IN THE HIGH COURT OF JUSTICE

1990 R No 860
1989 H No 3689

QUEEN'S BENCH DIVISION

ROYAL COURTS OF JUSTICE
THE STRAND
LONDON

Monday 30th November 1992

Before

THE HON. MR JUSTICE FRENCH

ELIZABETH REAY

Suing on her own behalf and as
Mother and Administratrix of the
Estate of DOROTHY REAY (deceased)
and as Widow and Administratrix of the Estate
of GEORGE REAY (deceased) (Plaintiff)

V.

BRITISH NUCLEAR FUELS plc (Defendants)

AND

VIVIEN JANE HOPE (Plaintiff)

V.

BRITISH NUCLEAR FUELS plc (Defendants)

APPEARANCES:

For the Plaintiffs: MR B A HYTNER QC
MR B F J LANGSTAFF
MR G S READ and MISS T GILL
(Instructed by Messrs Leigh, Day & Co. Solicitors, London)

For the Defendants: MR K S ROKISON QC
MR M G SPENCER QC
and MR C J BUTCHER
(Instructed by Messrs Freshfields, Solicitors, London)

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FIFTEENTH DAY'S PROCEEDINGS

MONDAY, 30TH NOVEMBER, 1992

STEPHEN JAMES EVANS Recalled:

Cross-examined by MR. ROKISON (Cont.):

MR. ROKISON: Good morning, Prof. Evans.

THE WITNESS: Good morning.

Q. MR. ROKISON: Prof. Evans, you will recall, no doubt, that on Friday afternoon I had asked you if you could do some homework over the weekend, for which I apologised then and I apologise again now, but have you managed to do that?
A. I have.

Q. Could you let us have the result of your study, please?
A. Yes, I have labelled it Fifth Report, although I am entirely unfamiliar with the processes of the law, but there is a copy for the Judge and two copies per side, as you might say.

Q. That is very helpful. Thank you very much.
A. I have labelled them for you, but I do not know quite what the legal status is, as you might say, of such a report. Very clearly, neither side has seen it and I have not discussed it with anyone.

MR. JUSTICE FRENCH: We will simply call it Evans No. 5 for the moment, if you do not mind.

MR. HYTNER: May I just say this, my Lord? Mr. Rokison has indicated he does not propose to be cross-examining Prof. Evans on this report at the moment and, my Lord, we are content to leave it for the moment. My Lord, I should say this, and it is only fair that I should, that I have now reviewed the correspondence, I have reviewed the transcript of Friday afternoon. My Lord, at an appropriate stage this matter will have to be dealt with. My Lord, we have regarded it as inappropriate to interrupt Mr. Rokison's cross-examination to deal with it but, my Lord, it is simply that it should be known that it is a point that will not go away and will have to be dealt with at a later stage.

MR. JUSTICE FRENCH: Yes, I do not for the moment see what has to be dealt with but then I shall learn in due course.

MR. HYTNER: Yes.

MR. ROKISON: Sorry, your Lordship is obviously far quicker than I am this morning. I was not quite sure what it is that my learned friend was saying would have to be dealt with.

MR. JUSTICE FRENCH: Nor am I. Indeed, I was saying I shall learn in due course what it is that has to be dealt with.

MR. ROKISON: Oh, I see.

MR. HYTNER: My Lord, if Mr. Rokison does not know, I shall indicate. It is not a mystery. Questions were put to Prof. Evans relating to the reasons why he has not done calculations. A discussion took place relating to an agreement that had been made and a statement, certainly an indication, was given by Mr. Rokison as to what the Defendants understood. My Lord, as I say, the matter is there in correspondence and, my Lord, we will wish to comment upon it.

MR. JUSTICE FRENCH: Yes, if it needs exploring, it will relate to some conversation which bears upon the homework of Prof. Evans.

MR. HYTNER: And correspondence.

MR. ROKISON: I see, yes. I now understand what the point was. It is a question of the conversations between Prof. Evans and Dr. Wakeford, as I understand it.

MR. HYTNER: And correspondence.

MR. ROKISON: And correspondence relating to it.

MR. JUSTICE FRENCH: It may turn out to be very important, but we will leave that point aside.

MR. ROKISON: I think it probably will not, but we shall see.

MR. JUSTICE FRENCH: Perhaps I should have said it may or may not turn out to be very important.

MR. ROKISON: No doubt, in this case, my Lord, further correspondence will follow, but for the moment may I just say thank you for having done this. Obviously, just glancing at it, there is some explanatory material as well as the tables produced and obviously I will want to look at it with those advising me before I ask you questions about it.

THE WITNESS: I understand.

MR. ROKISON: Thank you very much.

THE WITNESS: Am I allowed to comment that I also produced the graphs that his Lordship requested, but I have not got copies of those yet. They were done this morning, but they are for teaching purposes, as you might say.

Q. MR. JUSTICE FRENCH: Yes. Perhaps if you kindly hand that down, copies will be made. I can then look at it for my own education and anybody else can make of it what they wish to make.

A. It is not very well labelled so far and it is only labelled by hand on the overhead, in case you wish me to do that. I have got paper copies which I can get relabelled and, if it is not a matter for today, I can produce a better copy a little later, just as you wish, my Lord.

MR. JUSTICE FRENCH: I suspect Prof. Evans will be in the witness box longer than the rest of today.

MR. ROKISON: Oh, yes.

MR. JUSTICE FRENCH: Then there is no urgency in the matter, thank you, Professor.

What I am going to do with what I call Evans No. 5 is to put it in the back of my folder of Prof. Evans' reports.

MR. ROKISON: My Lord, yes, I have done the same.

THE WITNESS: Each page is labelled Evans 5.

MR. JUSTICE FRENCH: Is it? Good, thank you. On we go then.

MR. ROKISON: Thank you:

Q. Prof. Evans, I leave that to one side for the moment and come back, if I may, to ask you some questions about your first report. Do you have a copy of that in front of you?

A. I do.

Q. And, on page 3, where you start, in true scientific style you start with your conclusions or, at least, a summary of your conclusions, which I will come back to at various stages, but, as you say, your work for the purposes of this case has concerned considering epidemiological work in the United Kingdom concerning the possible association between child leukaemia and NHL and nuclear plants, especially Sellafield. It appears from page 4 of your report and, indeed, from your evidence that you are now a medical statistician. Is that an appropriate description of your work?

A. That would be correct, yes.

Q. And, as we saw from your impressive curriculum vitae, you actually took your degree in physics, chemistry and maths. Is that right?
A. That is correct.

Q. And until 1973 you worked in computing and data analysis?
A. Yes.

Q. It was in 1977, I think, that you turned to the field of medical statistics?
A. Formally, yes.

Q. You had been doing some medical statistics before?
A. I had.

Q. I see, and, as a medical statistician, you started at the London School of Hygiene and Tropical Medicine and then moved to the London Hospital Medical School, where you now are when you are not in the witness box?
A. I was in the London Hospital Medical College before the MSC as well.

Q. Would I be right in saying that you are not an epidemiologist?
A. I would not normally describe myself as an epidemiologist, depending on the audience. I am in the Department called Epidemiology and Medical Statistics and so the border between them, I would lie somewhere between the extreme statistician and the extreme epidemiologist and I would regard myself as lying in that grey area, shall we say, between the pure medically qualified epidemiologist - and some people say you have to be medically qualified to be an epidemiologist. That is not everybody's view, of course. So I am certainly not medically qualified.

Q. No, I was going to ask you about that, but many epidemiologists are medically qualified?
A. Yes, possibly the majority.

Q. And some are not?
A. Some are not, no.

Q. But, as you say in your report, even if you would not describe yourself as a pure epidemiologist, you do work with epidemiologists?
A. Yes.

Q. As you say, your primary function is relating to - is it the design of epidemiological studies and statistical analysis?
A. Yes, I think that is fair.

Q. Normally, it would be for the epidemiologist to assess and interpret the results of a study and draw conclusions from it?
A. I think that is going too far.

Q. How would you like to qualify that?
A. Because I would think that, as a medical statistician, I would be very interested in determining the conclusions of the study.

Q. You would assist the epidemiologist?
A. I would assist the epidemiologist.

Q. By giving advice in relation to the statistical aspects?
A. Yes, but also the logic of the study as well.

Q. And the logic. Yes, I see. I want to go right to the back, if I may, of your first report to page 30 and paragraph 86 and to the very last sentence of your first report, when you say that:

> "On the basis of the findings of the Gardner paper, I consider it statistically likely that the fathers' occupational radiation exposure caused or contributed significantly to Vivien Hope developing NHL and Dorothy developing acute lymphatic leukaemia."

You reach that conclusion, it appears, firstly, primarily at least, on the basis of the Gardner study. Is that correct?
A. Very importantly, yes.

Q. You say here "on the basis of the findings of the Gardner paper"?
A. Yes, but obviously that comes after a large section of report that has covered a number of other issues.

Q. Oh, indeed, but in reaching your conclusion, at least you primarily reached that conclusion on the basis of the Gardner study?
A. I think I find it difficult to say "primarily". I do not know quite what you mean by that.

Q. Is this a sentence that you wish to qualify? As I read it, your concluding sentence is that your statistical conclusion is said to be based on the finding of the Gardner paper. Is that right or wrong?
A. I think that the statistical issue, very directly, is yes.

Q. You reach that conclusion as a statistician. You reach that conclusion statistically from that study?
A. Yes, if you read the first sentence, it is referring to the fact that they received large doses.

Q. Oh, indeed.
A. And I think that what I would say is that, given the conclusions of the Gardner paper and my re-analysis - sorry, I have to go back to June, but the analyses that had been done at that stage certainly - that the large dose that they had received meant that it was statistically relating to them most based on Gardner?
A. Yes.

Q. If we return to your summary at page 3 - and one problem is that summaries, because they are summaries, are not always wholly accurate - but the last sentence of your summary says this:

"Given the above...."

and we see what you have said above, where I have referred you to the first sentence, which is the work that you have done for your report. You then deal with the Gardner study. You say:

"It is unlikely that the excess of childhood leukaemia cases around Sellafield is a chance occurrence."

You then refer again to the Gardner study, it being of very high quality, which, on the basis of current knowledge, suggests the most plausible explanation, and then you say:

"Given the above and the 'high' radiation doses received by the Plaintiff's fathers in the course of their employment at Sellafield, I am of the opinion that radiation from the plant caused or materially contributed to the diseases of Dorothy Reay and Vivien Hope."

Is that, similarly, a conclusion which you express as a statistician, statistically from the Gardner study?

A. Yes.

Q. And you are not purporting, I think, in this report, in that summary or in your conclusion, to express opinions as an epidemiologist?

A. No.

Q. As opposed to as a statistician?

A. No, my wife is an epidemiologist and I would wish to draw a distinction obviously. She is medically qualified and I am not drawing on her knowledge directly, for example, in regard....

Q. No, and I do not think - we have not at least heard so far - that she is to be a witness in this case.

A. Definitely not.

Q. Although who knows!

A. Definitely not.

Q. At the bottom of page 4 of your report, Prof. Evans, you very fairly make it clear that you have not specialised in the field of radiation linked cancers. Have you been concerned in any studies relating to the cause or causes of cancers?

A. Yes.

Q. What are they?
A. I have been involved in some studies in oral cancer. I think you will find Reference 26 in my c.v. on page 43. I have been involved with spinal metastases in cancer, which involves breast and lung cancer, and that is a reference at 27. I do not think that I immediately come up with another one.

Q. I see. Thank you for clarifying that. You have not been concerned with any studies concerning leukaemia?
A. No.

Q. MR. JUSTICE FRENCH: What is a metastasis?
A. When you have a primary cancer, then it may spread and a secondary cancer can grow in various different spots, and that is metastasis. It is where....

Q. So a metastasis is a Greek word for secondary cancer?
A. Essentially.

Q. And you said metastases from spinal cancer?
A. Yes, particularly in lung cancer and breast cancer, patients tend to get a secondary cancer in their spine.

Q. So it is spinal cancers as metastases for breast and lung?
A. That is right. Breast, lung and other cancers, but primarily breast and lung.

Q. MR. ROKISON: You told my Lord that you had not been concerned with any studies relating to leukaemias and you have not been concerned with studies concerned with the effects of radiation?
A. No.

Q. You are obviously aware, Prof. Evans, that there are epidemiologists in the United Kingdom and elsewhere in the world who have specialised for many years in the studies of the cause or causes of cancers and, in particular, leukaemias and possible links with radiation?
A. I am aware.

Q. Although you say - one might say, "Well, he would say that, wouldn't he" - although you say, in paragraph 6, that you do not believe that your lack of relevant experience is a disadvantage, do you not consider that, in casting a critical eye over studies that have been done, that your lack of experience in this field would place you at a disadvantage as compared with such experts as I have referred to, at least so far as drawing conclusions as to causation is concerned?
A. I think that there are some disadvantages, but there are also advantages, and that is why, for example, the British Medical Journal has had a statistician on their so-called "hanging committee" for a long time and, as you are, no doubt, aware, Dr. MacRae has been acting in place of Prof. Gardner because of his illness, and I have also acted there. So I think that a medical statistician -

and I have been on the editorial board of a variety of journals and I have refereed many articles involving cancer. So I am asked to provide a critical eye as part of my professional work on that, even though I am not a specialist in the field, and one would look for an opinion elsewhere in addition to mine.

Q. Yes, I did not mean in any way to denigrate your function at all, but you would be looking at such studies critically from the statistician's viewpoint?
A. Yes.

Q. MR. JUSTICE FRENCH: Can I just insert a question? It may be that there was a piece of shorthand capable of misunderstanding. When you referred to "hanging committee", do you mean a committee to decide whether a paper was to be published or not?
A. Exactly.

Q. MR. ROKISON: Your contribution, which, of course, would be a valuable contribution, would be to look at such papers critically, as you say, from a statistician's viewpoint and to consider whether, from that viewpoint, the study had been well designed and executed. Would that be right?
A. And whether the conclusions were appropriate to the design and the analysis.

Q. Statistically?
A. I think that I cannot draw a demarcation dispute there because I do not only deal with the numbers and, if you would like to read some of my referee's reports, I very often stray beyond the bounds of strict numbers, as you might say, because that is part of one's function to do so, to try and be more generally critical as a scientist.

Q. Yes, I see, and that is what you have tried to do in this report?
A. This is what I tried to do with, very clearly, a non-medical background and a statistical perspective.

Q. Can we come to page 5 of your report, where, in paragraph 7, you set out the purpose of your report - to review the epidemiological work, which you there describe, so as to be able to make an assessment of whether radiation was implicated as a cause, and you say that that is based on applying your own experience to a critical reading of the literature?
A. Yes.

Q. Is this an area which you had read into before you applied yourself to it for the purposes of this litigation?
A. No.

Q. And, as you say, you undertook a computer search so that all the relevant literature could be produced to you?
A. Yes. When you say "all", the computer does not find all.

Q. No. Would I be right in thinking that you have read all the UK reports which are referenced in your report?
A. Yes.

Q. So far as the non-UK reports are concerned, have you read any of those reports?
A. Yes.

Q. Which ones have you read?
A. I have read, for example, a paper in the New England Journal of Medicine on leukaemia following radiotherapy. I have read the reports themselves of people like Scott Davis and Ken Kopecky.

Q. Yes.
A. I have read reports that have appeared, on the whole, in the Lancet or the British Medical Journal which refer to work done elsewhere in Europe and that sort of thing. Some of the stuff that came out from Germany following Chernobyl, that sort of thing.

Q. Yes, so it is basically UK published work, the reports of Dr. Scott Davis and Dr. Kopecky and the New England study of radiotherapy?
A. Yes, I tend to try and read the New England Journal of Medicine as well in general, so I will....

Q. You may have picked up something there?
A. I will have picked things up along the way.

Q. Yes, I see, but it is not a matter on which you specifically comment in your reports?
A. No.

Q. And I take it that it would be more appropriate if I question Dr. Scott Davis or Dr. Kopecky about the....?
A. I have no doubt whatsoever.

Q. Tell me, have you read any of the studies concerning the follow-up of the survivors of the atomic bombs in Hiroshima and Nagasaki?
A. I have read some of the reports, but not in great detail.

Q. Was that for the purposes of this case or do you just happen to have come across them and glanced at them in the past?
A. I have largely done it for the purpose of this case. I would not have remembered in any detail anything prior to this case.

Q. I see. May I come back to your summary on page 3 and, having referred to the Gardner report in the second sentence, you reach a conclusion that it is unlikely that the excess of childhood leukaemia cases around Sellafield is a chance occurrence. Was that a deliberate omission of NHL or was it an accidental omission of NHL?
A. I would regard it as an accidental one.

Q. Oh! What is the basis for your conclusion that there was or is an excess of NHL around Sellafield?
A. My conclusion would be that, combining the data, as Draper did in his OPCS study, that he finds such an excess when they are combined.

Q. I see. So it is only when combined, so far as you are aware?
A. Absolutely.

Q. Thank you. Again, and the trouble with cross-examination is that my function is to be critical, and perhaps hyper-critical, and I apologise for that and I hope you will not take it personally, but where again in that sentence you refer to the "excess around Sellafield", would it be more correct to say "the excess in Seascale"?
A. No, I think I would mean that the boundary that one would draw seems to be a geographical one that is not simply Seascale.

