Wednesday, 22 June 2016
(10.00 am)
Housekeeping
MR JUSTICE BLAKE: Yes. Good morning.
MR HEPPINSTALL: Good morning, my Lord.
If we could just have a discussion about SB22. It has grown overnight.
MR JUSTICE BLAKE: Yes. Is it going to stop growing?
MR HEPPINSTALL: I hope so, especially as we approach our last witness.
MR JUSTICE BLAKE: Yes.
MR HEPPINSTALL: They are mainly papers that I think are going to be put to Dr Haycock and he has had an opportunity to look at them overnight, for which we are grateful.
MR JUSTICE BLAKE: Well, I think we are going to have to draw a line because to some extent these learned papers are obviously very good for one's self-education into these topics, or not as the case may be, but ultimately they are mediated through the comments of the witnesses that we hear or the experts are able to evaluate them.
MR HEPPINSTALL: Indeed.
MR JUSTICE BLAKE: Bearing in mind the stage we are at, I think the time has come down.
Since all the experts who are here to assist the

Tribunal are here to assist the Tribunal, it does seem to us that if, once we've absorbed lines of inquiry that we think are relevant we then realise that there are questions, we may want to have to pose questions to experts. But we'll do that through transparent, normal channels so you can see what's going.
MR HEPPINSTALL: That's fine.
MR JUSTICE BLAKE: But we don't want more interesting questions and hypotheses and possibilities to emerge from learned papers because we do have to bring a close to it, but at the same time we want to do the best job that we can on the material that is available. So there we are.
MR HEPPINSTALL: Can I just check that there are -- all three parties have added --
MR JUSTICE BLAKE: Oh right, so it's a general update, is it?
MR HEPPINSTALL: I think we have got to hand ours out, although one e-mail --
MR JUSTICE BLAKE: I think our hard-working clerk has updated mine, although I suspect what last appeared as an index is no more than an indicator of what once it was.
I have also a feeling that I've departed from one of my annexes. I think I was meant to take them all and

relocate them elsewhere in the SBs but I've kept them all in SB22, so I suspect we might be a tab out. Otherwise, it was working yesterday.
Where are we up to now?
MR HEPPINSTALL: Operation Dominic went in at 14 yesterday.
MR JUSTICE BLAKE: Yes.
MR HEPPINSTALL: Then at 15 there's a male breast cancer incidence and mortality risk by Mark Little, put in my the Hogan Lovells appellants.
MR JUSTICE BLAKE: Yes, I have that. If we just slow it down and then my colleagues will have a chance to have their files updated.
MR HEPPINSTALL: Yes.
MR JUSTICE BLAKE: 16 is "Radiation exposure from CT scans in childhood"?
MR HEPPINSTALL: Yes.
17, I think we are to learn about, I hope in cross-examination because it appears to be an authorless, explanatory note, but I think Mr ter Haar is going to produce that in cross-examination.
MR JUSTICE BLAKE: Does that relate to the fact that Mr Hallard did revise calculations on Mr Abdale and we weren't entirely sure --
MR HEPPINSTALL: No, I don't think so.
MR TER HAAR: My Lord, it is our document. It's

authorless -- I can explain -- done by one of the members of the Hogan Lovells' team. It's simply an exercise in taking Dr Haylock's calculations set out in appendix 2 of his report and taking the first of those whom I represent, that's Mr Abdale, and feeding in what happens if you change the assumptions. It is purely mathematical.
MR JUSTICE BLAKE: Yes, I think interesting. Thank you very much.
MR HEPPINSTALL: We'll see how Dr Haylock responds.
MR JUSTICE BLAKE: Well, I am glad someone has done that.
MR HEPPINSTALL: 18, a paper by Greenland and Robins.
MR JUSTICE BLAKE: Thank you.
MR HEPPINSTALL: 19, another paper by Greenland, Bulletin of Atomic Scientists.
20 Beyea and Greenland, "The importance of specifying the underlying biologic model".
29, now I think this is from the BS appellants, "Chromosome Aberrations Determined by FISH in Radiation Workers from the Sellafield Nuclear Facility".
21, sorry.
MR JUSTICE BLAKE: 21, and we've seen some papers by some of these authors in the BS, tab 6.
MR HEPPINSTALL: I think so.
MR JUSTICE BLAKE: Right.

MR HEPPINSTALL: 22 is --
MR JUSTICE BLAKE: Chromosome aberration.
MR HEPPINSTALL: Chromosome analysis.
23 is a World Health Organisation paper.
MR JUSTICE BLAKE: Now, I think -- well, we do not have a tab at the moment.
MR HEPPINSTALL: Ah, I think there are some new tabs for you (Handed).
MR JUSTICE BLAKE: All right. So these are blank tabs. Oh right.
23?
MR HEPPINSTALL: Is what has just been handed to you.
MR JUSTICE BLAKE: "Ionising radiation health effects of Chernobyl."
MR HEPPINSTALL: Yes. Again that's from the BS appellants.
MR JUSTICE BLAKE: Yes.
MR HEPPINSTALL: Then 24 and 25 are going to be two documents from the Secretary of State which I'll hand up.
MR JUSTICE BLAKE: I think bundle 22 is now beginning to complain that it is --
MR HEPPINSTALL: 24 is the Health Protection Agency's response to the Rowland paper, because I detect that there is going to be some cross-examination of Dr Haylock at that point. It struck me if someone

wanted his organisation's response to Rowland, the relevant expert agency have already provided it. It's from the library B12/157.
MR JUSTICE BLAKE: It's from the library. This is moving from these documents to this bundle, yes.
MR HEPPINSTALL: Then at 25, this is "A history of the New Zealand Navy and the British nuclear test programmes" by JAB Crawford. It's a disclosed document. I can't actually now recall whether I handed it up to the last FTT but it didn't go into the bundles. So this is not in the library, but, rather than speculating on what the New Zealand sailors did, this actually tells you what they did.
MR JUSTICE BLAKE: So this is a bit of historical data?
MR HEPPINSTALL: Yes, it's a history commissioned by the New Zealand Government, by Headquarters New Zealand Defence Force in 1989. Crucially there is a table at the back which tells you --
MR JUSTICE BLAKE: Can you just hold that whilst I catch up? We now just have tab 24.
MR HEPPINSTALL: Yes.
MR JUSTICE BLAKE: Hang on.
MR TER HAAR: I am losing track of this. My learned friend is rattling through this, handing up documents to you and then coming late to us. So at the moment I do not

have the references. I wonder if my learned friend would start again with his reference so I can identify what it is.
MR HEPPINSTALL: So at tab 24, "Comments on New Zealand nuclear test veterans study by cytogenetic analysis" by Rowland.
If you look at the end, this is from the Radiation Protection Division, Health Protection Agency, 25 July 2007, so the predecessor body to Dr Haylock's current employer.
That was in the library of documents at B12/157.
Then tab 25. It's quite a long document and that's why I think it's set out in this format, a bit like the transcript although it does make the text hard to read. But the first page gives us the document UIN, if anybody is interested, so we know this has been disclosed in these proceedings. Then you'll see, actually, how it comes into the Secretary of State's possession because in fact --
MR JUSTICE BLAKE: 2005?
MR HEPPINSTALL: The New Zealand Defence Force was asking for the Chief of Defence Staff's assistance with some information. Then over the page, you start to get into the report itself.
MR JUSTICE BLAKE: Okay. Well, I think we have what it is.

MR HEPPINSTALL: Fine.
MR JUSTICE BLAKE: You don't want us to read it before we hear Dr Haylock?
MR HEPPINSTALL: No.
MR JUSTICE BLAKE: But I think I see it's a typescript document.
MR HEPPINSTALL: Yes, there's a very useful table at the end.
MR JUSTICE BLAKE: The table at the end that you were -- hang on ... is that appendix 11?
MR HEPPINSTALL: Sorry, I just shut my bundle.
MR JUSTICE BLAKE: Appendix 2. No.
MR HEPPINSTALL: Appendix 1 gives you the test. Then I think the location of the -- yes, appendix 2, exactly, it tells you where they were during the tests.
MR JUSTICE BLAKE: Right. Okay.
I anticipate that's all relevant when we come to look at the Wahiban(?) --
MR HEPPINSTALL: Yes, I just thought instead of speculating about what they did, someone has thought about it and written it down.
MR JUSTICE BLAKE: So that's the last update, is it?
MR HEPPINSTALL: Yes, let's hope so.
MR JUSTICE BLAKE: Right, well, let's just see what the bundle 22 is looking like groaning under the weight of

this learning. I think probably that's getting close to absorption point. Does anyone anticipate or expect that any further data is to be supplied to us of this sort? No? Okay. Good.

Right.

MR HEPPINSTALL: Dr Haylock.

MR JUSTICE BLAKE: Yes. Just give me just one moment to put this ... He can come up.

DR RICHARD HAYLOCK (affirmed)

Examination-in-chief by MR HEPPINSTALL

MR JUSTICE BLAKE: Right, are you happy to give your evidence standing up?

A. Erm --

MR JUSTICE BLAKE: If you need to sit down or you would be more comfortable, I think you may be here for a substantial part of the day but others know better. If you do sit down, make sure you can keep your voice up so we can all hear you.

THE WITNESS: Okay.

MR JUSTICE BLAKE: Although the record is being taken it's quite useful to understand you as we go along as well. Yes?

MR HEPPINSTALL: SB2, tab 2.21, please. You'll find the SB bundles to your right and it should be the second from the left.

Turn to tab 2.21. Is that your first report this to Tribunal?

A. Yes, it is.

Q. If you turn to the last page of that report, just under section 2.5 -- Mr Battersby -- so not the references, just before then, the last substantive page, there's a heading "Statement of truth" and your signature. Do you see that?

A. Yes.

Q. Is that still the case today?

A. That's still the case.

Q. Then 2.22, please. You were asked questions by Dr Christopher Busby and Group Captain Andrew Ades. Are these your answers to those questions.

A. They are.

Q. Then, finally in the next tab, 2.23, you provided a supplementary report to the Tribunal in respect of a report provided by Professor Howard relating to the Rabbitt Roth survey carried out in 1998. Is that your supplementary report?

A. It is.

Q. Then, again, on the last page, you signed another statement of truth. Is that still the position set out in that statement of truth today?

A. It is.

MR HEPPINSTALL: I have no further questions.

MR JUSTICE BLAKE: Thank you very much.

Cross-examination by MR TER HAAR

MR JUSTICE BLAKE: Yes.

MR TER HAAR: Dr Haylock, good morning.

A. Good morning.

Q. You may well be aware that I represent some 12 appellants listed starting with Mr Abdale.

A. Mm-hm.

Q. And the questions I am going to ask are on behalf of those appellants.

Do you still have that bundle still open? Could you go back to tab 2.21, where you'll find the first of your reports.

A. I have it.

Q. Unfortunately it's not paginated. It's one of the annoyances of the Word system, I find, that you have to remember to press the "paginate" button. But at any rate, could you go through to the end of your report, and then on after the very helpful references and glossary to the appendix which has your curriculum vitae.

A. Uh-huh I have it.

Q. Now, you set out there your academic qualifications. A first degree and a second degree from the University

of Leicester and then a doctorate from the University of Nottingham?

A. That's correct.

Q. Your first degree in mathematics is pure mathematics presumably?

A. That's general mathematics so it incorporated some aspects of and pure, applied and statistics.

Q. Then you did an MSc in medical statistics and information technology?

A. Yes.

Q. To what extent would that involve you learning about medicine as opposed to learning about the application of statistics to medicine?

A. It involves nothing about medicine itself. It's just about the application of statistics to medicine.

Q. Then you did your doctorate in statistics with a thesis title that starts to make me go cross-eyed but certainly looks to me like a very mathematical exercise in statistics.

A. That's correct. It's a very mathematical part of statistics, yes.

Q. Then you set out your employment. And do I get the impression right that your role is to take epidemiological data and with the expertise you have of statistics, but particularly statistics relating to

illness and morbidity and mortality, to process the information which comes to you?
A. Yes, that's correct.
Q. And clearly you are very dependent upon medical judgments made by others in order to make sense of the statistics that you are considering?
A. To some degree, in that, for example, we rely on death certificates so we rely on the accuracy of those, and on the accurate diagnosis of cancer incidences so that's probably the two main respects in which the statistics rely upon directly medical expertise.
Q. Yes. Obviously -- I say "obviously", perhaps you'd agree with me -- in assessing statistics often a degree of history and common sense has to come into play in order to try to understand where the bias might be in the data you are considering?
A. Absolutely, yes.
Q. So if we look back in the main body of your report at the third page, the top of the page starts:
"The epidemiological evidence for deriving the risks to adverse health effects."
Do you have that page?
A. Yes.
Q. You are there dealing with the lifespan study or the LSS. Clearly in considering the LSS you have to

understand the history of the people who are being considered?
A. Indeed, yes.
Q. And I'll come back to some aspects of that in due course.
Now, just so I can see where your assistance to this Tribunal starts and ends could I ask you please, to take up bundle SB11. To give yourself space do put the bundle with your report in it back on the shelf to make a little bit more space.
Turn to the very first divider, please.
A. Mm-hm.
Q. You should find there a relatively short report from Dr Brenner who we can see if we go to page 6 of this is not only a PhD but also a DSc, and he is a Professor of Radiation Biophysics, Director of the Center for Radiological Research at Columbia University Medical Centre?
A. (Nodded assent).
Q. Was he somebody of whom you are aware? Do you know his reputation?
A. I know him by reputation. I think I've met him once or maybe twice.
Q. In two of the experts' reports filed by the Secretary of State in the earlier proceedings -- I think

you are aware there were earlier proceedings --
A. Mm-hm.
Q. -- Dr Brenner was described by Dr Lindahl as eminent and by Dr Darroudi as very well respected. Would those descriptions of Dr Brenner accord with what you know of him?
A. As far as I know, yes, I would.
Q. His report comments upon -- this report as he has here is primarily concerned with the Wahab and Rowland Report on chromosomal abnormalities which have been identified -- there's some argument about it, but identified among the New Zealand naval representatives.
A. Mm-hm.
Q. Would I understand that your speciality is not one which would enable you to comment upon what a radio-biophysician has to say about that study?
A. That's correct. I'm not an expert on fish or any of these techniques, so I would not want to comment on that sort of study.
MR JUSTICE BLAKE: Fish and mFISH --
A. Fish and mFISH.
MR JUSTICE BLAKE: -- are therefore --
A. Related.
MR JUSTICE BLAKE: -- medical analyses as opposed to statistical analysis?

A. They are biological techniques, not medical techniques.
MR JUSTICE BLAKE: Biological techniques.
A. Biological techniques.
MR TER HAAR: I am going to take you in a moment to a paper which sets out the history of it, which you may or may not be able to confirm from the interplay between your specialisation and the pure medical world.
Getting closer, though, to your field of expertise is the next tab, that's to say tab 4, where we have a report from Professor Parker.
Just so we can see what her expertise and experience is, her report goes through to page 116, so if you get to the very last page of her report. It's a long way on.
Immediately after that you'll find her CV, personal details.
A. Yes.
Q. We can see her experience. She's also an epidemiologist coming from a rather more medical background?
A. Mm.
Q. Is Professor Parker somebody of whom you've heard, are you aware?
A. Only in connection with this case. I've not heard of her previously.
Q. But you would accept, wouldn't you, that she is somebody

who has a specialist interest in the epidemiology, in particular in relation to the effects of ionising radiation?

A. That may be so. But as I said, I've only known of her through this case. So I don't have independent evidence of that.

Q. But at any rate, you have no reason to doubt her expertise?

A. I have no evidence one way or the other, I'm afraid.

Q. But what she does do in her report is to carry out an extensive survey of the various epidemiological studies which have been carried out which are of relevance to especially whether or not radiation may or may not have caused some cancers and other diseases in what we call the nuclear veterans.

Have you been given the opportunity to read her report and look at her comments on those epidemiological studies?

A. I have looked at it but not in great detail.

Q. So for me to ask you detailed questions on this would be pointless?

A. I think so, yes.

Q. But when you reviewed it, albeit not in depth, was there anything which you came to the conclusion was outside the range of views which a competent epidemiologist

could hold?

A. Some of the views appear to be somewhat different to views I would probably have held given the same information.

Q. That's an answer to a slightly different question. That's why I put it as I did.

Obviously in science, as in other areas, views can differ.

A. Mm.

Q. What I am asking you is whether or not the views which she expressed are within the range of views which a competent epidemiologist could hold.

A. I think I would have to look at each point in particular. I think it's -- maybe some of the points she raised I would agree; others, possibly not.

Q. Well --

MR JUSTICE BLAKE: So just for me to understand your answer, you would agree that there is a difference between you?

A. Yes.

MR JUSTICE BLAKE: It's still a difference within a range of reasonable options but some you think would fall outside that?

A. Professor Parker makes a lot of different statements in this report, some of which I would say are potentially reasonable, others I would say are perhaps not.

MR JUSTICE BLAKE: Okay.

MR TER HAAR: Let's see if we can do this quite quickly in this sense. Go to page 113. She is commenting there upon, in section 11.1, upon the NRPB studies.

First of all, were you involved in the NRPB studies into the nuclear veterans?

A. Yes.

Q. I thought you were. And so maybe this is one area where you might not entirely agree with her. Can we just look at some of the points she makes here.

If you look at the numbering, you see in paragraph 1 it says this:

"As a result of inadequacies in the Ministry of Defence record keeping, it was not possible to fully and unambiguously identify all participants."

You'd agree with that statement of fact?

A. I don't have information on that. As I said, I am an epidemiologist statistician and I analysed the data we have. I was not involved in the original collection and setting up of the cohort. That occurred before I joined the organisation.

MR JUSTICE BLAKE: So you don't know whether there were missing participants or not?

A. No.

MR TER HAAR: I think it would follow from that that you're

not able to comment on item 2:

"Participants not included in the NRPB studies are likely to have a poorer outcome than those included."

A. I think you would have to know who were the people who were not included to be able to make that statement. I don't know how she knows that if they're not included in the studies.

Q. Well, let's look at her reasoning.

It's in a number of places but if we go back to page 4 and 5. She first of all at page 5 comments on the uncertainty of who the participants were at paragraph 2.1.6.

A. Mm-hm.

Q. And those certainly appear to be valid comments, do they not?

A. Like I said, I was not involved in setting up the cohort so I can't comment on how that was set up or what steps were taken to do it.

MR JUSTICE BLAKE: Can you comment on 2.1.7 where, as I read it, in summary she suggests that of the three NRPB reports the second excluded 514 who had been included in the first. That looks like that's something within the NRPB dealing with the data as opposed to what was the data provided to the NRPB in the first place.

A. It could well be. I would have to look at exactly why

these 514 were excluded to comment.
MR JUSTICE BLAKE: You don't have that at your fingertips?
A. No, I'm afraid not.
MR TER HAAR: Then if you go on to page 7, this is the section where she deals with the second point I was putting to you earlier that participants not included are likely to have a poorer outcome.
She says this at 2.1.10:
"There are a number of further challenges in determining the extent to which the health outcome of the participants have been affected by their involvement in atomic weapon experiments. Some of these are the inherent difficulties of retrospective epidemiological studies and some were created and exacerbated by the inadequacy of the record-keeping at the time in the aftermath of the experiments. In particular: that the group of all participants is unknown means that the questions to the extent and nature of health detriments that may have been experienced by participants cannot be fully answered. The table on page 16 illustrates the extent to which the groups identified in the NRPB studies do and do not overlap and emphasised that there remains uncertainty in identifying the group of all true participants. This is discussed briefly here in more detail in section 2.1.10."

MR JUSTICE BLAKE: Isn't this section 2.1.10?
MR TER HAAR: Yes.
MR JUSTICE BLAKE: I marked that up and I am confused of (inaudible) here.
MR TER HAAR: Yes.
We can see, though, that she does, further on in this section, actually deal with some of the problems.
If we go on:
"The group of participants included in the NRPB second and third reports was estimated by its authors to include around 85 per cent of all participants. It is important to also acknowledge that those categorised in the NRPB studies as participants will also include some non-participants. This again is discussed in more detail later in this document at section 4.3.4. The extrapolation of any findings from this 85 per cent sample to the larger group of all participants depends critically upon whether that missing 15 per cent of missing participants was likely to be similar or different to the known 85 per cent in terms of their exposure and experience of health outcome. There are two major pieces of evidence to suggest that two groups were probably different. Firstly, as detailed extensively in the NRPB R214 there were challenges in identifying the records of those participants who had

issues with their health which had been drawn to the attention of their employers. These records could have been removed without any flag being put in place to point either to their existence or to fact they had been removed and for what purpose. Thus this creates a situation where less well participants were less likely to be included in the NRPB studies."
That's the first point she makes. That's at least a plausible view, isn't it?
A. Yes, it is.
Q. Then she goes on:
"Further evidence is provided by Rabbitt Roth in her report ..."
Her more recent publications.
Then if we go a bit further down:
"Roth provides evidence of under ascertainment of serious health outcomes such as multiple myeloma, overall in the NRPB study, and especially in the estimated 15 per cent of participants not included in the NRPB studies. Roff proposed that the rate of some adverse outcomes (i.e multiple myeloma), was twice as high in the excluded 15 per cent of participants than in the 85 per cent included. This may be contributed to by the fact that ascertainment of RAF participants, who may well have had the greater radiation exposures, may have

been as low as 74 per cent."
Just stopping there, I know that on behalf of the Secretary of State there are substantial caveats expressed in relation to Rabbitt Roth, but the specific point that she deals with in the last sentence there would be significant, wouldn't it, if the RAF participants were under-represented?
A. If their mortality rates were different to the rest of the other servicemen it potentially could, yes.
Q. Well, one of the reasons why it's suggested that that might be a problem is this. Some of the hapless RAF chaps were told to fly right the way through the middle of the nuclear cloud and therefore on any view got absolutely massive exposure to radiation and there is some evidence before the Tribunal that that resulted in incidences of very severe cancer cases. Are you aware of that?
A. Only in general. I've not looked at those cases in any detail.
Q. I think --
A. It's not relevant to the kind of work we do.
Q. You are coming from a different world in order to give such assistance as you can. I totally understand that.
But I think what you are agreeing with at any rate is this. On the first point, if there had been

effectively some form of pre-selection of the records in the sense some had been extracted because people who are ill needed to get treatment, if so that would be a valid point to take into account?

A. It would be. I can't comment on whether that actually happened because, as I said, I was not employed and I didn't -- I was not involved in the actual setting up of the cohort. As I said, the important thing is that the cohort that we see is a representative and unbiased sample of all those people at the tests. That was obviously the aim of the people who were setting it up. Whether they succeeded, I don't know.

MR JUSTICE BLAKE: Who was involved in setting it up?

A. My predecessors at the NRPB.

MR JUSTICE BLAKE: So that would be an NRPB selection process.

A. Yes.

MR JUSTICE BLAKE: Rather than simply something that had gone on before it gets to the NRPB.

A. No, it was an NRPB --

MR JUSTICE BLAKE: But it's not within your personal knowledge?

A. No.

MR JUSTICE BLAKE: Is it within the institutional knowledge of the NRPB?

results. What I am suggesting is that when you look at the exercise they've carried out it had the disadvantage, as this Tribunal has the disadvantage, of having to look back historically 50 years or however many years it was then, and inevitably if you are trying to look back that far the records are not likely to be as complete as you might hope for, so the controls are not up to, if you like, the best standards of an epidemiological piece of research?

