IN THE HIGH COURT OF JUSTICE

QUEEN'S BENCH DIVISION

1990 R No 860
1989 H No 3689

ROYAL COURTS OF JUSTICE
THE STRAND
LONDON

Friday 4th December 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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EIGHTEENTH DAY'S PROCEEDINGS

FRIDAY, 4TH DECEMBER, 1992

MR. JUSTICE FRENCH: Mr. Langstaff?

MR. LANGSTAFF: My Lord, I thought we had better tell your Lordship the present plans, the present intentions as to witnesses before Christmas.

MR. JUSTICE FRENCH: Yes.

MR. LANGSTAFF: My Lord, it is a matter which has been under review over the last day and a half and the parties have been in contact. As your Lordship knows, it had been intended to call Prof. Savitz before Christmas and, to that end, dates had been allotted him on what seemed to be the likely timetable. Things being as they are, and more often than not perhaps are, that timetable has not been adhered to as we might have hoped and so the original dates have had to be abandoned and Prof. Savitz is now available to come on 20th and 21st January of next year.

My Lord, if by then the rest of the Plaintiffs' epidemiological witnesses have given their evidence and have been cross-examined, I understand it will cause my learned friend no difficulty. Prof. Savitz deals with what might be called a discrete area. The question that he addresses is that of analogy, and he is the only witness who does so from our side.

My Lord, it would seem likely that on Monday your Lordship will hear from Dr. Scott Davis. He is likely to take, we think, a little more than two days, probably three, although it is unlikely to be more than that, and will be followed by Dr. Kopecky. My Lord, he is likely to be a little shorter, I suspect, because he covers much of the same territory, although from a slightly different perspective.

My Lord, it looks as though Wednesday will be the reading and preparing day next week, subject to any comments that your Lordship might have.

MR. JUSTICE FRENCH: Dr. Savitz may well straddle that day.

MR. LANGSTAFF: Dr. Scott Davis, my Lord, yes.

MR. JUSTICE FRENCH: I am so sorry, yes. I beg your pardon. Dr. Scott Davis may well straddle that day.

MR. LANGSTAFF: My Lord, it is likely that Dr. Kopecky will, therefore, go into the last week before Christmas. My Lord, in that week the Plaintiffs would

hope to call Prof. Alberman and to recall Prof. Evans, subject to his availability, which I have not had an opportunity of discussing with him, so that his evidence is complete before Christmas.

MR. JUSTICE FRENCH: So you are hoping to fit in Dr. Alberman and, shall I say, Prof. Evans, slice 2?

MR. LANGSTAFF: My Lord, yes. My Lord, it looks to us as though that is as far as we are likely to go with witnesses before Christmas. The other witnesses that we would have would be too long to fit in without straddling the Christmas vacation, and plainly that is undesirable.

MR. JUSTICE FRENCH: Yes.

MR. LANGSTAFF: My Lord, one of them, Prof. Duncan Thomas, comes from North America, so plainly it is undesirable to bring him over on a wing and a prayer, as it were, and Prof. Day had, in any event, been scheduled for next term.

MR. JUSTICE FRENCH: So Duncan Thomas, Day....

MR. LANGSTAFF: And then Savitz next term.

MR. JUSTICE FRENCH: Of course, I can only rely on your informed view, which necessarily must be better than mine.

MR. LANGSTAFF: My Lord, yes.

MR. JUSTICE FRENCH: So I am content with that. Have you any observations?

MR. ROKISON: My Lord, I am content. May I just mention two things in relation to it because I want to get on as quickly as possible, my Lord.

Firstly, we were - I do not say we were promised - we were told that we would be getting Prof. Duncan Thomas's report on the new figures on Wednesday. I simply say we have not got it yet. Both sides have difficulties. I am not saying it was a promise and I am not complaining. All I am saying is that, to some extent, my ability to complete cross-examination of Prof. Evans in relation to the further figures may be influenced by what Prof. Thomas seeks to build upon them. So I simply say, through your Lordship, that we are very anxious to get that as soon as possible.

MR. JUSTICE FRENCH: I suppose, although it has its undesirabilities, it is something that would not be disastrous if it were left until slice 2.

MR. ROKISON: If what were left until slice 2, my Lord?

MR. JUSTICE FRENCH: The cross-examination that you speak of upon the figures.

MR. ROKISON: No, it is going to be left until slice 2.

MR. JUSTICE FRENCH: Oh, I see. You are looking that far ahead?

MR. ROKISON: Oh, indeed, yes, I am, because we have no idea what it will say. So I merely put in that caveat.

MR. JUSTICE FRENCH: You are putting in a plea?

MR. ROKISON: It is not a plea yet. It is simply a caveat. Prof. Evans said this morning to me that he would be not very happy if his evidence were to be held over until next term and, with respect, I would entirely agree and we will co-operate, subject to my learned friend wanting to call other witnesses at other times, in order to try to ensure that Prof. Evans' evidence is finished this term, but that must be subject to the fact that I am, by that time, in a sense, fully briefed so that I can absolutely finish it and that will be that. So I simply put down that marker, if I may.

The other thing is we have been considering it, my Lord, and, all other things being equal, I think that the parties would appreciate perhaps the reading day in the last week of term being the Monday.

MR. JUSTICE FRENCH: Yes. I will not....

MR. ROKISON: Which happens to be the last day of term, of course! (Laughter) That really knits in with what my learned friend said. If we can finish whoever is the last witness by the end of the previous week, then I think neither of us see any point in bringing another witness in, especially one from across the Atlantic for a day's evidence, which will not be completed.

MR. JUSTICE FRENCH: Yes. On the other hand, if there is a prospect of finishing a witness on the Monday, I shall not regard that as a reading day.

MR. ROKISON: No, my Lord, I feared as much! Thank you, my Lord.

MR. JUSTICE FRENCH: So what we are looking to, on the balance of probabilities, if I can use that expression, it is likely to be this coming Wednesday that is the reading day for next week?

MR. ROKISON: Yes, my Lord.

MR. JUSTICE FRENCH: Thank you.

STEPHEN JAMES EVANS Recalled:

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

Q. MR. ROKISON: Prof. Evans, may I just tell you which topics we have got to deal with and tell you what my timetable is for this morning to try to finish you by lunchtime? I have got to deal with McKinney with you further. I have also to deal with the Scottish Urquhart study. I have to deal with the Louise Parker paper and Dr. Wakeford's first statement and I have to deal with the Draper paper and Dr. Wakeford's second statement, and I also want to ask you a few questions about Kinlen and Alexander, the viral theory which you mention in your report. So we have got five topics effectively and, if I want to finish by lunchtime, we have got five periods of half-an-hour and so I shall get on my starting blocks now!

Before I come back to McKinney, may I just ask you one more question, which I ought to have raised with you yesterday when we were considering the Gardner study, and that is simply this: that one notices in the Gardner study that - and I am looking now at the Results paper, G 88, I think - that we see from Table II that there was a significant association, so far as both leukaemia and leukaemia and NHL are concerned, with maternal age being equal to or greater than 40?

A. Yes.

Q. And it was area controls only for leukaemia only; area and local controls for leukaemia and NHL?

A. Yes.

Q. Where one has some finding such as that, the question arises as to what, if any, adjustment one should make in relation to testing another hypothesis specifically for the possible confounding influence of that association?

A. Yes.

Q. Do I express it accurately?

A. Yes.

Q. You certainly, as I read your second report, considered that would have been a proper thing to do?

A. Yes.

Q. In paragraph 31 of your second report - and I do not want to take you through it now - but you do that exercise in paragraphs 31 and 32 and you show there the extent to which it affects the P values of your regression slopes?

A. Yes.

Q. Of course, I am going to deal with the up-to-date figures in the next tranche?

A. Yes.

Q. But would I be right in thinking that that is not a matter you have taken into account for the purposes of your fourth report and your final figures, or have you done that adjustment?
A. It is not in the fourth report, no.

Q. Third report, I am sorry?
A. Third report.

Q. Third report. It is not in that?
A. It is not in that.

Q. You have not done it?
A. No.

Q. Is it right to say, although as we have seen in another study - for example, the big Cook-Mozaffari study - they took into account potential confounding factors?
A. Yes.

Q. That it is not something that Prof. Gardner did in his study?
A. He did not, no.

Q. Would you agree that it would be better, particularly in view of the fact that it appears to be a statistically significant association, to have done so?
A. No, because I think that the categorisation of the data is what is leading to the confounding and not a continuous term, which is my argument in paragraph 32.

Q. I can see that you say, and you have told my Lord, that your opinion now is that the regression slope is the right way to do it?
A. Yes.

Q. And that, despite the fact you thought Gardner had done as good a job as he could, he presented his data in a way which was not the best?
A. No, it may be the best for communicating to epidemiologists and the general public, but there is a distinction between the best detailed analysis and that which is the best for communication. Most people have difficulties with regression slopes.

Q. Yes, I accept that.

Q. MR. JUSTICE FRENCH: Can I pause? There was a longish answer, which was followed immediately by a question, and I am not criticising that, but I would rather like to record it. The long answer related, I think, to paragraph 32 and the potential confounding factor?
A. Yes.

Q. Is that enough for you to repeat it, Prof. Evans?
A. Yes. In paragraph 32 I looked at age as a potential

confounding factor when it was used as a continuous value. I did not find it to be a confounding factor.

A

Q. Thank you. Is that complete?
A. That is complete, yes.

Q. MR. ROKISON: You say you did not find it to be a confounding factor. Do you mean by this that you did not find that that was the true association, as opposed to the parental preconception dose? Is that what you mean by saying you did not find it to be a confounding factor?
B
A. No.

Q. What do you mean by saying you did not find it to be a confounding factor?
A. A confounding factor.....

Q. MR. JUSTICE FRENCH: Take this steady, if you would?
C
A. Yes.

Q. By not finding it to be a confounding factor....?
A. A strong confounding factor, can we....

MR. ROKISON: Now I understand what you are meaning.
D

Q. MR. JUSTICE FRENCH: Would you continue, nonetheless?
A. Sorry, can you repeat the beginning of my sentence, my Lord? I am sorry.

Q. I have amended the first answer. "In paragraph 32 I looked at age as a potential confounding factor when it was not used as a continuing value. I did not find it to be a strong confounding factor." Right?
E
A. Yes.

Q. Then it goes on. "By 'not finding it to be a strong compounding factor' I meant...."?
A. I meant that it was not significantly associated on its own with risk of leukaemia.
F

MR. LANGSTAFF: My Lord, before your Lordship passes on, when your Lordship read out the answer that your Lordship had recorded, at the beginning of the first answer, "In paragraph 32 I looked at age as a potential confounding factor when it was used...." I think your Lordship has "when it was used not as a continuing term," and I had understood the witness to say "when it was used as a continuing term".
G

MR. JUSTICE FRENCH: What I wrote was "when it was used as a continuing value".

MR. LANGSTAFF: My Lord, in that case, I stand corrected.
H

MR. JUSTICE FRENCH: I may have misread it back, I do not know. Probably not. Yes?

Q. MR. ROKISON: As I understand your paragraphs 31 and 32, what you found is that, if you made an adjustment for that weak confounding factor, on your answer - yes?
A. Yes. Shall I try and tell you what a confounding factor is? That would be a help.

Q. Yes, if you like?
A. A confounding factor is one that is associated both with the outcome of interest and with some other risk factor, so it requires that maternal age itself is associated with the risk and that the maternal ages of the cases is different from that of the controls.

Q. Yes, but that is shown in Table II. What Table II shows you is that there is a statistical association between being a leukaemia case, if you like, and having a mother who was more than 40 years old when you were born?
A. Yes, but that is as a result of categorising it at age 40, which is entirely arbitrary.

Q. I see that and, of course, the way in which you introduce that potential confounding factor and make allowance for it may depend upon whether you are taking a regression slope and then you would consider maternal age as a continuing adjustment to your continuing regression slope?
A. Yes.

Q. If I can put it that way?
A. Yes.

Q. Whereas, if you were doing it by categories, you would make an adjustment, in a sense, category by category?
A. Yes, but I think that is a weak way of doing it.

Q. I appreciate that, but all I am putting to you is this, that when you did it on a continuous basis, although it did not deprive your P value of statistical significance, it did have some noticeable effect on your P values
A. Yes, but then I could enter perhaps the colour of hair of the mother and that might have the same effect.

Q. It might, but that was not something which was introduced as a potential confounding factor. Maternal age was considered because maternal age is sometimes related to diseases of children?
A. It is.

Q. Without being more specific than that, and that is why it was one of the hypotheses that was tested?
A. Yes.

Q. You having made that adjustment in paragraphs 31 and 32 on a continuing basis to your regression slope?
A. Yes.

Q. First of all, do you not agree that it would perhaps have been better if Prof. Gardner had made such adjustment as should have been made - and that will depend on the appropriate calculations - to his dose category assessments?

A. No, I do not think that is necessarily true.

Q. What I do not understand, and it is, I am sure, my shortcoming, is, if it is appropriate to do the exercise if you are looking at a regression slope and looking at it on a continuing basis, why it is inappropriate to do it if you decide to assess and present your data categorically?

A. I am not saying that it is inappropriate to do it. I am not saying that it is a requirement that you do have to do it. In fact, Gardner, I think, discusses the fact that he has not adjusted and, having said that he has not adjusted, this is acceptable. Papers published in the literature are not necessarily exhaustive. You cannot necessarily cover every possible thing, otherwise papers would become very, very much larger and editors' pressure on you is to publish the simplified version.

Q. MR. JUSTICE FRENCH: They might be less read?

A. And they might, indeed, be less read.

Q. MR. ROKISON: But they might be less accurate?

A. They may be.

Q. Of course, this was not a paper that was being produced for publication as such, although, of course, it was published, but it was a study that was carried out pursuant to the recommendation of the Black report?

A. Yes.

Q. We do not make a strong point of this, Prof. Evans, but those instructing me suggest, and I suggest to you, that where one is dealing with pretty marginal results, then where you have a statistically significant association with maternal age, it would be appropriate to make whatever adjustment arises from that because it could have an effect on the overall picture?

A. I think if you had some idea that age 40 was very important, you might, but you might also look at the fact that in the McLaughlin study maternal age was entirely unimportant and this might be one of those chance findings.

Q. It might be, but it is a finding. I quite accept, as you say, here you have a statistically significant association.

MR. JUSTICE FRENCH: What, between maternal age and leukaemia?

MR. ROKISON: Yes, my Lord.

Q. MR. JUSTICE FRENCH: Do you accept that proposition?
A. I accept it when you have categorised it at age 40. If you do any other categorisation, or virtually any other categorisation, you do not find it to be so.

Q. If you categorise - categorise what?
A. A mother's age.

Q. If you categorise a mother's age at age 40?
A. Yes, using a boundary of age 40.

Q. Using a boundary of age 40, the result is statistically significant?
A. Yes.

Q. MR. ROKISON: To take age 40 is not arbitrary in the same way as dose categories, is it, because the age 40 is quite often taken - at or around 40 - as being an age which may make a difference between healthy offspring and an offspring which develops disease?
A. I know of no biological situation which has a step change at age 40.

Q. That is not actually what I asked you. What I said to you is that the age of 40 is often taken as being an age where it is said there may be an increased risk of certain diseases?
A. That may be.

Q. MR. JUSTICE FRENCH: As something you know, is it often taken within your knowledge?
A. I would not say often, but certainly taken, yes. It is sometimes 35 and sometimes 44.

Q. MR. ROKISON: You said it as "only if you take the division at 40" there is any statistical significance. Have you actually done the exercise to show that that is the case or is that purely a guess on your part?
A. If I turn to page 424 of the Gardner results paper, on the right-hand column, around the middle of the paper, it says:

> "Relative risks around unity were also found for maternal age at birth of 25 or older compared with under 25. For mothers of 40 or older, however, when examined in a comparison of all age groups - that is, <25, 25-29, 30-34, 35-39, and >40 years - relative risks were about 4."

Q. Yes?
A. So it is only the above 40 where there is any raise. 35-39....

Q. No, what it shows is that, if you compare mothers above 40 with any of the other age groups, whether it be below 25, 25-29, 30-34 or 35-39, you will find that there is an enhanced relative risk of about 4?

A. Yes.

Q. To say, as I understand you are saying, "Oh, well, supposing you were to compare mothers of, say, 38 and over with all those other categories, there would be no excess, or no statistically significant excess." You cannot say that, can you?

A. I can say that it is true for 35.

Q. No, you cannot, can you?

A. Because I did it. That is my recollection.

Q. Oh, I see, you did it?

A. I believe that I did that thing because I read what he said there and I examined the maternal age, but it was clear that it was.....

Q. Is it not somewhat extraordinary, if you did it at a time when you were looking at the categories, that it is something which you did not mention at all?

A. No, I do not think that is necessarily true.

Q. I see, but the position is, whether it is an arbitrary choice or not, that here, in the highest category which they choose, which is equal to or above 40, one finds a statistically significant result, which shows a fairly consistent pattern, whether you are dealing with area or local controls - there are differences - and you say, "Oh, well, do not take that into account because it is something that probably occurred by chance." What I do not quite understand is why you simply brush that one aside and say, "Oh, well, that is chance, so we do not take that into account, even though it is statistically significant," in circumstances where, in relation to paternal preconception irradiation, as I understood your first report, although not your evidence before my Lord, that provided you have a statistically significant association, it proves causation?

A. I certainly did not suggest that. I would challenge you to find where I have said in my written report that statistically significant associations prove causation. If I did write that, I would be very, very surprised. I certainly do not hold to that view. Can you give me chapter and verse, as you might say?

Q. No, to be fair, that was how we read your process of reasoning in your first report, but we can perhaps leave it there. What I do suggest to you is this, that where you have a statistically significant association with one of the hypotheses you are testing, when you are looking to test the strength of another hypothesis, it is right, it is better, to take the first statistically significant association into account in order to test to what extent it is a confounding factor, whether a strong one, a weak one or not at all?

A. When I began doing the analysis on maternal ages, when I began doing the analyses on the doses, I was entirely open-minded. It was entirely possible, as far as I was

concerned, that maternal age was all of the explanation for the risk and so, when I then looked at it, my own view was, and my pre-specified way of doing it, because I am not somebody who likes presenting data and analysing data in groups - it is beloved of epidemiologists and less so of statisticians - then I looked at maternal age as a continuous term and I then found that it was no longer associated. That puzzled me and that left me slightly surprised, so I then investigated, as far as I was concerned, to see whether I had made a mistake, whether there were some errors and, as you may notice, I studied maternal age such that I did, indeed, find that Prof. Gardner had had a computer program that miscalculated mother's age in some instances.

So I studied it very carefully and I found that there was, indeed, a step change at age 40 and I thought that that was not a sensible biological hypothesis and, in particular, as I had pre-specified that I would analyse by maternal age, that maternal age on its own was not associated and, therefore, by my definition, is not strictly a confounder, but it is possible, I stated, that it is a weak confounder, even though it is not statistically significant.

Q. However, if there is some threshold, if there is some step change, you say that you rejected that as being biologically implausible?
A. Yes.

Q. But if there was such a step change, then it would be, would it, a strong confounding factor?
A. Yes.

Q. MR. JUSTICE FRENCH: Just a moment. It is possible that age 40 plus is a weak confounding factor?
A. No, that maternal age itself....

Q. MR. ROKISON: As a continuous term?
A. As a continuous term, yes.

Q. MR. JUSTICE FRENCH: It is possible that maternal age as a continuum is a weak confounding factor?
A. Yes.

MR. JUSTICE FRENCH: Then there was a further answer.

MR. ROKISON: Yes, the further answer, perhaps I could put the question again:

Q. It would have been a strong confounding factor on the assumption of a threshold or step change at or about the age of 40, but you rejected that as being biologically implausible. Is that right?
A. Yes.

Q. MR. JUSTICE FRENCH: It would have been a strong confounding factor....?
A. I am not totally happy with the word "strong" but certainly a confounding factor.

Q. It would have been a confounding factor had there been a step change at 40?
A. Yes, if there was some biological process.

Q. MR. ROKISON: Assuming a step change or threshold at 40?
A. Yes.

Q. I think you said at about the age of 40?
A. Very close to the age of 40.

Q. MR. JUSTICE FRENCH: A threshold very close to age 40 - maternal age 40. Yes?
A. I think my paragraph 33 puts it as clearly as I would still wish to do - of that second report:

> "In conclusion maternal age does not appear to be a major feature since it does not show a continuous relationship with risk of leukaemia. It is also not widely acknowledged in other studies to be of importance and I know of no prior hypothesis that age >40 (but not age >30) would be a particular risk factor for leukaemia in subsequent children."

Q. MR. ROKISON: Then you go on:

> "It may be noted that total dose will be likely to be slightly confounded with maternal age so this reduces the overall impact."

A. Yes.

Q. So that, even as a continuous factor, if you make the adjustment for the association which was found in the study population, then it slightly reduces the overall impact of the hypothesis which is being put forward as the principal hypothesis?
A. Yes.

Q. We can leave that then, thank you, and I would like to come back - I apologise for having omitted that yesterday and, my Lord, for taking it rather out of turn, but I thought it best to get it out of the way. Can we come back to McKinney and try to clear that now? May I just remind you, and you may have looked at yesterday's transcript to see, in a sense, where we had got to with McKinney.

MR. ROKISON: I do not know whether your Lordship has M 172, my Lord.

MR. JUSTICE FRENCH: I have got Day 17. Page?

