHS to how it is now. It has changed for the better, to being open and encouraging criticism or critique so that one can learn and improve. I felt that the NHS GGC approach was less attuned to practice today about accepting criticism and how one uses that criticism and critique compared with how it used to be. Way back when I first started, it was like that, but it certainly was less attuned to what I have become accustomed to. As a result of the response by NHS GGC, a number of very minor changes were made to the panel's draft report.

62 Had I been a senior manager at NHS GGC throughout the process, I would have made it an imperative for me to understand what the terms were of this next process, what the terms were, the objectives, I would have striven to get as much information as I could, and then tried to steer, dare I say, "correct," any deficiencies I felt or omissions in the process. I did not recall that taking

place and it felt more like a reactive process, responding to our report, the meeting, and the draft and so on, rather than a proactive response.

63 Within the Overview Report we had highlighted the issue of typing evidence and in particular queried the typing evidence that, according to NHS GGC, showed that the Stenotrophomonas cases were not linked to each other or the water system. We came to the view that this was an example of NHS GGC using Whole Genome Sequencing as a method of excluding a link rather than establishing it. Of course, NHS GGC were critical of this, but for me it seemed they were concentrating on the information they had to refute a potential link. However, that information was flawed in terms of its robustness on each of those three pillars that I referred to and there was no acknowledgement of that lack of robustness in that attempt to refute the links. When you read their report, you can clearly see a defensive approach to this, you see zero next to "definite cases" that fulfil the definition of, "The environment is the proven source." Then you say, "Well, you have been unable to come up with 'definite' and look, this is our typing evidence. We are agreeing with that. There are no 'definites'," as opposed to, "One in three of the cases, based on the grounds of probability, were linked to the environment." The lack of "definites" does not rebut those one in three cases.

64 I have been asked by the Inquiry for my thoughts on NHS GGC response, in particular their comments stating that the CNR ought to have considered additional comparison data from similar units and trends in infection along the years. I would respond by saying that we did not need to go and look at what was happening in a range of other hospitals. Faced with the epidemiological data that showed clear clustering in time and place of these bacteria, it was not relevant what was going on elsewhere. If I found the same thing in another hospital, then they have an issue, but it may be a completely different issue, and I do not need to look there. The epidemiology stands on looking within this institution, or place, or this limited number of places within this institution. The Expert Panel were not saying, "There's been an increase overall in Gram-negative environmental bacteraemia." That might be the case, but that was not our remit. Our task was to match a potential environmental source to each

individual episode. Therefore, looking at what was going on in other hospitals was not necessary.

65 From their response, NHS GGC's position was that there was no baseline for infection. The onus here is on them to make clear what they mean by that, but if what they mean is that we did not look far enough back in time to establish a baseline, then I would refute that. We have five years' worth of data; if you plot what is going on in the x-axis for five years and suddenly there is a peak here and then there is a peak there, your baseline is there. You have enough data in that five-year period to be able to spot clustering in time and place, because a five-year period provides, effectively, a running baseline.

66 I have been asked by the Inquiry for my thoughts on a letter dated, 1 March 2021, from Jane Grant in which she states, that "NHS GGC believe that the CNR indicates that the health board should have approached these issues they were facing in a different way, despite following advice and guidance from national experts and agencies." I recall the Expert Panel being a bit confused by that statement. I think we understood what was being said was that "we did what we were told, so if this report's saying that we shared something different, that is not our fault, because we did what we were told." I do not know precisely what Health Protection Scotland (HPS), told NHS GGC to do in detail. We reviewed the information that we had, whether that was the incident management reports, the clinical way of reporting the infections, the typing, the sampling, whatever. Either the advice they got previously was incomplete or they were just told that "everything's hunky-dory, just continue," which seems incredible given the number of reports and the consistent levels of concern that were expressed. A lot of what we were referring to are basic measures to minimise risk, either from the point of view of how you investigate infection clustering in time and place and/or carrying out surveillance, water sampling, etc, based on how you would carry out an IMT and a follow-up to an IMT and so on. I do not know what level of detail other organisations and bodies went into, but all we can look up is some product of what happened. I do not think it is fair to take a defence line that might be, "We did what we

were told," because I cannot believe they were told the things that we put in our reports because, if they were, then they did not do them.