A. Yes, the standard of research is dependent directly on the quality of the data.

Q. Can we go back to the conclusions, so back to page 113. The third point which she makes:

"Controls included in the study were likely to be healthier than all eligible controls."

Do you have the reference?

A. Sorry --

Q. Page 113, paragraph 3, towards the top of the page. Do you have it?

A. "Overall summary of conclusions."

Q. Then paragraph 3:

"Controls included in the study were likely to be healthier ..."

A. Uh-huh.

Q. The point being raised here is that by and large what

A. Yes.

MR JUSTICE BLAKE: Presumably epidemiology has to identify what it's doing to verify --

A. The very first thing of a study is to identify what the group of people you are going to study is. As an epidemiologist the overriding factor is to make sure your sample is unbiased. Even if it contained 50 per cent or 60 per cent or 70 per cent of the overall population the important thing is that it's an unbiased sample and it doesn't choose particular high dose people or low dose people or all RAF people or all Army. It needs to be unbiased in the sense that it is representative of the population that it is trying to represent.

MR JUSTICE BLAKE: Right. But you would accept that if, in selecting the sample that is going to be used for the study, people who had reported health conditions to their employers and for some reason those health records had been removed and therefore they were not visible as such and had been excluded from the study that would be a potentially biasing factor?

A. Yes, it would. Whether that happened or not --

MR JUSTICE BLAKE: I have that bit.

MR TER HAAR: Just to be clear, I'm not in any way suggesting that the NRPB deliberately skewed the

the Armed Forces did was to collect young, healthy men to go off to go and do hard work in the South Pacific, and hence the point she makes, which appears to be a valid one, I suggest, that "controls included in the study were likely to be healthier than all eligible controls".

A. Well, that would not have been the aim of the selection of the controls from an NRPB point of view. We would aim to have the controls as close in their selection as to the cases as possible apart from the fact of going to the test. Whether this happened or not, as I said, it's down to the people who set up the cohort in the first place and I was not one of those. So --

Q. Item 4, a statement of what the study actually carried out:

"Only cancers and deaths were included in the definition of health outcome."

Can you confirm that?

A. Yes.

Q. That's because, and again it's perfectly natural, that's where the main focus of concern had been, but nevertheless it means it wasn't a full across-all-diseases study?

A. No, no.

Q. Then at 5:

"Cancers and deaths in participants were incompletely ascertained because (a) there was no cancer registration in the UK before 1971; (b) post 1971 cancer registration was incomplete (c) death registration was incomplete; (d) some participants were lost to follow-up due to poor quality identifying information; (e) deaths and cancers occurring abroad were not identified."

Again, all of those seem to be sound points, don't they?

A. But the issue would be if these issues applied differently to the cases and the controls. Because I would not expect any of these issues to apply differently to the cases and the controls, the numbers and the data you get out are still comparable between the two.

Q. They are comparable but with what has to be recognised as substantial caveats?

A. I would not say "substantial".

MR JUSTICE BLAKE: So as long as problems about cancer registration are consistent between your control and your sample group --

A. In a sense the two groups are still comparable.

MR JUSTICE BLAKE: It doesn't skew the results. It's only if your study group or your control group is disproportionately affected by --

A. Yes, then it would not be a fair comparison.

MR JUSTICE BLAKE: Yes.

MR TER HAAR: Well, the problem is, just taking the first point -- no cancer registration in the UK before 1971 -- without such hard data it's very difficult to be sure that your control is comparable. It may or may not be?

A. I have no reason to assume it wouldn't be. We do a lot of work with cancer incidence statistics and from what I've seen from other studies it appears to be very reliable. And a point to take into consideration is that decades ago, cancer was more quickly fatal so the difference between -- sort of before 1971, the difference between cancer incidence and mortality would have been much less. The chances are that you've got a cancer you died fairly quickly. That's not becoming the case these days. People survive cancer a lot longer, so the difference between cancer incidence information and mortality information is beginning to diverge. But in these early days, particularly for main causes of cancer, that was not the case.

Q. Well, let's --

MR JUSTICE BLAKE: Cancer registration is a specific form of registration of death as recorded? What is cancer registration?

A. Sorry, there is a National Cancer Registry in the UK.

So you have a cancer diagnosis, that is then passed to the registry and is put on your record.

MR JUSTICE BLAKE: But on your death certificate if you die presumably it would tell you --

A. If you die of cancer, it would also say exactly the same thing. You might be diagnosed with a lung cancer, that incident is passed to the registrations scheme.

MR JUSTICE BLAKE: Right, but does cancer registration -- sorry to interrupt you but just to try and catch up my understanding -- cancer registration includes people who are diagnosed with cancer but die of it?

A. Yes.

MR JUSTICE BLAKE: Got you.

A. But before 1971 the difference in those two was quite small but is now diverging.

MR JUSTICE BLAKE: Yes.

MR TER HAAR: Well, I don't want to spend too much time on this but let's take point (d):

"Some participants were lost to follow-up because of poor quality identifying information."

That would be a concern, wouldn't it?

A. It would. I believe the proportion lost to follow-up, though, is very small and again there would be no issue unless that differed, that proportion differed significantly, between the two populations, the cases in

the controls -- sorry, the veterans and the controls.

Q. Let's look at point 6:

"As a result of inadequacies in the Ministry of Defence's monitoring procedures and record-keeping, the data on radiation exposure was unreliable."

Then she sets out four features.

Now, this is outside your expertise, isn't it, as to whether the record-keeping of exposure was unreliable?

A. Indeed.

Q. Over the page, 11.1.3, it says this:

"These limitations of the NRPB studies and the cumulative misclassification of participant status, exposure status and health outcome (death and cancer) status severely limit their ability to identify and quantitate any deficit in health outcome of the participants in the atomic weapons testing exercises. Thus these studies did not report an increase in solid cancers and other health outcomes in participants cannot be taken as evidence that such excesses have not occurred."

I think from what you said you disagree with that view?

A. I would.

Q. You can see the logic by which she's come to that view?

A. I can see, based upon her point over the page, why she

would come to that view, yes. But I don't agree with it.

Q. I understand that.

But can we go on though to 11.1.4 and this is where I want to see what the interface is between your expertise as a statistician and the medical world.

The background to this is do you remember we stopped at Dr Brenner's report, the American gentleman who comments from the point of view of a radiation biophysicist on the Wahab and Rowland survey?

That's now referred to here and elsewhere in this report by Professor Parker.

"The main findings of these studies were an increased rate of leukaemia and an apparently transient increase in the rate of multiple myeloma in participants. Leukaemia is considered to be the most radiogenic adult cancer and these results are consistent with an effect of radiation exposure."

First of all, you wouldn't disagree with that, would you?

A. No, that's -- that seems correct.

Q. "Given the limitations of the NRPB studies and the fact that misclassification as occurred acts to obscure any real effect, the implication of these findings is that the radiation exposure in the participants was likely to

be higher than that recorded."

There, I think, you differ from her?

A. In a sense our study, where we're comparing the veterans to the controls, takes no account of what radiation exposure the veterans might have had. The whole idea is to compare the population -- the two populations -- and at a population level see: do they differ? And we do see a difference in the rate of leukaemia.

Q. Then if we go on:

"That exposures were underestimated is consistent with the findings of chromosomal changes in New Zealand participants (Wahab et al, 2008), high rates of leukaemia in New Zealand participants (Pearce et al, 1996), chromosomal changes [I think that should be 'in UK participants', the letter is referred to] ... and elevated rates of several cancers including leukaemia and solid tumours in Australian weapons testing experiment participants (Carter et all, 2006)."

Now, would I be right in thinking that those comments in that sentence need to be assessed by somebody with medical expertise in order to understand exactly what those studies are showing?

A. I'm not an expert on the Wahab paper to comment on that and certainly not on the chromosomal changes bit. So ...

MR JUSTICE BLAKE: You can't comment?

A. No, I can't comment.

MR JUSTICE BLAKE: Could I just see I've understood your answers on the two parts of 11.1.4.

You disagree with the last sentence, but if the premise of all the criticisms and observations made at 11.1.1 were factually sound, would the accumulation of those omissions and problems -- I'll call them that -- justify the conclusion that it severely -- what was it? -- severely limits the ability of the NRPB to identify and quantitate any deficit in the health outcome?

A. I think it would depend upon the extent to which any of these factors she alludes to were present.

MR JUSTICE BLAKE: I am asking you hypothetically. If you don't think you can answer it, don't, but I mean if all these factors were present would that have the capability of justifying "severely limit"?

A. In sufficient severity, yes.

MR JUSTICE BLAKE: Right.

Secondly, then going down, leaving all that debate on the back-burner for the time being, what I think I understand that second part of 1.4 to be saying is I think there are problems with the extent you can rely upon the NRPB studies, and if elsewhere you are getting

evidence of high rates of chromosomal changes and/or leukaemia in biological studies, if I can call them that -- biological, medical -- of other veterans, if you did have that, again is that capable of raising questions about the NRPB studies?

A. In our study we do acknowledge that there is a higher incidence of leukaemia amongst the participants compared to the control. So that is an agreement with what was said in Australia.

MR JUSTICE BLAKE: So the factor of higher leukaemias wouldn't in your view severely limit your study? Indeed, that is what you are saying?

A. No, we agree with that.

MR JUSTICE BLAKE: Okay.

A. With regard to the chromosome aberrations, the issue is just because you are seeing increased chromosome aberrations doesn't necessarily mean you are seeing increased cancer.

MR JUSTICE BLAKE: Right. Okay. Can I just record that. (Pause). Yes?

MR TER HAAR: Could I just pick up on that last point, Dr Haylock?

A. Mm-hm.

Q. I think you may be help this far. There are at least two schools of thought as to the extent to which

chromosomal aberrations may be an indicator or a link to cancer. There are some who say that it can lead to cancer or be a sign that cancer will follow, some who say you can't make that link.

Do you agree with that as a general proposition first of all?

A. Yes.

Q. But what I think is common ground is that certain forms of chromosomal aberrations, in particular translocations, are a powerful indicator of exposure to ionising radiation?

A. As I said, I'm not an expert on that, but my belief is that there is a -- there is -- that is the case. But I'm not an expert on chromosome aberration.

Q. You may at least be able to help this far: and the received wisdom is that that sort of chromosomal aberration is an indication not only of exposure to ionising radiation, but to a high dose of ionising radiation?

A. I'm not prepared to comment on that. I'm not --

Q. Outside your territory?

A. Yes.

Q. Fair enough. We have papers in the files which support that view.

What I want to do is go on to another report. This

is at tab 7 of this bundle, and this is from Dr Mothersill.

Now, again, in order to identify who she is, if you go to the end of her report you get to numbered page 27, and then there's a unnumbered page after that with her signature on it. Then you should find her CV immediately after that.

Now, she originally appears to have come from studies in Dublin to do with zoology, but then she has moved, if we see her employment history, through into the world of medical physics having from 1983 to 1985 being a lecturer in medical physics and radiation biology. She was seconded half-time to run the Radiation Research Group at St Luke's Hospital, and later at the Nuclear Energy Board in Dublin.

995 to 2003, Scientific Director of the Radiation and Environmental Science Centre at DIT. I'm not sure what DIT --

MR JUSTICE BLAKE: Dublin Institute of Technology.

MR TER HAAR: Thank you. I should already have got that.

Then we see from 2003 to the present, to 2010, she was Professor and Tier 1 Canada Research Council Chair at the Department of Medical Physics and Applied Radiation Sciences at McMaster University, Hamilton, Ontario.

Again, a rather more medical and biological background than yours?

A. Mm-hm.

Q. What she tells us -- this is where I think your world and the world of the more medically qualified come together -- is about problems of uncertainty in what she describes as the low dose regions. Can I take you back to page 6, please.

Paragraph 3.3, "Old paradigm":

"The old paradigm basically holds that there is a linear relationship between radiation dose and biological effect."

Now, this is slap bang in your territory, isn't it, as epidemiological research?

A. Yes.

Q. It's the result of epidemiological studies over the years. Would you agree?

A. Yes.

Q. "It holds that DNA is the critical 'target' for radiation damage and that the DNA double strand break is the critical lesion. The number of double strand breaks can be directly related to the dose. Arising from this DNA damage, chromosome aberrations can occur due to changes in the sequence of DNA bases (code sequences). It should be noted that the old paradigm held that low

dose chronic irradiation does not necessarily have as great an impact as a brief higher dose exposure - a division factor of 2 was applied to the 'dose' if this was accumulated of (sic) a long period. The direct relationship between dose and DNA damage lent weight to the LNT model which was supported by high dose epidemiological data from the Japanese A-Bomb survivors who had an increasing rate of cancer incidence as the dose received increased. To determine risk at low doses, the high dose data were extrapolated to zero dose where there was zero effect. Of course in the low dose region, it was not easy to assign causation to radiation exposure due to the high background instance of cancer and other diseases associated with radiation."

Now, first of all, as a statement if you like of what certainly has been conventional wisdom would you agree with what she says in paragraph 3.3?

A. Some of it.

Q. What do you disagree with?

A. I think from an epidemiological perspective the bit about the exact mechanism is not particularly relevant. We are looking -- we don't need to know what is the particular mechanism to define the best mathematical relationship between risk and dose. There we are going through a mathematical process of finding in some sense

the best model which represents the data.

So knowledge about any underlying mechanisms or hypotheses about them don't necessarily have to figure on that.

So the epidemiological process is of trying to fit a model to the data and finding the best model. We don't necessarily need to know about what the underlying mechanism is.

Q. Except for this: that if you understand the underlying mechanism you may need to re-visit the conclusions which come from the pure application of mathematics?

A. Yes.

Q. That I think is what she then proceeds to do, and tell me when it stops being in your territory, paragraph 3.4:

"Uncertainties in the low dose region."

I think before I go into this you would agree with me that the low dose region is an area where statistics have real problems?

A. There are difficulties because of the lack of statistical power, yes.

Q. And I'll come back to what UNSCEAR have to say about it. You probably know the 2006 UNSCEAR report where they deal with this at some length, don't they?

A. Yes.

Q. Let's look at what Dr Mothersill had to say:

"While LNT predicts that some cancers or other diseases will occur due to low dose it cannot say which cancers were or were not due to radiation exposure."

That's obviously true?

A. Yes.

Q. "The difficulty is the variability of response at low doses. There is no doubt that high doses of radiation are toxic and carcinogenic and some extrapolation from a high to low dose can be made."

So far, so good?

A. Yes.

Q. "The difficulty lies in determining when this extrapolation is no longer valid."

Again, yes?

A. (Nodded assent).

Q. You are nodding?

A. Yes.

Q. You agree?

A. Yes, sorry.

Q. The transcript doesn't pick up a nod.

MR JUSTICE BLAKE: If we get an answer then we can record it.

MR TER HAAR: "The precautionary principle and the route taken by all radiation protection groups has been to say that any radiation dose has a potential to cause harm."

I think again that's a correct statement?

A. Yes.

Q. It is probably obvious from your point of view.

"The difficulty with this position is that we are exposed to natural radiation constantly from the earth, from food and tobacco, from each other and from air travel. We are also exposed to medical radiation. For this reason, effective thresholds have been set at which epidemiological studies have not had the power to detect any adverse risks to a population."

So if we just look at -- I think the Tribunal are probably aware of biostatistical power but can we just look at that for a moment.

If you survey a million people and discover that 990,000 get a cold every winter you can say people are likely to get colds in the winter with a fair degree of certainty.

If you study a million people and you find that one person gets a cold, it's very difficult to draw any conclusions from that.

The difference -- I take a very extreme case -- is statistical power?

A. Statistical power is the ability of the study you're referring to detect an alternative -- whether an alternative hypothesis is true compared to your null

hypothesis. So it's quite a complex thing, statistical power, it's not necessarily straightforward.

Q. Unbelievably complicated. Some of the formulae I've seen in some of these papers make very complicated reading, let's put it that way.

A. Yes, it is a complicated --

Q. That is your speciality, isn't it, the application of these very high-powered algorithms and mathematical calculations to statistics?

A. Yes, we can do that.

Q. Let's come back to this.

"For this reason effective thresholds have been set in which epidemiological studies have not had the power to detect any adverse risks to a population. This does not necessarily mean there is no detrimental effect to an individual within that population."

Again, that's obviously right, isn't it?

A. Mm-hm.

MR JUSTICE BLAKE: You accept the previous sentence:

"For this reason effective thresholds have been set at which epidemiological studies have not had the power to detect any adverse risk to a population"?

A. We can look at individual studies and see the point at which -- the lowest point at which we can see a statistically significant effect. And that, for that

study, then represents the lowest dose at which we can make a comment. Other than that we are extrapolating using higher dose data down to lower dose regions on the assumption of the model.

MR TER HAAR: It's that assumption --

A. If it's a linear model then we're assuming a linear extrapolation down to the very low doses.

Q. It's that assumption that Dr Mothersill describes as "the old paradigm", isn't it?

A. I wouldn't say it's necessarily old. I think it's fairly current still, I would say.

Q. We'll come to what she says about that in a moment.

Anyway, so you agreed with the sentence:

"This does not necessarily mean there is no detrimental effect to an individual within that population."

Then she goes on to say this:

"It is known that some subgroups of the population are exquisitely sensitive to radiation and it is likely that some of the background levels of disease in the population are in fact caused by the above listed exposures to radiation.

"Although radiation epidemiology looks at abnormalities such as cancer, which have genetic changes and can be screened for, there may also be epigenetic

abnormalities."

Can you explain to the Tribunal, what is an epigenetic abnormality?

A. No.

Q. You can't.

MR JUSTICE BLAKE: "Epi" means big, doesn't it, large?

DR RAYNER: Around.

MR JUSTICE BLAKE: Around.

A. I think it relates to the way in which genes are expressed. But that is as far as I'm prepared to go. That's not my field.

MR JUSTICE BLAKE: All right.

MR TER HAAR: She gives an example which gives us some indication:

"An example of this is peanut allergy or asthma where there is an abnormal response to a stimulus but no detectable genetic abnormality. All this leads to uncertainty as to the effects of low dose exposure."

So, stopping there, I think you would agree with, so far as it's within your expertise, everything in that paragraph?

A. Not entirely, no. I don't know quite how she comes to the conclusion that some people in the population are exquisitely sensitive. That doesn't ring true to me.

Q. Let me give you an example.

Young people, children, according to some studies, are more sensitive to radiation from CT scans.

A. Mm-hm.

Q. That would be an example of what she describes as a group exquisitely sensitive?

A. I would say all children are equally sensitive. I think you couldn't define a particular sub-population of children who are exquisitely sensitive. There is variation in the sensitivity of people across the population that we see as a result of the large-scale studies, but to say that particular groups of people are exquisitely sensitive, no, I would disagree with that.

Q. Would you at least go this far: certain human beings appear to be more sensitive to radiation than others?

A. That might well be possible. I haven't seen good evidence to that effect.

Q. Well, let's come back to that.

What she says going back to the end of this paragraph:

"All this leads to the uncertainty as to the effects of low dose exposure. This is compounded by efforts to relate endpoints measured at the molecular level to frank disease in the individual or efforts to link small clustered incidences of disease to a specific radiation exposure."

Now, that may be getting towards the outer limits of your expertise as a epidemiologist?

A. We are not measuring endpoint at a molecular level. Our endpoints are deaths and incidences of disease.

Q. You are looking at effects across the whole of the population?

A. Yes.

Q. Now let's go to why she describes it as a new paradigm. 3.5:

"Within conventional radiobiology, as accepted in the 1950s continuing through to the late 1990s, there was no room for epigenetic effects because the traditional concept of radiobiology was based on target theory. In order to work, radiation had to hit a defined target within the cell assumed to be DNA. An assumption about the numbers of targets hit could then be made from measurements of dose and dose rate."

Now this I imagine is outside your territory, isn't it?

A. Mm-hm, yes.

Q. She then goes on over the next couple of pages. If we go to page 8, she has a historical review of how methods of analysis from a radiobiological point of view have changed. And she does an extensive survey which goes all the way through to page 12.

Now, I imagine on this you really would have to bow to the expertise of the radiobiologists?
A. This work is outside my field of expertise. I can make no comment.
Q. When we come to page 12, she there pulls together the result of the references and makes a number of propositions, starting towards the bottom of page 12 where she says:
"Looking at matters in a more thematic way ..."
Do you have that?
A. Yes.
Q. That runs on to the end of page 14.
Now, of the points she makes it would seem to me -- tell me if I'm wrong -- that you are not in a position to agree or disagree perhaps with 1, 2 and 3. Do you want to just quickly cast your eye over those? They appear to be outside your territory.
A. Mm-hm, they are.
Q. It may be that paragraph 4 at page 14 is getting closer to your territory.
A. Mm-hm, yes.
Q. She refers to criticism of the epidemiological research undertaken after the Hiroshima and Nagasaki bombs.
A. Yes.
Q. Is that within your territory?

A. Yes.
Q. What we were told by Professor Sawada last week -- let me take two parts of it. I don't think you were present when he gave evidence, were you?
A. No, I wasn't.
Q. Well, I'll be corrected if I summarise it wrongly, but he points to two areas of research. One in particular deals with mortality rates, and what he says is this: that in the LSS studies there is research for the mortality rates which show an increased level of mortality within an area of a 1 kilometre radius from the hypocentre or epicentre of the explosion. This is at Hiroshima.
A. Mm-hm.
Q. That it had always been assumed that that mortality rate applied in that area, but that now it appears that the increased mortality rate extends at the same level on a radius 2 kilometres from the epicentre, which would suggest that -- and his figure is a mortality rate of 20 times what would be otherwise expected, as I understand it, from the control group. He says that suggests that the level of radiation in the 1 to 2-kilometre zone must have been a great deal higher than has so far been assumed.
First of all, are you aware of that point which has

been made about the LSS study?
A. Yes.
Q. It appears to have some validity, doesn't it?
A. No.
Q. Why not?
A. Because it's inappropriate to consider the geographical location as a measure of dose. The LSS studies that I use and the data we use, the dose is assigned on an individual basis according to the -- well, now the DSO2 dosimetry system and previously the DS86 and backwards. There you are looking at individual measurements of dose.
Now just because -- so it takes account of the person's shielding at the location they were exposed. So just because somebody was a particular distance away, their shielding would have an effect upon what dose they received. So it's not appropriate to compare exposure in a particular geographical location because that may apply to the area but it may not apply to the person.
The appropriate way to look at this is to use the individual doses that are derived for each person and apply that to the rates of disease.
Q. I think that's missing the point, with the greatest of respect. As I understand the point being made it is that the mortality rate, increased mortality rate in the

1 to 2-kilometre zone, which is identical to that within the 1 kilometre zone, is consistent with people in the 1 to 2-kilometre zone being exposed to much higher levels of exposure, of dose, than had previously been assumed. Therefore, the study as to mortality rates casts doubt as to what the dose was?
A. I would disagree with that. I think the geographical distribution of the dose is not as reliable an indicator of dose as individual measurements.
Q. But you don't have individual measurements. The Americans come along, drop a bomb and some years later you start studying. You don't have a reliable indication of dose, do you?
A. We don't have a reliable geographical indicator of dose. We didn't have dosimeters when bombs went off at particular locations on the ground. So we're -- you know, I don't believe his assertion that he can accurately judge these things.
Q. Well, I think what you said is very helpful because it actually illustrates many of the difficulties with the LSS study. Can we just highlight some of them. Others may have others to join to the list.
But first of all, of course, it wasn't a cohort sample in the sense of looking ahead. I remember once being told about the difference between "trohoc" and

"cohort" samples. I don't know if you ever use that expression, where cohorts ideally you look ahead with the sample of population; trohoc is where you look backwards. Sir Richard Doll is the man who first thought of that distinction. Sir Richard Doll is thought of as being the father of epidemiology in this country.

