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PUBLIC UTILITY COMMISSION

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

Pages 1069 through 1135 Hearing Room No. 1
State Office Building
Broad and Spring Garden Streets
Philadelphia, Pennsylvania

Wednesday, November 20, 1991

Met, pursuant to adjournment, at 10:00 a.m.

BEFORE:

HERBERT SMOLEN, Administrative Law Judge

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C O N T E N T S

<u>WITNESSES</u>	<u>DIRECT</u>	<u>CROSS</u>	<u>REDIRECT</u>	<u>RECROSS</u>
Philip Cole				
By Mr. Watson	1071	---	---	---
By Mr. Dillon		1078	---	---
By Ms. Dusman		1081	---	---
		1126		
By Mr. Sugarman		1105	---	---
		1129		

E X H I B I T S

<u>NUMBER</u>	<u>FOR IDENTIFICATION</u>	<u>IN EVIDENCE</u>
<u>Philadelphia Electric Company</u>		
✓ Rebuttal Statement No. 2 (Cole)	1078	1078

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P R O C E E D I N G S

1
2 ADMINISTRATIVE LAW JUDGE HERBERT SMOLEN: This is a
3 further hearing in the matter of the Philadelphia
4 Electric Company Woodbourne-Heaton line, Docket A-110550,
5 Folder 055.

6 Mr. Watson, or Mr. Bonney, are you prepared for
7 your next witness?

8 MR. WATSON: Yes, Your Honor.

9 I call Dr. Philip Cole.

10 Whereupon,

11 PHILIP COLE

12 having been duly sworn, testified as follows:

13 JUDGE SMOLEN: Please have a seat. Keep your voice
14 up. Welcome to the PUC. State your full name and
15 business address for the record.

16 THE WITNESS: My business address is 203 Tidwell
17 Hall at the University of Alabama at Birmingham.

18 JUDGE SMOLEN: Mr. Watson.

D I R E C T E X A M I N A T I O N

19
20 BY MR. WATSON:

21 Q. Dr. Cole, do you have in front of you a copy of
22 a document entitled Rebuttal Testimony of Dr. Philip Cole
23 and also labeled Philadelphia Electric Company Rebuttal
24 Statement No. 2?

25 A. I do.

1 Q. Do you have any typographical additions or
2 corrections to make to this document?

3 A. Yes. There are two typographical errors.

4 JUDGE SMOLEN: Before you start, is there an extra
5 copy of the testimony? What I want to do is for
6 Mr. Sugarman's sake I am going to make an extra copy of
7 the corrections and when he comes in I will give it to
8 him. We can save some time that way.

9 Go ahead.

10 BY MR. WATSON:

11 Q. The corrections?

12 A. Yes. There are two typographical errors in the
13 testimony that I would like to correct now. The first
14 one is on page 19, line 42. The name spelled "Branford"
15 should be changed to "Bradford", with a "d".

16 Page 20, line ten, the word "the" should be
17 deleted.

18 Q. Dr. Cole, are there any other additions or
19 corrections you would like to make to your testimony to
20 bring it completely up to date?

21 A. Yes. The November 1 issue of the American
22 Journal of Epidemiology was published yesterday and it
23 contains the final version of a report that is commonly
24 referred to as the Peters study. I have had a chance to
25 review that final report and as a result of that there

1 are a number of changes which convert the preliminary
2 nature of my comments regarding that study to much more
3 firm comments. So there are several changes that I would
4 like to make for that reason.

5 Q. Let me just ask you, were there any material
6 changes between the final report of Peters and the
7 information that you have in your testimony about the
8 Peters study?

9 A. No. The changes are not material. They
10 basically represent a firming up of results with which I
11 was already somewhat familiar.

12 Q. And which are referred to in your testimony?

13 A. Yes, sir.

14 Q. Could you give us the specific page and line
15 numbers for those changes?

16 A. All right. There are some ten or so of these.
17 Page 12, line ten is the first one. The word
18 "five" should be changed to the word "six".

19 Page 12 --

20 JUDGE SMOLEN: Go a little slower here.

21 THE WITNESS: Certainly.

22 (Pause.)

23 JUDGE SMOLEN: Go ahead.

24 THE WITNESS: Page 12, line 12, I would add to the
25 end of the line the words "and London (1991)."

1 BY MR. WATSON:

2 Q. Could you tell us what London has to do with
3 Peters?

4 A. The final report of what is referred to as the
5 Peters study actually appears under the name of Stephanie
6 London as the senior author. Peters is also included in
7 the authorship.

8 Q. Am I correct in understanding that is one and
9 the same study?

10 A. That's the same study, yes, indeed.

11 Q. Please continue.

12 A. Page 16, line six, I would ask that we insert
13 the words "one of" before the expression "the best". So
14 it now says "should be considered one of the best".

15 Page 17, beginning on line 35 with the word
16 "since", I would strike the rest of that response from
17 the word "since" to the very end. And in that same
18 location add the sentence "I have also reviewed the final
19 report of the Peters study."

20 JUDGE SMOLEN: Read the whole thing, then.

21 THE WITNESS: Okay. The whole response now reads
22 as follows: "I was a member of the advisory committee
23 which sponsored the Peters study and was present at the
24 advisory committee meeting at which Dr. Peters presented
25 his preliminary results," deleting the remainder now and

1 adding, "I have also reviewed the final report of the
2 Peters study.