67 We took the decision at the outset that we would not review other reports, as we did not want to be biased by what had been said. Obviously, we knew there were concerns about the environment and potential infection sources, but had we read reports that kept saying the same thing or lines of evidence, I think that would have naturally swayed us. So, we did not do that. I have not, after carrying out our investigation and writing our report, gone back to see what was agreed and disagreed. To my recollection, there was only one other report in detail that we referred to, but we did that post investigation as we were writing our report. So, what was said or not said, what was done or not done, was not part of our remit. Our remit was to review a cohort of bacteraemias, and we did that.

CNR Conclusions

68 I have been asked by the Inquiry the extent to which the expert panel's ability to come to findings was restricted by the limits of the datasets. I feel that the process took longer than it needed to have taken. Obviously, the pandemic drastically affected the initial predicted timescale, and we acknowledge that in the report. But undoubtedly the difficulties in obtaining complete datasets delayed us, as it meant we had to go around the process again. We had to review every patient twice, and sometimes more than twice because we were getting new information after we had done the first review. We have to assume that we were eventually given all the information that we requested, and that there was available. We could have done with more, if a major part of the criticism was that we did not dig enough. If the question is, "How were our conclusions affected by the information provided?" I think our conclusions came late compared to when they should have been made, but the substance of our conclusions stand.

69 In regard to those limitations, I have been asked what evidence was available in the ‘Most likely’ group (as described in section 5.6 on page 69 of the Oversight Review) that was absent in the remaining ‘possible’ cases. More often than not, the “Most likely” classification was not given when there was more information available than for the ones where there is not enough information. Rather, it was the weight of evidence based on time and place, based on similar organisms recovered from water. It is the ticking of those boxes that strengthens the balance of probabilities, as opposed to there being an absence of data. There is no straight-line association between how much information is provided and the degree of likelihood of association, but it is a contributory factor.

70 We had strong opinions based on the evidence available, and there was consensus. The Expert Panel was just the three of us and I cannot recall any instances where we had two versus one. It was, to the best of my knowledge, all three of us in agreement.

71 I have been asked by the Inquiry what evidence would lead to the conclusion that a case was unrelated; was there insufficient evidence to prove even a possibility of a link or was there evidence that positively excluded the link? On reflection I would say it was probably a bit of both. For example, if we get an environmental-type, Gram-negative organism occurring within 24 hours of admission to hospital then the likelihood is that someone has acquired that organism from the water. For it to be from the hands of a healthcare worker, or a contaminated intravenous line, all within 24 hours, is stretching the bounds of probability. That would be a good example where the basic epidemiology would say, “This is unlikely to be hospital acquired,” using a 48- to 72-hour window of community association. There could also be an organism you see once, and once only, in a patient’s blood, and you do not see it at all in any of the albeit incomplete surveillance data. During the CNR there was one case where we were unable to determine causation. I cannot remember which particular bug it was, but we just did not have enough information available in the timeline. There were some key bits missing and, therefore, to guess those would have would have clearly been inappropriate.

So, some of those basic tenets of when it satisfied time and place and likelihood of community source, for example, were missing.

72 I have been asked by the Inquiry if I am aware of NHS GGC reporting that they were able to link one of the three cases of mycobacterium chelonae to the environment, and if this was achieved because of whole-genome sequencing or Variable Number Tandem Repeat- VNTR. My recollection is that this was based on VNTR typing, where they saw that match. If all that had been done here, there would have been no whole-genome sequencing available and we would just be fingerprinting. There are a few examples of successful defence saying that fingerprinting is not enough to match a criminal to an offence. So, it was not, from recollection, whole-genome sequencing based that provided a match.