So the first problem you have is that obviously the Japanese on the ground didn't set up those circumstances for a proper controlled test or examination of what was about to happen to them?

A. Indeed.

Q. So things like what exposure there was on the ground have to be reconstructed on inevitably incomplete data, doing the best you can?

A. Absolutely.

Q. The second problem is that insofar as you are extrapolating information from the LSS study, the study didn't start for five years?

A. Mm-hm.

Q. By which time not only had many people died in the initial explosions from the acute effects of radiation but it's reasonable to suppose that many had died for what may be somewhere on the borderline between deterministic results and stochastic results; would you

agree with that?

A. It's possible.

Q. Thirdly, there is powerful evidence that certainly in the 1940s, it may not be so true now, the incidence of cancer generally and particular cancers was different in the Japanese population from, for example, in the western world?

A. That remains true today.

Q. Not -- it's changing, but as the Japanese eat more hamburgers.

A. Yes.

Q. But that is also a problem if you are extrapolating from the Japanese data to cancer rates and exposure in other parts of the world?

A. The issue is how you extrapolate the risk and how you apply the risk from one population to another population. There are two ways of doing it. We can either define -- we can either do it additively or multiplicatively. So, for example, in the Japanese population you might find that you see -- for a particular dose you might see an increase of double the baseline rate in a particular population.

So the question is, if the baseline rate in the population you want to estimate risk to, say a western population, then if the baseline is the same then we

would say that's okay; if the baseline was half, should you still apply that factor of 2? Or should you look at the absolute number of cancers per head of population in the Japanese for a given dose and apply that to the other population? Do you apply the risk absolutely or relatively across the populations if the underlying rates are different?

If they are the same, it doesn't matter, you end up with the same answer, but if they're different then you end up with different answers.

At the moment we don't have an ideal -- we don't know exactly which is the best way of doing that for all cancers. Over time, more information has become available and for some cancers we now have a better idea. For others we don't, and in those cases we simply take an average of the two. So we transfer the risk and then simply take an average to accommodate our lack of knowledge --

MR JUSTICE BLAKE: You've explained this in your report?

A. I do, yes.

MR JUSTICE BLAKE: About absolute and relative, 3 and 4?

A. Yes.

MR TER HAAR: What it comes to is this, a bit like on the NRPB studies we were looking at earlier: obviously statisticians are doing the best they can but with what

is undoubtedly a very difficult exercise of comparison?

A. But it's not the same for the NRPB studies because we're not trying to estimate risk in a different population. We're trying to estimate it in the same population so the issue doesn't arise.

Q. I understand. Certainly if you are trying to extrapolate from the Japanese experience you have problems with the historical data as to what is being reported about exposure initially?

A. (Nodded assent).

Q. You have problems with the control against affected persons because the study started late?

A. The study started late, which means that it's -- if we wanted to use the Japanese lifespan study to estimate risks very early on after exposure then that would not be appropriate. But to use the study to estimate risks many years later then that should be fine.

Q. Then the final point, and it is the one we have just been exploring, is you have to at least make some allowance for the fact you have a different population?

A. Yes, and that's been extensively thought about and ICRP have made their recommendation, which is to do it on an average basis. But for some cancers now in the latest iteration of the ICRP recommendations we have some better information about that average and it's been

weighted in some cases for certain cancers to take account of more knowledge that we have.

Q. Could we then go back to this report, please, back to page 14. The next paragraph, 3.7 --

MR JUSTICE BLAKE: Just before you move on, Mr ter Haar, I've been recording your question and a little while ago, some little ago when we were dealing with the reference to Professor Sawada you said that there were two areas of concern.

MR TER HAAR: You are quite right, my Lord. I haven't dealt with the second one.

MR JUSTICE BLAKE: The mortality rates. So it would be helpful --

MR TER HAAR: I didn't deal with the second point, and I apologise. That's very helpful.

The first point which Professor Sawada made to the Tribunal related to mortality rates in the 1 to 2-kilometre zone.

I think from what you said you were aware of that point but you don't agree with it?

A. I don't agree with it because I think estimating dose on the basis of geographical location isn't a sound way of doing it.

MR JUSTICE BLAKE: You told us that.

MR TER HAAR: The second point I think you will give the

same answer to, which is this.

He pointed to the evidence in the LSS in relation to depilation or epilation, i.e. losing your hair, and he pointed to the projections that certain levels of exposure are necessary in order to cause hair loss. He pointed to the fact that the statistics as to those at particular locations who had lost their hair and those at certain distances of what the projected exposure to dose was and said if you put the two together you actually had inconsistency in the LSS between on the one hand the depilation rates and on the other hand projected exposure levels.

First of all, are you familiar with that comment upon the LSS study?

A. Not particularly, no. I'm not, I'm afraid.

Q. Certainly it sounds logical, doesn't it? If people have been assuming on the one hand you need a certain level of dose in order to lose your hair, and on the other that there's a fall-off in exposure geographically and you'll find that people at the outer end of that geographical limit are also losing their hair, the two things don't seem to go together, that's point he's making.

A. On the face of it.

Q. That was the second point and certainly you don't know

about that criticism but you can see that, if true, it does cause some questions?

A. On the face of it, yes, but I think often you have to look deeper into these issues to really understand them.

MR JUSTICE BLAKE: Before we move on and leave Professor Sawada behind, which I know we are going to be doing shortly, you've read the article, have you, that's cited at page 14.4 which is the starting point of these questions, I think?

A. I have seen it. I haven't studied it in detail.

MR JUSTICE BLAKE: Have you read the report that he prepared for us which includes I think at tab 6 the epilation graph?

A. No, I haven't read it in detail.

MR JUSTICE BLAKE: And have you read the evidence that we've managed to get out with some difficulty in translation --

A. Well, I read what I could of it but it didn't all make a lot of sense to me, I'm afraid.

MR JUSTICE BLAKE: Right. Do you want to just have a look at some stage at the graph in his evidence to us which is I think what the questions that have just been put us -- I mean, if you can't make any more sense of it that's the end of it, but if you can it might conceivably be of interest to have your comment.

A. Okay.

MR TER HAAR: My Lord, I note the time.

MR JUSTICE BLAKE: Yes.

MR TER HAAR: I note the question which your Lordship has just asked.

I don't know whether you would be able to do it in the the break we take for the shorthand writers but just in case you can, in bundle SB2, it's a different bundle from the one you have there, you'll find Professor Sawada's report --

MR JUSTICE BLAKE: 2.6.

MR TER HAAR: -- at 2.6, and the graphs that we've just been talking about are to be found at pages 11 and 12.

A. Sorry, what was the tab?

Q. I apologise. 2.6, towards the beginning.

MR JUSTICE BLAKE: Yes, it was figure 6, wasn't it? That was the epilation?

MR TER HAAR: One needs to take 4, 5 and 6 together and the text in between.

MR JUSTICE BLAKE: Yes. Do you see page 11?

MR TER HAAR: I don't think the witness is there yet. Do you have divider 2.6, first of all?

A. Mine starts at 2.14.

MR HEPPINSTALL: 2.6 in your SB1.

A. Oh, in my SB1.

MR TER HAAR: Does that have 2.6 in it?
MR JUSTICE BLAKE: Right at the very back.
A. Yes.
MR JUSTICE BLAKE: Let's see if we can get there.
MR TER HAAR: Then if you go to another page, 11.
A. Yes.
Q. You'll find two graphs at page 11. Those are figures 4 and 5 and then another one at page 6. You may need to make sense of it to read the text which starts at the bottom paragraph of page 10 and then goes through, I think, to just above figure 6.
Now, it may be possible, in the quarter of an hour we have, for you to master it, if you can. I think that's what the Tribunal is asking for your assistance on.
A. Okay.
MR JUSTICE BLAKE: Thank you very much. Quarter to 12, then, but if you had longer do you think you could do better?
A. I'll see what I can do, my Lord.
MR JUSTICE BLAKE: If you are in the middle of some interesting calculation if you let us know we'll give you more time.
A. Unfortunately I don't believe there are any calculations I can do on this.

MR JUSTICE BLAKE: I mean, if you can't you'll let us know as well. There's not an expectation but it would be of interest if you can give us any assistance. I think that's as high as I can put it.
A. Thank you.
MR JUSTICE BLAKE: Quarter to 12 or such other time as you think you might usefully need.
(11.32 am)
(A short break)
(11.47 am)
MR TER HAAR: Dr Haylock, any progress?
A. I'm sorry but this is just not do-able in the time I have available, my Lord. It's just too complicated and too --
MR JUSTICE BLAKE: Right, okay.
A. It doesn't make sense enough, I'm afraid.
MR JUSTICE BLAKE: It doesn't make enough sense for you to comment.
A. No. If I had enough time to analyse it in great detail, possibly, but I'm afraid --
MR JUSTICE BLAKE: Righty ho.
MR TER HAAR: Unless the Tribunal has any more questions on that, could you put that bundle aside, please, and I am going to come back to Dr Mothersill's report in a moment but I want to take you first of all bundle SB21, which

I hope you have there, tab 30.
A. Mm-hm.
Q. If you could just open up the first page, you probably immediately recognise what this is, but it's annex A to the 2006 UNSCEAR report.
A. Uh-huh.
Q. I imagine this is a document with which you are very well familiar.
A. I've seen it once or twice before, yes.
Q. Were you one of the authors?
A. No, I wasn't.
Q. But anyway, could you go, please, to the introduction which is after the table of contents, page 17 at the bottom of it.
A. Yes.
Q. I just wanted to, again in the context of the Japanese experiences, look at what UNSCEAR had to say.
Paragraph 3, the top of the right-hand column:
"Although resolving inconsistencies in the dosimetry for the survivors of the atomic bombings has reduced one source of uncertainty in estimating cancer risks to a population from doses of radiation, a considerable numbers of other sources of uncertainty remain. A major one relates to extrapolating risks from the moderate dose but high dose rate exposures received by survivors

of the atomic bombings to low doses and dose rates."
That's a comment with which you'd agree, wouldn't you?
A. Yes.
Q. "This is also true for interpreting data on many therapeutically exposed groups. The topic has long been controversial and was discussed in annex G, 'Biological effects at low radiation doses', of the UNSCEAR 2000 report. There's also uncertainty related to extrapolating cancer risks to the end of lifetime. In particular, about half of LSS cohort is at present still alive. IN estimating lifetime risk factors from the data on this cohort, it Is vital to determine the pattern between radiation dose and expression of cancer risk for those who were exposed in childhood and who are now reaching the age at which larger numbers of cancers would be expected to arise spontaneously. Another source of uncertainty relates to the transfer of radiation-induced cancer risk estimates between populations with different underlying rates of cancer. For example, the rates of lung and breast cancer for the Japanese population tend to be lower than for many North American and Western European populations, whereas rates of stomach cancer tend to be much higher. The available evidence, most recently reviewed in the UNSCEAR 1994

report, did not suggest that there is an easy resolution of this problem."

You would agree with that? It's a difficult area of statistics?

A. Yes, as I said, in the latest ICRP/103 some progress in the time since this has been written, some progress has been made and for some cancers now ICRP recommends that the transfer of risk is not just done on a straight average between relative and absolute but it advises on certain weighted averages according to various cancer types.

Q. Can we go on to looking at the low dose problem as I can call it. Go, please, to page 24. You see the numbers are in the top left-hand corner, paragraph 15.

"Where the dose levels are low two other phenomena affect the study results. The first occurs because epidemiological studies are based on natural human populations with their extraneous variability in genetic make-up, diet, lifestyle and other exposures, rather than having tightly controlled experimental conditions. This means that there may be subtle differences between exposed and unexposed groups in some unmeasured factors that affect cancer risk. For a high dose study with a large expected radiation effect such variations are fairly inconsequential, but for a low dose study with

a small expected radiation effect the magnitude of such extraneous variations may equal or surpass the size of the expected radiation effect. Hence, for a low dose study there is great potential for a false negative or false positive result, and little way of even knowing whether such an effect has occurred. This reduces the credibility of the results. Assessment of the pattern of results in low dose studies may sometimes provide indications of artefactual findings. For example on the basis of an analysis of results for non-malignant respiratory disease relating to smoking which exhibited negative trends with a radiation dose. Muirhead and others suggest that smoking may confound the radiation dose response relationship in some smoking-related cancers."

16:

"Secondly, for a low dose study with small numbers of cases or deaths expected, and therefore within adequate statistical power, if any result for RR is found to be statistically significant its magnitude is in all likelihood a substantial over-estimate of the true risk. For instance ...(reading to the words)... with 19,100 spontaneous breast cancers during the same period. If the study were on a cohort of a million such women the statistical power would still be only at a

little above 5 per cent. Adequate statistical power is usually taken as at least 80 per cent. If such a study were to be repeated numerous times for the occasions when there was a nominal statistically significant excess the RR estimates would be about 9 times greater on average than the true relative risk. However, in a single given study the authors will usually derive the best estimate of the true risk from their own estimate, which is likely to be a substantial overestimate."

So arguments can go either way but low dose epidemiology is really difficult stuff is the message.

A. It is. Yes. Yes.

Q. And then specifically in relation to the Japanese studies, if we go to the next page, page 25, we see there, don't we, at paragraph 21 and following some discussions of the problems with extrapolation from the LSS studies?

A. Mm-hm.

Q. If we go on to page 29 we have there a section headed "Transfer of radiation risk estimates between populations and interactions of carcinogens", and after a lengthy analysis of the problems with the LSS study and extrapolating it to other nations, at paragraph 46 on page 31, the authors say this:

"Much of environmental, nutritional and occupational

cancer epidemiology is concerned with identifying risk factors that might account for some part of the variation of site-specific underlying cancer rates among populations. While there has been much progress the problem is vast."

Is the author's conclusion:

"There is only limited information on the interaction between radiation dose and lifestyle or constitutional factors in terms of cancer risk."

They might have made some improvements in analysis since but it's still a vast problem, isn't it?

A. There are problems. One of the things we do to try and determine if the things we see in one study are real is by comparing across different epidemiological studies. If we see a consistent pattern of a disease being at a raised risk across different large populations then that gives us some confidence that that is a true effect and not something that's occurred by random chance.

In terms of risks to the UK population, the study of radiation workers in the UK that was started by the NRPB many years ago, that does provide reassurance that the overall risk we see in the LSS population do agree with the risks we see in the -- and the worker population, who have considerably lower doses.

So organisations like UNSCEAR and ICRP don't base

their pronouncements on single studies. It's a consensus across studies. If we see one study showing something then that's interesting but doesn't necessarily prove conclusive. We would like supporting evidence from other studies. There are now larger worker studies around now, which are beginning to provide good quality evidence to either support or refute what we see in the bomb survivors. So yes, it is still difficult but it's not completely impossible.

Q. Well, not completely impossible -- it isn't actually completely possible because there are difficulties but what you can do is make a best estimate?

A. Yes. All the studies we have, we're all the time making the best estimate of the risk we see on the basis of the data that we have and as that data changes and improves so our estimates evolve.

Q. I think it may be that where in a sense your answers to my questions reveal a difference between us is this. What you are I think constantly striving for -- in an ideal world you'll strive for certainty but we know you can't get certainty in these epidemiological fields, you'd agree with that?

A. (Nodded assent).

Q. I think you are nodding.

A. Yes.

Q. So the next best you can do is to look at degrees of probability?

A. Yes.

Q. What you are doing is taking as many samples as possible to see what level of probability you can arrive at as a matter of scientific consensus?

A. But it's not just as simple as a bigger sample means more certainty. It's about the size of the effect you are looking for. If we're looking for a very tiny radiation effect on top of a potentially large underlying baseline effect that is much more difficult to do, compared to a larger radiation effect.

Q. I quite agree.

I was really at a much wider level of generality than that. The process you are doing is throughout the whole range of diseases you are trying to increase the levels of confidence in the data you have, but with some areas, i.e. because the diseases are very rare or because your statistical sample is very small, you end up with real difficulties?

A. You will find it increasingly difficult to detect risk in those circumstances, yes. If you don't detect a risk then the issue is: is that because there is no risk or is that because your sample or your study wasn't sufficiently statistically powerful enough to see a risk

that is actually there?

Q. So you may have a problem that the disease itself is rare, which causes a statistical problem, do you agree?

A. It can do, yes.

Q. You may have a problem also sometimes when the disease is not rare, or indeed in some ways that's the most difficult area because if for example you know that six out of 10 people are going to die of a heart attack, trying to identify whether a seventh might die of a heart attack is much more difficult because -- I think statistically you are probably using larger numbers -- if you have a very common disease which can be caused by a number of factors, to identify what the additional risk is from, for example, radiation is a difficult statistical exercise?

A. It depends upon the size of that additional risk. If it's a large risk then no, it's not difficult. If it's a small risk, then yes, it is.

Q. Yes. So you can have problems from a shortage -- I'll start again.

You can have difficulties from a rarity of disease, statistically, and you can have difficulties from the fact that a disease is very common. Both can potentially cause problems?

A. Yes. One thing we can do is if a study doesn't show us

a statistically significant effect we can look and see what is the smallest excess risk that we can exclude on the basis of the data we have. So we might be able to say: okay, we can't -- we don't detect a risk at a -- we might be -- we see no difference between two groups. We might say: well, our data supports that the true difference should therefore be less than some higher value.

Q. In particular in relation to what I've been asking you about, which is low dose effect, the problem with low doses is it's much more difficult to see a statistical conclusion which shows what the effect of -- as you go down towards the bottom of the dose range, what the actual statistical effect is of a low dose?

A. Yes, as the size of the effect you are looking for reduces in comparison to the underlying baseline rate it becomes more difficult, yes, which is why at the moment we use extrapolation from higher doses.

Q. I understand that. But can we go on back to UNSCEAR and go to their conclusions at page 137, quite a long way on.

On this occasion the numbering is actually at the bottom of the page. Have you found it?

A. Mm-hm.

Q. Can we go to paragraph 589, please.

A. Yes.
Q. In summing up, UNSCEAR say this:
"The increased statistical precision associated with the longer follow-up and the resulting larger number of cancer cases observed in the above studies have also been useful in the examination of dose response relationships, particularly at lower doses. For example, the most recent data for the survivors of the atomic bombings are largely consistent with linear or linear quadratic dose trends over a wide range of doses. However, analyses restricted solely to low doses are complicated by the limitations of statistical precision, the potential for misleading findings owing to any small undetected biases and the effects of performing multiple tests of statistical significance in attempting to establish a minimum dose at which elevated risks can be detected."
All of that, I think, reflects the statistical problems we've just been talking about?
A. Yes, that's correct.
Q. "Longer follow-up of large groups, such as survivors of atomic bombs, should hopefully provide more information at low doses."
Now is the part I really want to take you to.
"However, epidemiology alone will not be able to

resolve the issue of whether there are dose thresholds for risk. In particular, the inability to detect increased risk at very low doses using epidemiological methods does not mean that the underlying cancer risks are not elevated. However, the high dose radiotherapy studies of patients indicate that for some cancers, for example bone, connective tissue, rectum, uterus and small intestine, any risks of doses at below several grays, if they exist, are small."
What that leads to is this, isn't it: that where you are dealing with low dose analysis, epidemiology is a limited tool for identifying whether risks exist or the level of risks? You have to turn to other tools in order to see what statistics you get. Is that a fair general proposition?
A. There is a limit below which epidemiology will not be able to detect risks at low doses without making assumptions about the overall dose response relationship at higher doses. Essentially, as your doses get lower and lower, essentially your extra bit is lost in noise of the rest of it. So it does become more difficult, which is why we look at the whole pattern across the whole dose response relationship from higher doses downwards.
We do make assumptions about how dose can be

extrapolated and that gives us the ability to estimate risks at lower doses. But if we're just talking about estimates based upon low dose information on its own, no, it's unlikely to be able to do that in a short to medium term.
Q. Yes. It's at that stage in a sense that the enquirer after truth turns to other sciences in order to see whether the questions raised by or left open by the epidemiological research can assist?
A. Yes, in a sense that is happening now. We're trying to look at the mechanisms, the actual biological mechanisms by which radiation causes cancer. So as Professor Thomas mentioned about the multi-stage nature of cancer -- initiation, promotion or progression -- we are looking at: can we develop biologically inspired models that will take the place of things likely LNT? At the moment you can fit those sorts of models but they don't provide any extra information.
Q. So would this be a fair summary. There's a recognition that particularly in this low dose area, or low dose region, there's a necessity to carry out further research, that is ongoing -- and when I say further research, cell sciences and biological studies -- those are ongoing but the questions have not yet been resolved?

A. We can always do better but I think we have quite a good handle on what the risks are, certainly down to a few tens of millisieverts now. It's on a population level that the estimates are quite solid at that point.
Q. Could you then put away the bundles you have and go back to bundle 11. I don't know if you put it away or you have it still out.
We were in divider 7. This is Dr Mothersill again. Can we go, please, to page 16, paragraph 4.3:
"The new meaning of the linear non-threshold (LNT) model.
"Given all the new uncertainties, the LNT model cannot be called an LNT hypothesis any more. It is clearly not correct to say a linear extrapolation describes low dose radiation effects."
I think you probably wouldn't put it as firmly as that, but you do say that as an epidemiologist you are making assumptions as to whether there's a linear effects in low doses; do you agree?
A. I would disagree with her statement that it's not correct to say linear dose describes the low dose radiation effects. I think it does describe them as best we can at the moment.
Q. I think "as best as we can" is exactly where she's if you like -- she says that it's --

A. It's not just -- we're not talking about, for example, just a single study saying this. A number of large epidemiological studies also have the same result.
Q. But none of them get you to showing that there is a linear effect at low doses. All you can say is that there is a linear relationship down to a certain level of dose?
A. Yes, that's true.
Q. And the new paradigm, which is basically how she describes the radiobiological work that she had explained -- do you remember we looked at that earlier, leading to the conclusions which she drew at paragraphs 1, 2, 3 and four at the bottom of page 12 and 13 and the top of 14? So that's the new paradigm she's referring to.
"The new paradigm contains complexity and unpredictability. There are arguments and data to support any relationship between dose effect at low doses, but the reality is that any outcome can happen to an individual and there are ample data showing effects at low doses."
Now, just taking that last part of that sentence, it's right, isn't it, that there is a considerable body of data which can be interpreted as showing effects at low doses?

