COMMONWEALTH OF PENNSYLVANIA

PUBLIC UTILITY COMMISSION

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Letter of Notification of :
Philadelphia Electric Company : Docket No.
Relative to reconstructing and :
rebuilding of the existing 138 kV : A-110550F055
line to operate as a Woodbourne-
Heaton 230 kV line in Montgomery and
Bucks Counties.

Further hearing.

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Pages 1368 through 1532 Hearing Room No.
State Office Building
Broad and Spring Garden Streets
Philadelphia, Pennsylvania

Friday, February 7, 1992

Met, pursuant to notice, at 10:10 a.m.

BEFORE:

HERBERT SMOLEN, Administrative Law Judge

APPEARANCES:

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C O N T E N T S

E X H I B I T S

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P R O C E E D I N G S

ADMINISTRATIVE LAW JUDGE HERBERT SMOLEN: This is a further hearing in Docket A-110550F055.

Are there any preliminary matters which any Counsel desires to discuss prior to the taking of this additional testimony?

MR. BONNEY: No, Your Honor.

JUDGE SMOLEN: Then let's proceed.

MS. McCLOSKEY: Thank you, Your Honor.

The Office of Consumer Advocate calls David M. Rosenbaum, and he has been previously sworn.

JUDGE SMOLEN: You remain under oath, sir.

Whereupon,

DAVID M. ROSENBAUM

having previously been duly sworn, testified as follows:

MS. McCLOSKEY: Your Honor, I would like to ask that OCA Statement No. 2A be marked for identification. That is the surrebuttal testimony of David M. Rosenbaum.

JUDGE SMOLEN: That will be OCA 2A. So marked.

(Whereupon, the document was marked as OCA Statement No. 2A for identification.)

MS. McCLOSKEY: Thank you.

DIRECT EXAMINATION

BY MS. McCLOSKEY:

Q. Dr. Rosenbaum, do you have before you what has

just been marked as OCA Statement No. 2A?

A. Yes, I do.

Q. Does this consist of five pages of questions and answers?

A. Yes.

Q. Did you prepare this testimony?

A. Yes.

Q. And do you have any additions or corrections to this testimony?

A. No.

Q. Is this testimony true and correct to the best of your information, knowledge and belief?

A. Yes.

Q. And if I were to ask you these questions and answers today under oath would your answers be the same?

A. Yes.

MS. McCLOSKEY: Your Honor, what has been marked as OCA Statement 2A has previously been provided to all parties, Your Honor, and I have provided two copies to the court reporter. I would move for the admission of OCA Statement 2A subject to any timely motions and cross-examination.

JUDGE SMOLEN: Without objection, it is received.

(Whereupon, the document marked as OCA Statement No. 2A was received in evidence.)

MS. McCLOSKEY: Dr. Rosenbaum is available for cross-examination.

JUDGE SMOLEN: Fine. Let's go with Mr. Sugarman first.

MR. SUGARMAN: Thank you, Your Honor.

CROSS-EXAMINATION

BY MR. SUGARMAN:

Q. Mr. Rosenbaum, at page two of your testimony, lines 15 to 17, you essentially go back to a point that you made earlier in your main testimony. I just want to clarify is -- well, you use the word proof and put it in quotes. I think you explained in your previous testimony what you mean by the problem of proving an EM/F or anything else in epidemiology. But what I want to ask you at this time is is epidemiology commonly relied upon to make public and private decisions relating to safety in terms of health effects?

A. Sometimes. It is one of many things that are relied on.

Q. And the other question I had was on page four of your testimony, lines three to six, you state that, "neither the Savitz study nor the Peters study indicate there is a 95 percent probability that there is a true positive correlation." You put the word true in quotes. Can you explain why you put the word true in quotes?

A. Sure. Yes. The assumption in statistics is that there is a true value, a true relationship, and you are trying to find it. And what you are actually able to calculate is something that is an estimator of that. And there is always some chance that your conclusion from the estimator will be wrong and one -- two things, I guess, that you are trying to do is, one, you are trying to make a conclusion and then trying to estimate what the chances are that your conclusion is wrong.

The reason I put true in quotes is just to indicate that what is being calculated is what is called an estimator of what you are looking for and not the actual value, which is somewhat different than -- which is different, than, for example, if you were going to do a physics experiment in which you might actually be calculating the quantity itself. Even though there would be an error in that estimate, it still would be different than the actual calculated quantity.

Q. So does the word true have the same problem for you that the word proof does? Is the problem comparable? In other words, that we are talking associations?

A. It is a different situation. I don't know how to say whether it is comparable or not. The only reason I put true in quotes was to indicate that these calculations are calculations of the estimator and the

intent of making, for example, a range, a competence interval and assigning a probability to it is in some sense to estimate what the chance that the true value is in that confidence level.

Q. Towards the end of page four you state on lines 16 to 19, "I do not believe that the results of the Wertheimer & Leeper, Savitz and Peters studies indicate that if better and better studies are done the reported association between wire code classification and childhood leukemia will eventually go away." Do you have a belief as to whether, quote, better and better studies, end of quote, will maintain the association?

A. I don't know. I was only commenting there on the argument which was made by Dr. Cole that these three studies indicate that there is a logical conclusion from these three studies that if better and better studies are done the reported association will go away. I don't think that these three studies do make a logical argument that the reported association will go away. I can't predict the future and I don't know whether it will or not, but I don't think there is an argument from these three studies that it will go away. And that was my comment there.

Q. Is there any body of information or opinion that you are aware of that would attempt to draw an

inference as to the probability that three studies such as these and the others that you mentioned previously which suggest an association could all end up going away, so to speak, in later studies or later proofs, other forms of demonstration? Is there any body of knowledge that would enable you to place a probability estimate on that happening?

A. There are people who do what is called meta analyses but I don't think they are rigorous studies. They are an attempt to make use of a large body of information which is not exactly comparable and to be reasonable about it. But they involve a lot of assumptions. They are a perfectly reasonable thing to do and an useful thing to do and they are part of the general discussion, but I guess what I'm saying is I don't think they lead to a rigorous conclusion.

The only way you get rigorous conclusions from epidemiology in such a situation is to have --I mean, you don't get rigorous conclusions. If there is sufficient weight of evidence over a long period of time in some direction, then there becomes a consensus eventually that that is right. And it is obviously strengthened by biological information, and the perfect example is smoking and lung cancer, in which evidence accumulated for a very long time. There were physicians in the '30s

who were already worried about the possibility that cigarette smoking was causing lung cancer. But it took a very long time -- I know some -- but it took a very long time before there was any consensus on it.

Q. And you think that meta analysis as to this problem is not yet ripe?

A. No. I think it is an useful tool. It is one of the useful tools in the whole discussion.

Q. Right.

A. I mean, I think it is an useful thing to do.

Q. And it is a recognized specialty or field?

A. I don't know if it is a field. I don't know whether there are people who just do meta analyses. It is something that statisticians do. I have read some in this field. And it an useful thing to do.

Q. Finally, on page five of your testimony you comment on the concept of prudent avoidance, where the decision, the construction decision precedes the studies as to the cost and effects of possible mitigative measures. And you then refer to the fact that calculations were made here after the line was virtually finished and you say, "It is like driving on a long trip with bad brakes and no seat belts and calling it prudent because you arrived safely." Do you mean by that sentence to associate yourself with any conclusion that

this line would be safe?

A. If in the first place, as someone who has been involved with these decisions for a long time I honestly don't know what you mean by safe.

Q. Well, I am only picking up on it because you used the word. And I just want to make sure you didn't mean to assert that as to this line by reason of using that in your analogy.

A. No, I didn't mean. I have no -- I am not sure what the word safe in that context would be. In this context it is just if you arrive safely then you're safe. I mean, if you don't get in an accident, you don't get in an accident. In the other case it is not exactly clear to me what safe means. So I guess in that sense I don't really understand your question.

All I meant to say here is even if you have a situation in which you do something -- it is possible to have a situation in which you act imprudently and in fact no harm gets done. But it is not very good policy is to do that because if you keep doing it that is not always going to be the result. That was the only thing I was trying to say. It was not a conclusion about this line. It had to do with public policy.

MR. SUGARMAN: That's what I thought. I just wanted to be sure. Thank you very much.

Thank you, Your Honor.

JUDGE SMOLEN: Mr. Dillon.

MR. DILLON: No questions, Your Honor.

JUDGE SMOLEN: Ms. Burket.

MS. BURKET: No questions, Your Honor.

JUDGE SMOLEN: PECo. Mr. Watson.

MR. WATSON: Thank you, Your Honor.

CROSS-EXAMINATION

BY MR. WATSON:

Q. Hi, Dr. Rosenbaum. It's good to see you again.

A. It's good to see you.

Q. I may be asking you some questions about the Savitz study. Do you have a copy of it with you?

A. Yes. Are you talking the 1988 Savitz study?

Q. Well, yes.

A. Savitz, Rachtel, Barnes, John and Tvrdik?

Q. Wachtel.

A. Wachtel. I'm sorry.

Q. Right. A portion, one part of it, is published here in the American Journal of Epidemiology.

A. Yes.

Q. Now, could you turn to the paragraph of your testimony beginning on page two, line 27?

A. You are talking about my surrebuttal?

Q. Yes, sir.

A. Yes.

Q. When you are talking about wiring code in these studies we are talking about, first, reference to the wires outside of a house?

A. Yes.

Q. Is that correct?

A. Yes.

Q. And would it be correct to say that the researchers would go to the house and look at the wires outside the house and based on a visual inspection assign the wires into a wire code category?

A. In the Peters study actually the actual assignment was done by computer. But it was certainly based on observation of people.

Q. Visual inspection of what was there?

A. Yes.

Q. And in the Savitz and Peters studies a house could be assigned to any one of five categories, is that correct?

A. I think that is right. I could check it but I am willing to believe that. That is how I remember.

Q. Maybe this will refresh your recollection and get it out for everybody as well. Would the names of the five categories be, buried or underground as the first category, which I believe Savitz used one and Peters used

the other?

A. In some studies they were combined.

Q. Buried or underground was one category, very low current configuration, low current configuration, high current configuration and very high current configuration. Would they be the five categories?

A. I remember the five categories. I just want to check to make sure that -- in some things they were combined in different ways.

Q. Maybe it would help, in some instances, for example, when they may have been doing a calculation they may have combined one or two categories.

A. Yes. That's what I meant.

Q. Okay. And I guess I am asking the question before that, in the sense is that the five categories they started with?

A. Yes.

Q. And the Savitz and Peters studies, each report the number of houses of cancer patients in each category and the number of what we call control houses or houses with no cancer patients in each category?

A. I think so.

Q. And the odds ratios you refer to in your testimony are statistical comparisons of those numbers?

A. Yes. Well, they involve other things, too,

like the number of people who -- yes. I mean, they involved how many people got ill as well in the categories.

Q. So the studies report the number of houses in each category, the number of cancer patients in those houses in each category, the number of non-cancer patients in each category and they do various comparisons?

A. Yes.

Q. And the statistical comparisons they do would be referred to as the odds ratios in these studies?

A. Yes.

Q. I would like to refer you again to the paragraph of your testimony beginning on page two, line 27.

A. Yes.

Q. And I believe it says there in substance, and this is not all of it but I would like to refer you to this portion of that, "Third, and perhaps most important, I believe it is not entirely fair to say that, quote, he -- referring to Peters -- "found an association between the wiring code and childhood cancer that is weaker still than the association seen by Savitz."

A. Yes, but there is a discussion which goes with that.

Q. I was going to keep reading.

"The Peters study had a higher odds ratio, both adjusted (1.73) and unadjusted (2.15), than the Savitz study (1.54)."

A. Okay.

Q. Just referring to you that portion of your testimony.

A. Okay.

Q. Do you understand Dr. Cole's previous testimony to be that as the quality of the studies improves a pattern of weaker odds ratios provides support for the lack of an association between wiring code and cancer?

A. Well, that in general was what I was commenting on. I could look up his statement, but as I recall Dr. Cole's remark was somewhat stronger than he provides support for. I don't have it in front of me, but I could look it up.

Q. For example, maybe it would help, do you have his testimony?

A. Yes, I do.

Q. Page 18, lines 18 through 22, it begins with "with regard to".

(Witness perusing document.)

A. Yes. I have it. Yes.

Q. And I believe there he says that --

A. Suggests that there is no true association between EM/F and cancer -- again suggests that there is no true association between EM/F and cancer underlying the statistical reports.

JUDGE SMOLEN: I can't hear you. I'm sorry.

THE WITNESS: Again suggests that there is no true association between EM/F and cancer underlying these statistical reports.

JUDGE SMOLEN: Go ahead.

BY MR. WATSON:

Q. Referring there to Savitz and Peters?

A. Yes.

Q. Now, would you agree that the Savitz and Peters studies are an improvement over earlier studies?

A. Yes.

Q. So your disagreement with Dr. Cole is that when you compared the odds ratios in the Savitz and Peters studies you didn't think that the odds ratios went down?

A. No. That is not a fair statement. That is not my disagreement.

Q. Let me take you back just to make sure we understand this to page two, line 27.

A. Yes.

Q. Let me refer you particularly to this phrase so I can ask you about it: "Third, and perhaps most

important, I believe it is not entirely fair to say that he (Peters) found an association between the wiring code and childhood cancer that is weaker still than the association seen by Savitz. The Peters study had a higher odds ratio, both adjusted (1.73) and unadjusted (2.15), than the Savitz study (1.54)."

A. Yes.

Q. Do you still stand by that statement?

A. Only if the rest of the page is included as explanatory material, which it clearly was meant to be. Would you like me to expound on -- why don't I finish my answer.

Q. Are you referring to then there is a parenthetical that follows that that says, "The odds ratio is a measure of the relative risk of exposed people compared to unexposed people."

A. I am not referring to the parenthetical.

Q. Are you referring to the following sentence? "For example, an odds ratio of 2.0 means that the risk of the exposed is twice that of the unexposed."

A. No. I am not referring to anything in that parenthetical at all.

Q. So you are not referring to anything in the remainder of that paragraph?

A. No, when I was -- not in what I was just saying

a minute ago, no, I'm not. I was referring to the things in the next paragraph. I think it is fair to consider the statement, the entire testimony, as an unit which explains the thing fully rather than take a single sentence and isolate it.

Q. So you are saying that we have to read the sentence that I read before we get to the parenthetical, we have to read that in the context of the next paragraph of your testimony?

A. In the context of the entire statement that I made.

Q. And the entire statement?

A. Absolutely.

Q. Okay. I understand.

Now, taking into account all of your testimony, include including that next paragraph, taking that into account, do you still stand by your statement about the odds ratios, the prior statement?

A. The odds ratios are just numbers and the odds ratios are certainly right. The interpretation of the odds ratios needs to be done, and I tried to do that in the following.

Q. I understand.

Dr. Rosenbaum, didn't you make a mistake in the odds ratio you picked from the Savitz study to compare to

the odds ratios in the Peters study, the 1.54?

A. I made the point in the next paragraph that Peters' and Savitz's figures compared different things, if that is what you mean. That is the first sentence in the next paragraph.

Q. When you conducted your comparison of the two studies, you picked two odds ratios from the Peters study, 2.15 and 1.73, correct? Page three, line two.

(Witness perusing document.)

A. Yes. I mean, I have to go back and look -- yes, I did. Yes.

Q. Do you want to go back and -- do you need a moment to go back and look at something?

A. That would be helpful because I didn't memorize all the numbers.

MR. WATSON: Why don't we give the witness a moment to check that.

JUDGE SMOLEN: Yes, certainly.

(Witness perusing document.)

BY MR. WATSON:

Q. I am not suggesting, by the way, that 2.15 and 1.73 are transcription errors or anything. I am just referring you to those at this point.

A. It's always possible that they are, but I hope not.

(Witness perusing document.)

A. It would save time. Do you remember which -- I can find it, but which table of the Savitz study the 1.54 came from? That might speed things up.

Q. Table 7, page 34.

(Witness perusing document.)

A. Yes. Okay.

Q. Have you found it, Table 9?

A. Yes, I have found Table 9 and I have the tables from the complete study.

Q. And you transcribed those correctly, 1.73 and 2.15?

A. Yes.

Q. Okay. So just to make sure we are on track here, when you conducted your comparison of the two studies you picked two odds ratios from the Peters study, 2.15 and 1.73?

A. Yes.

Q. And the odds ratios that you picked from the Peters study basically compared the houses that the researchers thought had the highest exposure to EM/F?

A. Right.

Q. The very high current configuration category, with the houses that the researchers thought had the lowest exposure to EM/F, buried and VLCC, very low

current configuration, correct?

A. Yes, which meant underground and very low, yes. That's right.

Q. And didn't Savitz report an odds ratio that compared the highest exposure category, VACC, with the lowest exposure category, buried?

A. Yes, that's right. But what it says -- yes, he did. It says there are -- there are two things on Table 7. One is two level wire codes at diagnosis and the other is very high versus buried wire code, which didn't include very low. It included only buried.

Q. Just buried?

A. Which is the point I was trying to make, that they used different categories.

Q. Right.

Now, I guess one of the reasons for making this kind of comparison of extreme values, that is, the highest with the lowest, is that you might be able to better see an effect of exposure if you compare the highest and lowest?

A. If that was -- if that is what a dose meant, yes.

Q. And isn't that commonly what you do in these studies, is compare highest categories versus lowest categories, in other words the extremes, so that you

might be better able to see an effect of exposure?

A. That is certainly one common thing to do, yes.

Q. Now, the odds ratio that Savitz reported when he compared the highest and lowest exposure categories was 2.75, correct?

A. Yes.

Q. That would be page 34, Table 7?

A. That's right.

Q. But if we look at page three of your testimony where you discuss comparison of the two studies, that is not the number you picked from the Savitz study, is it?

A. That's right. The number was 1.54, which is a different grouping, which is -- neither of these groupings are the same as...

Q. Now, the number you did pick from the Savitz study was calculated from data from all five of the wiring code categories, comparing three categories taken as a group against the other two categories taken as a group, correct?

A. Yes. The bottom group in those categories included very low and buried, which was the lowest category in Peters, and also low --

Q. And the Peters -- excuse me.

A. -- against very high and high, whereas Peters only used very high. As I pointed out in the next

paragraph, they were divided differently. I mean, there is not an exact comparison possible because they group them differently.

Q. Now, the Peters study does not report an odds ratio based on dividing all five categories into two groups and comparing those two groups like the number you gave for Savitz, does it?

A. No. But I believe one could in fact from the Peters data calculate such as a thing. One can calculate the odds ratios but it would be a lot of work by hand. But one can calculate the odds ratios that are comparable in the two studies, actually. I didn't do that, but you could do it.

Q. We've got Savitz, Peters crude and Peters adjusted, basically, don't we? That is what we are dealing with here?

A. Right.

Q. Now, the odds ratios that you picked from the Peters study were 2.15 crude, correct?

A. Yes.

Q. Let me make sure I get this right. From your testimony?

A. Yes.

Q. 2.15?

A. Right.

Q. And then 1.73 for adjusted?

A. Right.

Q. So that would go here.

Now, these were very high compared to underground and very low, is that correct?

A. That is what the Peters study was, right.

Q. That is what these Peters numbers that you chose came from?

A. Right.

Q. Okay.

Now, do you remember a few minutes ago when we identified the 2.75 odds ratio from Savitz from page 34?

A. Yes.

Q. Now, that number was a comparison of very high?

A. Right.

Q. To --

A. Buried.

Q. Buried. And that number was 2.75?

A. That's right.

Q. It was very high to underground, actually. Wasn't that the phrase they used?

A. The Savitz called it very high to buried. But I'm willing to believe that buried is underground.

Q. Very high to buried. So from looking at the extremes we might put that 2.75 here, but we probably

ought to put a footnote, shouldn't we, just to be accurate so we have this little difference that says very high compared to buried. Would that be an accurate way for relating the data, as long as we put the footnotes? Because what you are noting, really, is that they grouped a couple of these for these two, they group the two bottom ones, don't they?