73 Whole genome sequencing (WGS) and Variable Number Tandem Repeat (VNTR) methods are both based on the DNA content of the microbe. They use different ways to look at the fine detail of the DNA sequence (code). WGS can provide more information/detail about the DNA code than VNTR and so may be more suited to determining a rule in/rule out for matching microbes; however, this is not always the case. Both methods are generally accepted as being proficient at ruling in/ruling out matches between microbes.

74 Within the NHS GGC response to the CNR they indicate that the CNR expert panel finds it hard to accept NHS GGC's challenge to its findings on causation. During the five-year timeline from which we were working, a number of significant interventions took place. This included ward relocation, which is a major decision, chlorination of water systems and additional extra decontamination and so on. It is hard to believe these measures were taken unless NHS GGC seriously entertained the possibility that the water was a potential source of infection for patients. To then say, "Ah, but there's no definitive proof because there are no cases that match your case definition here, and we did some post-hoc whole-genome sequencing based on incomplete datasets, therefore quid pro quo;" there is a lack of logic there. There is one set of arguments being used to take these very big, significant

decisions and a different set of logic to say, “Ah, yes, but the environment” you know, “there is no proof here.” The grounds of probability, for me, suggest there is ample concern that the environment was linked to a substantial number of these bacteraemias. To say that there is no definitive proof, I think, is ignoring a large part of the evidence story here. The question for NHS GGC would be why you would undertake such significant measures and spend likely large amounts of money if you did not think there was a problem.

HAI Monitoring and reporting

75 During our review we engaged with a number of key staff involved in IPC at NHS GGC who advised us that they had been denied access to water sampling and testing information despite multiple requests. This information was coming from whistle-blowers, with whom we met on more than one occasion. Clearly, we had to be careful about what we were told, and what we said in those meetings, and I believe we were careful. I know that, by the very nature of whistleblowing, it is potentially one side of the story, but some of the things we were told made us feel very uncomfortable about what allegedly took place. This was one of those things. If I was Infection Control staff which this person I am referring to was, and part of my responsibilities is to reduce infection risk to patients to a minimum, and I am denied access to basic, information, it would make my job untenable. I would want to know why am I being denied this? Is there something wrong with these data? Has it turned up something that no-one wants to tell me? Is the data incomplete such that I would want things done in a different way? Would I be critical of individuals? Would I be critical of managers? Am I protecting someone or something? I have never come anywhere near to experiencing this in my career. I might have to ask twice on occasions for such information, but I have never been denied.

76 This made very uncomfortable reading of the situation. Obviously, we do not know what the reasoning might have been, but I cannot think of a valid reason why multiple requests were denied, not just to an interested party but to someone who could not fulfil his or her role adequately. It is not difficult to say, “We haven’t got the information you’re asking for,” or, “It’s written in double Dutch, and we’ve got no-one who can translate double Dutch.” I am being facetious but why would that exist? It is fundamental to enable this person and the rest of the IPC team to minimise risk to patients, so I cannot think of a valid reason why multiple requests would be denied.

Declaration

I believe that the facts stated in this witness statement are true to the best of my knowledge, information, and belief. I understand that this statement may form part of the evidence before the Inquiry and be published on the Inquiry’s website.

Appendix A

A33448007 – Bundle 25– QEUH Case Note Review report March 2021
A43237055 – Bundle 25 Case Note Review team- rebuttal of NHSGGC response.

Appendix B- CV

International Responsibilities (selected)**:**

1. Delivered >140 invited lectures at international meetings between 2002-19.
2. Editorial Advisory Board Member Clinical Infectious Diseases, 2020-
3. Non-Executive Director, Phico Therapeutics, 2018-.
4. Member of Scientific Advisory Board of AiCuris, 2018-.
5. Member of Wellcome Trust CARB-X panel on novel antimicrobials 2017.
6. Co-Lead of EU Innovative Medicines Initiative’s COMBACTE-CDI consortium, 2017-.
7. International Advisory Board for Lancet Gastroenterology & Hepatology, 2016-18.