3 JUDGE SMOLEN: Following the word study --

4 THE WITNESS: That is the end.

5 JUDGE SMOLEN: What would the next thing be? "At
6 present", or is that also stricken?

7 MR. WATSON: Your Honor, everything is stricken
8 from the word "since" through "findings".

9 JUDGE SMOLEN: So the final two sentences are also
10 stricken?

11 THE WITNESS: From the word "since" to the end of
12 the original response is stricken, and then I add in a
13 sentence.

14 JUDGE SMOLEN: All right. What is the next one?

15 THE WITNESS: Going now to page 18, line one,
16 perhaps it would be easiest if I simply read this
17 sentence now. There are two changes in it.

18 JUDGE SMOLEN: All right.

19 THE WITNESS: "Not enough design information and
20 data were available." So we change "are" to "were".
21 "Not enough design information and data were available
22 from the Peters study at the time I originally prepared
23 this testimony." And then it continues as is, that is,
24 "to conduct a detailed epidemiologic evaluation."

25 I'm sorry. There are two more changes in that

1 response. The next one is 18-three. The word "cannot"
2 should be changed to "could not". And at 18-four, change
3 "is" to "was".

4 Page 18, line nine, delete, please, the first
5 clause "with the limitations just expressed". Delete
6 that and add the word "now" after the word "can". So
7 that it says, "I can now state my..." and delete the word
8 "preliminary". "I can now state my opinions."

9 Eighteen-16, delete the word "preliminary".

10 Eighteen-19, delete the word "preliminary".

11 BY MR. WATSON:

12 Q. Are there any more additions, Dr. Cole?

13 A. I just want to check here to make sure I have
14 not omitted any.

15 (Witness perusing document.)

16 Yes, there is one more addition that I would like
17 to make, and that is on page 31. After the reference to
18 the work of Linet I would like to add in a reference to
19 the work that represents the final report of the Peters
20 study. So that reference which would go in there would
21 read as follows: "London, S.J., Thomas, D.C., Bowman,
22 J.D., et al., Exposure to Residential Electric and
23 Magnetic Fields" -- that was Exposure to Residential
24 Electric and Magnetic Fields -- "and Risk of Childhood
25 Leukemia."

1 Next, the reference to the journal itself is as
2 follows: first is the abbreviation "Am." That is for
3 American. Next "J", for Journal, and finally "Epidemiol"
4 for Epidemiology. That is underlined, "American Journal
5 of Epidemiology".

6 Next is the year, 1991,

7 Next, the volume number, 134:923-37.

8 That concludes the changes that I would like to
9 make to my testimony.

10 JUDGE SMOLEN: Just give me a second here.

11 (Pause.)

12 JUDGE SMOLEN: We are ready to go?

13 BY MR. WATSON:

14 Q. Dr. Cole, if I were to ask you now while you
15 are on the stand under oath each of the questions set
16 forth in this document entitled Rebuttal Testimony of
17 Dr. Philip Cole, would your answers be the same as set
18 forth therein with the corrections you have noted today?

19 A. Yes, they would be.

20 MR. WATSON: Your Honor, we move that the testimony
21 of Dr. Cole be admitted into evidence subject to timely
22 objections.

23 JUDGE SMOLEN: All right. It is received subject
24 to that qualification.

25

1 (Whereupon, the document was marked
2 as PECO Rebuttal Statement No. 2
3 for identification, and was
4 received in evidence.)

4 MR. WATSON: I pass the witness, Your Honor.

5 JUDGE SMOLEN: All right, Ms. Khanwalkar. No
6 questions?

7 MS. BURKET: No, Ms. Burket has no questions.

8 JUDGE SMOLEN: I'm sorry. I'm reading from a list
9 that I have. It's Mr. Dillon who is from PP&L.

10 MR. DILLON: That's correct, Your Honor.

11 JUDGE SMOLEN: Do you have any questions?

12 MR. DILLON: I do have two quick questions.

13 JUDGE SMOLEN: Go ahead.

14 CROSS-EXAMINATION

15 BY MR. DILLON:

16 Q. Good morning, Dr. Cole. My name is Jesse
17 Dillon and I represent the Pennsylvania Power & Light
18 Company, an electric utility here in Pennsylvania.

19 Could you please turn to page 26 of your testimony?
20 I believe at lines 12 through 20 you are discussing a
21 study by Dr. Matanoski involving linemen, telephone
22 linemen, is that correct?

23 A. Yes.

24 Q. And you state at line 15 that preliminary
25 results indicate two to three more cases than expected of

1 male breast cancer. And my question for you is is that
2 two or three times more cases than expected two or three
3 out of how many? Could you expand on that statement?

4 A. Yes. First I would like to point out that the
5 Matanoski study was an offshoot of a larger investigation
6 that she had conducted on leukemia mortality, and she did
7 this study, which is a follow-up study of line and
8 non-line workers in the state of New York.

9 The results of that study are still in a
10 preliminary form. However, we now have more specific
11 information, and it is as follows:

12 In the entire group that she followed up there were
13 approximately 205,000 man-years of follow-up experience,
14 a man-year being one man followed up for one year. And
15 she observed two cases, not two or three, but two cases
16 of breast cancer among the men that generated these
17 200,000 or more man-years.

18 There is no formal expected number presented in the
19 report of this, which I have seen, which is published as
20 a letter and not in final form. However, it is contended
21 by Matanoski that these two cases represent an excess.