A. Yes.

Q. And in epidemiology the grouping from the bottom is called a referent group, isn't it?

A. Yes.

Q. And so we are just making a footnote so that we note that the referent group is slightly different because it doesn't have the other categories.

A. I don't know how big it is. I don't know that it is slightly different. It's different.

Q. Well, slightly different in the sense not talking about numbers but different in the sense that are these two bottom categories here and we are noting here there is only one bottom category that you used.

A. Right. They are different.

Q. Yes. Slightly is probably meaningless in that context.

JUDGE SMOLEN: Let me interrupt for a moment. For the record, you have referred to Peters crude and by that

I assume you mean Peters unadjusted as referred to in the witness' testimony. Is that correct?

MR. WATSON: Yes, Your Honor.

JUDGE SMOLEN: All right.

BY MR. WATSON:

Q. Now, are you expressing the opinion that we can't really compare the 2.75 number from Savitz with the numbers from Peters because the referent group in Savitz contains only the bottom wire code category and the reference group in Peters contains the bottom two wire code categories?

A. You can't compare them directly. They have to be somehow adjusted. And I made the point in my testimony that in fact they used different categories. I mean, they grouped things differently.

I think it would be reasonable -- I mean, if your point is, I mean, because they did group thing differently, I think both things are reasonable to discuss, actually, the two level wire code and the very high versus the very low. I mean, they are both relevant and they are different categories than Peters used.

Q. Now, in your testimony you give us a formula and say it is relatively easy math to calculate odds ratios, I believe.

A. Well, it is straightforward.

Q. Straightforward.

A. I mean, you can calculate from Peters and Savitz comparable odds ratios for comparable categories. It would take a long time to do it here with a lot of algebra, I mean, by hand. And I didn't actually do it but you could do that. But you wouldn't then have, anyway, the confidence interval. But you could actually calculate the comparable odds ratios.

Q. Could you look at page 34, Table 7 of the Savitz study that we have been referring to?

A. Yes.

Q. Now, you could calculate a lot of different odds ratios from the data in Table 7, couldn't you?

A. Yes. He did calculate a lot of them. There are a whole lot of them listed there.

Q. But at least here they don't give you the raw data for the very low category in Table 7, do they?

A. No.

Q. They just have low and high?

A. No.

Q. Right. They don't have very low?

A. No.

Q. They just don't give you the numbers there.

And so to calculate the odds ratio from Savitz that is precisely identical to the Peters odds ratio that we

have been discussing you need the raw data for the very low category, as you have indicated?

A. Yes. In fact it is given, I believe, in Table 6.

Q. But it's not for leukemia alone in terms of the comparison you made there, is it?

A. No.

Q. They just don't have it all in this?

A. As I recall, I thought about doing it and either in Peters or in Savitz I believe there is enough data to calculate the odds ratios the way the other one did it. And in fact I set out to do it but -- I didn't actually complete the calculation, but I could do it.

Q. So you could do it and you could do a head-to-head comparison here?

A. Only with odds ratios. You still wouldn't have the confidence interval.

Q. But you could do a head-to-head comparison of the odds ratios. So we could compare head-to-head the 2.75 and the 1.73, or the 1.73 on the one hand with the same numbers from Savitz on the other?

A. Yes.

Q. If you wanted to?

A. You could do such a thing, yes.

Q. Now, you are aware, aren't you, that the Savitz

study was conducted as part of the New York powerlines project?

A. Yes.

Q. And so I assume you are aware as part of that project Dr. Savitz prepared a report that contained the data from his study?

A. I assume he did. I have not seen anything more than the published paper. We may have it in the office but I have not read anything more than the published paper.

(Pause.)

A. I mean, if it's any help I might quote a sentence that's already in the record. "If the effect increases monotonically this would make Savitz' results weaker than Peters' results."

Q. Let me ask you to take a look at this and verify that we are talking about the same thing.

A. I won't have time to study it.

Q. I'm not asking you to study it at the moment.

A. I see that it is a report.

Q. All I'm really asking you to do is asking you to look through and see that we are all talking about the same Savitz study here. Because Savitz has, you know, other studies and we want to keep it straight when we make the reference to, quote, the Savitz study.

A. All right. The paper in the front of this is the same paper that we are talking about.

Q. Just look at the following stuff if you want to.

(Witness perusing document.)

Q. Now, if you could go past the copy of the published study.

A. Yes, I'm past it.

Q. To where the typewritten text begins in the supplement. Do you see that beginning on page four there is further discussion of the Savitz data?

A. Yes. There is an abstract on page four.

Q. Could you flip to page 19 of the typewritten text there?

A. All right.

Q. Do you see the data presented in tabular form beginning on that page?

A. Yes. I see there are data on that page, yes.

Q. Now, could you continue on to page 38 there?

A. Yes.

Q. Do you see at the at the top of the page it says Table 15, wire codes (five levels) cases of child control?

A. Yes, that is what it says.

Q. Do you see below that, still on page 38, in

Tables 15(b) and 15(c) data are provided for cancer and wire codes two years before diagnosis?

A. Yes.

Q. If you turn to page 39 do you see that data are provided for cancer and wire codes at diagnosis?

A. Yes.

Q. Now, if you could keep your place in the supplement where you are now, and turn to page 34 of the Savitz publication portion of this.

A. All right. That is Table 7?

Q. Yes.

A. All right.

Q. And Table 7 is labeled cancer risk in relation to wire configuration code for K subgroups at time of diagnosis, correct?

A. Yes.

Q. Now, just for purposes of confirming the data between those two pieces of material that are all actually bound together as one, would you take a look at the following: in Table 7, what is the total number of control houses for all five wiring code categories?

MS. McCLOSKEY: Your Honor, I would object at this point. I don't quite understand what the relevance is of comparing a document that is not in the record and the company has not indicated they are going to put it in the

record to data in a published study that we already have information from that study in the record. I would ask for an offer of proof at this point as to where we are going with this data comparison of raw data in a study that won't be made part of the record.

MR. WATSON: Your Honor, the witness made a comparison, as we have shown here, and has indicated that the comparison that he has actually made may not match up, and then we have another comparison that he said may not match up. But he says, as he testified, you could make a head-to-head comparison and he has relied upon the Savitz study for that purpose. And he said if you had the very low numbers we could make the head-to-head comparison. I'm simply going to ask him if we could find those numbers that would allow us to do this.

MS. McCLOSKEY: I think that, then, it is asked and answered. He has already said it could be done. He has not done it for purposes of his testimony and his testimony is not based on a head-to-head comparison. So I would say it is asked and answered.

MR. WATSON: I don't think that has been asked and answered. This is cross-examination --

JUDGE SMOLEN: Overruled. You can go ahead.

MR. WATSON: Thank you.

BY MR. WATSON:

Q. In Table 7 what is the total number of control houses for all five wiring code categories? Would it be 207 plus 52?

A. Yes.

Q. Which is a total of 259?

A. Right.

Q. On Table 15(d) of the supplement, look at the top of the page?

A. What page are we on in the supplement?

Q. Thirty-nine.

A. All right.

Q. And the total number of controls for all five wiring code categories there is given as 259?

A. Yes.

Q. Now, in Table 7 -- that is, in the AJE published version of Savitz -- in Table 7 what is the total number of leukemia cases for all five wiring code categories combined? Would it be 70 plus 27, or 97?

A. Yes.

Q. And in Table 15(e) the total number of leukemia cases for all five wiring code categories combined would be 70 plus 27, or it is just listed here as 97?

A. Yes.

Q. Are you willing to accept that Table 7 in the American Journal of Epidemiology article and Tables 15(d)

and (e) in the supplement both report the same set of data from the Savitz study?

A. Yes, but --

Q. One just has more? More data?

A. I'm willing to accept it. But in all fairness there may be lots of explanation of this data in the report, which I have not read. There is a long text here which I have never seen before. I don't know what caveats or explanations or qualifications there might be of this data in the text because I haven't read the text.

Q. But it's the same data? That's really all I'm getting at.

A. It seems to be. Those numbers are the same, anyway.

Q. I believe you said in your testimony calculating odds ratios is relatively easy? Page three, line 30.

A. Yes.

Q. So, odds ratio equals...

(Mr. Watson writing formula.)

Q. Would you check that and make sure that I have correctly recorded the formula you set out for us in the testimony for calculating the odds ratio?

A. Yes, you have.

Q. Now, am I correct that basically what we are

going to be doing is multiplying and dividing four numbers if you did this calculation?

A. Yes.

JUDGE SMOLEN: To the right of the slash is the denominator?

MR. WATSON: Yes, that is a good point. The slash mark means that what is after it is the denominator.

BY MR. WATSON:

Q. Is that correct?

A. Yes. And the stars are meant to indicate multiplication.

Q. Yes, the little star-like symbols mean to multiply?

A. Yes.

Q. And in this situation N(E,D) refers to the number of leukemia cases in the very high category from the Savitz study data, is that correct?

A. It's a general thing. If you are taking the very -- it is the number of exposed depending on how you want to define it, who actually came down with the disease of interest, which in this case is leukemia.

Q. I'm being a little more specific, but that is what it refers to, at least as to this study?

A. Okay.

Q. Can you turn to page 39 of the Savitz

supplement, Table 15(e)?

Now, would it be accurate to say there are seven leukemia cases in the very high category?

A. Yes.

MS. McCLOSKEY: Your Honor, I would object again at this point. I believe that Mr. Rosenbaum has said repeatedly through his testimony that even though the odds ratio can be calculated, the confidence interval cannot and that that was an important part of it. So this exercise is essentially not relevant to his testimony and to the presentation in his surrebuttal.

MR. WATSON: Your Honor, I would argue that it couldn't be more relevant.

JUDGE SMOLEN: The formula itself is presented in his testimony. He is testing it. So I think it is proper. If you have anything on redirect that you may want to bring out that other factors have to be considered, you can do so. But let's go ahead.

BY MR. WATSON:

Q. Looking at Table 15(d) would you agree there are eight controls in the very high category?

A. Yes.

Q. And that would correspond in this case to N(E,H)?

A. Yes.

Q. Now, on Table 15(e) would you agree that there are 28 leukemia cases in the buried category and five in the very low category?

A. I'm sorry. Are we on (d) or (e)?

Q. 15(e).

A. All this has to do with (e)? Because the number in the very high category --

Q. Controls are in (d), cases are in (e). Here I'm referring to cases in 15(e).

A. Okay. Yes.

Q. Okay.

A. Yes.

Q. And there are 28 leukemia cases in the buried category?

A. In the buried category.

Q. And five in the very low category?

A. Right.

Q. And by adding those two numbers we get the value --

A. Thirty-three.

Q. And that corresponds to N(U,D)?

A. Yes.

Q. Now, let's go back to Table 15(d). Would you agree that there are 88 controls in the buried category and 17 controls in the very low category?

A. Yes.

Q. Adding those two numbers we get the value of --

A. One hundred and five.

Q. -- N(U,H)?

A. Yes.

Q. Now, then, would I be correct that the equation we must solve is seven times 105, divided by 33 times eight?

A. Yes.

Q. Seven times 105 would be 735?

A. That's right.

Q. And 33 times eight is 264?

A. That's right.

Q. And 735 divided by 264 equals 2.78?

A. Yes.

Q. So if we are just going to do a flat head-to-head comparison of the categories with whatever explanation one might offer, just a head-to-head comparison, the number, instead of 2.75, would be 2.78 based on Savitz's data, correct?

A. That is the calculation we just did. I think it's -- I don't know what is in the text so I don't know -- it would have been a lot easier if I had a day to look at this or something. That is what he you get out of these numbers. I don't know what qualifications there

might be of the data in the text because I haven't seen it.

Q. Understood.

But we are just comparing the two numbers you used from Peters and we are comparing the comparable number, including the same categories, from Savitz?

A. It's the same categories.

Q. At least that much?

A. But I don't know what qualifications there are in this document that there might be in that 2.78, because I haven't read it.

Q. Understood.

Let's see if we can find another way, then.

(Pause.)

Q. Earlier we were talking about the Savitz number of 2.75. Right here, right?

A. Right.

Q. What that turned out to be, didn't it, was very high compared to buried?

A. Yes.

Q. Correct?

A. That's right.

Q. So that is 2.75. And this time we don't need a footnote, do we, since we have the category that describes what that was?

A. The footnote only has to do with I don't know what explanatory material goes with it.

Q. Right. We put this footnote in to show that this number when we stuck it in here originally didn't have quite the same categories as the column heading.

A. Right.

Q. But over here we have changed the column heading so we don't need the footnote anymore. But it says the same thing as the column heading, doesn't it?

A. Right.

Q. Now, could you turn to the Peters study, page 933, Table 7?

A. Okay.

Q. Do you agree that in the very high category there were 42 cases?

A. Can I just look at this for a minute?

Q. Sure.

(Witness perusing document.)

A. Yes. Okay.

Q. So if we were following your formula, N(E,D) will be 42?

A. Yes.

Q. And still on Peters, Table 7, do you agree that there are 24 controls in the very high category?

A. Yes.

Q. And so that corresponds to N(E,H), correct?

A. Right.

Q. And on Table 7, would you agree that there are 11 cases in the underground category?

A. Yes.

Q. And that corresponds to N(U,D)?

A. Yes.

Q. And on Table 7 do you agree that there are 11 controls in the underground category?

A. Yes.

Q. So the equation we have to solve is 42 times 11, divided by 11 times 24?

A. That's right.

Q. And 42 times 11 equals 462. Do you want to check me on that?

A. You can just cross out the 11s. It's just 42 divided by 24.

Q. Well, I was going to just march through it here just for completeness.

And 11 time 24 equals 264, correct?

A. The result is 42 over 24.

Q. Or 462 over 264. And if you divide those it equals 1.75?

A. That's right.

Q. Now, let's look back over here. So very high

compared to underground, 1.75. Have I placed that in the correct spot?

A. It seems to be, yes.

Q. Am I correct that the Peters study does not provide enough information to calculate the adjusted odds ratio for this particular one?

A. Yes.

Q. NA for not available.

Now, could you please turn, Dr. Rosenbaum, to page two, lines 11 through 17 of your testimony?

A. Okay.

Q. Let me ask you one other thing about this. Don't the numbers in this first column comparing these get weaker as you move from Savitz to Peters?

A. As a matter of fact, that is what I said in my testimony.

Q. Okay.

A. What I said was if the effect increases monotonically it would make Savitz's results weaker than Peters'. That's what I said. If that is the point you were trying to get at, I already said it.

Q. Excuse me. Maybe -- didn't you say that the Peters study had a higher odds ratio both adjusted, 1.73, and unadjusted 2.15, than the Savitz study, 1.54?

MR. SUGARMAN: I object. I don't see that.

MR. WATSON: Page three, line two.

MR. SUGARMAN: Oh, back to page three.

A. Yes. Those were numbers quoted. But as we went through before, they have to be taken in the context of the entire -- the numbers were not meant to be taken all by themselves. They were meant to be taken in the context of the entire piece of testimony.

BY MR. WATSON:

Q. Okay. So when we compare the odds ratios head-to-head in this category, very high compared to underground or very low, and we use the extremes of Savitz don't the numbers decrease has they go from Savitz down to Peters?

A. Yes.

Q. And when we do a head-to-head comparison with the exact categories of the two studies, don't the numbers decrease from Savitz down to Peters?

A. Yes.

Q. Now, referring to page two, lines 11 through 17 of your testimony you are basically saying, aren't you -- one of the things you have said is that the obvious thing to say is that all three studies observed an association between wiring configuration and childhood leukemia?

A. Yes.

Q. Would you agree that the question of which

result is obvious is a matter of professional judgement?

A. I don't even think -- it is just a matter of judgement, yes.

Q. Professional judgement under the circumstances?

A. Right.

Q. For instance, one might consider it obvious to say that in the Savitz study the observed association between wiring code and leukemia was not statistically significant?

A. That depends on what you mean by statistically significant.

Q. Well, what if I meant the same thing that you meant in your testimony when you used the phrase statistically significant?

A. What I did was I quote what they use. It is true, which is just what I said, that it's not significant at the 95 percent confidence interval.

Q. And that is the normal confidence interval that we use when we refer to the phrase statistically significant?

A. I actually discussed that at some -- I don't know who "we" is that uses it, but in fact the idea that the 95 percent level is the one that ought to be used is not a statistical judgement at all, nor is it an epidemiological judgement and my understanding of it --

and I think this is right -- is that it was actually determined by a political scientist or something like that who did a poll of people to see what they would consider, the average person would consider significant, what sort of risk they would consider a significant difference.

And studies are often done, for example, at a 90 percent level. Since it is a continuum, it would be ridiculous to say that something at the 94 percent level was not significant and something at the 95 percent level was. It's a very small difference.

Q. But would it be fair to say that when we refer to statistical significance at least among scientists it is commonly accepted that we are referring to the 95 percent confidence level unless otherwise pointed out?

A. No. I think it is fair to say that among scientists the level is always specified.

Q. And referring to the Savitz study, what level did Savitz use?

A. We used the 95 percent level. But he could clearly have done with the same data a calculation at whatever level he chose.

Q. And when we look at the level that he chose to report the results of his study, would it be accurate to say that the observed association between wiring code and

leukemia was not statistically significant?

A. It's not significant at the 95 percent level and that is the level he reported it at.

Q. Would it be accurate to say that once Peters got beyond the crude stage and adjusted his data the observed association between wiring code and leukemia was not statistically significant?

A. Yes. At the same level.

Since you brought that up, I would like to say something about the adjustment process. The adjustment process --

MR. WATSON: I don't have a question.

JUDGE SMOLEN: Well, I'm going to let the witness explain his answer.

MR. WATSON: If he wants to go ahead, that's all right.

A. You can only adjust for things that you have reason to suspect. And there may be other -- for example, one of the things that he adjusted for was incense use in the house. Someone else might not have thought of adjusting for that. Similarly, there may be other things that should be adjusted for which he didn't see.

BY MR. WATSON:

Q. When we talk about adjusted, those are Peters'

own adjustments of his data as distinquished from yours and somebody else's?

A. Yes, they are. And they are based on a study he had done of a large number of these cases for a different purposes before he did this.

Q. Can you take a look at your testimony, page four, line 25, to page five, line three?

A. Yes.

Q. I think it begins, "In contrast, 13 of the 14 odds ratios".

A. Let me read the whole paragraph.

(Witness perusing document.)

A. Yes. Okay.

Q. Now, there you say that 13 of the 14 odds ratios for wiring code in Table 7 are above 1.0, correct?

A. Yes, that's right.

Q. Wouldn't it also be accurate to say that only two of those associations were statistically significant at the confidence level used by the researcher?

A. Yes. But I would like to -- can I explain the answer?

JUDGE SMOLEN: Yes.

A. There is nothing holy about any particular level of statistical significance that is chosen. If something turns out to be significant at the 95 percent

level that in and of itself does not prove the association, nor does it prove that -- nor is there a dramatic effect if it is slightly below 95 percent, a dramatic difference. So I think that not very much import should be given to exactly whether it is above or below 95 percent.

I mean, the actual confidence level that it is at is important in judging the worth of the result, but it is important to take, as I think the company witnesses have said already in this case, it is important to look at all the data and try to get a sense of what it all means.

Q. And just to be sure with Savitz, none of the reported associations between wire code and leukemia are statistically significant, is that correct?

(Witness perusing document.)

A. Not at the 95 percent confidence level. I didn't do the calculation but they might very well be at the 90 percent level, for example.

Q. Using the calculations that the researchers themselves selected. Asking it just on that basis. I'm not asking you to look at other numbers. Just the ones that the researchers themselves selected.