8. Panel member for MRC UK-China Antimicrobial Resistance Partnership Initiative, 2016.
9. Member of Scientific Advisory Board EU Innovative Medicines Initiative's COMBACTE-NET consortium, 2015-.
10. Expert Panel for Swedish Medical Research Grant applications (all medical specialties), 2015-20.
11. Chair of global publication committee *C. difficile* monoclonal anti-toxin antibody studies (Merck), 2015-18.
12. Consultancy advice / lead clinician for phase 3 studies roles for the development of multiple novel antibiotics including linezolid, tedizolid, tigecycline, ceftaroline, dalbavancin, ceftaz-avibactam: 2000-17.
13. Chair of expert group/publication (*C. difficile* infection in Europe) on key issues for healthcare policy makers across Europe (2012-).
14. Lead of pan-European *C. difficile* surveillance projects (diagnosis, EUCLID; resistance, ClosER) (2012-).
15. International Editorial Board, Journal of Hospital Infection (2011-).
16. Editorial Board Infectious Diseases in Clinical Practice (USA) (2010-).
17. Invited member of *Clostridium difficile* guidelines working group for Infectious Disease Society of America (2010-). 2010 guidelines published: 2016/17 guidelines in progress.
18. Global Scientific Advisory Board for cadazolid, Actelion (2009-18).
19. Advisor to Wellcome Trust's Technology Transfer Strategy Panel (2008-).

National Responsibilities (selected)**:**

1. Clinical advisor to Centre for Health Economics, University of York for the (UK NICE) evaluation of the value to the NHS of ceftazidime with avibactam and cefiderocol for treating severe aerobic Gram-negative bacterial infections (2020-21).
2. Expert advisor to NICE for update of CDI treatment guideline (NG199, July 2021) 'Clostridioides difficile infection: antimicrobial prescribing' 2020-21.
3. Member of two SAGE sub-groups (Hospital-Onset COVID-19 Infection; and Environment & Modelling) and chair a working group (Hospital Environment) of the latter. The Scientific Advisory Group on Emergencies (SAGE) gives advice to the UK government on SARS-CoV-2 / COVID-19 (2020-).

4. Co-chair of Technical Validation Group for COVID-19 diagnostics for UK/NHS use (2020-).
5. Invited Expert by NHS Scotland to participate in Case Review for the Queen Elizabeth University Hospital (Glasgow) (2020-21).
6. NHS AMR Programme Board (2019-).
7. Expert advisor to NICE antimicrobial evaluations project, Univ. of York (2021-).
8. Expert advisor to NICE panel for guidelines on treatment of C. difficile infection (2020-21).
9. Chair of Public Health England sub-group to update guidelines on treatment of C. difficile infection (2018-19).
10. Chair of PHE sub-group to review delivery of C. difficile typing/fingerprinting in England (2018-).
11. Technical Advisor to Policy Research Unit in Economic Evaluation of Health & Care Interventions: 'Framework for Value Assessment of New Antimicrobials.' (2017-18).
12. Newton Prize reviewer (2017-19).
13. NIHR DH/NHS Capital Funding Antimicrobial Resistance review panel (2017).
14. Member of the Medical Research Council Infections and Immunity Board, 2017-21.
15. Chair of Public Health England Rapid Review Panel, 2014-.
16. Deputy Chairperson of Antimicrobial Resistance & Healthcare Associated Infection (ARHAI) (2011-18).
17. Chair of ARHAI sub-groups on HCAI surveillance (2012-14); Surgical site infection Surveillance proposals (2013-14); revised MRSA screening in NHS (2013-14); Antibiotic prescribing diversity (2014-).
18. Expert Advisor to EPIC3 project: Evidenced-based Practice Infection Control guidelines (2012-13).
19. Expert Adviser to National Institute of Clinical Excellence, NICE guidelines (Broad spectrum antibiotics and *C. difficile* infection risk, 2015; Infection Control, 2014; faecal transplantation, 2013-14); evidence summaries (fidaxomicin, 2013; fosfomycin, 2014; telavancin 2014).
20. Advisor on healthcare associated infection diagnostics for Technology Strategy Board (2009-10).