"The possibility that there are no health effects at low doses is very remote."
Well, I think you're assuming that there may be effects at low doses. Do you agree?
A. Yes.
Q. "The purpose of the LNT model now is to provide a tool for regulation in an environment of uncertainty, and on a scientific analysis the LNT dose effect relationship has been rejected by various radiological bodies asked to consider the evidence."
Which is the CERRIE minority and the majority reports of 2003, 2004 and the French Academy of Sciences.
Now we have heard about CERRIE in particular. Would you go this far: that there is at least a body of opinion which the CERRIE minority reflects?
A. I wouldn't say it's scientific opinion in terms of based upon evidence.
Q. Now, let's go on because I am going to suggest --
MR JUSTICE BLAKE: What about the French Academy of Science?
A. I'm not aware of that, my Lord.
MR TER HAAR: Well, we can look to the references and find the reference in due course.
Go on to page 17, the following page:
"The cause of the uncertainty is simply that the

A. The body of epidemiological data supports that there are effects in line with the LNT.
Q. Well, I think we've already been through that. At low doses you have in fact no epidemiological evidence other than at higher doses there is a linear relationship?
A. And at no dose there is no risk. A fixed point at the bottom as well.
Q. You are making a mathematician's assumption that because the line keeps on coming on down and gets to a certain point there's a gap where you get to 0?
A. The LNT model that we fit is the one that fits the data best at the moment, if you fit it all in one go.
Q. Because you have to make an assumption between 0 and the level at which epidemiology kicks in, if I can put it that way?
A. Yes, you could in fact fit a number of different relationships in that region -- many have been suggested -- but if you look at the statistical epidemiological data, none of those other models fit any better than LNT.
Q. One of the things to always bear in mind from our point of view is that this Tribunal is dealing with doubt, reasonable doubt, not balance of probability which is an important concept for the Tribunal.
Can we go back to page 16. She says this:

simple DNA damage paradigm does not hold at low doses."
Now, I stop there, I am going to come on to the rest of that sentence in a moment. That is a radiation biologist's assessment of whether the DNA damage paradigm, as she calls it, can stand with modern biological research.
Just to remind you of what she's talking about, if you go -- keep a finger in page 17 and go back to page 6 -- there she describes what she calls the "old paradigm", but it's a biological theory as to how radiation damage is caused in DNA.
What she has done, if we just remind ourselves, is she has gone from that which she calls the old paradigm through the biological research that's taken place, which she sets out at some length, as we saw, over pages 8 to 12. So that is based on what she says, that the simple damage paradigm does not hold at low doses.
Now, as an epidemiologist you can't agree or disagree with that, I assume, that's a biologist's conclusion?
A. It is.
Q. She then says:
"Therefore, dose and effect cannot be linearly related."
So what I think she is suggesting is, if you take

this zone where epidemiology cannot provide an answer, she says in that area, the biologists enter, and so it can't be the old DNA assumption, the old paradigm. Again, that's outside your territory?

A. Yes.

Q. Then she goes on to say:

"Which way the curve will go depends on other factors including genetic background and environmental conditions."

Then I suspect you will be able to agree with paragraph 4.5 but let's see:

"Relating dose effect to harm to risk.

"This is the key issue. It's always controversial, and in dose ranges where epidemiology is a weak tool it is usually difficult to assess whether a dose produced a specific adverse consequence in any individual. The reverse relationship, that an adverse health effect is caused by a dose, is also difficult to assess."

Just stopping there, you'd agree with that, wouldn't you, so far?

A. Epidemiology is not a tool really to estimate risks to individuals. We are mainly concerned with populations, to avoid the variability of individuals within a population. So I can agree that when she's talking about an individual, yes, it is difficult because

an individual is not always representative of the population.

Q. I am grateful. You may have saved me several minutes of cross-examination on something else there, but I totally are with what you've just said.

Can we go on, though, and I think this is where again we step outside your territory.

"However, some of the above tests are more suggestive of a link between dose and effect than others. The gold standard is of course chromosome aberrations as these are evidence of a fixed genetic change in dividing cells which is relevant to both cancer and hereditary effects but induction of cancer-associated proteomes, stress proteomes or genomic changes in cancer-associated genes are also important, even those these are not necessarily fixed and transmissible."

I think what we are getting to is this. In this low dose region you have to sort of give way to the radiobiologists and the people who understand about cell aberrations and that sort of science?

A. I think the issue is if we want to be -- if we want to refine the LNT further in that region it will take biology to do that. But at the moment, the evidence we have so far suggests that LNT is the best relationship

that we have to describe a risk in that region at the moment.

Q. We've been over that. You say that as an epidemiologist rather than as a cell scientist?

A. Well, the evidence shows that at the moment, yes.

Where she talks about the gold standard here, I think she's assuming that just because you have chromosome aberrations that's going to go on to cause cancer, that there is some direct relationship. I don't believe that's been shown to be the case. Just because you have chromosome aberrations does not necessarily mean you're going to go on and contract or die of a radiation-induced cancer.

Q. Let's put it the other way round. It doesn't necessarily mean -- I don't need to debate that with you, others might do -- but that there is a school of thought among suitably experienced people that there is a connection between chromosomal aberrations and cancer is the case, isn't it?

A. Yes, undoubtedly that there is some sort of relationship, but it's not going to be a straightforward one, I don't think.

Q. Your answer there illuminates really the whole of the debate we're having, which is this: that over the last 20 years, something like that, the rapid advances in the

study of cells, the study of chromosomes, has enabled a great deal more light to be cast on to causes and possible causes of cancers and other diseases. We can take that as a general statement. You don't disagree with that?

A. No, I don't disagree with that.

Q. All that the epidemiologists can say is whether or not particular effects appear to be statistically significant?

A. Mm-hm, correct.

Q. And what you are doing is looking at the rates of certain effects in the population as a whole?

A. Yes.

Q. So let's assume that we have a population of a million, 1,000 people are going to get a particular form of cancer each year. You may well be able to find from epidemiological studies that one of those may well be affected by -- may be in whole or in part the result of exposure to radiation. I mean that's the way that epidemiology approaches the problem.

A. Well, for a particular individual we couldn't say that, no.

Q. Absolutely.

So when you are looking at your figures in your report and you say that there's a 99.9 per cent chance

of such a disease being caused by other causes, the other side of it is that you can't say that a particular condition was not caused by radiation, because there's always that 0.1 per cent.

A. The evidence says what it says, that there is a 99 whatever per cent chance that it's caused, therefore whatever remains is the possibility it was caused by other things.

Q. Putting it in the vernacular: it's perfectly possible it was caused, I just don't know?

A. Something else.

Q. But the important point from my point of view is it would be a perfectly accurate way of describing the result of that: it's perfectly possible, I can't tell you?

A. Yes.

Q. Can we just look together at a paper which has gone into the bundle today. Could you have bundle SB22. You may find it easier to get rid of some of the paper that is piling up.

MR JUSTICE BLAKE: Have we finished with tab 11 for the time being?

MR TER HAAR: We have indeed, I think probably for the rest of my cross-examination.

So bundle 22, please, tab 19.

Have you managed to -- is it in that bundle?

A. No.

Q. It hasn't been updated, I apologise.

MR JUSTICE BLAKE: We've been updating ours, but yours has not been. (Pause). (Handed).

Can you helpfully slot it into tab 19?

Thank you.

MR TER HAAR: Now I think from what Mr Heppinstall said, you were provided with a copy of this paper overnight.

A. I was.

Q. And I am going to concentrate on this one, but I think you were given, I think it's four papers by Dr Greenland, either solely authored by him or written by him with others.

A. Okay.

Q. First of all, is Dr Greenland somebody with whom you are familiar?

A. No.

Q. He appears to be working in your sort of area of epidemiology with a special interest in --

A. There are quite a lot of people who work in epidemiology, though.

Q. But you had an opportunity to read this last night?

A. I had a read of it, yes.

Q. And it may be that the shortest way of looking at this

is to deal with this: that if what you are faced with is trying to work out whether a particular individual suffered a particular disease as a result of exposure to radiation, which is what we are concerned with, if what you are trying to do is to establish something on the balance of probability, that causation issue on the balance of probability, then epidemiology may be able to assist because you can look at what are the chances of that person having got the same disease in any event?

A. Epidemiology can provide some measures that you could use to do that.

Q. If on the other hand you are looking at simply whether there's a possibility that a particular disease was caused by radiation, unless you come to the conclusion there's a zero connection then your epidemiological study always produces a result: for this person it might have been caused by that -- that disease might have been caused by that risk, maybe radiation. That's the nature of epidemiology?

A. If we have a risk model to relate that disease to a dose, then yes we, would come up with a probability of causation and unless that was zero then there would be some chance.

Q. By definition it's always possible?

A. It has to be, unfortunately.

Q. Yes. What Dr Greenland is drawing attention to in his papers is that there's a tendency to use epidemiology, I think he would say somewhat misleadingly for a number of reasons. First of all, an epidemiological study of mortality may not tell you whether somebody would have lived longer but for the disease. So that a pure mortality study, you may say this person died of cancer aged 60, but you can't tell from epidemiology whether he would otherwise have perhaps have lived on to 70.

A. You can do a lifetime risk calculation which you can estimate the expected loss of life, if somebody dies of a particular -- of a radiation-induced disease.

Q. And in particular if we're dealing with the particular problem this Tribunal is facing, which is whether somebody's condition was caused or exacerbated by radiation, in order to get a pension, it's a particular problem, isn't it, because the data on which you based your evidence is dealing with either morbidity figures, i.e. what percentage of the population will get a particular disease, or mortality, what percentage of the population die of a particular disease? But you're not dealing if you like with the middle territory of how many people might get the disease earlier than they otherwise would because of the combined effects of radiation with the rest of their life?

A. The probability of causation calculation assumes the person has died of the disease and calculates the probability that a particular exposure was the cause.

What you're asking can be calculated. You can do essentially a -- similar to a mortality calculation where you can estimate loss of life expectancy, also in terms of incidence data, you can do loss of cancer-free -- cancer-free life. And the difference between the two would give you an estimate of I think what you're asking.

Q. Yes.

A. The number of years you would be alive but having suffered a cancer.

Q. That's not what you've been asked to do?

A. No.

Q. To look at, in the case of individuals, whether they might have got cancer anyway but they might have got it later or might, in the case of people who have died, might have lived longer than they did; that's not what you've addressed?

A. No. We know the person has died of whatever disease and we're looking at, given that fact, what is the probability that it was caused by a certain factor?

Q. Thank you.

I think the final area I want to ask you about is

this. It's not clear from your report what materials you were given before advising.

In these formal proceedings my clients filed a document which referred to probabilities and possibilities, which was what we call in the law courts effectively a pleading, a statement of case. That drew attention to the evidence in relation to each individual of papers which suggest that their particular cancer, bladder cancer or other conditions, was capable of being causally connected to radiation.

My impression I is you weren't given the opportunity to look through that and to consider what my clients were saying on the basis of identified scientific papers.

A. I don't believe I was given that document.

Q. No. So I think what you've done is simply to say, "Taking this particular condition", so let's take bladder cancer which Mr Abdale suffered from, "the best statistical fit I can find is that there's a 99 per cent, 99.9 per cent chance at the radiation dose I've been told to assume"?

A. I used the doses provided by Mr Hallard.

Q. So that was your starting point?

A. Yes.

Q. And you did not go on to consider whether or not the

scientific papers referred to by my clients would at least open up an area of debate which might cause you to think again about the figures that you had taken?

A. I think the process I went through, having determined what the disease was we were talking about, was to review what I consider to be the quality peer reviewed evidence to find an appropriate risk model for that disease to relate it to radiation dose and then I used that model to calculate the probability of causation in the case.

Q. Now, it's no part of what I want to do to suggest that you were careless or anything of that sort. You were doing it on the basis of what you were instructed to do.

But if I understood it right, you were not asked to carry out this exercise, which was in the case of each person that you were considering, to set out for the benefit of the Tribunal if there was an alternative body of scientific evidence with which you might disagree and which could lead to an opposite conclusion? That was not part of what you were asked to do?

A. No.

Q. It's fair to say, isn't it, that in this area of cancer research -- not only cancer research but we concentrate on that -- there are a very wide spread of opinions, held by people with great expertise?

A. There are a wide variety of opinions. Some of those are supported by evidence, some are not.

Q. But what is also true, though, is that there are opinions in particular of evolving theory which are not yet of sufficient certainty for you to be able to include the results in your analysis. Do you understand the point I'm making?

A. Mm-hm.

Q. People are coming up with credible hypotheses which need to be proved?

A. They need to be shown that the evidence supports them or not.

Q. But it is in the nature of this sort of work, isn't it, that very often a hypothesis will emerge and it takes some time to prove or disprove the hypothesis?

A. That might be the case.

Q. And in the nature as you described the task you carried out you have not brought forward for the benefit of the Tribunal hypotheses which are as yet unproved, you've dealt with the world of what you regard as being proven hypotheses?

A. I have used the models which are the models that are currently supported best by the epidemiological evidence.

Q. I understand that. I think that is actually an accepted

proposition I put to you, because you are making a selection from the wide range of material available to you?
A. Mm-hm.
Q. That inevitably means that there is some part of the material which you are putting on one side because you don't regard it as being the best evidence?
A. Mm-hm.
Q. The very last thing I want to ask you about is this.
Again, into the bundle, 22, not yours, I think, but for others, have been placed some calculations which were done by a member of the solicitors who are instructing me.
Those are now to be found at tab 17. You might have it separately.
A. Sorry?
Q. You had the calculations separately.
A. I have the calculations I was given last night.
Q. Yes.
MR JUSTICE BLAKE: Right. At the end of this you could probably put that in tab 17. It doesn't really matter because no one else is going to be looking at tab 17 after you.
A. I've scribbled all over it, I'm afraid.
MR JUSTICE BLAKE: Then you keep it.

MR TER HAAR: Now I am not going to suggest that this was done by a scientist but it's done by a very clever young man. What he has attempted to do is simply to take the figures that you have put in your appendix and make different assumptions first of all as to the dose rate.
First of all, as a matter of mathematics, do you have any reason to disagree with how he has done the mathematics?
A. I looked at this last night and I tried to reproduce the figures that he or she has done. I agreed with the excess relative risk calculations but I couldn't agree with the absolute risk calculations. I seem to be an order of magnitude different in my values compared to those there.
However, I do have to concede that because those are so small, the absolute risk values are so small, that doesn't actually materially make a huge deal of difference to the overall results.
Q. So --
A. But I can't -- I tried to see if I could work out why they were different, and I couldn't.
Q. There may be an explanation.
At any rate, if we're looking at the figures which are highlighted in yellow, whilst there would be some modest difference because of that factor, they are of

the right order; is that right?
A. They are of the right order, yes.
Q. This is all a matter then for submission as to what the right dose rate to take is.
A. Yes.
Q. The other factor is whether or not you take the DDREF of 2 or 1.
A. Yes.
Q. And in the explanatory note we can see the author says this in the first page under the heading "The appropriate DDREF":
"In his report Dr Haylock applies a dose rate effectiveness factor [that's the DDREF] of 2, in effect halving the radiation disease risk to take account of the extrapolation to a very low dose. Dr Haylock does not define in his report what he means by very low dose, but suggests that it is a dose below a few tens of millisievert. In his reply to the BS panel questions dated 7 March 2016 Dr Haylock characterised a low dose as 1 below 100 millisieverts or 100 milligray."
Can you first of all confirm, what do you mean by a very low dose?
A. This is a slightly vague term, I have to admit. There is a grey area in the middle where one might sometimes apply it or not, depending upon what your particular

personal preference is on these things. But I would say that in terms of the doses that we're talking about here, i.e. I was talking about 4 millisieverts, that I consider to be a very low dose indeed and therefore I would apply a (inaudible) of 2 to it.
So I think that the border is between, say, 50 and 100 millisieverts, but it's not entirely clear, I have to admit. People have different interpretations on it.
Certainly in terms of the doses I was given for the appellants, I consider that they fell into the very low dose category.
Q. You follow that through, if we go back to the calculations --
MR JUSTICE BLAKE: So anything below 15 millisieverts for present purposes is in your view very low dose?
A. Indeed, yes.
MR JUSTICE BLAKE: Right.
I may have another question, but let's complete the cross-examination.
MR TER HAAR: So if we go to the calculations, what I think this means is if we take the first page of a 4 millisievert rate, there you would say 2 is the appropriate DDREF?
A. Yes.
Q. Whereas if we go to the next page, where you are up to

170, we would be looking at the bottom figure DDREF of 1 because you are above your threshold?

A. I would say so, yes.

Q. Similarly obviously if you go to the last calculation of 431, there in fact only one figure is taken.

So on your analysis, 4 millisieverts the probability causation in fact worked out at slightly less than a DDREF of 2 but it's of the order of 0.075 per cent.

If you are wrong, or if views differ about DDREF, it's 0.15.

Otherwise, at the higher levels we are taking it's 6.6 per cent, roughly, at 170 millisieverts and 13, almost 14 per cent at 431, there or thereabouts?

A. Yes.

MR TER HAAR: Would you just forgive me for a moment, my Lord.

MR JUSTICE BLAKE: Of course. (Pause).

MR TER HAAR: I am grateful. Dr Haylock, thank you very much for your time. I have no further questions for you.

MR JUSTICE BLAKE: Just before we continue with questions from Dr Busby, can I just go back to the very low dose issue.

A. Uh-huh.

MR JUSTICE BLAKE: And try to take that back to the earlier

questions about one of the challenges to the science of epidemiology generally.

I have the proposition that one of the ways you proceed is to abstract and use the data from high dose radiation in the assessment of low dose exposures. You told us about that.

I also have at the other end of the scale when it's zero, it's zero.

A. (Nodded assent).

MR JUSTICE BLAKE: I think the debate is between the gap between that and where you can reliably use high dose extrapolation or where the model seems to work consistently.

So what is the size of the gap? Is it anything below 100 millisieverts is into the debatable territory? Or is it higher? Is it 200 millisieverts or is it 50 millisieverts?

A. I believe that the LSS can inform us down to around 100 millisieverts.

MR JUSTICE BLAKE: Right.

A. Below that we take the LSS and we apply this factor. I have confidence that that is an appropriate thing to do because we have evidence from the UK workers' studies, the study that we have started by NRPB.

MR JUSTICE BLAKE: Yes?

A. And that has, at the last analysis, looked at 179,000 UK workers, who received -- I think the average dose there was down to 25 millisieverts over their lifetime. And if you apply a DREF of 2 to the lifespan study and you draw a straight line down to zero and you look at the risks we get from the worker study then those two straight lines virtually coincide for solid cancers and for leukaemia.

So at least in a broad sense we see very good agreement between what we see at the lifespan study at high doses and applying a DREF of 2 to what we see at the much lower dose worker studies that we have in the UK.

MR JUSTICE BLAKE: That could be 25 millisieverts?

A. The average is 25 millisieverts. Some is lower, some is higher, some is higher, obviously. But, yes.

So that gives me confidence that, using the models that we get from the LSS and applying a DREF of 2, would in a broad sense be appropriate for somebody from the UK population.

MR JUSTICE BLAKE: Yes.

A few moments ago, I think wrapping up some of the propositions that Mr ter Haar was exploring with you, you explained that you use what you considered the most appropriate model.

A. Uh-huh.

MR JUSTICE BLAKE: And you didn't, or you weren't asked to go and see whether, if you used a wholly different model, whether that would have challenged or undermined your results.

A. That's correct.

MR JUSTICE BLAKE: You said, I think, it's the best model.

You're aware of other models, or are you not aware of any other model?

A. There are a range of good and bad models out there, my Lord.

MR JUSTICE BLAKE: Right.

A. I could have calculated probability of causation on the basis of a whole range of models and presented a whole range of possible probabilities of causation but I was not asked to do that. I used my judgment and chose the model I thought was best.

MR JUSTICE BLAKE: Right. It's a question of trying to get some sense, if this is possible, and it may be we're limiting -- as to what you may have discarded. I think if you see a model which you think is a bad model, I don't think that would be much use in you calculating a dose using a model which you assess to be bad. But are there models which are different to the one you used which you don't necessarily think are bad?

A. There are a range of other models. For instance, the BIR Committee in America has produced some models I could have directly used a model from our workers' study. But, again, that would be based upon just a single study. I defer to the judgment of ICRP and its recommendation in 103 that it recommended the models of the lifespan study as being the best currently available for estimating risk, and as in general they do agree with what we see in our UK worker study, that gives me confidence that they could be applied.

MR JUSTICE BLAKE: Yes. Just give me a date for the last time the ICRP continued to issue that recommendation.

A. It was 2009, I believe.

MR JUSTICE BLAKE: 2009.

A. I would have to double-check but I think it's 2009.

MR JUSTICE BLAKE: It rings a bell. But I do not have it at my fingertips.

In 2009 they would have at least seen the BIR study?

A. Yes, they reviewed all the material.

MR JUSTICE BLAKE: And the lifetime worker study?

A. Yes.

MR JUSTICE BLAKE: And they are still saying what they say?

A. Yes.

MR JUSTICE BLAKE: Since that date, if it was 2009, has any other candidate come on which has not yet been reviewed

by ICRP which might pose questions to the sufficiency --

A. The only significant publications that I can recall at the moment relate to the International Workers Study, which is called INWORKS, and that was a follow-on to the 15 country study which -- these studies aim to bring together worker cohorts from around the world to, again, pool data, increase statistical power to detect risk again at lower doses. So the 15 countries study took place a while ago. More recently we've had INWORKS. That had a slightly smaller overall population but the population was followed for longer and there were more deaths and cancer incidence in it. That study also was in broad agreement with the lifespan study which again gives me confidence that those models were appropriate.

MR JUSTICE BLAKE: Right. So INWORKS, at least, far from perhaps challenging ICRP 2009, is another form --

A. It doesn't challenge it --

MR JUSTICE BLAKE: -- of supporting data?

A. Yes, yes.

MR JUSTICE BLAKE: The 15 countries?

A. That was an earlier version of it and, yes, that broadly agreed as well.

MR JUSTICE BLAKE: All right.

A. There were some slight difficulties with that one.

MR JUSTICE BLAKE: All right. Thank you. That just deals

with matters immediately arising in the back of my mind. I didn't want to forget about it. Yes, Dr Busby, do you want to make a start now?

DR BUSBY: I can do, my Lord.

MR JUSTICE BLAKE: Shall we do 10 minutes?

DR BUSBY: Yes.

Cross-examination by DR BUSBY

DR BUSBY: Dr Haylock, good morning.

A. Good morning.

Q. I think you have said what your area of expertise is but I wonder if you could just distinguish between biostatistics and epidemiology. Is there a distinction that you could make?

A. They are fairly loose terms that are -- people call themselves epidemiologists but there isn't necessarily a particularly well-defined definition of that term. People come from either a medical background or often, as I do, from a mathematical statistical background. I would suggest perhaps biostatisticians would come from the latter, more of a statistical --

Q. Would it be fair to say that you are an extremely clever and competent processor of information that comes to you from other people with regard to, if you like, the inputs to you, if I can imagine you as a gigantic computer, like Deep Thought, the input would be so many

people have cancer in such and such population of such and such ages and their doses were such and such and you go -- all sorts of things happen in your head and the answer comes out?

A. I'm afraid I'm not sufficiently clever to be expert in all these various fields so, no, I do rely upon the quality of the data that's provided to me.

Q. Quite. So your conclusions in this case in particular are totally dependent on the input, if you like, if we can call it that?

A. Of course.

Q. That might not perhaps be so certain in the case of someone that you might call an epidemiologist rather than a biostatistician because they would consider all sorts of epidemiological aspects of the study, more perhaps than you would?