A. They reported -- the Savitz numbers are not significant at the 95 percent level, which is what he

reported. But one could do the calculation at any level with the same data.

Q. Would it also be accurate to say that when Savitz and Peters actually measured magnetic fields they did not report any statistically significant associations between the measured fields and cancer, including leukemia?

A. Yes. Again, if we are using significant in the same way.

MR. WATSON: Thank you.

JUDGE SMOLEN: Do you want some time?

MR. WATSON: Your Honor, one moment. I think for the record we ought to mark these as cross-examination exhibits.

JUDGE SMOLEN: Do you have them in reduced size?

MR. WATSON: I will produce a reduced size version for the record and for Counsel for all parties.

MS. McCLOSKEY: I would object to their admission into the record, Your Honor. They are numbers that the witness has not accepted. He has clearly explained that there may be qualifications to those numbers and to that data which he is not familiar with because he has not had an opportunity to review the entire study which he was handed.

JUDGE SMOLEN: Well, that might go to the weight.

But he actually used some of these numbers in his own testimony. 2.75, 2.15, 1.73 was used.

MS. McCLOSKEY: The 2.15 and 1.73, only two of the five numbers.

JUDGE SMOLEN: Yes, two of them were used. That's correct.

MR. WATSON: Here is 2.75 used here again. And, Your Honor, he's the one that made the comparisons.

JUDGE SMOLEN: Let her finish.

MS. McCLOSKEY: Your Honor, Dr. Rosenbaum did not use 2.75. That is a number that you had him calculate from a table which he was not able to review the entire study and determine whether there were qualifications on that number. The number he used in his study came from the reported Savitz data and it is not the 2.75 number.

MR. WATSON: Your Honor, I beg to differ. I think he used it on page four, line 14.

JUDGE SMOLEN: 2.75 is used there in the witness' testimony. So I'm going to permit it to be marked.

What number are we up to?

MR. WATSON: This would be PECo Cross-Examination Exhibit 5(a).

JUDGE SMOLEN: So marked.

MR. WATSON: And this where we show the calculations would be PECo Cross Exhibit 5(b).

JUDGE SMOLEN: So marked.

MR. WATSON: Your Honor, I propose that what we will do is type this up and send it to everybody rather than take pictures. I think in the record all the numbers are clear and if somebody had a problem they could tell us.

Is that acceptable to everybody?

MS. McCLOSKEY: That is fine.

JUDGE SMOLEN: It's acceptable to me. You may do it that way.

MR. WATSON: Thank you, Your Honor.

JUDGE SMOLEN: And we will receive them.

(Whereupon, the documents were marked as PECo Cross-Ex. Exhibits Nos. 5(a) and 5(b) for identification, and were received in evidence.)

MR. WATSON: Thank you.

JUDGE SMOLEN: Now, you have completed your cross-examination?

MR. WATSON: Yes, sir.

JUDGE SMOLEN: Do you need some time for redirect?

MS. McCLOSKEY: Yes.

JUDGE SMOLEN: Let's take a ten minute break.

(Recess.)

JUDGE SMOLEN: Back on the record.

Ms. McCloskey, go ahead.

MS. McCLOSKEY: Thank you, Your Honor.

REDIRECT EXAMINATION

BY MS. McCLOSKEY:

Q. Dr. Rosenbaum, if you could turn to page three, line two, of your testimony, where you utilized the odds ratio from the Savitz study of 1.54, could you please explain why you utilized the 1.54 odds ratio from the Savitz study?

A. There were two leukemia odds ratios listed in Table 7 of Savitz and perhaps I should have used both of them. Neither one of them had categories that were exactly comparable to the Peters study. And I pointed out in my testimony that you could actually calculate actually the same things that we have calculated here just from the data in the Savitz and Peters studies. But I didn't do that and I did not have that -- it's a long task to do it without the data that is in this report that was just handed to me by Mr. Watson a little while ago and I didn't have that so that I didn't actually do it. That is one of the two numbers that he reported. You could have also used the other one.

Q. And if you could turn to page four, lines 11 through 14 of your testimony -- I guess it continues on to line 15. Based on the calculations and the odds ratios that you report in your testimony there, is that

statement accurate?

A. Actually, I realized after a long time when Mr. Watson was questioning me right at the end that I had reversed the two names in that sentence and that was what it was all about. And indeed the rest of the paragraph goes along with the fact that there is -- I mean, the rest of the paragraph is right and in fact explains that.

It should say in the sentence that begins on line 11 if the effect increases monotonically this would make Peters' results weaker than Savitz's results. Which is why it says, indeed, when Savitz compared his wire code with very high wire code his leukemia ratio goes up to 2.75. And I'm sorry for the error.

JUDGE SMOLEN: Let's get it clear on the record what you mean in that sentence, which starts "if the effect".

THE WITNESS: What I meant was, since I had not calculated it, but what I meant was that if you do calculate comparable categories you would probably find that Peters' results were weaker than Savitz's results. Which is why the next sentence starts with indeed, and it explains that.

JUDGE SMOLEN: Go ahead.

MS. McCLOSKEY: Thank you, Your Honor.

BY MS. McCLOSKEY:

Q. Now, referring now to PECo Cross-Examination Exhibits 5(a) and 5(b), which are the charts, do you know whether the numbers that represent the cases and controls that Mr. Watson used from the Savitz study to calculate the odds ratios in those cross-examination exhibits are comparable to the numbers for cases and controls in the Peters study?

A. They are certainly not directly comparable because, for example, the age ranges in the two studies were different. In the Peters study the age range was from zeor to ten and in the Savitz study it was from zero to 14. And that is another problem that makes comparability difficult just as the different categories -- the way they chose to group the five categories was different.

Q. And the calculations of the odds ratios that appear on PECo Cross-Examination Exhibit 5(a), which is the chart actually under the other chart, in reviewing those calculations of odds ratios, does that change the conclusion in your testimony?

A. No. The conclusion about -- not at all. The conclusion that I made was a limited one. The only conclusion I was -- what I was trying to say and what it says is that the results of the Wertheimer & Leeper, Savitz and Peters studies do not indicate that if better

and better studies are done the effect will eventually go away. That was the point I was trying to make.

You cannot take three studies which represent at most two comparisons between the first and the second and the second and the third -- so there are three studies but there are only two comparisons involved. You cannot take two comparisons and make a general long range trend out of it, particularly when the categories are not even comparable, for example same. The age ranges were different. And when you include the Wertheimer & Leeper study there are lots of other things that were different. So in many ways you are just comparing Savitz with Peters, which is only one comparison in which there were a lot of things different. We have already mentioned an additional one here, which was the age ranges that they looked at were different.

So that certainly does not indicate any kind of trend at all and there is no logical deduction to be made from those two studies that if you keep doing more and more and better and better studies -- and by the way, I think the Peters study was very well done -- that it will go away.

My general point was, on the contrary, the most striking thing about the three studies is that they all reported positive associations. And there is at least as

much reason to believe from those three studies that continued studies would continue to report positive associations as there is that continued studies would make the effect go away. That was the point I was trying to make.

MS. McCLOSKEY: I have no further questions, Your Honor.

JUDGE SMOLEN: Anything else?

MR. SUGARMAN: Maybe two -- one anyway -- if Your Honor please.

RECROSS-EXAMINATION

BY MR. SUGARMAN:

Q. On page four, the sentence that you indicated the names were reserves, I would like to ask you, it may be self-evident to your mind but to say it on the record, is the opinion that you have expressed in this sentence as it was typed and submitted the opinion that you hold today and that the mistake was made in transcribing the names? Or has your opinion changed?

A. No. My opinion about the whole subject has not changed at all, and the discussion that follows the sentence where Savitz's and Peters' names were reversed in fact explains the right order.

Q. In other words, it's anomalous. If the sentence were intended to be as it were submitted in

writing, the remaining part of the paragraph would be anomalous?

A. The next sentence would make no sense at all.

Q. Right. So it is a merely an error in calling out the words, so to speak?

A. Yes. Unfortunately, once you do something like that, you tend to read what you think is there after you read it over and over and over again.

MR. SUGARMAN: I don't have any further questions. Thank you.

JUDGE SMOLEN: Anything else of the witness?

MR. WATSON: Nothing, Your Honor.

JUDGE SMOLEN: The witness is excused. Thank you very much for appearing and testifying.

(Witness excused.)

MS. McCLOSKEY: Your Honor, the Office of Consumer Advocate calls David Janes to the stand.

JUDGE SMOLEN: Now, you previously testified in this proceeding, sir?

MR. JANES: Yes, sir.

JUDGE SMOLEN: All right. You remain under oath.

Whereupon,

DAVID E. JANES

having previously been duly sworn, testified as follows:

MS. McCLOSKEY: Your Honor, I would like to have

marked for identification OCA Statement No. 1A.

JUDGE SMOLEN: So marked.

(Whereupon, the document was marked as OCA Statement No. 1A for identification.)

MS. McCLOSKEY: Which is the surrebuttal testimony of David E. Janes, consisting of two pages of questions and answers and one page of references, a bibliography.

JUDGE SMOLEN: That is marked for identification as OCA Statement No. 1A.

DIRECT EXAMINATION

BY MS. McCLOSKEY:

Q. Mr. Janes, do you have before you what has been marked as OCA Statement 1A, consisting of two pages of questions and answers and a page of bibliography material?

A. Yes, I do.

Q. Do you have any additions or corrections to that testimony?

A. No, I don't.

Q. Was the testimony prepared by you or under your direct supervision?

A. It was.

Q. And is this testimony true and correct to the best of your information, knowledge and belief?

A. It is.

Q. And if I were to ask you these questions today under oath would your answers be the same?

A. They would.

MS. McCLOSKEY: Your Honor, the parties and Your Honor were previously provided with Mr. Janes' surrebuttal testimony and I have provided two copies to the court reporter. I would move at this time for the admission of OCA Statement No. 1A subject to any timely motions and cross-examination.

JUDGE SMOLEN: Hearing no objection, it is received with those qualifications.

(Whereupon, the document marked as OCA Statement No. 1A was received in evidence.)

MS. McCLOSKEY: Mr. Janes is available for cross-examination.

JUDGE SMOLEN: We will start with Mr. Sugarman.

MR. SUGARMAN: Thank you, Your Honor.

CROSS-EXAMINATION

BY MR. SUGARMAN:

Q. Mr. Janes, at page two, lines 15 through 17, you indicate that you attach a special importance to the recently published study of Demers, et al.?

A. Yes.

Q. And I have a couple questions to ask you about that study and the importance that you attach to it.

The Demers study was a 1991 study that was published in the American Journal of Epidemiology, is that correct?

A. Yes.

Q. Do you have any familiarity with whether the American Journal of Epidemiology is a peer review publication?

A. I believe it to be a peer review publication.

Q. And do you -- was the study in question available when the hearings in this case started? Did you have the opportunity to consult it for purposes of your initial testimony?

A. No.

Q. So do you have any idea approximately when in 1991 it was published?

A. It should say.

(Witness perusing document.)

A. My copy does not show a date -- here it is. It was received for publication in February of 1991 and in final form in April, 1991. And when it actually hit the street would be sometime later than that.

Q. You had an opportunity, obviously, to review Dr. Gelmann's testimony, is that right?

A. Yes, that is true. I have seen Dr. Gelmann's testimony.

Q. Do you find any reference in Dr. Gelmann's testimony to the Demers study?

A. I don't recall.

Q. Now, lastly, I think, you indicate you attach a special importance to it. And again, the reason may be self-evident to you, but to get it on the record and for the benefit of those of us to whom it may not be self-evident, given the rest of your testimony, I mean, that is why you may think it is self-evident, but could you put it in context for us why a study of association between occupational exposure to electromagnetic fields and breast cancer in men is particularly relevant to Dr. Gelmann's testimony?

A. Let me try to put that in the context in which I intended this. I was addressing the issue of whether there was a requirement for additional research.

Q. Right.

A. Which is the question. And these two studies that are referenced here, the Demers study and Wilson's work, both relate to effects in humans.

Q. Of and on?

A. Well, they relate -- the Demers study relates to an incidence of cancer in males classified into categories exposed to electromagnetic fields. Wilson's study indicates that there are at least in some sensitive

individuals there may be some changes in melatonin concentrations that are associated with certain kinds of low frequency fields that arise from the use of electric blankets.

I don't want to push that any further than that, other than to say that it raises enough of a question, at least for me, from a public policy standpoint, that I would want to follow up and see what the relationship of the animal studies I referred to earlier in the melatonin piece and these particular things, whether or not they fit together.

Q. That is what I wanted to get you to say in one sentence, if it is a correct understanding of your testimony, and that is that the studies show effects of electromagnetic fields on melatonin levels and that melatonin levels affect incidence of chemically induced breast cancers in rats.

A. You could walk through that.

Q. Right.

A. At this stage of our knowledge, I would not make direct connections between those events.

Q. No, I'm not suggesting that you would. But I'm saying that those two findings which you referred to in your testimony are the reason why it is particularly important to do more research in the melatonin area as a

result of the Demers study and Wilson work, is that right? That is, the fact that EM/F affects melatonin and that melatonin affects incidence of chemically induced breast cancer in rats. Those two facts are the reason why the Demers work and the Wilson work is important as suggesting the need for additional research, am I correct?

A. All of those elements are equally important.

Q. Okay. Right. But it is the combination of them?

A. It is the collection of them.

MR. SUGARMAN: That's the point I wanted to make. Thank you.

I have no further questions.

JUDGE SMOLEN: Mr. Dillon.

MR. DILLON: No questions, Your Honor.

JUDGE SMOLEN: Ms. Burket.

MS. BURKET: No questions, Your Honor.

JUDGE SMOLEN: Mr. Watson.

MR. WATSON: Thank you.

CROSS-EXAMINATION

BY MR. WATSON:

Q. Hello, Mr. Janes. It is nice to see you again.

A. Good morning again.

Q. Would you take a look at page one, lines 27

through 30, of your surrebuttal testimony?

A. Lines 27 through 30? Yes.

Q. Here you discuss magnetic fields and melatonin, is that correct?

A. Correct.

Q. Now, this study that you are referring to by Lerchl used a 33.7 hertz magnetic field, not a 60 hertz magnetic field like this powerline, is that correct?

A. That's correct.

Q. Now, was this the study where the researchers sacrificed the rats, removed their pineal glands and then exposed the pineal glands after having been removed to EM/F and conducted the rest of the experiment?

A. Yes.

Q. And you are not suggesting that these results have been shown to occur in humans?

A. No.

Q. Now, could you turn to page two, lines 17 through 19, of your testimony where you discuss some research by Wilson?

A. Yes.

Q. Incidently, Wilson measured the change in melatonin in nanograms, is that right?

A. I have to look.

Q. Try page 263 of the study.

(Witness perusing document.)

A. He expressed his results in nanograms per milligram of creatinine. Now, what he actually measured, he measured some mass normalized to the mass of creatinine that appears in the urine. So he expressed his results in terms of nanograms per milligram of creatinine.

Q. Look at page 266, Table 2 there.

A. All right.

Q. Under exposure period, four, post exposure.

A. Uh-huh.

Q. Can you tell us what the units are that he is expressing the mass in?

(Witness perusing document.)

A. Well, the table doesn't indicate but the text does, as I recall, and they normalized to creatinine because the variances were smaller. That is what I recall from the text. The table does not indicate what the units are at the moment. I have to -- let's see if we can tie the things together. You will have to give me a moment.

Q. Sure.

(Witness perusing document.)

A. Page 264, Table 2, shows the group means in corresponding log transformed data expressed as nanograms

of 6-hydroxymelatonin sulfate per milligram of creatinine.

Q. So in terms of the measurement we are talking nanograms? I'm just trying to identify the starting point here. Nanograms of something?

A. I'm not sure because -- and I don't have the raw data in front of me. But once you divide by milligrams in the denominator, I don't know what the enumerator was actually measured in. It is a ratio. And since it is nanograms per milligram --

Q. But look at page 264 at the bottom where it says results.

A. Uh-huh.

Q. Could you read that first sentence there for us that starts with "Table 2"?

A. "Table 2 shows the group means in corresponding log transformed data expressed as nanograms of 6-hydroxymelatonine sulfate per milligram of creatinine.

Q. And the results that he reported were changes on the order of two, three or four nanograms, if you know? Per kilogram of --

A. Per milligram of creatinine.

Q. Per milligram of creatinine, correct?

A. Correct.

Q. Milligram.

A. You are dealing with a ratio. And if he doesn't give you the enumerator or denominator you are not going to be able to derive out of the study what the actual units of measure were.

Q. Am I right that a nanogram is one-billionth of a gram?

A. Ten to the minus nine, yes.

JUDGE SMOLEN: I can't hear you. Keep your voice up.

THE WITNESS: I'm sorry.

Yes, a billionth.

BY MR. WATSON:

Q. Could you look at page two, lines 10 and 11 of your testimony?

A. Page two, lines 11 --

Q. Ten and 11.

A. Yes.

Q. I believe there you begin to characterize this portion of your testimony as a summary.

JUDGE SMOLEN: Wait. Off the record.

(Discussion off the record.)

BY MR. WATSON:

Q. Mr. Janes, let's try that again. Let me refer you to page two, lines 10 and 11 of your testimony.

A. Yes.

Q. There you characterize that portion of your statement as a summary?

A. Uh-huh.

Q. You begin with the phrase "in summary". I would just like to ask a few questions about the scope of that summary.

Now, would it be fair to say that you are not saying that it has been proven that electric fields alter melatonin levels in humans?

MR. SUGARMAN: I object to that. The implication of the question is too strong, I think. It is that -- it is asking the witness to either agree or disagree with a statement that is -- that suggests an opinion on the subject one way or the other. He has not expressed an opinion on it one way or the other.

If the witness agrees with the question or responds -- it's a yes or no question. If the witness responds yes, then he is agreeing that this doesn't prove it. He hasn't said it doesn't prove it. If he disagrees with the question -- it is like those typical questions that there's no good answer to.

MR. WATSON: It sounds like to me that the witness after --

JUDGE SMOLEN: Let's hear the question again.

MR. WATSON: -- after this exposition can say

whatever he wants.

MR. SUGARMAN: Well, don't ask a yes or no question.

MR. WATSON: Your Honor, I think I'm entitled to ask him whatever question I want.

MR. SUGARMAN: Your Honor --

JUDGE SMOLEN: Wait a minute. One voice at a time. I have not made a ruling. You've got an objection. I've asked for the question to be repeated.

MR. WATSON: I would like to have the reporter read the question back.

JUDGE SMOLEN: All right.

(Whereupon, the reporter read from the record as requested.)

MR. SUGARMAN: I withdraw the objection. I thought he asked a different question.

JUDGE SMOLEN: Please answer the question.

A. No. I didn't say that it proves that.

BY MR. WATSON:

Q. And you are not saying that it has been proven that magnetic fields alter melatonin levels in humans, are you?

A. No.

Q. And as I understand your testimony, you are not saying that it has been proven that magnetic fields alter

melatonin levels in animals, correct?

A. I think we are into a definition of proof and what one means by saying proven. There are certainly a number of studies which indicate that melatonin levels are -- magnetic fields alter melatonin levels in animals.

Q. And I'm asking you whether you are now testifying that that has been proven, that magnetic fields alter melatonin levels in animals.

MR. SUGARMAN: Objection, asked and answered.

BY MR. WATSON:

Q. Referring to animals completely, all animals.

MR. SUGARMAN: Objection, asked and answered.

MR. WATSON: It has not been asked and answered, Your Honor. He didn't answer that.

JUDGE SMOLEN: If it has been asked and answered then the worse it is is surplusage. So in order to save time I'm going to let him go ahead and answer it.