22 It's not clear to me that it would, the reason
23 being that the incidence rate of male breast cancer is
24 about one per 100,000 man-years, and as I say, she has
25 200,000 man-years. So the expected number for that

1 series should be in the vicinity of two, a little more or
2 less depending upon the age structure of the men that she
3 actually followed up. So at the present time I would
4 have to say that until there is clarification of that I
5 really don't know that we can interpret the Matanoski
6 study. It is clear that the expected number would be in
7 the vicinity of the observed number but just how close
8 they are, I really don't know.

9 MR. DILLON: No further questions, Your Honor.

10 JUDGE SMOLEN: All right. Ms. McCloskey.

11 MS. McCLOSKEY: Ms. Dusman will be conducting the
12 cross-examination.

13 JUDGE SMOLEN: All right.

14 MS. DUSMAN: Your Honor, I have no objection to the
15 changes made in Dr. Cole's testimony. However, although
16 we were aware of the article in the issue of the Journal
17 of Epidemiology that he referred to, of the Peters study,
18 we do not have a copy of it and I would ask whether he
19 could produce a copy of it today.

20 MR. WATSON: We have a copy. We just got it faxed
21 to us. We would be happy to share one with you.

22 MS. DUSMAN: In addition to that, we noted that
23 there was a page missing from a transcript which was
24 provided us in response to our discovery. It is a minor
25 detail but Mr. Smith has offered to check to see whether

1 they have that with them. And since we are anticipating
2 a break anyway, I would reserve the right to
3 cross-examine on either the missing page of the
4 transcript or the Peters study following the break.

5 JUDGE SMOLEN: There is no objection?

6 MR. WATSON: No objection.

7 JUDGE SMOLEN: All right. Then we will proceed in
8 that fashion.

9 MS. DUSMAN: Thank you.

10 CROSS-EXAMINATION

11 BY MS. DUSMAN:

12 Q. Good morning, Dr. Cole.

13 A. Good morning.

14 Q. My name is Dianne Dusman and I am here on
15 behalf of the Office of Consumer Advocate.

16 MR. WATSON: Excuse me once again. I just noticed
17 one other change that might be consistent with the
18 changes Dr. Cole made on the Peters study that change
19 basically the words from preliminary to now. Could I
20 just bring that to everybody's attention for what it is
21 worth?

22 JUDGE SMOLEN: Page 26?

23 MR. WATSON: Yes, page 26 line 12. It says "As
24 with the Peters study not enough information is
25 available," and I think that has now changed based on

1 what Dr. Cole said.

2 JUDGE SMOLEN: Why don't we let the witness say
3 that.

4 MR. WATSON: Is that an appropriate correction?

5 THE WITNESS: Yes, that is entirely appropriate and
6 apparently I omitted to mention that when I went through
7 my list.

8 MR. WATSON: So how would you change that?

9 THE WITNESS: So page 26, line 12, reads as
10 follows: just delete the expression "as with the Peters
11 study," capitalize the word "not", so it simply says now,
12 "Not enough information is available about the data and
13 design of the Matanoski study."

14 MR. WATSON: Thank you.

15 I'm sorry for the interruption but I thought we
16 ought to straighten that out.

17 BY MS. DUSMAN:

18 Q. Dr. Cole, with respect to the Matanoski study,
19 do you know when to expect that final results will be
20 available?

21 A. No, I don't.

22 Q. You state at page five of your direct testimony
23 that the purpose of your analysis is to offer a
24 professional opinion as to whether the E/MFs associated
25 with the Woodbourne-Heaton 230 kV transmission line will

1 cause cancer. Is it accurate to state, then, that your
2 testimony is immaterial to whether any other adverse
3 health effects might be caused by E/MFs surrounding the
4 line?

5 A. I would like to focus on the question of cancer
6 as a potential adverse effect of E/MF.

7 Q. In other words, you are making no statement as
8 to whether E/MF has a causal connection to other adverse
9 health effects?

10 A. I would restrict my testimony to the question
11 of E/MF and cancer.

12 Q. Would you turn to your testimony at page six,
13 lines three to eight, where you address the meaning of
14 the word cause. Is it accurate to state that when you
15 use the term cause you also intend to include the meaning
16 increased the risk of disease?

17 A. I'm sorry. I don't understand. It appears
18 that the response already includes those very words.
19 Maybe -- I must be missing something.

20 Q. Is it true that throughout your testimony when
21 you use the word cause you also mean to express your
22 opinion as to whether cancer is -- the risk of cancer is
23 increased?

24 A. Well, without reviewing each place, my response
25 to that would be probably yes, it is conceivable to me.

1 I don't recall that there would have been a place where
2 perhaps by example I might have used cause to mean a
3 causal reduction. But I don't specifically recall any
4 such thing. So generally yes, to cause is to increase
5 the risk in general unless it is specifically clear
6 otherwise.

7 Q. Since you have just stated, then, that your
8 testimony is intended to be limited to whether cancer is
9 caused by exposure to E/MF, you are also expressing no
10 opinion, then, as to whether E/MF exposure increases the
11 risk of other adverse health effects?

12 A. That's correct.

13 Q. Does your review, Dr. Cole, make any
14 distinction between electromagnetic fields as a potential
15 contributing factor to the development of cancer and E/MF
16 as a possible contributing factor to the growth of cancer
17 cells?