MR. ROKISON: My Lord, where we had got to at the end, at 81 at G, we were looking at the McKinney paper and your Lordship may recall that the tables are set out in a somewhat confusing way and I am afraid that some of my questioning fell into the trap of confusion, as, indeed, I think we were in fieri delicto, Prof. Evans, in that respect, but I think we sorted it out in the end.

MR. JUSTICE FRENCH: So I have got to get Common Bundle M 172, did you say?

MR. ROKISON: Yes, my Lord, it is.

MR. JUSTICE FRENCH: Do I need the transcript as well?

MR. ROKISON: It is simply this. The position is....

MR. JUSTICE FRENCH: Yes or no. Do I need it?

MR. ROKISON: Yes, my Lord, please, unless I were simply to read your Lordship one answer. It is just where we had got to. At 81 G we had agreed that, of the certain ionising radiation cases which come within the 15 who had a preconception radiation exposure, at the bottom left-hand side of Table III - of those 15, there were four cases, as appears from Table V, or within those that come within certain ionising category of that 15, there are four cases. So, of that 15, it is made up of either ionising or non-ionising and it is made up of categories of certain, possible, unlikely.

There are only four cases which are certain ionising and of those two of them were Gardner cases and therefore there were two of them that were not. There were certain ionising cases in addition to Gardner, one being a Gateshead and Humberside case and the other being not a Gateshead and Humberside case, and therefore a West Cumbria case?

A. Yes.

Q. So that if one comes back to the way in which you had dealt with McKinney in your first report at page 24, paragraph 67, just to recap, you have agreed with me that the last sentence was in error in that the statistically significant relationship was not found between preconception exposure to ionising radiation and leukaemia, but it was found between exposure to all forms of radiation, ionising or non-ionising, and it included categories of the ionising exposure being stated to be unlikely?

A. Yes.

Q. As well as possible?

A. Yes.

MR. JUSTICE FRENCH: I have already amended paragraph 67 to read "exposure of fathers to ionising and non-ionising ..." You are now, as a result of that answer, proposing that I add "ionising, doubtful ionising and non-ionising"?

MR. ROKISON: It was called "possible ionising" and "unlikely ionising".

MR. JUSTICE FRENCH: Ionising, possible ionising and unlikely ionising.

MR. ROKISON: As well as non-ionising.

MR. JUSTICE FRENCH: And non-ionising?

MR. ROKISON: As well as non-ionising, yes.

MR. JUSTICE FRENCH: So all those categories must be inserted after the words "fathers to ionising ..." is that right?

MR. ROKISON: Yes. It is better, with respect, to say "exposure of fathers to radiation which includes those categories, certain ionising, possible ionising, unlikely ionising and non-ionising".

Q. MR. JUSTICE FRENCH: Do you accept that, Prof. Evans?
A. Yes.

MR. JUSTICE FRENCH: Thank you.

Q. MR. ROKISON: The position is that, quite apart from the fact that one only has four certain ionising cases, only two of which are non-Gardner, one has four non-ionising cases?
A. Yes.

Q. But as far as the others are concerned, one does not really know whether they have had any radiation at all?
A. No, but one doesn't know about the non-ionising radiation either.

Q. What we know about non-ionising presumably is that they were exposed to radiation which was non-ionising?
A. We know that. We don't know whether they didn't have any, clearly.

Q. I was saying that as far as the other categories are concerned, possible ionising and unlikely ionising, we do not actually know whether they were exposed to radiation at all?
A. No.

Q. You point out in paragraph 68, very fairly, that no doses were stated, therefore no dose response relationship could be assessed?
A. Yes.

Q. And the study was based on interviews of case parents which is notoriously subject to recall bias?
A. Yes.

Q. Where you say in paragraph 68:

> "In addition the conclusions of the part of the study dealing with preconception exposure to radiation were based on only seven cases, some of which could have been included in the Gardner study"

the position is that it is made pretty clear that as far as preconception only is concerned, of those seven cases you only have two of certain ionising?
A. Yes.

Q. It makes clear, with respect, how many cases and controls within each category were or were not within the Gardner study?
A. It does.

Q. You said in your evidence yesterday that really you relied for support on the letter rather than the report itself?
A. Yes.

Q. Would you agree that just looking at the report itself, you cannot really say that that gives support to the Gardner hypothesis because the non-Gardner numbers of certain ionising are so very small?

MR. JUSTICE FRENCH: I am sorry, if you could take it step by step, it would be much easier for me to record.

MR. ROKISON: It is my fault for not following your Lordship's pen, I am sorry.

MR. JUSTICE FRENCH: If you were to get the answer and then add the "because ...", it makes notetaking rather easier.

MR. ROKISON: I am sorry, my Lord.

MR. JUSTICE FRENCH: It is not your fault.

MR. ROKISON: It is, my Lord, because I ought to be watching to see what it is that your Lordship is noting and what your Lordship is leaving to the transcript.

MR. JUSTICE FRENCH: Never mind; could you break the question down into the preliminary and then the "because ..."?

Q. MR. ROKISON: The first point that I was making, Prof. Evans, was that you said in your evidence yesterday that you really relied upon the subsequent letter to the BMJ rather than this study paper itself?
A. Yes.

MR. JUSTICE FRENCH: Yes, I have got that.

A Q. MR. ROKISON: Looking just at the paper itself, bearing in mind all the points that we have looked at, one cannot really say that this gives any support to the Gardner hypothesis, can one?
A. My phrase was "only marginal support".

Q. Is not even that putting it a bit high, bearing in mind the small number of cases, the doubts about the certainty
B of ionising radiation and the overlap with Gardner? It is really minuscule, is it not?
A. I would still hold to marginal, but somewhere between marginal and minuscule if you really wish.

Q. Very well, I will not pursue that further. Can we then look at the letter to see what difference that makes?
C A. Yes.

Q. That is a letter by Alexander, Cartwright and McKinney?
A. Yes, perhaps most easily found at P4, page 276.

Q. That is very helpful, thank you.
A. Page 275 and 276.

D Q. We start at the bottom on the right where it says "Editor"?
A. Yes, that is page 275 at the bottom on the right.

MR. JUSTICE FRENCH: I have got 275.

MR. ROKISON: That is where it starts, my Lord, where it says "Editor".

E Q. They say:

> "We reported last year an analysis of a case-control study that we had conducted in three areas of northern England (West Cumbria, Gateshead, and North Humberside). Significant associations were found for childhood leukaemia and certain exposures of
F fathers before the affected children were born; these included exposure to 'radiation.' After the analysis was completed, review of the occupational histories reported at interview permitted classification ..."

Then they set out that classification which we have seen in Table V?
G A. Yes.

Q. So it may be that that is why there was the confusion, because they produced Table V on the basis of information which they got after they had produced Table III?
A. Yes.

H Q. "The numbers of fathers exposed to radiation were very small, but this aspect of the study was important because of other contemporary reports".

That is Gardner and Urquhart that they refer to.

> "Gardner et al's results were not published until after our interviews were completed. Thus they cannot have led to any recall bias related specifically to the prenatal period. Nevertheless, there was geographic overlap and the time period and methods of control selection were similar so that our results were not independent of theirs. We have now been able to cross check our entire study population against their data base (which includes more recent cases and controls added since the original report). We excluded all subjects who were present on their data base and analysed all remaining case-control sets that were discordant for paternal radiation exposure (in one of the three time periods) using exact methods of analysis with the statistical package EGRET.
>
> The table provides exposure classifications for the subjects in this analysis. The overall numbers are reduced, as was anticipated; ..."

because they have cut out the overlap with Gardner?

A. Yes.

Q. "in particular, the numbers classified as certainly exposed to ionising radiation have become almost negligible (one case 'exposed' before conception and one control exposed postnatally, both with extremely small lifetime doses)."

Can we just look please at the Table "Exposure of fathers to ionising and non-ionising radiation"? What one finds, if one cuts out the Gardner cases - and really I think this is comparable with Table V, is it not, in the report?

A. Yes.

Q. One finds that there is one case before conception only of certain ionising radiation?

A. Yes.

Q. There are no cases that cover all periods of preconception, periconception and during gestation, so that means that of any period including preconception, there was only one case of certain ionising radiation?

A. Yes.

Q. That case of certain ionising radiation, as they say, had a total lifetime dose of 1.11 mSv?

A. Yes.

Q. Which is a very very small dose, is it not, as a lifetime dose?

A. Negligible compared with environmental exposure.

Q. Indeed. As far as the possible category is concerned, it is slightly confusing, in fact, because you can see that in the description under this they have taken the wrong terminology. In the Table they call it "possible", but in the description underneath they refer to it as "uncertain" - do you see?

A. Yes.

Q. But it looks to be the same category?

A. It does.

Q. "Uncertain" are "others whose job description is consistent with exposure to ionising radiation (industrial radiographers and contract workers at nuclear sites)"?

A. Yes.

Q. So we do not know what, if any, radiation doses those had?

A. No.

Q. If they were industrial radiographers who had substantial doses, then those doses would have been recorded, would they not, at a central registry?

A. I believe they certainly should have been, but I can't say they would have been.

Q. Can I just refer you back, in that respect, to the paper itself? I am sorry to do that, my Lord, but it relates to this point.

MR. JUSTICE FRENCH: Yes.

Q. MR. ROKISON: If you look back to the paper itself at page 682 on the right, two-thirds of the way down, what they say is this:

> "For the 25 fathers who reported radiation exposure in the preconceptional and periconceptional and gestational periods a subclassification of their exposure to ionising radiation was produced after checks with the National Registry for Radiation Workers and British Nuclear Fuels, Sellafield, Cumbria. This was not originally envisaged, but was completed to clarify the study findings. Exposures were recorded as certain for those who had had a total gamma dose according to the national registry or British Nuclear Fuels."

Pausing there, if somebody had a recorded dose either at the National Registry for Radiation Workers or British Nuclear Fuels, then they were put as "certain"?

A. Yes.

Q. If an industrial radiographer had a significant dose, it ought to have been recorded at the National Registry for Radiation Workers?

A. I thought that was precisely what I had said, that it should be; you had said that it would be.

Q. Yes, of course.

A "Exposures were recorded as possible for other contract workers on nuclear sites and industrial radiographers."

So the position is that with regard to those who were possibles, it was not just that they were the industrial radiographers and contract workers, but they would have been the industrial radiographers who did not have a dose B recorded at the National Registry?

A. Yes.

Q. They say:

"This category was chosen after consultation; we acknowledge that these occupations may be incompletely registered by the national registry. C The unlikely category included people who reported ionising radiation exposures in occupational settings, such as education and medicine. The remainder reported non-ionising radiation exposure and included radar and radio operators".

So if one comes back to what we are left with in the letter, we are left with one case of a recorded dose in D any context at all?

A. Yes.

Q. Who has a dose which you agree was minuscule or totally insignificant as against background radiation?

A. Yes.

E Q. And we have got a total of, embracing the preconception period, four cases where radiation exposure was possible but not recorded anywhere?

A. Yes.

Q. Then we go down to the unlikelys and so on. I would respectfully suggest to you that, far from strengthening what one finds in the original report, if one goes F through this exercise of seeing what the categories are and what the dose was of the only certain ionising radiation case, the only one with a recorded dose, it reduces any marginal support which the paper provided to no support at all?

A. No, I disagree.

G Q. What do you say ---

Q. MR. JUSTICE FRENCH: You say that you disagree?

A. I disagree.

Q. Can I ask why?

MR. ROKISON: I was going to.

H

MR. JUSTICE FRENCH: I was perhaps putting it rather more shortly, dare I say!

Q. Why?
A. The last paragraph of the letter, page 276 at the bottom left-hand corner ---

Q. Where are you going to, the bottom left-hand corner, "These results yield ..."?
A. Yes, it is exactly that sentence.

Q. MR. ROKISON: Which one?
A. "These results ..."

Q. MR. JUSTICE FRENCH: "I disagree that the letter reduces support from the study to nil" - that is the effect of the answer, is it not?
A. Yes.

MR. JUSTICE FRENCH: I am writing, Mr. Rokison, "I do not agree that the letter reduces support from the study to nil", and now we are having the because.

MR. ROKISON: My Lord, we are.

Q. MR. JUSTICE FRENCH: What is the because?
A. I agree with the author's statement there that it is a modest, independent contribution.

MR. JUSTICE FRENCH: Yes.

Q. MR. ROKISON: With respect, my Lord asked "Why?" and you say "Because somebody else said so", but what I am asking you to do is actually look at what the results are and not just to accept what the authors say, because authors having produced their report, it might be difficult for them to say "Whoops, now we have checked, this doesn't tell you anything", so they write their letter and they qualify it as hard as they can by referring to it as a modest independent contribution which must be interpreted with extreme caution, with the numbers being very small, with almost none of the exposures having been validated, etc. But I am asking you modestly to apply yourself independently to the data and to the results, and I am suggesting to you that although they qualify that first sentence to a substantial degree in the last paragraph, on analysis it really adds nothing when you look to see what you are left with?
A. I disagree, and you would like me to go on and say why: I think the really important thing is that these are independent of the Gardner findings ---

Q. MR. JUSTICE FRENCH: Wait a minute. "The really important thing is that study plus letter are independent of Gardner"?
A. Well, the letter itself makes it independent. The study was not independent.

Q. "The really important thing is that the letter is independent of Gardner"?
A. Yes, and that there is a significant association of leukaemia risk with paternal exposure to all kinds of radiation.

Q. MR. ROKISON: You agreed with me yesterday that the Gardner hypothesis was a hypothesis which involved ionising radiation?
A. Yes.

Q. You were relying on this report, as I understand it, as being the only positively consistent study supporting Gardner, the Gardner hypothesis?
A. Yes.

Q. If you looked only at either ionising radiation where it is recorded, or possible ionising radiation even though it were not recorded when it ought to have been, then you do not have eleven cases at all?
A. No.

Q. You in fact have five cases?
A. Yes.

Q. Only one of which has a recorded dose, which is minuscule?
A. Yes.

Q. And the others have no recorded doses in circumstances where, if they had any doses of any significance, they ought to have been recorded?
A. Yes.

Q. There are no doses stated expect for the certain case?
A. Yes.

Q. There is no dose response relationship?
A. There cannot be.

Q. Of course not. The numbers are now not only small but they are very, very small, because your "certain ionising" is only one case?
A. Yes.

Q. Even though it might be independent of the Gardner study, what I suggest you are left with when you look to see what there is that is independent of the Gardner study, is nothing, or nothing of any significance at all?
A. I think it is of some significance.

Q. If anything, given that these cases would be very unlikely to have a substantial ionising radiation dose, it would tend to be contrary to Gardner, would it not?
A. I think you are calling for speculation there.

Q. Well, you are speculating, are you not?
A. You say "It is very unlikely that ..."

Q. I am not relying on any legal presumptions, Prof. Evans, but, as a matter of common sense, your speculation that this adds something depends upon these fathers having been exposed to significant doses which were not recorded in circumstances where, if they had received significant doses, they ought to have been recorded? Is that not right?
A. I am not sure whether it does depend on that, but I can see that certainly if that were true, it would undoubtedly support my view.

Q. But if it were not true ...?
A. If none of those people did have doses, and indeed that person whose recorded dose was 1.11, then it would be no support, I do agree with that.

Q. I will leave it there, if I may.

Q. MR. JUSTICE FRENCH: "If none of the other four ...", is that sufficiently accurate?
A. Yes.

MR. ROKISON: Yes, my Lord.

Q. MR. JUSTICE FRENCH: What I am writing is, "If none of the other four in fact had doses ..."

Q. MR. ROKISON: May we add this qualification: "if none of the other four had <u>significant</u> doses ..."? It is rather difficult. It has nothing to do with statistical significance, but doses greater than minimal doses?
A. Yes.

Q. MR. JUSTICE FRENCH: "If none of the other four in fact had doses greater than minimal ...", I suppose "greater than minimal over environmental ..."?
A. Yes.

Q. "... then Alexander et al gives no support", is that right?
A. Yes.

Q. Even in the letter?
A. Even in the letter.

MR. ROKISON: My Lord, I will leave it there.

Q. Can we move please to the next study to which you refer, which is the Urquhart.

Q. MR. JUSTICE FRENCH: Is that in the new bundle.

MR. ROKISON: My Lord, it is U248. My Lord, I note that there was one other point which was made by Prof. Evans in the course of his evidence in relation to the McKinney study which I perhaps ought to have mentioned.

MR. JUSTICE FRENCH: Yes, we will go back to that.

Q. MR. ROKISON: You mentioned in your evidence, Prof. Evans - and I do not want to read it to you, other than to give my Lord the reference for the purposes of the transcript cross-check, which is Day 13 at 72D - some documents which had been discovered on discovery which showed that there had been a potential source of radiation in the Gateshead area, is that right?
A. Yes, that is my memory.

Q. Do you recall what that source of radiation was?
A. No.

Q. Do you recall if it was some potential source of radiation from the nuclear industry?
A. My recollection is that it was a company called Parsons, but I don't know.

Q. There are two points that I want to put to you in relation to that. We have seen that as far as British Nuclear Fuels are concerned, of ionising radiation we only have the one case with the minuscule dose; the other cases are cases who were either construction workers, effectively, or industrial radiographers?
A. Yes.

Q. We do not have any of the cases which relate to any other ionising radiation exposure?
A. I am sorry, British Nuclear Fuels doesn't have?

Q. No, we do not in that letter. Of course they lump them together here, but assuming that the cases are cases which are not covered by Gardner and are therefore those that are dealt with in the letter that we have looked at, I suggest to you that those cases which are either possibles or certain ionising radiation, which we have discussed, are unlikely to have anything whatever to do with the activity in Gateshead to which you refer?
A. That could be. I have no knowledge on it.

Q. I think there had been a cluster at Gateshead which had given rise to some report. My instructions are that there was a report of Knox which reported a cluster between 1951 and 1960. Does that ring a bell with you?
A. It rings a bell.

Q. Without looking at the documents, it appears that the documents, if they are the documents that we think you were referring to, relate to an engineering facility which was not opened until 1962?
A. That could be.

Q. But you really cannot recall the details?
A. I can't recall the details.

Q. Then I can leave it there. I was going to ask you about Urquhart. I wonder whether we can take this fairly

shortly. What the authors of this report did was to examine the reported cluster around Dounreay?

A. Yes.

Q. And to test whether or not the Gardner hypothesis of paternal preconception irradiation could explain that cluster?

A. That was one of the things they tested, yes.

Q. I agree it was one of the things. What they did was that they took leukaemia and NHL together?

A. Yes.

Q. But they looked only at the age up to 15 and they looked at the period 1970 to 1986. Of course, as we have seen, the cluster at Dounreay was a cluster which was limited in both time and space?

A. Yes.

Q. MR. JUSTICE FRENCH: 1970 to 1986?

MR. ROKISON: Yes, that appears in subjects, my Lord.

Q. They did not restrict it, I think, to those ---

A. I think it is actually 1968 to 1986. I think the summary is wrong.

Q. MR. JUSTICE FRENCH: If you go five lines from the bottom of the left-hand column, you have got 1968 to 1984?

A. Yes, that was what COMARE looked at, but I think if you look at the first sentence, "The subjects and methods..." on the right-hand side, it is 1968 to 1986.

Q. MR. ROKISON: You are right, it is two-thirds of the way down the right-hand side:

> "All registered cases of leukaemia and non-Hodgkin's lymphoma in children resident in Caithness during 1968-1986 were included in the study ..."

You can see that the main outcome measures, which I think are the pathways or possible causative factors that they considered, are:

> "Antenatal abdominal x-ray examination; drugs taken and viral infections during pregnancy; father's occupation; father's employment at Dounreay and radiation dose; distance of usual residence from the path of microwave beams, preconceptional exposure to non-ionising radiation in the father; and other lifestyle factors".

The results were:

> "No raised relative risks were found for prenatal exposure to x-rays, social class of parents,

employment at Dounreay before conception or diagnosis, father's dose of ionising radiation before conception, or child's residence within 50m of the path of microwave transmission beams. Results also proved negative for all lifestyle factors except an apparent association with use of beaches within 25 km of Dounreay. However, this result was based on small numbers, arose in the context of multiple hypothesis testing, and is certainly vulnerable to possible systematic bias".

Their conclusion was:

> "The raised incidence of childhood leukaemia and non-Hodgkin's lymphoma around Dounreay cannot be explained by paternal occupation at Dounreay or by paternal exposure to external ionising radiation before conception. The observation of an apparent association between the use of beaches around Dounreay and the development of childhood leukaemia and non-Hodgkin's lymphoma might be an artefact of multiple testing and influenced by recall bias".

A. Yes.

Q. One sees on page 688 that there were fifteen cases of leukaemia and NHL during the relevant period below age 25, and one case was registered in the subject age 23, and that the fourteen other cases ---

Q. MR. JUSTICE FRENCH: Where are you reading from?

MR. ROKISON: My Lord, it is the paragraph about three-quarters of the way down the right-hand side of 688.

MR. JUSTICE FRENCH: "Completeness of Response"?

MR. ROKISON: Yes, my Lord, that is right.

Q. You will see that there were fifteen cases below the age of 25, but fourteen of them were clustered under the age of 15, as one sees at the end of that paragraph?

A. Yes.

Q. Eight cases were resident within the 25 km zone, and for one diagnosis in 1970 the remaining seven were 1979-86, and that was the time and space cluster which had been found?

A. Yes.

Q. Then:

> "Two of these seven cases were in children born outwith the Caithness area and the remaining five were in children born within the 25 km zone."

One sees on page 689, Table III, "Numbers of cases and controls and odds ratio or Fisher's exact p value, by paternal occupation recorded on birth certificate", and

you will see that occupation at Dounreay, both for those within 25 km and those outside, showed a negative odds ratio?