Q. You say the boundary you would draw seems to be a geographical one which is not simply Seascale?
A. When you say "Seascale", that has boundaries that are drawn by Local Authorities and obviously, as a relatively small geographical area, my view is that there is an excess that seems to extend a little beyond that.

Q. We will have to look at the studies, of course, in some detail, but what is the basis for your conclusion that the excess of leukaemia or leukaemia and NHL together extends beyond the village of Seascale?
A. Again I think I would want to turn to Draper's studies and I would agree that the evidence is strongest for Seascale, but that there appear to be excesses in the larger geographical area and that the excess among workers at the plant appears to be higher, and so to draw a geographical boundary can be a little difficult.

Q. Are you saying that the excess among workers in the plant who do not live in Seascale, that there is such an excess? Are you saying that?
A. If I look at all the workers together, there appears to be an excess.

Q. Where do you look at all the workers together? Where do you find the study which considers all the workers together?
A. I would suggest that the Gardner study itself has some evidence for workers as a whole rather than just Seascale, but I would agree that the evidence - and I have said there - that around Sellafield and, by that sense, Seascale is primarily where the excess for which there is evidence that it is not a chance occurrence is largely Seascale.

Q. Obviously, as I say, we will have to look at the studies to see to what extent it is restricted to Seascale, but a number of studies do emphasise, I think, and, indeed, it

is emphasised in the Black report, that there appears to be an excess in Seascale. There does not appear to be an excess in Ennerdale, Whitehaven. The excess in Millom appears to be wholly driven by Seascale. It is perhaps an obvious question to ask, but did you ask yourself, in reaching your conclusions, why it should be restricted to Seascale or certainly, on your evidence, is primarily centred at Seascale? Is that a question that you considered?

A. Yes, it is.

Q. Where do we find in your report a discussion of that question?

A. I think that the answers can largely be only speculative and I have attempted to avoid speculation

Q. So is the answer that you do not pose or discuss that interesting question in your report?

A. I do not pose that question, no.

Q. Do you not think that, before reaching a conclusion in relation to causation, that it might have been sensible to have considered that question?

A. I think that the best data that we have in regard to causation is the Gardner case control study and that did not come to the conclusion that Seascale was the cause of the excess because within Seascale itself the only cases occurred to parents, fathers, who had been irradiated.

Q. Indeed, but the corollary is that one asks the question, what about all the children of irradiated fathers who did not live in Seascale?

A. That Gardner did not have a look at in great detail.

Q. No, he did not and that you did not consider either in reaching your conclusion, did you?

A. To my knowledge, there was no data available and I believe that Dr. Wakeford's study potentially has that data available, but he has not presented it either.

Q. We will come to that much later in your cross-examination. I will ask you about your comment that he has not presented the data, but it is fair to say that it is a question you asked yourself. It is a question which you did not discuss, and it is a question which, in reaching your conclusion, you really ignore. Would that be fair?

A. I think that the Gardner study, to some degree, does address the question and, insofar as Gardner concludes that Seascale is not the cause, then it is not an issue of ignoring it.

Q. Again we will have to look when we look at Prof. Gardner's report, but I would suggest to you that Prof. Gardner, perhaps prudently, does not express any opinion as to what was or was not the cause?

A. No, he suggests a number of possible causes.

Q. And I would suggest to you that Prof. Gardner does not say, either in terms or implicitly, that Seascale itself is not the or a cause or does not contribute the or a cause?
A. Do you mean living in Seascale or do you mean Seascale as a name or Seascale as a place?

Q. I mean living in or being born in or being conceived in Seascale?
A. Undoubtedly, Gardner suggests that being born in Seascale is strongly associated with the risk of leukaemia, whereas living in Seascale, in terms of the children there, he demonstrated from his schools cohort was much less likely to be associated.

Q. Indeed.
A. So I think that he does have something to say, when you put those three studies together, about Seascale.

Q. MR. JUSTICE FRENCH: The three studies, for the avoidance of doubt, are principally the Gardner report, 1990, but introducing as background and perhaps confirmation or whatever you like to call it two of the four earlier studies, namely the two cohort studies?
A. The two Gardner cohort studies, yes.

Q. MR. ROKISON: Yes, the schools cohort and the birth cohort studies?
A. Exactly.

Q. Which again I shall ask you to look at. Again looking perhaps hyper-critically at your summary - I will ask you about the quality of the Gardner study later on, but you say, "on the basis of current knowledge, suggests the most plausible explanation for the excess." May I ask you, when you are talking about "current knowledge", were you referring there to other epidemiological studies?
A. Yes.

Q. You were not there considering the question of biological plausibility or anything of that kind?
A. When you say anything of that kind....

Q. I do not know. Biological plausibility, genetics and so on?
A. Those were not the highest considerations in my mind at that point, no.

Q. MR. JUSTICE FRENCH: But were you excluding them?
A. No.

Q. MR. ROKISON: Is it something you took into account?
A. Yes.

Q. Did you consider yourself qualified to take that into account?

A. Yes, because I think that, while I accept I am not at all convinced they should have made me a professor at all, but that is for other people to judge, I think that we know very well that leukaemia is caused by radiation. We have a reasonable view that leukaemia might be caused by benzene and, beyond that, we do not know absolutely and certainly that leukaemia is caused by any other mechanism in human beings. We have various speculations, but we know about radiation. We are convinced. The scientific community as a whole is, and it is almost convinced on the issue of benzene and leukaemia, though the evidence there is rather weaker. So, in that sense, I took into account something of biological plausibility, but I would agree that I am not an expert in the genetics and the detail.

Q. As far as the link between radiation and leukaemia is concerned, it is something which is a link which is acknowledged somatically, is that right?
A. Yes.

Q. You are not aware of any similar conclusions drawn by the scientific community in relation to NHL?
A. I cannot give you the original reference, but my recollection is that Draper suggests that there might be.

Q. MR. JUSTICE FRENCH: Could you pause there, please? Draper suggests that NHL might be somatically linked?
A. Yes.

Q. Again please pause. For the avoidance of doubt, is the word "somatic" used at least in this context in distinction to genetic link?
A. Yes. My understanding of it would be that radiation on a a particular individual is known to cause leukaemia in that individual.

Q. That being so, I hope all of us can agree that unless we use "somatic" in some different sense, that is the sense in which it will be used? Is that acceptable?

MR. LANGSTAFF: My Lord, yes indeed.

MR. ROKISON: That was certainly the sense in which I was using it, my Lord.

MR. LANGSTAFF: My Lord, if I may just interpose, I believe the word "genetic", one sees in the reports, is used in two different senses. In some senses the word "genetic" is taken to mean inherited; in other contexts it is taken to mean a change in the genes, which then leads to leukaemia.

MR. JUSTICE FRENCH: Perhaps I am painting with too broad a brush - I do not know - but it seems to me that the word "genetic" is apt to cover them both, and one is by a defective gene and the other is by a mutated gene.

MR. LANGSTAFF: My Lord, yes.

MR. JUSTICE FRENCH: I see Mr. Rokison does not agree.

MR. LANGSTAFF: My Lord, I think the difference is really the inheriting mechanism on the one hand and the simple change, whether it be a mutation or a defect in the gene, on the other hand, which may have a link - I put it no higher than that - with the somatic expression of leukaemia.

MR. JUSTICE FRENCH: Have I misunderstood this: that both forms of what I have been calling genetic link refer to leukaemia part of whose mechanism is a defective gene, and that may be defective either because of a defect in the parental gonads which produce it or a mutation caused by radiation, benzene, whatever.

MR. LANGSTAFF: My Lord, I think that is right. I think one finds, for instance, anticipating the evidence of the Defendants' Prof. Evans much later in the case, that there is an issue between some of the genetic experts as to the degree to which certain forms of damage apparent within the genes at the time of the expression of leukaemia is caused somatically or is inherited genetically.

MR. JUSTICE FRENCH: I think it is Dr. Smith, our background expert, who defines somatic effects as "the effects of radiation on the body of the person or animal exposed", as opposed to genetic effects. So it looks as though I have at least some authority for drawing that broad distinction.

MR. LANGSTAFF: My Lord, I entirely accept that the different experts appear to be using the word "genetic" in the two different senses, and I thought it useful to mention that at this stage so that one does not ---

MR. JUSTICE FRENCH: As long as we all understand that under the label "genetic" there comes mutation by irradiation and mutation by inherent causes, we shall not get into trouble. Is that right?

MR. LANGSTAFF: I think that is right, my Lord.

MR. JUSTICE FRENCH: Do you agree, Mr. Rokison?

MR. ROKISON: I do, my Lord. I am afraid I had adopted a rather more simple approach, contrasting somatic and genetic as Dr. Hilton-Smith had. I do not quite understand the difference between something that is inherited and something that arises from a mutation or damage in the germ line, but no doubt it is something which will be explored at a later stage in the case.

MR. JUSTICE FRENCH: I know you are an expert in the criminal law, Mr. Rokison, where we have to deal quite frequently with inherent causes of various forms of mental malady.

MR. ROKISON: Yes, my Lord. I do not think that continuing the debate is going to help your Lordship.

MR. JUSTICE FRENCH: No, but we do understand that somatic means direct?

MR. ROKISON: Certainly, my Lord.

Q. In making your statement in your summary that on the basis of current knowledge, the Gardner case suggested the most plausible explanation for the excess, did you consider the A-bomb studies and in particular the dose response relationship and the work of Little in which he had expressed the view that the dose response relationship produced by Gardner appeared to be inconsistent with the A-bomb data?

A. Yes, I did consider that. There is a great deal of difficulty with dose response relationships in regard to radiation.

Q. Is that a matter about which you speak from experience in saying that?

A. Yes. I think that one has to be aware that we use radiation to cure cancer as well as it being a cause, so radiotherapy, with which I am familiar to some degree, is something that can be used to treat cancerous cells so that the dose at some stages can indeed prevent the occurrence or cancer or kill off cancer cells.

Q. Which is it doing?

A. Well, it would be certainly appearing to kill cancer cells and is used to treat it. I wouldn't say "prevent". On the whole, we don't go round giving people radiotherapy as a preventative measure, but we certainly use it for treating people with cancer. So the whole range of doses which people can receive in regard to radiation has a rather strange shape to it, in that it turns over.

Q. I do not understand. Perhaps my Lord does; I do not. Perhaps you could explain that further. We use radiation in order to treat cancers because radiation is employed in order to kill cancer cells?

A. That's right.

Q. I think we all agree about that, but what has that got to do with dose response relationship?

A. Because the doses at which that is happening are rather different. It may well be - and it undoubtedly is - that radiation at some different dose causes those very cancers. So we have the same thing both causing a cancer and being used to treat it, so we would not expect to find something that caused it being any good in its

treatment. At very, very high doses of radiation, we find that cancer cells are killed. Now I would not regard myself as a great expert in this, but I have examined with radiobiologists in Membership of the Faculty of Royal College of Radiologists, and for Radiotherapy and Clinical Oncology, and certainly my understanding from them is that they would agree that when we talked in one of my earlier diagrams about the dose response curve being a straight line and going up and then eventually levelling out, it actually moves back down again in some senses.

Q. With respect, this is not a matter in relation to which you have any personal expertise? This is something that you have learned in your conversations with others with whom you work, is that right?
A. Yes.

Q. When you were considering the relationship and the possible inconsistency between the Gardner conclusions or the Gardner hypothesis and the A-bomb data, how did you apply that acquired knowledge to that question?
A. Because the A-bomb data is very dependent upon assuming a particular form and shape for the dose response curve, and the amount of human data that we have in relation to that is almost entirely restricted to the A-bomb data.

Q. I think that you yourself in your reports, in considering dose response for the purpose of the Gardner study and the follow-up to the Gardner study, have expressed the view that a linear dose response relationship best fits the biological data, is that right?
A. Yes, and again you will notice that I say that that is on the basis of advice received from others.

Q. Yes, quite.
A. I would have to agree that I am not an expert on that.

Q. It is simply this: what I do not quite understand still - and it may be that I am being very slow and I apologise if I am - is how you were able to reconcile the Gardner hypothesis with the A-bomb data in the light of, as you say, the work of Little and others which you considered which suggested that they were inconsistent?
A. I think I have made clear in one of my paragraphs my view on that in particular, that the difficulty is - and I think I make it in regard to my comments on the Black Report, in paragraph 21 - that it is dependent on knowing what the doses really were to the individuals involved at Sellafield and also knowing the shape of the dose response curve, in other words what I have referred to there as the model, and I think that these are subject to a great deal of uncertainty.

Q. Surely what you are talking about there is modelling in relation to environmental dosimetry, is it not?
A. Not necessarily. The NRPB made an assessment of radiation doses to children, and you are assuming that

the model that one has for that is related to the model that one has for Little and the work following up the children of those who were exposed in the Atom Bomb.

Q. I simply do not understand that answer. Look at page 10 of your report. We will come to it in due course. You say, in relation to conclusion c, being one of the significant conclusions arising out of the Black report:

> "The radioactive emissions from Sellafield into the environment were too low to account for the size of the leukaemia excess observed in the village. This was based on the NRPB's assessment of the radiation doses to the children of Seascale, assuming that the data from other studies, particularly the atomic bomb data, could be used to estimate the rate of leukaemia for a given dose".

Then you say that you consider that conclusion is weak. The point, as I understand it, that you are making there is that modelling may well not accurately assess the dose to the environment. Is that not right?

A. No, I am saying that modelling may not accurately assess the effect on individuals, because the model is that you use as a predictor the radiation dose that an individual may receive and, as your outcome, their getting a cancer or a leukaemia.

Q. With respect, this is simply not what you say. Let us read this paragraph together:

> "I consider that the last conclusion is very weak. All models are prone to error and this places too much reliance on the NRPB model and assumes doses are known within relatively narrow limits. The Black Committee make the point that there are 'unavoidable uncertainties on dose in this situation'. From my own limited knowledge, it seems to me that the true level of radioactive emissions will never be known precisely, and any error will nearly always lead to an under-estimate, since by their nature having measured a given amount of radiation, we know the level is as high as that reported and the unknown can only increase the levels. In this case, the reported levels of radioactive emissions discussed in the Black report were rapidly discovered to be wrong and had to be increased."

What you are dealing with there is simply saying that you think the conclusion is weak because the environmental emissions and therefore the radioactivity in the environment will never be known for certain and depends on modelling which may be inaccurate. That is the point that you are making there, is it not?

A. I don't know whether you understand what I mean by the model that we are referring to as a whole. There are two things: one is to know what the doses that individuals

have; the other is that by "model" there, I mean the relationship, such as we have discussed in regard to the Gardner study of additive or multiplicative model, between dose and leukaemia risk, so the first sentence of c. is talking about the relationship between radioactive emissions from Sellafield and the size of the leukaemia excess. So the model not only involves the radioactive emission but also the equation, and Dr. Little also has some series of equations in there. The assumption that the situation with the atomic bomb is generalise-able to that in Sellafield, to me, is not a very good scientific viewpoint; they are not comparable.

Q. So having had no experience in relation to leukaemia, having had no experience in relation to the effects of radiation, you having read studies particularly by Dr. Little where there is a comparison made between the apparent dose response on the Gardner hypothesis and the a-bomb data, you conclude that he is wrong, is that right?

A. I don't recall going into his conclusions in great detail. However, he is making a series of assumptions in doing that, that are very clearly known and clearly expressed, and that is that the a-bomb data is a similar situation, is reliable, is something that we really know very well, and that the doses to the individuals themselves are really very well known. I do know from personal experience, from the time that I worked in the Atomic Energy Authority, that those of us who were interested in research very often took off our badges when we were doing something dodgy. I certainly went in and I know for a fact that I took off my radiation badge when I went into the electron linear accelerator because I knew I would be getting a dose, and we didn't want to frighten the health physics people by putting the dose up. This was something done by researchers commonly at that time, and other colleagues did the same thing, so I know that I did that. The point that I am making is that Little and Co. are assuming that the doses in the a-bomb data are well known and that the doses to the workers are really very well known. The logic also ---

Q. I do not want to argue the toss on this aspect of the case with you particularly. I was concerned whether it was something that you had borne in mind, taken into account?

A. I think that I did.

Q. The differences in the dose response when you compare the Gardner hypothesis with the a-bomb data are quite enormous, are they not?

A. They are considerable, yes.

Q. There is no question of the victims of the a-bomb, the survivors of the a-bomb, having removed any badges. You know that an enormous amount of research has been done over a large period of years in order to try and assess the doses as closely as possible, do you not?

A. I do, but I also know that, for example, in the a-bomb data the spectrum of radiation that they received was entirely different. I also know that they are Japanese and I know from my data on Japanese babies that they are quite different to British babies and quite different to Swedish babies.

Q. I see. These again are matters which you took into account?
A. All of these things you end up putting together in that sort of way and you form a judgment. I wouldn't wish my judgment alone to be the one on which it was based, and I would expect others to, but I am asked to form a judgment on it and I do so to the best of my rather poor ability.