18 A. No, it does not. I consider both of those to
19 be in the definition of a cause.

20 Q. At page 16 you direct your attention to lines
21 26 through 30. You state at that place in your
22 testimony, quote, a few irregular statistically
23 significant associations in a case-control study are to
24 be expected even when nature is in a null state. Is that
25 an accurate paraphrase of your testimony?

1 A. Yes, Ma'am.

2 Q. What do you mean by when nature is in a null
3 state?

4 A. I mean by that that there is in fact and in
5 truth no relationship between the factor under study and
6 the disease that is under study.

7 Q. What would you use to draw a conclusion that
8 there is no causal connection in fact between a
9 particular factor and a human disease?

10 A. The ability to defend the proposition that
11 there is not a causal relationship between a factor under
12 study and a disease rests upon a number of different
13 kinds of information. One would be a series of studies
14 that are perceived to be of good quality and valid which
15 failed to show any such association. And second is the
16 absence of information either from animal studies or from
17 other kinds of investigations to the contrary, that is,
18 supporting the idea that there is a causal relationship.

19 Q. Can you give me an example of an instance in
20 which, as you have stated here, nature is in a null
21 state?

22 A. And in which the data in the study appeared to
23 be contrary to that? That is, positive?

24 Q. No. I just want an example of an instance in
25 which you would consider that nature is in a null state.

1 A. Okay. Saccharin does not cause cancer of the
2 bladder in human beings. That is a null state.

3 Q. You have drawn that conclusion from the items
4 you listed in your earlier answer?

5 A. That is correct.

6 Q. So in this particular circumstance you have
7 only the materials that you have referred to within your
8 testimony to determine whether nature is in a null state,
9 is that correct?

10 A. When you say this particular circumstance are
11 you speaking about this material contained on lines 26
12 through 30?

13 Q. I am talking about in general when you want to
14 discern whether nature is in a null state.

15 MR. WATSON: Your Honor, I think we have at least a
16 very confusing question at best. We have a question and
17 then -- a question by the witness and a follow-up
18 question.

19 JUDGE SMOLEN: Well, this is a sophisticated
20 witness and if he does not understand the question I
21 think he can say he does not understand the question.

22 Do you understand the question?

23 THE WITNESS: Your Honor, I think I understand the
24 question but I don't understand the context of it,
25 whether I am discussing E/MF or the nature of

1 establishing the absence of causality in general or what.
2 That is where I am a little confused.

3 MS. DUSMAN: I will go forward, Your Honor.

4 BY MS. DUSMAN:

5 Q. The particular reference on this page, are you
6 making this statement in terms of the Savitz study?

7 A. Yes.

8 Q. Does the Savitz study concern a connection
9 between childhood cancer and electromagnetic fields?

10 A. Well, it -- yes, it does.

11 Q. Now, other than the materials that you have
12 referred to within this rebuttal statement, do you have
13 any others to use in order to determine when nature is in
14 a null state or not?

15 A. May I ask, when you say how do I determine
16 whether nature is in a null state or not with regard to
17 the question of electromagnetic fields and childhood
18 cancer?

19 Q. Yes, sir.

20 A. Okay. My perception that nature is probably in
21 a null state in this regard is based primarily on my
22 familiarity with the epidemiologic literature but I also
23 have some degree of familiarity with the other literature
24 as well.

25 Q. When you say other literature, what are you

1 referring to?

2 A. I am referring to animal studies and cellular
3 studies.

4 Q. Later in your testimony at page 18 you discuss
5 your preliminary opinions -- well, now you discuss your
6 opinions -- about the Peters study, do you not?

7 A. Yes.

8 Q. And Peters also found an association between
9 wiring code and childhood cancers did he not?

10 A. He found a weak association between wire code
11 and not childhood cancers, no. Leukemia. That study was
12 restricted to leukemia.

13 Q. If one more study were completed as the Peters
14 study was just completed of that same caliber which
15 showed an association between wiring code and leukemia,
16 would that additional study cause you to reconsider your
17 opinion as to this issue?

18 A. I would reconsider my opinion any time that I
19 am exposed to any new information. But I would like to
20 point out that an opinion such as I hold on this issue,
21 which is now many years old and based on more than 50
22 studies, is not likely to change a great deal on the
23 basis of any one study. It is much more important to
24 look into the studies and to perceive the patterns of
25 consistency or inconsistency among them.

1 For example, we have may have left the impression
2 that the Peters study is clearly positive with regard to
3 wire code. It most assuredly is not. There are
4 inconsistencies in the data. There are changes in the
5 data that reflect correction for other factors. And also
6 the association is somewhat weaker than that which was
7 seen in the Savitz study, which in turn is weaker than
8 that which was seen in the original paper, that is, the
9 Wertheimer 1979 paper.

10 So to me the question is not whether or not one
11 more study might be positive, but rather, whether or not
12 it can alter my perception of the long-term time trend in
13 the results of these study, which is that as the studies
14 are improving in methodology and size and focus they are
15 getting weaker and weaker. To me that is the issue, not
16 whether or not there is one more positive study.

17 Q. Would you agree, then, that consistently weak
18 associations can lead you to draw an inference as to
19 causation?