A. I don't agree with that.

Q. You don't think that's true?

A. No. I have lots of expertise over many years looking at different studies and reviewing their qualities and issues.

Q. Right. But if, for example, there were some questions over the input end of what it is that you've done, so for example the doses that you have received from Mr Hallard, if they were shown to be wrong or uncertain,

then your conclusions would be uncertain or wrong in the same proportion roughly?
A. Obviously.
Q. Right.
Similarly, I'm not sure if you were here the whole time that we were discussing these issues, if the concept of dose was wrong -- I mean dose is the covariate that you use, I mean perhaps the number that you use in your calculations -- if there was a question mark over the validity of the concept of dose, would that also affect your conclusions?
A. I think I've used the dose -- the concept of dose I've used as provided by -- the dose provided by Mr Hallard is the same as the concept of dose that is used in the carrying out of the epidemiological studies. So they are in a sense consistent. If they are all wrong, then of course it's all meaningless but I don't believe that's the case because of the fact that we seek consistency across completely different populations which would be difficult to imagine if the whole concept was completely wrong.
Q. But these different populations that you look at are really mostly people who are exposed to external doses, are they not?
A. The worker populations, a good proportion of them, my

best guess is perhaps around a half, are also exposed to internal or are monitored for exposure to internal radiation.
Q. They are monitored?
A. They are monitored. Whether --
Q. But you don't know what the actual internal radiation doses are, do you?
A. Not in the UK worker study I referred to just now, no.
Q. In fact not in any studies?
A. Some studies have measured it, yes. For example, the studies of the Russian radiation workers at the Mayak plant, there are estimates of the internal exposures.
Q. But those estimates are not based on dose -- on analytical data, are they?
A. Yes. They are based upon bioassay data that is then fed into a dosimetry model to estimate doses.
Q. Would you agree that there are various questions over those studies because they are retrospective studies and many people in the cohorts will have died or not be included in the studies?
A. The studies are not without issues, I admit. I --
Q. Well, we don't have enough time to go into all this in great depth but I think my point was that if there were some concerns about the concept of dose it would affect your conclusions, wouldn't it?

A. Yes.
Q. Yes. Okay.
Now, I think you said -- I just want to be sure about this -- you don't really have any expertise in other relevant areas like medicine, biology, environmental modelling on exposure?
A. No, my background is mathematical statistics.
Q. You are, as I said, I sort of processing machine for information?
A. I take that as a compliment.
Q. At the mathematical end of it -- yes, right.
Now, your calculations also depend upon the safety of essentially what we should call the model of the ICRP. There are various versions of this but they are all roughly the same.
A. I chose the ICRP models to represent the best models I could use to do the calculations I did.
Q. But again, if the ICRP model were wrong for the specific -- and you will have heard enough evidence here that we are arguing that it is -- if it were then again your own conclusions --
A. Of course. If the model is substantially wrong then my calculations would be wrong and my calculations are based upon the model.
Q. That's right, okay.

Are you a member of the ICRP?
A. No, I'm not.
Q. Have you any relationship with the ICRP?
A. I had -- for the latest recommendations, ICRP/103, I was asked to reproduce the calculations of lifetime risk to check that I got to the same answer as the chap who calculated the risks in there and I --
Q. Sorry, yes. Your predecessor, Dr Cooper, was a member of the ICRP. He was also the head of the National Radiological Protection Board at the same time.
A. Uh-huh, correct.
Q. So is it fair to say that the outfit that you come from has very close connections with the ICRP and with all of these risk agencies that you use for your calculations, that you rely on for your calculations?
A. Thankfully I work with some quite eminent people in their fields and ICRP seeks out eminent people to do its work, and therefore they have been asked. Yes, we do have close relationships --
Q. Quite right, what you might expect. But I think my point, where I was going with this is that I want to ask you about what you consider might be a sort of culture of scientific thinking that in fact -- or might I suggest to you, ask you for your comment, that ways of seeing scientific information or evidence or data might

be coloured or biased -- I'm not saying in any bad way, but just culturally by the group of people to whom the scientist who is making the assessment belonged?
A. I would like to think that my colleagues are reputable scientists who base their judgments on the evidence and are not coloured in that way.
Q. Of course. I'm not suggesting --
A. It's a matter of personal integrity of the individuals, I believe.
Q. No, no, I'm not -- don't get me wrong. I'm not trying to cast any aspersions here. I'm just asking about a more philosophical or perhaps psychological point which is relevant to all of this, which has to do with the ways in which you see the world being coloured by the people that you associate with, if I can put it like that.
A. I suspect that's true of everyone.
Q. Yes, of course, of course, but in this case it may be more important.
A. As I said, I think that the ICRP prides itself on the fact that it bases its judgment on evidence and not anything else.
Q. But what is emerging in the discussions here and in the submissions that we have made and the evidence that we have seen, particularly from Professor Schmitz

Feuerhake, who has been in this game if you like since about 1975, is the idea that there are groups of people who have an interpretation of the facts. I think nobody disagrees about the facts, although there are various tiny disagreements, perhaps, but the overall facts are up there if you like on the notice board but we have groups of people that make an interpretation of those facts in one direction, and another group of people, in this case I am suggesting the ICRP and the National Radiological Protection Board and all these eminent people that have another view.
My question is, is it possible that the other side, as it were, is actually right and that the ICRP way of seeing it all, you know, might be unsafe? Is that possible?
A. ICRP supports its position with evidence based upon scientific research. If there was evidence to support -- reputable evidence to support the other propositions, I would like to hope that ICRP would consider it along -- in the same way. I don't know that it would not be the case.
MR JUSTICE BLAKE: Let's get the answer. I think that's probably an appropriate time to break for lunch.
I'll just record that. (Pause)
We will resume at 2 o'clock please.

Now, you are in the middle of giving your evidence, which means you can't talk to anyone about your evidence until it's concluded. Thank you. We'll see you at 2 o'clock.
2 o'clock. Are you going to conclude your cross-examination by 4.30 this afternoon?
DR BUSBY: Probably not, my Lord.
MR JUSTICE BLAKE: Well, in which case I may need some information as to when you think you are going to.
DR BUSBY: Well, we'll see where we get to.
MR JUSTICE BLAKE: Yes, we'll see where we get to, but we should also be seeing what you're aiming at.
DR BUSBY: Yes, my Lord.
MR JUSTICE BLAKE: Thank you.
(1.05 pm)
(The short adjournment)
(2.00 pm)
MR JUSTICE BLAKE: Yes.
DR BUSBY: When we broke for lunch I was asking you about scientific culture and how it might colour the way in which one saw -- as a scientist, interpret the information.
From time to time would you agree that science or the scientific world view of something can change? Throughout history we've seen large examples of how

a particular theory has been overthrown by some new evidence.
A. Mm.
Q. Now in your opinion, could that be the case at the moment that there is what's called a paradigm shift in the understanding of radiation and health over the last, say, 10/15 years, perhaps?
A. New hypotheses come along all the time but they are treated only as hypotheses until they are supported by evidence. The current system we see by ICRP is supported by evidence. There has not to my knowledge been any new system that has come along which has demonstrated that it is any better than what we already have with reference to the evidence that we have.
Q. I'm not suggesting that there's anything that might be better than what we have but perhaps it is that the evidence is now showing that what we have is wrong.
A. No, I disagree.
Q. You don't think so.
You don't think that the evidence put forward in this case by Professor Mothersill, for example, who is perhaps the world's greatest authority on genomic instability, might have shaken the foundations, if I can call it that, of the ICRP risk model for low doses?
A. No, the ICRP risk model is based upon epidemiological

evidence.
Q. Right.
A. That evidence still supports the model.
There may be other hypotheses but they have not demonstrated they are better than what we already have at the moment.
Q. Well --
A. There are still hypotheses.
Q. Of course. Let's look at some evidence now that goes the other way.
So, for example, you obviously know about the Sellafield leukaemia cluster, the leukaemia clusters around all nuclear sides which occur at doses which are very, very small; is that correct?
A. There are clusters around some sites, but there have also been shown to be clusters around sites where power stations might have been built but never were. So ...
Q. But is it not true that the sites where power stations might have been built but never were are all by the sea?
A. Most of our power stations are by the sea.
Q. But that's not an answer to the question.
MR JUSTICE BLAKE: Well, it may be.
Do you want to go to specific evidence or are you asking a general question as to whether the leukaemia cluster evidence undermines the ICRP model?

DR BUSBY: I think that's my point, my Lord.
MR JUSTICE BLAKE: No, no, hang on. It's a question you can ask, not a statement.
Let's just put it again. I think I have your answer but I think generically, without going into any particular detailed study but Sellafield was mentioned, it is put to you that the leukaemia cluster evidence points to something wrong with the ICRP model.
A. No, I disagree with that, my Lord.
MR JUSTICE BLAKE: You are sufficiently familiar with the leukaemia cluster evidence to make an informed judgment on that question?
A. Yes, I believe so. The estimated doses from the studies are way lower than could possibly be accounted for.
DR BUSBY: But, with respect, that is exactly our point here in this Tribunal. That is what we are looking at. Because here again it's argued that the doses are far too low for the effects which are clearly observed and which we will talk about later on this afternoon.
A. You're making the assumption that the clusters around the power stations are caused by radiation exposure. That's not necessarily the case. In fact, evidence might suggest that that's certainly not the case.
Q. I think my point wasn't whether it was the case or not the case. It was that you said that there was no

evidence that the model was wrong. All I am asking you is whether there is evidence that the model is wrong, but you deny the evidence on the basis that the dose is too low, which is the model. It's rather a circular argument, it seems to me.
A. I think I've lost the point there. Can you ask the question again, please?
MR JUSTICE BLAKE: No, I think you have answered it.
You do not accept that the leukaemia cluster material is evidence of radiation at very low doses.
A. Correct.
MR JUSTICE BLAKE: That's the answer.
DR BUSBY: Sorry, my Lord --
MR JUSTICE BLAKE: That is the answer, and that is therefore an answer consistent with his previous answers that there is no evidence, there's a hypothesis.
DR BUSBY: Well, sorry, my Lord, I have to say that it's evidence that if there is --
MR JUSTICE BLAKE: No, his answer is that it's not. Now that's his answer and that's what you have. You are not going to improve upon that by assertions as to its quality or nature.
DR BUSBY: Could we go to some more evidence which is SB7/121.
Are you familiar with this paper?

A. I only have the top page. I have read it.
MR JUSTICE BLAKE: You only have the top --
DR BUSBY: You only have the abstract. You should have the full paper.
MR JUSTICE BLAKE: If this has been a recent update, updates don't get into the witness' bundles unless someone does it. Do we have a spare of this? This is the full paper, is it? (Handed)
You are not going to be deprived of that, Mr Heppinstall?
MR HEPPINSTALL: No, no, don't worry.
MR JUSTICE BLAKE: Right.
Have you read this paper before?
A. I've read this paper before, my Lord.
MR JUSTICE BLAKE: Right, okay, you now have the full paper as well as the abstract.
A. Yes, my Lord.
MR JUSTICE BLAKE: Okay.
DR BUSBY: Well, like the Sellafield paper, here is a paper that also appears to show evidence that there was an effect in northern Sweden from the Chernobyl accident. Do you agree with the authors of this paper that that is so?
A. No.
Q. No. Why not?

A. It's a generally a poor quality paper and I don't believe the fact that it uses geographical distribution of doses in place of actual individual doses to be a good point, and therefore I'm not convinced by the evidence in it.
Q. Do you recall if the ICRP -- and you can see here that this was written in 2004 -- do you recall if the ICRP included discussion of this evidence in its 2007 report?
A. I do not recall.
Q. Well, I mean, it actually didn't, but if it didn't, would you find that unusual or unacceptable?
A. No, because I don't believe it's a good quality study.
Q. Quite. So the ICRP probably also don't consider it's a good quality study?
A. I'm not a member of ICRP to respond to that.
Q. But my point is that scientists, therefore, who have a particular view of things can decide whether a study, or what I might call the facts are acceptable on the basis of their decision whether the study is good or not. So they can exclude something from their particular world view.
A. Yes.
Q. Do you think that's acceptable, that you can actually exclude facts from your world view on the basis of a subjective decision?

A. I think if you review a paper and you feel that the evidence isn't of sufficient quality then you should reject it and that is the case, I believe, with this paper. It doesn't meet the threshold for good evidence.
Q. So it's therefore possible that a particular view about whether some area is right -- we're talking about the ICRP risk model now -- can be, if you like, put into a bubble and any evidence that shows that it may be wrong can be just excluded on the subjective decision of the people in the ICRP who don't like it, if I can put it like that?
MR JUSTICE BLAKE: Well, he has explained the answer. I don't think you are going to get much change from this kind of question. It's not a question of "don't like it". It is suggested that the evidence supporting the conclusion is not sufficiently robust to sustain the conclusion, if I understood your answer correctly?
A. That's correct, my Lord.
MR JUSTICE BLAKE: If, therefore, the method, the methodology and the conclusion, is insufficiently robust to sustain the conclusion, it's not considered evidence which requires a response from ICRP. Yes?
A. Yes, my Lord, yes.
DR BUSBY: Thank you, my Lord.
I think that was the point that I was trying to make, anyway.
MR JUSTICE BLAKE: Well, we have that answer.
DR BUSBY: Okay.
Just to be certain that, yes, you don't believe that there's a paradigm shift at the moment?
A. Not at the moment.
MR JUSTICE BLAKE: You've explained the two reasons I've recorded why you have issues with this paper.
A. Yes, the main reason is that doses are derived from -- the assumption is that people in a particular area are receiving a certain dose based upon the deposition in that area. Now, the problem is the deposition -- the dose received by a person does not necessarily correlate well with the deposition in a particular area. Individuals move around in an area. If you take some sort of area average it's not very good. Therefore, the dose measure is a poor quality.
MR JUSTICE BLAKE: So deposition evidence is not by itself sufficient to make an assessment of effective or other dose?
A. Yes, that's my ...
MR JUSTICE BLAKE: Right, well, I record that.
Yes, we're moving back to another topic, are we, Dr Busby?
DR BUSBY: We're cantering on, my Lord.

Yes, we leave that business about the paradigm shifts. I just wanted to ask Dr Haylock about it.
Now, there are certain rules for experts which have been discussed in this Tribunal. As I understand it, one mandate is that an expert report should discuss any different conclusions or opinions and explain why they were not adopted and employed.
Have you read the expert reports in the statement of case of the appellants?
A. I have.
Q. You have. Why have you not addressed any of the arguments and information in those reports?
A. I was asked to do particular calculations for this Tribunal. I have done those. I've also answered the questions that were put to me by yourself and others. And that's it, so ...
Q. But when you originally were asked to provide a report were you directed not to go to any of the arguments?
A. I provided explanation for why I did what I did, in that to me that was sufficient. I mean, I explained why I chose particular models and on what basis I did that. That seemed to me to be appropriate.
Q. Well, it may be appropriate but it doesn't seem to be the way in which experts are supposed to write expert reports in a legal framework as I understand it, but you

weren't told that. You didn't read the CPR35 rules for experts?
A. Yes.
Q. You did read the CPR35 for experts?
A. (Nodded assent).
MR JUSTICE BLAKE: Your statement says so. You confirm at para1.2.
A. I did.
MR JUSTICE BLAKE: I think if this is a legal debate, Dr Busby, we might reserve it for submissions, and there may be a difference between experts who are invited to make calculations applying a conventional model and other expressions of opinion. If you want to ask any further questions by all means but I think probably you are straying into legal rather than evidential inquiry.
DR BUSBY: You are quite right to point out, my Lord, that I know nothing about the law but I did understand that you directed the Secretary of State's experts to provide a response to all of the points that were raised in the statement of case.
MR JUSTICE BLAKE: That sounds like a legal submission to me.
DR BUSBY: Very good. I'll leave that then.
Could I ask you if it emerged that evidence of a major new source of exposure suddenly appeared during

the course of these discussions, would you accept that your conclusions might be invalidated by that?
A. That could be possible, yes.
Q. Possible, yes. For example, yesterday we learned from Mr Hallard that he had not included exposures to carbon-14, and he told us the amount created in the test was 1500 moles, which is a rather strange way of looking at it, but this is the same as 10 to the power 15 becquerels which is not a small dose.
If you accept that it's 10 to the 15 becquerels -- and you can do the calculation or anybody can -- this must represent a significant missing component and dose, especially as we are all made of carbon and so are all the things people were eating on Christmas Island and so forth --
MR JUSTICE BLAKE: Question, please. Try to cut out the statement if you possibly can.
DR BUSBY: Would you say this knowledge, if it were true, had had any effect on your own calculations of causation?
A. I'm not an expert on dosimetry and I would need to see what difference including that source made to the overall effective dose that I would use on the calculations. If it made a very small difference, of course it would have no effect on my calculations but

I'm not a dose expert to make that determination.
Q. Thank you. Yes, of course, it's an input/output problem, isn't it?
Right, I'm going to move on now to epidemiology and uranium.
Because Professor Thomas said to us that she wasn't really an epidemiologist and she referred my questions to her that I should ask them of you. So we're going to go there now.
The excess relative risks that you employ in your probability of causation are based on the ICRP, so the ERRs which therefore depend on the Japanese lifespan studies, is that roughly right?
A. That's right.
Q. I think Mr ter Haar asked you if you had read Professor Sawada's criticisms of the lifespan study which show that internal exposures to uranium and other fallout and washout were invisible to the epidemiology because all of the exposure groups were equivalently contaminated.
So my question is -- well, first of all have you read Professor Sawada's criticisms?
A. To the extent that they made any sense, yes.
MR JUSTICE BLAKE: I mean, he has clearly given a paper -- a witness statement which has some difficulties in

language --
A. Yes.
MR JUSTICE BLAKE: -- re-reading it again. Just pause there.
DR BUSBY: Sorry.
MR JUSTICE BLAKE: I think that he has published other pieces on this topic which perhaps don't have those difficulties. What have you read of Professor Sawada?
A. I've seen the paper we referred to this morning.
MR JUSTICE BLAKE: The 2007 paper?
A. Yes. I don't think I've seen others.
MR JUSTICE BLAKE: He has published on this topic apart from 2007, hasn't he?
DR BUSBY: No, my Lord. He published a paper in Medicine Conflict Survival in 2007 and since then most of these have been unpublished reports.
MR JUSTICE BLAKE: There are two Sawadas, are there?
DR BUSBY: Yes. We are going to go to the earlier Sawada now.
MR JUSTICE BLAKE: Sorry, there is another Sawada who has published on this topic.
So the witness that we heard last week, it is 2007 and his witness statement, those are the two sources of data?
DR BUSBY: Yes.

MR JUSTICE BLAKE: Right.
DR BUSBY: The black rain paper is by a different Sawada.
MR JUSTICE BLAKE: That's where I've gone wrong. Thank you.
DR BUSBY: Professor Sawada's report to this appeal made quite a clever analysis of the non-cancer effects. I know you've just read it and you said it's very difficult to understand, but it's really quite a simple thing that he did. He looked at the non-cancer effects, he looked at epilation and diarrhoea and things that happen almost immediately after the bombing, and he found that they occurred at distances which were too far from the hypocentre for any external radiation dose.
Well out, 6 kilometres away, 7, 8, 9, 10 kilometres, people -- their hair was falling out, their teeth were bleeding, they had diarrhoea. They had all of these effects that other people got when they were exposed to external radiation. So they were radiation effects, well known radiation effects.
If you accept that -- and it's in his report which you've looked, that's basically his point -- what he's saying is there must be something there, some sort of radiation exposure that is not the external gamma radiation from the initial explosion.
Would you accept that?
A. No. I have looked at the DS86 dose calculations for the

lifespan study done by RERF and their conclusion is that there was not significant effects of fallout on the people -- on the survivors in comparison to their external doses. And, therefore, I'm not sure what Professor Sawada shows but I don't think it's necessarily what he thinks it shows, that there is -- there is a significant fallout effect.
Q. Well, the evidence he is using is actually taken from the Radiation Effects Research Foundation disk which gives the data for non-cancer effects, for these epilation and diarrhoea effects specifically for those.
A. These are early effects.
Q. Yes, early effects, correct.
A. Uh-huh, but the lifespan study is concerned with late effects, which are consistent with the type of effects we're interested in for this Tribunal.
Q. Of course. But that wasn't Professor Sawada's point.
His point is that if -- well, let's put it this way. Let's say that we looked at the rates of epilation and diarrhoea, 8 kilometres away from the hypocentre and we found that they were significantly high. This is what he found. This is in the data. So his interpretation was that these were radiation effects but they were too far away from the hypocentre.
Well, is it not a logical conclusion that there must

have been therefore enough, if you like, radiation effects from some source at 6 to 8 kilometres away from the hypocentre? He is suggesting it's the black rain. He is suggesting that what happened is that there was an explosion and therefore rain fell out of the explosion and by that time of course the rain fell at some distance away from the hypocentre and that's his explanation for these radiation effects, these immediate ones.
A. I think I would put to you that the RERF has looked at the same data, if you are saying it's their data, and come to a different conclusion.
Q. I don't think they have. I have not seen any paper where they have done.
A. The DS86 dosimetry system does not include any component for fallout or effects relating to black rain or that sort of thing. They do not consider that it's a significant component of dose. The only significant component of dose that's been ascribed from the DS86 system and the subsequent DSO2 is from the direct effects of the bomb.
Q. I think that's the point that Sawada is making, that that particular type of dosimetry which is based on external radiation dose, joules per kilogram, is not safe.

A. But DS86 does look at internal exposures and the effects of fallout and comes to a different conclusion.
Q. Well, would you say that if you did come to the Sawada conclusion it was something that was a possibility?
A. If, yes.
Q. No, I am saying -- I don't mean that. I mean when you look at the Sawada conclusion that there must have been something there in the way of radiation or genotoxic exposure or internal radiation or whatever --
A. I'm afraid I would want to look at Sawada's data myself before I would make that sort of conclusion. I wouldn't trust his calculations unless I'd seen them myself.
MR JUSTICE BLAKE: You do not have enough material from what we've tried to give you over the short break?
A. It's challenging.
MR JUSTICE BLAKE: So, just so I understand, if you were being asked to examine those four charts we drew your attention to, what material would you really need to conduct your own review?
A. Well, I would want to see the original data from which those charts were drawn and their sources and their reliability.
MR JUSTICE BLAKE: So --
A. But --
MR JUSTICE BLAKE: -- you want to see the data supporting

the charts --
A. Yes.
MR JUSTICE BLAKE: -- before you are doing any calculation?
A. Yes, and I think the premise on which they are working that you can relate dose to distance from the hypocentre is not necessarily correct and open to quite a lot of error.
DR BUSBY: Well, the data is freely available from the RERF. Given that this is such an important issue, do you think it possible that the Health Protection Agency might ask you to do just that?
A. I don't think it is an important issue. Dr Sawada or Professor Sawada has a hypothesis but RERF, who have looked at dosimetry a number of times over the years, I think we're on the third or fourth iteration now, have come to a completely different conclusion. They do not believe that there is significant other exposure to the survivors apart from the direct exposure from the bomb at the time.
Q. So they don't believe that there's any fallout, is that what you're saying?
A. As I understand it.
Q. Well, we're now going to go and look at some evidence that there is some fallout at SB7/110.
MR JUSTICE BLAKE: We are in the same volume, I think, if we

go back to tab 110. This is the other Sawada who is the co-author of this paper.
A. Yes.
DR BUSBY: I also thought this was the same Sawada, my Lord, but then somebody told me it wasn't.
MR JUSTICE BLAKE: Well, I am glad to know I am in good company.
A. I'm afraid I'm not familiar with this paper, my Lord.
MR JUSTICE BLAKE: No. Right, the witness is not familiar with the paper.
What questions do you want to ask? You need to read the whole paper before you can answer any questions about it at all?
A. It's looking like it's a fairly physics-related or dosimetry-related paper and, as I said before, I am not a dosimetrist or physicist.
MR JUSTICE BLAKE: Well, have you read the abstract at the beginning?
A. This strikes me as a paper that would be understood by dosimetrists, my Lord, and not by an epidemiologist.
MR JUSTICE BLAKE: Right. That doesn't look like it's going to be a fruitful line of inquiry.
DR BUSBY: Nevertheless, my Lord, I would just like to read one sentence here at the end of the abstract which says:
"The results of this study suggest that

manifestations of fallout from the Hiroshima atomic bomb are still detectable in the black rain area."
That's just a statement that anybody would understand, Dr Haylock. I mean you don't need to be an expert to understand that. You could say that maybe this paper is wrong or the scientists got their sums wrong or they used the wrong instrumentation or something like that, but you would agree that this a paper that does draw attention to the existence of fallout some distance from the hypocentre including uranium?
A. It may do, but without having read it or having any indication of its quality I wouldn't know whether this was a meaningful statement or not.
Q. Well, I think that's as far as we can go with that.
MR JUSTICE BLAKE: Yes, I think it's probably as far as we can go with this witness. This is apparently a 1983 paper in the Journal of Radiation Research, is it?
A. Yes.
MR JUSTICE BLAKE: Right. So that's a recognised journal on this topic?
A. Yes.
MR JUSTICE BLAKE: Given its age and the place where it was published, will this have come to the attention of ICRP?
A. I would think so, my Lord, yes.