A. One less than 1, yes.

Q. Yes, that is what I mean. That means that on that hypothesis working at Dounreay would tend to have a protective effect?

A. Yes.

Q. In other words it would be less than one would expect from the total numbers being considered?

A. Apart from the fact that the confidence interval is so wide it is compatible with a rise, but nevertheless if the 0.58 were correct or the 0.38 were correct, either of those would imply what you say.

Q. I quite see that where you have small numbers you have wide confidence limits, that it is not incompatible necessarily with a rise, but that in a sense your best estimate, your point estimate, is that there is a reduction?

A. Yes.

Q. Where one looks at Table V, one gets "... ionising radiation dose before conception", and they set out the number of cases and controls within the various dose brackets, which are the same ones as were taken for Gardner, but in this case there was only one control in the total lifetime dose who had in excess of 100 mSv, and no cases, and there was only one case and no controls who had a six month dose of more than 10 mSv?

A. Yes.

Q. So the numbers are much smaller than they were in the Gardner Study?

A. Yes.

Q. Which is a point which you quite rightly make. If you look at Table IV, one gets, "Numbers of cases and controls and odds ratio or Fisher's exact p value by paternal employment in nuclear industry at conception of child and estimated ionising radiation doses before conception", and we have seen the "Father employed in nuclear industry ...", that is taken from the Dounreay figures in the table above, and the odds ratio, 0.38, that one finds that the father's radiation dose, comparing less than 100 with more than 100, one finds that you can get no confidence limits, no odds ratio, because there were not any cases who had more than 100, is that right?

A. Essentially.

Q. Shall we look at the "Results", and what they say about them:

> "Information on parental occupation was obtained from birth certificates and from the computer files

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of workers employed at Dounreay and elsewhere in the nuclear industry. Information available from questionnaires was used as confirmation. Table III shows the paternal occupation for cases and controls at the time of birth of the child for the main industrial groups found in the Caithness area. For three cases the fathers were employed in the nuclear industry at the time of the birth of the child .... One of them was described as an electrician, one as a process worker, and one as a charge hand. All these fathers were employed at Dounreay at the time of conception of the child and two were fathers of cases resident within 25 km .... No significantly raised risk was associated with employment in farming or fishing. Information derived from the occupational records was used to identify periods of employment in the nuclear industry of fathers and cases and controls before conception of their children. No raised risks were observed in respect of these periods of employment. None of the fathers of cases had an accumulated external ionising radiation dose >100 mSv before conception of the child; the fathers of three cases who were employed in the nuclear industry each had a lifetime dose <50 mSv (40 mSv, 29 mSv, and 17.4 mSv respectively). One father of a case had a dose of >10 mSv ... in the six months before conception; the two other fathers had doses of 3.7 mSv and 0.7 mSv respectively. No significant differences were observed between cases and controls with respect to these external radiation doses."

Then the discussion we find at page 690:

"In their case-control study of leukaemia and lymphoma among young people in West Cumbria Gardner et al observed higher relative risks in children whose fathers were employed at the Sellafield plant and, in particular, in those whose fathers had had high radiation dose recordings before their conception. Dounreay and Sellafield installations are the only nuclear reprocessing plants in Britain, and both have a reported excess of childhood leukaemia in their immediate surrounding areas, which, in the view of [COMARE] seems unlikely to be the result of chance. In view of the findings of Gardner et al there seems to be a prior hypothesis of a possible association between paternal employment at Dounreay and the development of leukaemia by their children, but no raised relative risks associated with such employment were observed in this study.

A particular focus of concern in the committee's report was the seven cases occurring between 1979 and 1986 in children resident in the 25 km zone around Dounreay. A primary objective of the study was to determine whether any of these cases could be

explained by possible risk factors identified in earlier studies. Six of the fathers of these cases were not employed in the nuclear industry before conception of the child."

So those cases that had comprised the Dounreay cluster, as had been identified, being seven cases within that time and distance cluster, six of those were not employed in the nuclear industry at all before conception.

> "Thus, although the results of the current study do not contradict the hypothesis developed by Gardner et al, this particular hypothesis clearly does not explain the excess incidence of childhood leukaemia and non-Hodgkin's lymphoma observed in the 25 km radius circle around Dounreay from 1979-1986."

Just pausing there, that must be right, mustn't it?

A. Absolutely.

Q. So the position is that the numbers are small and we do not suggest, and I think nobody suggests, that this demonstrates that the Gardner hypothesis in relation to the population studied by Gardner is wrong, but what it does show beyond doubt is that to the extent to which there was a cluster at Dounreay, it could have had nothing whatever to do with parental preconception radiation dose?

A. Yes.

MR. JUSTICE FRENCH: Just pause there. So on the one hand it does not support Gardner and on the other hand it demonstrates that the Dounreay cluster was not due to paternal preconception dose?

MR. ROKISON: To be fair, what I put is that it does not prove that the Gardner hypothesis is wrong, and we do not suggest it does but it does not give any support to it and what it does show is that the Dounreay cluster could not be caused by that.

MR. JUSTICE FRENCH: I have got that, yes. It does show that the Dounreay cluster was not caused by paternal preconception dose, right?

MR. ROKISON: Yes, my Lord.

THE WITNESS: Yes.

MR. ROKISON: My Lord, I do not really think there is much point in questioning further about that. There is agreement between us and I can get on.

Q. Can I just ask this one question? I think you did say in your evidence that the Urquhart study was compatible with the Gardner hypothesis because the confidence limits, the confidence intervals, were so wide that it was compatible with Gardner. It may be that in that respect you were

looking at Table IV but it is not compatible with Gardner in the sense that the Gardner hypothesis could be the or a cause of the Dounreay cluster. Do you agree?

A. No, I do not think that is the point at all. The Gardner hypothesis is that paternal preconception irradiation might cause leukaemia. That is a possible thing and that, for example, if you wish to emphasise something, the six month dose before conception has an infinite odds ratio, the highest odds ratio we have yet seen but of course that is very little evidence, and if you were to look at some of the other categories ....

Q. That is because you have no controls who had it?

A. That is right. Similarly the odds ratio of zero in the lifetime dose is similarly little evidence either way.

Q. I agree with that, with respect, and that is why we say that it does not, in a sense, disprove the Gardner hypothesis because the numbers are so small and the confidence limits so wide. But it is not compatible in this sense, and I know you say that is not the important sense, but you are not saying that "Oh well, the Gardner hypothesis could have caused the cluster"?

A. By no means, no, I am not suggesting that at all.

Q. That is all I wanted to clarify. Thank you very much. You next refer to the "Birth and School Cohort Study around Dounreay", which is Black et al in 1992, and you summarise what it says. You are not saying, are you, that this provides any support to the Gardner hypothesis?

A. No.

Q. Then I think we can leave it. The conclusion, therefore, you reach from the four studies which you have looked at, let's bear in mind what those four studies were: they were the Draper Childhood Cancer Research Group Study?

A. Yes.

Q. Which you summarise at the end there and I asked you some questions about it yesterday. You say that it is not supportive of the Gardner hypothesis although it does not directly contradict it. We looked at McKinney and we have looked at the Dounreay Study, and you say:

> "I conclude from the above four studies that although support for the Gardner hypothesis is limited, his findings have not been either disproved or reduced in cogency. None of the studies published provides any strong evidence for an alternative mechanism."

It is right, isn't it, that the only support that you rely upon from any of these subsequent studies is such support as you think can be derived from the McKinney letter?

A. Yes.

Q. We have looked at that. You say they do not provide "strong evidence for an alternative mechanism", and may we just look, because you yourself do consider a possible alternative mechanism in your next few paragraphs, and in particular the viral hypothesis which is proposed by Prof. Kinlen?

A. Yes.

Q. May we look at those briefly, the Kinlen papers, to see what the hypothesis is and the extent to which it could explain any of the clusters which have been observed? I think the first of the studies is 10th December, 1988, K147. This first paper that we look at, you see that Prof. Kinlen is a Professor at the Cancer Research Campaign's Cancer Epidemiological Unit at the University of Edinburgh. Is he somebody who is known to you?

A. Yes, he is actually in Oxford now.

Q. Yes, but at the time of these papers he was at Edinburgh?

A. The first two, yes.

Q. He is considering a possible alternative explanation for the Seascale and Dounreay clusters, isn't he?

A. Yes.

Q. I do not think it is necessary to read all of it but may I just take you to some parts of it. First of all his summary:

> "Increases of leukaemia in young people that cannot be explained in terms of radiation have been recorded near both of Britain's nuclear reprocessing plants at Dounreay and Sellafield. These were built in unusually isolated places where herd immunity to a postulated widespread virus infection (to which leukaemia is a rare response) would tend to be lower than average. The large influxes of people in the 1950s to those areas might have been conducive to epidemics. The hypothesis has been tested in Scotland in an area identified at the outset as the only other rural area that received a large influx at the same time, when it was much more cut off from the nearest conurbation than at present - the New Town of Glenrothes. A significant increase of leukaemia below age 25 was found (10 observed, expected 3.6), with a greater excess below age 5 (7 observed, expected 1.5)."

So what he did was he considered this hypothesis, which is a hypothesis in a sense which he then tested blind somewhere else, in a place which he selected as having as near as possible the relevant characteristics which he saw in Sellafield and Dounreay?

A. Yes.

Q. He says, at the top of the first page, 1323, on the right:

"This unit ...."

- that is, the Edinburgh unit -

> "... has for some time been investigating the possibility that certain distinctive aspects of the Dounreay and Sellafield areas that owe nothing to radiation are relevant to the causation of leukaemia elsewhere. These aspects involve the combination of geographic isolation and population influx. The hypothesis involves three elements: 1, that influxes of population into rural and isolated areas are conducive to epidemics of certain infections; 2, that Sellafield and Dounreay are extreme examples of isolation and population influx; 3, that some unidentified virus or viruses can cause childhood leukaemia. The last mentioned is itself hypothetical; but it is plausible, and if it is correct the possible relevance of 1 and 2 obviously deserves careful examination."

Then he discusses the point more fully and describes what he has in mind:

> "1. People who live in geographically isolated places represent one of the few groups who may escape appreciable exposure at usual ages to common and widespread infective agents. To such groups 'incomers' can introduce infective agents with dramatic consequences. The following interrelated factors may be relevant here. A lower level of natural immunisation to the agent may occur either because of the fewer opportunities for person-to-person transmission than in more urban areas or because the population is not large enough to maintain the disease in endemic form. Age at exposure too may be grater in rural than in urban areas, and this can influence the form of certain viral infections such as maternal rubella and paralytic poliomyelitis. The dose of the agent may also be important, as it is in several animal models of viral oncogenesis, and when there is large-scale mixing of susceptibles ...."

and so on. Then he describes Dounreay:

> "2. The nuclear power station at Dounreay is unquestionably isolated."

and he describes its location. He says:

> "The isolation in the later 1940s and 1950s of Sellafield, near Seascale, is perhaps less obvious. Although quite close to the Lake District, the West Cumbrian coastal strip was by comparison little visited by tourists. Always recognised as distinctive and somewhat separate by the people of

Carlisle and Lakeland, the small industrial towns of Whitehaven, Workington, and Maryport on the coast were nevertheless of sufficient size to make the West Cumbrian population fairly self-sufficient. Sandwiched between the sea to the west and the Lakeland mountains to the east, access to Seascale was mainly from the north along the coast, which in turn was reached from the main regional centre of Carlisle to the north-east ...."

and so on.

> "But in addition to their relative isolation, the populations of Thurso and Seascale were unusual in increasing greatly between the 1951 and 1961 censuses."

and he sets out the figures. Then:

> "3. The possibility that childhood leukaemia is of viral origin has often been raised ...."

and he discusses two rare types of adult leukaemia and the viruses associated with them:

> "Furthermore, specific viruses are known to cause leukaemia in cats and other animals."

Then he refers to the Epstein-Barr virus infection and he says, at the bottom of the page:

> "Spread of the agent may not depend mainly upon persons with leukaemia but, as in infectious mononucleosis and Epstein-Barr virus infection, upon a common infection in apparently healthy people to which leukaemia is a rare response."

He says:

> "The obvious test of the above hypothesis is to determine whether there is an excess in other isolated areas which have seen a considerable influx of population but which lack any man-made potential source of radiation."

Then what he does is explains then how and why he chose the town of Glenrothes. Then he sets out his methods on the next page, 1325, and how, near the bottom of that column:

> "The observed numbers of deaths from leukaemia and lymphoma in each 5 year age group and quinquennial period were compared with expected numbered, obtained by applying Scottish national mortality rates ...."

and then they set out the results in Table III. If one looks back to Table III, on page 1324, what one finds is that for leukaemia there is a statistically significant

increase in Glenrothes, 1951-1967, in the 0-4 age group, as well as 0-14 and 0-24, and one finds a just statistically significant increase in 0-4 in the years 1951 through to 1985. Perhaps one ought to read, in the discussion, because of criticisms which are thereafter made:

> "Ideally the above hypothesis should be tested in an area similar to Thurso. However, because the town increased more in population and is also further away from a sizeable centre than any other burgh, the comparison area had to be the only other rural area of Scotland to experience a large influx of people and which was even moderately separated from a conurbation - namely ... Glenrothes .... Most of this period preceded the opening of the Forth Road Bridge in 1964, an event that had important economic and demographic effects on Fife. The finding of a significant excess of childhood leukaemia in this area therefore supports the hypothesis. Chance is an unlikely explanation, since an excess was postulated before the data were collected; nor can it be attributed to underestimation of the population in the relevant age groups. Even if the official total population estimates of Kirkcaldy DC in the years 1956-60 had been underestimated, and, most improbably, the 1961 census population had been reached 5 years sooner with no further increase until 1961, an excess would still have been evident at ages below 5 ...."

So he is dealing in advance with problems of the connection of Glenrothes by the bridge, and the population estimates. Then he sets out what the excess would have been on the hypothesis he there makes. He refers to the influx to Glenrothes, that it:

> "... occurred before that in Thurso .... The former (1951) residents of Thurso, on the present hypothesis, include most of the 'susceptibles' whereas the incomers ... include the carriers and infecteds. By contrast in Glenrothes the largest group of incomers came from other parts of Fife, many being as likely to be susceptibles as the original inhabitants ...."

In others words, he refers to differences between Thurso and Glenrothes' age groups and time periods that show increases in leukaemia; he refers to

> "Classical epidemiological reasoning about infectious disease ... applied here to childhood leukaemia, a disease often suspected of being viral in origin."

He refers at the bottom of that page to the originally reported Dounreay excess and the boundary points, which we have looked at. Then:

> "No such choice was possible in the present study for, whereas the 1981 populations can be calculated for areas similar to the former local authority areas, it is impossible to estimate the pre-1971 populations of post-code units. With improvements in survival, mortality has become an inefficient measure of childhood leukaemia incidence, but this was not so in the 1950s when the disease was uniformly fatal. The previous study of leukaemia incidence near Dounreay was based on 4 cases in, and 2 near, Thurso in the period 1968-84. 2 of these 4 patients have died and are included in the present study. The 1.78-fold increase in mortality from leukaemia over the whole period since the Dounreay reactor was completed is not appreciably different from the estimate based on incidence over the more restricted period ...."

He says:

> "The Glenrothes cluster of childhood leukaemia seems to be the first instance of a particular cluster being found as predicted by a hypothesis specified before the data were collected. It is difficult to escape the conclusion that at least some of the excesses near Dounreay and Sellafield have a similar (infective) explanation since they represent more extreme degrees of isolation combined with population influx. It is the locally born children near Sellafield and also near Dounreay, as found in this study, who show the increase of leukaemia and not those born outside but attending school there. This is consistent with there being fewer outside contacts, among the former, producing more susceptibles than among the children of incoming workers. It may also reflect an interaction between age at exposure and dose of virus, as seems to occur in feline leukaemia. But the pattern of transmission in cases even of known infection can be extremely elusive.
>
> The absence of space-time clustering commonly reported in leukaemia is consistent with the notion that leukaemia is a rare response to a common, possibly subclinical infection, the studies having been conducted mainly in fairly stable populations in which the virus-host equilibrium had not been disturbed."

and then he says that it "deserves renewed attention".

That was then followed by a further study by Kinlen, Clark and Hudson in 1990, K148, and that was published in the Lancet. There you can see that he extends the study substantially and considers the

> "Evidence from population mixing in British New Towns 1946-85 of an infective basis for childhood leukaemia.

> Mortality from leukaemia under age 25 was studied in British New Towns to test the hypothesis that leukaemia represents a rare response to a much commoner (but unrecognised) infection, the transmission of which is facilitated when large numbers of people come together. The density of children was higher in the rural, but lower in the overspill, New Towns ...."

He draws a distinction between New Towns which effectively are overspills from large towns, as opposed to new ones which are set up in a rural setting, doesn't he?

A. In this paper.

Q. "Residents of the rural New Towns had greater diversity of origin than those of the overspill towns of London and Glasgow. These two factors would encourage a greater rise in the postulated underlying infection in the rural towns, and in these a significant excess of leukaemia at ages 0-4 was found in 1946-65. In both sets of towns there was a significant deficit in other age groups consistent with immunising effects of the relevant infection. There are parallels with feline leukaemia virus infection ...."

and so on. Then he introduces it by setting out the hypotheses. Then one sees the methods; there were 14 New Towns which were designated and 9 of these were overspill towns which received a well mixed group from a nearby city, and 5 rural towns which aimed to build up the population of industrial development areas that had sprung up away from conurbations. They set out how they find their details and so on. I would like to come to 579 where he takes two periods ....

MR. JUSTICE FRENCH: Page 579? I must be looking at the wrong document. 147 is correct and 148 is wrong. I am missing 148 and have got two 149s.

THE WITNESS: I can give you my 148, my Lord. (Handed)

MR. JUSTICE FRENCH: I have now got a 148, Mr. Rokison, but I was listening as you read ....

MR. ROKISON: In that case, my Lord, if your Lordship could go to page 579, Table V, "Observed to Expected Ratios of Deaths from Leukaemia", and the two periods taken, Periods A and B, your Lordship sees set out in Table VI below. Period A was 1946-65 and Period B 1966-85.

Q. What one finds is that there is a statistically significant increase in Glenrothes for the ages 0-4 and 0-24, and one finds that there is a statistically

significant increase in 0-4 in other rural New Towns, both in Period A a Periods A and B taken together. If one lumps them together and takes all rural, embracing Glenrothes and other rural, again one finds statistically significant increases in the 0-4 age group for 0-4?

A. Yes. If we were to go through the exercise that Mr. Rokison has gone over McKinney or over Gardner, we would find that all the excesses 0-24 are driven by the 0-4s, but the Glenrothes was not actually pre-specified - he had already found the cluster in Fife and he then examined Glenrothes and therefore ....

Q. That is a different point. I will come to that.

A. I see it as terribly unimportant from my report's point of view.

Q. I accept that 0-4 overlaps with 0-24, of course, and when you split it up the 0-4 has a statistically significant increase, 5-25 does not, so that the statistically significant increase you get in 0-24 is driven by 0-4?

A. Yes.

Q. I quite accept that. If one looks at individual towns, they are set out in table 6, and one finds that there is an elevated incidence in three towns, Glenrothes, Corby and Aycliffe in 0-4. For the first period it is significantly increased, whereas if one looks at the overspill towns in the bottom part of the table, one sees lower incidence figures which are reflected in table V. Is there anything more one should say by way of general description of what the tables show you?

A. No.

Q. I am trying to give a fair summary as we go through because it may be the only time my Lord will be invited to look at this paper. Is there anything you want to draw to attention? Perhaps I should have asked you whether there was anything you wanted me to draw attention to in the last paper that I omitted to refer to?

A. Well, the thing I would be concerned about is that there is a concentration on particular periods and the evidence and so on is largely determined by drawing the boundaries in the way that you wish to.

Q. Drawing the boundaries in terms of the age groups or in terms of the years?

A. In terms of both the years and the age groups, and the findings are...

Q. Is that really so, bearing in mind...

A. I think it leads to some difficulty because...

Q. It is taking very long periods, comparative long periods for both of these studies?

A. Yes.

Q. Taking more than one age group from which it does appear that it is the youngest age group where the effect seems to be the most marked?
A. I would agree that the effect, if there is an effect, and there could well be an effect, is most marked 0-4. However, for example, in table 3 of the previous paper, the excess 0-4 is 7 and the excess 0-14 - in other words 5-14 - there is no excess at all, rather a deficit, because there is only 1 case.

Q. I think he says that. He draws attention to that...
A. No, he draws attention in his summary to a significant increase below age 25.

Q. No. I did read it to my Lord, the passage where he says that it is concentrated in the...
A. With a greater excess.

Q. ...and that is followed by a deficit?
A. Well, in the summary of that paper he doesn't emphasise that.

Q. I am sure we looked at that.
A. The deficit you are referring to is in the second half of the time period, 1968-75.

Q. No, it isn't. Well, we can find it.
A. I can find you the bit where he says, on page 1325 in the middle of the "Results" paragraph, it says:

> "In the second half (1968-85) there is no excess - indeed, a non-significant deficit, with 1 death observed and 5.18 expected."

Q. Forgive me, that wasn't the passage.
A. In the previous bit of his results he says:

> "...there is a significant excess of leukaemia deaths below age 25 in the Glenrothes area...This is mainly due to an excess at ages below 5."

Q. Yes.
A. Almost entirely due to, but the only significance is entirely due to that.

Q. Yes. Well, I would accept that.
A. I think you have an asymmetric approach to the papers that you looked at. It is quite understandable. I don't think I am called on to dissect these papers.