20 A. Oh, I would -- if I understand this question
21 correctly, I would agree with you. It is possible that a
22 large body of consistent information to the effect that
23 an association is weak would permit that association to
24 be accepted as causal. However, I don't know of any
25 example of that but one, and even that one is somewhat

1 dubious. So in general weak associations just don't
2 muster up. They tend to fade away.

3 Q. Dr. Cole, are there epidemiological studies
4 which identify groups of individuals which are more or
5 less susceptible to cancerous conditions?

6 A. I think it would be fair to say that most
7 epidemiologic studies do not focus on the relationship
8 between susceptibility and disease but, rather, exposure
9 to external agents. However there some studies, very
10 few, at least very few that I am aware, that do try to
11 isolate what we might refer to as susceptibility, usually
12 implying that it's a genetic trait or a consequence of
13 some very early environmental exposure.

14 Q. Is it possible, then, that variations in
15 susceptibility to the development of cancerous conditions
16 among population subgroups can interfere with the
17 consistency in epidemiological study results?

18 A. It's possible but it is not considered a major
19 distorting factor.

20 Q. Dr. Cole, if further research were performed
21 which in your opinion resulted in a demonstration that a
22 biologically plausible mechanism existed connecting E/MF
23 exposure and cancer, would that cause you also to
24 reconsider your opinion as to whether the epidemiological
25 studies demonstrate a causal connection between the two

1 things?

2 A. I don't think it would cause me to reconsider
3 my opinion as to whether or not the epidemiologic studies
4 demonstrate the relationship. But it would cause me to
5 reconsider my overall opinion as to the credibility of
6 the idea that magnetic fields cause cancer.

7 Q. Well, you stated in your testimony that you
8 interpret the epidemiological studies and consider at the
9 same time whether there is evidence of a biologically
10 plausible mechanism. Are you now stating that you would
11 not reconsider your opinion as to those studies in the
12 event that the state of the science changed?

13 MR. WATSON: Objection, Your Honor. Argumentative.
14 And I don't think it accurately states what the witness
15 said.

16 THE WITNESS: I don't --

17 JUDGE SMOLEN: Wait a minute. We have an
18 objection.

19 Do you want to be heard on that? The objection is
20 that it is argumentative. It's asking why.

21 MS. DUSMAN: I don't think asking this witness a
22 why question is argumentative at all. He has stated his
23 rebuttal statement in terms of looking at the studies
24 concomitantly. I asked him whether he would change his
25 opinion about the epidemiological studies if further

1 evidence as to a biologically plausible mechanism were
2 available. And then he said --

3 JUDGE SMOLEN: And now you are asking him for the
4 reason why he wouldn't change his opinion.

5 MS. DUSMAN: He said no, he would not.

6 JUDGE SMOLEN: And now you are asking for what
7 reason would you not change the opinion?

8 MS. DUSMAN: That's correct, Your Honor.

9 MR. WATSON: I think he said, no, he would not
10 change his opinion if there was a biological plausibility
11 theme brought to the forefront, but wouldn't change his
12 opinion on the epidemiological studies themselves. But
13 it might change his opinion overall. And I was concerned
14 with the misstatement of what he previously said, not
15 intentional, obviously.

16 JUDGE SMOLEN: Under those circumstances let's try
17 the question again and see if we can get it straightened
18 out.

19 BY MS. DUSMAN:

20 Q. Would the existence of a study which in your
21 opinion showed a valid biologically plausible mechanism
22 cause you to change your opinion as to this subject
23 overall?

24 A. It is not likely that the appearance of a study
25 such as you have described would cause me to change my

1 opinion if we are speaking about my opinion being the
2 response to the question is there or is there not an
3 established cause-effect relationship between E/MF
4 exposure and cancer in human beings.

5 On the other hand, such a piece of information
6 would certainly enter into my overall consideration of
7 the issue.

8 Q. Dr. Cole, I have a few questions for you
9 pertaining to ecological comparison, which you testify to
10 at page 20, lines seven to 28.

11 Would you direct your attention to the final
12 sentence of the first full answer where you state, "This
13 type of ecological comparison, while subject to
14 limitations, can provide important information as to a
15 suggested relationship." Would you tell us what
16 limitations you are referring to in that sentence?

17 A. The major difficulty in trying to interpret
18 ecological comparisons as causal is their
19 non-specificity. That is, they basically represent
20 either time or place or time and place comparisons. And
21 when one makes comparisons over, as in this example, over
22 time it is not just thing that changes, not even one
23 relevant thing that changes.

24 For example, while cigarette smoking was going up,
25 so was air pollution. So this could be a detraction from

1 this idea that this particular ecological comparison is
2 valid.

3 So that is the problem with ecological comparisons,
4 non-specificity of association.

5 Q. Directing your attention to lines 19 to 20,
6 where you state that you use power consumption as a
7 surrogate for E/MF exposure, are you stating that a
8 ten-fold increase in power consumption results in a
9 ten-fold increase in exposure to electromagnetic fields?

10 A. No.

11 Q. What do you mean, then, by this?

12 A. Nobody knows how much E/MF exposure a human
13 being has even today, unless that individual human being
14 were to wear and use personal dosimetry for an extended
15 period of time. We have such information for a few days
16 for a few people. We certainly had no such information
17 back in 1950. So I would not assert that, for example,
18 there has been a ten-fold increase in E/MF exposure
19 between 1950 and 1960 or 1980.