MR JUSTICE BLAKE: And ICRP includes amongst those who constitute it or advise it, physicists?
A. Most definitely, my Lord.
MR JUSTICE BLAKE: And dosimetrists?
A. Yes.
MR JUSTICE BLAKE: Who can evaluate these conclusions?
A. I would think so, my Lord.
MR JUSTICE BLAKE: Even though you are not the person to do that?
A. Yes, my Lord.
MR JUSTICE BLAKE: Right.
DR BUSBY: Would you be surprised, Dr Haylock, if I were to say to you that this paper doesn't appear in any of the documents of the ICRP or the United Nations Scientific Committee or the Biological Effects of Ionising Radiation Committee or indeed any of the committees with which your paradigm, if I can call it that, is associated?
A. That may be because it wasn't considered of sufficient quality to be included. But, as I said, I'm not an expert on this field so I would not be able to comment on that.
Q. Yes. Thank you.
But once again we see that, if you like, evidence may be excluded -- I am just making the general point

here that I made before -- if someone doesn't like it; they say it's not considered to be of sufficient quality.
MR JUSTICE BLAKE: Well, you say "doesn't like it"; he says the evidence supporting the conclusion is not of sufficient quality to justify it. I think you've got to use his terminology if you are going to cross-examine about it, otherwise you are introducing your version of what's going on which the witness probably doesn't agree with.
DR BUSBY: Can we then go to SB7/107.
Would you agree this paper, another paper about residues at test sites, finds excess plutonium and uranium in human bones of the people who lived in --
MR JUSTICE BLAKE: Are you familiar with this paper?
A. I'm not familiar, no. I've not been asked to look at it and I've not. I'm not an expert on the --
MR JUSTICE BLAKE: Do you just want to read the abstract at the beginning to see whether it's something you can answer questions about? (Pause)
A. I've read it.
MR JUSTICE BLAKE: Okay.
DR BUSBY: Yes. My question here is: do you agree that this paper provides evidence that people who live near the test sites, or were near the test sites, ended up with

excess uranium and plutonium in their bodies?
A. I would question whether that -- I would agree with that. It says "the levels within the range found in human bone samples for other countries solely due to global fallout" for the plutonium. And for the uranium it says the levels were consistent with the UK but 10 times higher than those residents of New York City and Japan. So I don't think your statement holds true.
Q. Well, I am just saying that -- I just asked if you thought that it meant that there was uranium in the bodies of people living near test sites?
A. If I understand correctly, we all have uranium in us because it's something we take in on a regular basis.
Q. But this is a particular isotopic ratio of uranium which identifies it as from fallout.
A. I'm not an expert on uranium to comment.
Q. Okay. We'll move on from there I was just trying to get -- we're going along this route of uranium genotoxicity.
We are finished with that one now.
My next question is this: if uranium had an anomalous genotoxicity, such that it could not be properly assessed by the ICRP model -- if, if, if, I am not saying it has, if -- would it follow that the excess relative risks per sievert obtained from the lifespan

study would be wrong?
A. Not necessarily. The lifespan study provides estimates of risks for external gamma radiation. It would depend upon the distribution of the uranium in the participants as well. If the distribution was uniform or random across the population that would not necessarily invalidate the risks we get from external gamma radiation.
Q. Very good. So the risk -- so you would say that the ICRP model, the LSS analysis, if you like, is one of external radiation?
A. Absolutely, yes.
Q. And is invisible with regard to internal radiation, it cannot give us any information about internal radiation?
A. The RERF have determined and proposed that the LLS were only exposed to external gamma and that is the dose that they provide and therefore the models as a result of that of course only relate to external gamma.
Q. And they don't relate to uranium?
A. No.
Q. Well, let's now have a look at some studies which link exposure to uranium to apparent genotoxic effects. If I can take you to -- well, first of all, before I do that Professor Thomas characterised the uranium effects -- there is some argument about how it works,

and there are a lot of papers which I won't take you to because you are not an expert in that area and we will just go nowhere with it. Professor Thomas characterised the genotoxic effects of uranium as due to heavy metal genotoxicity and Professor Howard referred to electron particle effects. I just want to be sure that you don't have any position on this, it is not your area of expertise.
A. Not at all.
Q. Let's go now to some epidemiological studies which we did put to Professor Thomas and she referred us to you and I will now quickly put these to you as an epidemiologist.
Now, these are studies of a number of different people who were exposed to uranium. I want to start with uranium miners at SB7/124.
We have here both the abstract, but we also have the paper there but it may not have made it into your bundle.
A. I have the paper.
Q. It has?
MR JUSTICE BLAKE: Do you know this paper?
A. I had it handed to me the other day and I quickly glanced at it but no more than that.
MR JUSTICE BLAKE: So before the other day you weren't

familiar with it?
A. No, I was not.
MR JUSTICE BLAKE: Yes.
DR BUSBY: Well, the finding of the paper which is in Radiation Research -- would you categorise Radiation Research as a reputable journal?
A. Yes.
Q. You would. It's the sort of journal in which a lot of nuclear stuff is published. They publish -- the finding is in the title:
"Unexpected rates of chromosome instabilities and alteration of hormone levels in Namibian uranium miners."
If you could just quickly look at the numbers there and the P values and let us know as an epidemiologist if you also, if you had been one of the referees here, would have considered agreeing to it being published.
A. Whether I would agree to it being published, I would have to read the whole paper. But my initial thoughts looking at this are that I see that the numbers are fairly small and therefore there's a risk that when you do see things with small numbers that they are occurring simply by random chance and not because they are indicative of some effect.
MR JUSTICE BLAKE: Just remind us of what the kind of

numbers were involved in this study.
A. Sorry, I think we seem to have 75 in the cohort and 31 in the control group.
MR JUSTICE BLAKE: The control group.
A. So I would question are these numbers representative of the groups from which they were drawn?
MR JUSTICE BLAKE: Yes.
DR BUSBY: In epidemiology how does one deal with seeing whether a finding in a smallish group like that is statistically significant or not? What methodology do you use?
A. We usually, if we see something in a study that we were not particularly looking for then we usually refer to that as a hypothesis-generating study, and in which case we would note it as an interesting fact but would want to see it replicated in other studies, ideally in other populations or larger populations before we would accept that it was not simply a chance finding.
Q. So what does it mean here when it says about two-thirds of the way down:
"A threefold increase in chromosome aberrations in the miners compared to non-exposed controls was recorded. P is less than 0.0001."
What does that mean?
A. Very statistically significant.

Q. So what does that mean? What does statistically significant mean in this context then? What are the odds that it could have occurred by chance, say, 1 in 20?
A. 1 in 10,000 in this sample.
Q. So you would have had to do 10,000 of these studies in order to get the result that they got by chance; is that fair?
A. But we would want to know: is this sample representative of the population from which it was drawn? If not, then it doesn't have any implications for the larger population.
Q. You didn't answer my question there.
A. Sorry, could you ask it again then, please.
Q. That you would have had to do -- what did you say, 10,000? You would have to have done 10,000 of these studies in order for this result to have appeared by chance, is that right?
A. It means if you repeated this study 10,000 times you would expect to see as extreme or more extreme results less than 1 in 10,000 times.
Q. So therefore it is unlikely that this is a chance finding, would you agree?
A. It could be -- it could be a perfectly appropriate finding in this sample, but that does not mean this

sample is representative of the population as a whole and therefore that this result applies more widely. It certainly is a result relating to this particular sample. The question is: does that apply more widely?
Q. Well, I mean at minimum you would say it was -- I think you did say -- that it was a hypothesis-generating study?
A. Yes.
Q. In which case we can look at some other studies that show similar sorts of things on the basis that we have generated a hypothesis that exposure to uranium causes chromosome damage.
A. In a sample of Namibian uranium miners.
Q. Well, in a sample of -- could we not agree on a sample of people exposed to uranium?
A. In very specific conditions. These uranium miners might well also be exposed to other things as well. For example, they might well be exposed to radon. Has that been controlled for? I couldn't say from looking at it. But I know from experience that exposure to uranium, often at the same time as you are being exposed to uranium often you are being exposed to radon in the same situation. Has that been controlled for?
Q. I think they did look at that. Anyway, without spending the whole day on this, let's just look at another

example of chromosome damage which is from your own area. Well, perhaps not your area. Well, anyway, the nuclear industry and that is SB7/106.
MR JUSTICE BLAKE: It looks like 106 I've just got as an abstract.
DR BUSBY: Oh right. So have I, my Lord. I think maybe --
MR JUSTICE BLAKE: Well, you know what we've been asking for, ever since Friday week.
DR BUSBY: We thought we put it in, my Lord, according to my daughter. Oh, we didn't, all right.
MR JUSTICE BLAKE: If we are all in the same boat, I think the possibility of a random error is statistically insignificant, without trying to give evidence, of course.
DR BUSBY: I am sure Dr Haylock could put a P value to it, my Lord.
MR JUSTICE BLAKE: Right.
DR BUSBY: All right, well, we can't use that one then. Let's go to SB7/119.
MR JUSTICE BLAKE: "Chromosome aberration analysis in peripheral lymphocytes of Gulf War and Balkans War veterans", Schroeder, 2002.
DR BUSBY: Are you familiar with this one? Sorry, you haven't got there yet. (Pause)
MR JUSTICE BLAKE: What's your 119?

A. Sorry, my Lord?
MR JUSTICE BLAKE: 119.
A. Yes.
MR JUSTICE BLAKE: I think you have the abstract, page 1. Flip that over and then you have the full paper, I hope; yes?
A. Yes, my Lord.
MR JUSTICE BLAKE: Right.
DR BUSBY: Well --
MR JUSTICE BLAKE: So the first question, are you familiar with this paper?
A. I am not familiar with it, no, my Lord.
MR JUSTICE BLAKE: Right.
DR BUSBY: Well, this paper, I'm not sure how far we can go with it in the same way, but anyway if the --
MR JUSTICE BLAKE: Shall we let him read the abstract to see whether this is a topic on which he might be able to help you with questions and answers? It's depleted uranium, is it? (Pause)
A. I've read the abstract. My first thought would be that they were looking at a very small group of volunteers here that were exposed. Again I would question whether such a small sample size enables us to draw the conclusions that are more widely applicable.
MR JUSTICE BLAKE: We have --

A. 16, my Lord.
MR JUSTICE BLAKE: 16 volunteers. Yes.
DR BUSBY: Would you consider this a hypothesis-generating study?
A. Potentially.
Q. So we have two hypothesis-generating studies?
A. Yes.
Q. And they are the same hypothesis?
A. I would have to read them to confirm that.
Q. Well, the hypothesis is that exposure to uranium causes chromosome aberrations.
A. I think this is depleted uranium. I'm not quite sure that's exactly the same as in the other study, is it?
Q. The other one is uranium and this is depleted uranium.
A. Are they the same?
Q. Yes.
A. I'm afraid --
Q. Essentially they are the same, yes?
A. Well, I'm not familiar enough to comment on that, whether these do actually generate the same hypothesis.
Q. Okay. So let's go to another study that is also about uranium and Gulf War veterans, and that is SB7/93.
We seem to have the wrong reference here, my Lord.
MR JUSTICE BLAKE: I think you do.
MS BUSBY: Yes, so that is the right one but this is not in

our bundle in the right place.
MR JUSTICE BLAKE: "Prevalence of birth defects among infants of Gulf War veterans in Arkansas, Arizona, California, Georgia, Hawaii and Iowa"?
DR BUSBY: Yes, that's the one.
MR JUSTICE BLAKE: Right. Do you have that, 93?
A. Yes.
MR JUSTICE BLAKE: Do you have the Areneta and others paper?
A. Yes, I do.
DR BUSBY: Our hypothesis-generating study of uranium miners took us to another hypothesis-generating study of uranium-exposed -- DU, it's the same thing -- soldiers, and now we go to some more DU-exposed soldiers but we're now looking not at chromosome damage but at birth defects.
Now when I took this to Professor Thomas she said we should ask you about this and ask you whether you thought this was a reputable --
MR JUSTICE BLAKE: Have you read this paper?
A. No, my Lord.
MR JUSTICE BLAKE: Were you asked to read it?
A. No. Sorry, my Lord.
MR JUSTICE BLAKE: Do you want to read the abstract and see what it's all about? Sit down by all means, if you prefer. (Pause)

DR BUSBY: Well, you'll see here that the population base was 684,645 veterans who were deployed in the Gulf War. Is that a large enough study to overcome the problems of small numbers?
A. It might be, depending upon the size of the effect you are looking for.
Q. Well, they found a significant effect on birth defects so I think that's all we have. I mean, do you agree from what you've seen --
A. From what I can see from some very small writing, yes.
Q. Yes, I know this is all a bit -- so yes, that's a "yes". So this is a paper that -- we've now gone from uranium in miners to uranium in Gulf War veterans now to birth defects in Gulf War veterans.
A. Could I point out, though, that birth defects is nothing to do with cancer. You're talking about defects in offspring of people. It bears no relation to cancer and the risk of cancer in individuals who were exposed themselves.
Q. I am not suggesting that that is the case. We're just looking at the ICRP risk model and --
A. The ICRP risk model says nothing about this.
Q. The ICRP risk model does actually give a relative risk for birth defects.
A. Okay, but not in relation to the kind of models I was

using to -- birth defects. Okay.
Q. I think, yes, well, we'll explore this relationship between the birth defects and the cancer. But all I am trying to do at the moment is to go along to show that maybe uranium has not been adequately examined with regard to its genotoxicity.
You would agree, would you not, in passing that cancer is essentially a disease that follows from genetic damage, from damage to DNA?
A. I'm an epidemiologist, not a medic or a biologist, so I deal in deaths and cancer incidences and doses and the calculation thereof but I would not agree to your statement because I don't have that --
MR JUSTICE BLAKE: You can't comment upon the causes of cancer?
A. I'm not qualified in that area.
DR BUSBY: Okay. Just to continue with this quickly, we want to look at Mr Kang which is SB7/98.
When you are ready. I understand that you are not -- do you not have the main paper?
A. I only have the abstract, I'm afraid.
Q. The main paper, this one is there, so it must not have gone into your bundle. Can somebody help? He doesn't have this in his bundle. Well, he can have mine.
MR JUSTICE BLAKE: Hang on a moment. (Pause) (Handed)

A. Thank you.
DR BUSBY: Well, this is another study --
MR JUSTICE BLAKE: Are you still reading this one?
A. I think I've read as much as I can.
MR JUSTICE BLAKE: Okay.
DR BUSBY: This is another study like the Areneta study that you just looked at, and mainly I'm not asking you if it's in your area of knowledge with regard to birth defects and so on but basically as an epidemiologist would you consider this to be a reasonable study because we asked Professor Thomas and she referred us to you.
A. Certainly the size of the study looks good, but again that could only be confirmed with proper calculations. But it does appear on the face of it to have been a properly conducted study, but ...
Q. And it was a study by the Environmental Epidemiology Service of the Department of Veterans' Affairs, Washington, and the Food and Drug Administration Department of Health and Human Affairs, Washington, and the Office of the Under Secretary for Health, Department of Veterans' Affairs, Washington.
Would you consider those to be people that you might listen to or --
A. They sound good, but I have no personal knowledge of these organisations myself.

Q. No, but you wouldn't see them as dodgy characters if I could put it like that?
A. As I said, I have no personal knowledge of them to evaluate that.
Q. Okay, thank. But you might consider then that this is another hypothesis-generating study?
A. What hypothesis are you suggesting.
Q. Well, that people who were exposed to something in the Gulf War had a higher risk of congenital malformation in their children?
A. It is suggesting that people who were at the Gulf War have a higher risk but as far as I can see it's not attributing that to anything.
Q. No, indeed, I'm not suggesting it is, but it's one more -- if I may put it -- supporting piece of evidence that there may be something wrong with the assessment of risk of harm of genetic damage from uranium?
A. No, I disagree. It says nothing at all about uranium. How can it --
Q. Well, it's supporting evidence. If uranium causes some damage then you go and look at people who are exposed to uranium amongst other things --
A. It's not saying these people were all exposed to uranium.
MR JUSTICE BLAKE: I think what the witness has said is that

from examining the abstract, as I understand is what you've done, it shows that there is some outcome of people who were employed in the Gulf War but it doesn't show what caused that outcome, therefore you need to have some evidence to say it was uranium exposure that caused the outcome before you can go back to say there is a failure in the risk model assessment of ICRP because of uranium exposure. Do I have that right?

A. I believe so, my Lord, yes.

DR BUSBY: Can we go to SB7/122 now.

A. I have not seen this publication before and I would question actually whether it is actually a peer reviewed publication looking at what it says on the front that it's a faculty publication. But maybe I am wrong there.

Q. I think it is a faculty publication, yes, rather like the ICRP risk model.

MR JUSTICE BLAKE: We're not going to get involved in that kind of skirmishing, Dr Busby, so withdraw that. I am going to police you to make sure that you are confining this part of the hearing to asking questions of witnesses whose expertise falls within the proper scope of the questions. You are not going to be making statements, they will be ignored.

DR BUSBY: Thank you, my Lord.

Well, how shall I put it? The authors of the study

are all based at the University of South Carolina. Would you just discount this study? Because then we can move on if you do because it has not been published in peer reviewed literature.

MR JUSTICE BLAKE: He hasn't had much time to look at it. What do you want to --

DR BUSBY: I wanted to ask him if he thought it was evidence -- more evidence -- that uranium -- because these are very low levels of uranium in groundwater -- caused cancer at doses that would not be expected on the basis of the ICRP risk model.

A. I would caution drawing conclusions from a study like this because it appears to be an ecological study, where you are comparing risks in an area to exposure in an area and that's a thing which can only be done with great caution because you're assuming that everybody in the same area has the same exposure and that is almost certainly not the case.

Therefore, drawing these sorts of conclusions can be very dangerous indeed.

MR JUSTICE BLAKE: That's a point you made previously.

A. I would not want to put any weight on this, I'm afraid.

DR BUSBY: None at all?

A. Well, if I had the opportunity to read it in detail I might find some interesting points. But on the face

of it these sorts of studies are usually of poorer quality because they don't have individual estimates of the exposure and assuming that the average for a particular area is appropriate to be assigned to individuals and that's in general not the case.

Q. So you discuss -- not discuss -- you dismiss this sort of ecological approach that somebody in a particular area can be categorised in terms of some quality relating to that area?

A. I said that the issue is that in this sort of study you're assigning the same value of exposure to everybody in a particular area, and an example like this with groundwater if it's being used for drinking, for example, not everybody would drink the same amount. People would not spend the same amount of time in the area. The variability -- if you tried to do the relationship between personal exposure and this average it would be -- there's not a good relationship between the two.

It is not impossible that you can do good quality ecological studies but trying to draw any conclusions is quite challenging, I'm afraid.

Q. Yes. Well, I mean the LSS study is exactly such a study, is it not?

MR JUSTICE BLAKE: That's a statement.

A. I disagree, I'm afraid, because the LSS does have individual dose estimates --

DR BUSBY: How were they obtained? How were the individual dose estimates obtained?

A. My understanding is they were obtained by using a model and on information gleaned from individuals at interview when the study was set up.

Q. Yes, but the model, was it not -- was it not true that the model was based essentially on the distance the individual was from the hypocentre as Mr ter Haar suggested to you earlier?

A. But importantly also on shielding and it's more complex than that, most certainly.

Q. But it is still an ecological model, is it not?

A. No, I disagree because there are individual personal dose measurements.

Q. There were no dose measurements, Dr Haylock.

A. Well, calculated doses on an individual basis, not on assigning a particular dose to everybody who was in a particular area. That does not happen.

Q. As I understand it that's exactly what they did and then later on they modified it with various shielding co-efficients on the basis of questionnaires but the initial dose was defined in terms of distance from the hypocentre.

A. But we are not using the initial doses. We're using three iterations further down the line in terms of the DSO2 dosimetry system which has been refined and evaluated over a period of time.
Q. Professor Sawada was involved in actually building up the DSO2 system and we do know that the way in which it works is that essentially the doses were based on the distance of the person and then that was modified by shielding, so essentially the main -- nobody wore dosimeters, did they?
A. No, definitely not.
Q. So all of this was done retrospectively on the basis of two major components. One is how far they were and this was carried out on the basis of Nevada test site measurements with dosimeters with a similar bomb. Do you agree with that?
A. I don't have any personal knowledge of that, but --
Q. Then what they did was they built concrete walls and they put dosimeters behind concrete walls and so therefore they got various shielding of components and then refined the dosimetry to the point where we have it today. But the essential dosimetry was based on distance and then it was refined afterwards. Would you agree with that?
A. Yes, and shielding as well.

Q. So it is essentially, it is -- although it's maybe a bit more sophisticated it is what you call an ecological study?
A. But each individual as I understand it reported where they were at the time of the bomb so that each individual would have an individual dose measurement. We're not talking about basing individual measurements on an average of a particular area. So I still maintain they are individual measurements and not measurements defined on an area basis.
Q. Well, I think that's as far as I can take that one.
Let's just go to another one of these uranium studies which is SB6/85.
MR JUSTICE BLAKE: So we can put volume 7 away, I hope, and take out volume 6.
DR BUSBY: There are two papers in this tab. I am interested in the Canu 2008 one.
MR JUSTICE BLAKE: I think I only have one. Anyway, Canu, that's what we have, "Characterisation of protracted low-level exposure to uranium in the workplace"?
DR BUSBY: Yes.
MS BUSBY: Just behind it is a second paper, I think, my Lord.
DR BUSBY: It's the one that you mentioned, my Lord. That's the one that I am interested in. (Pause)

MR JUSTICE BLAKE: Do you know this one?
A. I have seen it before, yes, my Lord.
MR JUSTICE BLAKE: Right, okay. Hold fire then. Yes. He has seen it before.
DR BUSBY: Are you familiar with the work of Dr Canu?
A. Only as a result of this --
Q. This particular one?
A. Yes.
Q. She's written a whole series of papers on this issue.
A. Yes.
Q. Well, I won't go on and on about it. It's just one more piece of evidence that there are --
MR JUSTICE BLAKE: What conclusion do you want to put to the witness to comment on?
DR BUSBY: Do you agree that this paper and maybe the other ones you've seen --
MR JUSTICE BLAKE: Deal with this paper.
DR BUSBY: -- suggests that there is an anomalous risk from exposure to uranium and in this case we're talking about French uranium workers, in this case there is an excess risk of --
MR JUSTICE BLAKE: Just pause there. The question is, I think: do you agree with the authors of this paper that there is an anomalous risk to radiation in this study of French uranium workers?