Q. No. All that I ought to have invited you at this stage at least to have done would be to point out any aspects of the papers as we are going through them that you think ought to be particularly referred to my Lord. I accept that this hypothesis as reflected in these two papers is considering the effect of an influx of population on the young in a rural area. If we can come back to the

second paper which we were looking at. It is page 579. We have looked at the tables. If I could pick it up half way down the right hand column:

> "The ratios of observed to expected deaths from leukaemia by age group in the two groups of New Towns are shown in Table V...There was a pronounced excess of deaths from leukaemia at ages 0-4 in Glenrothes during period A which accounts for most of the excess previously reported for the whole of Kirkcaldy DC in this age group. There was also a significant excess in this age group during period A in the other rural New Towns but no significant excess during period B. In the overspill towns there was no excess during either period. Analysis by individual rural New Towns for the age group 0-4 showed that the excesses during period A were greatest in Glenrothes, Aycliffe and Corby."

That is all accurate, isn't it?
A. Yes.

Q. Then he shows, by reference to Table VII, that it is not confined to a single cell type, although the only significant excess was lymphatic leukaemia, nearly all specified as acute during period A, and we find that set out in the first column?
A. Yes, not very specific.

Q. Not very specific in terms of...?
A. In terms of being only confined to one cell type, but I don't think it is an important point.

Q. Very well. Then in the "Discussion" he refers to:

> "An infective basis has been established in some animal and (rare) adult human leukaemias and has often been considered for childhood leukaemia."

Would you agree with that?
A. Yes, I would agree with it.

Q. His conclusion or observation on the right hand column:

> "It is clearly difficult for an epidemiological study to produce evidence that leukaemia is a rare response to an unrecognised infection when the agent in question has not been identified."

You would agree with that I take it?
A. Yes. I think the key difficulty is what triggers the rare response.

Q. Also what is it a rare response to? There are two questions, aren't there?

MR. JUSTICE FRENCH: I thought, in my simplicity, it was the same question.

MR. ROKISON: Firstly, what is it a response to? What potential viral infection? What is the virus that it is in response to, and secondly, what is it that gives rise to the unusual response to it.

MR. JUSTICE FRENCH: Yes, I see.

MR. ROKISON: He is postulating here that whatever may be the virus, being a virus which can be leukaemic, he is postulating that the rare response to it may arise from a lack of relevant herd immunity in a rural population, which is then faced with the influx of a large population from outside:

Q. Would that be a fair description of the idea?
A. No, I don't think he is saying that the rare response is because of the rural community. He is merely talking about the spread of that virus being more likely there and also the immunity to it being there. Maybe we are saying the same thing.

Q. Yes. It is the lack of immunity to it?
A. Yes, it is the lack of immunity to it in those people who are resident in the rural areas.

Q. Yes, I think the difference between us is a semantic one only. Then he says:

> "This study adds to support for the infection hypothesis."

Would you agree with that?
A. I think it adds slightly to it.

Q. "Interest in the possible relevance of population mixing to the aetiology of this disease was first aroused by the excesses recorded near two isolated nuclear power stations (Sellafield and Dounreay). Linked to their isolation have been population influxes and a high turnover of residents - all factors that have figured in the epidemiology of infections."

Do you agree with that?
A. Yes.

Q. Then he says:

> "New Towns differ in several obvious respects from these areas. In the case of Aycliffe, Peterlee and Glenrothes the findings cannot be explained by invoking isolation or their rural nature...since no communities of any size previously existed there; the effect may, however, have operated within subgroups of incomers."

What he is postulating there is if you get a lot of people coming there may be some subgroups who have not

built up the relevant herd immunity to some viral infection which may be carried by others?

A. This is typical of those medical hypotheses that you can come up with...

Q. Indeed.

A. ...for any pattern of data.

Q. With respect, I quite agree. I think it is there. It is at 581. I thought I had read it somewhere where he makes the point, at the top right:

> "An infection that is only rarely complicated by leukaemia must subsequently confer a measure of protection against that infection and therefore also against its complication, leukaemia. An epidemic of such an infection should therefore be followed by a deficit of leukaemia."

A. Yes.

Q. That was where he actually specifically raises that point. Then he says:

> "Those considerations are relevant to our most striking findings..."

Then half way down that column he refers to the fact that the excesses are confined to preschool children:

> "...in whom the patterns of many different infections are greatly affected by older children."

We come to 582 where on the left he refers to what might be called the Greaves theory, which is slightly different, I think; the Greaves theory being, if I don't describe it inaccurately, that the peak of common ALL which one finds in ages 3-4 is possibly due to the fact that it is at that time that children who might have been protected within the environment of the home and so on, they are first exposed to outside viral infections?

A. Yes. As far as I understand it, it also involves a mutagenic effect that the Kinlen hypothesis does not in the same way.

Q. No, I think the gloss that may be put upon it, is that it is consistent with that, that you could have a mutagenic effect which makes the child more vulnerable?

A. It is not my own area of expertise.

Q. What he says is:

> "Greaves has proposed that acute lymphoblastic leukaemia around its peak incidence at ages 3 and 4 is due not to specific viruses but to mutations that are made more likely by delayed exposure to various non-specific infective agents, as well as to other immunological challenges."

Then he says:

> "Correspondingly much of our data could be interpreted according to the mutation hypothesis. However, this hypothesis has not predicted the deficits we observed in myeloid and lymphatic leukaemia. In addition, the fact that the excesses in the rural towns did not spare children below the age of 1 suggests that delayed exposure to infections is unlikely to explain all of our findings.
>
> The findings suggest rather that an infection underlies childhood leukaemia that has certain similarities to feline leukaemia virus infection."

At the bottom of the page he says:

> "Our findings further support an infective basis for childhood leukaemia (not only the lymphatic type) and specifically for an infection that is promoted by greater levels of social contact particularly between people from previously widely separated communities."

That is his second paper to which you refer. There is then a paper in the BMJ in 1991.

MR. JUSTICE FRENCH: Is this another Kinlen?

MR. ROKISON: Yes, my Lord. K.150, my Lord. I am sorry, it is the British Journal of Cancer in March 1991.

MR. JUSTICE FRENCH: That is K.150 - "Contacts between adults..."?

MR. ROKISON: Yes.

Q. Is this a paper you have read?
A. I don't think that I referenced it.

Q. This is a paper in which Kinlen et al...
A. Yes, I did. It was 35.

Q. Now, as you rightly say, at the epidemiology research group in Oxford. What they discuss here is the possibility of a similar infective origin, explaining the excess which was observed in the area of Reading, which we looked at when we were looking at Burghfield?
A. Yes.

Q. If one can just read the Summary:

> "The increasing tendency for people to work outside their home community - one of the most striking of modern demographic changes - has relevance to a

recent aetiological hypothesis about childhood leukaemia: that a community's immune response to an underlying infection can be disturbed by increases in new social contacts. This was tested in the only 28 former county boroughs in which accurate comparisons of workplace data from the 1971 and 1981 consensuses are possible - because their boundaries were left unaltered by the major reorganisation in 1974. After ranking the districts according to extent of commuting increase, a significant trend in leukaemia incidence was found at ages 0-14 and a suggestive one at ages 0-4. Among ten similar sized groups of county districts ranked by commuting increase, the only significant increases of leukaemia in 1972-85...were in the highest tenth for commuting increase. These excesses persisted after excluding Reading, a major part of the area where an excess of leukaemia has been linked to the nearby nuclear establishments at Aldermaston and Burghfield."

Then he refers to what I might call the Kinlen hypothesis and his earlier papers. He says half way down:

> "This possibility has therefore been investigated in all the former county boroughs of England and Wales..."

That is the possibility that the increased tendency for people to work in communities away from where they live, commuting having been common place, and the number of boundary crossings and the fact that:

> "Such journeys in the area of work itself provide opportunities for new contacts that may be relevant to the above infected-base hypothesis."

Therefore they investigated it in the former county boroughs of England and Wales which remained after the reorganisation:

> "Only in such areas can comparisons be made of commuting levels across town boundaries in 1981 with those in 1971, using census data for those years. These boroughs include Reading, part of an area of Berkshire and Hampshire where an increased incidence of childhood leukaemia...has been reported and had been linked by some to the nearby nuclear establishments at Aldermaston and Burghfield."

He refers to Roman, which we have looked at.

One sees the results are summarised, starting at page 550 and he notes, about a dozen lines down:

> "It is striking that the only two (Gloucester and Lincoln) with significant excesses of childhood leukaemia in any age group are among those with the greatest commuting increases ranking 2nd and 3rd highest respectively."

Then he also observed in Table III, simplifying the findings:

> "In the absence of any a priori basis for grouping the county districts, we have chosen the maximum number of similar sized groups (in terms of child years under age 15). That the number of groups was ten was determined by Liverpool which happens to have both the lowest rank of commuting increase and the largest population. In the category with the greatest increase in commuting level, highly significant excesses of leukaemia are present at ages 0-4 and 0-14...
>
> The district with the greatest increase in commuting is Reading which forms part of an area of Berkshire and North Hampshire...
>
> However, this district is not solely responsible for the significant excesses in the highest tenth, for they persist after excluding Reading..."

I don't want to spend time with you, obviously, in the witness box reading through the whole of this paper. He refers to Table V at the bottom of page 551, which shows the data for ages 0-4, analysed by change in commuting and change in population.

> It concludes:
>
> "A significant excess of leukaemia is present only in the group of county districts forming the highest category for both measures."

That is the change in commuting and population. He again refers at page 563, on the left in the middle to the fact that the county with the greatest increase in commuting is Reading:

> "This is also the only district entirely contained within an area in which an excess of childhood leukaemia had already been observed..."

He concludes in the next paragraph:

> "It is likely that cases of leukaemia outside Reading but within the West Berkshire excess are also related to commuting increases..."

He notes, at the top of page 553, on the right:

> "It may be noted that none of the other four county districts in group X are within 10 km (even 25 km) of a nuclear installation."

Can we come to the last page - I am really skipping through it in order to try and pick out its main findings and conclusions:

"Investigations by COMARE indicate that the tiny amounts of radiation released into the environment from Aldermaston and Burghfield are too small to cause the increased incidence of leukaemia in their vicinity, which they were unable to explain. However, they regarded a hypothesis about disturbances of herd immunity by population mixed as irrelevant because there had been no sudden population influx into a somewhat isolated area as in the first reported test of the idea. The present study offers an explanation that is consistent with that hypothesis and with the observations in new towns. Moreover it is supported by the finding of similar excesses in areas without nuclear installations but which, like the area around Aldermaston and Burghfield, have recently experienced large increases in commuting levels."

He says:

"These findings are consistent with other evidence about the relevance of increases in social contact to the aetiology of childhood leukaemia and present further support for an infection-based hypothesis for the disease."

Do you disagree with that?

A. I think a key bit is the previous paragraph.

Q. What, the relevant question?

A. Yes.

Q. "A relevant question is whether the prevalence of any infectious disease has also increased in the districts that have experienced the greatest increases of commuting. Published data are limited to only a few infectious diseases, but none show any clear increase in the highest tenth group. However, it may be noted that none of those diseases show a close similarity to the type of disorder to which childhood leukaemia is postulated as belonging."

A. Yes.

Q. So there is no general increase in disease, but there is apparently...

A. There is no general increase in infectious disease and if this is an important aspect then one would expect to find infectious diseases being more prevalent in those places, if infection is the important mechanism.

Q. Whereas, as you say, if preconception irradiation and some germ-like damage is the likely cause then one would expect to find a widespread of other cancers, stillbirths, infant mortality and so on in the same areas?

A. Assuming that they have the same mechanism, yes.

Q. Your note in the Addendum:

> "A correction for multiple comparisons omitted from Table II changes the significance levels..."

So they have carried out that exercise?
A. But of course they don't report it terribly carefully.

Q. Yes, but they do it, don't they?
A. Yes. Of course, in a geographic study of this kind, and the way he has decided to group things, it is very, very vulnerable to that sort of problem.

Q. Finally, perhaps we can just look at the last of the Kinlen papers, which is Kinlen and Hudson, K.149, which is the study of childhood leukaemia and polio in relation to military encampments during the period of National Military Service. Was this one you read?
A. Yes.

Q. Perhaps one can look at the abstract:

> "To determine if any excess of childhood leukaemia was associated with the large and increasing numbers of national military servicemen in 1949 and 1950, particularly in rural districts. This would be a further test of the hypothesis that childhood leukaemia can originate in an infection, the transmission of which is facilitated by an increased number of unaccustomed contacts in the community."

They studied children under 15 in England and Wales and the results were:

> "In 1950-3 but not subsequently, a significant excess of leukaemia in children under 15 was found in the fifth of county groupings with the highest proportions of servicemen. This was due mainly to a significant excess in children under 2 years (and especially in those under 1 year) in rural districts. It was confirmed among the tenth of local authority districts with the highest proportion of servicemen. These rural areas showed significantly more notifications of, and deaths from, poliomyelitis among children than the rural average.
>
> Conclusions: The findings support the infection hypothesis. That the excess of leukaemia was greatest in children under 1 year suggests transmission of infection among adults and thence to the foetus."

Do you have any comments about that paper?
A. This was the Kinlen paper I didn't cite in my report. My comment on that would be that it is really rather odd that the leukaemias occurred among the servicemen's children when you would actually expect it to be

occurring among the indigenous community if that hypothesis was correct.

Q. Of course, either is possible?
A. Either is possible, but the mechanisms keeps changing. The hypothesis is a bit like the soap in the bath. When you sit on it it zips somewhere else.

Q. Well, just pausing there, is that fair? The hypothesis is this, isn't it: that the hypothesis is that carriers of some viral infection meet with a population which has not developed a herd immunity to that infection?
A. Yes.

Q. That combination gives rise to an excess in childhood leukaemias...
A. But it should be in the ones who have not developed the herd immunity.

Q. Who have not developed the herd immunity. Certainly. However, it is possible that if you have an influx of population into an area where there may be a viral infection to which the local community has built up a herd immunity, then exactly the same hypothesis can operate, can't it? What is needed is the meeting of a population, one of which has the viral infection and has built up a herd immunity, and the other of which has not built up the herd immunity? Is that not right?
A. Yes.

Q. There is no magic in which population happens to be coming in and which one is there already. The hypothesis could work whichever was the case?
A. It could do, but it would also require then a rather different pattern. I would have to say I am not an expert in this area but it seems to me that the pattern of the commuting and the pattern of the rural excesses seems to require subtly different hypotheses.

MR. JUSTICE FRENCH: You would have to postulate a military camp full of people who came from the first place, wouldn't you? A military camp tends to have a lot of heterogeneous populations?

MR. ROKISON: No, you would not. That is exactly what you would not do, my Lord:

Q. The position surely is this, that my Lord saying that what you have to postulate is the number of people coming in who all have the same lack of herd immunity. However, it is not a question of having the same herd immunity. It is the fact that if you have a viral infection in a particular region, those who are in that region will build up some herd immunity to that viral infection? This is the hypothesis. If you then get an influx not necessarily from one place, but influx from other places, those may not have built up the same herd immunity.

MR. JUSTICE FRENCH: In theory, of course, it is perfectly possible. Well, all I was talking about was the relative likelihood and I think that is what Prof. Evans was talking about. I may be quite wrong, do pursue it.

MR. ROKISON: Well, my Lord, I don't know that Prof. Evans, any more than I... We can deal with this with the geneticists and medical experts in due course.

Q. I am just trying to reach common ground as to the understanding of what the theory is, but the hypothesis could operate, leaving aside for the moment questions of likelihood, but the hypothesis could operate whether you have your local community who have no herd immunity to a viral infection which is introduced from outside, or whether you have a community coming in from outside who do not have a herd immunity to a viral infection in the location to which they come?
A. Yes.

Q. That is all?
A. Well...

MR. ROKISON: Then we can leave it there. I will not spend long on that. I will try very hard to be as quick as I can because I know my learned friend wants a little time for re-examination this afternoon and I have yet to deal with Draper and the Louise Parker paper.

MR. JUSTICE FRENCH: Yes. We had lots of Draper on Wednesday, but there is more to come?

MR. ROKISON: There is the 1992 paper I have got to deal with, my Lord.

MR. JUSTICE FRENCH: Yes, very well.

(Luncheon adjournment)

Q. MR. ROKISON: Prof. Evans, we finished this morning by looking at various Kinlen papers and we discussed the broad nature of the hypothesis. Do you accept that that hypothesis is a possibility?
A. I do.

Q. It is a possibility which could, in whole or, at least, in part, explain the Seascale excess?
A. My judgment is that it could more easily in part explain it, but would be unlikely to explain it in whole.

Q. The reason why it would be unlikely....?
A. It could, nevertheless, explain it.

Q. Yes, and it could explain Dounreay?
A. Much less likely. The time period for the excess relative to population movements is not right with Dounreay.

Q. But that presupposes that it follows swiftly upon a sudden population explosion, if I can put it that way, rather than the fact that where you are dealing with nuclear installations you may be dealing with a mobile population, as is clear you were in Seascale?
A. But most of the excess in Dounreay has not anything to do with employees to do with Dounreay.

Q. No, I see. Yes, you are quite right. It could explain in part an excess in Reading?
A. I find that much less convincing, but it could.

Q. And the answer is it is an interesting hypothesis, which is not biologically implausible and which merits further investigation?
A. Yes.

Q. I suggest that, in those characteristics, it is really comparable with the Gardner hypothesis?
A. It is a very non-specific hypothesis and it requires some other triggering mechanism, and it seems to me it is much more likely that it provides the general background of susceptibles, if it is correct, and that I think that, if radiation is contributing, it would be a trigger that initiates that rare response. That is, I think, what I said there.

Q. Yes, if radiation is a cause, then it could operate together with some viral cause such as that?
A. Yes.

Q. I entirely accept that, if radiation is a cause, but looking at the hypothesis as operating on its own, without necessarily any radiation, or unusual radiation, it may be a hypothesis of a much wider application?
A. It could be.

Q. As is demonstrated by the new towns study, for example?
A. As I said, I am a little suspicious of the selection criteria and that sort of thing.

Q. But, as you have agreed, if the Gardner hypothesis cannot explain why the excess was in Seascale, then that too will need some other factor in order to account for the Seascale excess?
A. But I think that the Gardner hypothesis does go a long way to explaining the particular circumstances.

Q. So you say, but it does involve, does it not, the Gardner hypothesis being sufficient to explain why it was, or perhaps still is - we can look at Draper - but why there is an excess concentrated in the village of Seascale?
A. Yes, among the fathers who had high doses.

Q. I think you have agreed, and we are going to look at the Wakeford paper, that if it be the case that there are significant numbers of fathers with high doses elsewhere than in Seascale, with no excess of leukaemia, one would have to find some other explanation for Seascale?

A. Not necessarily. It depends on what you mean by significant numbers.

Q. It depends on the numbers, does it not?
A. Yes.

Q. Just looking at your detailed comments about the Kinlen papers, if we can look very briefly at your paragraph 79, you say:

> "The main weakness of Kinlen's papers arise from their being cross-sectional studies."

Do you mean there geographical or ecological studies, as they are sometimes called?
A. Yes, they are ecological studies.

Q. As, indeed, are many, many of the studies which we have looked at relating to possible clusters round nuclear installations?
A. Yes.

Q. I do not quite understand your next sentence?
A. It is saying the same thing, that he has not done a case control study in which he defined people as individuals who were susceptibles and who then were found to have the disease at a higher rate.

Q. Yes, I see, but I think I am right in saying - we discussed it this morning - that his hypothesis does not, in a sense, postulate that identifiable individuals, because of some peculiarity, are susceptible, but rather that either the local or the incoming population as a whole may be susceptible if it comes up against a viral infection against which it has not built up a herd immunity. Is that not right?
A. If he were to retain that through thick and thin and always hold the position that you could not define it, his hypothesis would no longer be scientific. It would not be a testable hypothesis in the limit.

Q. This is the problem, is it not?
A. But it is a problem, undoubtedly.

Q. It is not, in a sense, a criticism of the studies that he has done. It is simply that the hypothesis which he has come up with and tested, if it is a hypothesis which cannot, at the moment at the least, identify the particular susceptibilities so that it can be linked in with particular individuals, would not be susceptible of a case control study in the same way as, for example, parental preconception irradiation would?
A. It would certainly be more difficult.

Q. You deal with the Cartwright criticisms. Are these a matter of great import? There are a number of them?
A. They are of import, but what do you mean by "great import"?

Q. The problem is simply this: are you aware of the fact that those criticisms by Cartwright, which were in a letter to The Lancet by Cartwright, which I think is only referenced in your report?
A. Yes.

Q. I think it is S. Evans, Reference 36.

MR. JUSTICE FRENCH: So this is E 96?

MR. ROKISON: No, it is not, my Lord, because it is not in the Common Bundle. Because it is only referenced in one expert's report, it will be under Prof. Stephen Evans' references, No. 36.

MR. JUSTICE FRENCH: I have that at the back, but I have not got the letter, have I, or have I?

MR. ROKISON: If your Lordship has a copy of his 36, you will have it in that reference bundle.

MR. JUSTICE FRENCH: Yes, thank you.

MR. ROKISON: It was a letter, my Lord, to The Lancet, 14th January, 1989, from Cartwright, Alexander, McKinney and Ricketts, who are a team we have seen before in that or different order.

THE WITNESS: Yes, one might describe the various Mafias in this field!

Q. MR. ROKISON: This is the Leeds Mafia. Is that right?
A. Yes, migrated partially to Southampton now, I believe.

MR. JUSTICE FRENCH: Yes, you could call them a triad quite happily! Yes, now we are going to your Reference 37. Is that right?