21. HPA (PHE) Healthcare-associated Infection & Antimicrobial Resistance Programme Board (2008-).
22. HPA (PHE) Regional Microbiology Network Lead *C. difficile* infection (CDI) (2007-).
23. Appointed as member of ARHAI - advisory committee to the Department of Health (2007-). Lead for CDI.
24. Lead Examiner for Royal College of Pathologists (MRCPath) Medical Microbiology Practical Examination (2001-02, 2005, 2008, 2010, 2013). Assisted in re-design of MRCPath Part II examination. Lead for setting of Scientific Paper Evaluation questions - MRCPath Part II (2004-).
25. Lead / UK Investigator 15 clinical trials of new anti-infective drugs (1999-). Advisor on trial design/results/ registration issues.

Research Awards: In the last 5 years ~[redacted].

Selected Research Grants:

1. Biological Research Centre, Leeds. One of six theme leads – Infection and Antimicrobial Resistance. NIHR (2022-27) ~[redacted] (Theme Lead).
2. Mechanisms of spore engulfment in C. difficile. Medical Research Council (MR/V032151/1) (2021) - ~[redacted] (Co-Investigator).
3. Reducing the infectivity of SARS-CoV-2 on PPE gowns used in healthcare environments. Engineering and Physical Sciences Research Council (EP/V056921/1) (2021) - ~[redacted] Lead
4. Detection of SARS-CoV-2 in faeces. JP Moulton Foundation (2020) - [redacted] (Lead).
5. Health Protection Research Unit in Healthcare Associated Infection. NIHR 2019-24 - [redacted] (named academic partner to U of Oxford/PHE).
6. Transforming Antimicrobial Research with Gut model Evaluations for Therapies and Diagnostics (TARGETED AMR). NIHR (200633) (2019-21) - [redacted]) (Lead).
7. Development and Validation of in vitro Healthy and Dysbiosis Human Microbiota Models to Facilitate Early Phase Antimicrobial Development. Centers for Disease Control. (2018-19), [redacted] (Lead).

8. Combatting Bacterial Resistance in Europe – Clostridium difficile Infections (COMBACTE-CDI). European Union Innovative Medicines Initiative (2017-) – [redacted] in kind EFPIA contribution (Co-Lead).
9. Multisite study of environmental contamination in hospital washrooms according to hand drying method. European Tissue Symposium. (2017-) - [redacted] (Lead).
10. Accelerating development of infection diagnostics for patient management and reduction of antibiotic misuse. MRC. (2016-), [redacted]. Co-app.
11. Principal/UK Investigator 14 clinical trials of new anti-infective drugs, 1999-2015.
12. Novel test for rapid bacteraemia detection. Spectral Platforms (2015-), [redacted]. Lead.
13. Health Protection Research Unit (Oxford), HCAI & AMR. NIHR (2013-16), [redacted]. Co-app.
14. Antibodies to treat severe CDIs. TSB (2013-16), [redacted]. Co-app.
15. WGS for patient care/surveillance. HICF, Wellcome/DoH (2013-16), [redacted]. Co-app.
16. Pan-Europe CDI surveillance (Co-Lead), resistance (Lead), diagnosis (Lead). European CDC [redacted], Astellas [redacted], Astellas [redacted] (2010-14).
17. Modernising Medical Microbiol via WGS. NIHR (2009-13), [redacted]. Lead on C. difficile. Co-app.

Invited Oral Presentations: >250, including >100 international.

Publications: 13 Books, 25 Chapters, ~600 Publications, >300 Abstracts.