Q. Yes. You were asked to express a general judgment and conclusion on causation, were you?
A. I was asked to say - and it was very clearly in regard to this case - did my assessment of the literature mean that it was likely that these particular individuals were affected, and I would say that my judgment was yes, whereas for certain other individuals I would say much less likely.

Q. Is your conclusion that you express in the last sentence of your summary one which you reach with confidence?

Q. MR. JUSTICE FRENCH: Where are we, page 3?

MR. ROKISON: Page 3, my Lord.
A. I understand that the position is that in some senses we, for the purpose of this litigation, need to say either that radiation caused or materially contributed to the disease of these people or it didn't, either one or the other.

Q. You say "<u>we</u> are required to say ..."?
A. I am sorry, his Lordship is required to say that.

Q. His Lordship at the end of the day has the burden of making a judgment on that issue, but it does not mean to say that every witness has to express a conclusion, especially if that conclusion involves areas of expertise which he does not happen to have?
A. I would entirely agree with that, but I would regard it from a scientific point of view that one wishes to have - and obviously I am straying again out of my own area of expertise - what I believe Scottish law would call "not proven". In scientific terms, in terms of the standard of proof that we require in science, I would say that the situation is not yet proven. We require a very, very high degree of proof in regard to that. However, I am of the opinion that radiation from the plant caused or materially contributed, and I was asked to give an opinion on that, and I think it is more likely than not that radiation contributed to the diseases of those two individuals in a way that it didn't to other individuals who were potential subjects of litigation.

Q. You did not think it would have been prudent for you to have said, "I am a medical statistician and reaching a conclusion on causation is something which involves a number of disciplines in which I do not have the necessary expertise, and therefore I would not think it appropriate to express a conclusion"? You did not consider that?

A. But most of the people who do express such views do not have expertise in the statistical analysis of their data, so they perhaps should not express opinions either. I think that we need to do this in a collaborative way. I would agree that I would not wish my opinion to be taken as the only one, and I think that on the medical bits of this my evidence is very clearly weak, I would agree with you. Nevertheless I think it is entirely within my purview to give an opinion generally, just as I am asked to give an opinion on a paper - should this be published or not? - and I do that very regularly.

Q. I see. In drawing a conclusion as to causation from a statistical association, there are recognised criteria which should be applied, are there not?

A. There are, yes. I teach my students that regularly.

Q. Although there have been variations on them, basically they are the Bradford/Hill criteria, are they not?

A. Yes. The Professor of Medical Statistics who was not medically qualified set out those criteria, yes.

Q. No doubt in reaching your conclusion you considered those criteria?

A. I did.

Q. Where do we find that discussed in your paper?

A. For example, I discuss in very particularly in regard to the dose response issue. I discuss very briefly issues of biological plausibility, and I think that that probably appears in one of my later reports specifically that the issue of biological plausibility is relatively limited. In paragraph 10 I discuss some of the biological background, and I would rely heavily on Sir Richard Doll's review that was restricted to leukaemia in children and obviously at that stage did not mention non-Hodgkin's lymphoma. So biological plausibility is part of the aspect of that. We would go through the idea that strength of association is one of them; we would go through the idea that specificity is one of the criteria, and so on. I think that Dr. MacRae has set out those criteria really very clearly.

Q. Certainly, but I was concerned about your report. Some of them you discuss as we go through the report. You do not consider those criteria in any sort of systematic way when moving from your ---

Q. MR. JUSTICE FRENCH: You do not tick them off with a checklist?

A. No, I do not.

Q. MR. ROKISON: You do not consider them when moving from your statistical association to causation?
A. No, and I think that given the experience which I now have of the law, which I didn't have when writing that report - this is my first occasion - I would have learned perhaps from Dr. MacRae's experience and I would have gone through with that kind of checklist approach. I can see that that would be helpful to the Court, whereas one wouldn't do that in discussion with one's scientific colleagues.

Q. I see. Did you read the report of Prof. MacMahon?
A. I did.

Q. Have you heard of Prof. MacMahon?
A. Undoubtedly.

Q. He is, is he not, an eminent epidemiologist?
A. He is.

Q. And was Professor of Epidemiology at Harvard University and Chairman of the Department of Epidemiology at the Harvard School of Public Health?
A. Yes.

Q. Can I ask you just to look at one passage in his report and comment upon it? You will find it in a bundle looking like this, I think. I was going to look at page 21.
A. Is this his first report?

Q. Yes, it is. This is within a passage which starts at page 14, Prof. Evans, where he is dealing with the assessment of causality?
A. Yes.

Q. He deals with a number of considerations, one should bear in mind, and he concludes at page 21 where he says this near the top:

> "The types of evidence that will be drawn on in considering the inferences from the material described in the six previous paragraphs comes from a wide range of disciplines. In evaluating the causal nature of an epidemiologic association it is important not only not to put sole reliance on any one study, as mentioned earlier, but also not to limit consideration to evidence from only one field. The totality of the evidence must be weighed with due regard to the strength of individual studies".

You would agree with that?
A. I would agree entirely.

Q. May I just put one further extract from a report now to see whether you agree with it and that is Dr. Scott Davis?
A. Do I need to go back to MacMahon?

Q. No, you do not. Page 17, paragraph 30, where he is dealing with the question of consistency, where he says this:

> "Before a causal link can be claimed with much confidence one would like to see an overall consistency among results from not only ecological studies but case-control and cohort studies as well."

Then he goes on to deal with biological plausibility. Would you agree with that?

A. I would.

Q. Could you tell my Lord which are the studies which you particularly rely on as showing consistency with the Gardner study and the Gardner hypothesis as developed in your re-analysis of his data?

A. You are asking me to do something as to where I stand now rather than...?

Q. Well, of course, you are giving your evidence now but I was just wondering which studies you rely upon as being consistent with Gardner so as to enable you to take this step from a statistical association to a conclusion on causality?

A. I think I would regard McKinney as one of them. I think I would regard the continuing existence of clusters around nuclear facilities that have been found, and clusters around places like Gateshead where one didn't realise, or it doesn't seem to be well described, that there has been exposure to radiation around there. I would take those as the main objects for the consistency.

Q. We will look at those in due course. Of course, the Gardner hypothesis postulates a possible link between paternal preconception irradiation and leukaemia in the offspring?

A. Yes.

Q. To what extent do the studies which suggest that there may be - may be - clusters around some nuclear facilities, to what extent are they supportive of that hypothesis without any reference whatever in any of those studies to paternal doses?

A. They are supportive in the sense that if the hypothesis is correct, then the pattern of data you see is consistent with that sort of hypothesis.

Q. Would it be right to say...

MR. JUSTICE FRENCH: Can I just record that?

Q. Supportive in that if the hypothesis re. parental radiation be correct, it is consistent with the cluster findings?

A. That is right.

Q. MR. ROKISON: Unless one knows, in relation to the cases of leukaemia which are found in any of those studies, what, if any, dose the relevant fathers may have had, one can draw no conclusions whatever, can one?
A. Clearly, if there is no data, one can draw no direct conclusions.

Q. Let's take an example if I may. I will come to it in detail, but let's take Burfield. Now it is suggested that there may be a cluster around Burfield, if one takes a 10 km circle radius from Burfield and thereby takes in the town of Reading, where it appears there is an excess beyond national average of child leukaemias. On one view of the matter that could be said to demonstrate a cluster around Burfield. On another view of the matter, it can show there is an excess in Reading. How does that, for example, give any support whatever to the Gardner hypothesis?
A. Once again I have to come back to saying if - and we do have to agree that we do not yet know - if the Gardner hypothesis is correct then it is quite likely that some of the parents who work at Burfield live in Reading and that it is not entirely unreasonable that if the hypothesis is correct you might see an excess of childhood leukaemia there.

Q. Of course. If the Gardner hypothesis is correct, then one needs to go no further for the purposes of this case. However, as I understand that what Dr. Scott Davis is saying, is in assessing whether there is any causal link you should not just look at one study, but that it is necessary to have a number of studies which demonstrate a consistent pattern. What I am asking you is, by way of example, how you can say that the fact there is an excess of child leukaemias in Reading which happens to be within 10 km of Burfield which has minuscule discharges of radionuclides, how that can possibly demonstrate a consistency with Gardner so that one can go from your statistical association to draw your conclusion as to causation?
A. First of all, of course, if there are no emissions from Burfield then it is indeed unlikely that environmental radiation might be a cause. It then means that it is certainly possible that paternal exposure could be a cause. I am not trying to say that there are a large number of studies, all of which are consistent with Gardner. I would entirely agree in the sense that they have all studied exactly what Gardner has studied and had the same findings.

Q. But there are none are there?
A. I think the McKinney study and the letter that followed it in the BMJ, which demonstrated something independent, showed that. I think I would entirely agree with you that the scientific community at large is not yet certain whether Gardner is correct or not, but I would go back and say that my opinion is it is more likely than not that this is so. We have not demonstrated it to the

exclusion of everything else, but then there have been almost no studies which have been done in exactly the circumstances as the Gardner study, which do not show an association. Again, when I talk about something being "consistent with", the hypothesis itself appears to me to be an explanation for what is happening at Seascale and appears to be consistent with a variety of other findings, none of which looked at paternal dose, but which it is possible may be explanation, where the environmental radiation on its own appears much less likely as an explanation.

Q. The only study that you place any reliance on, which has tested the Gardner hypothesis, and which on your evidence produces support for the Gardner hypothesis, is McKinney?
A. Of the UK studies.

Q. Well, which of the foreign studies?
A. Well, I am saying that. I am not an expert in the foreign studies. You would have to go to someone like Scott Davis for a better overview.

Q. However, if one asked you, assuming that a consistent study is a study which comes up with a confirmation of the same or a similar hypothesis, the only study you rely upon as being consistent with Gardner is McKinney?
A. Yes.

Q. Do you know Dr. Draper?
A. Only by repute.

Q. He is, of course, Director of the Childhood Cancer Research Group at Oxford?
A. Yes, a medical statistician at Oxford.

Q. Indeed, but a medical statistician who has devoted a great deal of his time and efforts to investigating causes of child cancer in general and leukaemia in particular?
A. Yes.

Q. You yourself refer to and, indeed, draw support from his latest draft paper?
A. Yes.

Q. I think you have it in a bundle called P.4 at page 30?
A. Yes.

Q. We looked at this on Friday of last week. One sees from the abstract to which you were referred, that the purpose of the exercise which he was undertaking and reporting on here, was effectively a reappraisal of the epidemiological findings of the Black Committee. He considered, amongst other things, the Gardner study, and he considered whether one could draw any conclusions as to causation?
A. Yes.

Q. His conclusion is at page 15 of the draft report. I am taking the numbering from the top left hand corner.
A. Page 45.

Q. Where he says this in the middle of the page:

> "In conclusion, having considered a number of hypotheses and discussed them, we confirm there is good evidence for an increased incidence of lymphoid leukaemia/NHL among young people in Seascale though we are unable to identify the cause of this increase nor can we say that the new data and analyses presented here either support or detract from the conclusions of Gardner et al."

A. Yes.

Q. That is a conclusion with which you would not agree?
A. No. I would agree with that conclusion.

Q. You would agree with it? Forgive me, and perhaps I am being very simple, but I thought that you were yourself drawing a conclusion identifying the cause of the increase and applying it to the two young people who are the subject matter of these proceedings?
A. In terms of scientific philosophy we can have two different approaches. One is to be almost entirely agnostic about causes and to attempt to be as cynical as possible about any possible finding. As medical statisticians that is what we are often requested to do. However, for the purposes of this litigation I think that a different philosophical stance is called for because as scientists we tend to say we will not believe in something until we have exceedingly strong evidence for it and until then we will say we don't know.

If we have gone and looked very, very hard for evidence that would demonstrate, and had consistently failed to find that, then eventually we will say there is evidence against. However, for a lot of the time we will sit in a grey area. I think this is what Draper is doing at this stage, whereas I was asked to say that if Gardner is, in some senses, true, what consequences does that have? One of the consequences is that you would expect to find the excess of lymphoid leukaemia and non-Hodgkin's lymphoma and Draper seems, for some reason, to combine them of course here again, just as I did...

Q. I don't think that is quite right but we will come back to that. It is a point you have made and I note you make it again.
A. He says, "...there is good evidence for an increased incidence..." and what he says is it neither supports nor detracts from. Nevertheless, he has not, in this study, looked at data on paternal exposures. In some senses he can't either support or detract from. However, if Gardner is true, then we would expect to find that the excess around Seascale should continue because paternal exposures and the activities of Sellafield have not ceased entirely.

Q. I was asking you first of all about his first of two conclusions. Well, he reaches three conclusions there, doesn't he, on analysis. First of all he confirms that the excess is there?
A. Yes.

Q. Nobody disputes that. Secondly, he concludes:

> "...we are unable to identify the cause of this increase..."

It was that I was asking you about particularly because that seems to me, and you will correct me if I am wrong, to be inconsistent with the conclusion which you reach at the end of your summary. As I understand it your explanation is that he is applying a different standard to you, is that right?
A. First of all, of course, he was not looking for causes. He has made no attempt in this study whatsoever to look at any possible causal factors.

Q. This simply isn't true. He starts on page 13 and he says:

> "We consider below some of the main hypotheses that might account for the findings presented here."

He considers environmental radiation. He considers the Gardner hypothesis specifically on page 14. He considers the McLaughlin study and he considers other suggestions such as the Kinlen suggestion of virus. He considers all those and his conclusion is that now or a matter of only a couple of weeks or so ago when this final draft was produced, that his conclusions - no doubt as much as he would like to - but he is unable to identify the cause of the increase?
A. Yes, but what I would go on and say is that all of that is in the discussion of his paper and he has not looked at any data on viruses. He has not looked at any data on paternal exposure to radiation. What he has done, and he has not looked at any date on effects of environmental radiation, what he has done is examine whether chance is a likely explanation. That was essentially the purpose of his paper...

Q. Well, I don't accept that...
A. The rest of his discussion does not relate to data he has collected.

Q. Indeed not, but what he does is, he follows up the Black Report by bringing up to date the incidence of various diseases in the area which was the subject of the Black Report. He then discusses the possible causes, including - if it is not a contradiction in terms - chance, but he discusses the Gardner hypothesis. He discusses environmental exposure. He discusses the viral theory. His conclusion is, after considering all those, his very last paragraph where he sets out his

conclusion before the acknowledgements, is a conclusion that one is not yet able to identify the cause of the increase. I suggest to you that if you accept that, as you said you did, and you agreed with it, it is inconsistent with the conclusion which you reach in your report, unless you are simply applying a different standard?

A. I am in some senses applying a different standard because I was asked to assess "Yes" or "No", and as far as I was concerned I was not offered the option of "I don't know."

Q. I see.

A. What Draper is saying is, "I do not know. We do not yet know." That, in a scientific sense, is true, we are not absolutely certain.

Q. If you had been given the option of "I don't know", would you have said, "I don't know"?

A. "I don't know" would be "No" in the sense of being certain would have been the option I would have taken.

Q. You didn't feel that in writing your report it was open to you to express your conclusion in that sort of way?

A. I think it could have been, but I think that my understanding of the process of the law, which I am sure is entirely fallible, was that I was asked to give an opinion one way or the other and my judgment, and it would be different for different scientists, obviously, on the same basis of the evidence, my judgment would come down in favour of Gardner being a good explanation for a number of findings.

MR. JUSTICE FRENCH: Can I just say something here, again, for the avoidance of doubt?

Professor, unless somebody persuades me, and I doubt if they will try, that this is the wrong view, I have to decide on the balance of probabilities. Now that balance may be a bare balance in some cases. It may be more distinct in other cases. However, it is the balance of probabilities. Forget, for the purpose of the decision I have to make, about beyond all reasonable doubt, forget about scientific proof, or such proof as would satisfy the scientific community, and bear in mind that it is the balance of probabilities.

Now perhaps you could approach Mr. Rokison's questions and your answers with that in mind. I hope that is helpful.

At some stage I would certainly wish to ask you the specific question: on the balance of probabilities, in your opinion which? However, I won't do it now.

Q. MR. ROKISON: While we are on it, you relied upon the Draper paper of 1992 as lending support to the Gardner hypothesis and your adoption of it?

A. In the sense that it removed, in my view, the possible explanation that chance, which you said nobody believed in chance a little while ago I seem to remember, but certainly some people believed that chance was the explanation for the Seascale excess.

Q. I said no such thing. All I said was that everyone acknowledges there is an excess.
A. Everyone acknowledges there is an excess - I'm sorry - but that chance as an explanation for that was then ruled out. If chance had been an explanation that was a reasonable explanation, then Gardner is entirely unnecessary, whereas I found that Draper has not entirely, but largely, ruled out chance as being the likely explanation.

Q. You agree, I think you said, that the new data and analyses which he presents in this paper do not either support or detract from his conclusions?
A. The data that he presents do not support or detract from his conclusions directly, no.

Q. May I just put one more... Well, perhaps I can leave it. I was going to put another document to you but I don't think it is necessary in view of your answers. Can we move on in your report?

THE WITNESS: May I ask or a short adjournment, my Lord?

MR. JUSTICE FRENCH: Yes, twenty past twelve.