MR. ROKISON: 36, my Lord:

Q. This was comments in relation to the Scottish new town, the Glenrothes study, was it not?
A. Yes.

Q. Which was the first one we looked at, where the authors of the letter say that the Kinlen paper is a valuable contribution to research. I wonder whether you would identify as we go through what it is that you want to adopt, or perhaps you can just tell us. You say:

> "I also consider the criticisms by Cartwright et al concerning Kinlen's data sources for his first paper are valid."

I wondered what criticism you adopt?
A. It is basically from half-way down on the case ascertainment and also on inter-census population projections that are reasonable.

Q. As far as the inter-census population projection is concerned, that was a matter which Kinlen himself had anticipated in the paper and really dealt with, did he not?
A. Yes, but he did it as best.

Q. Yes, he dealt with it at page 1325 in that study and what he effectively said was, "I used the information that was available and I made certain assumptions, but even if you make the worst possible assumption against me, it is still statistically significant"?
A. Yes, the other issue is that for the later time periods, again inevitably, he has depended on mortality data.

Q. Yes, which others had done?
A. Which others have done, yes.

Q. The problem about registration and mortality is that, if you take mortality figures, you remove the possible confounding factor of registration bias or different standards of registration in places that you may be comparing, which we saw in some of the English studies, whereas if you take mortality, as time goes on, since treating leukaemias, as many other cancers, has improved, that the incidence of leukaemia is not always reflected in the mortality data?
A. No, and so some of his deficits that fit in with his theories may be partially associated with improvement in treatment. I agree that the expected values are also calculated that way, but they are minor criticisms. They are not major ones.

Q. It may be that it is a little unfair to call them criticisms. It is really observations which mean that, in those respects, you have to look at it with a little caution?
A. Yes.

Q. Then I would accept that and we can leave it. I do not know whether you are aware of the fact that in a subsequent edition of The Lancet the following month that Kinlen answered the Cartwright paper?
A. Yes.

MR. ROKISON: I do not know that we have in the bundle Kinlen's answer to it, but we have some copies of it, my Lord. I think that there was an administrative error in the fact that nobody referenced it and, therefore, it may be convenient if we could put it in so that your Lordship could put it into the bundle immediately after the Cartwright letter.

MR. JUSTICE FRENCH: Yes.

MR. ROKISON: May we call it 36A?

MR. LANGSTAFF: I wonder if I might have a copy.
MR. ROKISON: Of course, yes.

My Lord, I am not going to take time going through that letter now, especially in view of the very fair answers I have received from Prof. Evans in the last few minutes:

Q. I think we have dealt with your next paragraph, where you say that, even if the Kinlen hypothesis were applicable, it would not necessarily rule out a radiation linked cause operating in synergism with it?

A. No.

Q. And I think that nobody would dispute that that is possible?

A. I agree.

Q. It could obviously be, as Prof. Greaves has indicated, that a viral hypothesis, as postulated by Kinlen, could operate in synergism in the way in which it has been suggested by Prof. Greaves, and we will ask Prof. Greaves about it, but if the Kinlen hypothesis is a hypothesis which operates in places and areas in which there is no unusual exposure to ionising radiation, then, in those circumstances, one would draw the conclusion - it is perhaps obvious - that it does not need ionising radiation in order to operate?

A. Yes.

Q. And, conversely....

MR. JUSTICE FRENCH: Can I just have a pause to note that? If Kinlen hypothesis operates in the absence of radiation....

MR. ROKISON: The way I put it was, my Lord, in places where there is no abnormal excess of ionising radiation.

MR. JUSTICE FRENCH: In the absence of excess of radiation over environment - will that do - that will suggest that it can operate without radiation.

MR. ROKISON: Indeed. It is obvious.

MR. JUSTICE FRENCH: It is nearly obvious.

MR. ROKISON: Yes, but, nonetheless, may be....

MR. JUSTICE FRENCH: I am not criticising spelling it out. Yes?

Q. MR. ROKISON: It would also follow, would it not, that, conversely, if it needs some synergism, interaction with radiation to operate and did so operate in Seascale so as to bring about the excess which undoubtedly exists, or existed, it does not follow that the same cause or causes operated outside Seascale where there was no excess?

A. That went so slowly that I found it difficult, but I think you are quite right. What you are saying is that a possible explanation for the absence of an excess in places where there are high doses elsewhere could well be that the virus infection was not there.

Q. No, could well be that an explanation as to why there is no excess elsewhere is that, in order to produce your leukaemias to any excess, you need a synergism between these two factors?

A. That could be, yes.

MR. JUSTICE FRENCH: Is it sufficiently accurate to compress it by saying you may not need a synergy to produce leukaemia?

MR. ROKISON: No, that is not at all what the witness said, my Lord. It is totally different.

MR. JUSTICE FRENCH: Do not crush me into the ground, Mr. Rokison.

MR. ROKISON: I am sorry. Forgive me, my Lord. I did not mean to be impolite. I am terribly sorry, my Lord, but....

MR. JUSTICE FRENCH: Just tell me where it is wrong. Tell me how I should put it.

MR. ROKISON: It just is not the point I was putting really and it is my fault because - you are quite right - I said it so slowly because I was thinking as I was going along that Prof. Evans lost the train of it anyway as well. May I start again to try to establish....

MR. JUSTICE FRENCH: Yes, please. I will cross out everything I have written on this topic. That is after, "That will suggest that it can operate without radiation."

MR. ROKISON: "That will suggest...."

MR. JUSTICE FRENCH: I will tell you what I have crossed out. It is everything after "If Kinlen hypothesis operates in the absence of an excess of radiation over environment, that will suggest that it can operate without radiation."

MR. ROKISON: Yes.

MR. JUSTICE FRENCH: Right. Now then, what should I write down?

MR. ROKISON: Then I said that, in a sense, conversely, if you need a synergism between, for example, radiation and the Kinlen virus theory....

MR. JUSTICE FRENCH: I shall simply put "radiation and virus".

MR. ROKISON: Yes, if you need a synergism radiation/virus, which operated in Seascale so as to bring about the excess in Seascale, it does not follow that either of the contributory causes applied to cause leukaemias outside Seascale.

MR. JUSTICE FRENCH: Do not go too fast. It does not follow that either of the putative contributory causes?

MR. ROKISON: Yes, if you like, my Lord. Operated to cause leukaemias outside Seascale where there was no excess.

MR. JUSTICE FRENCH: Caused leukaemia outside Seascale in places where there was no excess?

MR. ROKISON: Well, on the assumption, as may be established probably already on the facts, where there was no excess.

MR. JUSTICE FRENCH: But that must be in places where there was no excess.

MR. ROKISON: Yes, indeed.

MR. JUSTICE FRENCH: Or in populations where.....

MR. ROKISON: Places and/or populations. It is populations within areas.

MR. JUSTICE FRENCH: In places and/or populations. Let me read that through, please, to myself. There may have been some independent explanation for the latter situation. I mean, is that the shape of the question?

MR. ROKISON: It could be. Leukaemias occur all the time and everywhere. It is a comparatively uncommon disease, but the fact is that leukaemias occur all over the place:

Q. That is right, is it not?
A. Yes.

Q. And, as we said at the outset, very little is really known about the cause or causes of the disease?
A. Yes.

MR. JUSTICE FRENCH: So would I be right in adding, "It may be due to some utterly different cause"?

Q. MR. ROKISON: That would be right, would it not?
A. It may, yes.

Q. Did you agree basically with the point that I put to my Lord?
A. It is all so very hypothetical.

Q. Yes, it is?
A. If you wish to try and make the same point, you could say that there is radiation everywhere and there is cosmic radiation and it may require that someone has both the infection and happens to be hit by a cosmic shower of the right type, and that might also explain the distribution of radiation.

Q. It is, as you say, very hypothetical and speculative?
A. Yes.

Q. But, subject to that, you would agree with the point that I was putting, would you?
A. Yes.

Q. Thank you. Can I leave, please, the Kinlen hypothesis and its possible application?

MR. JUSTICE FRENCH: With pleasure.

MR. ROKISON: Thank you, and I apologise again, my Lord, for being rude. Having watched "Rumpole" last night, I feel rather chastened! My abject apologies, my Lord! (Laughter)

Q. Just looking very quickly, I do not think it is necessary to deal with your next point. I do not think anybody is suggesting that statistical artefact is relevant in the present case. We have already dealt with 83, being this question of potential sites. I have already asked you about this. This is the Cook-Mozaffari, Darby and Doll paper. Of course, just pausing there, if there is an association - a real association as opposed to an apparent only association - a genuine association between the incidence of leukaemias and sites chosen for potential nuclear installations, which may have - may have - similar geographic characteristics as those which are actually used for nuclear installations, then that would tend to point towards some sort of factor such as Kinlen rather than a factor relating to the operation of the installation, which ex hypothesi was never there in the potential sites?
A. Yes, provided you knew that there were no workers who had been exposed to radiation in those areas, yes.

Q. I can understand that, if there were a number of people who came to live in those areas who had been exposed to radiation elsewhere, then you could not rule out an occupational exposure possibility?
A. Yes.

Q. But, subject to that, you would agree?
A. I would.

Q. I wonder, could we look, I think it is reference A5 in the Common Bundle, which is a paper by Alexander et al in 1990. Is that a paper that you looked at at all?

A. Not for the purposes of any reports.

Q. May I just refer you to it briefly? It is again the same team virtually, again in a different order, I think?

A. Yes.

Q. "High rates of leukaemia in children and young people have been associated with features of community isolation and population growth."

I think that is the Kinlen theory?

A. Yes.

Q. "Incidence data collected by two specialist registries were used to compare incidence rates at ward level with relevant ward characteristics derived from routine census and Ordnance Survey data for England and Wales. An excess risk of childhood acute lymphoblastic leukaemia was found for wards which are farthest from large urban centres. The excess was greatest for wards of higher socioeconomic status and for children aged 1-7 years (the childhood peak), for which a two-fold excess was seen. These findings in general support the hypothesis that childhood leukaemia has an infectious aetiology."

They refer to Kinlen's hypothesis and you see what they do. They take the Leukaemia Research Fund DCS, which is their Data Collection Survey, and the registry of leukaemias and lymphomas for England and Wales, which was published in an atlas form, and they related that to aspects of community lifestyle identified from the 1981 census. As you see, under their methods, the study related primarily to 438 cases of childhood ALL (0-14 years) diagnosed in 1984-88 and registered by the DCS. The Yorkshire Region Children's Tumour Registry is also held at the Leukaemia Research Fund Centre and they applied subsidiary analyses to 200 cases diagnosed in the Yorkshire Health Region. So they did, it appears, a sort of national survey and a local Yorkshire survey:

"ALL has a pronounced peak in early childhood (ages 1-7) and the aetiology for this group may well differ from that for children of other ages. All analyses were repeated for this age range only."

Their "Ward lifestyle indicators", they took socioeconomic status; commuting to work distance; urban-rural status; settlement type, distance to built-up area and isolation; and so on.

What one finds under the Results is, on page 1463:

"The individual effects of each of the primary lifestyle classifications are shown in table I," which is on the previous page. "The risk of ALL was greater in rural than urban wards and higher than lower socioeconomic groups, but neither of these differences achieved significance. The town and village wards had higher risks than the built-up and other wards especially for children aged 1-7. For this age group differences by settlement type were of borderline significance. The risk increased greatly with distance from a built-up area; these differences were highly significant. The analyses were repeated with adjustments for county of residence; most risks were slightly reduced but for distance to built-up areas they were increased.

When these factors were combined in one analysis the effects of urban-rural status and socioeconomic status disappeared and that of settlement classification was no longer significant. However, the effect of distance from a built-up area remained significant. Examination of the DCS data showed that this effect was concentrated in the wards classified as towns and villages. The classifications are highly intercorrelated, with the majority of wards in the group farthest from a built-up area being both of socioeconomic status and classified as villages or towns. The remainder represent a heterogeneous group."

The Discussion, where they say:

"There are many reports of small clusters of childhood leukaemias. Many are anecdotal and uninterpretable, whereas some have been subjected to intense investigation."

No doubt, that includes Seascale:

"A common feature is that, although an environmental leukaemogen is present at low levels, the observed excesses in risk are incompatible with plausible ecological models for dispersion and biological models for exposure and disease induction.

Another common feature is a professional population living in relatively isolated communities."

Then it refers to the suggestion that:

"....disregulation of herd immunity arising from an influx of young professional people into an isolated community produced conditions under which childhood leukaemia was more likely to occur."

It refers to Kinlen and to Greaves, and you will see that this is another, in a sense, variation on the hypothesis, but particularly concentrating on the combination of an

isolated rural area together with a high socioeconomic community?

A. Yes.

Q. One might think that Seascale was really an extreme example, where, as was emphasised in the Gardner cohort studies, to which we have looked, you have a very high socioeconomic class; you have a highly mobile graduate population?

A. Yes.

Q. And where you have a comparatively isolated community with a substantial influx from outside. It really has all those features, does it not?

A. Yes, the difficulty with emphasising the geographical isolation, the higher socioeconomic groups tend not to be isolated. They tend to travel a great deal more.

Q. They do, but, of course, in Seascale, as I say, what you do have is you have an isolated rural area; you have an influx of a population from outside?

A. Yes.

Q. Which is a highly mobile, high socioeconomic class community?

A. Yes.

Q. So that, if one were to try to think of - I am sure there will be others, but if one tried to think of a place which appears to have all these factors coming together, it would be Seascale, would it not?

A. Yes, and that is partly why they present the idea. You cannot have it both ways.

Q. It is not, with respect, is it, simply presented here as an idea that this may be the real cause of the Seascale cluster. It is presented as a more general paper, applying to isolated rural areas as a whole?

A. I think you will find that reference 1 of Kinlen in regard to new towns, and Darby and Doll in reference 17 are both following Sellafield discoveries and Dounreay, so....

Q. Undoubtedly, I do not, of course, quarrel with you at all by saying that this idea, the hypothesis, is effectively a follow-on and a development of the Kinlen hypothesis?

A. You have a circular argument, with respect, Mr. Rokison, that you cannot say that, having thought up the idea in response to the observations, that you can then turn round and say, "Ha, this idea fits in with that observation in Seascale" because it was, indeed, partially the circumstances in Seascale that gave rise to the ideas in the first place.

Q. Surely what it is doing, is it not, is to effectively come up with a hypothesis and to test it in quite a large test by reference to a large number of rural areas?

A. That's an entirely different point. You were asking me to say wasn't Seascale an example par excellence of exactly this situation, but the whole point was that the example par excellence was the thing that led to the theory in the first place.

Q. Where do you find this from?
A. I find it very clearly in the discussion. "Another common feature is a professional population living in relatively ---

Q. Where are you reading from?
A. The second paragraph of the discussion on page 1463:

> "Another common feature is a professional population living in relatively isolated communities. This feature led Kinlen and Darby and Doll to suggest that disregulation of herd immunity ..."

and so on.

Q. But the Kinlen hypothesis was a hypothesis which was, agreed, generated in order to try to consider what might be the causes of the leukaemia clusters at Sellafield and Dounreay. He then tests it in Scotland by references to Glenrothes; he extends it to ---
A. He tests it in an area in which he knew about there being a cluster in Fife.

Q. Maybe, but then he tests it by reference to a larger study of new towns in England?
A. Those are all different points. As far as I understand it, what you asked me was whether Seascale was an example, using my words again, par excellence of such a thing, and I would say yes, but it was that very example that gave rise to these theories.

Q. You may or may not be right. I agree it may have ---

MR. JUSTICE FRENCH: I think this is all pretty marginal anyway, with great respect.

MR. ROKISON: It may be. This is not marginal. Whether or not it was Seascale which generated it ---

MR. JUSTICE FRENCH: This discussion is very marginal.

MR. ROKISON: I quite agree.

MR. JUSTICE FRENCH: Is it circular, is it not circular? With great respect, so what?

MR. ROKISON: That may be the answer and perhaps that ought to be the question, but I will not ask it. Very well, we will leave that, if we may.

Q. I am sorry, I said we are going to leave that, but there is just one point that I would invite you to look at in the text. If one looks at page 1464, perhaps I can just pick it out in the second column of 1464. They say:

> "The modest effect of area socioeconomic status is consistent with previous results".

They refer to Cook-Mozaffari and say:

> "The observation that associations are more apparent with area than with personal socioeconomic status is supportive of an interpretation involving community behaviour. It is widely believed that risk of childhood leukaemia is higher in rural than in urban areas, but typical reports find small non-significant excess risk similar to our own. Interpretations have included ..."

and they set out differences in water supply and so on and so forth. Then he refers to Knox:

> "Knox reported urban-rural differences in the age distribution of childhood lymphoblastic leukaemia in northern England similar to those in the DCS data. It is reasonable to suppose that urban-rural status functions as an inadequate proxy for the isolation that we have highlighted here. We used Ordnance Survey cartographical classifications, which must also be imperfect. The strength of the associations we detected suggests that substantial personal risks may be involved".

So they put it quite high, do they not?

A. They do.

Q. Very well, thank you. Can we now move on and ---

A. Am I allowed, as you have just introduced a report that I haven't seen and not referenced by any of your experts, to say that they also note, "We have already reported a statistical association between mean household radon exposure and county risk..."?

Q. There are a number of hypotheses, are there not?

A. Yes.

Q. Of which this is one?

A. I am sure it your job to be selective.

Q. It may be that performing our respective functions, Prof. Evans, it is our task, each of us, to be selective?

A. I am sorry, I don't regard it as my task to attempt to be selective; I would hope not.

Q. Well you did not come up with this one, did you, with Alexander?

MR. JUSTICE FRENCH: This is not taking us anywhere.

MR. ROKISON: It is not, I know.

MR. JUSTICE FRENCH: Let us move on.

MR. ROKISON: My Lord, I agree. Until I complete my cross-examination in relation to the final figures of Prof. Evans, my Lord, it seems to me that it is of no value to go to his final overall conclusions, because they must depend upon the final figures and what conclusions he seeks to draw from those.

Q. Prof. Evans, may I just deal now with two outstanding topics: first of all, the Wakeford/Parker evidence, that is the statement of Dr. Wakeford appending the Louise Parker draft paper?

MR. JUSTICE FRENCH: I have put away everything except Prof. Evans.

MR. ROKISON: I do not think you even need that, my Lord. All your Lordship needs for the moment is the first statement of Dr. Wakeford which exhibits and appends a draft of a paper by Louise Parker et al.

Q. You were asked about this paper in your evidence-in-chief?

A. Yes.

Q. On Day 14. If I can just ask you generally in relation to the paper and to Dr. Wakeford's statement which accompanies it, I think you understand that the point which is being made in this paper and in Dr. Wakeford's statement is to the effect that the Gardner hypothesis cannot, or at least cannot alone, explain the Seascale excess because there was a comparatively large collective radiation dose outside Seascale; and, not only that, but that there was a large proportion of the workforce who had high doses above 100 mSv outside Seascale; that if one were to apply the Seascale relative risk, dose response relationship, to those outside Seascale, you would expect to find a substantial excess of leukaemias - I think somewhat over 50 - but you do not find any excess at all; and, as I say, for that reason, either the Gardner hypothesis is not truly associated with the excess or it must be acting together with some other factor which does not operate outside. Does that accord with your understanding of the gist of this paper?

A. What I understand the gist of this paper is, is to take the extreme value of the Gardner excess and apply that, and I would have to agree with that, if you take the extreme value of the excess.

Q. The extreme value is, am I right in saying, only taken in respect of greater than 100 mSv, is it not?

A. I must confess, this paper arrived so very late and as such an unpublished thing, I have much greater difficulty with this than with a number of the other things that I have had time to read.

Q. With respect, I understand that difficulty. It is quite important. You were asked about the paper itself and, if I may, I am going to take you through Dr. Wakeford's statement, which is comparatively short, which accompanies it and to a large extent seeks to explain it and put it in its context.

A. I have to confess that I think it is much more relevant to the re-analysis and I don't understand why it has to be dealt with here and now, but that's not my business perhaps.

Q. Perhaps not; that may be a fair comment. It seemed to me that it is relevant to the question as to whether the Gardner hypothesis is applicable to the Seascale excess or explains it, and that is a question which arises either on the Gardner study as published or on your re-working of the figures, so I would like to ask you about it if I may.

MR. JUSTICE FRENCH: Of course you may.

Q. MR. ROKISON: You know who Dr. Wakeford is?

A. I do now, yes.

Q. He sets out the background in paragraph 3. In paragraph 4 he says this:

> "One of the puzzling aspects of the results of Prof. Gardner's study is the implied concentration of risk due to paternal doses in children born in Seascale. This is not what would have been expected from what was known of the geographical distribution of residences of the Sellafield workforce. The majority of the workforce live and have lived in the communities to the north of Sellafield (such as Whitehaven) rather than in Seascale. Also whereas in Seascale the majority of Sellafield employees are professionals, the majority of the industrial workforce live to the north of Sellafield, and it is this latter group of workers who have tended to have the highest doses. This peculiar feature of Prof. Gardner's study was noted by some of the Sellafield workforce at the time of the publication of the Gardner study in February 1990".

That is factual background on which you obviously cannot comment?

A. Yes.

Q. "The feature has also been noted by a number of the expert witnesses in this litigation ..." -

and they are referred to.

"6. As a consequence of the Gardner report a number of studies were proposed. Among these studies was a proposal by Dr. Louise Parker and Professor Alan Craft of the Department of Child Health at the University of Newcastle to identify all children born in Cumbria to Sellafield employes and to follow up these children to determine their status with respect to various health effects."