A. No, it has not been proven that magnetic fields affect melatonin levels in all animals.

BY MR. WATSON:

Q. And would it be fair to say that you are really talking primarily about rats as the animals involved in these studies?

A. Primarily. Not exclusively.

Q. Now, could I direct your attention to page two,

lines one through four?

A. Yes.

Q. And there you make reference to Dr. Reiter, Russell Reiter?

A. Yes.

Q. Now, you refer to long-day and short-day breeders in this statement?

A. Uh-huh.

Q. Just to keep the record clear, you are talking about animals but not about humans, is that correct?

A. Correct.

Q. Now, this article by Dr. Reiter that you have cited here, it's not actually a study with data, is it?

A. No, it is not.

Q. And did you personally review the primary articles and the data that are discussed in Dr. Reiter's review article?

A. Not all of them.

Q. Now, the Reiter article suggests that this synchronization of long-day and short-day breeders occurs through a melatonin effect on gonadotrophins, correct?

A. Probably more complex than that. But the indication is that elevated melatonin levels provide a signal to the animal as to what the light/dark cycle is, and animal behavior is governed accordingly and probably

mediated through hormones.

Q. And the particular actions that they are talking are gonadotrophic actions, that Dr. Reiter is referring to, would that be correct? I refer you to page 226 of his article.

A. Yes. Principally, yes.

Q. So you are basically reporting an article by Dr. Reiter that you read. You are not now testifying as an expert on melatonin and any relationship it has, if any, between it and sexual function?

A. No. I'm not testifying as an expert on sexual function. I will mention that I have discussed this work with Dr. Reiter.

Q. Would you take a look at page two, lines four through eight of your testimony where you discuss a study by Tamarkin?

A. Yes.

Q. Now in this study Tamarkin administered the melatonin in milligram doses, actually 2.5 milligrams per kilogram of body weight, correct?

A. Let's look because there were several dose levels in the Tamarkin study.

(Witness perusing document.)

A. He used a range of -- let's see...

(Witness perusing document.)

A. The dosage of melatonin that I find quickly scanning through this is 500 micrograms. I don't have immediately at hand the --

Q. Could I refer you to the abstract, the fourth line down. It says melatonin and then --

A. Two-and-a-half milligrams per kilogram?

Q. Right.

A. That works out about right. Yes, that's fine.

Q. Okay. So he basically administered the melatonin in milligram doses? We can at least say that?

A. Now we are back to ratios again.

Q. Well, milligram per kilogram doses?

A. Yes.

Q. And a milligram is one-thousandth of a gram?

A. That's correct.

Q. Can you tell us how many years people have been researching melatonin and cancer?

A. Not specifically, but not -- it's not a new avenue of study.

Q. In fact, this Tamarkin paper that you cited was published in 1981?

A. Yes. And there was earlier work than that.

Q. And Wilson published his first papers in this area as early as 1981, didn't he?

A. I could check. That seems to be my

recollection. If it is critical, I could look.

Q. Well, you have a copy of the Battelle book that we have referred to earlier, page ten?

A. Not with me at this time.

Q. Well, let me share this with you.

A. It seems to me that the early work is around 1981.

MR. WATSON: Do you want to see this again?

MR. SUGARMAN: Let me just take a look.

A. It was published, '81, '83, '86 and '89.

BY MR. WATSON:

Q. So he has research going back to '81, Wilson.

(No audible response.)

Q. Now, you have suggested that there should be some further research in this melatonin area, correct?

A. Correct.

Q. Can you tell us how many years people have been researching the physiological role, trying to figure out the physiological role, of the pineal gland?

A. No. It is an old, old question but I wouldn't try to quantitate that for you.

Q. Now, you are familiar with Tamarkin's work. I'm going to just ask you if you are familiar with this work by Tamarkin. Would you just take a look at this and see if we talking about the same Tamarkin, the same

researcher and the same research that you are talking about?

A. They are both from the same place. I presume it is the same individual.

Q. Take a look at the second full paragraph of that.

A. All right.

Q. Does it say there systematic investigations of the mineal and pineal gland began in the 1880s?

A. That is what it says.

Q. Do you accept that as true or do you know if that is accurate?

A. I have no basis to disagree with it.

Q. Now, I believe you referred, Mr. Janes, to a study by Demers.

MS. McCLOSKEY: Excuse me. It just struck me. You had mentioned that this is the same research. You are referring to the area of research, not the same research article that was in the testimony? In other words, was your question was the Tamarkin study the same article that you handed him that he cited in the testimony?

THE WITNESS: No. I think the question I answered was whether this article --

JUDGE SMOLEN: Wait, sir. This was a question by Counsel to Counsel.

MS. McCLOSKEY: Yes, just before we moved off that I wasn't sure that he had answered that, that we were not discussing the same article, but discussing the same area.

MR. WATSON: I think he said we are discussing the same area and it is the same researcher. I didn't hear him say it's the same article. And I asked him, then, a question about this generic statement about how long they have been researching this and he said he didn't know. And so I figured in those circumstances I would just leave it there. There was nothing else to pursue because he couldn't confirm the statement and the article.

MS. McCLOSKEY: Just for the clarity of the record, I just want to be clear that the article that he was looking at in reference to your question was not the same article cited in his testimony.

JUDGE SMOLEN: Well, you can ask him that when you get around to redirect.

MR. WATSON: Counsel, based on what he said, that was my understanding.

MS. McCLOSKEY: I just thought there was a question he had been asked that he didn't answer. I'm sorry.

MR. WATSON: Your understanding and mine are the same, then. I think what you said was right.

MR. SUGARMAN: I want object to the statements in

the pending question, as I understand it, about research into this area as being hopelessly vague and talking about subjects that have nothing to do with this case. The witness was recommending additional research on the uncertainties about the role that melatonin plays in carcinogenesis as related to electric fields. And the questions -- when he talks about research in this area, the research that he is asking about has nothing to do with -- is much broader.

JUDGE SMOLEN: I don't think we have an open question.

MR. SUGARMAN: I thought there was a question on that.

JUDGE SMOLEN: There is no open question.

MR. SUGARMAN: Fine. I'll wait for a question.

MS. McCLOSKEY: I'm sorry. I thought he asked a three part question and only answered two.

JUDGE SMOLEN: Let's have a question.

BY MR. WATSON:

Q. Dr. Janes, you refer to a study by Demers, correct?

A. Yes.

Q. The Demers study is an epidemiological study, correct?

A. That's correct.

Q. And previously you said very candidly that you don't claim to be an expert in the field of epidemiology?

A. That's correct.

Q. At page two, lines 16 and 17, of your testimony you referred to the Demers study as having reported, quote, an association between occupational exposure to electromagnetic fields and breast cancer, close quote. Do you see that?

A. In men.

Q. In men. Yes.

Now, Demers did not actually measure or calculate any magnetic fields or collect any EM/F exposure data of any kind, did he?

A. Not in the narrow sense that I think you mean exposure. He collected occupational work histories.

Q. And so he didn't report an association between actual exposure to EM/F and male breast cancer. What he reported dealt with occupational categories and breast cancer?

A. That's correct.

Q. In fact, didn't Demers say in his study that the design of the study precluded any formal evaluation of cancer risk in relation to even the probability of exposure to EM/F? And I can refer you to page 345 of his study.

A. Why don't we do that so I get the context.

(Witness perusing document.)

Q. Look in the left column under the heading discussion.

A. Uh-huh.

(Witness perusing document.)

Q. And look in the last sentence. It begins with "however".

A. "However, the lack of exposure measurements and the high variability possible within each occupation preclude any formal evaluation of breast cancer risk in relation to either intensity of exposure or probability of exposure."

MR. WATSON: Thank you, Dr. Janes.

THE WITNESS: You keep promoting me. It is Mister. But thank you anyway.

MR. WATSON: That is all I have, Your Honor.

MR. SUGARMAN: I have a couple of questions.

RECROSS-EXAMINATION

BY MR. SUGARMAN:

Q. Are you familiar with the Electric Power Research Institute?

A. In a general sense.

Q. What is the Electric Power Research Institute.

MR. WATSON: Objection, Your Honor. Beyond the

scope of the cross examination.

MR. SUGARMAN: It goes to the question of the suggestion that additional research is not needed. EPRI is recommending additional research and stating that additional research is needed. EPRI is another recognized authority on the subject of electrical issues. In fact, it is the authoritative arm of the electrical power industry for research --

MR. WATSON: Your Honor --

MR. SUGARMAN: -- funded by PECo, among others.

MR. WATSON: He was not asked any of that. The witness' testimony has no reference to that at all.

MR. SUGARMAN: First of all, he is not my witness. So if Mr. Watson can cross-examine him --

JUDGE SMOLEN: Well, it is friendly cross-examination when you are cross-examining this witness.

MR. SUGARMAN: I wouldn't say I'm fighting him, but...

JUDGE SMOLEN: He is not your witness. I'm going to let it go a little bit. So I will overrule.

BY MR. SUGARMAN:

Q. What is EPRI, Mr. Janes?

A. I don't know that I can give you a definitive answer. My understand of what EPRI is, it stands for the

Electric Power Research Institute and it is an organization in California that is supported by contributions from utilities that engages in a wide range of research of things related to transmission and distribution of electricity, and included in that is some work on the effects of 60 hertz electric and magnetic fields.

Q. Are you familiar with whether EPRI had anything to do with the Peters study?

A. I believe that EPRI was a sponsor --

MR. WATSON: Objection, Your Honor.

JUDGE SMOLEN: Now we are going far afield so I'm going to sustain.

BY MR. SUGARMAN:

Q. Has EPRI, to your knowledge, taken a position on the need for additional research?

MR. WATSON: Objection, Your Honor. This is beyond the scope of the cross examination.

MR. SUGARMAN: It is directly related to the scope of cross-examination.

JUDGE SMOLEN: Overruled.

MR. SUGARMAN: Thank you.

A. Do you want to restate the question?

BY MR. SUGARMAN:

Q. Has EPRI taken a position, to your knowledge,

on the need for additional research?

MR. WATSON: The same objection.

JUDGE SMOLEN: Overruled.

A. I think the only way to be responsive to that is to say I know EPRI funds research on the biological effects of power frequency fields.

Q. Is it funding research at this time?

A. To my knowledge, yes.

Q. Are you familiar with the Institute of Electrical and Electronics Engineers?

MR. WATSON: Objection, Your Honor.

JUDGE SMOLEN: I understand, the same objection.

MR. WATSON: Beyond the scope. He didn't testify on it and I didn't cross-examine him on it. This examination is limited to the cross-examination.

MR. SUGARMAN: The cross-examination went to the need for additional research.

MR. WATSON: No, it did not, Your Honor?

MR. SUGARMAN: It didn't? Then I move to strike the cross-examination --

JUDGE SMOLEN: Just a minute. One voice at a time. Let Mr. Watson finish.

MR. WATSON: This is beyond the scope of the cross-examination of this witness -- and also his testimony, but it is certainly beyond the scope of the

cross-examination. There was no reference to either one of these subjects, certainly not to the Institute of Electrical and Electronics Engineers in any form or fashion.

Counsel is simply trying to use this witness to try to backdoor information in that he otherwise could not get in that this witness did not testify to. And he has already cross-examined him once.

JUDGE SMOLEN: Go ahead. Again, responding to Mr. Watson's objection.

MR. SUGARMAN: This is the first area of questions that have attacked the need for additional research in the area. If Mr. Watson will stipulate that he was not questioning the need for additional research in the area of the effect of EM/F on cancer by way of melatonin, then I will withdraw my question.

MR. WATSON: I'm going to stipulate that the questions that I asked him are the ones that the reporter transcribed.

JUDGE SMOLEN: I'm going to overrule now. Ask the question.

MR. SUGARMAN: Thank you.

BY MR. SUGARMAN:

Q. Are you familiar with the Institute of Electrical and Electronics Engineers?

MR. WATSON: The same objection.

JUDGE SMOLEN: Overruled.

A. Yes.

BY MR. SUGARMAN:

Q. What is that organization?

A. It's a professional organization of engineering and related type scientists who have interest in a broad range of electrical engineering problems, everything from computer science to power transmission. I'm a member of that society.

Q. Are you familiar with the United States Activities Board of that organization?

MR. WATSON: Objection, Your Honor.

JUDGE SMOLEN: Overruled.

BY MR. SUGARMAN:

Q. Do you know what it is?

A. Yes, I know what it is.

Q. What is it?

A. But just marginally. It is the United States Activities Board and it is one organization, I guess, within the IEEE that takes positions on things. But I don't follow that part of it very carefully.

Q. Are you familiar with the entity position statement issued by the United States Activity Board with respect to the biological effects of power frequency

electric and magnetic fields?

MR. WATSON: Objection, Your Honor. The witness did not testify on this statement. He did not at all in his surrebuttal testimony let alone was he asked about any such statement in cross-examination.

JUDGE SMOLEN: Here I'm going to sustain. It's going very, very far afield, particularly in light of the witness' answer to the previous question.

MR. SUGARMAN: I'm only asking him whether -- it goes to the rebuttal to the cross-examination about whether additional research is needed. If Mr. Watson is not challenging the need for additional research, which he has already refused to stipulate to, fine. But since he apparently is, I'm entitled to on rebuttal, since he raised that for the first time now --

JUDGE SMOLEN: This is not rebuttal. This is recross. It is recross.

MR. SUGARMAN: I'm entitled to ask the witness --

JUDGE SMOLEN: Ask your question again. I sustained the last one. Ask this question again -- or another question.

BY MR. SUGARMAN:

Q. Do you know whether the United States Activities Board is an authoritative entity of the Institute of Electrical and Electronic Engineers,

authorized to issue position statements.

MR. WATSON: Objection, Your Honor.

JUDGE SMOLEN: That is sort of two questions that you asked. Break it up again. We will sustain that one.

BY MR. SUGARMAN:

Q. Do you know if the United States Activities Board has the authority to issue position -- or to take positions on behalf of the institute, or to take positions that are associated with the institute?

MR. WATSON: Objection, Your Honor.

JUDGE SMOLEN: Overruled.

A. I really don't know.

BY MR. SUGARMAN:

Q. Do you know whether the United States Activity Board has taken a position on whether there is a need for additional research in order to define safe and unsafe levels of human exposure to power frequency fields?

MR. WATSON: Objection, Your Honor. The witness' testimony --

JUDGE SMOLEN: He didn't ask what the study was but whether he knows of a position having been taken.

MR. WATSON: I understand, Your Honor. But my objection goes to the point that the witness did not testify to that on the cross-examination. Mr. Sugarman has something for rebuttal where he is trying to

introduce extraneous material that this witness never covered.

JUDGE SMOLEN: I understand your argument.

MR. WATSON: It goes way beyond.

JUDGE SMOLEN: It does. However, in administrative proceedings where we are not really bound by strict technical rules of evidence, we liberally construe them. So I'm going to overrule at this time and let the witness answer. If he remembers the question.

A. I would have to respond this way. Some time ago, and I don't know how long, some entity within the IEEE issued a position statement with respect to research. Now, whether that was the U.S. Activity Board or some other group, I don't know. I have seen it, but it's been a long time. I don't recall.

BY MR. SUGARMAN:

Q. Let me show you the document and ask you if it refreshes your recollection that this is the document that you saw.

MR. WATSON: Your Honor, I object to this. He is introducing extraneous materials that this witness did not testify on at all.

JUDGE SMOLEN: Yes. I know your objection is strenuous and vigorously expressed.

Now, I haven't heard Ms. McCloskey, her position

with respect to this.

MS. McCLOSKEY: Your Honor, up until the point I believe Mr. Janes has attempted to answer these questions and he has expressed that he is not familiar with that information and then it goes back to a long time ago. It is not information that he presented in his testimony but he did address the need for additional research.

JUDGE SMOLEN: Yes, I recognize that he addressed that need and he was asked questions about it with respect to his testimony on page two, that that testimony was related to a requirement for additional research. I believe that was the witness' testimony.

Is that your recollection?

MS. McCLOSKEY: That's correct, Your Honor.

JUDGE SMOLEN: And he has already testified to do that.

MS. McCLOSKEY: That's correct, Your Honor.

MR. WATSON: Your Honor, could I just make the point that the reference to additional research was his statement about additional research to resolve the present uncertainty about the role that melatonin plays in carcinogenesis. Just melatonin.

And, I would point out, that did I not ask him about that statement.

JUDGE SMOLEN: I understand.

We don't want to turn this into a direct testimony, Mr. Sugarman. And that is what apparently you are doing or attempting to do.

MR. SUGARMAN: I'm trying to basically offset any effect that Mr. Watson's cross-examination may have had.

JUDGE SMOLEN: I understand what you are trying to do. But we have to be guided by some rulings of evidence no matter how liberally the Commission construes them.

MR. SUGARMAN: Agreed. I just wanted to clarify -- I mean, in a sense -- I mean, he is not my witness as one can readily see and I may be aligned with him to some extent or my clients position may be aligned to his position to some extent; to some extent it may not.

The issue, I think, is whether this line of questioning is fairly related to Mr. Watson's line of questioning.

JUDGE SMOLEN: The witness testified, and I will ask the witness to correct me if my recollection is incorrect, that you have very limited knowledge, if at all, regarding the particular division or bureau of EPRI about which you were questioned. Is that correct?

MR. SUGARMAN: This is not EPRI, Your Honor.

JUDGE SMOLEN: Well, excuse me. The other.

MR. SUGARMAN: Electrical and electronics.

THE WITNESS: There are a large number of entities

inside the IEEE. The U.S. Activities Board is one. And I just confess that it is not something that I have looked at very hard. So I don't know what the role of the U.S. Activities Board specifically is. I could guess, but --

JUDGE SMOLEN: Well, we don't want you to guess.

THE WITNESS: Okay.

JUDGE SMOLEN: I think in view of that answer that limits this witness' ability and qualifications to tell us what the U.S. Activities has done. I know you can present him that paper. You can present him comic strips to read from. But that is not within this witness' knowledge. So in that sense I'm going to sustain.

BY MR. SUGARMAN:

Q. Let me go back to the specific questions that Mr. Watson asked. You were asked about how long there has been research into physiological role of the pineal gland. Do you remember that line of questioning by Mr. Watson?

A. Yes.

Q. Can you tell me how long there has been research into the role of the pineal gland as related to melatonin and cancer causation?

MR. WATSON: Asked and answered, Your Honor. 1981.

MR. SUGARMAN: Where?

JUDGE SMOLEN: I will let him answer again. Overruled.

A. The role of melatonin and cancer, apart from electromagnetic fields, I think was investigated at least as early as '81 by Tamarkin and perhaps by some others earlier than that.

BY MR. SUGARMAN:

Q. My question went to pineal gland as related to melatonin and EM/F exposure. Maybe I didn't put that in there so let me rephrase the question.

My question goes to, and it goes back to my earlier cross, the combination of the factors, pineal gland, melatonin, EM/F and cancer. How long has the relationship between those elements been under research?

A. I think I understood your question the first time and that is what I was trying to address.

Q. I'm sorry. Go ahead.

A. I'm not aware of any direct studies of electromagnetic fields on pineals and cancer all in the same study.

Q. Thank you.

A. And I think that is what I called for here, was to close that gap.

Q. Thank you. You have anticipated my next question.

Now, Mr. Watson asked you about the use of rats and you agreed with him that the studies primarily have utilized rats. Is there a consensus in the scientific community in general in designing biological research to use rats as relevant to human effects?

MR. WATSON: Objection, Your Honor. Lack of foundation. It has not been established that this witness could sit here and speak for the biological research community. In fact, I believe he has testified that he is not in that arena.

JUDGE SMOLEN: Sustained.

MR. SUGARMAN: I will lay a foundation.

BY MR. SUGARMAN:

Q. In your direct testimony in your initial appears here how did you qualify yourself and how were you -- what did your qualifications -- what did you set forth about your qualifications?