20 What I mean to assert here is that during this
21 period of time there was a ten-fold increase actually in
22 per capita power consumption, not just power consumption.
23 And it seems reasonable to me to believe that E/MF
24 exposure would, if not parallel the increase in power
25 consumption, it would at least follow the same trend in

1 time, that is, upwards.

2 There are a number of observations that would
3 support that. For example, the average American home
4 built in 1950 had a 100 amp or 125 ampere power service
5 put into it. The average American home built in the
6 1980s has a 250 to 300 ampere power service. It is also,
7 I think, fair to say as a general observation that we use
8 more electricity today than we did 30 years or 40 years
9 ago.

10 Q. I believe initially in your answer you stated
11 that no one can possibly know whether the population
12 overall has experienced an increased exposure to
13 electromagnetic fields.

14 A. Well, I don't know if I used the actual words
15 "no one can possibly know". What I can see is this: I
16 don't know and I have not been able to identify anyone
17 who does know.

18 Q. In doing your comparison, then, is it accurate
19 to state that you didn't consider the societal changes
20 that have occurred since 1950, for example?

21 A. Perhaps I could ask for some clarification of
22 what you mean by the societal changes.

23 Q. Well, I will rephrase the question. Did you
24 factor in any societal changes in doing this ecological
25 comparison?

1 A. No.

2 Q. You referred earlier to increased ampere
3 service. Does that necessarily increase exposure to
4 electromagnetic fields?

5 A. It could be contended that it does not
6 necessarily increase exposure. But it would be very
7 difficult for me to believe that it does not increase
8 exposure. The purpose of upgrading the service is to
9 allow the residents of the home to use more electric
10 power. That is the purpose of it.

11 Q. Doesn't that comparison assume that a person is
12 in their home consistently day in and day out for a long
13 period of time?

14 A. There are many factors that can be brought to
15 bear on the ecological comparison. You speak about the
16 time that people spend in the home. But in addition we
17 could point to the fact that more people work in offices
18 today than they did in 1950. A lot fewer people work out
19 of doors. These are facts.

20 Q. In doing your ecological comparison --

21 JUDGE SMOLEN: Wait. Let's see if the witness --

22 THE WITNESS: I am finished.

23 JUDGE SMOLEN: Okay. Go ahead.

24 BY MS. DUSMAN:

25 Q. Why did you limit your comparison in your study

1 to the 20 percent increase in childhood leukemia from
2 1950 to 1960?

3 A. Well, actually I -- maybe I don't understand.
4 It was not an effort to limit it to 1960. The remainder
5 of the response deals with the period of time subsequent
6 to 1960 or at least I intended that it would. I tried to
7 make a distinction between 1950 through '60 and 1960 and
8 after for what I thought was a good reason, namely there
9 was a rise between 1950 and 1960.

10 There was a rise in most forms of cancer in most
11 age groups. And this was almost certainly attributable
12 to improvements in the quality of diagnosis and
13 certification of cause of death. From 1960 on, most of
14 the modern diagnostic techniques -- not all, but most --
15 were in place and any rise that would have occurred since
16 1960 is more likely to be genuine than artifactual. But
17 there has in fact not been any further rise in childhood
18 leukemia.

19 Q. Do you agree or disagree with the statement
20 that overall the evidence as to association between
21 electromagnetic fields and cancer now available is too
22 weak to allow firm conclusions either way?

23 A. I would agree with that. It is not my own
24 position but I can understand how other people would hold
25 that position.

1 Q. You agree with that statement but it is not
2 your position in this case?

3 A. It is not my own position, that's correct.

4 (Pause.)

5 A. Perhaps I should just add one thing to my
6 previous answer. My problem with the answer is the idea
7 of firm conclusions, implying that one will never be able
8 to change. This is the difficulty that I have with it.

9 Q. Do you agree or disagree with the statement
10 that only in rare circumstances would it be justifiable
11 to infer a cause-effect relationship from a small number
12 of epidemiological studies?

13 A. Absolutely.

14 Q. At page 12 of your testimony -- you absolutely
15 agree with that statement?

16 A. Absolutely.

17 Q. At page 12 of your testimony, line ten, this
18 reflects that you have looked at six published
19 residential case-control studies regarding E/MF and
20 childhood cancer?

21 A. That's correct.

22 Q. Would you consider that to be a small number of
23 studies or a reasonably large number of studies?

24 A. Madam, I don't consider that particular group
25 of studies to be a set in and off themselves. They are

1 part of a much larger set. There are, for example, adult
2 residential studies and a number of other different kinds
3 of studies of the question of E/MF and cancer in human
4 beings. So even if we were to speak about just
5 residential studies, this would not comprise the whole
6 set.

7 Q. Well, let's turn for a moment to the
8 conclusions that you express at the end of your
9 testimony. Would you agree or disagree that you can hold
10 opinions as to particular conclusions with varying
11 degrees of certainty?

12 A. Yes.

13 Q. Have you ever said I am 100 percent certain of
14 a particular statement of fact?

15 A. Are we speaking about cause-effect
16 relationships or empiric facts like there is a table in
17 front of you?

18 Q. I am asking you in general whether you have
19 ever said that you are 100 percent certain of a
20 particular fact.

21 A. Yes, I have.

22 Q. With regard to your opinion on the relationship
23 between power frequency electric and/or magnetic fields
24 and cancer in humans, can you assess your degree of
25 certainty as to that opinion?