You agree that that would seem like a sensible thing to do?

A. Yes.

Q. "7. As a consequence of this proposal I suggested at a meeting with Dr. Parker and Professor Craft in 1990 that their study could be conveniently extended to consider the question of the geographical distribution of paternal preconceptional doses and they accepted that this would be an interesting and useful addition to their proposed study.

8. The principal funding for the study has come from the UK Coordinating Committee for Cancer Research".

A. Can I comment?

Q. Yes, please do as we go through.

A. My recollection was - and again you are asking me to do this without having access to people - that there was funding for this that came from British Nuclear Fuels directly, from my recollection, in the discovery documents, and that this aspect was the third of three aspects suggested by Dr. Parker and Prof. Craft.

Q. I think both of those things that you say may very well be right.

A. So the principal funding I would agree with.

Q. Dr. Wakeford was involved with it, and we know that Dr. Wakeford is a BNFL employee?

A. Yes, but there was actually specific money in addition.

Q. That may or may not be the case.

"9. The study commenced in 1990 and, although various aspects of the study are continuing, the data relevant to the geographical distribution part of the study has effectively been completed in the last few weeks."

You are not suggesting that because a British Nuclear Fuels employee was involved in the study or because there may have been some funds provided by British Nuclear Fuels, as they do for a number of scientific researches, that means that its results are therefore suspect, are you?

A. Certainly the Journal of Clinical Epidemiology and some of the other journals require that to be stated so that people can draw their own conclusions, but I am not ---

Q. I am asking you what conclusion you would ask my Lord to draw?
A. What I would draw from that is that I would regard it that to have not mentioned that some funding came from there is being less than fully relevant to the study.

Q. What I asked you was a different question. It was this: are you suggesting that because Dr. Wakeford is a British Nuclear Fuels employee and because it may be - I do not know but it is your suggestion - that some funds, albeit not the principal funds for the study, were provided by British Nuclear Fuels, for that reason it should somehow be discounted?
A. No, I don't think it should be discounted.

Q. "The study commenced in 1990 and, although various aspects of the study are continuing, the data relevant to the geographical distribution part of the study has effectively been completed in the last few weeks. The attached document is a preliminary draft of the report, as there are a (relatively small) number of additional traced fathers who have yet to be incorporated into the final analysis. However, I do not anticipate that this addition of individuals will affect the outcome in any way.

10. The study involved an enormous exercise of computerising the Cumbrian birth register from 1950 to 1989 and examining the personal dossiers of many thousands of Sellafield employees. Using (a) information on birth certificates and (b) personal details of individuals employed at Sellafield, it was possible to match the fathers of children born in Cumbria with men who had been employed at Sellafield. In this way effectively all children born in Cumbria to a Sellafield employed father between 1950 and 1989 have been identified.

11. The only practicable source of dosimetry data for the approximately 15,000 children of Sellafield fathers was the database of annual recorded external whole body radiation dose summaries, the database which was also used by Professor Gardner and his colleagues (albeit Professor Gardner was involved with a far smaller number of case and control fathers) for the purposes of his 1990 study".

We know that for the purposes of this litigation there has been further research which has resulted in amended dose figures?
A. Yes.

Q. For the purposes of this case?
A. Yes.

Q. But in so far as one is seeking to draw any comparison with the results of the Gardner study, then the same database for the Gardner study was used, and of course

you would appreciate that it would be a fairly daunting task to carry out the same, more precise exercise of trying to assess the more accurate doses of the fathers of 15,000 children for the purposes of this study?

A. To the same detail, I imagine yes, very difficult.

Q. For the purposes of making any sort of broad comparison with the Gardner hypothesis and the Gardner study, then it would be legitimate to take the same database, would it not?

A. I don't know enough detail about the database and nobody has offered me the data, so I can't comment.

Q. It was simply that you made an observation about this in your evidence-in-chief?

A. Yes, I would have just expected that one should use a methodology that was as up to date and as appropriate as possible, rather than something that appears to have been agreed, and it is not my own area of expertise to say that there were problems with that.

Q. I do not know whether you know the detail of the evidence?

A. No, I don't.

Q. It has been agreed that as far as external whole body radiation doses are concerned, the film badges were agreed as being as accurate as one could reasonably say at all times after, I think, 1960?

A. Right.

Q. It goes on:

> "12. I understand that for the purposes of this litigation experts have agreed a number of adjustments to pre-1963 recorded photon doses for a number of the fathers who were included in Professor Gardner's study. In addition, I understand that the experts have agreed that for a relatively small number of these fathers neutron doses should be added to the recorded doses. The internal doses have always been recognised as not being included in the epidemiological database (indeed considerable effort has been expended over the past few years to assess these doses with the aim of incorporating them on the database) and again for this litigation the dosimetry experts have agreed internal doses for the 'Gardner fathers'. The adjustments made for the purposes of this litigation are not reflected in the epidemiological database used in this study".

So it is comparable to the original Gardner database, if I can put it that way?

A. Yes.

Q. "13. However, I note from Dr. Strong's agreed statement that in general the contribution of the neutron and internal doses to the testes dose

equivalent assessed from recorded photon doses is small, as are the agreed adjustments to the recorded photon doses."

We can leave that. Then we have this:

"14. The results are set out on pages 11, 12 and 13 of the draft report. The key points are as follows.

15. The Cumbrian birth register contains details of approximately 270,000 live births registered within Cumbria over the 40 year period 1950-1989. 15,308 of these children were linked to males who were employed by BNFL and/or the UK Atomic Energy Authority at Sellafield at or prior to the child's birth. The children of the Sellafield workforce were born throughout West Cumbria, although the majority were born in the communities to the north of Sellafield."

Those have been earlier defined?

A. Yes.

Q. "Of these 15,308 children, 8,886 have a paternal total preconception dose, and 7,244 have a paternal 6 month preconceptional dose.

16. In radiation epidemiology the term 'collective dose' is frequently used."

I think you queried the whole concept of this in your evidence-in-chief, or not?

A. No, I just said that it was an area that I was not familiar with, essentially.

Q. Are you aware of the fact that it is frequently used?

A. None of the papers that we have discussed so far have used it. Would you agree?

MR. ROKISON: It is a phrase that is used in Black, my Lord. It is actually defined in the glossary of terms.

MR. JUSTICE FRENCH: I do not much mind where it was used. Let us move on. Whether he is familiar with it or not, I do not mind.

MR. ROKISON: Very well, my Lord.

Q. You understand the concepts, do you?

A. I believe I do.

Q. It is the total dose to which a particular population may be exposed?

A. Yes, for a group of individuals.

Q. It is a concept which is generally used internationally for the assessment of doses, risk estimates, and so on?

A. Yes.

Q. And recommended limits, do you know that?
A. Yes, in the calculation of recommended limits rather than the application of them, I would have anticipated.

Q. Certainly, but you arrive at collective doses, collective risks, and then you deal with recommended limits, and of course ---
A. I am sure the ICRP documents would be full of that, yes.

Q. Indeed.

> "The collective dose for a group of individuals is the sum of their individual doses. If a particular risk is directly proportional to the dose, then the collective risk for a group will be directly proportional to the collective dose for that group."

That must follow, must it not, if it is directly proportional?
A. Yes.

Q. "To distinguish collective dose from individual dose the unit 'person.Sievert (Sv)' is used as a measure of collective dose. The collective paternal total preconceptional dose associated with the children of the Sellafield workforce is 514 person.Sv, a mean dose of 58 mSv per exposed child."

So what one does is takes the total collective does and apply it as a mean or average across each exposed child?
A. The 8,886.

Q. That is right. Similarly for the six months, it is done over the 7,244?
A. Yes.

Q. "For the 819 children born in Seascale, the collective paternal total preconceptional dose is 35 person.Sv ..."

Q. MR. JUSTICE FRENCH: Can I just hark back a line or two?

MR. ROKISON: Of course, my Lord.

Q. MR. JUSTICE FRENCH: When Dr. Wakeford says "The collective total paternal preconceptional dose associated with the children of the Sellafield workforce is 514 person.Sv, a mean dose of 58 mSv per exposed child", is that - and I am simply seeking information, I hasten to emphasise - per parent of exposed child, or is it really per exposed child?

MR. ROKISON: Your Lordship is right first time. It is more precise to say "per exposed parent of a child whose parent was exposed", so to speak. It is the parent, the father.

MR. JUSTICE FRENCH: Per father?

MR. ROKISON: Per father. What that is doing, as I understand it, is taking the 514 Sv. and simply dividing it.

MR. JUSTICE FRENCH: I think I understand it. I just wanted to make sure that I was understanding fully and correctly.

MR. ROKISON: Yes, your Lordship is quite right.

Q. Then that is compared with Seascale:

> "For the 819 children born in Seascale, the collective paternal total preconceptional dose is 34 person.Sv (7% of the entire collective total dose), a mean dose of 48 mSv per exposed child. Similarly, the collective paternal 6 month preconceptional dose allocated to Seascale is 3 person.Sv (6% of the entire collective 6 month dose), a mean dose of 4 mSv per exposed child. Thus the average paternal preconceptional doses to children born in Seascale were less than the average doses attributable to the workforce as a whole."

If those figures are accurate, that would be true, would it not?

A. Yes.

Q. "For children falling within the highest total and 6 months dose categories (namely greater than or equal to 100 mSv and 10 mSv respectively), 6% and 5% of the respective collective doses are associated with Seascale - again the average for Seascale is less than the overall average".

That is something which comes from the Tables which are at the back of the report, so there is a similar pattern if one takes the high dose categories alone?

A. That would appear to be so.

Q. "17. In summary, the fractions of collective total and 6 month paternal preconceptional doses associated with the births within the civil parish of Seascale between 1950 and 1989 are 7% and 6% of the respective collective doses for births in West Cumbria, reflecting the proportion of all births which occurred in Seascale.

Conclusion

18. As is demonstrated by the discussion section of the draft report, the distribution of the cases that might be attributed to their paternal preconceptional doses, in particular the concentration of cases in Seascale, is clearly at variance with the distribution of the collective dose.

> 19. This may be illustrated by attributing the 4 Seascale leukaemia cases with paternal preconception doses to the collective paternal total preconceptional dose for Seascale offspring of 34 person.Sv which would give 0.12 excess leukaemia cases per person.Sv paternal total preconceptional dose ... Applying this excess absolute risk coefficient to the 472 person.Sv of collective dose in West Cumbria outside Seascale would produce an expected number of excess leukaemia cases of 56 .... This figure is plainly inconsistent with the maximum number of 4 leukaemia cases born in West Cumbria outside Seascale which might be attributable to paternal preconceptional doses".

Do you follow the exercise which has been carried out there?

A. Yes, again it says there are four Seascale leukaemia cases with paternal preconceptional dose, and I understood that there were five.

Q. Whether there are four or five does not make much difference?

A. No, it doesn't make much difference.

Q. In fact, Dr. Wakeford suggests that it would be more inconsistent if you take them as being five. I think what he is doing is taking the ---

A. The four published in Gardner.

Q. That is right, this is based on the Gardner position and the Gardner data?

A. Yes.

Q. I understand now that in the re-working there is an additional case within the high dose category, but you see the exercise that is being done, and if the figures are correct, the conclusion must follow, must it not? (Pause)

Q. MR. JUSTICE FRENCH: Do you want time to reflect on this?

A. I would prefer to have time to reflect on this. I think it is possible.

MR. JUSTICE FRENCH: Very well.

Q. MR. ROKISON: If you want to qualify that answer at a later stage ---

MR. JUSTICE FRENCH: I certainly would like time to reflect upon this before understanding any answer that might be given about it.

MR. ROKISON: Yes, my Lord.

MR. JUSTICE FRENCH: Because Prof. Evans requires time, a fortiori.

MR. ROKISON: I see that, my Lord. I think it is a good idea if I take him through it and put the points to him, and if he wants to come back later, the problem about any piece of litigation is that sometimes witnesses have to deal with evidence which is to be given and therefore has not been explained by the giver of that evidence.

MR. JUSTICE FRENCH: Of course, yes.

Q. MR. ROKISON: It goes on:

"20. This study has generated the group of all children born in Cumbria to fathers employed by BNFL and/or the UK Atomic Energy Authority at Sellafield and receiving an occupational radiation dose before conception, and has used the same, or very similar, pertinent sources of data as those used in the case-control study of Gardner. It shows that the suggestion that the paternal preconceptional doses of children born in Seascale are sufficient to explain the excess of childhood leukaemia cases in the village is incompatible with the absence of any indication of similar excess in the much greater number of children with such doses born outside Seascale.

21. I conclude on the basis of the report that it is highly unlikely that the Seascale childhood leukaemia cluster is due to paternal preconceptional radiation exposure and this must cast further doubt on the causal interpretation of the statistical association between paternal preconceptional radiation exposure and childhood leukaemia reported by Professor Gardner and his colleagues".

Again I will understand if you say that you need further time to think about it, but if the figures are correct, or more or less correct, and if it be the case that there is a comparatively greater collective dose in the population of children with Sellafield fathers outside Seascale, but you do not find a comparable excess or any excess as you do within Seascale itself, then it must, must it not, put a great big question mark against the hypothesis?

A. The difficulty is that the excess at Seascale is a small number of cases in a relatively small population. The excess that might be elsewhere in Cumbria, might be, let me just say if there were a mechanism working, would be less easy to detect because it would be in a much larger population. So I have an uneasy feeling about this study, whereas had what would seem to me to be entirely simple to do, because the person.Sv approach is one that is used where you only have aggregate data, you have no data on individuals, you do not have what each individual has been exposed to, and what I find disturbing about this is that I don't have the individual data that Dr. Wakeford has that says for each person with a particular

dose whether they had leukaemia or not. I think that a table of that would suffice to make me agree with you. So I am struggling with the idea of why - and perhaps, forgive me, I may even have a degree of suspicion that says "Why should you pool together data which has been very very carefully collected and I produced individually?" and then suddenly you throw away all this precise data and throw it into a big pot and count the large numbers. I don't see that that is the right way to analyse the data, so I am a little fearful, shall we say?

Q. May I just pick up one or two points? One is that you said earlier that if you had the same effect happening outside Seascale, then it would be in a larger population and it would therefore be more difficult to detect?
A. Yes.

Q. But if you had the same thing happening outside Seascale to the same degree, based on the same dose response, then it may be in a larger population but it would be a very much larger number of cases, would it not?
A. The cases would be larger, yes, but the relative risk would appear not necessarily to be the same.

Q. It ought to be comparable?
A. It ought to be comparable.

Q. Therefore, if you have a larger population, you will have a larger number of cases?
A. Yes.

Q. If the excess that you would be likely to find in Cumbria outside Seascale would be somewhere over 50, you would be likely to find them, would you not?
A. I haven't thought my way through all of that. I haven't looked at the population numbers in regard to that.

Q. What we do know, of course, is that Gardner carried out a study in relation to West Cumbria, did he not?
A. Yes.

Q. Which includes Whitehaven?
A. Yes.

Q. Which is the large section of population. Have you, in relation to the Gardner figures, considered what the picture is that is presented by the non-Seascale cases and their controls?
A. The non-Seascale cases, I seem to recall we are talking about two, were we, from Black Table 1, 2.1 or something?

Q. What we find in West Cumbria is a very small number of non-Seascale cases?
A. Yes.

Q. Yet, if this evidence is correct, one has ---
A. I would agree with you, but I think you should also have sympathy with me that I find it very puzzling as to why

individual data are not presented. The individual data showing this pattern would be very convincing indeed.

Q. It is suggested that I ask you to look at Table 1, which you will find immediately after the references in the draft. That shows the geographical distribution by reference to maternal residence of the eight leukaemia and two NHL cases included in the West Cumbria case-control study, and known to have a non-zero paternal preconception dose received at Sellafield, so those are those with a dose, is that right?

A. Yes.

Q. What that shows you is that the NHLs are given in brackets, so you have got three, and then in the middle dose within Seascale you have got one, none outside - this is for total - and for greater than 100, you have got three within Seascale and one outside; and you get a similar pattern with the six month paternal preconceptional dose?

A. Yes, I would have to agree that, given what these things are saying, this undoubtedly is potentially very strong evidence, but I am very puzzled why an analysis that appears to be almost opaque to the point of obfuscation has been done. There are some simple methods of analysis that could use the individual data. I believe when I first saw the report I requested to have that data and it was refused.

Q. Forgive me, I am not sure about this - Dr. Wakeford is looking somewhat mystified - but could you be specific as to what it is you suggest should better have been done with the data and what data you require, what you need to know, before you can look at this critically?

A. This is essentially potentially a cohort study, rather like the birth and schools cohort study, of people who are employed and not employed at Sellafield with their doses, and the right way to analyse that, given all the information that we have from the description here, would probably be by a Poisson regression, as it is called, and not a case-control study.

Q. I am sure Dr. Wakeford understands exactly what you are saying.

A. That you should look at a Poisson regression, and it seems to me that that could be very convincing indeed. I am just puzzled as to why it hasn't been done, and when I am puzzled as to why very competent people haven't done what would be the classic analysis for this sort of data, I am left uneasy, I have to just confess that.

Q. I think your position is that you have no experience of dealing in this area of epidemiological studies, as you have said yourself ....

A. I am sorry, I think I do have experience of dealing with cohort studies. I do not have experience of dealing with person Sievert data where you have no individual data, you just look at data over populations, where you look at

things like radon exposures and you do not know what a particular individual living in a county has. That, I agree, I have no experience in but this sort of data, you know in principle what every individual, all these 15,000 children, you know exactly what their fathers had.

Q. But how does it help you - I do not want to canvass this too far but I want there to be clarity between us so that this perhaps can be sorted out before you come back for your further cross-examination, so to that extent it is very useful for us to have canvassed this - we are not quite sure what you are saying is the exercise which should be done - you give it a label, but what exactly are you saying you do? Supposing that you have your however many thousand exposed children there may be, or children of exposed fathers, 8,886, and suppose that you then list out all the total preconception doses of each of those and you find that there were, for example, 40 of them that were over 150, 30 of them over 100 and so on, what does this tell you?

A. It is just that there are absolutely classic methods, described in Breslow and Day, for example, Volume 2, analysis of cancer studies, analysis of cohort studies, that will describe Poisson regression and how you do it, and as it happens the famous package EGRET is able to do, not only conditional logistic regression but is able to do Poisson regression, and I would have expected a design of study like that to have that sort of analysis, and so I have been thrown a little by something of that kind. It is applying methodology that should be applied where you have no knowledge of individual doses, to a situation where you do have knowledge of individual doses.

Q. We hear what you say. Perhaps you will consider further - obviously you say you need more time to consider this and no doubt we will consider that answer and we will produce some response to it.

Q. MR. JUSTICE FRENCH: And I think if you want any data you should ask for it and I am sure that every effort will be made to supply it. I will be jolly surprised if it is not.

MR. ROKISON: My Lord, my instructions are that what we do know, what we do have and what we could provide, although obviously it would be in a numbered form in order to preserve confidentiality - it would not be a question of anybody being identified - we do have on a person by person basis the relevant doses. What we do not have, I am instructed, is doses as to which of those may be cases. We have information as to how many cases there were, but not necessarily being able to relate, to identify the cases from among that data.

Q. For the purposes of doing your regression analysis, would that be sufficient, simply to have a list of all the doses for the fathers of children living outside Seascale, as opposed to those living in, or born in, Seascale? Is that what you would need?

A. No, and I agree, if they do not have that then I can understand why they rushed out to do this ....

Q. It is not a question of rushing out.
A. Sorry.

MR. JUSTICE FRENCH: Tendentious words are being used on both sides and I think should be ignored on both sides.

MR. ROKISON: Very well, my Lord. I will try and exercise self-control, my Lord.

THE WITNESS: I am sorry, my Lord. What it suggests here, in the methodology in Dr. Wakeford's statement at paragraph 10, is that:

> "Using (a) information on birth certificates and (b) personal details of individuals employed at Sellafield, it was possible to match the fathers of children born in Cumbria with men who had been employed at Sellafield."

So the data has been matched to individual children with their birth dates and all the other things.

Q. MR. ROKISON: So I am told, yes.
A. And yet the information regarding the length of time they have been followed up from the work that Craft and Parker have done, in terms of the follow-up of cancers, has not been done, so you do not have any individual information on individual cases.

Q. I am told that exercise is only, at this stage, complete for West Cumbria but not for all those who have had fathers employed at Sellafield?
A. Yes, well I think it would be sensible to present again, given that it has been pointed out so strongly that this is comparison with Gardner, to present that data for West Cumbria.

Q. I think you have made the point, those here have heard that point, and it can be pursued outside Court if necessary through the solicitors or through counsel, in order to be able to clarify what it is that you would like, if you want to carry out any exercise in order to be ....
A. No, I do not think I need to but I am sure that there is a way of doing the analysis that is not difficult.

Q. I see. I think that we will leave that for the moment on the basis that we are bound to have to come back to it, Prof. Evans. Can I lastly take you to the latest Draper paper, please, which is in P4, page 30? This is a paper which I think is the latest draft, and it is the form in which it has been submitted but has not yet been reviewed or published. What Draper et al did, is this right, is not just to cast a critical eye over the epidemiological

findings in the Black Report, but what they did was to continue the examination of the Seascale population and to see whether the increase or the excess in malignant disease continued after the end of the period covered by the Black Study?

A. Yes.

Q. What we find in, I think, Table III, is that the authors summarise cancers of various types among persons aged 0-24 resident in Seascale at diagnosis, from 1953 onwards. Is this right, that the first ten cases were cases which were identified in the Black Report?

