The Sheku Bayoh Public Inquiry

Witness Statement

Professor Sebastian Lucas

Taken by [redacted]

Via MS Teams

on Thursday 8th December 2022

Witness details

1. My name is Sebastian Lucas. My details are known to the Inquiry.

Professional Qualifications and Background

2. My qualifications are MA, BM BCh, FRCPath, FRCP.

3. I have been a consultant histopathologist since 1980, with wide experience in general diagnostic histopathology and autopsy pathology. I have been a Member of the Royal College of Pathologists from 1978, and Member of the Royal Colleges of Physicians since 1975. I have published widely on all aspects of histopathology and autopsy practice, particularly in infectious diseases.

Signature of witness…… [redacted] ……..

4. Also since the late 1990s, I have taken a special interest in the morbid anatomy of sickle cell disease. I have done around 100 autopsies now on individuals with sickle cell disease, and autopsies on uncountable numbers of individuals with sickle cell trait, because trait is so common. I have been involved in medico-legal court work on many sickle disease deaths and, specifically, five previous deaths in custody or stress-related deaths among men with sickle cell trait.

5. I became a professor of pathology in 1995 at Guys and St Thomas' Hospital, retiring in 2012. From 2012 to 2022, I worked part time, at GSTT Hospital and also local mortuaries in Milton Keynes, Bedfordshire, Luton, and Buckinghamshire. I stopped practical working altogether in October 2022. Since then, I teach pathology trainees on autopsy pathology interpretation by lecture, and I lecture to medical students on general pathology. And I review autopsy pathology cases at the request of coroners, pathologists, families, and legal firms.

COPFS Instructions For Report

6. I have been asked if I recall receiving a letter of instruction or briefing papers or other materials. The briefing stuff I had, and the person to whom I responded was someone called Alasdair MacLeod, Procurator Fiscal.

7. I have been shown the email from Leslie Brown and Ashley Edwards (CC'd) from COPFS instructing my report.[1] I have been asked whether I recall this email. The email makes reference to 4 reports being forwarded to me for consideration. My report lists the following 4 reports as being presented to me *"The original autopsy report (F542/2015); The supplement dated Sept 2017 that confirms that BAYOH has sickle cell trait; The expert witness report of Dr E Soilleux (11th Feb 2016) and her supplementary report (10th May 2018)".* I have been asked if I was provided with any other documents. No.

[1] COPFS-02468

8. I have been asked whether there would be any other material that I would have expected to see in the instruction from COPFS. No, and the circumstances of the death, the autopsy report, versions of it, someone else's opinion and I was sent the histology slides also. I don't have the slides anymore. I must have sent them back a long time ago. I have been asked if there were reports by forensic pathologists in a case like this, would I expect to see such reports. Yes, I would.

9. The email has a list of bullet points for me to consider as part of my report:

    - *"To comment on the supplementary report by Liz Soilleux and in particular her conclusions on the cause and mechanism of death and the significance of sickle cell carrier status*
    - *Whether the apparent sickling of cells occurred ante or post mortem*
    - *From your own experience conducting autopsies in persons who are sickle cell carriers the extent to which sickling of cells is noted generally*
    - *Whether it is possible or likely that sickle cell carrier status contributed to the cause and mechanism of death in this case having regard to all of the relevant factors set out in the reports."*

    While I don't recall this email, I have listed these bullet points in my report at page 1 and I subsequently addressed all of those issues.

FINDINGS OF THE REPORT

Histopathology

10. I have been referred at page 2 of my Report[2] where I summarise my review of the histology slides of Mr Bayoh. I have been asked to explain my findings in relation to each.

[2] COPFS-00084

Bone Marrow

11. In relation to bone marrow, I confirmed that there are no sickle cells. I have been asked if that is normal in a case such as Mr Bayoh's. Yes. If you've got sickle cell disease, you virtually always have sickle cells in the marrow, but in sickle trait, not necessarily.

Brain

12. The brain has a bit of sickling cells there. SRBC here is shorthand for sickle cell red blood cells. Mr Bayoh has died, ultimately, of brain failure due to lack of circulation of blood and oxygen. So red neuronal change is the pathology-speak for that brain neurons are dying. So if a person is strangled, that's what you see. If you've had a heart attack and the heart is not restarted within three minutes, the brain dies, and that's what you see. It's a non-specific finding of lack of oxygen and blood going into the brain.