1 A. Is there some particular statement in here that
2 you are asking me about? Or are you asking a more
3 general statement?

4 Q. I am asking as to the opinion that you express
5 on this page of your testimony.

6 A. Which page is that?

7 Q. Page 28.

8 (Witness perusing document.)

9 MR. WATSON: Is that line 40, Counsel, which you
10 are referring to?

11 MS. DUSMAN: Yes.

12 MR. WATSON: Or wherever.

13 MS. DUSMAN: Yes, referring to line 40.

14 BY MS. DUSMAN:

15 Q. Can you assess your own degree of certainty
16 with respect to your opinion that E/MFs have not been
17 demonstrated to be carcinogenic?

18 A. Yes. I think I will try to be brief on this.
19 We ordinarily look at the spectrum of credibility of
20 carcinogenesis or causality to exist on a spectrum
21 between, let's say, zero and 100 percent where zero would
22 mean the relationship has not been demonstrated and 100
23 percent meaning that I am absolutely sure that it has
24 been demonstrated. So that is the spectrum in which we
25 ordinarily think of things, between zero and 100 percent.

1 But I point out that in causal thinking that is
2 only half the spectrum. There is another half to the
3 left which is between zero and minus 100, which means
4 between not demonstrated and demonstrated not to be
5 causal.

6 So the three points on the spectrum are minus 100:
7 E/MF has been demonstrated not to cause cancer in human
8 beings; zero: we are relatively uncertain about whether
9 it has been demonstrated not or demonstrated to cause
10 cancer; and to the extreme right we have plus 100, which
11 is E/MF has been certainly demonstrated to cause cancer
12 in human beings.

13 Now, on this full spectrum of the credibility of
14 ideas, my position is minus 70. I believe that it is
15 much more likely to be true than not that E/MF will be
16 demonstrated not to cause cancer in human beings.

17 Q. Okay. Do you assess your degree of certainty
18 as to that opinion at the same level for childhood
19 cancers as you do for adult cancers?

20 A. Yes, I do.

21 Q. Do you agree with me that in this context you
22 have drawn a causal inference -- I'm sorry -- you have
23 drawn not a causal inference but you have drawn an
24 inference as to absence of cause?

25 A. Well, I haven't actually drawn the inference by

1 which I would understand to mean once again this firm
2 conclusion idea, that I am absolutely sure that E/MF does
3 not cause cancer in human beings. I am 70 percent of the
4 way there. I'm not 100 percent of the way there.

5 Q. I understand.

6 In your opinion do you need the same amount of data
7 to draw an inference as to the absence of a causal
8 relationship as you do to draw an inference as to the
9 existence of a relationship?

10 A. Madam, I don't think it is a question of
11 amounts of data, if I interpret your question literally.
12 If I interpret your question a little more generally to
13 mean is it as easy as or easier or more difficult to
14 establish the minus 100 position, that is, the null state
15 as opposed to the plus 100 position, then I would say
16 that most people hold the opinion, and so do I, that it
17 is more difficult to establish the null state with
18 certainty.

19 Q. Is there a difference between holding an
20 opinion and having a belief?

21 A. This is not an actual distinction that I have
22 actually faced. When we speak about holding opinions
23 with regard to cause-effect relationships I can only say
24 that I like to perceive these opinions as existing on the
25 spectrum that I have described to you and whether or not

1 that should be called a belief, I don't have any
2 objection to that as long as it is understood that the
3 belief, like the opinion, can change.

4 Q. I would like you to focus on lines 40 to 42 on
5 page 28 of your testimony. Now, you have stated that you
6 are of the opinion that E/MFs have not been demonstrated
7 to be carcinogenics. And let's take these things one
8 thing at a time. You have said that you are 70 percent
9 certain of that opinion?

10 A. No. They are not exactly the same, and I
11 don't --

12 Q. Would you clarify for me what you meant by,
13 then, that you are 70 percent of the way towards feeling
14 that there is a 100 percent certainty that that is the
15 case?

16 A. It occurs to me that the best way I could do
17 this would be to phrase two different questions, that is,
18 am I of the belief or do I know of anyone who is of the
19 belief that electromagnetic fields have been demonstrated
20 to be carcinogenic in human beings. To my knowledge
21 there is no one who would answer that question in the
22 affirmative. There may be someone but I don't know of
23 any such person.

24 MS. DUSMAN: Your Honor, I would ask that that be
25 stricken and direct the witness to answer the question I

1 put to him.

2 JUDGE SMOLEN: Well --

3 MR. WATSON: Your Honor, I think it was a fair
4 attempt to try to get at the point Counsel was asking.

5 JUDGE SMOLEN: I am going to overrule the
6 objection. The witness says he does not know of anyone,
7 which is something within his knowledge. So I will
8 permit that to stand.

9 Let's go on with the rest of your answer.

10 A. In trying to be responsive I was simply
11 pointing out that from my perception the issue here has
12 related to two questions. One is where am I on the right
13 side of this spectrum, and if that is the only spectrum
14 we can talk about, if that is your wish, then I am at the
15 zero position. I simply offered the opinion in response
16 to your previous question, which was that in fact we
17 perceive in causal thinking that the spectrum has a left
18 side to it. And that is in fact where I am to be found
19 these days.