A. My training is in biophysics with emphasis on molecular level interactions, particularly protein acids. I have an ABT degree in biophysics. I have done experimental work on radiation effects on systems. I directed research programs within both the Department of Health and Human Services and the Environmental Protection Agency relating to ionizing and non-ionizing, including 60 hertz, fields. And I have been engaged at

least during the last part of my career in the public policy aspects of trying to set standards and in reviewing research for providing protection to public health.

Q. In your work in supervising research -- or funding research?

A. I have done both.

Q. In your work in supervising research, funding research, doing research and making public policy or giving advice as to public policy implications as to research, have you maintained a familiarity in a general sense with the appropriateness of using rats as relevant material in order to ultimately derive human effects conclusions? By ultimate I mean directly or indirectly down the road.

A. The rat is one model that is commonly used to look at effects. There are a great deal of -- a great number of factors that go into selecting a particular animal system to investigate an effect. So that one tries to tailor the chosen animal to the effect and the kind of physiology or biochemistry or whatever you are looking for. Rats are commonly used in this type of experiment, not exclusively and not appropriately for all things.

MR. SUGARMAN: Thank you. No further questions.

JUDGE SMOLEN: Do you have anything?

MS. McCLOSKEY: Could I have just a moment, Your Honor?

JUDGE SMOLEN: Yes.

(Pause.)

MS. McCLOSKEY: We have no redirect, Your Honor.

JUDGE SMOLEN: Anything further of this witness?

MR. WATSON: Nothing, Your Honor.

JUDGE SMOLEN: The witness is excused. Thank you very much for appearing and testifying.

(Witness excused.)

JUDGE SMOLEN: I have 12:45. Do we want to break at this time for lunch and then return? What is the pleasure of Counsel?

MR. WATSON: Why don't we break. It is going to take a little while.

JUDGE SMOLEN: All right. Let's come back at two o'clock.

(Whereupon, at 12:45 p.m., the hearing was adjourned, to be reconvened at 2:00 p.m. this same day.)

AFTERNOON SESSION

(2:00 p.m.)

JUDGE SMOLEN: Back on the record.

Mr. Watson.

MR. WATSON: Thank you, Your Honor.

I call Dr. Philip Cole.

JUDGE SMOLEN: Dr. Cole, you have previously been sworn in this proceeding,, have you not?

DR. COLE: Yes, sir.

JUDGE SMOLEN: You remain under oath.

Go ahead, Mr. Watson.

MR. WATSON: Thank you.

Whereupon,

PHILIP COLE

having previously been duly sworn, testified as follows:

DIRECT EXAMINATION

BY MR. WATSON:

Q. Dr. Cole, have you reviewed the written surrebuttal testimony filed in this proceeding by Dr. David Janes?

A. Yes, I have.

Q. And were you in the hearing room earlier today to hear Mr. Janes' testimony?

A. Yes, I was.

Q. I would like to begin by asking you a few

questions about the Demers study which was referred to in Mr. Janes' testimony. Are you familiar with that study?

A. Yes, I'm.

Q. Is Demers a epidemiological study?

A. The Demers study is an epidemiologic study, yes.

Q. In your previous testimony you pointed out that several designs and methods have been used in the epidemiological studies of EM/F. Do you recall that?

A. Yes, I recall testifying to that effect.

Q. Just to keep the record clear, what design or method was used in Demers?

A. The Demers study is a case control study.

MR. WATSON: I just want to make sure we are talking about the same study here.

MR. SUGARMAN: Have you got a copy of this that I could have?

MR. WATSON: No.

MR. SUGARMAN: No problem.

BY MR. WATSON:

Q. Is this the -- I'm just going to show you this to make sure we are all talking about the same thing. Is this the Demers study that I have asked you about whether you are familiar with? Is this the one?

A. This is the one that I have understood you to

be asking about, yes.

Q. Entitled Occupational Exposure to Electromagnetic Fields and Breast Cancer in Men?

A. Correct.

Q. Now, in his testimony Mr. Janes stated that the Demers study reported an association between occupational exposure to electromagnetic fields and breast cancer in men. Do you recall that statement?

A. Yes, I do.

Q. Now, let me ask you this. This says that this study -- actually, a statement -- this Demers study was funded by a grant of NCI, Paul Demers was supported by a training grant in environmental epidemiology and biostatistics from the National Institute of Environmental Health Sciences. Can you tell us what a training grant is in that respect?

A. A training grant from NIEHS is typically used to support a student who is in the process of earning a degree, typically a doctoral degree but not always. I would infer that this supported Dr. Demers' work towards his PhD. degree.

Q. Would you have a familiarity with epidemiological research on breast cancer with men and women?

A. My major epidemiologic interest has been in the

area of the epidemiology of cancer of the breast.

Q. And how many years have you been involved in that?

A. I have worked in that area virtually the entirety of my professional career. My first paper in that area was published in 1969 and I believe I have published 30 or more papers on epidemiology and causation of cancer of the breast.

Q. So 20 some years of experience?

A. Twenty-five or so.

Q. Can you tell me how long has research been going on regarding the causes of breast cancer and more specifically here possible --

MR. SUGARMAN: I object.

MR. WATSON: Could I finish?

JUDGE SMOLEN: Let him finish the question.

BY MR. WATSON:

Q. -- and more specifically here, possible endocrine causes?

MR. SUGARMAN: I object to the question. We had this discussion in the context of Mr. Janes' testimony. Mr. Watson asserts that he is not asking questions that are relevant to the question of whether EM/F -- whether there is an association between EM/F and cancer and these questions certainly are not, and therefore it is

irrelevant to this proceeding how long studies have been going on relating to cancer of the breast and epidemiology. It is only relevant how long they have been going on with respect to EM/F and cancer of the breast.

MR. WATSON: Your Honor, in fact, I believe the points made by Dr. Janes refer specifically to a relationship between pineal melatonin and breast cancer and the question of whether in the first place, or in the second place more specifically, whether EM/F would affect pineal melatonin which would in turn affect breast cancer. I'm asking about that research specifically, and I'm asking about the first part of the connection and I will ask about the second part of the connection. It makes no sense whatsoever to suggest that we can somehow limit it only to the question of whether EM/F affects melatonin. Entirely relevant to that question is whether melatonin at all is associated with breast cancer.

MR. SUGARMAN: Your Honor, the issue is not whether we can ask one question or the other. The only asserted relevance for the issue of how long research is going on is the Applicant's contention that the research has gone on long enough. In order to make that contention we have to know what research we are talking about and the fact that research may have been going on in this more general

area for 150,000 years is irrelevant if we have just started to look at the key connection which is the ultimate relevant one. The first one is irrelevant without the second, and Mr. Janes in his testimony pointed out that you have to look at the whole of the interrelationship in order to test whether you need research. The fact that research may have established or may have gone on for some period of time in the general area is utterly irrelevant to this proceeding.

We are not talking about whether it is necessary as a part of the research. We are talking about whether the fact that it has gone on for a long time obviates the need to do anymore. The fact that one half of it has gone on for a long time is of no relevance.

MR. WATSON: Your Honor, Mr. Sugarman is welcome to argue that it is of no relevance. But I don't think that is the issue here.

JUDGE SMOLEN: I'm going to overrule the objection. The question stands. You may answer.

A. We generally consider that the modern era of breast cancer causation and the study of that dates from about the mid-1920s. At that time attention focused both on the prospect that the disease was caused by endocrine factors and also by a virus. In the ensuing 65 or so years attention has focused almost entirely on the

endocrine causes of this disease.

BY MR. WATSON:

Q. Now, how did the researchers in the Demers study decide whether to categorize a person as exposed to EM/F?

A. The researchers in the Demers study used a somewhat inadequately described method for characterizing individual people. They say that in part they used classification systems that had been suggested by prior investigators. However, they don't actually lay out this classification scheme.

From my perspective what has been used in the Demers study is what I would call a title classification. That is, they characterize all the individuals, cases that have been with breast cancer and controls that have been without, according to the occupational title they used for a part of their working life.

Q. Then I guess more specifically, based on your review of the Demers study, did it include any actual calculated or measured magnetic field data?

A. No. There was no measurements of magnetic field that were made and no effort was made to impute to the categories what the likely level of magnetic field exposure in the category would be. As a matter of fact, the paper makes it clear that in any given category there

could be a substantial proportion of people who would be exposed and a substantial proportion who would not, and this would apply to virtually all of the categories.

Q. What is the significance of that from the standpoint of epidemiology?

A. The significance is that the categories which are claimed to reflect different levels of exposure to magnetic fields actually may not and in my judgement probably do not.

Q. Now, let's look for a moment at the data that Demers did report. Can you generally tell us was the study of male breast cancer, female breast cancer or both?

A. This was a study restricted to men and breast cancer.

Q. And did they report a dose-response relationship?

A. Well, the Demers study has three categories of major limitation. Specifically to answer your question, they attempted to look at dose-response. But I would say more fundamentally the study design is somewhat lacking in that there was this use of occupational titles which could not be validated and which could not be related to actual exposure.

In addition, in the design area, they also used

random digit dial as an approach to selecting controls. While this is an acceptable approach to control selection it is probably the weakest one. For one reason, you cannot determine what the actual response rate was among the controls.

So much for the design.

When we get on to the major analysis they do report an overall association between some of these categories and breast cancer. However, they attempted to look at the relationship between breast cancer risk and what we could call dose-response or degree of exposure in two ways. The first way that they attempted to look at it was what we might describe as an intensity measure. That is, they presumed that the different categories would reflect different levels of intensity of exposure and they did not find any relationship of disease risk with increasing intensity of exposure.

Then, secondly, they attempted to look at duration. That is, to see whether or not the risk went up as they went up the scale of duration of employment in these supposedly exposed occupational categories. And there was no effect there either, none at all. No relationship of the disease risk, then, with either intensity of exposure or of duration. So in both senses of the word there was no dose-response effect.

Q. What is the importance of whether or not they show a dose-response?

A. In epidemiologic research because of its non-experimental nature and the inability to control the circumstances of exposure, dose-response is perceived as an extremely important, virtually crucial, characteristic of the data if it is to be interpreted as supporting a cause-effect relationship. The absence of it, while not necessarily eliminating the association, would make it extremely dubious and I don't think most epidemiologists or most scientists, for that matter, would be willing to interpret as causal a data set that does not demonstrate a cause-effect relationship -- I'm sorry -- that does not exhibit a dose-response relationship.

Q. Did the Demers study provide data on the age of the subjects when they were exposed to EM/F?

A. Yes. This brings us to the third major limitation of the Demers study. The finding was that while there was some association between breast cancer risk and these occupational titles, this association had these two rather surprising characteristics: one, that it was restricted to people who held these jobs prior to age actually, that is, even if these job titles were associated with risk it was not so for people who began the work after age 30. And secondly, the risk was

restricted to those people who held the jobs for more than 30 years. So we had the rather strange thing that only those people who were exposed to an early age for very long periods of time were exhibiting any increased risk.

This is extremely problematic because it doesn't jibe with the general perception of how carcinogens act. But most especially, as you may be aware, this particular question that is in front of us today, that is, the carcinogenicity of electromagnetic fields, has been advocated on the basis of the idea that the magnetic fields act as a late stage carcinogen, that is, as a promoter. Well, if you were to take the Demers study as valid, and I don't, but if you were to take it as valid then it would suggest that electromagnetic fields act only as an initiator, that is, an early stage carcinogen. So this is directly contrary to the existing positive epidemiologic information and is frankly, from my perspective at least, not credible.

Q. Let me refer you to a statement by Dr. Janes on page one, lines 24 and 25, of his testimony.

A. Mr. Watson, I don't have his testimony.

Q. It's one sentence. Let me read it.

"In my opinion additional research is needed to resolve the present uncertainties about the role

melatonin plays in carcinogenesis."

Let me just ask you this, does the research presently support a role for melatonin in causing breast cancer?

A. None at all. The vast majority of research suggests that some other hormones and endocrine substances may be related to breast cancer but I know of none that supports the idea this melatonin is.

Q. Have you reviewed the written surrebuttal testimony filed in this proceeding by Dr. David Rosenbaum?

A. Yes, I have.

Q. And you were here in the hearing room earlier today to hear Dr. Rosenbaum's testimony?

A. Yes, I was.

Q. Let me refer you to page one, lines one through 27.

A. I do have a copy of this.

Q. Oh, you do. Okay. Page one, lines one through 27.

Now, I would like to refer you with respect to that material to the term identified there as irregular statistically significant associations.

A. Yes. Fine.

Q. Just to clarify the record, what do you mean by

the term irregular statistically significant associations?

A. I would like to say that the context in which I was using the word was a discussion of some of the Savitz results and particularly some of the Savitz results that as I understood it were intended to address the question of whether or not there was a dose-response, that is, whether or not the odds ratio, the estimate of risk of cancer, increased in some fashion as you went up the wire code scale from, let's say, underground or buried all the way up to very high current.

In that context I was using the word irregular to mean that while in some of the data sets there was a suggestion of a dose-response, although I must say that in most of them there was not, but in some there was a suggestion of a dose-response, that even when there was such a suggestion the association was irregular, that is, it did not mount in a steady fashion as one moved from one category on up to the next.

Q. Are you referring, when you say mount in a steady fashion, are you referring to increases in exposure and increases in an associated factor?

A. I was referring to increase in the wire code category from what I refer to as category one, buried or underground, on up through the intermediate categories to

category five, VHCC or very high current configuration. And what I was trying to say there, I think, was characterized more technically by Dr. Rosenbaum himself when he used the word monotonic, that is, progressively upwards in this context. What I was saying was that this association was not monotonic, that is, it was irregular.

Q. So the Savitz data does or does not report a monotonic increase?

A. There is, to the best of my recollection, nowhere in the Savitz results, neither in the original contractors report, which I think has been referred to here today as a supplement, nor in the published paper, nowhere in any of those documents do I believe there is a reported monotonic dose-response relationship. And to the best of my recollection, despite many examinations of the dose-response question, there are only one or two non-monotonic or irregular such relationships.

Q. What effect does this irregularity, as you have used the term irregular, have on your interpretation of the Savitz study?

A. As I mentioned before, the concept of dose-response, sometimes referred to in epidemiology as internal consistency, that is, that the data are self-supporting and mutually reinforcing, is crucial to the interpretation of epidemiologic research because of

its non-experimental nature. The absence of dose-response is something that seriously undermines our willingness to interpret a data set as supporting a cause-effect relationship.

Q. How does that particular irregularity here that you referred to apply to the Savitz study?

A. The Savitz study, as I say, despite examining a number of possible dose-response relationships found only one or two and they are irregular and in fact take virtually all of their strength from the highest exposure category. So there is no consistency within the data.

Q. By consistency in the data, are you making a reference to the higher the --

A. The higher the wire code configuration, the higher the risk.

Q. Okay. And what are you telling us about that in terms of the Savitz study? The higher the wire code the what?

A. Well, firstly, lest this gets too confused, regularity, consistency and monotonicity are all synonyms for the very same idea. And what I'm saying is that in the Savitz data this attribute is absent.

Q. So as we go up to higher wire code --

A. We do not go up in risk.

Q. Now, in the field of epidemiology is there a

generally accepted level of statistical significance?

A. In epidemiologic studies, and I would say in science in general, you can use several different levels as the criterion of statistical significance. By all odds, the five percent level, or the 95 percent level as it is sometimes called, is by all odds the most commonly used. If there is another one that is in some frequent use it is the 99 percent level. This is an even more stringent test of statistical significance so anything that fails at the 95 percent level must necessarily fail at the 99 percent level.

Now, it is true that the 90 percent level is occasionally used. But in my experience I would say that is extremely rarely used, virtually never in study design, that is, when requesting funds for research one never proposes that the results will be tested at the 90 percent level. That would not be acceptable most of the time to most sponsors.

When the 90 percent level is used in presentation of results it is a mark of weakness in the data. It means that the investigator has had to revert to a lower level of meaningfulness in order to present his data. So presenting results at the 90 percent level of statistical significance is very rarely used, I would opine virtually never by an investigator except when he absolutely has

to.

Q. Now, in his testimony Dr. Rosenbaum made some comments about your analysis of the data in the Wertheimer, Savitz and Peters studies. Do you recall those comments?

A. Yes, I do.

Q. Just take a look at page two, lines 19 to 25, of Dr. Rosenbaum's testimony and just briefly review it.

(Witness perusing document.)

Q. Now, are you in complete agreement or disagreement with Dr. Rosenbaum on this point, his statement there?

A. I think that the statement that he makes here is at least from my perspective usefully divided into two major parts. The first one I understand to be this: it is not necessarily true that if we do more research of a true cause-effect relationship the strength of that cause-effect relationship will necessarily go up. I agree with that. I agree with the implication, which is that as research goes on and the estimate gets better and better it may go up or it may go down as long as it is focusing on the truth. So I don't believe there is any discrepancy between us there.

He then goes on to say that -- to imply that the fact that the associations in question, that is, between

childhood cancer and leukemia, get weaker and weaker, as we move from the Wertheimer to the Savitz to the Peters study, the mere fact that that association gets weaker is not to be taken as evidence that it will eventually disappear. Well, in a sense I agree with that too. It is not necessarily so. But in my experience it always has been so.

And the view that I have and I think which most epidemiologists have is that associations are commonly found which are not truly valid. They are created by the inadequacies of study design and that as refinement occurs the association gradually fades away. I don't know of any instance where we have seen an association get weaker with the passage of time in epidemiologic data and yet eventually come to be accepted as valid; whereas there are many examples of the reverse, that is, associations are supposedly found, they get weaker and then they fade away.

But I think the most important thing here and which we should not lose sight of is that the association in question as I was describing it is not between wire code and leukemia, it is between magnetic fields and leukemia. The wire codes are but a surrogate or marker for magnetic field.

And if we look at these three studies we see that

the Wertheimer study was the strongest one by far and it had absolutely no measurements of magnetic fields. The Savitz study was substantially weaker than Wertheimer in the wire code and also showed no association with such magnetic field measurements as were made. Granted, they were minimal. And then we come to the Peters study, which had as one of its major goals estimating the relationship between measured magnetic fields and disease. And here we have the weakest association of all, both in terms of wire code and none at all with regard to the measured magnetic fields.

So from my perspective we have at least three points over a period of 13 years and what we see is the wire code association gradually fading away and the association with the factor of actual interest, which is what everyone would have studied from the beginning if they could have, essentially non-existent.

Q. Now, could you turn to page two, line 27, through page three, line two, of Dr. Rosenbaum's testimony?

A. Yes.

Q. And were you in the hearing room earlier today to hear Dr. Rosenbaum's testimony about this statement and the context in which it was given and his clarification of what he said?

A. Yes, I was.

Q. Could you tell us what the 2.15 and the 1.73 numbers from the Peters study represent in terms of a comparison?

A. Certainly. Let's begin with the 2.15. This was a comparison of the relative risk of disease in what I have called category five, that is, VHCC, with what I would call categories one and two, which is buried and low current configuration wires. So this is a comparison of relative extremes, that is, of the highest to the lowest in the Peters study. And what it says is that the data appear to show that children who lived in the highest current configuration homes have about 2.1 or 2.2 times the risk of leukemia as compared to children who lived in the lowest two categories. That is the unadjusted.

The adjusted has exactly the same interpretation. It relates to the very same groups. It is just a more refined estimate that has gotten rid of sources of distortion in the estimate.

Given that we have both of them available to us, I would know of no reason to use the unadjusted.

Q. Now, is it acceptable practice in the field of epidemiology to group two categories like buried and very low current together when you are comparing extremes?