DR BUSBY: Yes.
MR JUSTICE BLAKE: That's the question.
A. There does appear to be an elevated risk. My concern about this paper is that it's not based upon what I would say proper dose measurements. It's based upon what we call a job exposure matrix system and a categorical quantification of dose, so not real measurements of dose. So I would say that it's potentially hypothesis-generating but certainly not any more than that.
MR JUSTICE BLAKE: I missed the first part. I have: "My concern is the quantity of dose ..."
A. It's not a numerical measure of dose, my Lord. It's based upon what we call a job exposure matrix system whereby we look at what jobs people do --
MR JUSTICE BLAKE: Job exposure?
A. -- and the exposure a typical worker might have and then those are applied to other workers and therefore we have that sort of system.
MR JUSTICE BLAKE: So the work proceeds on a hypothesis of what a particular job function might be exposed to?
A. Yes. I think if I remember correctly we're talking about low, medium or high exposure but there's no indication that this is related -- I do not believe there is anything as sophisticated as taking into

account the duration of exposure, the duration of employment and things like that in the exposure. So at the best I would categorise it as a hypothesis-generating study.

DR BUSBY: So this is another hypothesis-generating study and the hypothesis is that uranium might be anomalously genotoxic, is that correct? That would be the hypothesis that it's generating?

A. No, it's simply a hypothesis that you are seeing excess risk in particular groups exposed to particular levels.

MR JUSTICE BLAKE: I think if you go to page 276, just navigating through this paper of which I have absolutely no knowledge, do you see there's a heading "Conclusion"?

A. Uh-huh.

MR JUSTICE BLAKE: Do you want to just read that? (Pause) Okay, is there anything else you want to say about this paper?

A. Only that the authors do recommend at the end that further investigation is required and that this is not a definitive study by any stretch of the imagination. I believe that is actually beginning to happen now, my Lord.

MR JUSTICE BLAKE: Right. Any other questions you want to pose about this paper, Dr Busby?

DR BUSBY: Well, I just wanted to ask if you thought that

the authors of this paper, who worked differently for Oakridge in Tennessee and for the French nuclear industry, would be accepted to be, if you like, acceptable scientific --

MR JUSTICE BLAKE: Are they credible experts in this --

A. The only one I know personally is Margot Tirmarche, and I believe she would be considered as a suitably expert person. I don't know the others.

DR BUSBY: So Dr Haylock, they obviously thought there might be a problem and that all of these hypotheses we have been looking at --

A. Like said, this is the kind of --

Q. -- were sufficiently generated for them to get --

MR JUSTICE BLAKE: Right, it's a hypothesis --

A. This is a hypothesis-generating study, yes.

MR JUSTICE BLAKE: I think that will probably do on that topic.

DR BUSBY: Well, we were just going to -- I think Dr Haylock has already admitted that there is a DoReMi study --

MR JUSTICE BLAKE: On this paper, yes?

DR BUSBY: On this paper, yes.

MR JUSTICE BLAKE: We are going to take a break. Take a seat for a moment. We'll come back at half past. Now, we will carry on this afternoon until 4.30. If you know there are epidemiological papers that you are

going to put to this witness this afternoon, can you make sure he is alerted to them so that if you want him to refresh his memory from them over the break you can pass that information on to Mr Heppinstall. I would have expected by 4.30 that you would have made very substantial progress in your cross-examination. If you haven't concluded I am going to indicate now that you've got to conclude by 11 o'clock tomorrow if we start at 10. Yes?

DR BUSBY: I can do that, my Lord. I think I'll possibly not conclude today.

MR JUSTICE BLAKE: Well, I'm just trying to set some parameters and then that would enable any re-examination, I hope, within reason, and anything else that we need to discuss before one o'clock. Yes?

DR BUSBY: Yes.

MR JUSTICE BLAKE: That's just some timing. So see how far we get. Obviously the more the witness is alerted to these papers you may get a better quality answer.

DR BUSBY: Thank you, my Lord.

MR JUSTICE BLAKE: Half past then. The same rules apply.

(3.15 pm)

(A short break)

(3.30 pm)

MR HEPPINSTALL: My Lord, I only rise at this stage perhaps

through you to enquire of Dr Haylock how far he has got with the list. It was quite a long list that was handed to him. I'm told that it's to the end of the cross-examination, not to the end of the day, and it looks to me like it fills a whole page of A4.

MR JUSTICE BLAKE: Well, it's too little, too late, isn't it, or too much, too late?

MR HEPPINSTALL: Can you indicate which ones you've read in the last 15 minutes so Dr Busby knows?

A. I know about half of these, I suppose.

MR JUSTICE BLAKE: All right. We'll just see how we go. I think you are going to be back tomorrow morning. Right.

DR BUSBY: Well, we talked about various uranium hypotheses-generating studies and that took me to -- I was about to show you -- I was about to go to SB7/112 and ask some questions about this.

MR JUSTICE BLAKE: So we are back to SB7 again.

DR BUSBY: This is DoReMi, my Lord.

MR JUSTICE BLAKE: Yes.

DR BUSBY: You are familiar with this document?

A. I am.

Q. So what I want to ask you about this is whether you think that, if you like, the risk community, the radiation risk community, has now concluded that it's

possible that there may be some anomalous radiogenetic effects or radiological effects from uranium which are not currently included in the ICRP model, if we can call it that?

Is that a fair --

A. I would say --

Q. You don't think so?

A. No, I would say that this particular document proposes a study that would provide more information on the relative risks of uranium exposure in comparison to external gamma exposure. It has the biological and statistical aspects to look at a range of things. I wouldn't say it specifically says anything about genotoxicity. It's looking at a range of different --

MR JUSTICE BLAKE: It is a document proposing a study rather than drawing a conclusion on existing evidence.

A. It is proposing a study. It is not a study in itself. It is saying: is a study possible?

MR JUSTICE BLAKE: Yes.

A. I believe it concludes it is possible.

DR BUSBY: I think my question is: why are they doing it now?

A. Because we would like more information, more direct evidence, for the difference, if there is one, in the risk from external gamma exposure in comparison to

internal exposures such as uranium to which occupationally exposed people receive exposures.

We currently rely on the factor of 20 to relate alpha radiation to gamma. This sort of study would provide direct evidence, at least the statistical part of it would provide direct evidence as to whether that was correct or whether there were grounds to look elsewhere or reject it.

Q. Would you agree that the decision to go down this route to put a lot of money into this -- and let's be clear it's an awful lot of money going into this.

A. I thought it wasn't an awful lot of money. I wish it was.

Q. Are you yourself involved in this research?

A. Yes, I wrote part of this document.

Q. You wrote part of the document, yes.

So why did you decide to -- why did the group, this DoReMi group, why did they decide to look at uranium in particular?

A. Because there was a high level expert group a few years prior which looked at the areas that the European Commission would like to fund research on, going up to the next 20-odd years, and that group decided that this was an area they would like more evidence on.

That is why this group that I was part of was put

together to look at: was it even feasible to do a study to try and give us more information?

Q. So the fact that this was all done is quite coincidental and nothing to do with any of these hypothesis-generating studies that I drew attention to?

A. It may well be, it may not. I don't know. I was not part of that high level group that decided the direction of European research into the future.

MR JUSTICE BLAKE: So as I understand your answer, for some reason you are not party to the idea of whether a study was considered to be a relevant inquiry, and they then convened a group to see whether such a study was possible?

A. Well, the European Commission DoReMi offered funding for proposals, and I was party to the group which put forward this proposal which said: we would like to see if a uranium study is possible.

MR JUSTICE BLAKE: Right. But why were you asking the question whether a uranium study was possible?

A. Because we know that both miners and radiation workers in the UK, and other European countries for that matter, are exposed to uranium and the current assumption is that we can use this factor of 20 to translate the risk from alpha to equivalent risk to gamma from uranium.

But we know that that value of 20, it's an overall

value used for all alpha emitters but obviously the Commission here is seeking additional evidence as to whether that value is appropriate or whether this sort of study might provide evidence that a different value would be better.

MR JUSTICE BLAKE: Right. So the inquiry is testing the hypothesis as to the way you assess alpha risk from gamma risk?

A. Yes, it's saying: is this value of 20 that we use appropriate or could there be a better value?

MR JUSTICE BLAKE: Right.

DR BUSBY: A better value for uranium?

A. For uranium.

Q. Not just for alpha emitters but for uranium?

A. We're focusing on uranium here, yes.

Q. Is this possibly a concession or at least an attempt to try and see if that value is wrong, the value you use now?

A. Yes, absolutely.

Q. So it might be wrong?

A. It might be wrong, indeed, but we won't know until we look at some evidence.

Q. Of course, but you know that the law relating to these appeals has to do with reasonable doubt?

A. (Witness shrugged).

MR JUSTICE BLAKE: Don't worry about the law.
A. The value of 20 that we use currently is the best value that we have on the basis of the evidence that we have at the minute. We would always like more evidence. This was a project to try and provide more evidence, to see if more evidence was feasible.
MR JUSTICE BLAKE: So this was submitted March 2015?
A. Yes.
MR JUSTICE BLAKE: Is it going forward, do we know?
A. I am myself at this very time working with some of these other people here to put forward a proposal to the current corps from the European Union to do this very study.
MR JUSTICE BLAKE: Right, so you put this one in but you have to go back and knock on some door again?
A. Yes. We are asking for 5 million euros to do the work but the Commission will have to decide whether they would like us to or not.
MR JUSTICE BLAKE: So we don't know if it's going to happen and if it does happen it will take some years presumably.
A. The project we are planning will take three years.
MR JUSTICE BLAKE: Yes. The chances are we would have written our determination by then.
Okay, thank you.

DR BUSBY: We've discussed in some way a lot of studies which suggest anomalous genotoxicity from uranium and therefore we should at least provisionally think there may be a causal relation and this is why this study is being undertaken?
A. The study that this study proposes might reveal something, indeed, but I am not a biologist to know about genotoxicity of uranium.
MR JUSTICE BLAKE: Page 58 and 59 appear to be the conclusions.
A. Mm.
MR JUSTICE BLAKE: Is that a relevant place to search for what you are telling us all about?
A. To a certain degree, my Lord.
MR JUSTICE BLAKE: But you've added to that in your --
A. Again the technology for looking at the biological aspects of radiation exposure is moving on abound and I believe that some of the suggestions have been adapted in the light of new and recent developments.
MR JUSTICE BLAKE: In the light of what?
A. New developments. For example, when we were talking about -- Professor Thomas talked about mFISH being superseded by whole genome analysis.
MR JUSTICE BLAKE: Yes.
A. And I believe that maybe that sort of thing is occurring

in this, that as the cost of more comprehensive and wide-ranging assays goes down we can afford to do more of them in these sorts of studies.
MR JUSTICE BLAKE: Yes, all right.
DR BUSBY: So I am going to ask you about epidemiology. This is to do with this. So it's not just a digression.
In epidemiology do we not normally go from the effects to the cause? For example, in public health if you had a Chinese restaurant and people suddenly started to get ill you would start with the illness and then you would go and see what the cause was, is that right?
A. It sounds plausible.
Q. That's the normal epidemiological approach.
In this case, though, it's not been done like that. What's happening here is that we're going from the effect to the cause, so in other words the doses we use, to decide whether the effect is possible; is that reasonable?
A. That sounds -- yes. We only have limited information and therefore we are basing our study on what information we do have.
Q. So what you are doing is you are comparing acutely exposed Japanese survivors exposed to high levels of external radiation and you are using that information to decide whether or not people who are chronically exposed

to uranium internally can or cannot have a real effect as a result -- a causal effect. That's essentially what you are doing, isn't it?
A. No, these studies would have subgroups within them of workers who are -- or mainly workers who are only exposed to external gamma radiation. They will provide a separate estimate of the risk from external gamma with which we can compare the risk we might see from uranium in the same overall group. So we're not having to use a separate population, a Japanese population, to estimate our excess probability of risk from gamma radiation. We've got within the same overall population of workers.
In the UK we have many thousands of radiation workers over the years and some have been exposed to just gamma, some have been exposed to gamma and other radionuclides. A few have only been exposed to things like uranium, for example, Springfields plant in the UK. And so we are able to get estimates of both uranium and gamma from essentially the same overall population of workers. So we're not doing what you are suggesting, going to the Japanese and --
Q. But the ICRP risk model is not predicated on any study of internally exposed uranium workers at Springfields; is that right?

A. No.
Q. Sorry, I wasn't talking about DoReMi there. My daughter ...
MR JUSTICE BLAKE: Right.
DR BUSBY: So I mean, I think my point is by implication here are we not comparing apples and oranges? You know, if you like nuclear workers externally exposed -- agreed -- and people like Hiroshima survivors, externally exposed -- agreed -- but we're comparing these people with chronic internal exposure to uranium in the Namibian miners, in the Gulf War veterans, in the studies by Guseva Canu, and in the whole range of studies I put to you, it's a different kind of exposure. It is an internal exposure to uranium compared with a dose which is devised on the basis of external radiation?
A. Which is why this sort of study that is just looking within UK workers -- sorry, European workers, shall we say? It's going to be UK, French and --
Q. Uranium workers.
A. It's why looking at just that population is a good idea. It avoids the problems of having gamma estimated in one population and uranium estimated in another population. As you pointed out, I don't believe that the lifespan study tells us anything about internal exposures. We

are using this value of 20 to look at the ratio of the harm from alpha to gamma and that's derived separately.
Q. Thank you, yes. Good.
Now we saw a number of studies which showed excess risk for chromosome aberrations and congenital disease after internal uranium exposure, or partly in some cases internal uranium exposure but also possibly other exposures which you categorised as hypothesis-generating.
Can I now take you to the Rowland studies of the New Zealand veterans which is SB7/123.
MR JUSTICE BLAKE: Now, behind tab 123, I believe -- are you there?
A. I am, my Lord.
MR JUSTICE BLAKE: You should have original article, Wahab, Nickless, et cetera, and Rowland, and then five pages in do you have a second document in the tab, a cytogenetic analysis?
A. I do.
MR JUSTICE BLAKE: Right. So far, so good. Yes. Are you familiar with these papers?
A. I have glanced at the first one but not the second.
DR BUSBY: Your --
MR JUSTICE BLAKE: So you know from the first one what the content is?

A. Yes.
MR JUSTICE BLAKE: Is there an epidemiological aspect to these papers that you can help us with?
A. Not a lot, my Lord.
MR JUSTICE BLAKE: So it's all primary biological modelling?
A. Mostly, and I would point out that the numbers are again very small in this.
MR JUSTICE BLAKE: Yes.
A. It's --
MR JUSTICE BLAKE: Well, I'm not sure -- but I don't know -- that this paper is put forward as an epidemiological paper as opposed --
A. I wouldn't have said it was in the first instance. It might use some epidemiological techniques and there is a dose response relationship there I can see but primarily I don't believe it is.
MR JUSTICE BLAKE: So it's an inquiry into a particular group of servicemen.
Right, well, anyway let's see how we get on. What questions would you like to ask?
DR BUSBY: Do you agree that this study is a significant piece of evidence that the New Zealand veterans, at any event, were exposed to a genotoxic agent, something that caused chromosome aberration?
A. I'm not prepared to comment on that. I would need to

read it in detail. I'm sorry.
Q. All right.
MR JUSTICE BLAKE: If you did read it in detail, would you be able to comment?
A. I might well be able to, yes, my Lord.
MR JUSTICE BLAKE: Right. Well, I think this is one that I would like you to read overnight, please.
A. Okay, I'll do that.
MR JUSTICE BLAKE: I think it's probably best to read this one and the second document in the tab as well, because I think that gives more information. It may be there is some other information in other documents that a cautious approach would direct you to. But I'm sorry -- if you are able to read it tonight we may want to turn to this theme tomorrow.
Thank you.
A. I will try.
MR JUSTICE BLAKE: Yes.
DR BUSBY: We'll leave that aside and go to something that is more in your line of work, which is the -- hang on -- the linearity of the dose response curve. The ICRP has this LNT model in which a line is drawn through all the data points as you go through the origin -- that's correct -- and that's called the linear no threshold model.

Now, can I first take you to -- before I do so, can we agree that cancer is essentially -- it starts as a genetic disease, that it's a mutagen-driven effect?
MR JUSTICE BLAKE: Are you able to assist us on this?
A. That's my understanding but only as a layman, my Lord.
MR JUSTICE BLAKE: He has no expertise upon it. I think he's told us that already, but yes?
DR BUSBY: So can I take you to Professor Schmitz Feuerhake's genetic paper, SB6/89. This is also as an epidemiologist because we discussed -- we tried to discuss this paper with Professor Thomas but she made various remarks about it and essentially asked us to talk to you about it as an epidemiologist, which she said she wasn't.
MR JUSTICE BLAKE: Do you have that?
A. I have the paper, my Lord. But I am not familiar with it.
MR JUSTICE BLAKE: Right.
DR BUSBY: Have you read this paper?
A. No, I'm afraid I haven't.
Q. We asked Professor Thomas to --
MR JUSTICE BLAKE: I don't think it's got through that this witness -- do you want -- this is a paper of some significance to your cross-examination, Dr Busby?
DR BUSBY: Yes, it is.

MR JUSTICE BLAKE: Shall we get him to read that overnight and return to it tomorrow morning?
DR BUSBY: Yes, we could.
MR JUSTICE BLAKE: Would that be --
A. I'll do my best, my Lord.
MR JUSTICE BLAKE: Without overdoing it, yes. This paper has emerged more than once in the course of this hearing. So if you are able to offer any assistance we'd like to know what it is.
DR BUSBY: It is mainly I want to ask you about your expert view of it as an epidemiologist. There were some epidemiological points raised by Professor Thomas which she said that she would hope you might be able to give more --
MR JUSTICE BLAKE: So you want to ask this witness to comment upon the epidemiology used in the construction of this paper, yes?
DR BUSBY: Essentially yes, my Lord, yes.
MR JUSTICE BLAKE: Right.
Thank you. Shall we pass over that one then? Where else did you want to go?
DR BUSBY: So one thing that I can put to you which is in this paper which I could ask you about anyway with regard to the -- we can go along with this question of the linear no threshold response.

What exactly is the basis of the linear no threshold response? Why do you think they adopt it? Do you have any position on that?
A. It represents the simplest model which best fits the data.
Q. But is that really true? Is there --
A. I believe so.
Q. -- no other model that fits the data better?
A. We can compare models statistically and see how well they fit in comparison to each other and the linear no threshold represents the simplest of those models.
Q. What does it mean by "model" in this context?
A. So --
Q. May --
MR JUSTICE BLAKE: Hang on. Do you want an answer to the question?
DR BUSBY: I thought he was huffing and puffing a bit, my Lord. I thought it might make it easier.
MR JUSTICE BLAKE: The thing is if you are going to ask a question, give him a chance to respond before you move on to the next one. Are you able to answer that question?
A. I can try, my Lord.
MR JUSTICE BLAKE: Right.
A. Okay, so if we imagine we have our two axes, our dose

across the bottom and our risk up the side. We have data plotted on our graph. We would like to determine the best fitting relationship to that data that best describes it. So what we would do is let's say we fit a -- choose a relationship we are going to try first. Let's call it the linear relationship. So effectively what we're doing is sticking a drawing pin with a piece of string in in the bottom left-hand corner at 00 and moving our line until we find where the string would go to best describe the data.
Now we do that in terms of in a computer. And in order to find the best position on the line we have essentially a quality a fit function, we have a mathematical function which describes how well that straight line describes the data.
The computer will maximise that function, so find the straight line which best fits the data. So we can do that and get an answer to that question.
We would then choose a different function -- call it linear quadratic -- so we essentially have some sort of curved line and we would repeat the process. We would choose the parameters of that function so that it maximises this quality of fit function.
And what we would expect to see is that the quality fit function, the quality fit value of the more

complicated function is a bit better. So the more -- the linear quadratic which has more parameters fits the data slightly better. But the question is: does it fit to the data sufficiently more betterer (sic) than the linear given the fact it's got an extra parameter? So we look at the difference in the quality of fit function and refer that to a statistical distribution and come out with a P value which represents how much better the more complicated function fits the data than the simpler function. If it fits it sufficiently more better then we would adopt that in preference to the simpler function. If not, we would stick with the simpler function.

That process can be repeated many times with as many different functions as you like and that's essentially what we do until we've exhausted all the potential functions and find what we consider to be "the best".

MR JUSTICE BLAKE: Am I on the right lines when I think I seem to have read somewhere that the papers show other models, such as a minimum threshold or a curved line response, have in the past been examined as well?

A. Yes, yes. The point is that you could fit a model with a threshold but it would have an extra parameter in it but it would have to be seen to be fitting significantly better to be worthwhile having that in. If it doesn't

do that, we stick with the simpler model.

MR JUSTICE BLAKE: So a simpler model unless a more complicated model is significantly better --

A. Yes, that's right, my Lord.

MR JUSTICE BLAKE: -- in fitting the data?

A. To date we find that essentially the linear model for the low dose region fits best.

DR BUSBY: But the best model would be one where you just took a piece of string and you stuck a load of pins in the data points and you wound the string around the data points, wouldn't it? That would be a much more accurate model?

A. Indeed, if we had a parameter in the model for each item of data it would fit perfectly.

Q. It would be a difficult mathematical model to construct but it would do it?

A. It would perfectly represent the set of data you have but would be useless for predicting risk in other populations.

Q. You could, for instance, apply a version of that which is called a low S model, a local regression model?

A. There are various different types of regression like that, yes.

Q. They get closer and closer to the string around the data points is my point.

A. As I said, there are different ways you can fit the model. There are variations.

Q. I think what I am getting at is that in order to do this, in order for the ICRP to do this or for you to do this or for all these various models to be fitted, somebody has to decide on a range of models that seems plausible; is that not right?

A. That is correct.

Q. Nobody decides on a model that they think is implausible.

A. Obviously not.

Q. Why would they?

A. Indeed.

Q. So they have a preconceived notion of various things to do with what happens when radiation increases?

A. But I think it's clear if you look at the data from a lot of these studies they are plotting -- the point of the data gives you an idea of models that you think are likely to fit well and models that are not.

Q. One of which could have been the spring around the data points model?

A. Well, that would always be a good fit --

Q. The best fit, in fact?

A. -- to that specific set of data, but it would not be good for predicting risks in other populations. That's

why I say that in terms of when we're doing the fitting process in order to add an extra parameter the model has to fit statistically significantly better than the simpler model.