Kidney, Liver and Heart

13. This paragraph in relation to the kidney histology essentially says there's an awful lot of sickle cells present, but he's not died of kidney failure. Kidney failure takes an awful long time to kill you. It takes about a week. So that's just an observation at the time of death, there's a lot of sickling going on. Similarly, there's a lot of sickling in the liver and the heart. The heart seems to be normal, otherwise.

Thyroid and Adrenal

14. Again, the thyroid and adrenal tissue is normal apart from the fact there's a lot of sickling.

Lung Tissue

15. This is the crucial one in people who have died under restraint and exertion and you can say that sickle really is very important here.

16. Now, in this particular case, you will notice I've been quite precise, that whoever did the autopsy took six samples of lung, I assume from six different parts of the lung.

17. The lung has five lobes, so probably it's one piece from each lobe, plus one extra, but I don't think it was stated exactly where they come from. Some bits don't have any sickling and some bits do, which is why it's not quite so clear-cut as some of the other more obvious ones, where all the lungs are very sickled, and you can say this is a classic example of the scenario called the Acute Chest Syndrome, and this is well-known in sickle disease.

Acute Chest Syndrome and Sickle Cell

18. It obviously happens much more commonly in people with sickle disease, and what it means is that in the lung blood vessels, the cells sickle, and they simply can't move. If you can't have blood moving through the lung, the lung stops, the heart stops, and you die. It's called a crisis due to Acute Chest Syndrome. Mr Bayoh has some aspects of the Acute Chest Syndrome, which is why I think sickle contributed. Having thought this through, I would now say my bottom line is that I think we should probably move sickle trait down in the cause of death list from being in part 1 into part 2, as a contributor but not the main cause. Exactly the extent to which it contributes is entirely subjective. There's no absolutes here.

Sickle Cell & Toxicology

19. I have been referred to my commentary on the histology at pages 2 and 3 of my report:-

Signature of witness…… [redacted] ……..

> *"There is no doubt that HbAS[3] persons have died in custody or under stress (heat, dehydration, exercise) where the main pathogenesis is a sickle crisis affecting the lungs (particularly), the kidneys, skeletal muscle, and the heart, and this has led to death.*
>
> *There is no doubt that the vast majority of persons with sickle cell trait, who undergo life's daily stresses, do not suffer any such syndrome. The critical aspects are therefore the level of stress and accompanying elements such as dehydration, drugs, alcohol intake, muscle activity and body temperature."*

20. I have been asked about the reference to alcohol intake on page 3. I have been advised that the toxicology analysis was negative in respect of alcohol despite the references in some reports to Mr Bayoh drinking alcohol in the hours prior to death. I am asked Mr Bayoh's consumption of alcohol was of any consequence to my conclusions. No, not at all – this is just a general statement. I don't mention alcohol in relation to the cause of death.

21. I mean, it's a small point. If you drink a lot of alcohol or if you have done, you often end up a bit dehydrated. In terms of direct alcohol toxicity being of relevance, no.

Sickle Cell Count in Critical Organs

22. I have been referred to paragraph 2 of page 3 of my report:-

> *"In evaluating the clinical pathology, the critical aspects are the amount of sicking of red cells and in how many critical organs. There is no rigid morphological case definition – we must acknowledge – that separates harmless sickling from harmful sickling: it is inevitably somewhat*

[3] sickle cell trait

subjective, and informed by the observer's previous experience. And it must be acknowledged that changes in the body's tissue post-mortem can contribute to sickling of red cells. But the quantity of sickling here tells me that this is much more than just post-mortem sickling; it happened peri-mortem as part of the death processes."

23. In relation to this, I am asked what the amount of sickling cells in critical organs indicates to me. The critical organs in the body are the heart, the brain and the lungs in terms of acute events. You don't die acutely of liver failure or kidney failure or anything like that. So it's heart, brain and lungs that are key. Mr Bayoh has sickling in his brain tissue, some sickling in the heart tissue, but the crucial one is the lung.

24. I have some reservations about this, actually, because I would have, in a sense, been happier if all the slides of the lung showed sickling, but actually only half of them did. So there's a partial effect here, and if I wanted to emphasise quantitation, I would actually stick with that to say it's partial, which is why I think it's a contributor.

25. Reflection on my report four years on, I don't think sickle cell trait is quite as important as I thought it was perhaps back in 2018. That's partly influenced by lots of discussions I had with coroners in England about how we should be phrasing causes of death; what is important and what is less important, what goes into part one, which is the main thing, and what goes into part two as a contributor. If I was doing this case again now, I would move the reference to sickle cell trait part 2 of the death certificate. I would not frame the entire story of his death around sickle cell trait. I would just simply put that in on the end as small extra factor; as explained earlier, that it may well have shortened his life expectancy by a couple of minutes, given the stresses he was under at the time.