(Short adjournment)

Q. MR. ROKISON: I am wondering whether you could have available Common Bundle B13 which is the Black Report? I just wanted to ask you about one observation in that report to see if you would agree with it. On page 34, paragraph 2.46, and just to put it in context, Prof. Evans, the Black Committee has been considering the various geographical studies reflecting the existence of the excess, or the cluster, and what Sir Douglas Black and the authors say at 2.46 is this:

"Most cases of child leukaemia are of unknown cause."

Pausing there, with all the qualifications, as we know, that you are not a doctor, but on your understanding that would be right?
A. Yes.

Q. "Therefore caution is necessary in interpreting the results described above. An observed association between two factors does not prove a causal relationship."

Would you agree with that?
A. I would indeed.

Q. If you could put that to one side for the moment, but I will be coming back in just a moment to Black, in paragraph 9 of your report, starting at the bottom of page 5, you refer to the various stages of studies which would be likely to carry out and over on page 6 you very helpfully set out the annual incidence rates for leukaemia in children under 15 and the rates for NHL. Although, again, it strays a little bit perhaps outside your field because you are not experienced, as you have said, in relation to leukaemia studies, you will have observed in a number of studies, particularly studies before Gardner, that the dividing line between child leukaemia and adult leukaemia is normally taken at the age of 15?
A. Yes. This is largely because of the Office of Population, Censuses and Surveys dividing their data on population at that age.

Q. I would suggest to you that is not the reason for that division; it may be a reason but it is not the predominant reason in relation to studies of leukaemia. For child leukaemia and cancer it is because there seems to be in observation a dividing line at about that age between certain sorts of cancers which develop in a certain way in children and other sorts of cancers which tend to develop later in life?
A. Yes. My understanding would be that 15 would not necessarily be exactly the best time for that one but given that OCPS produces their data that way it is nearly coincident with it. My understanding is that you might move it a little lower in age.

Q. Indeed. The point, in fact, is made in the Black Report, if you look, at paragraph 2.9 on page 12 where the authors of the Black Report are referring to the background to their report, and in particular the television programme and the researchers for the television programme having identified this apparent cluster?
A. Yes.

Q. At paragraph 2.9 Black says:

> "An exaggeration of the problem might have arisen in the way that the above data were used because the age group reported was defined by the ages of the discovered cases. This is exemplified also in the statement in paragraph 2.6 with the choice of the age of 18 years as the upper limit. A statistically sounder method is first to define the age range of interests (0-14 years of age is most commonly used for childhood cancer) and then to ascertain the number of cases which fall within this defined range."

and you would agree with that, that would be a sounder method of doing it?

A. Absolutely.

Q. I think you made the point in your evidence in chief, that one has to be somewhat cautious in interpreting the result of a study or studies which relate to a cluster which has been identified in advance of the study?

A. Exactly, yes.

Q. This is, I think, why you drew particular comfort from the 1992 Draper paper, because to some extent the Gardner Study had resulted indirectly via Black from the YTV programme and the cluster identified in that programme?

A. When you say, "I took comfort from", you say it as if I had some very strong prior hypothesis or vested interest in that being so. What I take it you mean is that it was in agreement with the overall opinion that I had.

Q. I cannot remember exactly how you expressed it, but you drew a contrast between the Draper paper, where Draper was looking to see whether the excess continued?

A. Yes.

Q. As opposed to the Gardner Study which because it had been spawned, if you like, via Black from the YTV programme, was investigating a cluster which was already identified and known to exist?

A. The difficulty that you are saying there is that what I would regard as dangerous is the YTV programme itself and the calculation of incidence rates, whereas Gardner was not attempting to calculated incidence rates and is a slightly different point, whereas Draper was calculating incidence rates, and if you define your boundaries of age and geography for your calculations of incidence rate, after you have found the cluster, then you are likely to mislead yourself, whereas in regard to Gardner he was not attempting to define incidence rates but having found the cluster to investigate causes, and that was something different. So I do not think that Gardner can be criticised. You are implying a criticism of Gardner by his judicious selection of ages which I do not think would be a fair criticism.

Q. I was not criticising Gardner in any way, I want to make that clear. I am not criticising the Gardner Study but simply saying, as indeed Black said, that it would be statistically sounder to define the parameters of your study without reference to a cluster which has already been identified.

A. In terms especially of investigating incidence rates, yes.

Q. But generally that would be so as well, would it not?

A. If that were to be taken to the extreme it would say to you, you must not investigate any clusters that are found.

Q. It is a question of which parameters you choose ....
A. Yes.

A

Q. And if you draw your parameters around your known cluster then one has to ....
A. Then the incidence rates so calculated will be misleading.

Q. And it may be that other results ought to be looked at with caution, especially if one is to apply them to people who are outside the scope of that cluster?

B

A. I am not quite sure what your question is there. Can you try and rephrase that for me?

Q. In this case, as Black pointed out, an age range which was taken which would not be perhaps the normal the age range which one would consider?
A. No.

C

Q. That age range was taken for the purposes of the Gardner Study ....
A. No, I am sorry, I do not think that is true.

Q. The Gardner Study took an age range which was such as to embrace those that were within the cluster, was it not?
A. Yes, but the Black Report itself chose other age ranges rather than that which was taken by the YTV programme and

D

I think that Gardner used age ranges defined by Black and not by the Yorkshire Television Company.

Q. Indeed that is right, but they took an age range which was wide enough in order to embrace the cluster which had been discovered, as indeed is set out in Table 2.1 on page 13?
A. That would be the only logical thing to do but not to use

E

the YTV's range and to believe in their incidence rates calculated from 0-18, they used a wider age range which would be more sensible.

Q. However, for the purpose of investigating possible causes, had one not had the YTV programme one would have been likely to look to, in respect of childhood leukaemias, 0-14, and adult leukaemias, 15 and above?

F

A. Not necessarily because Draper and others show that in some instances leukaemia is raised in certain areas in the age range 0-24 and it might be sensible to look over the age range 0-24.

Q. Could you help as to what it is you are referring to in that answer?

G

A. Sorry, not Draper. It is one of the Darby, Sarah Darby's studies and others, that looked at a wider age range. They looked at 0-24; they looked at cancers as a whole.

Q. You may be right. It is not something I have immediately to hand.
A. I obviously do not have it immediately to hand either. If you would like me to return to that I will find it

H

during lunchtime and find you the 0-24.

Q. Yes, thank you. The Black Committee make the same point at the end of paragraph 2.10 in relation to the other parameters, don't they?
A. Yes.

Q. "The same comments apply to similar selection of certain calendar years, disease categories and age ranges for study."
A. Absolutely, yes.

Q. The point really is this, that it is better, if I can use that rather general word, to draw the parameters of your study without reference to incidence that you know about?
A. Exactly right.

Q. It is also, and we will come on to this, having drawn the parameters of your study it is very important, is it not, to stick to those parameters?
A. Yes.

Q. May I go on to paragraph 10 of your report where you refer to a review by Sir Richard Doll? I asked you about Prof. MacMahon and would you agree that Sir Richard Doll is one of the leading epidemiologists in the world?
A. I would indeed.

Q. I take it you would respect his opinions on matters epidemiological?
A. I would hold them in the highest regard but that does not necessarily mean that everything that he says is necessarily true. If this is done on the eminence of the witnesses then I might as well leave [illegible] instantly.

Q. I won't ask you to do it - you have only just done that about 25 minutes ago!
A. For good this time! Sir Richard Doll is a very eminent man indeed.

Q. As you rightly say, and we have just looked at a passage in Black to that effect, we do not know very much about the disease of leukaemia. Perhaps I can ask you in this regard to just glance at the earlier Draper paper which you relied upon, which is D64, I think.
A. D 64 is an earlier draft of the paper we were just looking at.

Q. Would you bear with me a moment?
A. D63 is also by Draper.

MR. JUSTICE FRENCH: Can I put Sir Douglas Black away?

MR. ROKISON: My Lord, yes. My Lord, I have just looked at the reference that I was going to put to Prof. Evans and I am not going to bother, my Lord. I am sorry to have put your Lordship and the witness to the burden of looking at it but I think it is a point which is not really in issue:

Q. It is in this paragraph, I think, in paragraph 10 of your report, that you refer to, I think the only point in your report where you refer to, the follow-up to the atomic bomb survivors?
A. Yes.

Q. I think in the course of your evidence, when you were explaining to my Lord the differences between the various sorts of epidemiological studies, I think you said that a prospective cohort study is in theory the best of all but certain stances do not often arise where one can carry one out? Would that be a fair summary?
A. The best of all in the absence of an experiment.

Q. Yes, quite, and one of the advantages which did flow from the tragedies at Hiroshima and Nagasaki was the fact that one was able to establish a very substantial prospective cohort study?
A. Yes, but then the question really is, is it a cohort study of the relevant exposure? That cohort is no good for us in regard to heart disease.

Q. Indeed, I am not suggesting that it enabled one to study everything but one had two populations who were exposed to radiation in circumstances which could not be matched experimentally and that one was able to follow up what happened to that population and to the next generation?
A. Yes.

Q. It is probably the biggest cohort study which has ever been carried out, isn't it?
A. I am not sure on that. I have been involved in a cohort study of 17,000 children in Britain. Maybe the atomic bomb survivors was much larger, I cannot remember.

Q. Yes, it was over 70,000.
A. Over 70,000, it may well have been, certainly among the largest.

Q. You refer to, and there is no issue, that radiation can cause leukaemia somatically. You then say that there is evidence that radiotherapy used as a treatment for cancer may be associated with the development of second primary tumours, and that again would be a somatic effect of that radiation?
A. Yes.

Q. "The association with leukaemia is known in children who have been diagnosed as having cancer."

I am just not quite sure what you mean by that? It is just clarification that I seek in relation to that statement.
A. I think that the association with leukaemia is known in children who were treated for an early cancer, they have themselves had a slightly increased risk of getting leukaemia subsequently, that is what I think I meant. I agree it is not very clearly expressed. It is just

reiterating the same point. It refers in both instances to somatic exposure.

A Q. Yes. You say in relation to Sir Richard Doll's review, that it was written before the Gardner Study and therefore he does not discuss the link between paternal radiation exposure?
A. Yes.

Q. Does it follow from that, and it may follow from answers that you have given already, that you are not aware that
B before the Gardner Report and the Gardner hypothesis, that the link between, or the possible link between leukaemia and paternal radiation exposure was something which was generally recognised as being a possible link?
A. It certainly occurred to the Black Committee as a whole and it had occurred to me prior to Gardner being published, so I do not quite understand what the question is. It is certainly not generally recognised then as
C being definitely so but as a possibility it was known prior to Black, but Doll did not mention it.

Q. It was described by Prof. Greaves, who you will have heard of?
A. Yes.

Q. And probably know?
D A. No, I do not.

Q. Prof. Greaves, when referring to the Gardner Study, referred to it as being a remarkable result. Would you agree with that?
A. Yes. Do you mean that he said it, or do I agree that it was a remarkable result?

E Q. No, you can accept if I tell you that he said it or wrote it, that that is the case.
A. Do I agree that it was a remarkable result? No, I do not agree that it was a remarkable result in the sense that it was one of the possible explanations.

Q. What he also said, in the same document as this, was that
F occupational exposures may not be the whole story, since there is no obvious reason why workers exposed to higher levels of radiation at the plant should live close to the plant in Seascale Village, and in fact most workers at the plant do not, and proximity to the plant or living in Seascale might itself be a risk factor. He goes on:

> "The association with living close to Sellafield has
G not been explained but provides at least a hint of additional exposure of either the father, mother or child."

Do you agree with that?
A. No.

H Q. In what respect would you disagree?

A. I think one might almost be forgiven for saying that paternal radiation was not the explanation if you look in Gardner's 1990 paper, when all but one of the Seascale leukaemias had paternal exposure. We now know that all of them did and that in itself is a surprising finding in some senses, and the fact that in Seascale none of the excesses to children whose parents were unexposed, but all of the excess, indeed all of the cases as far as I can tell, have been to a father who was exposed, and the diagram that Gardner gives, where there was an unlinked case, now has that case linked and having a substantial dose, and higher than the controls.

Q. But the point which Prof. Greaves is making is not so much saying those who were in the village whose children had leukaemia had high doses, but the point that there was no obvious reason why those who were exposed to higher levels of radiation should live in Seascale, and if as he states they did not then one has got to look at some other factor in order to explain why the excess was in Seascale. Do you follow?

A. I do. I think that all of those things are speculation. If you wish to look at a reason, as far as I understand a lot of the buildings built in Seascale were for young research workers of slightly higher social class than others, and who might be inclined to do as I was and that is take their badges off before going into nasty hotspots of radiation. When you are enthusiastic and your belief is in research you have a slightly cavalier attitude towards your future health. It could be that is an explanation. It is not an explanation for which I have any evidence but you asked me for an explanation.

Q. I see, but you agree that one has to find an explanation?

A. I agree that it would be a reasonable thing to look for an explanation, yes.

Q. It is not just reasonable to look for it, unless one has a concentration of high dose fathers in Seascale then one has to look for an explanation for the Seascale cluster other than, or beyond paternal occupational dose?

A. It could be that their paternal occupational dose was measured in a biased way, as I have implied or it could be an interaction of something else with Seascale, it could be, as I am sure Prof. Greaves would believe, an interaction with a virus. It could be an interaction with environmental dose. All of those things are speculation; all of them are possibilities and I do not know by any means which of them is the correct explanation. I would agree that it would be reasonable to look for such a thing.

Q. One has to find something else, either operating in synergism with the parental exposure or operating in synergism with something else other than the paternal exposure, or perhaps operating on its own?

A. You say you have to. I am not aware of any studies that have had the power to detect importantly raised risks

which have been carried out anywhere to be able to follow them up. I do not agree that the atomic bomb data is similar but one could carry out an entire case control study of all the employees - or indeed a cohort study - of all the employees of the Atomic Energy Authority and follow up their children. To my knowledge that has not yet been done so we have not yet got the data on that. So to say that you require that there is something extra in Seascale I do not agree with. I think that it could well be that there is something extra in Seascale, I do not know, but it does not require it.

Q. You say it does not require it but if you have an excess in Seascale and if the Seascale workers do not have a monopoly or anything like a monopoly of the high doses then in order to explain why you have your excess in Seascale and not elsewhere where the high dose workers live, you must, must you not, find some cause or additional cause peculiar to Seascale?

A. You have said that there is no excess in workers elsewhere and the data on that are limited.

Q. We will come to that in due course, but if it be the case that Seascale does not have a monopoly or indeed the majority of the high dose workers living there, if you only have your excess in Seascale, you would agree on that hypothesis that you have to find some other explanation for the Seascale excess?

A. I think that you are pushing the data on the dose distribution and you are clearly referring to the Parker et al study. I think you are pushing that beyond its reasonable conclusion, and I don't agree.

Q. If the hypothesis I was putting to you were correct ---

A. If we have a study which demonstrates that overall among large numbers of people who are exposed to, let us say, 100 mSv radiation or more occupationally, and if we have studies done elsewhere that demonstrate no risk, then indeed what you are saying is entirely true and the explanation must lie in something special about Seascale.

Q. We can look at a later stage to see to what extent there is a disagreement between us as to whether that information exists.

A. Right.

Q. MR. JUSTICE FRENCH: This is, I think, an important answer and one that I should record. I am conscious that this is a paraphrase and very likely a clumsy one, so please correct it in any way you think right. What I have written is this, and I will read it straight through: "If there is evidence that other workers not living in Seascale received doses equivalent to or greater than those of the Seascale fathers, yet their children had not increased risk, we must look for something additional in Seascale to account for the cluster"?

A. Yes.

Q. That is sufficiently accurate?
A. That is sufficiently accurate.

MR. JUSTICE FRENCH: Thank you.

Q. MR. ROKISON: May I come on to paragraph 13 of your report where you start to deal with the studies leading up to the Black report? I think the first publication following the Yorkshire Television programme was a letter from Craft and Birch to the Lancet on the 3rd December 1983, which I think is C47.
A. Yes, that is correct.

Q. Is that a letter entitled "Childhood Cancer in Cumbria" by Craft and Birch?
A. Yes.

Q. You will see that they refer in the first paragraph to the Yorkshire Television programme?
A. Yes.

Q. Then they refer to the Manchester Children's Tumour Registry and Northern Children's Cancer Registry and so on?
A. Yes.

Q. In Table 1, they summarise the incidents of cancer and leukaemia in children before their fifteenth birthday in a number of areas?
A. Yes.

Q. What one finds from that is that there appears to be no general excess of malignant diseases or ALL in children under 15 in Cumbria when compared with other northern areas, and if one goes down to Table 2 where one finds Copeland being the district within which Sellafield is located (or Windscale as it then was) one finds similarly no general excess of malignant disease or ALL in Copeland as opposed to other local areas?
A. Sorry, you are asking me to say that that is what it says or do I agree?

Q. That is what it appears to show on these Tables, is that not right?
A. In some senses, this disagreed with Black, yes.

Q. Never mind whether it disagreed with Black; this was pre-Black?
A. Yes.

Q. MR. JUSTICE FRENCH: That is what the Tables are showing, is that right?
A. That is what the Tables are showing.

Q. MR. ROKISON: Indeed if one looks at Table 2, one finds for ALL for Copeland that for 1973 to 1977 and 1978 to 1982 the incidence seems comparatively low when compared with other areas, for example South Lakeland?
A. Yes.