Q. In all these models it's assumed -- and this is part of the model approach -- it must be assumed that there is a monotonic increase in effect with dose; that is to say the more dose, the more effect. It doesn't have to be more effect linearly, it could be more effect in all sorts of ways, but there is always more effect. Is that right?

A. It doesn't have to be, no. You could have a quadratic relationship where it does that sort of shape.

Q. Do you know of any model that's attempted to be fitted like that?

A. No, because that doesn't fit that sort of data well.

Q. Well, let's just look at some of the things that we are going to talk about tomorrow in terms of the effects of genetic damage causing congenital malformations.

Now with cancer there are lots of problems because it takes a long time, do you agree, between the exposure and the manifestation of the disease and lots of things can happen in between, so that kind of epidemiology is rather more tricky, would you agree?

A. We recommend when we're talking about leukaemia that it

is likely to occur no sooner than two years after exposure and potentially up to certainly 25 and maybe even 40 and in fact the latest indications of the lifespan study are that it may even be longer than that as well. For solid cancers, the sort of lowest estimate we have is five years, and my preference is to go for ten years to be the shortest period of time. But then cancers could occur from then any time during the rest of the persons's lifetime.

Q. Lots of other things can happen between the exposure and then?

A. Absolutely.

Q. Lots of control-confounding possibilities?

A. If you're a smoker or if you happen to be a sky diver you might find --

Q. Yes, but with regard to congenital malformation which occurs in people who have been exposed to radiation that occurs pretty soon after the exposure, doesn't it? It's not a long gap?

A. I don't know the answer to that question.

Q. No.

Well, I just want to take you through this linear dose response for congenital malformation because earlier you agreed that the ICRP does actually provide a risk factor for heritable damage, a doubling dose?

A. It does, yes.

Q. And that the assumption again is that there's no safe dose and then it continues in a linear way, would you agree?

A. I must admit I can't recall off the top of my head.

Q. Never mind.

So let's just imagine the increasing exposure of some parent and then the sperm and the egg and then the fertilised egg in the womb and then all the way up through to the child that is born with the congenital malformation.

A. Mm-hm.

Q. Now, as we increase the dose from zero, would you agree that the effect would increase?

A. It seems plausible. I must admit it's not something I've studied in detail. It's not my particular speciality.

Q. I just want to go through this thought experiment with you. So then if you increase the dose even more then you might have more of an effect -- increasing the dose you get more and more effect; yes?

A. It's possible. As I said, I'm not a particular expert.

Q. So we have a monotonically increasing effect with dose here from zero?

A. Possibly.

Q. Now at some point you get a miscarriage or the child dies in the womb and there's an abortion and so the baby is not born and there's no effect; would you agree with that?

A. It might well be the case.

Q. In other words, it's not a linear no threshold, it's a linear -- it goes up and then it will come down again, will it not?

A. It might do.

Q. So if you fitted -- if you didn't know that that was the case and you were trying to investigate, say, for the ICRP or in order to provide a paper that might inform this area, you found that there was no increase after a certain dose and you drew a straight line through it, the line would be wrong, wouldn't it, because it would take into consideration something that couldn't exist? As a mathematician now I am asking you.

A. As a mathematician, possible.

Q. Yes.

So I think what I am getting at is to ask you whether you think it's possible that the linear no threshold dose response might not be the correct model to apply to the data points.

A. For congenital abnormalities?

Q. Certainly for congenital abnormalities but for cancer as

well.

MR JUSTICE BLAKE: Deal with congenital abnormalities first, if you can.

DR BUSBY: Congenital malformations.

A. I believe the evidence is quite poor on this subject at the moment. I'm not even sure ICRP does fit a linear dose response relationship to congenital abnormalities. As far as I'm aware they talked about a risk of 1 in 500 live births per -- in the first two generations. I'm not sure that they even do put such a linear dose response on.

So I'm afraid I'm not sure I agree with you.

Q. Well, okay.

MR JUSTICE BLAKE: Then do you want an answer about cancer?

DR BUSBY: Well, I was going to -- before you answer about cancer I was going to ask you if you thought maybe the similar sort over effect -- we've heard from Mr Hallard about hot particles and about high doses to cells and so on. He says that above a certain dose the cells are killed and therefore they can't become cancer.

So my question to you, is it possible that that also is a component of an understanding of cancer? In other words, at high doses to cells there would be a reduction in effect at some point?

A. I wouldn't say there would be -- in terms of when you're

talking about exposure of a whole body we don't see the stochastic effects, late effects, we don't see a reduction and we do see in the lifespan study a plateauing of risk above very high doses, which we do ascribe to cell killing. But I wouldn't say it decreases, no.

Q. I mean, if we look at the nuclear worker studies, for example, which I'm sure you're very familiar with, are they not -- in terms of excess relative risk per unit dose, are not the excess relative risk per unit dose highest at the lowest doses?

A. We don't fit that sort of model, we are fitting a relative risk to the whole dose relationship. We don't compare excess relative risk at the bottom with an excess relative at the top. We are fitting one excess relative risk model to the whole thing --

Q. But this is a decision you've made, isn't it?

A. -- is linear at the moment.

Q. This is a subjective judgment, though. If we'd have tied a bit of string around it we can just have ignored all the high doses. But the effect at low dose would be higher than the effect at high dose, that's my question.

A. But, like I said, we don't do that because it doesn't make sense because then that model would be useless at predicting risk elsewhere.

Q. Well, it would be jolly useful for people who got low doses, wouldn't it?

A. No, because it's specific to that set of data. The more points you put on that line to make it specific to that set of data the less use it is to predict risk in other situations because it becomes more and more specific to the data you have.

Q. Well, let's say you didn't have any data above 20 millisieverts. Let's just say that you just didn't.

A. Right.

Q. And you decided to use the data up to 20 millisieverts to define risk. I think my question is would that risk factor not be much higher than it is if you take the line, using that between the data points where you go to the high doses?

A. We actually do, usually as part of our studies -- and I think I put it in one of my responses to one of your questions -- that in the INWORKS project where we were looking at leukaemia risk we do actually fit an excess relative risk linear dose response to the whole dataset, and then we step down, removing the higher doses and only using the lower dose data, and looking at does the linear dose response relationship change when we do that? In fact, for the leukaemia that we saw in the INWORKS paper there, it doesn't, it remains very stable

indeed. Although, of course, the uncertainty on the dose -- on the slope -- does increase because we have less powerful data.

But there is high consistency between the slope we get just using the low doses and the slope we get using all the doses.

Q. Well, I want to actually follow this up by going to the Cardis 2005 --

MR JUSTICE BLAKE: You don't want to ask about cancers, I take it.

DR BUSBY: This is about cancer, my Lord. I take his answer, but I think we ought to look at some data.

MR JUSTICE BLAKE: Do you want to ask him first a general question about whether the LNT model might not be the correct model for cancers, or do you not?

DR BUSBY: I thought he had answered that.

MR JUSTICE BLAKE: No, he hadn't. You had been asking about other things, as I understand it. I asked you whether you wanted to deal with cancer and you said you wanted to continue with genetic --

DR BUSBY: We were just talking about cancer just now, my Lord. But, anyway, yes, then I will ask you that question, since it appears that you haven't answered it.

Is that do you believe that the linear no threshold model is an accurate representation of the effects of

radiation when we look at cancer effects?

A. I believe at a population level it is the best we have on the available data, yes.

Q. Bear with me a minute, I am trying to find this paper. I have it written down here but I got a bit wrong footed because we had to leapfrog.

Yes, here it is, SB6/68. Would you say that this was an authoritative study?

A. Yes.

Q. In fact, it is not your own study that you had in your report -- your INWORKS study -- it's a subset of these people, isn't it?

A. This is an earlier -- this is based on an earlier set of data. This looks at 15 countries. The INWORKS study takes three of those countries, USA, UK and France, and uses data sets with longer follow-up together. So this had 400,000 workers, I believe. We had only 300,000 in the INWORKS study.

But because of the longer follow-up we had more person years and more cancer deaths and instances. So, in a sense, we believe that the later study is more statistically powerful than this.

There were also, unfortunately, some problems with the Canadian data in this study, which means that some of the results might well be not as good as they

possibly could be.
Q. What was wrong with the Canadian data?
A. There was some missing data, as I understand it, there was a subsequent paper published that confirmed this fact that they had missed out people with, if I remember correctly, with a group of people with low doses, that would have skewed the results. I would have to double check on the precise paper, though.
So, if you look, it does, somewhere in here -- I would have to look carefully to find it -- it does note that and notes that the overall value of risk changes notably if you take out the dodgy Canadian data.
Q. Well, who decided that it was dodgy then?
A. The Canadians themselves subsequently published a paper saying that they thought it was.
Q. The Canadian study does actually show a higher relative risk, doesn't it?
A. I believe so but, as I said, there was problems with the data that I believe invalidated it at the time.
Q. It's not that the problems with the data was that it showed a higher relative risk?
A. It was missing a proportion of the workers --
Q. It's all right --
A. -- I can refer to the paper, if you like.
Q. No, no, it's okay.

MR JUSTICE BLAKE: Canada is mentioned at 404, table 2, risk estimates, and over the page. So whether they explain why. Yes, I think it is at page 405, the first paragraph. Do you see page 405?
A. Yes.
MR JUSTICE BLAKE: First column. If you take up "risk estimates in the two countries with the largest numbers of deaths, a significantly reduced risk received the cohorts"...
A. Yes. They did a bit where they excluded one country at a time, and if we excluded the Canadian data then you ended up with a different level of risk.
MR JUSTICE BLAKE: Yes.
DR BUSBY: Right. Well, the first thing that I want to ask you about this is if we look at table -- no -- figure 1, page 404. If we look at -- well, it's hard to see -- but if you look down at the lines that are drawn through this, the various lines that are drawn through this, would you agree that, although they may be the best regression line, if you were to put the bits of string over the data points you would find quite a high point at low doses, would you not?
A. I'm not sure I understand what you mean here.
Q. Well, if you take the central estimate, the little circle for leukaemia excluding CLL -- in fact, we can go

to table 1 and see the exact numbers here -- the data points themselves appear to show that the risk is quite high at the very lowest doses.
In fact, if you take the leukaemia -- take the leukaemia little black circle here, quite close to the origin, and if you go along to the right with it, you find that you have to get to 250 millisieverts before you hit the same level?
A. Mm-hm. But I think you'll note that there's a very wide confidence interval on that point.
Q. Of course, yes, there's a wide confidence interval that gets larger as you get towards larger doses, but the confidence intervals are smaller at the lower dose, aren't they, because there are more people --
A. In general. But that raised point you are pointing to there, if I can see correctly, leukaemia lymphatic, is quite wide. I believe it extends below the horizontal there. So it would not be seen to be, in itself, statistically significant.
Q. But none of these are significant, are they? It's the line that is significant. It's the regression line.
A. Yes, the important thing is the regression line. Here we're looking at just comparing small pieces of data in each dose group.
Q. I think my point is -- I'll ask you this again -- that

the assumption of the regression line is what gives you your significance. Is that right?
A. It's one way of doing it, yes. We are looking at -- here we're comparing -- likely they'll have compared a regression line with just a flat line showing no change in risk with dose.
Q. Yes, but my point is --
A. The fact is that the --
Q. Sorry, go on.
A. -- the fact that the confidence intervals -- the width of the confidence intervals tell you whether that is statistically significant or not. So if the confidence interval dips below the horizontal then that raised line is not different from no risk at all across dose.
Q. Well, let me put it this way. Let's say that there was some mechanism which we don't understand which causes a high level of risk at very low doses. But then, at higher doses, it fell back to the normal regression line, if you like, because the cells have been killed or, for some reason which we don't understand but might discover one day, or maybe isn't even true.
The choice of the regression line, the choice of the linear no threshold model, would completely ignore that, it would just go straight through it and assume it was an outlier. Is that not right?

A. It would go through that, it would ignore it, yes. What we would need to do is to compare the straight line regression line with a line which has some function to describe what you are suggesting in it --
Q. But nobody has done that.
MR JUSTICE BLAKE: Let him answer the question.
A. Again, as I said, it would be a matter of statistically comparing the relative fit of those two models to see if the data that you are suggesting at that low point is strong enough to say that your alternative model fits the data better than the linear dose response model.
DR BUSBY: But what if there was just -- I mean, even as it is, the confidence intervals, as you pointed out, are pretty large.
A. Mm-hm.
Q. So it would be quite hard to get statistical significance for such a data point. Nevertheless, it would not necessarily be -- it would not necessarily be correct to dismiss it on the basis of a preconceived notion about what the dose response should be. That's my point.
Does that make sense?
A. I can see it makes sense, yes. I mean, you could fit, as I say, you fit any relationship you like, but we are using the dataset as a whole dataset and trying to

describe the best relationship overall.
The statistical power of the data at the bottom end, on its own, is relatively low. But if there were extremely high risks at low doses like that then I would expect you to actually be able to see that. But obviously it doesn't appear to be the case.
Q. Well, they wouldn't be extremely high risks, would they, not in absolute high terms, but they would be extremely high risks in terms of the excess relative risk per sievert?
A. Yes.
Q. We can distinguish between those two things.
So, in other words, let's say there is a, I don't know, say a 5 per cent excess risk, or let's say 10 per cent excess risk at a dose of 20 millisieverts, for example. Just as a matter of argument here. Then we can convert that into an excess relative risk per sievert by multiplying by 50. Is that correct? That's how it's done?
A. Mm-hm.
Q. So if that were real, if there were such a thing, and you were to then come along and put that excess relative risk per sievert into your probability of causation calculation -- and we're going to come to that at some point -- you would get a much higher probability of

causation if you use the excess relative risk at low dose than if you took the excess relative risk from the gradiant of the line.
A. Yes, but that's not what the data supports, the data does not support that.
Q. Well, let's look at the data and see if it does support that, shall we? Let's look at table 1. Let's look at solid cancers. If we look at table 1 and we go -- this is a table where, unlike in your INWORKS paper and in most of these papers, the actual numbers are not given, you give a graph. We'll come to that as well because I have some comments about the graph.
But, before we go there, let's look at this.
We're looking at solid cancer. So there are 4,770 solid cancers in these 1,993 nuclear workers. Yes? That's the second column, it says "cause of death, solid cancer, number 4770".
A. Yes.
Q. There we have "observed" and "expected". We see that in the less than 5 group, observed and expected, there are fewer observed than was expected by some small amount.
A. Mm-hm.
Q. But then if we go to the next group, which is 5 to 10 millisieverts, it goes up - 512 over 493. So that is an increase, is it not?

A. It is.
Q. Well, if that were real -- and I know you are going to say, well, this is all (inaudible) of chance and so forth -- but if it were not, if it were real, and if you go down those columns -- and I am not going to do it now because it will take all day -- you will find many many cases where there are higher risks in the low dose region which then come down and then go up again. Would you accept that? I mean, without me having to go through it all and us having to tediously look at it? Or maybe you would like to look at it overnight and come back on it?
A. There may well be.
Q. Right.
MR JUSTICE BLAKE: Does that have relevance to the linear no dose threshold --
A. I don't think so, I think it's picking out little bits of data. Whereas what we're doing is looking at the data overall, which is where the statistical power is. These individual dose groups -- there's not a lot of statistical power in each little bit of information on its own, you need to put all the information together to get the statistical power to demonstrate the dose response.
DR BUSBY: This effect is very clear in the LSS study of

colon cancer. Do you remember the graph for colon cancer in the LSS? I haven't brought it along, but maybe you remember it?

A. Not off the top of my head.

Q. No. Well, I won't take this much further. My only point is that if you did -- if you were to use all of the low dose effects and to compare -- and to use those as the driving force to produce your excess relative risk per sievert, it would be much higher if than if you used your higher no linear threshold line through all of the points?

A. One other thing to point out is the fact that these groupings of dose here, I mean we're talking about estimates of dose for these people. It seems to -- when you look at a table like this you get the impression that you have a particular dose that falls in the category and the person's dose is the person's dose, end of story. That's not necessarily the case. The person's dose is the best estimate we can, we can estimate for that person. We have a degree of uncertainty on that as well, which you don't see in this table.

So I think (inaudible) as well. There's uncertainty in both ways.

Q. Of course. I have one more question, my Lord, and then

we can wind it up for today, if that is acceptable.

MR JUSTICE BLAKE: Right.

DR BUSBY: We'll just go back to the same paper now, we'll go back to figure 1. This your graph. Not your graph, but Dr Cardis. The figure 1 is the Cardis graph, it is on page 404.

A. I'm afraid, I'm lost.

MR JUSTICE BLAKE: You are lost. I think you are about to be asked a question in the same paper in tab 68, page 404, figure 1.

A. I've got you. Sorry.

MR JUSTICE BLAKE: Hold fire until we get the question.

DR BUSBY: This is -- are you ready? -- this says:

"Excess relative risk by dose category."

It has these nice straight lines.

It goes through the origin, zero.

But that's not true, is it? I mean, the excess relative risk is actually not zero because these people are healthy and their relative risk is in fact not the same as the national population, it's already been factored in. Is that not right? They are healthy workers.

MR JUSTICE BLAKE: Do you understand that question?

A. I think so. I mean, this is an internal -- an internal estimate -- an internal study -- so we're not comparing

these people in any sense with the general population. So it's perfectly appropriate for this to go through 00. It's in relation to the underlying risk within this group, not within -- not comparing that group to any sort of bigger population.

So it doesn't matter what the underlying -- how the underlying risk in this group might vary according to the general population, it should still go through 00, even if these were healthier compared to the general population, because they're all the same, in a sense, healthy people, all the same radiation workers. You've not got radiation workers with radiation dose and a matching group of controls who were not radiation workers, they're all radiation workers.

DR BUSBY: But none of them have zero dose, have they?

A. Probably not.

DR BUSBY: No, not probably not.

A. The best estimates might not be. Again, it's certainly possible that -- actually, that's not true, I'm sorry, they might well have zero dose. We have radiation workers who are monitored for whom they've never had a dose shown on their badges. So effectively they do have zero dose. Our best estimate of their dose is zero, so that's not true, I'm mistaken.

Q. Well, if we go back to table 1, it says less than 5 in

table 1.

A. Yes.

Q. So that's the first data point you have is less than 5, it's not zero.

A. We group the people in that bottom end together.

Q. Exactly. So this data point that says zero is not real?

A. Don't forget, this is only -- the group's data you see here are only for interpreting, they are not actually used in the generation of the dose response. It's the individual data there that are used.

Q. I think my point here is, apart from the fact that you haven't got any zero dose people, or at least not in the study, only --

A. -- do you mean by zero occupational dose, I think we probably do our best estimate of some of the people in our cohort is they have had zero dose, they've worn dosimeters, they've done their job, and none of the dosimeters have come back with a reading. Therefore, their dose is what we term below the limit of detection, or whatever that might be. So our best guess is they do have zero dose.

Q. I think my point is that what you call zero dose --

MR JUSTICE BLAKE: What's the question?

DR BUSBY: The question is, is it true that what you call zero dose and what is actually zero dose are two

different things?
A. Zero occupational dose, do you mean, or zero dose --
Q. No, zero dose. I mean, everybody gets a dose, don't they?
A. Yes.
Q. Yes. So --
A. This is relating to -- this is on top of the dose that we all get, and this is in relation to occupational dose --
Q. Yes, and that's in a range of 0 to 5 millisieverts?
A. We group people in that range --
MR JUSTICE BLAKE: That's enough now. We've had five supplementary questions beyond your last point.
Take a seat for a second so we can just discuss tomorrow. Now, we've reached 4.30, so we will adjourn for tomorrow. As I've indicated, you are going to have to complete whatever other course you are going to take by the time we reach the break tomorrow at 11.30.
On the back-burner we have the Rowland paper, the Wahab Rowland paper, insofar as you can comment upon epidemiology. If you can't comment on it, having read it, that's it, no one is going to ask you to (inaudible) you out of your expertise. And the Feuerhake paper to which we referred.
Now, I'm told you presented quite a long list of

reading. I'm afraid it's not fair or reasonable to expect overnight all of those documents to be looked at. Is there one other or two other documents which is going to be an important part of where you are going to go tomorrow?
DR BUSBY: We can -- yes.
MR JUSTICE BLAKE: Which are those?
DR BUSBY: I would have to indicate those -- we don't have the list, my Lord.
MR JUSTICE BLAKE: Don't you know what you are going to go to? (Pause)
DR BUSBY: SB22/22.
MR JUSTICE BLAKE: Just mark them down. (Pause). You have two, you can choose two more.
DR BUSBY: Can't we have four, my Lord?
MR JUSTICE BLAKE: I don't know whether that is going to be reasonable. If you put four down -- you certainly know more --
DR BUSBY: I will put them in order of importance, shall I? There we are.
MS BUSBY: Some of them it is only --
MR JUSTICE BLAKE: Well --
DR BUSBY: Some are very short.
MR JUSTICE BLAKE: Well, that's what I've indicated. Right. Do you have that?

Can you give that back to the witness, please.
(Handed)
So, in the course of this afternoon, we've had two papers that we've reserved over. If you are able to read those and see whether you can offer some assistance. As I say, if it's apparent that the subject matter of the paper, the epidemiological element is very limited or marginal and there is nothing you can say or very limited you can say, don't struggle on with the questions which you are not competent to answer.
If you can deal with the next most important ones. But there's a limit to what you are expected to do. Obviously, if you whizz through all of those, because there's nothing that you can comment upon, you might be able to look at something else. But I don't know what's in the paper, I don't know what is on the list and I am not expecting you to do anything which is unreasonable given the circumstances of the case. But, since you'll be our last witness, and you are coming to an end, if there is anything else you can assist us on, I would be grateful. Yes.
MR HEPPINSTALL: My Lord, the other matter I wanted to raise was the contractual relationship between the Government Legal Department and the stenography company. We have to give 24 hours notice to avoid charge, I am told.

I think the company has been stood down for Friday and Monday. I just wanted to make sure that that was a safe decision that had been --
MR JUSTICE BLAKE: Yes, I am not going to re-visit those.
MR HEPPINSTALL: Fine. So we are not sitting Friday, not sitting Monday.
MR JUSTICE BLAKE: Yes. The present proposal, assuming that the time limits I've just indicated to Dr Busby will enable you to complete re-examination before one o'clock comfortably and any other tidying up issues that we can deal with, we will terminate tomorrow round about the lunch and adjournment.
MR HEPPINSTALL: Yes.
MR JUSTICE BLAKE: We will come back on Tuesday. We will not be sitting on Thursday afternoon, Friday or Monday. I hope, however, that each of you will be thinking about how to present your final submissions.
MR HEPPINSTALL: Yes, my Lord.
MR JUSTICE BLAKE: And that, giving you some time, will facilitate the presentation for final submissions. We may also have some discussion that what, in a world -- what we consider would be helpful to us -- but I'll reserve that until, perhaps, five to one or some other such time tomorrow.
MR HEPPINSTALL: I am grateful, my Lord.

MR JUSTICE BLAKE: It may be that what I would like is not going to be what I am going to get. No doubt there is a graph of expectations versus satisfaction.

Is that clear?

MR HEPPINSTALL: Very clear, my Lord, thank you.

MR JUSTICE BLAKE: Right.

Thank you, see you tomorrow at ten o'clock. You have an indication of how long you are going to go for, and I hope you are able to work through that material without ruining your evening. Or some of it, at least.

Yes.

(4.35 pm)

(The court adjourned until Thursday, 23 June 2016 at 10.00 am)

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