A. Yes.

Q. And that relevant cases - I say relevant cases because there were some that were excluded from Gardner on various bases, as we know - up to and including number 10 were included in Gardner?

A. Yes

Q. So that it is numbers 9 and 10 which were the two NHL cases which were considered in Gardner? Sorry, 10 was included within Gardner - the position is that what Draper says about case 10 is that it was wrongly stated as being diagnosed in 1983, when in fact it was diagnosed in 1984, and was included in Black wrongly, ought not to have been included in Black but nonetheless was, but of course Gardner went up to 1985 and it was included within Gardner?

A. Yes.

Q. So that if one is looking at this in order to see how the excess as considered in the Gardner Report continued thereafter, on the assumption that one excludes in any event case 15, it does not matter because that is the Bristol case which either should have been in Gardner or should not, but was certainly in Gardner, and what one is therefore left with, post-Gardner, is four cases, is that right?

A. Yes, from 11 onwards.

Q. 11 onwards being one ALL, one NHL, one Hodgkin's disease and one pinealoma?

A. Yes. I cannot speak exactly as to whether number 10 was in Gardner or not but I think it was.

Q. Number 10 was in Gardner.

A. Yes, all right.

Q. So the cases which you find continuing are four, of which there is one NHL and one ALL, one Hodgkin's disease, which we have discussed before is not generally considered to be related to radiation, and indeed Gardner would tend to confirm that, and the pinealoma case. We see what is said in this report. Of course, one has to be a little careful when looking at this report, if one is seeking to consider the extent to which it either lends support to or does not lend support to the Gardner

hypothesis, in that this report tends to look to the period covered by Black by way of comparison, rather than the period covered by Gardner, which was a longer period which went up to 1985?

A. Yes.

Q. So that if one were looking to see the extent to which it confirms or otherwise the Gardner hypothesis what one finds is, if you look at the bottom of the first page, for the period 1985-90 there is evidence of an excess of total cancer for the group 0-24, that would then be based, he says, on four cases, two cases of NHL - it would be one case of NHL post-Gardner ....

MR. JUSTICE FRENCH: I do not suppose it matters, but in fact it is 1984-90, rather than 1985-90.

MR. ROKISON: No, my Lord, forgive me, what I was saying is this ...

MR. JUSTICE FRENCH: That is what it says.

MR. ROKISON: I know but the point I was seeking to make is this, that what the Draper paper is doing is looking at the period post-Black. That is not the same as the period post-Gardner. This is because the Gardner Study embraced later years than Black, so that the case that we have referred to as being case 10, although diagnosed in 1984, and therefore which ought to have been outside the Black period, was within the Gardner period.

MR. JUSTICE FRENCH: Does that mean that in order to get it accurate I have got to write, "for the period 1984-90", I have got to strike out "4" and put "5"?

MR. ROKISON: If one is considering it post-Gardner, yes.

MR. JUSTICE FRENCH: All right, I understand.

MR. ROKISON: This is not inaccurate, it is simply that if one is looking at this, in order to see whether what has happened since the Gardner Report confirms or otherwise the Gardner hypothesis, is it still operating in other words, but then one has to exclude cases which were embraced within the Gardner period.

Q. Although they mention the ALL case in Table 3, as far as this report is concerned they were taking their study up to 1990?

A. Yes.

Q. So that as far as their study is concerned, instead of having four cases post-Gardner, one has three cases post-Gardner, which is a pinealoma, an NHL and Hodgkin's disease. They say that there is an increase, but a significant increase, of other cancers, that is a pinealoma and one Hodgkin's, in the upper part of the age

range, so that those, as we see, are age 17 and 18, and of course the NHL case is Vivien Hope.

A. I was not aware of that.

Q. One only has to look at the age distribution pattern in cases 1-10, as opposed to cases 11, 12 and 13 to see that - it may just be chance or coincidence - it would appear that there is a different age distribution?

A. Yes.

Q. And whereas in the period studied by Black and Gardner the mainstream, if you like, of your cases, are child ALLs?

A. Yes.

Q. Here there is no ALL?

A. Yes.

Q. And no child?

A. Yes.

Q. And they go on to say that for the immediately surrounding area, the county districts of Allerdale and Copeland, excluding Seascale, and in the remainder of Cumbria, there is no evidence of an increased incidence of cancer at 0-24 in either period, so that the fact which was observed in Black and upon which, as you know, we rely, is something which continues, namely that whatever may be the position inside Seascale there remains no excess outside Seascale?

A. Yes.

Q. They conclude, even when they bring in, as they do for these purposes the other case of NHL, that during both the periods the incidence of malignant disease, particularly lymphoid leukaemia/NHL in young people, was higher than would be expected on the basis of either national rates or those for the surrounding areas. That cannot apply, can it, to lymphoid leukaemia in respect of the second period, post-Gardner?

A. No. Even on that basis the authors of the paper conclude that it seems that the increased risk is unlikely to be due to chance, but the reasons for it are still unknown. I think one can leave the next part, the introduction - on page 3 in the middle they make the same reservation in relation to the studies discussed in the Black Reports, namely that the results were vitiated by a biased selection of diagnostic routes, age groups, calendar periods and areas - and we have discussed that, drawing your parameters round your cluster?

A. Yes.

Q. And they then refer to the time limits of the Black Report. On the methods, coming to page 4, they divide their malignant diseases into categories which do not include NHL with all leukaemias but only lump it together with lymphoid leukaemia, is that right?

A. No, I think when you say all leukaemias, category (ii) is all other leukaemias, so if you were to include with all - they have put it in a category with lymphoid leukaemia and non-Hodgkin's lymphoma and so on.

Q. Indeed, but, for example, myeloid leukaemia would not be regarded as being in the same category?

A. No.

Q. Then one sees the areas which they analysed and they say, at page 5, under "Calendar Periods":

> "Data presented for 1963-83 and 1984-90. The latter period does not overlap with any of the analyses covered in the Black Report."

That should be qualified by the fact that Black actually included one case which was a 1984 case, and that is the one we looked at being case 10?

A. I may be mistaken but I did not think there was an analysis. I think he listed the case but I do not think there were any rates computed using that. It was listed, just as in this instance case 14 is listed but does not appear in the analysis.

Q. I do not quarrel with that. Then they deal, at page 8, with the results, "Cases of Cancer in Young Persons in Seascale since 1953", and they refer to Table 3 and say that they have, as far as possible checked the information for the cases listed in the Black Report, which we looked at, and cross references are given:

> "Ten cases diagnosed during 1963-90 ... are included in the present analysis. One from the most recent period ... is included in the Black report, but with the year of diagnosis given wrongly as 1983 instead of 1984."

That is where I got it from, do you see?

A. That may be, yes.

Q. But you are quite right, they say:

> "This case was notified to the Black advisory group during the course of their investigation and does not appear in any of the analyses .... We have therefore included her in our analysis for the post-Black period 1984-90 since this information should be regarded as testing rather than generating the 'Seascale hypothesis'."

Of course, if what they mean by the "Seascale hypothesis" is the Gardner hypothesis, then of course what one ought to be looking at are cases post-Gardner?

A. Yes, I do not think they mean the Gardner hypothesis because at page 15 they say that the data presented here neither supports nor detracts from the conclusions of Gardner, so they are not studying paternal doses, so it

cannot relate to the Gardner hypothesis. The hypothesis they mean is of an excess in Seascale.

Q. They say why they exclude the Bristol case, which we have looked at, and one finds where they are dealing with cancer incidence 1963-90 on page 9, they say in the second paragraph:

> "Thus there is no evidence over the period from 1963 onwards that the excess found in Seascale extends to a wider area around Sellafield though for the most recent period there is a slight increase in the incidence of lymphoid leukaemia/NHL in the 'rest of Cumbria', particularly in the age-group 0-4 years."

They deal with 15-24, where one finds that there are four cases, of which three were diagnosed in 1984-90, one NHL, one Hodgkin's and one pineal tumour, which we have looked at.

> "In the remainder of Copeland and Allerdale and in the rest of Cumbria the rates are unremarkable."

Then they deal with the higher age group, which I do not think matters. Then one looks to their discussion, at page 11:

> "Two principal questions are considered in this paper. First, do the findings of the Black Report relating to the period up to and including 1983 remain unchanged now that more comprehensive data sets and analyses are available? Secondly, did the excess incidence of childhood leukaemia in Seascale found in the various analyses summarised in the Black report persist in later years?"

They say, in the next paragraph:

> "The conclusions of the Black report are confirmed ...."

- up to 1983, and they conclude that the excess is unlikely to have arisen by chance, and again refer to the omission of case 15. Then they say:

> "For the period before 1984 our analyses rely on much the same evidence as the Black report, though more complete registration data are now available. There is, however, no way of overcoming the objection that analyses of Seascale data for this period are not amenable to any rigorous statistical evaluation because the area, age-group and types of disease to be studied were selected as a result of the observed 'cluster'."

The same point again as we have seen before?

A. Yes.

Q. They say:

> "This criticism cannot be applied to the results for 1984-90 ...."

because they did not know that there was a cluster there and they were looking to see if the cluster was continuing, and:

> "Even the case from this period that was included in the Black report (with the year of diagnosis wrongly given as 1983 rather than 1984) was diagnosed after concern had been raised about the high incidence in Seascale. For the age group 0-24 there is an excess of malignant disease which is highly unlikely to have arisen by chance. These more recent data therefore strengthen the suggestion that there is an increased incidence in Seascale for the age group 0-24 years though, while the original findings relate mainly to lymphoid leukaemia at ages 0-14, there were no leukaemias and only one case below age 15 during 1984-90."

That is only if one takes into account the 1984 case that was also in Black, otherwise there is none, and they say:

> "Of the four cases found in either period two had NHL, one had Hodgkin's disease and one a pineal tumour; the excess is mainly attributable to NHL. We have excluded from these analyses case 14 ...."

- because it was too late -

> "As regards other cancers ... there is a small, non-significant excess ... if the whole period is considered.
>
> There is no evidence that the raised incidence in Seascale extends to the two county districts nearest to Sellafield or to Cumbria generally."

So that the pattern to some extent continues and to some extent it is different. It continues insofar as there is an excess of malignancies in Seascale, and it continues insofar as there is no excess in any area outside Seascale, in any local area outside Seascale?

A. Yes.

Q. It is different, insofar as the predominant type of leukaemia or disease is different?

A. Yes.

Q. And it is far more diverse, heterogeneous, and also the age distribution is markedly different?

A. Yes.

Q. They then discuss the possible hypotheses to account for the findings. They first of all deal with chance and

consider that is unlikely and, as you have, they say it could have been by chance when the original cluster was discovered, but this further increase tends to count against the suggestion that it is chance, that is right, is it?

A. That is right.

Q. They say that secondly they deal with environmental radiation, and they suggest that the doses delivered to the child or foetus were far too low, and I think you said that you were inclined to agree with that, in your evidence-in-chief?

A. With the proviso unless either the discharges were grossly underestimated, or the assumptions made in computing the risk were grossly incorrect.

Q. Yes. Then they deal with Gardner, and over the page they say:

> "Again, the level of risk implied by this explanation ...."

- that is the Gardner hypothesis -

> "... seems inconsistent with the dosimetry and previous estimates of genetic risk."

Do you agree?

A. Yes.

Q. "It has been suggested that the measured dose of external radiation may in fact be a surrogate measure for internal exposure to radio-nuclides or to chemicals; such alternative explanations are still open to the objection that there is no generally accepted human data to support them."

Agree?

A. Yes.

Q. "The present analysis includes the geographical area covered by Gardner but follows it too closely in time to provide data to test his findings ...."

Agree?

A. Yes.

Q. And they make the point that only cases 12-14 in Table 3 were diagnosed after the period covered by Gardner, and this is perhaps of more importance:

> "... moreover all three were conceived before the parents moved to Seascale."

A. Yes.

Q. We know, in fact, that one of those is Vivien Hope, whose father did have a dose, even though she was not born in Seascale?

A. Yes.

Q. But we can look and see, and I have no doubt that you have looked at Dr. Wakeford's Second Report, when he gives certain information in relation to the doses of the others?

A. I happened to spot Dr. Wakeford's Second Report in the documents today. I had no idea of its existence.

Q. Then it refers to McLaughlin and I am not going to ask you about that at the moment. They then say:

> "Little is known about risk factors for childhood leukaemias and lymphomas."

and you agree with that, I think?

A. Yes.

Q. They refer to the Kinlen viral hypothesis, over the page, and they refer to the studies suggesting that childhood leukaemia is more common among higher socio-economic groups and suggest that the risk of childhood ALL is doubled in isolated towns and villages, and I think that is actually a reference to the Alexander paper, at which we looked earlier this afternoon, isn't it?

A. Yes.

Q. "In conclusion, 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."

The position, therefore, as far as Draper is concerned is that if one is considering whether the Gardner hypothesis is the right explanation or an explanation for the Seascale cluster, that the Draper paper really takes the matter no further?

A. Yes. It takes the matter fractionally further in that we agree that it removes statistical artefact as a likely cause, but we are agreed on that, so ....

Q. It makes the existence of the cluster less likely, you say, to be due to chance?

A. Yes, but it does not directly affect the Gardner hypothesis one way or the other.

Q. And if one were to find, as the second Wakeford paper suggests, that apart from the case of Hope none of the other further cases had any significant doses, then it would tend to suggest that to the extent to which the excess continued, it was not due to the operation of the Gardner hypothesis?

A. You are asking me to comment on a report I have not seen so ....

Q. Perhaps I can ask you just to look at it then. This really arises, I think, in response to - unfortunately what has happened in this case, which may be a good idea because it puts cards on the table, but reports are served in response to what is said in other people's reports, and I think that you said in your Fourth Report, in the last sentence, that:

> "The study is compatible with preconception radiation exposure playing a causal role in the cancer excess, although this is not directly addressed in the study because there is no data on paternal occupational exposures given."

The answer is, it may or may not be compatible depending on what the exposures were, is that right?

A. Yes.

Q. I think it was in response to that this was then served, and Dr. Wakeford says this:

> "The objective of the Draper paper was to examine the incidence of leukaemia and other malignancies in young persons resident in Seascale and the surrounding area ...."

and so on, and he refers to that as the 'post-Black period'. I do not think it is necessary to read further from paragraph 3. In paragraph 4 he refers to Table 3 and to the cases there set out. He points out that the:

> "... four post Black period cases occur in young adults ... rather than in children as occurred predominantly in the period studied by the Black Group."

and he points to the variety of different malignancies which we have looked at and there is no dispute between us. Under "Paternal Preconception Doses", he says:

> "As stated in my first report one of my areas of study over recent years has been the incidence of cancer in young persons in Seascale and I am aware of the identities of all fifteen cases presented in Table 3 ...."

Perhaps one ought really to have the Draper paper open so that one can simply cross-refer. It is Table 3 of Draper. Dr. Wakeford said:

> "I am aware that cases numbered 3, 5, 6, 8, 10 and 15 were included in the Gardner Study ... and that all of these cases have a paternal preconception dose. Cases 2, 4 and 9 were not included in this study because they were born outside West Cumbria. Cases 1 and 7 were not included ... because they were cases of malignancies other than leukaemia or lymphoma."

A. Yes.

Q. He says:

> "These two 'other malignancy' cases ...."

- namely 1 and 7 -

> "... do not have paternal preconception doses, Case 1 because he was born in 1948 ... and Case 7 because her father was not employed at Sellafield."

Then "Post-Black Cases":

> "The father of Case 11 ...."

- which is the pinealoma -

> "... was employed during the preconceptional period by the UKAEA at Winfrith. He was later transferred ... to Sellafield and his dose record shows a whole body total preconceptional dose of less than 5 milliSieverts and a six month preconceptional dose of less than 1.8 milliSieverts."

So small dose. Then:

> "The father of Case 13 (Hodgkin's disease ...) worked during the preconceptional period ... at Risley. He was later transferred ... to Sellafield and his dose record shows a whole body dose of less than 0.5 milliSieverts ...."

The father of Case 14 which is mentioned but not analysed because it arose after the period covered by the Draper Study, which was an ALL, although not a child ALL, was not employed in the nuclear industry prior to the case child's conception.

> "Dr. Draper has identified four cancer cases diagnosed in Seascale during the post-Black period. Of these four cases, two were 'other malignancy' cases and I have shown that in both these cases there is a low paternal preconceptional dose. In respect of the period studied by Black, there are only two 'other malignancy' cases diagnosed in Seascale and both these cases have zero paternal preconceptional doses."

He therefore concludes that your suggestion that:

> "... paternal preconceptional radiation is a cause of leukaemia and can account for the excess of childhood leukaemia cases in Seascale, extends to or applies also to other malignancies diagnosed in young persons resident in Seascale both in the Black and post-Black periods ... this speculation cannot be supported."

Would you agree that the last sentence of your report, if these figures are correct, that even though there may have been an excess of malignancies after the Black period, it is not an excess which would be explained by the Gardner hypothesis?

A. Yes.

MR. ROKISON: I apologise for having taken such a long time, but I think that is all that I wish to ask you at this stage. I want to ask you about McLaughlin. I want to ask you about your re-working of Gardner and perhaps come back to the Wakeford and Louise Parker point when we meet again, which we will try very hard to fit in at your convenience some time this side of Christmas.

MR. LANGSTAFF: Let me tell your Lordship by way of outline, the areas which it is intended that my re-examination will cover. Before I outline those areas, let me put it in context of time. My Lord, my learned friend Mr. Rokison when he began to look as though he might exceed his original estimate to me, was kind enough to agree that if I should be left with insufficient time to re-examine, that the appropriate thing to do in the interests of Prof. Evans and progress, would be to reserve those aspects I cannot cover today until Prof. Evans returns.

I am grateful to him for that and I told him in return that although he might be subject to a certain amount of general teasing from our side, we would not in any way seek to hurry him, and haven't done so.

My Lord, it leaves me in this position that there are a number of areas that it is plainly very important I should cover as soon as possible and I wish to indicate those areas to your Lordship so your Lordship knows what areas I am going to cover eventually. I am deeply conscious of the fact that when re-examination takes place after an interval, when there has been time to recollect, the impact may not be as great as when it comes immediately after the evidence has been given.

My Lord, the first area that I intend to cover, and I hope to cover this today, although it may take even though this is Friday, a little after 4.15, is to deal with the suggestion, for such it was when Mr. Rokison first made it and it has never been accepted by the witness, that the excess is limited to Seascale. I hope to show that is a complete misinterpretation of the data and a misunderstanding of what the various papers show.

My Lord, the second area which I propose to look at is the question of non-Hodgkin's lymphoma and whether or not the evidence available allows one to say, on a balance of probabilities, that is caused by radiation from Sellafield.

MR. JUSTICE FRENCH: With a view, apart from leukaemia?

MR. LANGSTAFF: My Lord, yes. NHL as distinct from leukaemia is caused.

MR. JUSTICE FRENCH: Viewed not together with leukaemia but viewed apart from leukaemia?

MR. LANGSTAFF: My Lord, the way I put it as distinct from leukaemia, so, yes.

MR. JUSTICE FRENCH: "As distinct from" means that you are not, in doing your figures, adding leukaemia to it?

MR. LANGSTAFF: My Lord, I think it is interpretation and argument in the light of all the circumstances rather than the figures.

Perhaps I should deal with that when I come to the questions.

MR. JUSTICE FRENCH: I am finding it difficult to see around what concept your are drawing your line.

MR. LANGSTAFF: Perhaps I can put it this way - I am conscious I must not stray too far into speech, as it were, as the witness is there.

MR. JUSTICE FRENCH: Well, do anything you need to assist my understanding, please.

MR. LANGSTAFF: My Lord, it was put to Prof. Evans that if one looked at the Gardner occupational data alone, one could not tell from that, statistically, that NHL was caused by parental preconception exposure. He then went on to accept that when looking at that alone one could not, on any philosophical basis, and by that he meant...

MR. JUSTICE FRENCH: I have got a note of it and I remember it all very well, Mr. Langstaff.

MR. LANGSTAFF: My Lord, that was looking at it alone and I shall ask him questions which I would hope would show your Lordship that looking at it not...

MR. ROKISON: My Lord, I know your Lordship has said if there is anything that makes it clear to your Lordship it would be of assistance, but for my learned friend to indicate in advance what areas he is going to cover and what answers he hopes to get, really does seem to me to be, with great respect, going beyond the bounds of starting a re-examination. He is now getting into saying what distinctions he is going to invite the witness to draw. That cannot be right.

MR. JUSTICE FRENCH: I am only trying to understand whether the topic you are going to re-examine upon is the proposition that NHL, viewed in isolation, is not

statistically significant. Is that the topic, or isn't it?

MR. LANGSTAFF: My Lord, with this qualification, that your Lordship says, "statistically significant", my Lord I hope to cover what that means with my next witness, but that it is a question of interpretation whether, in Prof. Evans' view, he can say...

MR. JUSTICE FRENCH: Well, I have tried and failed and it has made Mr. Rokison cross, so let's move on!

MR. LANGSTAFF: My Lord, I am sorry. I am trying to tread a very careful line and I am conscious of what Mr. Rokison has said. My Lord, I will then deal with the other studies and the question, in particular, of the Aldermaston and Burghfield areas.

MR. JUSTICE FRENCH: The other studies and what support, if any?

MR. LANGSTAFF: Yes. Then with case 106, and then with the various criticisms of the methodology of the Gardner study that have been put forward. My Lord, there may be a number of points which could properly be called miscellaneous which will come after that.