26. Again, that's guesswork. I can't quantify that seriously. No one can do that. This is a very recondite field. I have been involved in a number of inquests over

deaths involving sickle disease generally in patients with sickle cell disease and trait . On many occasions I have provided opinions that sickling played no role whatsoever; but conversely I have seen ones where it absolutely definitely did and, if they hadn't had sickle, they probably wouldn't have died when they did. I don't think this case comes into that category.

Sickle Cell Trait As "Part Of The Death Process"

27. I have been referred again to paragraph 2 of page 3 of my report:-

"There's no rigid definition that separates the harmless sickling from harmful sickling. It's subjective and informed by experience. […] But the quantity of sickling here tells me that this is much more than just post-mortem sickling, it happened perimortem as part of the death process."

28. I am asked what is meant in particular by "*as part of the death process".*

29. I'm comparing Mr Bayoh with all the other people who have had sickle cell trait and it did them no harm whatsoever. They've died of other things – diseases, accidents, whatever – and when I look at them, I see a certain amount of sickling. When I look at Mr Bayoh, I see more. It's simply a semi-quantitative estimation of how much sickling is going on, and that's all it is. I'm afraid it's subjective. There are no absolutes here, and you might find another pathologist to say, "Rubbish! We don't think that sickle could possibly have contributed," and I can't argue factually, I can just say, "My opinion, based on many years of experience in this field, is that I believe it contributed a bit."

30. I am asked to how I can tell that the sickling happen prior to death and contributed to the death, as opposed to it was sickling that occurred around the time of death as part of the process of dying. When people who have sickle traits, who have died of something else, die, you can get after death sickling that occurs because the body cools down and it becomes slightly acid, and that

can turn red cells sitting harmlessly in blood vessels into sickle cells, and we see that. There is a difference, actually. The sickle happening before as opposed to after death; this is partly based on the organ distribution of the sickling, and quantitation of the process, which is a subjective histopathological evaluation.

31. Almost certainly in Mr Bayoh, like in all the other cases, there's an element of both, because you don't autopsy people the moment they die or very rarely. So there's always going to be a little of additional post-mortem sickle trait, "it happens anyway sickling." I was making a comparison between the average sickle trait person who's died and this kind of case, like Mr Bayoh's, where there is more sickling, and it struck me it probably could have contributed to death. In other words, it happened in the time leading up to the point when his heart stopped. However, to emphasise for the again, in terms of importance and significance of the sickle cell trait, it's a lot less than the recreational drugs and the struggling and the restraint. Those are the important things here.

Respiratory Arrest

32. I have been informed that there is a suggestion that Mr Bayoh may have gone into respiratory arrest before he went into cardiac arrest. That's usual. The heart carries on when everything else stops and then it stops. Yes, that's pretty standard mode of death.

33. I am asked whether the sickling of cells I saw in the histology fits with respiratory arrest and then a cardiac arrest. Oh, yes. To the extent that one can pathologically distinguish, I think that's right.

Cause of Death

34. I have been referred to page 3 of my report:-

"I would suggest an alternative [cause of death]

1a. sudden cardio-pulmonary failure
1b. sickle cell trait, recreational drug use, struggle against restraint.

The last feature – struggle against restraint – can include positional asphyxia, but as a non-forensic pathologist, I do not wish to be drawn into a more detailed discussion in that area.

Importantly I do not think we can quantify the contribution of the three factors presented in 1b. and state, with rigor, that one is more or less important than the others. It is multifactorial."[4]

35. I think I would modify that statement now and make it a little less generous. Since I produced my report in 2018, I have been given much more information regarding the circumstances of Mr Bayoh's death information (i.e. more about the degree of restraint Mr Bayoh suffered), and more forensic opinion.

36. I would now actually put recreational drug abuse, struggle against restraint; I'm not going sort out which of those two is more important, but definitely put sickle trait in a different line as a less important phenomenon.

37. I am asked whether, but for sickle cell trait, Sheku Bayoh would have died. Yes. Now, I say that as a non-forensic pathologist. Just from what I've learned over 45 years of looking at dead people, and quite a lot of time looking at sickle patients particularly, it seems to be a very minor component here.

38. I am asked whether, in my view, sickle cell trait made a material contributed to Sheku Bayoh's death. Yes, but a small amount.