A. Oh, yes. It is a common thing to start from the bottom of the exposure scale and work up adding in one category or another as necessary to build up a sufficient number of observation in this group, which will be the crucial group. It is the referent category, the group to which all other risks are compared. So it is a very common thing to combine one or to combine two or even three categories so that the unexposed or referent group is going to be sufficiently large that it is a stable estimator of baseline risk.

Q. Now, can you tell us what the 1.54 number from the Savitz study represents?

A. Well, the Savitz estimate of 1.54 is not a comparison of extremes as is the Peters. It is a comparison of adjacent groups, that is, the bottom two groups to the upper three. So that it is a much less pure comparison. It compares groups that are much more alike than are the groups in the Peters study. So it necessarily represents an underestimate of any true association that would exist in the data.

Q. In the field of epidemiology is it appropriate to compare the odds ratios from the extremes in one study to the abutting groups in another study?

A. I really don't want to say that it is inappropriate or appropriate. I can only say that it

wouldn't be done very often in my experience. If you wanted to compare two studies where the data sets are sufficiently similar that they can be compared, exposure for exposure, it would be the more common thing in fact to compare like with like: apples with apples, oranges with oranges. If you want to compare the abutting groups, fine. But they should be compared from both studies. If you wanted to compare the extreme groups, fine. But that should be done for both studies.

I don't see that we can gain very much by taking the extremes from one study and comparing it to the abutting group comparison from another study. That comparison has to be tainted by the composition of the groups and where the dividing lines have been drawn.

Q. What data from the Savitz study were you referring to when you submitted your previous testimony regarding the pattern of data in these studies?

A. I felt that it was important because it was available and because it should show the association in its full strength to compare both studies with each other in terms of the extreme comparison that can be made. And for the Savitz study for leukemia that is 2.75 and for the Peters study that is 1.73.

Q. Now, could I direct your attention again to Dr. Rosenbaum's testimony on page four, lines 15 through

16. I'm referring specifically to the statement, "On balance one might call the associations found by the two studies (Savitz and Peters) fairly comparable." Do you see that?

A. I think they could be described as comparable or somewhat similar only in the sense that both are extremely weak by epidemiologic perceptions. But I at least am more struck by the discrepancy between them than by the similarity. Given that they are both weak, it is useful, I think, to investigate them closely.

Let's consider, for example, the 2.75 from Savitz versus the 1.73 from Peters. Now, I think what is important to do here is to subtract one from both of these measures. If we do that -- and I will explain why in just a second -- if we do that the Savitz estimate becomes not 2.75 but 1.75. That can be interpretted as saying that Savitz believed that he found that high current configuration multiplied the risk by 175 percent, a virtual tripling of the disease risk. An 175 percent increase.

But Peters says that he found only a 73 percent increase. So once we get rid of the baseline which is common to all of us and to all children, then we can see in the sharpness revealed against the background that Savitz was saying there was nearly a tripling and Peters

saying there was less than a doubling.

Now, in fact, if you compare those two measures of effect directly what you see is that the Peters estimate is less than half of the estimate of effect than was presented by Savitz. So rather than being similar, these two estimates differ by a factor greater than two. This would seriously diminish the ability to call them similar or comparable or mutually supporting.

Q. And in terms of can you describe in your own terms the direction of the data as we move from Wertheimer & Leeper to Savitz to Peters?

A. Yes. It is a little difficult to introduce Wertheimer into the comparison because she doesn't present dose-response and overall effects strictly for leukemia. However, based on my familiarity with the paper I would say that comparing the extremes in the Wertheimer, which is a four point scale, not a five point, she is completely in a different order of magnitude than the other two. Her estimate cannot be made with precision but it is almost certainly greater than five-fold. So from Wertheimer to Savitz we take a reduction from around five to around three, and then as we move from Savitz to Peters, we drop to under two. And in terms of effect measures, we go from four to two to less than one in terms of the relevant risk scale.

So from my perspective what we see here is a long term time trend, 13 years, three studies, at least two of which are very well respected, including by me, and we see this reduction virtually to the point now of the vanishing point in the Peters study.

Q. What does it mean when you say down to a relative risk of less than one? What is the significance of that?

A. If that is what I said I misspoke. I should that have said a measure of effect less than one. That would be a relative risk less than two. And in the Peters study it is a measure of effect of 73 percent or a relative risk or odds ratio of 1.73.

Q. Let me direct your attention to Dr. Rosenbaum's testimony on page two, lines 11 to 15. Would you agree that the obvious thing to say about the wire code data in these studies is that these studies observed an association between wire code and leukemia?

A. I think that is one thing that can be said. But I think that it is much more important to look into the data, to look into each of the individual studies and ask somewhat more detailed questions about what they actually say regarding this association between wire code and leukemia and more especially of magnetic field and leukemia.

What Savitz says, to my mind, with regard to wire code and leukemia is despite making innumerable comparisons I cannot find any consistent, that is, regular, monotonic statistically significant relationship between wire code and leukemia. And what he says with regard to magnetic field is despite the serious limitations of my magnetic field measurements, which I too acknowledge, I can find no relationship between magnetic field measurement and leukemia.

What Peters says is that despite my very refined study, much larger, much more focused in terms of its age group but overlapping largely with Savitz, despite the use of computerization to develop the wire code categorization, I find after reducing all of this data down once again a non-monotonic or inconsistent, that is, irregular relationship between wire code and disease risk, and it is weaker than that of Savitz; and I find now despite reasonably sophisticated magnetic field measurements, both spot and 24 hour for a substantial proportion of cases and controls, no relationship whatever between the magnetic field measurement and disease risk.

Q. Looking at the measured magnetic field data in the two studies taken together, what do you find?

A. Well, nothing at all. The Savitz study, again,

had rather limited measurements. It was not that easy back then to try to take those measurements. But he found essentially nothing.

And the Peters study, considerably more sophisticated in terms of measurement and larger proportion of subjects, also found nothing.

So what this tells me is that at least at the present time there is no evidence that magnetic fields, setting aside the question of wire code, there is no evidence that magnetic fields are associated with childhood leukemia.

MR. WATSON: Thank you.

I pass the witness, Your Honor.

MS. McCLOSKEY: Could I have just a minute?

JUDGE SMOLEN: Yes.

(Pause.)

JUDGE SMOLEN: Back on the record.

CROSS-EXAMINATION

BY MS. McCLOSKEY:

Q. Good afternoon, Dr. Cole.

A. Good afternoon, Ma'am.

Q. The first question, I think when you were concluding your discussion concerning Dr. Janes' testimony, if I heard you correctly, you stated that there was no research that you knew of that supports the

relationship between carcinogenesis and endocrinology, or in the endocrinology field?

A. I hope I didn't say that. Far from it. The links between hormones and cancer in both animals and humans is very well established. What I was trying to say was that relating to the hormone melatonin.

Q. And I believe you stated in your direct testimony that you are not an endocrinologist, is that correct?

A. I can't recall whether I stated it or not, but I'm happy to state at the present time that I'm not an endocrinologist.

Q. So your statement concerning melatonin and its relationship to cancer is based on your experience as an epidemiologist, is that correct?

A. It is based on my experience as an epidemiologist with particular interest in the hormonal basis of breast cancer. Several of my studies have included rather detailed hormonal measurements in relevance to this disease.

Q. Now, I believe you were asked a question by Mr. Watson concerning how long research has been going on into breast cancer and various hormonal causation of breast cancer. And you had stated it began probably in the mid-1920s. Do you recall that question and your

answer?

A. My recollection of the question was when did research into the causes of breast cancer begin. I don't recall that the question specified the nature of the targets.

And I said in response that we generally consider that the modern study of breast cancer began in the 1920s. Undoubtedly there was work prior to that time, but I know it only as case reports.

And I further said that in the 1920s, when there is interest in breast cancer and causation started, there were two major targets. One was a virus and the other one was hormonal factors.

Q. And in that research how much of that research has been done on the question of breast cancer in men?

A. The studies of breast cancer in men are relatively few. The studies of breast cancer in women, it has been contended that there are more studies that have been done on breast cancer in women than on any other form of cancer with the exception of lung cancer. The studies of men, I can't give you a number, but I would acknowledge that it is relatively few.

Q. And, Dr. Cole, how long have studies been going on into the relationship between electromagnetic fields and breast cancer in men?

A. The question specifically asks how long has research been going on into the relationship between electromagnetic fields and breast cancer in men? Ms. McCloskey, I don't mean to try to be excessively clever here. I have to tell you what I believe. I don't believe it has begun yet.

Q. So, for example, you don't consider the Demers study to be a study concerning electromagnetic fields and breast cancer in men.

A. That's correct, I do not. I consider it a study of occupational titles which have a ephemeral relationship to electromagnetic field exposure.

Q. And how long have occupational studies or other types of epidemiological studies been going on into occupations which may have an exposure to electromagnetic fields and breast cancer in men?

A. If I had to choose a time I would probably say about 1976 with the start of studies that have not yet come to full publication. They have been published as a letter but not full publication.

Q. And have there been any comparable studies as to breast cancer in women?

A. Dr. Wertheimer and her colleague, Leeper, did a study somewhat similar to their childhood cancer study which pertained to cancer in adults. And in that study

it was contended that there was an association with cancer in adults collectively. Now, that would have to include breast cancer in women because at the time the study was done and still today breast cancer is the most common form of cancer in women. So to that extent I guess you could say that the Wertheimer study pertains to this issue, even though the specific issue is not -- cannot be isolated in the data to my recollection.

Q. But that is the only study that you are aware of that relates to breast cancer in women?

A. Well, there are other studies that used a collective cancer end point. For example, the McDowell study related to all forms of cancer mortality so we imagine that breast cancer was in it. But I don't know of any studies that have specifically isolated this other than the Demers study. And as I say, that is deficient on the other end of the cause-effect relationship.

Q. Now, I believe you stated in reference to the Demers study that it was funded and you were inferring that it was funded as a training grant?

A. No, no. Not at all. The Demers study is not funded as a training grant. That is made clear from the footnote. I thought I was being asked about the specific support for Dr. Demers, who at the time I assume was Mr. Demers since it says he was supported by a training

grant. But I don't know that for a fact.

Q. And are you familiar with -- you don't have the study in front of you but I can hand it to you -- with David Thomas, who is the second author on that study?

A. Yes, I know David Thomas.

Q. Can you briefly tell us who David Thomas is?

A. Sure. David Thomas is professor of epidemiology at the University of Washington at Seattle.

Q. And when you were discussing some of the limitations of the Demers study I believe you noted that you found it problematic because the results reported did not jibe with the general perception of carcinogenesis and it does not support the promotion issue or cancer as a promoter? Do you recall that?

A. I think that is a fair representation of what I said, yes.

Q. And is it fair to say that those comments are limited to the findings in the Demers study which refer to breast cancer in men and not to all cancers?

A. Oh, any comment about the Demers study would have to be restricted to male breast cancer. That is all that the study relates to.

Q. Now, in response to Dr. Rosenbaum's testimony you discussed the levels of statistical significance, that researchers use a 95 percent confidence interval and

that is the most commonly used confidence interval?

A. In my judgement it is. I can't say that I've tallied this up but that would be my judgement, yes.

Q. And is it your opinion that there is a significant difference between something that is statistically significant at the 95 percent level versus something that might be statistically significant at the 93 percent level?

A. Ms. McCloskey, firstly I would have to say that I have never seen the 93 percent level used and I shall take it to be an example of whether or not one would attach great differences there.

Q. Yes.

A. As a foundation to my answer I would have to say that I think in modern epidemiologic analysis of data the role of significance testing is perceived as relatively minor. After all, it does not actually address the main issue, which is one of causality. It simply deals with a peripheral issue, which is whether or not chance may be held accountable for the result.

All of that have said, that is, that I hold statistical significance to be a relatively weak approach in the analysis of data, I would say that the distinction between whether something is statistically significant at the 93 or the 95 or the 90 is one that I would consider

pretty minor.

Q. Now, you have also stated several times that the dose-response is important in interpreting or reviewing an epidemiological study. Could you just for the record define for us what you mean by a dose as it relates to electromagnetic fields?

A. As you know, in the area of the epidemiology of health effects of magnetic fields, we have been plagued by our inability to actually describe dose or exposure. That is why we have used such surrogates as wire code and job title. Very, very few studies, maybe none yet, actually have a good estimate of dose or exposure in this area.

But what I would mean by it would be some measure that would describe in some reasonable summary fashion the total magnetic field exposure that an individual human being has had up through the time when he developed a cancer or, for a comparable subject, failed to develop cancer.

I might mention that as of now we don't have any epidemiologic studies in which electromagnetic field measurements have actually been made of the individual people in the studies.

MS. McCLOSKEY: I think that's all the questions I have, Your Honor.

JUDGE SMOLEN: Mr. Sugarman.

CROSS-EXAMINATION

BY MR. SUGARMAN:

Q. I was intrigued by your last answer, doctor, as to your interest in knowing how much exposure a person had had at the time of diagnosis.

A. Up to the time of diagnosis.

Q. Up to the time of diagnosis. What do you mean by how much exposure?

A. In each area of epidemiology that is reached a causal factor or suspect or potential causal factor, we try to develop some index that would allow us to characterize individuals as to how much they have been exposed.

Q. What do you mean by how much exposure?

A. I'm trying to get there.

Q. Okay.

A. In most areas I think it would be fair to say that the description of exposure comes down to trying to get an idea of intense was the exposure. In this context it would how high was the magnetic field and how long, that is, the duration of exposure, how many years on the job, whatever. And then by combining the two into some intensity times duration measure coming up with what we may refer to as a cumulative estimate of exposure. The

present body of knowledge has not arrived at that stage.

Q. Do you have an opinion about whether what you are calling a cumulative exposure as a product of two variables, intensity and duration, is more important than the degree of variation in exposure? That is, the number of times that it is turned on and off so to speak. Do you have an opinion on that?

A. Yes, I do.

Q. And what is that?

A. My opinion is this: that at the present time the available data would not substantiate one or the other or other alternatives, for example, peak exposure, as likely to be relevant or meaningful. However, we have a vast body of knowledge regarding how carcinogens act both in animals and in human beings and I know of no exception to the following: that risk is a function of cumulative exposure, with some very small exceptions implying plateauing of risk.

Q. But these exceptions --

A. Let me finish, if I may.

Q. Go ahead.

A. And this information that I have just said is true for chemical, for physical and for viral carcinogenesis. So it would be pretty bizarre, I think, if magnetic fields were to turn out to be a carcinogen,

which I think they won't, to think that it's going to be an exception to this near universal rule.

Q. Is not one of the exceptions to that near universal rule the role of initiators or promoters?

A. No.

Q. Isn't it true that once cancer is initiated the initiation need not be repeated?

A. That's correct.

Q. That the body will develop itself -- develop the disease without further initiation?

A. Well, I think you have asked two questions there. The answer is once initiation has occurred further initiation -- we're not sure what that is -- would not be necessary.

But my point is that the probability of initiation is a function of the degree of cumulative exposure to the initiator. This is especially true when you are speaking about numbers of animals and numbers of people as opposed to an individual.

Q. What study shows that cumulative initiation is the critical -- the cumulativeness of the initiator is critical?

A. Firstly, my specialty is epidemiology, not animal research.

Q. No, but I asked you to authenticate your own

answer where you said that the cumulative initiation is the relevant test. And I'm asking you give me a study that says that the cumulative amount of initiation is the relevant test of the likelihood of developing the diagnosis.

A. I believe you're asking me two questions. I would like to try to answer both of them, if I may.

The first one relates to human beings. This distinction between initiation and promotion is not something that we can draw in human studies. We accept the concept as an useful model for thinking about it, although now with some refinement.

With regard to animal studies, it is my understanding that initiation occurs in a dose-response fashion, but I'm not able to cite specific studies that would demonstrates that.

Q. All right. Now, with respect to humans, in your previous work haven't you acknowledged that initiators are a relevant factor in examining the development or the effect of EM/F in producing cancer?

MR. WATSON: Your Honor, could we have a citation to what Counsel is referring to?

MR. SUGARMAN: I have a right to ask him a question.

JUDGE SMOLEN: I'm going to overrule. He asked him

whether or not he so testified.

MR. WATSON: He said previous statement. He is required to identify the prior statement, isn't he? It's not guesswork.

JUDGE SMOLEN: I overruled. To me the question is did you so testify to your recollection.

THE WITNESS: I don't recall that I did or did not.

BY MR. SUGARMAN:

Q. Have you previously taken a position that initiators are a significant subset of potential causes of cancer, of potential EM/F causation of cancer?

A. Mr. Sugarman, I'm sorry. I really don't know what the answer to this question is.

Q. Now I will show you your prior testimony. Do you remember testifying in September, 1989, at the hearings on the Jersey Central proposed powerline on behalf of Jersey Central?

A. Yes, I do.

Q. And do you remember testifying at that time, "On the other hand, if EM/F were to be a growth enhancer or promoter but not an initiator the total risk of leukemia would remain about the same but some epidemiologic features of the disease would change. For example, we would expect EM/F exposed cases of leukemia, especially among children, to be younger than the EM/F

non-exposed cases."

MR. WATSON: Your Honor, can we have Counsel show the witness the quote so he can see the context?

JUDGE SMOLEN: Yes, I think that is appropriate.

BY MR. SUGARMAN:

Q. Do you remember giving that testimony? And you may look at the document before you answer as Counsel has requested. The bottom of page 11 of your prepared testimony.

(Witness perusing document.)

JUDGE SMOLEN: For the record, you mean his prepared testimony in another proceeding?

MR. SUGARMAN: That's right.

A. Mr. Sugarman, if the question is do I recognize this as in fact my prior testimony, the answer is yes, I do.

BY MR. SUGARMAN:

Q. So then you recognize that you have previously testified and stated that initiators are a separate subset in terms of analyzing the potential for EM/F to cause cancer?

A. I'm sorry. I don't understand the question. Could you repeat it for me?

Q. I'll repeat it. You agree with your own statement on page 11 that your own statement has implicit

in it that initiators are a separate subset and must be analyzed separately to evaluate the effect of EM/F in causing cancer in humans, that you have previously stated that opinion?

A. Mr. Sugarman, the concept of an initiator, a promoter, complete carcinogen and a growth enhancer has never been shown to exist in human beings. This is a set of constructs that we use to try to understand why it would be true that carcinogens would operate at different times in a person's life relative to the time of diagnosis.

Now, if the question is do I think it would be important to know whether EM/F is an initiator or promoter or complete carcinogen, the answer is only if one would accept it as a carcinogen, which I do not, or if one is attempting to propose the model whereby such activity would be demonstrated. It has been my understanding that those people who are proponents of the idea that magnetic fields cause cancer have generally urged the notion that it is a late stage carcinogen. Whether you want --

Q. You are off the subject, doctor. You are off the subject. We're talking about --

MR. WATSON: Your Honor.

MR. SUGARMAN: Wait a minute.

JUDGE SMOLEN: Stop. One voice at a time.

BY MR. SUGARMAN:

Q. We are talking about your statement and your definition of how much exposure a person would have and your contention that cumulative exposure is the preferred way to go. And I asked you whether episodic exposure or incidence of peaks might not be more relevant specifically with respect to initiators. It has nothing to do with age.

JUDGE SMOLEN: Well, I'm not sure that you did ask that question before.

But did you understand his last question posed by Mr. Sugarman.

THE WITNESS: Well, I'm sorry. I've lost my train of thought now. It was from my perspective rather complicated.

MR. SUGARMAN: It is rather complicated.

THE WITNESS: And I'm not sure I can reconnect my stream of thought. I was only -- now I do remember.

I was trying to point out that prior to the Demers study those people who have felt deep concern about the relationship between magnetic fields and cancer have suggested that it was a late stage carcinogen.

MR. SUGARMAN: Your Honor, that has nothing to do with my question. I object and I ask that the witness be

instructed to answer my question. He is making a speech that has nothing to do with my question.