Q. Perhaps it might be a convenient time to ask you next to look at two more letters which actually followed each other in the Lancet on the 28th January 1984, that being the letter from Gardner & Winter and the letter from Urquhart Palmer & Cutler. My Lord, perhaps we can check the references of those and turn to them after the adjournment.

MR. JUSTICE FRENCH: If that is the course you prefer, by all means.

MR. ROKISON: It might be convenient, thank you, my Lord.

MR. JUSTICE FRENCH: Is it in the same bundle that we should be looking?

MR. ROKISON: No, it will be in G, my Lord. It is G90.

MR. JUSTICE FRENCH: Can I put Bundle C away?

MR. ROKISON: Your Lordship can, yes.

MR. JUSTICE FRENCH: I am a bit puzzled. I have got C42 and I have got C29 to 51 which would embrace 42. It has been extracted.

MR. ROKISON: It may be that C42 is one on its own.

MR. JUSTICE FRENCH: I think that is what Mr. Butcher is telling me.

MR. ROKISON: Yes.

MR. JUSTICE FRENCH: Right, that explains it, thank you.

(Luncheon Adjournment)

Q. MR. ROKISON: I was going to ask you to look at the Gardner and Winter letter, which is G 90. Do you have that available?

A. Yes, I do.

Q. Can I ask you this? Is this a letter which you read for the purposes of your survey of the epidemiological literature in this case, or did you simply look at the reference to it in the Black report?

A. No, I think I will have read that.

Q. One can see what the title of it is - "Mortality in Cumberland during 1959-78 with reference to cancer in young people around Windscale". Again it refers to the Yorkshire Television programme and the fact that it is going to be the subject of a Government inquiry and the authors, including Prof. Gardner, present information

which they have available on death rates in the surrounding areas for the years 1959-78. They say:

> "We accept the statistics on cancer incidence might be more pertinent."

Then they refer to Craft and Birch, which we looked at this morning, and I think if we go to Table 1, which is simply "Mortality by cause of death and sex in Cumberland during 1968-78", one finds there, as observed compared with excess, that there is no very large difference between Cumberland and anywhere else during those periods and, in particular, in relation to leukaemia, in men there is a slight deficit, a very slight excess in women. Right?

A. Yes, I am trying to remember. I think that this is over a larger age range than just children, is it not?

Q. Certainly, yes, indeed. Yes, it is. I mean, it does not purport, I think, to be for children, at least at this stage, not the early part of the report?

A. No.

Q. And then Table 2 compares Ennerdale and Millom, Ennerdale being the district to the north containing Sellafield, just?

A. Yes.

Q. And Millom being the district to the south containing Seascale, just?

A. Yes.

Q. This table, I think, for my Lord's reference, is reproduced in the Black report as Table 2.16. What it shows is that for Millom, if you look on the right, one has a statistically significant excess of leukaemias in 1968-78 and an excess - not such a great excess - of cancers in the same period, which, to some extent, will arise from the leukaemias?

A. Yes.

Q. And one finds no excess shown for the Rural District of Ennerdale?

A. Yes.

Q. One finds that that is referred to about three-quarters of the way down the right-hand column, where they make the point in that paragraph that for Millom Rural District - it is about six lines down in the penultimate paragraph:

> "....the high rate was largely accounted for by the leukaemia figures, but this was not so for the statistically non-significant excess in Ennerdale."

Then they describe the leukaemias involved and, if one glances through that, one can see that there is a marked lack of specificity. Would you agree? There are two ALLs, three AMLs, one CLL, I think?

A. If you regard the sub-divisions of leukaemia as specificity, yes.

A Q. I agree, on that hypothesis. At the bottom of the page, they then refer to Table 3, which one finds over the page, and that is "Mortality from leukaemia under age 25 in Cumberland" and what does appear from that is that there is a significant excess in Wigton for 1959-67. Correct?
A. Yes.

B Q. But if one looks up to Whitehaven, one finds that there is no excess in Whitehaven for either of the periods concerned?
A. No.

Q. Indeed, although it is a very small number of cases, for the period of 1968-78, there is a deficit - 2 as against 2.9?
C A. Yes.

Q. May I ask this? It is nowhere, I think, highlighted in the Black report or, indeed, anywhere else, because they were not dealing, in particular, with a Whitehaven case, but was that a matter which you recalled and took into account when considering the Reay case?
A. No.
D

Q. I mean, were you, are you, aware of the fact that Dorothy Reay was born to a mother who was resident in Whitehaven at the time of conception and birth and lived all of her short life in Whitehaven?
A. I think that I was. I certainly am now.

E Q. Well, you are now. Did you perhaps think, when you wrote your report and reached the conclusions that you did, that Dorothy Reay had been born to a mother resident in Seascale?
A. No.

MR. JUSTICE FRENCH: Sorry, repeat the question.

F MR. ROKISON: I had asked whether Prof. Evans had thought at the time when he reached his conclusions in his report that Dorothy Reay had been born to a mother resident in Seascale and he said no:

Q. We see, for Wigton, as is pointed out at the bottom of the right-hand column on page 216, that the six deaths against 1.6 expected, all children under the age of 15, a
G statistically significant result. Can one draw any conclusion from that result?
A. Not a great deal.

Q. MR. JUSTICE FRENCH: Is this Table 2, near the bottom on the right?
A. Table 3, at the bottom, I believe, my Lord.
H

MR. JUSTICE FRENCH: Table 3, at the very bottom.

MR. ROKISON: It is Table 3, at the bottom, my Lord, yes.

MR. JUSTICE FRENCH: 15-24 age, or am I looking in the wrong place?

MR. ROKISON: My Lord, on page 217, at the top, on the left, is Table 3.

MR. JUSTICE FRENCH: Ah, that Table 3. Yes. That is under 25.

MR. ROKISON: And your Lordship will see that Wigton is at the bottom of that list.

MR. JUSTICE FRENCH: Yes, I have already seen the figures for Wigton and Whitehaven.

MR. ROKISON: What tells you that they are all under the age of 15 is the bottom of the right-hand column on the previous page, which makes that observation.

MR. JUSTICE FRENCH: So it makes it clear that, though it is headed under age 25....

MR. ROKISON: Yes, but it makes it clear that they were all actually under the age of 15.

MR. JUSTICE FRENCH: Yes, I am with you. So, although under 25 is the age studied, in fact, they were all under 15.

MR. ROKISON: In fact, they were all under 15:

Q. And I think you agree that you cannot draw any particular conclusion from that, and I think the authors of this letter, in the last paragraph of the letter, said that:

> "Studies of this sort will inevitably yield some excesses .... as a result of statistical fluctuation alone, and both the high rates near Windscale and the raised mortality levels in Carlisle and Wigton .... could have happened in this way."

A. Yes.

Q. No doubt, if Yorkshire Television had fastened upon the excess in Wigton and had it happened to have a nuclear installation nearby, that would not have been an end of it. Do you agree?

A. That certainly is calling surely for speculation on my part.

Q. MR. JUSTICE FRENCH: Yes, but it is a fair comment?

A. It is a fair comment.

Q. MR. ROKISON: Yes, I am sorry, it is perhaps not for me to make comments, I agree.
A. I would entirely agree with you that you cannot do your epidemiology through the media.

Q. No. I do not whether it is something which is easy for you to do, but those instructing me have said - they are all under 15 - that, for the 0-14 age group in Wigton, with six observed over 2.1 expected, it is suggested to me that that would produce a P value of 0.006?
A. Yes, very likely.

Q. Which would, on the face of it, appear to be a highly statistically significant cluster?
A. I do not think that that is sufficient reason to say it is a cluster, because, as you have pointed out, you have selected your area, your age grouping and the calendar years after looking at the data, and that is exactly what Yorkshire Television did and that, on its own, was not sufficient evidence.

Q. No, I think they did not. This is by contrast because here what they were doing was without any preconceived ideas of looking at Wigton in particular. They were simply looking at mortality from leukaemia under the age of 25 in Cumberland and they took a number of rural districts in Cumberland, of which one happened to be Wigton, and, indeed, they did their study on 0-25 years, but it turns out that this cluster, as I call it, was in the ages 0-14?
A. Yes.

Q. So I would suggest to you that what you have just said is quite wrong. This was something which was not discovered in advance, but was something which was revealed by this geographical study
A. What I wrote in paragraph 16 of my report, in the last sentence or last two sentences - I have slightly disagreed with Pomiankowski, if that is the correct pronunciation of his name - that:

> "There are an infinite number of ways of looking at an excess in terms of most appropriate age group, time period and geographical area. Interpretation of probabilities is not straightforward because adjustment must be made for all these multiple possibilities."

So, if that is what you are saying to me, I entirely agree with you.

Q. It was not, I think, what I was saying to you. What I was simply pointing out was this: that, unlike the Seascale cluster, which was discovered by journalists - I do not mean that in any pejorative sense?
A. Yes.

Q. That, unlike that cluster, "the Wigton cluster" - and I call it that in quotes - was something which was not identified in advance of this study, but it was simply that, in the course of doing a geographical study for the purposes of looking at mortality from leukaemia in young people in Cumberland, those preparing these statistics came up with a highly statistically significant association in respect of Wigton, in respect of a fairly limited period of years, and in respect of a limited age group, 0-14?

A. Yes.

Q. That demonstrates, does it not, the point that they are making in the last paragraph: one should not necessarily draw any inferences from that alone, but one simply looks at it and says, "Well, you would expect to find excesses or clusters if you are doing this sort of exercise"?

A. Yes.

Q. It is something which could well arise by chance, and probably did?

A. Yes.

Q. If we can go on in the same publication, the very next letter is the Urquhart, Palmer, Cutler letter?

A. Yes.

Q. This is John Urquhart, not to be confused with what has been called "the Scottish Urquhart", although I have no doubt John Urquhart is Scottish as well. James Urquhart is the Scottish Urquhart.

MR. JUSTICE FRENCH: Where are we going to now then?

MR. ROKISON: My Lord, it is the very next document.

MR. JUSTICE FRENCH: 91?

MR. ROKISON: The letter follows on from the Gardner and Winter letter. The next letter.....

THE WITNESS: ....is incomplete.

MR. ROKISON: ....which happens to be published in the same edition of The Lancet, is the letter from Urquhart, Palmer and Cutler. My Lord, it may be that, because of the way that the bundles have been arranged, by alphabetical order, that your Lordship will have page 217, but will not have the next page here. Is that right?

MR. JUSTICE FRENCH: Quite right.

MR. ROKISON: My Lord, unfortunately, you will have the whole document in Bundle U because the first author was Urquhart.

THE WITNESS: But there are only a couple of paragraphs on the second part. It is 250.

MR. JUSTICE FRENCH: Is there any mileage in my taking it out of U and adding it to G?

MR. ROKISON: It might confuse matters. Where I think there might be mileage, my Lord, is if we can simply copy for you page 218 so that you can add that to your reference in Bundle G.

MR. JUSTICE FRENCH: If that could be arranged fairly quickly, then we shall not forget to do it. Otherwise we might. As it is, I am going to U.

MR. ROKISON: I am sorry about that. It is U 250, my Lord. My Lord, if your Lordship would like to have Mr. Spencer's for the time being, then he can get another one. He might remember!

MR. JUSTICE FRENCH: If he can spare it, thank you.

THE WITNESS: May we put away G?

MR. ROKISON: Yes, by all means put away G. I am sorry about that, that you have to turn to another one.

MR. JUSTICE FRENCH: I thought it was going into G.

MR. ROKISON: Yes. Of course, the witness does not have that extra page.

MR. JUSTICE FRENCH: I alone need not put G away?

MR. ROKISON: No, your Lordship should either keep G or U, and I do not care which, because I am going to refer to this letter. I am sorry about the chaos:

Q. Do you have your report still open?
A. Yes.

Q. In paragraph 13 of your report, what you say is:

> "The researchers for the programme," which are these gentlemen, I think, or certainly Cutler was and may have been assisted by Urquhart and Palmer, "found that five cases of leukaemia had occurred in children aged less than ten years between 1963 and 1982...."

MR. JUSTICE FRENCH: Which page are we on?

MR. ROKISON: My Lord, this is paragraph 13 of Prof. Evans' statement, where he refers to this, on page 7, where he says:

> "The researchers for the programme found that five cases of leukaemia had occurred in children aged

> less than ten years between 1963 and 1982, where less than one would be expected if the rate near Sellafield was equal to the average rate in the United Kingdom as a whole."

It appears that, if you look at Table 1, one sees that they are taking 1963 to 1982, "Deaths per 100,000 person-years for England and Wales and for selected parts of Cumbria", and they take, across the top of it, England and Wales, Copeland, Millom, and Seascale and coastal villages. There, as I say, they take the years 1963-82. Then, when they deal with all malignancy cases, rates per 100,000 person-years, in Table 2, they take the years 1968-83 and, as you state, at the bottom of the first column of this letter, 217 on the left, they say:

> "In the areas closest to the BNFL plant and coastal areas where high levels of radioactivity have been found there is a significant excess of deaths from malignancy and leukaemia in the 0-24 age group."

And they refer to the data:

> "The 7 deaths in the age group 0-24 from all malignancies and the 4 from leukaemia recorded for Seascale and coastal villages and the 8 deaths from leukaemia in Millom are all significantly more than would be expected...."

and so on. So far as that is concerned, it is the case, is it not, that Seascale and the coastal villages are part of Millom?

A. Yes.

Q. So that the Seascale cases constitute part of the Millom cases. Is that correct?

A. I would think so.

Q. And the cases they are referring to, therefore, are the 0-24. If you look at Table 1, the 0-24, all malignancies, Seascale and coastal villages is the third figure down on the right? Is that correct? That is the 7?

A. Yes, 22.4.

Q. And the 4 from leukaemia, so that there are 3 malignancies other than leukaemia?

A. Yes.

Q. The 8 deaths from leukaemia in Millom, and there are 7 other malignancies in Millom?

A. Yes.

Q. If one goes down, one finds in the next paragraph this:

> "Craft and Birch's contention that clusters can occur by chance fails to meet the point that a cluster of cases may also be a sign that some

specific cause is at work. However, the 6 leukaemias diagnosed in children under the age of 15 and living in Seascale" - those are the ones mentioned in the television programme - "are not a 'cluster' in that sense. They are more an 'excess'."

Then one finds the details of these, where you get one died in 1956, another died in 1960. One then recovered. Then the next one died in 1970, died 1971, and another recovered. They say there were five cases under 10. So the position is that, in order to, in a sense, produce what they say is an excess rather than a cluster, they go back before the period which they have set in Table 1. Is that correct?

A. Yes.

Q. So that they take in Case A and Case B, which do not come within Table 1?

A. Yes.

Q. Also, of course, they take in two cases which have not resulted in death at all but where the child has recovered?

A. Yes.

Q. So that there is some overlap between them, but they are not the same cluster or excess that is being identified?

A. No, this letter is not of epidemiological quality.

Q. It is very difficult to puzzle it out, in fact?

A. That is, in some senses, what I think I was trying to say when I talked about an infinite number of ways of looking at an excess.

Q. Yes, I see that, because, in fact, just to show this example, if one were to start by simply setting one's parameter of saying, "I want to look at those that have died within a particular period," one would have found, in relation to the cases which were thrown up by the Yorkshire Television study, that, out of those six cases, they would only actually have come up with two, which are cases D and E, because two recovered and two were too early?

A. Yes.

Q. And it is an example - perhaps a clear example - of drawing one's parameters round the cases?

A. Yes.

Q. I think we can leave that then. There is nothing else I wanted to ask you about that. Having looked at that, could you now put that one away, please, and if my Lord would put G away, then I can come back to your report, if I may, your statement, in paragraphs 14 and 15, where you deal with chance and Poisson probabilities and you deal with the convention of taking 95 per cent confidence factor or P value of 0.05 as the test of statistical significance?

A. Yes.

Q. As you say, what one is considering is the likelihood of the association being demonstrated when, in fact, there is no true association at all. Is that right?
A. In this instance, there is no true excess.

Q. There is no true excess, yes. Well, it depends what you mean by excess. There may be a greater number, but it is something which may occur by chance?
A. Yes. There may be no difference in the true underlying rate.

Q. Yes, and it is the case, is it not - I think statistically it would be the case - that, if one is carrying out a number of tests in order to test a number of hypotheses, the more hypotheses one is testing, the more likely one is to come up with a statistically significant result by chance?
A. If they are independent hypotheses, yes.

Q. So that if one takes the 95 per cent confidence level or the P value of 0.05, would it be right that if you were to carry out 20 independent tests, testing 20 unconnected hypotheses, that the chances are that you will come up with one statistically significant result where there is no true excess by chance?
A. Yes.

Q. How, if at all, does one adjust or take into account that point if one is testing a number of hypotheses and reporting on one's results?
A. If one has some good evidence that the things that one is dealing with are independent, then there is a simple correction that you can have mathematically.

Q. I see.

Q. MR. JUSTICE FRENCH: Will you say that again? I am afraid, with your hand there, it makes you a little bit indistinct for my ears?
A. I am very sorry. If the hypotheses are independent, then there is a simple mathematical correction that can be made to adjust those P values.