There is one matter which perhaps I should raise now. It ought to be a matter of agreement between the parties, because those who have been instructing me prepared for the purposes of your Lordship in the hope that it might assist, a table showing all the cases of whom we have information who were referred to in Black, in Gardner, and in Draper, who are known to have been diagnosed in and around Seascale. The information has been agreed by the Defendants but for some reason they will not permit those instructing me to have it put before the court, so I am told. My Lord, that is a matter of some disappointment. I hope it will be possible to agree, and I say this in court. In fact there were 4 NHLs in total diagnosed in Seascale from 1950 to 1989; 2 in the period pre-Draper and 2 in the Draper period.

My Lord, I say that openly and I hope that that may be possible to be the subject of agreement before we come back before your Lordship.

MR. JUSTICE FRENCH: Yes, we must wait and see, but it is not strictly a matter for re-examination?

MR. LANGSTAFF: My Lord, no. May I then deal with the first of those matters, the question of whether the excess is limited to Seascale?

Re-Examined by MR. LANGSTAFF:

Q. Would you take the Black study? Prof. Evans, it was put to you on a number of occasions that the excess of leukaemias was limited to Seascale and there was no excess outside Seascale. It was put to you on a number of occasions as though that was something you had accepted, although the transcript showed you didn't, and it was said by learned friend just this afternoon, that it had been established, or almost established, or as good as established, that that was the case. Could I ask you to turn to page 26? There we see paragraph 2.29, Table 2.11. It appears to come from Urquhart 1984?

A. Yes.

Q. Could I ask you to put your finger in page 13, Table 2.1, whilst maintaining your place at page 26? Table 2.1 shows us the cases of leukaemia resident in Seascale since 1955 and aged under 25 years at diagnosis?

A. Yes.

Q. We can very quickly look through 14, 15 and 16, and see there are separately listed cases of leukaemia in Millom Rural District, case of lymphoma resident in Millom Rural District. That includes cases of lymphoma in Seascale. Also cases of solid tumours resident in Millom?

A. Yes.

Q. So a distinction is made between table 2.1 and table 2.2. Table 2.1 includes Seascale, table 2.2 excludes Seascale when we are dealing with leukaemias?

A. Yes.

MR. JUSTICE FRENCH: I am sorry, I wasn't alert to the fact we were looking at 2.2, as well as 2.1.

MR. LANGSTAFF: It is simply to point out...

MR. JUSTICE FRENCH: The result is I have not been following what you have been saying with regard to table 2.2. I have my finger in table 2.11 for some reason.

MR. LANGSTAFF: My Lord, I am going to go back to that.

MR. JUSTICE FRENCH: So let's start the exercise again, comparing 2.2 with 2.1.

MR. LANGSTAFF: My Lord, yes:

Q. Is it right, Prof. Evans, that table 2.2 deals with cases of leukaemia in Millom other than cases in Seascale?

A. Yes.

Q. Table 2.1 therefore shows us all the cases about Black was aware in Seascale alone?

A. Resident in Seascale alone.

Q. Keeping your finger at table 2.1, would you go with me to table 2.11? Does this deal with deaths as opposed to incidence?
A. Yes.

Q. If one looks at the bottom of the table does one find all leukaemias dealt with and the second from last and the last boxes to the right deal with Millom Rural District and Seascale and coastal villages?
A. Yes.

Q. It appears, as one looks across the table, working from left to right, that there is a narrowing down. You start with England and Wales. You then go to Copeland - Copeland being the area which includes Millom and Millom in turn includes Seascale and the coastal villages, as there defined?
A. Yes.

Q. We know that the figures in brackets after the rate which is shown in the table are the numbers of deaths upon which those rates are based?
A. Yes.

Q. It would follow that from 0-24, between 1963 and 1982, there were 4 deaths in Seascale and the coastal villages and 8 in Millom as a whole?
A. Yes.

Q. Seascale and the coastal villages, is that defined in the note underneath the table?
A. Yes.

Q. The coastal villages and Seascale are the five coastal parishes, which includes Seascale itself and four others. If you turn, keeping your finger at 2.11, turn back to table 2.1, it is possible to identify the deaths between 1963 and 1982 from leukaemia in Seascale to those under 25, is it not?
A. Yes.

Q. If one does that, one doesn't find 4, I think one finds 3. "Year of death" is the central column?
A. Yes.

Q. We can see there is a 1979, 1970 and a 1971 death?
A. Yes.

Q. So it follows, going back to table 2.11, that there were 3 deaths in Seascale and the coastal villages, and 5 other deaths in Millom Rural District?
A. Yes.

Q. Because, of course, Millom Rural District includes Seascale?
A. Yes.

Q. Now that being...

MR. ROKISON: My Lord, it is wrong because it is Seascale and coastal villages, and it just goes to show how, in re-examination if all you get from the witness is "Yes", it does not actually amount to anything. I object to leading questions because they don't help my Lord, not from any form at all.

MR. JUSTICE FRENCH: Yes.

Q. MR. LANGSTAFF: Prof. Evans, would you look at Millom RD at the bottom of the column there?
A. Yes.

Q. What relative risk is shown there?
A. 6.6 is not a relative risk but is deaths per 100,000 years.

Q. I beg your pardon. Is that, or is it not, greater than the rate for England and Wales?
A. Yes.

Q. What does that suggest to you about the rate in Millom Rural District compared to the rate in England and Wales?
A. It suggests to me that the rate in Millom Rural District is higher than that in England and Wales and would also be higher than that in Cumbria.

MR. JUSTICE FRENCH: Millom RD shows death rate 6.6 (8). This is higher, both than England and Wales, and did you say Cumbria or West Cumbria?

THE WITNESS: I was adding something that wasn't there and that is my knowledge that the death rate in Cumbria is lower than that for England and Wales as a whole.

Q. MR. JUSTICE FRENCH: Yes, I appreciate that, but it was Cumbria and not West Cumbria?
A. It was Cumbria and not West Cumbria.

Q. MR. LANGSTAFF: Would you turn now to table 2.13 on the facing page? Is this a table drawn from the investigations of Palmer, which compares "Observed" and "Expected" leukaemia deaths in Millom during the same period, 1963 to 1980?
A. Yes.

Q. I stand corrected. It is not the same period, it is two years less. Does it divide up the comparison of observed and expected deaths between the five coastal parishes on the one hand and the rest of Millom Rural District on the other?
A. Yes.

Q. If one looks at the rest of Millom Rural District, how many deaths does it appear were observed between 1963 and 1980?
A. Four.

Q. How many does it show were expected?
A. 1.62.

Q. Mathematically does that show that two and a half times as many were observed as expected?
A. Yes, but it is not statistically significant.

Q. MR. JUSTICE FRENCH: It is not what?
A. It suggests it is not statistically significant.

Q. MR. LANGSTAFF: When it says "p=0.08", in what percentages of cases would you expect to see a finding as high as that?
A. Eight percent.

MR. JUSTICE FRENCH: Just a moment. The rest of Millom RD shows 4 leukaemias observed, 1.62 expected, not statistically significant, but chances against...

MR. LANGSTAFF: I think he gave the chance for. I think he said it was 8%.

MR. JUSTICE FRENCH: The probabilities of chance, 8 over 100?

MR. LANGSTAFF: My Lord, yes:

Q. Can we do the same exercise for the five coastal parishes, as we did for the Urquhart paper? If one looks at the five coastal parishes, how many deaths were there observed?
A. Four.

Q. If one goes back to table 2.1, can you tell me how many deaths there were in the coastal parish of Seascale as opposed to the other coastal parishes?
A. Three.

MR. JUSTICE FRENCH: Five coastal parishes, 4 deaths of which (see table 2.1) were in Seascale.

Q. MR. LANGSTAFF: Can you say, without doing the calculations, is it possible to say anything about the likely statistical significance of there being 5 deaths in the period in Millom Rural District outside Seascale?
A. I think it is likely that that would be statistically significant.

Q. MR. JUSTICE FRENCH: I am sorry, that which will be?
A. That 5, when something around 1.7, shall we say, is expected, because I am adding the 1 death that has occurred outside Seascale in one of the other coastal parishes, to the 4 that were in the rest of Millom Rural District.

Q. Adding 1 outside Seascale to the 2 in the 5 coastal parishes, is that right?

A. No, there are 3 in Seascale and 4 in the 5 coastal parishes. That means there is 1 in the coastal parishes other than Seascale.

Q. Other than Seascale, yes, I see.
A. But they aren't included in the rest of Millom. If you add that 1, and were doing, as it were, a table of Seascale versus rest of Millom RD.

Q. So 1 outside Seascale to those in the remainder of Millom, right?
A. Yes.

Q. Namely?
A. Namely 4.

Q. Namely 4.
A. That the expected value will be approximately 2, and it is likely that that would be statistically significant.

Q. MR. LANGSTAFF: What do you say, having looked at the figures of Urquhart and the figures of Palmer, as to whether the leukaemias, the excess of leukaemias, was limited to Seascale?
A. I think there is some evidence that it is not limited to Seascale, but extends to the rest of Millom RD.

Q. Would you look now at page 29?

MR. JUSTICE FRENCH: Just a minute - "I think there is some evidence (see Urquhart and Palmer) that the 'cluster'" can I call it?

MR. LANGSTAFF: My Lord, yes.

MR. JUSTICE FRENCH: "Is not confined to Seascale, but extends to Millom RD."

Q. MR. LANGSTAFF: Would you turn to page 29? Would you look at the table at the top, table 2.16? We are told this derives from the researches of Gardner and Winter in 1984
A. Yes.

Q. Would you look again at the figures for Millom? Can I take you to the figures in respect of leukaemia, 1968 to 1978?
A. Yes.

Q. Does it show that 6 deaths were observed when 1.4 were expected?
A. For ages 0-24, yes. It is the bottom row of table 2.16.

Q. There are two time periods, 1959 to 1967 and 1968 to 1978.

MR. JUSTICE FRENCH: Aged 0-24, and all ages above that. Now what I am looking at is 1968 to 1978, is that

right, and I then go to Millom RD and I find "Observed 10", is that right?

MR. LANGSTAFF: No. Your Lordship is looking at cancer, I think. If your Lordship goes down...

MR. JUSTICE FRENCH: I should go to leukaemia.

MR. LANGSTAFF: My Lord, yes. It is the very last three entries on the right hand side.

Q. MR. JUSTICE FRENCH: So it is "Observed 6, Expected 1.4"?
A. Yes.

Q. MR. LANGSTAFF: Can we turn back to table...

MR. JUSTICE FRENCH: Can I just note that if it is important - 1968 to 1978, leukaemia, Millom RD, observed 6, expected 1.4.

THE WITNESS: That is essentially a relative risk of 4.35. They have expressed it as an SMR.

Q. MR. JUSTICE FRENCH: Of 4.35?
A. Yes.

Q. MR. LANGSTAFF: If one goes back to table 2.1, one can again go through the exercise of excluding the Seascale cases. What does table 2.1 show you about the deaths between 1968 and 1978 in Seascale?
A. There are three deaths.

Q. Would you look again and make sure that answer is right?
A. 1968 to 1978? Oh, sorry, 4 total. Place of death Seascale, do you mean?

Q. No. I am looking at the years of death. How many deaths were there between 1968 and 1978?
A. I'm sorry, 2.

Q. 1979 is outside. 1970 and 1971, and both of those are in Seascale?
A. Yes.

Q. If one returns with that in mind to table 2.16, what can one say about the deaths outside Seascale in the Millom Rural District in the period 1968 to 1978?
A. That it would appear there are 4.

Q. Is 4 greater than 1.4?
A. Yes, but you should not use 1.4 as the standard. You should use a number rather smaller.

Q. If we are excluding Seascale?
A. Yes.

Q. Even if one takes 1.4, would that be, or would it not be, an excess?

A. It would be an excess.

Q. It was put to you that there was no excess in Ennerdale. Would you look at the other half of the Gardner and Winter paper, the top of page 29?
A. Yes.

Q. For leukaemia deaths between 1968 and 1978, how many were observed?
A. At ages 0-24, four.

Q. How many were expected?
A. 3.3

Q. One has an SMR of 121, which indicates a relative risk of what?
A. 1.21.

Q. Is that or is that not an excess?
A. Yes, but it would appear that it is not statistically significantly different from 100.

Q. The text deals with this, I think, and the paragraph 2.31.

MR. LANGSTAFF: My Lord, I shall not trouble with that for present purposes:

Q. Leaving Black, I want to take you to one other paper, I am afraid, this evening, if I may. Can I say, having looked at the information there from those three studies reviewed, what do you say about whether the excess is or is not limited to the village of Seascale?
A. I think there is some evidence it is at least in the rest of Millom Rural District, that it may possibly extend to Ennerdale, but the evidence is not as strong for Ennerdale.

Q. The next paper I want you to look at, and it will be the last I take you to today, if I may, is C 42.

MR. LANGSTAFF: My Lord, it is the large Cook-Mozaffari study and your Lordship can put away Black.

MR. JUSTICE FRENCH: Yes, what are we moving to?

MR. LANGSTAFF: C 42, the large Cook-Mozaffari study:

Q. Before I ask you to open it up, can I ask you this?

MR. JUSTICE FRENCH: I am so sorry, I keep going to C 29-51 when I should be going straight to C 42. Right, I have got it.

Q. MR. LANGSTAFF: Can I begin by asking you this, Professor? After 1968, in examining the rate of

leukaemia in England and Wales, is it more appropriate to look at death rates or incidence rates?

A. I would judge that it is better to look at incidence rates.

Q. Are death rates able to tell us anything about persons who have caught the disease and have been treated and cured?

MR. JUSTICE FRENCH: Yes, I have got the picture that methods of curing leukaemia have greatly improved.

Q. MR. LANGSTAFF: Would you turn to Table 3, page 178? Is this a table, Professor, of all leukaemia, persons aged 0-24?

A. Yes.

Q. If you look at the bottom of the page, because that is the half of the page that you were taken to by Mr. Rokison. He established with you, and there is no dispute between us on this, that there are four areas set out within six miles. There is a blank for Seascale?

A. Yes.

Q. Sellafield, I should say, because there was no local authority area which fell within six miles?

A. With at least two-thirds of its population within....

Q. Yes, you are quite right. We were told the second column was Ennerdale; the third column was Whitehaven; and the fourth column was Millom?

A. Yes.

Q. You were taken to the bottom half of the page and you agreed that those figures there showed no excess so far as Sellafield was concerned, compared with its control. Do you remember the INS was the installation area?

A. Yes.

Q. And CON were the control areas?

A. Yes.

Q. What you were not taken to was the top of the page. Let me ask you about that. Is that an incidence table?

A. It is.

Q. Do we see three figures for Ennerdale - installation figures, observed, I think, OBS. Do you follow me across to the second column to the right?

A. Yes.

Q. OBS, that is the number observed, is it not?

A. Yes.

Q. So we see that in Ennerdale there were nine observed compared with six in the control area?

A. Yes.

Q. And presumably the SMR depends upon the exact numbers of populations in the subject area and the control area?
A. A. They are each related to a national standard for the age distribution of children and young people aged between 0 and 24.

Q. What do those figures, 140.7, tell us about Ennerdale?
A. That its risk of incidence is raised by 40 per cent, a relative risk of 1.4.

B

Q. What does that tell us about whether there is possibly an excess in Ennerdale?
A. That is some slight evidence of an excess in Ennerdale.

Q. MR. JUSTICE FRENCH: Wait a minute. I have got to imagine that Ennerdale is written above the second column?
A. Between 6 and 8, yes.

C

MR. LANGSTAFF: My Lord, yes. My Lord, it may be helpful simply to write Ennerdale, then Whitehaven, then Millom.

MR. JUSTICE FRENCH: I think I will do that. It is difficult to write with your hand on a big hoop! The next one is Millom.

D

MR. LANGSTAFF: My Lord, the first one is Ennerdale, the second one Whitehaven and then Millom.

MR. JUSTICE FRENCH: I have got that. Ennerdale column shows incidence 140 against 94:

E

Q. Is that right?
A. Yes.

Q. Which equals a 40 per cent increase, which may suggest an excess in Ennerdale?
A. Yes.

Q. MR. LANGSTAFF: If you go to the Whitehaven column, the next column, what does that tell us about whether there is or may be an excess in Whitehaven?
A. It is certainly consistent with the idea there could be an excess in Whitehaven. Certainly more than expected.

F

Q. How much more than expected?
A. About 60 per cent more.

G

Q. MR. JUSTICE FRENCH: Consistent with an excess in Whitehaven?
A. It is consistent with.

Q. MR. LANGSTAFF: And if you go to the next column, the Millom column?
A. Yes.

H

Q. What does that suggest about the whole of Millom?
A. It suggests for the whole of Millom that the relative risk is at least 2.8, 2.88.

Q. What do you say about there being an excess in Millom, on the basis of that?
A. That will include Seascale, but there is evidence for there being an excess in Millom.

Q. Can you very quickly, if you please, leave page 178 and go to page 91. This "Areas with at least two thirds of population resident within 8 miles", one could write at the top Ennerdale, could one not, on the same basis?
A. Yes.

Q. We are looking here at the figures for all malignant sites broken down into five year periods?
A. Yes.

Q. Broadly, one can see the same figures across the period 1961-65, 1966-70, 1971-75, 1976-80. Can one summarise very briefly for each of those periods whether this shows any excess of all malignancies for Ennerdale during those various periods?
A. This suggests that, for three of the four periods, that compared with England and Wales, there is an excess and compared with the control areas, there is an excess in every group of years.

Q. Page 95, if you would?

MR. JUSTICE FRENCH: Shall I make a note of this?

MR. LANGSTAFF: My Lord, if your Lordship would.

Q. MR. JUSTICE FRENCH: So this is Table 2, continuation, page 91. What is it that I am taking to connote Ennerdale? The entire table?
A. Yes, it is the area with at least two-thirds of population resident within 8 miles, so it is Ennerdale for the Sellafield row. It does not apply to Springfields and Capenhurst and so on.

Q. Yes, so that what I am really doing, for this exercise, is substituting Ennerdale for Sellafield? Is that it?
A. No, you are substituting Ennerdale for the area across the top there. Yes, if you like. Yes, I suppose, in that sense. Yes, I am sorry, my Lord. Yes.

Q. So if I put Ennerdale in brackets over Sellafield?
A. Yes, that would be reasonable.

Q. For each of the four year bands there is an excess over the controls and over the national average?

Q. Not for one of the years, 1966-70, there is not over national.

Q. In each year band there is an excess over controls; in each year band bar one, i.e. - which band?
A. A. 1966-70.

Q. There is an excess over nationally expected figures?
A. Yes, though this may not be statistically significant.

Q. MR. LANGSTAFF: By "statistically significant" are you dealing with significance as any particular P value?
A. Yes, for a 95 per cent confidence interval.

Q. Would you turn to page 95? Looking again at Ennerdale....

MR. JUSTICE FRENCH: Which slab of page 95?

MR. LANGSTAFF: My Lord, if one looks at the top:

Q. Again looking, Professor, if you would, at the incidence rather than the mortality?

MR. JUSTICE FRENCH: Again the top slab?

MR. LANGSTAFF: My Lord, yes.

Q. MR. JUSTICE FRENCH: We are notionally putting in Ennerdale, are we?
A. Yes.

Q. MR. LANGSTAFF: Does that show that for each of the five year periods there was a rate of incidence of leukaemia in Ennerdale in excess of the national figures?
A. Yes.

Q. I am sorry, Professor, you have not answered?
A. I am sorry, I said yes some time back.

Q. I beg your pardon. It must be my fault for not hearing you.

MR. JUSTICE FRENCH: I think the clock is beginning to win over fascination!

MR. LANGSTAFF: My Lord, yes.

MR. ROKISON: My Lord, I am content, insofar as anything is clear from a document, that if my learned friend wants to refer to it in due course, then he can do so without asking the witness, to save time. It seems to me that the issue here may be one which is actually investigated by those who carried out the study as to whether it is more appropriate to take registration on mortality, and why.

However, anybody can see what the figures tell you and their interpretation, it seems to me, is not a matter on which your Lordship, or any of us, requires assistance. I am content that if this exercise were

going to continue in this way, that it should be taken as read, my Lord.

MR. JUSTICE FRENCH: Yes, thank you, particularly as we are all getting tired and beginning to make a lot of mistakes that we otherwise might not.

MR. LANGSTAFF: My Lord, dare I ask if that would be a convenient moment?

MR. JUSTICE FRENCH: I think if you summon up your courage in both hands.

MR. LANGSTAFF: My Lord, may Prof. Evans be formally released until he comes back again? As I anticipated earlier today, I hope that will be some time this term.

MR. JUSTICE FRENCH: Yes. Well, certainly you may be released, Prof. Evans, and I look forward to seeing whenever it may be.

MR. ROKISON: My Lord, I assume that Prof. Evans will - although I have no objection to Prof. Evans talking to my learned friends in relation to his re-working of the Gardner study - I have assumed that so far as matters which I have covered in cross-examination is concerned, that he will be put in the usual purdah?

MR. LANGSTAFF: My Lord, any matter upon which I propose to re-examine Prof. Evans of course I will not discuss with him.

MR. JUSTICE FRENCH: Well, that, I trust, could have gone without saying.

MR. ROKISON: Yes, it is simply so Prof. Evans understands what the position is, my Lord, that is all.

THE WITNESS: Yes, I understand that. I am now allowed to talk about the re-analysis to...

MR. JUSTICE FRENCH: Yes, to counsel for the Plaintiffs. Very well.

(The Court was adjourned until 10.30 on Monday, 7th December, 1992)