JUDGE SMOLEN: I'm going to allow the witness to finish his answer and if it doesn't complete the answer to your question he's going to --

MR. SUGARMAN: It's clear, Your Honor, he is off on a different subject.

JUDGE SMOLEN: Go ahead. Finish your answer.

THE WITNESS: The conclusion of the answer is that the Demers study, if it is to be taken as literally valid, which I do not, has to be interpretted as supporting an initiator role. This is inconsistent with the role that has been attributed to magnetic fields by proponents of the association in the past.

JUDGE SMOLEN: Now, if the witness has not answered the question --

MR. SUGARMAN: Now I want to go back to my question, Your Honor.

JUDGE SMOLEN: Ask it, if you will, in simple stages rather than multiple questions.

MR. SUGARMAN: I was going in simple stages, Your Honor, but it takes a while to get through to the end because each stage has to be tested out.

JUDGE SMOLEN: All right.

MR. SUGARMAN: He doesn't respond directly.

MR. WATSON: I object to that, Your Honor.

MR. SUGARMAN: I'm sure you do.

JUDGE SMOLEN: Your objection is noted. Let's go to the next question.

BY MR. SUGARMAN:

Q. The area of questioning was what you mean by exposure. And you said you liked the concept of taking intensity times length or longevity of exposure. And I asked you why. And I asked you whether that would apply to initiators. And you said that initiators were not a separate subset for humans, they had never been shown to be a separate subset for humans. And I asked you whether you hadn't previously testified on the basis that initiators are a separate subset for humans.

JUDGE SMOLEN: All right, that's a question.

MR. WATSON: I object. That's not a question. That's argument. There's no question there.

MR. SUGARMAN: That's the question.

JUDGE SMOLEN: Wait a minute, now. Let me speak. As I understand the question, it is did you previously testify -- now finish it.

BY MR. SUGARMAN:

Q. That initiators are a separate subset for humans.

JUDGE SMOLEN: That's all. That is the question.

A. No.

BY MR. SUGARMAN:

Q. Can you explain why on page 11 of your prior testimony, then, you stated, if I may take it back from you again, that if EM/F were to be a gross enhancer or promoter but not an initiator the total risk of leukemia would remain about the same but some epidemiological features of the disease would change. For example, we would expect EM/F exposed cases of leukemia, especially among children, to be younger than the EM/F non-exposed cases."

Why were you talking about the difference between enhancers or promoters and initiators if they weren't separate subsets?

A. I think I can now make this clear if you will give me this. It is from the context. I will read the sentence, if I may.

"It would be of value to determine whether it is proposed" -- not my me, but by the advocates of this position -- "it would be of value to determine whether it is proposed that EM/F is an initiator, a promoter or a growth modifier."

That's all. I'm saying to those people who would propose there is such a relationship let them be specific and let's see how the data comply with their

specification.

Q. Very good, doctor. Except that on page ten you start out stating, "There are four concepts which have been referred to commonly in discussions regarding a proposed cause-effect relationship between EM/F and cancer. I review these," meaning the four concepts, "because the words that describe them, especially the word promotion, are occasionally misused. Ordinarily this does not pose a problem, but I believe it has posed a difficulty in the present circumstances. Below I will explain the problem as I see it."

"I offer the following definitions and will adhere to them in this report: transformation," and you define transformation. "Initiation," and you define that as, "The earlier part of the process of cell transformation and is considered to represent a series of irreversible changes in a cell." Then you define promotion and then you define growth modification.

Now, how did you define all those terms and why do you refer to the four categories that are commonly regarded if you don't recognize that there are separate categories?

MR. WATSON: Objection, Your Honor. Compound question.

JUDGE SMOLEN: Yes, I agree. It's a compound

question, but we have a very sophisticated witness and it's all related together.

Does the witness understand the question?

THE WITNESS: I think so.

JUDGE SMOLEN: Then try to answer it.

A. My answer is this: in laying out this model of carcinogenesis I was trying to illustrate what I referred to as the four concepts, which have herestic value -- probably don't have any theoretical value anymore, but have herestic value -- in requiring of a data set that it comply with these consents.

Now, if you were to ask me specifically do I believe that initiation exists as such my answer is no.

BY MR. SUGARMAN:

Q. And you don't see that as inconsistent with your testimony as I quoted it back to you?

A. I do not see it as inconsistent.

Q. The sentence here that you spoke in 1989 was, "There are four consents which have been referred to commonly in discussions regarding a proposed cause-effect relationship between EM/F and cancer." You say four concepts which have been referred to commonly.

Is there anywhere in here on page ten where you say those four concepts or the concept of those four concepts is invalid? Do you say that anywhere in here?

A. Probably not.

Q. Now, aren't there other initiators of cancer in humans such as -- I shouldn't say other. It presupposes that you agree with me on EM/F. Haven't genuine causes of human cancer been identified as attributable to an initiator from which the cancer evolves even if the initiator is terminated?

A. I know of no substance and I know of no physical force which has been demonstrated to be solely an initiator in human carcinogenesis.

Q. I didn't say solely an initiator. That's not my question. I said aren't there other substances which have been demonstrated to cause cancer even though the use of that substance or the exposure to that substance is terminated prior to the diagnosis?

A. The fact that a cancer would occur subsequent to termination of exposure would in no way be helpful in allowing us to distinquish that exposure as an initiator or a promoter or a growth enhancer.

Q. Thank you. But aren't there other substances, such as smoking tobacco, exposure or inhalation of asbestos, which have been shown to be initiators? Not solely initiators but initiators.

A. The three things that you mentioned are recognized as complete carcinogens. Yes, they have

initiating activity. But they do not have or are not thought to have solely initiating activity. That is, they function as complete carcinogens.

Q. Now, in terms of measuring the exposure and the ability to measure exposure in a dose-response, in order to evaluate the dose-response or estimate the dose-response relationship, isn't it a potential that those initiators -- let's not talk about EM/F, let's talk about tobacco and asbestos -- that those initiators or those substances -- let me drop the word initiators -- that those substances can -- that the relative likelihood of those substances leading to cancer can be a factor of the intensity of the dose and not the longevity?

MR. WATSON: Objection, Your Honor. Beyond the scope of examination. He has made it clear he is not talking about EM/F. In fact, it was excluded from the question.

MR. SUGARMAN: The same as the witness did, I'm using the relationship of other substances to cancer as a basis for designing an epidemiological study.

JUDGE SMOLEN: I know, but you specifically referred to tobacco and asbestos.

MR. SUGARMAN: Right.

JUDGE SMOLEN: I'm going to sustain it with respect to tobacco and asbestos. Perhaps you can rephrase your

question --

MR. SUGARMAN: Sure.

JUDGE SMOLEN: -- without reference to those items.

MR. SUGARMAN: Sure.

BY MR. SUGARMAN:

Q. What reason is there to exclude, if any, the potential for the intensity of a short-term exposure as being a relevant variable in determining the initiating ability of various substances that cause cancer?

A. None.

Q. What about the trigger -- I will call it a trigger or a multi-trigger -- exposure where the relevant dose might be the number of times that the substance is exposed to the subject, and the rapidity of those exposures as triggering and initiating cancer? What reason is there to exclude that?

A. None.

Q. In your study -- I think you testified the last time you were here that you are initiating a study, an epidemiological study relating EM/F to humans. Is that right?

A. I hope that what I said was that I'm initiating a study which will include that as one factor. It's not the primary focus of the study.

Q. I think you did say that.

What dose-response relationship are you going to measure?

A. We are going to use duration and a surrogate of intensity.

Q. What is the surrogate of intensity?

A. That hasn't been decided yet.

Q. Is it going to be wire codes?

A. Oh, no. It's an occupational study.

Q. And you are going to use a surrogate for intensity. What industry are you going to analyze?

A. This is electronics manufacturing.

Q. Why haven't you decided what surrogate to use?

MR. WATSON: Your Honor, this is way beyond the scope.

JUDGE SMOLEN: It's is far afield now. It doesn't matter. Sustained. Next question.

BY MR. SUGARMAN:

Q. Now, have you expressed any opinion as to whether or not your study is an exercise in futility?

MR. WATSON: Objection, Your Honor.

JUDGE SMOLEN: Sustained.

MR. SUGARMAN: It goes to the question of credibility of the witness. Here he's testifying there's no reason --

JUDGE SMOLEN: I sustained it. Next question. Try

it again.

MR. SUGARMAN: I'll ask it a different way.

BY MR. SUGARMAN:

Q. If you are satisfied that EM/F does not cause disease how can you in good conscience propose a study?

A. As I mentioned to you -- well, let me make two responses to this question if I may. The more important one and the practical one is that as I mentioned a moment ago the study is not focused on electromagnetic fields. It is actually primarily concerned with other aspects of the exposure and work history of these people.

And although I feel reasonably strongly that electromagnetic fields have not been and are not likely to be shown to be carcinogenic in human beings, I don't proclaim that it is true absolutely beyond the ken of research.

Q. I appreciate that answer. Thank you.

Now, you described the Demers study as performed by a student, as you indicated?

A. I don't believe I indicated that, no.

Q. Maybe I misunderstood your testimony. You stated that he was supported by -- that the individual was supported by a training grant?

A. The footnote to the paper indicates that Dr. Demers was supported by a training grant at the time

the research was done. All I was doing was in effect reading the footnote out of my mind.

Q. Is it of my relevance to you?

A. It is of some relevance. I would not say it is of great relevance but it is of some relevance that this was probably the first study that this person did.

Q. But this person was one of about 15 or 20 people who did the study, right?

A. That is probably not right in the most literal sense. I would infer from the nature of those people's affiliation that they didn't actually do the study but contributed data to the study, that is, made resource available.

Q. Contributed data to the study? What about Dr. Thomas?

A. I don't know what the role of the individual people is. I would assume that Dr. Thomas was fairly heavily involved in the study, but that is a supposition on my part.

Q. Was this study available to you at the time of your prior testimony in October? I believe it was October.

A. Mr. Sugarman, I'm sorry. I just don't remember whether I had it at that time or got it shortly thereafter or just what.

Q. Is the American Journal of Epidemiology peer reviewed?

A. Yes, it is.

Q. Do you know whether this study was peer reviewed?

A. I do not.

Q. Are you a reviewer for that journal?

A. Yes.

Q. I take it, then, you didn't participate in a review of this study?

A. I did not.

Q. Did you have any opportunity to hear a presentation of this study?

A. No, I did not.

Q. Now, you indicated that a title was used, which title is a term for the occupational category of the subjects in lieu of exposure. Did I understand you correctly?

A. I only tried to recapture my understanding of what is specified in the paper, that people were classified according to the job titles that they provided.

Q. Right. And you used the word title to describe that. Are you going to use titles in your study?

MR. WATSON: Objection, Your Honor.

JUDGE SMOLEN: Sustained.

BY MR. SUGARMAN:

Q. Have you done any studies using title as the basis for the dose?

A. Yes, I have.

Q. Doesn't epidemiology in general depend on, at least as related to case control studies, depend on comparing a range of subjects who have exposure with a control group that is intended to have no or less exposure but also different amounts? In other words, isn't it inherent in the control group versus the exposed group that is there is variation of exposure within each group in a case control study?

A. Mr. Sugarman, perhaps there was a mixture in your question of two different kinds of studies. In a case control study the comparison is of cases with controls.

Q. Right. Isn't there inherently a difference between the exposed group and the -- I'm sorry -- yes, the control and the exposed group?

A. No, sir, there is not.

Q. Well, what is the -- I'm sorry. Okay. You're talking about they are all exposed in a case control group, is that right?

A. No, sir.

Q. All right. Would you explain to me, then?

A. A case control study is a comparison of two groups of people, the cases who have the disease and the controls who do not have the disease. The object of the study is to determine the exposure profile for each of these two groups. In an ordinary case control study both groups would have people who are exposed and who are not exposed. So the study begins by a comparison of the relative frequency of exposure in the two groups.

Q. Thank you for the clarification.

What my question was is isn't it true that exposure is inherently variable in those who have been occupationally exposed and those who haven't? That there is no consistent level of exposure within the occupation or within the residence or whatever it is.

JUDGE SMOLEN: You have about three or four questions there, but I think the witness understand it. If the ALJ understands it, I'm sure the witness understands it.

A. I understand the question to be isn't it true in a case control study as well as in other kinds that even among exposed people there may be gradations of exposure ranging all the way from the non-exposed up to the very highly exposed. If that is the question --

BY MR. SUGARMAN:

Q. The question is not whether there may be but whether there inherently is given the complexity of life.

A. No, it's not inherent. There certainly are studies in which one series or the other, the cases or the controls, have been uniformly exposed or uniformly not exposed. Now, admittedly that is quite rare but it does occur.

Q. Do you know of any study design dealing with EM/F and human health that has -- any study design whether implemented or not, or one that you could think of, that would eliminate the variation in exposures?

MR. WATSON: Your Honor, I'm going to object to this. This is way beyond the scope of the direct examination.

MR. SUGARMAN: He testified about the dose-response relationship and about the dose --

JUDGE SMOLEN: The court reporter can take only one voice at a time, despite the fact that he has two hands.

Now, let's start all over again. We have a question and objection. In order to clear this record I'm going to sustain the objection and let you start all over again. Let's see if we can get one question which is relevant.

BY MR. SUGARMAN:

Q. In your testimony about criticizing the Demers

study because of the variation in dose, isn't it true that there is no way that an EM/F versus a health effect study could be designed which would eliminate the variation in dose?

A. Mr. Sugarman, I believe this question begins with a premise that is false. It says in effect isn't my criticism of the Demers study based on the idea that there is variation in dose.

Q. That is one of the criticisms.

A. Quite differently from that, my criticism of the Demers study is I don't know whether there is variation in dose or not, that I cannot deduce a perception of exposure as different even from one group to another much less from one individual to another from the information provided in the study.

Q. What do you mean by within a group -- I'm sorry. From one group to the other? Sorry.

A. As I recall in the Demers study, there are six groups of subjects classified on the basis of occupational title. I forget the exact labels that are put on these groups, but there is a representation on the part of the authors themselves that they do not know to what extent magnetic field exposure would have differed from one group to the other, with the exception of one group which I think they represent as probably highly

exposed.

Q. What does the sentence in the report mean, the following sentence: "The results for all jobs involving electromagnetic field exposure and for each of the five exposure groups relative to the jobs without exposure are presented in Table 2"?

A. Well, I take it at face value. I guess it means that if we look at Table 2 we will see the odds ratios in the various five or six groups.

Q. And so doesn't the data present a separate group which is not exposed versus the five or six groups which are exposed?

A. Indeed. But the paper makes it clear that among the five or so ostensibly exposed there is no way of knowing to what extent exposure was actually experienced, and this is particularly true among individuals in the categories.

By the way, I might add further, that the group that is called non-exposed is not necessarily non-exposed.

Q. And isn't that going to be true of every EM/F study?

A. Virtually certainly, yes.

Q. You indicated in your testimony in the New Jersey case that, "There is a clear tendency for positive

findings in the case control studies which is highly unlikely to be due to chance. Also, it is difficult to imagine how bias or confounding" -- and by bias or confounding you are talking about technical problems with the study, is that right?

A. Yes.

Q. "...could explain the consistent positive findings. However, one possible explanation of these positive findings is a propensity of investigators to report only positive rather than negative findings pertaining to EM/F and cancer."

Is that still your opinion?

A. Yes, sir.

Q. Is that your opinion of the Demers study?

A. I don't think one can hold an opinion such as that with regard to any one study. It is a question of whether or not there is a phenomena in favor of publication of positive studies. For all I know, the Demers study would have been published in it's present form even if it were negative. But whether it actually would have been or not, I don't know. After all, Demers examined numerous variables and we have not seen any publications with regard to any of the other variables as negative, at least not yet.

Q. You testified that there is no evidence in your

opinion to tie magnetic fields to -- no epidemiological evidence to tie magnetic fields to cancer. Was that your testimony?

A. I thought the testimony today related to melatonin.

Q. Oh, just melatonin?

A. Well, maybe -- I do not recall specifically making the statement that you have just suggested I might have made.

Q. It may have. It was your last answer and it was coming at me fast. I may not have gotten the word down.

Do you intend to testify that there is no evidence relating electromagnetic fields and cancer?

MR. WATSON: Your Honor, I'm going to object to this in terms of the use of the word evidence. We are in a courtroom. The witness is not a lawyer.

MR. SUGARMAN: I meant evidence in a scientific sense.

JUDGE SMOLEN: Now it is qualified.

MR. WATSON: On that basis I withdraw my objection.

JUDGE SMOLEN: All right.

A. My testimony is that there is a large amount of evidence now pertinent to the question. There is some on one side, there is some on the other. Thus for me the

question becomes how does one integrate this information and try to come to some statement as to what it shows today and what reality is likely to be. And from that perspective my own view is that the available evidence is just about equally divided between that which is supportive and that which is contradictory. And my experience leads me to say that that is a pattern for associations that end up fading away.

MR. SUGARMAN: Thank you.

I don't have any further questions.

JUDGE SMOLEN: Ms. McCloskey.

MS. McCLOSKEY: Your Honor, I just have one question on recross to clarify a point.

RECROSS-EXAMINATION

BY MS. McCLOSKEY:

Q. I believe in the cross-examination you at one point stated that risk is a function of cumulative exposure, and I believe you were referring to the risk of cancer and said that was true for chemicals, the physical and viral causes of cancer that we are aware of, and EM/Fs would then have to be an exception to this near universal rule. I would like to focus on the near universal rule.

What are the exceptions to the rule that the risk of cancer is a function of cumulative exposure?

A. One exception is ionizing radiation delivered to localized areas of the body where at high dose there may be a killing effect rather than a transforming effect. So that risk rises as a function of dose to a certain point and then plateaus and may even fall off.

MS. McCLOSKEY: Thank you.

That is the only clarification that I needed.

JUDGE SMOLEN: Mr. Watson, anything further?

MR. WATSON: Nothing, Your Honor.

JUDGE SMOLEN: Then the witness is excused. Thank you very much for appearing and testifying.

(Witness excused.)

JUDGE SMOLEN: Anything further?

MR. WATSON: I think we need to talk about a briefing schedule, Your Honor.

JUDGE SMOLEN: Yes, I know. But as far as evidentiary testimony.

MR. WATSON: No, Your Honor.

JUDGE SMOLEN: Then let's go off the record. We're going to talk about a briefing schedule.

MR. SUGARMAN: Before you do, Your Honor, may I make a motion on the record?

JUDGE SMOLEN: Back on the record.

MR. SUGARMAN: I would like to move that Your Honor determine, as I believe the Commission has authorized you

to do, that an effect has been shown so that we can go onto the second stage of the hearings. I don't think any briefs are necessary to conclude, as Your Honor has stated in the prehearing order, that there would be a first stage to this hearing and then a decision would be made about the issue of need and so on and so forth.

JUDGE SMOLEN: What is your motion?

MR. SUGARMAN: The motion is to make the determination orally.

JUDGE SMOLEN: On all this evidence in this record?

MR. SUGARMAN: I'm sorry?

JUDGE SMOLEN: On all the evidence in this record.

MR. SUGARMAN: Yes.

JUDGE SMOLEN: The motion is denied.

MR. SUGARMAN: I was only halfway through my motion.

What I was trying to say, Your Honor, was that there is a concern or has been a concerned expressed as to timing here. And it seems to me that in just the last sentence out of the last witness' testimony was that there is substantial evidence -- he said equally balanced -- that there is a relationship between EM/F and cancer and not. So why can't we have the second stage of the hearings?

JUDGE SMOLEN: Because I have denied your motion

and I want to receive briefs on the first stage of the hearing and make a determination there and let the Commission make whatever further determination it has to make.

So let's go off the record.

(Discussion off the record.)

JUDGE SMOLEN: Back on the record.