Q. MR. ROKISON: How does it work? Can you explain what the correction is? What is the correction you apply?
A. It is the inverse of the situation that, if we say that there are no excesses anywhere, then the probability of getting one is 1 minus the probability of not getting any, and we actually work it out as 1 minus 0.95 to the power n - all right - by laws of mathematical probability, and loosely you could multiply - if you have got 20 tests, then you can multiply the 0.05 by 20, but, of course, by the time you reach 20 it starts not to work.

Q. I see. So, if you are carrying out 10 independent tests, for example, similarly, you would multiply, would you, your factor by 10?
A. Loosely, in small numbers, that will actually work, but as soon as we get to large ones we can apply what is called a Bonferroni correction, after an Italian statistician.

Q. MR. JUSTICE FRENCH: Is that correctly described as the converse of the null hypothesis?
A. It is saying what is the probability of rejecting many null hypotheses, each of which are independent? That probability will increase the more tests you do.

Q. The probability of wrongly rejecting the null hypotheses?
A. At least one of those null hypotheses.

Q. At least one of the null hypotheses?
A. That is right. So, by my arithmetic, if we, in fact, have 10 tests, the probability that at least one of them will be significant is 0.40126, so it is not quite 0.5. So you are not quite multiplying by 10.

Q. MR. ROKISON: No, but, in those circumstances, you have got about 40 per cent chance that you are going to produce something that is statistically significant?
A. Exactly, yes.

Q. By chance?
A. Yes.

Q. Is that a matter that was taken into account in the Gardner case control study?
A. You are moving now from looking at excesses in a particular area to something in regard to the Gardner case control study. Gardner did not attempt to make any adjustment to his P values, I imagine because he was not sure exactly how many independent hypotheses he had.

Q. I will come to the detail of the Gardner study with you later, but it does appear, does it not, from the document which was appended to Prof. Gardner's statement that there were a number of hypotheses listed which were to be tested by his study?
A. Yes.

Q. And, indeed, in the report - I will come to the detail - but there are a number of hypotheses that are set out under four categories, I think?
A. If we went to the report and you were able to show me what those four categories were, I would be happy to discuss them, but I do not think he sets them out terribly clearly in terms of hypotheses in the tables of result.

Q. As I say, I will come back to the detail but, if one is testing a number of hypotheses, then the statistical facts to which we have just referred should be taken into account, should they not?

A. Particularly if they were not specified prior to the study.

Q. What I do not quite understand is why that should be so. Surely, if you are testing a number of hypotheses, you are bound to define them before you do your study?
A. No, or, at least, if you were doing your study among every geographical area, every rural district in the country, then you would have perhaps several thousand possible null hypotheses and, if you said you have listed all thousand of them, then that is not terribly sensible, but if you have listed four or five hypotheses, the usual practice in epidemiology would not be to adjust all those three or four, but if you had no prior hypotheses and, as is done, for example, in drugs surveillance, if you have a study which follows up adverse events of drugs among a very large number of possible drugs, then you will be very careful there to adjust your multiple probabilities. I am sorry, my Lord, I will put my hand down. You would adjust your probabilities there because there are a very large number of drugs that you are studying and, by chance, one of them will come up with more adverse events.

Q. But why does it make any difference in principle whether the hypotheses that you are testing, in a sense, are something which is inherent in the study because you are testing a possible cause and effect with a number of drugs? Why is it different if your hypotheses are defined in advance because, surely, the person carrying out the study will say to himself in advance, "I have got to test the possible connection with a number of drugs here and I should have thought the most likely that might be connected with it are, say, 20 and, therefore, I will test those 20 drugs"?
A. Yes, when it comes to 20, you want to make some adjustment, but you would not necessarily make a full Bonferroni correction if there were several hundred drugs and you had prior specified 20 of them. You would not do a full Bonferroni correction, but you might apply some other more limited correction.

Q. Simply to take account of the fact that, as I say, the more hypotheses you test, the more likely you are to come up with a statistically significant result by chance?
A. Yes.

Q. Are you aware as to whether any such allowance or adjustment was done in the Gardner study?
A. As far as I can tell, no such adjustment was made.

Q. Is it something that you have done in your re-assessment?
A. No.

Q. Paragraph 16, you touched upon earlier, Prof. Evans, Pomiankowski's paper - I do not know whether that is the right pronunciation either, but I am sure he will forgive us if it is not - where what you say is:

"There are an infinite number of ways of looking at an excess in terms of most appropriate age group...."

and so on. Would it not be more accurate to say "an infinite number of ways of seeing if there is an excess or to identify whether or not there is an excess in terms of appropriate age group, time period and so on"?

A. Yes, I am afraid I missed the subtle distinction.

Q. It is not particularly subtle. It is simply this: you seem to presuppose that there is an excess and, of course, if what you are looking for is to see whether there is an excess or not, then if you adopt some parameters, you may find one and, if you adopt other parameters, you may not?

A. Absolutely, yes.

Q. And, as we have seen, the Yorkshire Television team set their parameters round an apparent excess and, lo and behold, they found one?

A. Yes.

Q. You then go on to deal with the Draper review, which is D 62, which is a very large document indeed, and it is not a matter that I want to go into with you in any detail. If you will just bear with me a moment, I am just looking for the relevant part. I think you have said in your evidence that epidemiologists frequently looked at NHLs and leukaemias together and I think that you referred particularly to the Draper 1992 paper as an example of that?

A. Yes, but this study also in a number of instances combined....

Q. Can we perhaps just look at this study to see that? It is a large study and it is D62. It in fact comprises a number of studies and papers by a number of authors?

A. Yes.

Q. Could you please look at page 9 of that, which is part of a paper by Stiller et al, the National Registry of Childhood Tumours and Leukaemia Lymphoma Data?

A. Yes.

Q. One sees that Dr. Draper is one of the authors of that paper. One sees there that for the purposes of the National Register of Childhood Tumours and Leukaemia Lymphoma Data, they set out in the various Tables starting on page 9, lymphocytic leukaemia, acute non-lymphocytic leukaemia, NHL, lymphocytic and unspecified leukaemia, all leukaemia, NHL and unspecified lymphomas and all diagnoses, and one finds those same categories continuing through in those Tables?

A. Yes.

Q. If one looks at the discussion on page 9, they say under the heading "The leukaemia/lymphoma data" in the definition of eligible cases:

"Their diagnosis was leukaemia, NHL, Burkitt's lymphoma or unspecified lymphoma. These correspond to the categories I(a) to (e) and II(b) to (d) in the classification scheme of Birch and Marsden. The study is mainly concerned with leukaemia but NHL has also been included since NHL and leukaemia can in some instances represent different stages in the natural history of the same disease. In particular, the distinction between T-cell acute lymphoblastic leukaemia (ALL) and T-cell NHL is usually made in terms of the percentage of blast cells in the bone marrow, and the very rare B-cell ALL may be considered as a leukaemic manifestation of B-cell NHL."

Then they continue the discussion. The position is, is it not, that there are some NHLs and leukaemias which are difficult to distinguish from each other?
A. Yes.

Q. But it is by no means generally the case that leukaemias and NHLs would be lumped together as being, in effect, the manifestation of the same disease?
A. Well, they have lumped them together in this on occasions.

Q. I beg your pardon?
A. You are asking me to stray beyond the bounds of my competence.

Q. Fine. I am very happy with that answer. By all means feel - and I am sure you will - that you do not have to answer a yes or no, but you can actually say that so far as you are concerned it is not proven.
A. The "don't know" is entirely permissible here.

Q. The answer is that you do not know?
A. I don't know.

Q. Then I think we can put that away. You say in relation to the pattern of clustering and so on, you refer in paragraph 17 at the bottom of page 8 to there being some evidence for extra Poisson variation and more clustering in younger age groups. That means a clustering beyond that which you would normally expect from a Poisson distribution, is that right?
A. That's correct.

Q. The problem is that within a Poisson distribution one will get what appear to be clusters?
A. Entirely so.

Q. And they will appear by chance?
A. Yes.

Q. What you are saying here is that there are clusters which are, in a sense, even tighter than those which you would get from a Poisson distribution?

A. You have more in the tail of the Poisson distribution than you would expect.

Q. MR. JUSTICE FRENCH: I am sorry, would you say that again? You have more ...?
A. What happens is that the Poisson distribution will give us the probability of within an area getting nought or one or two or three or four and so on cases. So when we have got four cases, we can calculate the Poisson probability of getting that, assuming the rate is not different. What we find is that there is a little evidence that we are getting the fours, the fives and the sixes a little too often, given that the underlying rate over the country or over smaller areas is actually fairly homogeneous. So we are getting a few little clusters.

Q. Or too many little clusters?
A. We are getting too many little clusters, yes.

Q. MR. ROKISON: More clusters than you would get from a Poisson distribution?
A. That's right. May I just try and make a slight distinction here? One of the things which may have confused people is that within epidemiology as a whole, clustering has tended to come partly from the work of Prof. Knox, where you look at clustering both in space and in time, and in infectious disease epidemiology you look for clusters that are cases that appear not just in the same area but in the same period of time very strongly; whereas on the whole in regard to this, people are not looking for that. In that document, most people didn't look for distance apart in space and time, but Prof. Knox in that document of Draper looked at that, so his emphasis on clusters will very much depend on whether they are happening at the same time, because you will get a cluster of outbreaks of flu because they happen one after the other because the infections aetiology suggests that it will be passed from one person to another. So clusters very often imply short periods of time, but obviously in regard to leukaemias we are not talking about that sort of clustering in short periods of time. We don't get three or four cases diagnosed in one week in the same place.

Q. Could you tell me if this is right, Professor: you can get too many little clusters for the aggregate of them to be the product of chance?
A. Very well expressed, my Lord.

Q. MR. ROKISON: I think it was in relation to this part of your evidence that my learned friend asked you a leading question and my Lord, who is permitted to do that, asked you another leading question on the following page. Have you got the transcript for day 13, please?

MR. JUSTICE FRENCH: Page?

MR. ROKISON: 19, my Lord.

Q. Do you have it, page 19?
A. Yes.

Q. You will see at about F, my learned friend Mr. Langstaff reads this passage of your statement and then asks that question to which I make an intervention. Do you see the question?
A. Yes.

Q. "Does that hint at there being a pre-natal factor in the aetiology?" and there is then a discussion over the next page as to whether or not it is a leading question, and Mr. Langstaff asks you to answer it whether it is or not. You says this:

> "There is some evidence that the pattern in younger age groups is different to that in older age groups. If you are going to have a disease involving cancer in very young people, aged 0-4, they don't have very much time to have been exposed to something that might have caused it and so it is entirely possible that the exposure might possibly be pre-natal or have been caused pre-natally".

Then my Lord, quite rightly wanting to get it down accurately, asks the witness to clarify that, and below G the end of it is that it may be due to something before birth rather than after. Of course, it could very well be due to something which occurs early in life, could it not?
A. It could be, but the closer you get to nought, the more difficult it is.

Q. I appreciate that, but it is right, is it not, that as far as common child ALL is concerned, this is something which peaks quite early in life usually between the ages of 3 to 5?
A. Something like that.

Q. We know that Prof. Greaves - and there may be others as well - has a theory as to a possible viral cause of that peak?
A. Yes.

Q. When the child is first exposed to viruses after the protection of the home?
A. Yes.

Q. That is the sort of thing which could cause these sorts of clusters in young children?
A. It is one of the things that could, yes.

Q. We then go on to deal with the Black report itself ---

MR. JUSTICE FRENCH: Does the transcript go away again?

MR. ROKISON: The transcript can go away, my Lord. I keep forgetting to say that, I am sorry, my Lord.

MR. JUSTICE FRENCH: Otherwise, we get buried.

MR. ROKISON: I will try to remember. I think we should at the moment have a clear table apart from Prof. Evans' report.

MR. JUSTICE FRENCH: Thank you.

MR. ROKISON: We are now going to look at the Black report itself, so may I ask your Lordship to find the Black report again?

MR. JUSTICE FRENCH: Tell me where it is.

MR. ROKISON: B13, my Lord.

MR. JUSTICE FRENCH: When you actually look at a report having read a lot about it, it is rather like meeting somebody of whom you have heard a great deal and at last there it is, almost like an old friend!

MR. ROKISON: Yes, indeed.

Q. Perhaps we will just look at this first, Prof. Evans, at page 23 where there is this rather useful map, in which one can see, before we look at the various Tables, the limits of Millom and Ennerdale and Whitehaven and how all those three together make up Copeland, with Allerdale lying to the north. You were referred to Table 2.1 on page 13. They are the Seascale resident leukaemia cases since 1955 and aged under 25 years at diagnosis. Again one sees that in those cases one has a fairly wide variety of leukaemias in the right-hand column?
A. Yes.

Q. In fact, there is I think only one case there, in relation to the clustering that you have just been giving evidence about, of an ALL in a young child, which I think is case E?
A. Sorry, is this Table 2.1?

Q. Yes.
A. Case E?

Q. Sorry, case 6.
A. Yes.

Q. Just relating to the Urquhart, Palmer and Cutler letter, you will remember that they set out the cases in that letter being A to F, and I think that the cases are 1, 3, 5, 6 and 7 in this list, but the only one that is a child ALL is Case No. 6?
A. Yes.

Q. Keeping this available, in paragraph 20 you very helpfully summarise what you regard as being the most significant conclusions. We would not take issue with them on a., but at b. you say:

> "The rare disease of leukaemia did occur far more frequently than could be explained by chance in variation in the immediate vicinity of Sellafield".

Can you identify - perhaps you cannot - where you say in the report they say that?

A. It depends partly, I suppose, on whether you regard Seascale as not being adjacent to Sellafield.

Q. We have looked at this before. The fact is that it appears from the various studies that are referred to here that the excess is a Seascale excess, would you accept, which in turn drives, if I can use that expression, Millom, in the relevant Tables, which in turn (where included) drives Copeland? In other words, where one finds an excess for Millom, it is explained by Seascale; when one finds an excess for Copeland, it is explained by Millom, which in turn is explained by Seascale?

A. I think there is a subtle distinction to be made between saying that the excess is driven by... and saying that there may actually be, nevertheless, an excess in some of the other places, but I couldn't point you to chapter and verse on the details in that. If you assure me that the Black report does not mention this as being close to Sellafield, then I would have to agree with you.

Q. It does, but it refers to it as being close to Sellafield in this context: what they do say is that their terms of reference are to look into the recently published claims of an increased incidence of cancer in the vicinity of the Sellafield site, and then in particular they have to examine the evidence concerning the alleged cluster of cancer cases within the village of Seascale. It is true that they are examining the alleged increase in the immediate vicinity of Sellafield, but they are doing so by reference to the cluster in Seascale, do you follow me?

A. Yes. I think paragraph 6.2 of their conclusions says "The proximity of Sellafield .... is a factor which is not one which can be categorically dismissed, nor on the other hand is it easy to prove".

Q. Indeed, but it is the proximity of Sellafield to the village of Seascale, since you are reading from 6.2. Indeed they are saying that if you are looking to see the possible cause of a cluster, if we can call it that, in Seascale, one cannot simply dismiss the fact that Seascale is near Sellafield?

A. No.

Q. That is what they are saying, is it not, and nobody is suggesting that one should? If we need to go through the

various studies in order to see that this is the case, I think we can do so reasonably rapidly. The studies are set out and summarised in Table 2.5 on page 21. I certainly do not intend to invite you or my Lord to look at all of them, but one finds that at page 22 they say in paragraph 2.20:

> "Leukaemia mortality in Cumbria 1951-1978 was lower than national rates when all ages are considered together."

They give chapter and verse for that.

> "In West Cumbria, the age-standardised incidence of malignant disease among both men and women during 1969-77 was significantly lower than in England and Wales overall ... When leukaemia is considered, again the incidence was not significantly higher than expected in either sex.
>
> 2.22 This evidence, while reassuring in that it demonstrates a generally low incidence of malignancy in West Cumbria considers all ages together and relatively large geographical areas. It does not exclude the possibility of a localised excess of cancer in young people living near Sellafield".

Then they refer to Alderson, and again at the top of page 24 they refer to the fact that in the Alderson study the population was separated into 0 -14 and 15 -74, and they say:

> "As mentioned in paragraph 2.9, 0-14 is the commonly accepted age-span for studying childhood tumours".

Then they show the results in paragraph 2.24, where one finds Table 2.8 and 2.9. In Table 2.8, one finds that there is a statistically significant increase in the Copeland District for lymphoid leukaemias and all leukaemias, and this is for 1961 to 1980, correct?

A. Yes.

Q. What they say at the bottom of that Table is "Other sites studied and giving SMR/SRR not statistically significant were ..." SMR is standard mortality rate, is it?

A. Yes.

Q. And SRR is ratio and registration, is it?

A. Yes.

Q. Then one finds "liver, bone, thyroid, all lymphoid, multiple myeloma, monocytic leukaemia, other leukaemia, leukaemia unspecified, lung, Hodgkin's ..." and so on and so forth. So there are a very large number of malignancies which were considered and do not feature in that Table which gave no statistically significant increase in Copeland?