39. I am asked whether the drugs contributed to Sheku's death and whether the struggle against restraint contributed to Sheku's death. Clearly the original

[4] Page 3, Report, COPFS-00084

pathologist did. I don't feel I can answer that as that's question for a forensic pathologist.

40. I have been referred to my use of the term "positional asphyxia" and am referred to my statement that "As a non-forensic pathologist I do not want to be drawn into a more detailed discussion"[5]

41. Following that, I am asked what my limits of expertise are in relation to that, and whether it is simply because I am a non-forensic pathologist. I've used the term positional asphyxia as a cause of death or contribution to death in many a few cases over the years, but these are when there's no third-party involvement. This is people who have got drunk, taken drugs, and some have fallen asleep with the neck on the side of a rather sharp armchair, and have simply strangled themselves because they've been unconscious due to drugs and have died following positional asphyxia. That's the sort of circumstances I'm relatively happy with, but when it comes to other people creating the positional asphyxia, then I won't offer an opinion as I don't have experience of positional asphyxia causing death in these circumstances.

42. As you know very well, in ordinary coronial autopsy work in England and Wales, the answers are meant to be on balance of probability. Well, I do better than that, but when you're in court and when someone's potential imprisonment or not is at stake, that becomes beyond reasonable doubt, doesn't it? Simply, at positional asphyxia, I can't have an opinion on that sort of thing when there's third-party involvement. It's purely the forensic aspect. I don't see enough.

43. I am informed that the Chair will be seeking to determine whether there is evidence of asphyxia in relation to Sheku Bayoh's death. I am told this will rely

[5] Page 3, Report, COPFS-00084

predominantly on eye witness accounts of the restraint, weight, and force applied to Sheku's body by Police.

44. I am informed that there is also a question regarding petechial haemorrhages and whether evidence of such haemorrhaging is indicative of asphyxia.

45. I am asked that, in the event the Chair determines that there *is* evidence of asphyxia, how such asphyxia would have an impact on sickling.

46. If you're in a state of asphyxia, the blood oxygen level drops. There are lots of ways of arriving at asphyxia from hanging, strangulation, all sorts of things like that. The blood oxygen level stops and that's what precipitates sickling. So it's a final common pathway. The sickling is not interested in precisely why the blood oxygen is dropping, it just has dropped for one reason or another and then the sickling takes place.

47. I have been asked how I would separate that situation from someone who has sickle cell trait. Well, I've said it before, I'll just say it again. It's a comparison from memory of looking at people who have sickle cell trait who've died of other things, and they have less sickling going on in their body than he has. It's a semi-quantitative, historical, anecdotal comparison, actually, to be quite crude about it. That's what it is.

48. But I just want to emphasise that we know this sort of thing does matter because we know about deaths in athletes and in soldiers on exercises, and in those two scenarios – soldiers, athletes – asphyxia is not relevant. I'm merely making a point. In this particular scenario, how he arrived at the death was via asphyxia, in which, therefore, could and probably did precipitate a little bit of sickling by the time he died.

Signature of witness……  ……....

The Case Of Prince Fosu Death In Custody

49. I have been shown a chain of emails the first of which is from Alasdair MacLeod, COPFS to me dated 24 April 2020.[6] I am referred to my email of reply dated 25 April 2015 and the paragraph which states *"since this case, I have been involved in reviewing another death in custody in a person with sickle trait in London; in that particular case, I am confident that sickle trait played no role in the death, despite the opinion, the other way, of the forensic pathologists involved. At the inquest, the coroner believed me rather than the forensics."* I have been asked to provide a brief summary of this case.

50. Prince Fosu was a Ghanaian gentleman who died after being detained in in Harmondsworth Immigration Removal Centre awaiting deportation. He was experiencing a psychotic episode and he basically, I think, undertook a dirty protest, and didn't eat, didn't drink and died on 30 October 2012.

51. The Inquest took place in Fulham, West London in 2020. So it took eight years to get the inquest going. The man did have sickle trait. The forensic pathologist in the case said the main cause of death was cardio-respiratory collapse associated with sickle cell trait, 1A, just one line. Someone else, a very good Welsh forensic pathologist, Dr James said his cause of death, which I then agreed with much more enthusiastically, was cardiovascular collapse, dehydration, malnutrition, hypothermia, mania – subject to psychiatric consents; pathologists can't diagnose mania – and he put sickle trait under number 2 as a small contributor to the death. My opinion was that sickle trait did not played any role in his death; rather it was cold, hunger, malnutrition and neglect, because on histology there were very few sickle cells. He hadn't sickled enough. So it wasn't so relevant to his death. The inquest jury concluded that the medical cause of Prince's death was "A sudden death

[6] COPFS-02089

following hypothermia, dehydration and malnourishment in a man with psychotic illness", contributed to by neglect and multiple serious failures.