At an off the record discussion we have arrived at a briefing schedule which is as follows: initial briefs in hand March 17, 1992, all parties to file contemporaneously; reply briefs in hand April 7, 1992, the same, all parties to file contemporaneously.

With that, I assume everyone has closed their cases at least as to this portion of the case. We still have to receive as late-filed exhibits PECo Cross-Examination Exhibit 5(a) and 5(b). And if any other parties have any exhibits which have been identified and admitted but not distributed you are urged to do so, otherwise we can't consider them if the issues contained therein are raised on the briefs.

MR. WATSON: Can we have a date set so we know when we've got the complete package for that?

JUDGE SMOLEN: Well, I don't know if there are any outstanding.

MR. WATSON: I don't know if there are any either.

I'm just saying --

JUDGE SMOLEN: Do you have any?

MS. McCLOSKEY: I don't think we do.

JUDGE SMOLEN: I don't think OCA does. And I don't think the company does except for this these last two. I don't know if Mr. Sugarman has.

MR. SUGARMAN: I don't know of any.

JUDGE SMOLEN: Well, every party has to do his own work and his own research. If it's there, it's there to consider. If it's not there, then it's --

MR. WATSON: I was just going to suggest something so we all focus on it earlier rather than later to make sure it's in, such as a week from today.

JUDGE SMOLEN: All right. Let's put that on the record.

MR. SUGARMAN: Do you know of something of mine that's not in?

MR. WATSON: No, I don't, actually. But I don't want to start working on the thing and then somebody says, oh, gee, I forgot the deadline and it comes sailing in at the end.

JUDGE SMOLEN: I think it's an appropriate request. Let me put on the record that all exhibits which have been marked and admitted, if they have not been distributed to other Counsel must be done so within by

February 14, 1992.

MR. SUGARMAN: Well, Your Honor, the problem is my clients have not been able to afford to purchase the transcript of the later hearings yet and they are having to generate the funds to do that. So until that is done I may not have access to transcripts to be able to ascertain that.

JUDGE SMOLEN: Let me make this suggestion, and Ms. McCloskey or any other Counsel can cooperate. Generally in the front of the transcripts there is a page showing exhibits marked for identification and then admitted. So it is really a question of just checking a few of the transcripts. Even if it is by telephone call, perhaps Ms. McCloskey will accommodate you and check those transcript either with you or via telephone to see if there are any which have been identified or admitted.

MS. McCLOSKEY: We can do that, Your Honor.

JUDGE SMOLEN: I mean with respect to Mr. Sugarman.

MS. McCLOSKEY: With respect to Mr. Sugarman's, yes.

MR. SUGARMAN: Thank you.

JUDGE SMOLEN: So the date still stands, February 14, for late-filed exhibits.

Having said that --

MR. BONNEY: Your Honor, if you were about to

finish, one thing you noted about the size of the record made me think it might be helpful as just a reminder to instruct us with respect to the briefs that --

JUDGE SMOLEN: You mean the size of the brief? The number of pages?

MR. BONNEY: Well, I wasn't thinking about that as much as the transcript and other testimony references in the brief be made so that --

JUDGE SMOLEN: Well, I'm going to send out a briefing letter.

MR. SUGARMAN: And what are my clients to do if they can't afford to buy a transcript, Mr. Bonney? What are they to do?

MR. BONNEY: Your Honor, I think it is a Commission regulation that briefs have references to the record and it's impossible to follow the argument without it.

MR. SUGARMAN: Your Honor, I move that the company, which is paid by the ratepayers, be required to make a copy of the transcript available to the Protestants, whose property and health they are seeking to influence.

JUDGE SMOLEN: The transcript is available here. I have copies of the transcript and they can be referred to here in this office in our library.

MR. SUGARMAN: Will this office be open over the weekends, Your Honor.

JUDGE SMOLEN: No.

MR. SUGARMAN: I request that the company be directed, which is paid by my client's money --

JUDGE SMOLEN: To do what?

MR. SUGARMAN: To make a copy of the transcript.

JUDGE SMOLEN: Well, there are copyright laws. I think you have to check that out with the reporter. I don't believe that they can photocopy them.

MR. SUGARMAN: I don't mean by photocopying it. I mean by buying it from the court reporter.

JUDGE SMOLEN: Do you have any comment on Mr. Sugarman's request?

MR. BONNEY: Yes, Your Honor. As far as I know that request is unprecedented. I think it is completely out of line.

I would also note that Mr. Sugarman characterized us as the moving party in essence here, and I note that we already have a Commission order approving this line and this case is brought by the Protestants. If they want to bring this type of case and submit testimony and file briefs I think it is incumbent upon them to --

JUDGE SMOLEN: You made your point. I don't want to get involved in argument. We are going to try to make it as convenient as possible for Mr. Sugarman's clients. While we are not open here on the weekends, I have a copy

of the transcript. You can come up and look at it here in our library and use my set, at least up until the time I receive the briefs. At that point and even probably before, I will want to review those transcripts anyway.

Having put that question aside, I want to thank all our Counsel present for their well prepared cases and often heated cross-examination. It has been an interesting and enlightening case and of course I look forward to receiving scholarly briefs from each and every attorney here.

Thank you all very much for appearing here and attending. We will adjourn today's session.

(Whereupon, at 3:54 p.m., the hearing was concluded.)

C E R T I F I C A T E

I hereby certify, as the stenographic reporter, that the foregoing proceedings were taken stenographically by me and thereafter reduced to typewriting by me or under my direction; and that this transcript is a true and accurate record to the best of my ability.

COMMOMWEALTH REPORTING COMPANY, INC.

By: Robert J. Stonaker

Robert J. Stonaker

OCA Statement 2A
A-110550F055
2/7/92
OALJ, PA 1015

BEFORE THE

PENNSYLVANIA PUBLIC UTILITY COMMISSION

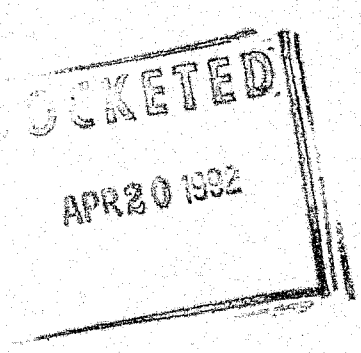
SURREBUTTAL TESTIMONY

OF

DAVID M. ROSENBAUM

ON BEHALF OF

PENNSYLVANIA OFFICE OF CONSUMER ADVOCATE



Risk Analysis Corporation
6723 Whittier Avenue, Suite 202
McLean, Virginia 22101

January 1992

SURREBUTTAL TESTIMONY OF DAVID M. ROSENBAUM

Q. Please state your name and business address.

A. David M. Rosenbaum, Risk Analysis Corporation, Suite 202, 6723 Whittier Avenue, McLean, Virginia 22101.

Q. Are you the same David M. Rosenbaum who testified before in this case?

A. Yes.

Q. What are the purposes of your surrebuttal testimony?

A. The purposes of my testimony are to comment on certain aspects of the rebuttal testimonies and cross-examinations of Dr. Philip Cole and Mr. J. Michael Silva.

Q. What are your comments on Dr. Cole's rebuttal?

A. I have two comments.

On page 16 of his rebuttal testimony, in discussing the Savitz study, Dr. Cole three times uses the characterization "irregular statistically significant associations", or "irregular associations" (lines 14, 23, and 39). The use of the term "irregular" before the term "statistically significant associations" strongly suggests to the reader that Savitz's results are considerably weaker and more suspect than would be suggested by the term "statistically significant associations" without this modifier. In fact, "irregular" has no technical meaning in this context, and what Dr. Cole means by it is unclear.

Q. What is your second comment on Dr. Cole's rebuttal?

A. One of Dr. Cole's main points is based on his statements that "the positive associations with the wiring code that Savitz did report are weaker than the associations reported by Wertheimer and Leeper" (page 17, lines 12–14) and "He [Peters] found an association between the wiring code and childhood cancer that is weaker still than the association seen by Savitz." (page 18, lines 13–15) . . . "With regard to the findings on wiring code, my conclusion is that the weaker association between the wiring code and cancers continues the pattern of better-done studies reporting smaller associations, which again suggests that there is no true association between EMF and cancer underlying these statistical reports." (page 18, lines 18–22)

There are several points to be made about this argument. First, by far the most important thing is that all three studies, including the very carefully done Savitz and Peters studies, found associations between wiring configuration and childhood leukemia. The obvious thing to say about the three studies is that they all observed an association between wire code classification and leukemia. That is not to say that they "proved" that EMFs cause childhood leukemia; as I pointed out in my direct testimony, it is not clear what it means to "prove" cause and effect with epidemiology.

Second, it is not necessarily true that a more carefully done study should get stronger results. For example, some of the technical problems with the Wertheimer & Leeper study may have introduced bias; *e.g.*, the non-blind wiring code classifications. In such a case, a more carefully done study might give weaker results even if there is a real effect. Thus, it is not clear that Wertheimer & Leeper's stronger results, compared to those of Savitz and Peters, is any evidence at all that the reported associations between wiring code classification and childhood leukemia are not evidence of a real effect.

Third, and perhaps most important, I believe it is not entirely fair to say that: "He [Peters] found an association between the wiring code and childhood cancer that is weaker still

than the association seen by Savitz." The Peters study had a higher odds ratio,* both adjusted (1.73) and unadjusted (2.15), than the Savitz study (1.54). [The Odds Ratio is a measure of the relative risk of "exposed people" compared to "unexposed people". For example, an Odds Ratio of 2.00 means that the risk of the exposed is twice that of the unexposed. An odds ratio of 1.00 means that there is no difference in risk between the exposed and the unexposed. An odds ratio less than 1.00 means that the risk of the exposed is less than that of the unexposed.]

The lower end of the 95 percent confidence interval** in Savitz is 0.90; the lower end

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* In order to define "Odds Ratio" we must define the following quantities:

- o N(E,D) = The number of those who were in an "exposed" group [in the Peters study, those from birth to ten years old with a particular wiring code other than "Underground and Very Low"] who got the disease [in the Peters study, leukemia]. [There are three such exposed groups in the Peters study: people with "Ordinary Low", "Ordinary High", and "Very High" wire code configurations.]
- o N(U,D) = The number of those in the "unexposed" group [in the Peters study, those from birth to ten years old with an "Underground and Very Low" wiring code] who got the disease.
- o N(E,H) = The number of those in an exposed group who did not get the disease.
- o N(U,H) = The number of those in the unexposed group who did not get the disease.

Odds Ratio = [N(E,D)*N(U,H)] / [N(U,D)*N(E,H)],

where * indicates multiplication and / indicates division.

** It is relatively easy to calculate estimates of numbers like odds ratios, what is often very hard is to determine how much confidence one should have in them. That is, what the chance is that a result, like an odds ratio, is really just due to random chance and does not indicate what the "true" situation is. A 95 percent confidence interval means that if the same "experiment" was repeated a great many times on exactly "equivalent" populations the "true" odds ratio would be within the 95 percent confidence intervals at least 95 percent of the time. It is of course not possible to repeat an epidemiological

(continued...)

of the 95 percent confidence interval in the Peters study is 0.82 adjusted and 1.08 unadjusted. [A 95 percent confidence interval is, roughly, the range in which the "true" odds ratio is found 95 percent of the time.] Since 0.90 and 0.82 are both below 1.0 neither the Savitz study nor the Peters study indicate that there is a 95 percent probability that there is a "true" positive correlation between wire code classification and childhood leukemia.

The situation is further confused by the fact that the Peters and Savitz figures compare different things. Savitz (his Table 7) compares (buried, very low and low wire code) with (high or very high wire code); Peters (his Table 9) compares (underground and very low wire code) with (very high wire code). If the effect increases monotonically (*i.e.,* the effect increases with each increase in wire code classification), this would make Savitz's results weaker than Peters' results. Indeed, when Savitz (his table 9) compares buried wire code with very high wire code, his leukemia odds ratio goes up to 2.75, but there are so few cases that the 95 percent confidence interval is very wide (0.94 to 8.04). On balance, one might call the associations found by the two studies fairly comparable. I do not believe that the results of the Wertheimer & Leeper, Savitz and Peters studies indicate that if better and better studies are done the reported association between wire code classification and childhood leukemia will eventually go away.

I think it is instructive to compare the odds ratios found for measured electric fields in these studies with those found for wiring code classification. The odds ratios for measured electric fields in Peters are either very close to 1.00 or substantially below 1.00 (page 932 of the Peters study). In Savitz the odds ratios for measured electric fields are all close to 1.00 (Tables 2 and 3). In contrast, 13 of the 14 odds ratios for wire code

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**(...continued)
experiment on a different, but exactly equivalent, group. Thus some assumptions have to be made. (One important assumption is that if the number of cases and controls in each "experiment" is large enough then the errors in the estimates of the "true" odds ratio will be distributed approximately normally.)

configurations given in Savitz's Table 7 are above 1.0, 12 are above 1.5, 5 are above 2.0, and one is 3.3. These sets of odds ratios for measured electric fields and wire code configurations provide a more convincing argument that there is no "true" association of childhood cancer with measured electric fields than that there is no "true" association of wire code classifications with childhood leukemia.

Q. Does this complete your comments on Dr. Cole's rebuttal testimony?

A. Yes.

Q. Do you have any comments on Mr. Silva's rebuttal testimony?

A. Yes. I would like to make a few comments on the concept of "prudent avoidance". The real issue is what is good public policy. As I stated in my direct testimony, (page 7, lines 8–19) the public should be involved in the decision making process before critical decisions are made. At the very least, companies considering power line projects should consider the cost and effects of possible mitigative measures before any construction decisions are made. This was not done in the case of the PECO 230 Kv line. All the calculations on alternatives were done after the line was virtually finished, and then only after a request from the Office of Consumer Advocate for such calculations. This is bad public and corporate policy. It is like driving on a long trip with bad brakes and no seat belts and calling it prudent because you arrived safely.

Q. Does this complete your surrebuttal testimony?

A. Yes.

OCA Statement 1A

A-110550F0055

2/7/92

BEFORE THE

PENNSYLVANIA PUBLIC UTILITY COMMISSION

SURREBUTTAL TESTIMONY

OF

DAVID E. JANES

ON BEHALF OF

PENNSYLVANIA OFFICE OF CONSUMER ADVOCATE

Risk Analysis Corporation
6723 Whittier Avenue, Suite 202
McLean, Virginia 22101

January 1992

SURREBUTTAL TESTIMONY OF DAVID E. JANES

Q. Please state your name and business address.

A. David E. Janes, Risk Analysis Corporation, Suite 202, 6723 Whittier Avenue, McLean, Virginia 22101.

Q. Are you the same David E. Janes who testified before in this case?

A. Yes.

Q. What is the purpose of your surrebuttal testimony?

A. The purpose of my testimony is to comment on the position taken by Dr. Edward Gelman on the need for further research into the biological effects of exposure to power frequency electric and magnetic fields.

In addressing the need for further research in his area (oncology and molecular and cellular biology, pp. 1141-1142 of transcript), Dr. Gelman takes the position that no additional research should be done on the relationship between electromagnetic fields and the causation or the promotion of cancer (pp 1147 of transcript).

In my opinion additional research is needed to resolve the present uncertainties about the role melatonin plays in carcinogenesis. As noted in my direct testimony (p.8 line 6 *et seq.*), exposure to 60 hertz electric fields suppresses night time concentrations of melatonin in the rat pineal gland. Also it has been recently shown that exposure of excised rat pineal glands to simultaneously applied static and 33.7 hertz magnetic fields depresses the in vitro synthesis of melatonin.[1]

While its role in human physiology may not be understood, melatonin is physiologically active in mammals. For example, melatonin synchronizes the annual cycle of reproduction in both long-day and short-day breeders, according to Reiter.[2] Melatonin also appears to affect the incidence of chemically induced tumors in rats. In a study by Tamarkin, melatonin significantly reduced the incidence of mammary tumors in rats treated with the tumor promoter DMBA and partially reversed the increased incidence of tumors in rats whose pineal glands had been removed.[3]

In summary, exposure to extremely low frequency electric and magnetic fields affects melatonin levels and melatonin levels affect the incidence of chemically induced breast tumors in rats. In my view, it is important to establish by direct experiment whether exposure to these fields affects tumor incidence in rats and then whether the results in this experimental animal system can be extrapolated to humans. This is especially important in view of the recently published study of Demers *et al.*, who report an association between occupational exposure to electromagnetic fields and breast cancer in men[4] and the suggestion from Wilson's work that low frequency electromagnetic fields may affect melatonin levels in some humans.[5]

Q. Does this complete your surrebuttal testimony?

A. Yes.

1. Lerchl A, Reiter RJ, Howes KA, Nonaka KO, Stokkan K-H (1991): Evidence that extremely low frequency Ca^{2+}-cyclotron resonance depresses pineal melatonin synthesis in vitro. Neuroscience Letters 124: 213-215.

2. Reiter RJ (1991): Melatonin: That ubiquitously acting pineal hormone. News in Physiological Sciences 6: 223-227.

3. Tamarkin L, Cohen M, Roselle D, Reichert C, Lippman M, Chabner B (1981): Melatonin inhibition and pinealectomy enhancement of 7, 12-dimethylbenz(a)anthracene induced mammary tumors in the rat. Cancer Research 41:4432-4436.

4. Demers PA, Thomas DB, Rosenblatt KA, Jimenez LM, McTiernan A, Stalsberg H, Stemhagem A, Thompson WD, McCrea Curnen MG, Satariano W, Austin DF, Isacson P, Greenberg RS, Key C, Kolonel LN, West DW (1991): Occupational exposure to electromagnetic fields and breast cancer in men. American Journal of Epidemiology 134(4): 340-347.

5. Wilson BW, Wright CW, Morris JE, Buschbom RL, Brown DP, Miller DL, Sommers-Flannigan R, Anderson LE (1990): Evidence for an effect of ELF electromagnetic fields on human pineal gland function. Journal of Pineal Research 9: 259-269.

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February 13, 1992

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BY OVERNIGHT MAIL

The Honorable Herbert S. Smolen
Administrative Law Judge
Pennsylvania Public Utility Commission
1302 Philadelphia State Office Building
Broad and Spring Garden Streets
Philadelphia, PA 19130

Re: Philadelphia Electric Company Exhibits, Woodbourne-Heaton Proceeding

Dear Judge Smolen:

Per your instructions at the February 7, 1992, hearing, enclosed with this letter are typed copies of Philadelphia Electric Company Cross-Examination Exhibits 5(a) and 5(b).

In addition, our records are unclear as to whether copies of Philadelphia Electric Company Cross-Examination Exhibits 1 and 2 were distributed to the parties. Copies of those Exhibits are thus also included with this letter.

Two copies of these Exhibits have also been forwarded to the Court Reporter.

Sincerely,

Ward L. Smith

Ward L. Smith

Enclosures

cc: By Overnight Mail

Robert J. Sugarman, Esq.
Tanya J. McCloskey, Esq.
Patricia Krise-Burket, Esq.

By First Class Mail

Jesse A. Dillon, Esq.
Paul R. Bonney, Esq.
Robert Stonaker

PECO Cross-Examination
Exhibit 5(a)

A-110550F055
2-7-92
RJS
Ph-A

	Very High Compared to Underground and Very Low	Very High Compared to Buried
Savitz	2.78 ~~2.75~~ *	2.75
Peters Crude	2.15	1.75
Peters Adjusted	1.73	NA

* Very High compared to Buried

PECO Cross-Examination
Exhibit 5(b)

A-111550F0055
2-7-92
RJS
Phila

Odds Ratio = [N(E,D) * N(U,H)] / [N(U,D) * N(E,H)]
[7 * 105] / [33 * 8]
735 / 264 = 2.78

[42 * 11] / [11 * 24]
462 / 264 = 1.75