A. Yes.

Q. Then one finds a comparison of SMRs and SRRs for Copeland and control locations in Table 2.9. One sees from that that it is in the 0-14 age group that one finds excesses, is that right?
A. Yes.

Q. Rather than in the 15-74 range?
A. It was actually in Cook-Mozaffari, however you pronounce his name, that the 0-24 ---

Q. No, it is her.
A. Her, I mean; it is Paula, isn't it? It was in one of her papers that the 0-24 excess around nuclear installations was. I found that in the Common Bundle.

Q. It is in Cook-Mozaffari?
A. Yes.

Q. We are coming to Cook-Mozaffari in a moment.
A. But you asked me to find that over lunch.

Q. You had referred to something ---
A. And I couldn't remember the chapter and verse.

Q. You thought it was Draper, I think?
A. That's right.

Q. And you were going to check. Perhaps I can go on for a moment to try and identify from these reports which are here summarised in Black where it is that one finds where the Copeland excesses, to the extent that there were excesses, are derived. Perhaps I should refer you to 2.27 on page 25, where they refer to the Manchester District Tumour Registry and Northern Cancer Registry. This is the Craft and Birch paper to which I referred you earlier?
A. Yes.

Q. Then they refer to the Urquhart, Palmer and Cutler paper, to which I also referred you?
A. Yes.

Q. That is at paragraph 2.29. Over on page 28 at paragraph 2.31, they refer to Gardner and Winter. Towards the bottom of page 28 of the report, about ten lines up from the bottom, they say:

> "If the under 25 group only is considered, there were apparent raised cancer death rates in both areas during 1968-78 but not during the earlier years 1959-67. In Millom Rural District the excess was largely accounted for by leukaemia, for which there was a four-fold excess in 1968-78 period, but this was not the case in Ennerdale Rural District. Looking at leukaemia deaths in young people under 25 in Cumberland during 1959-67, there were statistically significant excesses in Carlisle County Borough and Wigton Rural District ..." -

which we have just looked at?

A. Yes.

Q. Then one finds the Gardner and Winter Table, to which I have already referred you, set out as Table 2.16. At paragraph 2.33, they say:

> "The above results can be summarised as suggesting an approximately four-fold higher rate of leukaemia mortality in the under 25 year old population in Millom during 1968-78 - or twofold during 1959-78 - and an approximately 10-fold higher rate of leukaemia incidence in the under 10 year old population of Seascale (paragraph 2.4; Urquhart et al 1984). No unusual cancer rates are found among the over 25 year old population in Millom or Ennerdale".

Then they go on in the report, having dealt with the rural districts, to deal with smaller areas in the United Kingdom, and in particular look at the position of Seascale?

A. Yes.

Q. They say in paragraph 2.34, after dealing with the Seascale and Millom apparent excesses, "The findings are based on small numbers of cases", and you would agree with that?

A. Yes.

Q. They say, "Excesses were also reported in Carlisle and Wigton", which we have looked at, and "... we are aware of leukaemia 'clusters' reported in other areas of the country, not all in the neighbourhood of nuclear plants". Then they look at Seascale. In paragraph 2.36, it should I think be 675 electoral wards rather than 765, and the same in paragraph 2.37. They make the point in paragraph 2.37 that where they are dealing with all childhood cancers the Seascale cases are four in number, but when they deal with lymphoid malignancy, it is for Seascale the same four cases?

A. Yes.

Q. So it appears that there are no other childhood cancers other than the lymphoid malignancy cases considered for the purposes of these studies?

A. In Seascale, yes.

Q. At the end of paragraph 2.37, they say:

> "Again there was no tendency for wards with higher rates to be in West Cumbria rather than in other parts of the Northern Region".

A. Yes.

Q. Paragraph 2.40 over the page on page 33:

"It is important to note that in neither of the latter two studies were the rates in the areas of interest above the observed range, but they were close to the top. Thus, the Seascale incidence and Millom Rural District mortality rates for leukaemia among young people are unusual, though not unparallelled."

Then one finds various discussions as to what the possible causes might be. I was merely seeking, in quickly running through this part of the Black report, to really confirm that apart from the excess in Seascale, which in turn produces an excess in Millom, there is no evidence of any other excess in the West Cumbria region other than in Seascale?

A. Not from Black, no.

Q. Not from Black or any of the reports which Black sets out and summarises?

A. No.

Q. Very well, that is fine. I think we can close Black for the moment, if we may. May I bring you back to your report at paragraph 20? I have asked you about your point b. at the bottom of page 9. It is agreed, I think, that although Seascale is in the immediate vicinity of Sellafield, we can read that, as far as Black is concerned, as a reference to Seascale in the penultimate line, is that correct?

A. Sorry, the penultimate line of ...?

Q. The penultimate line of page 9 of your report?

A. Sorry, I was looking at the penultimate line of paragraph b.

Q. Is that right, so far as Black is concerned?

A. They mention Sellafield at least as many times in the conclusions if not more than they mention Seascale. I did a quick count.

Q. Forgive me, the purpose of taking you through the parts of the report that I have done where they are dealing with the excesses and so on ---

A. It would be chance variation in Seascale.

Q. What they are referring to here is leukaemia excess?

A. Yes.

Q. As I say, one could read for "the immediate vicinity of Sellafield", "equals Seascale" for these purposes?

A. Yes.

Q. I think there is no need for me to deal further with your conclusion b. They also conclude, as you point out in c., that the radioactive emissions from Sellafield into the environment were too low to account for the size of leukaemia excess, and it was that which we touched upon this morning, your comment upon that, of considering that

that last conclusion is very weak, and your explanation, as I understand it, is that it is twofold - first of all the doubts about the levels of environmental exposure doses?

A. Yes.

Q. Secondly, as to whether it is appropriate to measure risk by reference to standards which have been derived from other studies such as A-bomb?

A. Yes.

Q. Is that correct?

A. That's correct.

Q. I think that neither of those areas are areas in relation to which you are an expert, if I can respectfully put it that way?

A. No, but I think the logic that the more cases there are around Sellafield the less likely it is to be due to Sellafield seems to me a little weak, whether you are an expert or the common man.

Q. That is rather dangerous, is it not? You may find a lot of cases round Nelson's Column, but it does not necessarily mean that they are caused by Nelson's Column?

A. If you did and you found everybody falling down dead by Nelson's Column and you were convinced it was nothing to do with Nelson's Column, I think that would be a failure of an epidemiologist to at least consider that there may be something to do with it.

Q. He might consider that, or it could be pigeons?

A. In which case, it's something to do with Nelson's Column, in that case, but I agree with you, the point being that I think it is logically, and I suspect the Black Committee themselves would say that logically that is a weak conclusion. The more cases there are around Sellafield, the less likely it is to be due to Sellafield.

Q. Forgive me, I did not understand that comment?

A. The logic of that conclusion c. is that the more cases there are in the environment of Sellafield, whether it is Seascale or anywhere else, the less likely it is to be due to Sellafield, is to me a simple failure of logic.

MR. JUSTICE FRENCH: I think to everybody else as well. I think there is a misunderstanding here in some way.

MR. ROKISON: I think there may be, my Lord, as to what conclusion c. really is.

Q. It is your report but, as I understand it, you are attempting to summarise the conclusions or some of the conclusions of Black. In that conclusion c., as I understand it, the relevant conclusion is that emissions of radionuclides into the environment are too low to have

produced the excess of leukaemias on the basis of recognised risk estimates derived from experience such as A-bomb studies, is that right?

A. Yes.

Q. It does not follow from that, does it, that they are saying the more cases you have the less likely it is that they are associated with Sellafield. What they are simply saying is "We do not think the answer to this is radionuclides in the environment, because the levels are too low". That is all that is being said, is it not? There is nothing illogical about that, is there?

A. There is not in itself, but the consequence of saying that is that had there been ten times as many cases, they would have been even more sure that it was nothing to do with radiation. I think all I am trying to say in that is that it is a conclusion based on theory and not on data.

Q. With respect, it is based on data on the one hand and theory developed from data on the other. May I explain what I mean? It is based on data so far as the assessment of the environmental doses are concerned?

A. Yes.

Q. I do not know whether you were aware at the time when you produced this report, but I assume you are now aware of the enormous work which has been done for the purposes of this litigation in trying to assess as accurately as possible the maximum environmental exposure and doses to the relevant individuals?

A. Yes.

Q. You are aware of that?

A. I am aware of that, but not in detail.

Q. But you are aware that a very substantial exercise has been undertaken and that Professor Jones and Dr. Stather have produced reports based on that exercise in which they both express confidence that the figures they have come up with are maximum figures which are based on cautious assumptions?

A. Yes.

Q. It is not right, is it? You say in the second part of this paragraph, paragraph 21:

> "From my own limited knowledge it seems that the true level of radioactive emissions will never been known precisely..."

Well, to that extent I think we all agree that you are right and it will never be known precisely, but you go on to say:

> "...and any error will nearly always lead to an under-estimate, since by their nature having measured a given amount of radiation we know the

level is as high as that reported and the unknown can only increase the levels."

If one measures radiation, then the position is that that is the amount of radiation which you have measured, isn't it?

A. Well, if you measure the radiation, you, for example, cannot - we have no method of directly measuring neutrons. We have to make neutrons interact with something else in order to begin to measure them.

Q. Are you suggesting that there was neutron exposure in the environment?

A. I don't know.

Q. Have you any reason to believe there was neutron exposure in the environment as opposed to in limited places within the plant?

A. No.

Q. Leaving aside for the moment neutrons - although we don't accept that you cannot measure neutrons - I am merely trying to test what you are saying in this paragraph. You say:

"...having measured a given amount of radiation we know the level is as high as that reported and the unknown can only increase the levels."

What I am suggesting to you is if you measure something, then that is what is there, it is what you have measured, unless your measure is inaccurate?

A. Your measure may be inaccurate...

MR. JUSTICE FRENCH: I think you may be at cross purposes here. Surely the radiation has never been measured. What has been measured is that which was found and inferences, perhaps firm inferences, have been drawn from that which was measured, namely, the deposits, as to what the radiation was?

MR. ROKISON: Well, I don't want to argue that aspect of the case with your Lordship again, of course.

MR. JUSTICE FRENCH: Mr. Rokison, can I say this: it appears to me to matter very little whether the sort of commonsensical observation that error will nearly always lead to an under-estimate, whether it be true or false is going to help me very little.

MR. ROKISON: So be it, I merely challenge that in the context of this assessment of environmental exposure. That error, depending on what one means by error, will necessarily result in an under-estimate, simply because those who have carried out the exercise of making an assessment have made that assessment on the basis of a cautious assumptions which may have to be made - for example, how much local seafood people eat, how much of

it was caught locally - which they have taken. They have assessed it on the basis of assumptions which they believe to be over generous and therefore will result in an over estimate of dose.

MR. JUSTICE FRENCH: If Prof. Evans does not say if the measurements are accurate and if they are cautious, then any error will tend to under estimate. If he says no to that I shall not accept his answer. I think we are dealing with the realms really of commonsense and to explore them with a witness is not going to help me.

You may argue about it afterwards but I don't think exploring it with a witness is going to help me.

MR. ROKISON: Very well. I only do so because the witness strays into the area in his expert reports, my Lord.

MR. JUSTICE FRENCH: And you have got to follow him into every byway into which he strays?

MR. ROKISON: Not every byway, but it is part of the exercise, not only to test his expertise...

MR. JUSTICE FRENCH: I am not criticising you, Mr. Rokison. I am only indicating that I don't think this line is going to help me.

MR. ROKISON: Very well:

Q. I think you agree that both the areas with which we were concerned, that is, the accuracy of the measurements in the environment, or the assessment of radiation in the environment, and the appropriateness or otherwise of applying risk estimates derived from experience and, in particular, A-bomb data, that you do not claim to have any expertise on which you can assist my Lord in relation to either of those areas?
A. No.

Q. Then I think we can move on. Again, in the interests of saving time, Prof. Evans, I shall leave aside your comment in paragraph 24, which is no doubt as a result of something which you have been told for the purposes of your involvement in this case. Is that right?
A. No. I think I got that from COMARE II, or maybe it is COMARE I? Doesn't that report give the information about what Dr. Jakeman said?

Q. I see.

Q. MR. JUSTICE FRENCH: Whatever it is, you are talking about 440 grammes going up to somewhere about 20 kg?
A. Yes.

Q. I think it is COMARE II, but I wouldn't be sure.
A. It is one of the COMAREs. I think it may be COMARE I, that is my recollection.

Q. MR. ROKISON: I think it is COMARE I. We have looked at it and I don't want to canvass it with you. Can we move on to paragraph 25, please, where you say:

> "The further work that has been carried out...in the statistical modelling of the incidence of leukaemia and non-Hodgkin's lymphoma has served to confirm the genuine nature of the Seascale excess of leukaemia cases."

Do you mean any more there that there was indeed an excess?
A. No.

Q. You say:

> "Draper's review has the most up-to-date overview of the geographical distribution..."

That is a reference to not the latest Draper paper...
A. The Draper book.

Q. The Draper book that we looked at.

MR. JUSTICE FRENCH: What page are we on?

MR. ROKISON: My Lord, we are page 11:

Q. You say:

> "...it is clear from this study that the Sellafield excess is not a statistical artefact..."

May I just ask, and I know there was clarification in the course of your evidence in chief as to what an artefact was, but what do you mean by that in this context?
A. What I mean by that in this context is that had we relied only on the Yorkshire TV programme we might well have been convinced that it was a statistical artefact. By drawing the boundaries of time and space cleverly we can make the artefact appear considerable. If we had only relied on John Urqhart's letter that would undoubtedly be the best explanation.

Q. Yes, I follow. Again, I don't criticise you for this. We have already clarified this. You use the expression "Seascale excess" in the fourth line, but then three lines later you refer to the "Sellafield excess", but we are talking about the same thing?
A. Yes.

Q. Very well. Can we move on to the next part of your report where you deal with other studies and concerning possible excesses round other nuclear sites. You make

the point in paragraph 27 that the nature of the studies being geographical studies means they are perhaps a bit crude and may not help very much in finding what the causes may be?

A. Yes.

Q. Cook-Mozaffari. This is a large study which we find in Bundle C, 42. Was this the study you were referring to in relation to the age group?

A. No. There was a separate paper that I think was in the British Journal of Cancer.

Q. Published after this? Cook-Mozaffari, 1989?

A. Yes.

Q. Well, we will come to this. However, in relation to the point about age group, I wonder if you would be kind enough to look at page 36, paragraph 4.7? You see "Age-sex groups selected for study"?

A. Yes.

Q. If you move three paragraphs on you will see:

> "Tabulations of SRRs or SMRs have been made for the age-groups 0-9, 0-24, 25-74 and over...The fifteenth birthday is the usual upper limit for studies of cancer in children. In the present study the grouping 0-24 has been preferred, partly because deaths are being studied as well as incidence and tumours that were incident before the fifteenth birthday may cause death during the following decade, and partly because, in the YTV programme that reported an excess of cancer in young people in the vicinity of Sellafield, the age-group 0-24 was studied. However, there is some evidence to suggest that malignancies associated with irradiation <u>in utero</u> appear largely during the first decade of life..."

That is why they have taken 0-9. So as far as 0-24 is concerned, it was partly, even in this study, as a result of the YTV programme, as you see.

Could you look at page 235 please? What they are doing in that Appendix is giving the reference to the distance zones which have been used for the purposes of the various installations?

A. Yes.

Q. What one finds is that for British Nuclear Fuels at Sellafield at the top, then the distance zone, distance zone 2 is Ennerdale?

A. Yes.

Q. And the control they have chosen for Ennerdale is Castle Ward. Then one finds that Whitehaven is distance zone 3 and Millom is distance zone 4?

A. Yes.

Q. One finds on the previous page the definition of the terms used, the distance zones are:

> "1. Districts with at least two thirds of the population within 6 miles.
>
> 2. Districts with at least two thirds of the population within 8 miles, but not within 6 miles."

And so on?
A. Yes.

Q. Perhaps we can then look at a couple of tables? Page 178, which is part of Table 3, which is leukaemias. At the top one finds "Incidence" and at the bottom one finds "Mortality". If you look at, for example, BNFL Sellafield in the Mortality table, in the lower half of the page, you find the installation first and then the control referred to?
A. Yes.

Q. As it is set out, there is nothing that has been selected for within 6 miles, but then zone 2, which is between 6 and 8 miles, that is Ennerdale, then one finds that zone 3 is Whitehaven. Then one finds Millom. This demonstrates - it is a picture we have seen before reflected in the studies which have been summarised in Black, is that one finds for Ennerdale there is a deficit or deficiency compared with its control?
A. Yes.

Q. For Whitehaven there is a substantial deficit compared with its control?
A. Yes.

Q. Whereas by contrast in Millom one finds a - I don't use the word "significant" in the technical, scientific sense - but there is a substantial excess compared with the control?
A. Yes.

Q. If one looks at malignancies, except leukaemia, on the next page, on page 179, again one finds that zone 3 being Whitehaven, has again only about 50% of the incidence rate of its control area, whereas there is a slight excess in Millom?
A. Yes.