52. Conversely, in Bayoh's case, there's an awful lot of sickling going on, but I want to emphasise, this is highly subjective. I know that lawyers love having clear case definitions: someone's got a disease, or they have not. There are quite a lot of pathologist scenarios where you can say, for example, that there is a case definition for ischaemic heart disease, and if you don't fulfil it, that's not why they died. In sickle, and especially in sickle trait here, it is basically a slippery slope and there is no hard dividing line.

53. So the point is, in these sorts of scenarios, there are cases where, in my view, it's very obvious sickle significantly contributed to the death, and others where it played no role whatsoever, and you have a spectrum in between. It's a question of where Mr Bayoh is on this spectrum.

Dr Nat Cary's Report

54. I have had sight of Dr Nat Cary's report.[7]

55. I have been referred to the findings of Dr Cary's report indicating that Mr Bayoh was made profoundly hypoxic and acidotic by the struggling and restraint, and that he wonders whether that would, or could, provide for sickling. Yes, this is correct.

56. If you're going to sickle and, as explained, Mr Bayoh has sickled more than most sickle trait people do when they die, that's what causes it. Mr Bayoh might have sickled more than sickle trait persons might usually do from peri-mortem

[7] COPFS-00196

hypoxia, but it does not mean that he died because of sickling as an equal contributor to the restraint, hypoxia or acidosis.

57. Interestingly we're dealing entirely with forensics here. A lot of the information and the support for the concept of people with sickle trait dying simply because they have sickle trait comes from athletes. Another area is soldiers in the army on exercises, and the Americans learned this the hard way in the 60s and the 70s. An awful lot of troops in the American army are black, and they will go on forced marches in the summer and be dehydrated, not given enough water, and quite a few died, and that's when the American army changed their mind about what to do. It's not to stop individuals with sickle trait joining the army, they want them, but to make sure they're well hydrated and they don't overexert themselves, and that cut the death rate. So it happens under natural circumstances, not only under forensic. I have to say, if it wasn't for that evidence of the sportsman aspect of sickle trait very occasionally causing death, a lot of the other stuff would probably never have developed, because we needed something that was definitely non-forensic to be able to hang it on, then apply it into the forensic scenario.

Dr Soilleux

58. I have been referred to paragraph four of page three of my Report:

> *"Dr Soilleux presents a good diagram of the potential clinical pathology of death in this case, with which I do not significantly disagree apart from suggesting a more prominent component from the lung pathology."* [8]

59. Yes, I got it. What I'm really saying is that if sickle is going to contribute to death in this sort of scenario, it's the lung that is most affected and that's all I'm really saying. I don't think Dr Soilleux has experience of this particularly, because she won't have come across these cases before. The circulation through the lung

[8] Page 3, Report, COPFS-00084

is compromised, and if you're under a situation of restraint, also with all these drugs on board as well, that can in a sense shorten life a bit, but by how much is very difficult to say.

60. My conclusion is subtly but not terribly importantly different from hers.

Crown Email Containing My Report June 2018

61. I have been referred to my emails of 4 June 2018 to Les Brown of the COPFS, where I write *"I will not put this in my Report".* Do you know, if I was doing it again, I certainly wouldn't write that. What I can see of the scenario has obviously changed, because, I think, when I was sent all this original material, there was very little evidence or information or documentation about restraint. The death, in a sense, was slightly more spontaneous, but as I understand from what's happened in the last seven years or so now, actually, a lot more information has come out about what actually happened, and I wouldn't write that now.

62. I feel that this was an 'off-the-cuff' comment at the time.

63. I would like to recommend a book to the Inquiry which may be helpful, Sickle Cell and Deaths in Custody by Simon Dyson and Gwyneth Boswell. It's basically looking at North America and Britain, and the extent to which people who are black, who have sickle or sickle trait and died in custody of one sort or another, to what extent the system, which includes the pathologists and the investigators and the police, blame sickle for the deaths, when actually the deaths were caused by restraint or other things related to being in custody.

64. I believe 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.

Signature of witness…… [redacted] ……....

Date ……April 28, 2023 | 9:29 AM BST

Signature of witness…… [redacted] ……