Q. I think it is a pretty complex document and it may be easier in order to see what one derives from it to look at the Summary of this which was summarised in a paper by Forman et al.

MR. ROKISON: My Lord, I think we can put this document away and Forman will be F, number 82:

Q. You refer to this in your report from paragraphs 28 through to 30. In paragraph 30 you say:

> "Despite problems in the design of the study, the results showed that there was evidence for a statistically significant excess of lymphoid leukaemia cases in young people (0-25 years) in the vicinity of nuclear plants, although cancers as a whole were not strongly linked to living near to a nuclear installation. The excess existed although Sellafield was excluded from the analysis."

Sellafield was not excluded from the study, it was simply excluded from 1955 installations for the purposes of that analysis?

A. For one analysis, yes.

Q. If one looks at the Forman paper, and perhaps we can go to page 501 where one finds a reference in the Results section to cancers at ages 0-24. It is the second sentence, page 501:

> "The relative risk for lymphoid leukaemia is significantly elevated in cumulative zones 1 and 2 in the local authority areas grouped around the pre-1955 installations."

One finds these set out in Table 5, and Table 5 is on page 502.

A. My copy is missing pages 502 and 504.

MR. ROKISON: Oh, it has been copied on one side only. Does your Lordship have 502?

MR. JUSTICE FRENCH: No. I have only got two leaves. There should be three, I think.

MR. ROKISON: We will have leave this reference and come back to it tomorrow. There is no point in spending time now getting these additional pages copied. It would appear, your Lordship...

MR. JUSTICE FRENCH: Unless there happens to be a complete one in court?

MR. ROKISON: Well, the witness apparently has not got the even numbered pages either.

MR. JUSTICE FRENCH: If there be a full copy in court and if quickly photocopying it would work, then let that happen. If not, leave it over until tomorrow. I go straight from 499 to 501.

MR. ROKISON: It appears your Lordship doesn't have the even numbered pages.

MR. LANGSTAFF: I wonder if it may assist. We have tracked down another copy in the Forman Report in the references for Prof. Evans, which may well be in court in a red bundle. At Divider 13 there appears to be what is a full copy of the paper.

MR. JUSTICE FRENCH: It looks very like it, Mr. Langstaff. I don't think we have devised a description for these. I suppose Evans 1 Reference bundles - Evans.1 Ref.

Q. MR. ROKISON: Before we look at the paper, may I just draw your attention to page 499? You will see that in the middle column - this is Forman's Summary, he summarises in the second paragraph there the four distance zones which we have looked at in the Appendix to the main study. In the right hand column they refer to "Methods" and say:

> "The principal results, in our opinion, are those relating to mortality at ages 0-24 and 25-74 years for combined groupings...Other data are less relevant because:
>
> (a) Cancer registration data are of variable quality and..."

In particular in the latter part of that paragraph they refer to to the fact:

> "...the ratio of the number of cancer registrations to the number of cancer deaths was higher in the LAAs around pre-1955 installations...which suggests a general registration bias..."

Just to get it out of the way, in the Discussion, as far as cancers are concerned, which we find at page 502 on the right, they say:

> "The comparison of Tables 2 and 4a with Tables 3 and 4b shows that the SMRs for LAAs in the vicinity of nuclear installations or in the 58 selected coastal LAAs are significantly less than the SMRs in their control LAAs more often than the reverse. This, moreover, remains true if attention is concentrated on those types of cancer that have been particularly associated with exposure to ionising radiation, namely, leukaemia, bone cancer and multiple myeloma. This provides strong evidence that there is no generalised increase in cancer mortality around nuclear installations in England and Wales either in young persons or in adults."

Do you see that?

A. Yes.

Q. I am just wondering how that fits in with your summary of saying that cancers as a whole were not strongly linked to living near a nuclear installation?

A. I think in regard to that particular paper... Where am I in my report?

Q. Page 13. It is half way through the first line. I was just getting cancers out of the way. You refer to

lymphoid leukaemia cases in young people and then you say:

> "Although cancers as a whole were not strongly linked to living near a nuclear installation."

A. Yes. I perhaps should have written that as, "not linked at all."

Q. Yes. They make it clear here that nobody is suggesting that living near a nuclear installation gives any form of protection. However, on the other hand they make it clear - they put it quite strongly:

> "This provides strong evidence that there is no generalised increase in cancer mortality..."

They make it clear that that also includes if one is looking at those cancers which are associated with exposure to ionising radiation?

A. Yes. I think one of the things that they themselves point out is that the control areas do not seem to have adequately controlled, because the rates are lower than they should have been in those areas.

Q. Indeed, we will come to that in a moment. That is the point which they deal with and is later clarified in relation to the apparent excess in the one type of leukaemia, namely, lymphoid leukaemia in young people. However, as far as cancers, including leukaemias generally are concerned, they are making the point there appears to be no general excess at all? It is not just that it is not strongly linked.

MR. JUSTICE FRENCH: Well, the Professor has already said that it should read, "not linked at all."

Q. MR. ROKISON: They make the point again in the Summary on page 505, where again they put it in very strong terms near to the bottom of the left hand column when they say:

> "These data show conclusively that there has been no general increase in cancer mortality in the vicinity of nuclear installations in a 22-year period beginning several years after the opening of the installations that have released the largest amounts of radionuclides to the environment."

Is that something you had picked up from this paper?

A. Well, I was picking it up in the context of that and the following paper that had Cook-Mozaffari as a first author.

Q. Yes, which we will come to.

A. I have to confess I was looking at all three together, in a sense.

Q. The excess which one finds in young people of lymphoid leukaemia, appears from Table 5? Is that right?
A. Yes.

A

Q. One finds there it is zone 1, isn't it?
A. Yes.

Q. It is restricted to zone 1?
A. No, I thought it also included zone 4.

Q. It doesn't look as though it is from zone 4, does it?
B A. Table 5?

Q. Yes. If you look at zone 4, lymphoid leukaemia, pre-1955, you will find the standardised mortality rate of 91.29 with a relative risk of 0.94?
A. I am sorry, you are referring to just pre-1955

Q. Yes. I thought that was the point that was being made.
C A. Yes, but I thought you were referring to all of them, all CEGB, Winfrith and Sellafield.

Q. Oh, all installations? No, what I was referring to is the point that was being made, which I referred you to at 501 in the left hand column near the bottom, where they make the point that the relative risk for lymphoid leukaemia is significantly elevated and you refer to that
D as being a "statistically significant excess"?
A. Yes.

Q. In cumulative zones 1 and 2, grouped round pre-1955 installations. If you got to the table at Table 5 what one finds is that the only significant increase is in zone 1 pre-1955?
E A. Yes.

Q. For other zones your relative risk is not only not significantly raised, it isn't raised at all?
A. No, the SMRs are for 2 and 3, but they are not significant.

Q. The SMRs are, but the relative risk relating to your
F control areas are not?
A. Right.

Q. In no case is the increase significant?
A. Quite.

Q. Except for zone 1, pre-1955?
G A. Yes.

Q. If you look at page 503, and I think this is the point you had in mind a moment ago, where in the right hand column on page 503, about a quarter of the way down:

> "It should be noted that the excesses of lymphoid leukaemia in the LAAs around the pre-1955 installations depend, in large part, on particularly
H low SMRs in the control LAAs in zone 1."

A. Yes.

Q. Because what they are doing is comparing the installation with its appropriate control as set out in the main paper?
A. Yes.

Q. "Thus the combined SMR for the age group 0-24 years in the LAAs in zone 1 around the pre-1955 installations is 113.4, whereas that for the corresponding control LAAs is 54.1. For the age group 0-9 the corresponding SMRs are 113.7 and 28.8."

So the statistical significance is something which derives more from the low SMRs in the control than the high SMRs in the installation areas?
A. Yes.

Q. I think that that is a point which is subsequently dealt with in the later report. Is there anything else you seek to derive, Prof. Evans, from that report and this Summary?
A. From this report, or the later Cook-Mozaffari paper?

Q. Well, this one at the moment.
A. No.

Q. Can we just look finally in the Summary at 505 before we leave it, where in the middle paragraph what they say is this:

"Detailed examination of the few types of cancer that were relatively more common in the installation areas suggests that several of the differences were most likely to be due to chance, diagnostic artefacts or social factors rather than to any hazard specifically related to the installations."

Then they say, and I should point this out to you:

"One disease provides a possible exception: namely, leukaemia in the age group 0-24."
A. Yes.

Q. Shall we now look at the later paper which you refer to in paragraph 31?
A. Yes.

Q. That is the Cook-Mozaffari paper, 1989. It is C44. I think it has the same authors basically, but they have shuffled their names about. Let us see what you say about that in paragraph 31. You say that:

"... the researchers re-analysed the data for leukaemia and other cancers using a more conventional form of analysis using the rates in England and Wales as a whole .... "

So they do not have this difficulty about the local authority areas' controls, and you say that they also took account of a number of matters which you there set out, and :

> "The results confirmed those seen in the earlier study i.e. that there was an excess in leukaemia cases in the 0-25 year old population living within ten km of nuclear plants as compared to the rest of the UK."

Just pausing there, it is only a very small point and it does not really matter very much, but it is not that they were living within 10 km it is local authority districts which had more than 0.1% of their population living within 10 miles?

A. Yes.

Q. That was a fairly large area and they divided that, not in the same way as they divided it before, but they divided that into again four categories, didn't they?

A. Yes.

Q. Which you find if you turn to page 482, it is a convenient way of seeing it, in Table 5, where you see that they have districts with at least 0.1% of their population living within ten miles and then they do 0.1-9.9, 10-65.9, and at least 66%, so that unlike the other one, it is rather confusing, but in fact those who had the larger percentage of their population living within the 10 mile zone is in fact the last of the areas rather than the first?

A. Yes.

Q. One sees they start by referring to:

> "Reports of an increased incidence of leukaemia in young people in the vicinity of certain nuclear installations have caused concern ...."

and so on, and perhaps we can move on the page 479, where after they have dealt with the variables which they have taken into account, things like socio-economic classes and so on, one finds page 479, bottom left, refers to "Variation in risk in the vicinity of nuclear installations", and they refer to Table IV, and this is for all the regions, in other words, all those comprised within Table V, so it is those with at least 0.1% of the population within 10 miles, and they give values, as one sees from 479 bottom left, without adjustment and then with adjustment for all the factors that they have referred to. This is mortality, relative risk of death, and one finds in ages 0-24 that if they exclude Copeland, which is the Sellafield, one finds that the unadjusted leukaemias of all types is a relative risk of 1.11, and an adjusted risk of 1.14. If they include Copeland it is 1.12 and 1.15. Lymphoid leukaemia, one gets 1.16 and 1.20; 1.16, 1.21. The P values are there set out and

they are all significant by relation to their P values, is that right?

A. Yes.

Q. But if one looks lower down one finds that they are doing leukaemias, lymphoid leukaemia and other leukaemias, so those two together would make up the leukaemia cases, correct?

A. Yes.

Q. One finds that although there is a lower and slight excess in other leukaemias they are not statistically significant?

A. No.

Q. And one does find, interestingly enough, that Hodgkin's disease in the adjusted figure has, I think, the highest relative risk of all, is that right?

A. Yes.

Q. Although its P value is pretty marginal?

A. Yes.

Q. I think you will agree that Hodgkin's disease is believed to have no radiation relationship at all?

A. That is correct.

Q. One finds, as far as other lymphomas are concerned, there appears to be no excess?

A. That is correct.

Q. That would include, would it not, non-Hodgkin's lymphoma

A. I am not sufficiently familiar with the way they have built the breakdown there.

Q. They are doing all lymphomas, Hodgkin's disease, which is Hodgkin's lymphoma and other lymphomas - one assumes that other lymphomas are non-Hodgkin's lymphomas?

A. Among others, yes.

Q. Well, maybe, maybe not, but one finds that there is no excess at all in relation to that category. The discussion on that is on pages 478-479 which I think does no more than summarise what we see in the table. They refer at the bottom of page 479, the right-hand column, to Table V and they make the point, over the page, about a dozen lines down:

> "For none of the three categories with districts in more than one zone is there evidence of a significant trend in RR with increasing proportion of the population living within 10 miles of an installation."

They refer to lymphoid leukaemia and so on in the next paragraph and say:

"Neither for the four categories nor for the individual installations is there any indication of trend ...."

and one sees that from Table V where one finds, for example, "All installations", that there is actually a lower relative risk in relation to those with more than 66% of their population within 10 miles of the installation, there is a lower relative risk to that which you get with at least 0.1. That is the point they are making, is it?

A. I am afraid I have lost you.

Q. Have you got Table V?
A. Yes, I am sitting looking at Table V.

Q. If you look at "Leukaemia - all types ages 0-24", which is the left-hand part?
A. Yes, you just are referring to the first two lines?

Q. I was simply referring to the first two lines, the "All installations", and it is a pattern that we see in relation to other columns as well, but if one looks at "All installations", or "All excluding Sellafield", then one finds - ignore Sellafield for the moment - "Other pre-1955 installations", and what one finds is that it is the relative risk for those districts which have at least 66% of their population living within 10 miles is actually lower than the relative risk for those who have at least 0.1% of their population living within 10 miles?
A. Yes. There are no standard errors that are given to these.

Q. That is right. But insofar as one finds any trend ....
A. There is no trend.

Q. There is no trend. Insofar as one can recognise any trend at all it seems to be a trend in the direction which is not that which one would expect?
A. I think it very much depends on the pattern of living around an installation. That is the difficulty with using this geographical type of study. The trend you have is that living at all is leading to relative risks that are greater than 1. That is the trend that I notice.

Q. Is that a trend or simply a statement?
A. It is a statement if you like but all the installations, and all excluding Sellafield, have relative risks for leukaemias, all types, aged 0-24, wherever you are. If you were then to look at districts with no one living near or close to an installation then you would find a value of something like, let us say, 0.9 or something of that kind, and what is the interesting thing there to me is that all of them are showing a rise in relative risk. I would not want to make a great deal about it but it seems to me that you are trying to make a great deal about it in the other direction.

Q. I am not trying to make a great deal about it. Forgive me, I am not trying to do that at all. What I am merely seeking to do is to look with you at the study to see to what extent it supports the summary or conclusion or what you say about it. In the discussion they consider the reasons for the excess, at page 481, bottom right, where they say:

> "Several explanations of the increase in leukaemia in the vicinity of the nuclear installations are possible. First, it may be due to local environmental pollution by radiation. Against this explanation are the current assessments of annual radiation doses which, with estimates of the risks of leukaemia per unit dose, together imply that the doses received by populations living in the vicinity of nuclear installations are far below those that would cause any detectable increase in incidence."

- and they refer to Hughes and Roberts, Stather, and Darby and Doll -

> "The present data, moreover, fail to provide support for this explanation in two ways: no trend in relative risk is observed with increasing proximity to an installation as measured by the trend from low to middle to high-proportion zones ...."

and that is the point we have just looked at, and also they point to the fact that:

> "... the difference in excess risk between the district round Sellafield and those around the other installations is less than a factor of six ...."

and refer to the fact that the doses around Sellafield are higher than other comparable installations, or other installations. Then they deal with the second possibility, some other factor characteristic of the nuclear industry, which they say:

> "... cannot be investigated by geographical studies alone ...."

with which I think you would agree?

A. Yes.

Q. Then they raise the possibility that:

> "... the districts close to nuclear installations differ from those elsewhere in some other characteristic that is relevant to the aetiology of childhood leukaemia. That this should be so seems unlikely ...."

because they have made adjustments for such matters?

A. Yes.

Q. They go on to say:

> "Nevertheless, the causes of different types of cancer differ greatly and it is possible that there is some other factor that influences the incidence of childhood leukaemia that is not allowed for by these adjustments ...."

They refer to the fact that in relation to:

> "... the tendency for a higher mortality from leukaemia in young people in districts with relatively high proportions of their populations in social classes I and II this deserves further investigation partly because in Seascale, near Sellafield, where an increased mortality from childhood leukaemia was first established, the proportion of the population in social class I was most unusual namely 47% of the economically active male population ... compared with 5% nationally ...."

Is that a matter which you have weighed in the balance or considered in your consideration of the excess at Seascale?

A. Yes. It obviously is a possibility that the excess in Seascale is due to social class differences alone. However, all the studies that we have demonstrate that the gradient with social class is really quite small. If you look at Table III of this paper, on page 479, you will find that leukaemia, all types, and lymphoid leukaemia, are actually fractionally lower in social class I than they are in social class II, but if we combine I and II they tend to be a little bit higher rates than social classes III-V, so there undoubtedly is a social class I/II difference compared with other social classes and that is a factor that I do not think, and I think others who have studied it subsequently, explains the excess there. We certainly do not see social class factors giving rise to relative risks of 3 and 4.

Q. No. We will come to the Kinlen and Alexander papers, which I think you have looked at, a little later on but it is the fact, and it is something that you have observed, that Seascale is not just of a high social class population but it is extremely unusual?

A. Extremely unusual.

MR. ROKISON: I think we can now leave this study and it may be a convenient time for your Lordship to rise for today and we can come back and look at some more studies tomorrow.

MR. JUSTICE FRENCH: Yes, 10.30 tomorrow.

(Court adjourned until the following morning at 10.30 a.m.)