20 BY MS. DUSMAN:

21 Q. I understand.

22 At line 41 you go on to state, "I believe that the
23 230 kV transmission lines poses no threat of cancer to
24 persons in its vicinity. What I want to know is are you
25 as certain of that belief as you are of your opinion that

1 E/MFs have not been demonstrated to be carcinogenic?

2 A. Yes. I believe it is much more likely than not
3 that the line poses no threat of cancer.

4 MS. DUSMAN: Nothing further, Your Honor, except to
5 reserve the right to cross-examine on the additional
6 materials.

7 JUDGE SMOLEN: Do you want to redirect now if you
8 have any, or do you want to wait?

9 MR. WATSON: I think I would rather wait.

10 JUDGE SMOLEN: All right. Let's take a break now.
11 I don't know what time Mr. Sugarman is going to arrive.
12 He said it was going to be before 11. He was second on
13 the list. That was the message that he left in the
14 office.

15 So let's take a general break. Let's report back
16 in 15 minutes and see what happens at that time.

17 (Recess.)

18 JUDGE SMOLEN: Back on the record.

19 Mr. Sugarman, cross-examination.

20 MR. SUGARMAN: Thank you.

21 CROSS-EXAMINATION

22 BY MR. SUGARMAN:

23 Q. Dr. Cole, I am going to try and avoid
24 repetition. I have some idea of your prior
25 cross-examination so I will try to avoid repetition of

1 testimony you have already given.

2 As I understand it, you interpret -- and by
3 interpret, I mean understand -- the Wertheimer, Leeper
4 and Savitz and Matanoski and Peters work to show some
5 larger or smaller amount of association between wiring
6 codes and some disease or diseases, is that correct?

7 A. No, sir. I think it would be useful to exclude
8 Matanoski from that series, since the Matanoski studies
9 are of adults and don't relate to wire code whereas the
10 other three relate to children and do relate to wire
11 code.

12 Q. All right. And Matanoski shows some exposure
13 from line work appears to result -- and this is without
14 evaluating it, but based on its face -- result in
15 different patterns of cancer risk?

16 (Pause.)

17 Q. Is that correct? That is what the report says?

18 A. I'm sorry. I would not agree with that
19 statement as it was made and I don't know exactly what it
20 is that you are reading.

21 Q. I am reading from the report, Cancer Incidence
22 in New York Telephone Workers, Matanoski, et al. dated
23 November, 1989. And it states, quote, these data suggest
24 that different exposures from line work appear to result
25 in different patterns of cancer risk."

1 MR. WATSON: Are you offering this to refresh his
2 recollection as to what this study says?

3 MR. SUGARMAN: Well, he said he didn't know what I
4 was referring to. So I'm offering it to him to explain
5 what I was referring to.

6 I thought he testified that he was aware, in fact
7 he was on the advisory committee of the Matanoski, page
8 26, lines three through 20. So I assume he knew about --
9 he characterized it so I assumed he knew about it.

10 MR. WATSON: And the pending question is whether
11 this document says what you read?

12 MR. SUGARMAN: Yes. I want to make sure we all
13 understand the study the same way. I am not asking him
14 to agree with that conclusion but I am asking him if that
15 is his understanding of what the conclusion says.

16 MR. WATSON: I am not sure that is a conclusion.

17 MR. SUGARMAN: Well, that is why I am asking him
18 the question. He is the witness.

19 A. If I may simply make a comment or
20 clarification, Matanoski has done two studies.

21 BY MR. SUGARMAN:

22 Q. Right. You so testified in your direct.

23 A. I just needed a moment to understand which one
24 you are talking about and which particular comment.

25 Now, could you direct me to the comment that you

1 asked me about?

2 Q. Well, you are calling it a comment. Let's
3 avoid argumentative characterization and call it a
4 statement. It says here these data -- this is the next
5 to last sentence of the report. It says these data
6 suggest that different exposures from line work appear to
7 result in different patterns of cancer risk. The last
8 sentence, then, is the data are limited to New York
9 because at this time New York was the only feasible
10 population to study.

11 And I am asking you -- I wasn't showing you
12 originally -- the original question to you was is it your
13 understanding of Matanoski's report that it did purport
14 to find some association between exposure to
15 electromagnetic fields and cancer risk?

16 MR. WATSON: Your Honor, could we have at least an
17 identification of which Matanoski study?

18 JUDGE SMOLEN: Which one?

19 MR. SUGARMAN: The one on New York telephone line
20 workers.

21 JUDGE SMOLEN: Go ahead.

22 A. Mr. Sugarman, you have impressed upon me the
23 importance of the words, and you asked me about a report.
24 This is not a report. This is a representation of a
25 poster session.

1 Q. Of what?

2 A. A poster session.

3 Q. Does the representation of the post-assessment
4 by Matanoski as you understand it purport to find some
5 association between exposure to E/MF and cancer risk?

6 A. Does it purport to find such a thing?

7 Q. Right.

8 A. Yes. It purports to.

9 Q. All right. Thank you. May I take that?

10 A. Yes.

11 Q. Thank you.

12 Now, we have Wertheimer, Leeper, we have Savitz, we
13 have Peters, we have Matanoski, all of which purport to
14 find some association between some electrical fields --
15 magnetic fields, I should say -- and cancer, albeit in
16 different populations.

17 MR. WATSON: Objection, Your Honor. Counsel is
18 testifying. That's not established, that's Counsel's
19 characterization.

20 MR. SUGARMAN: That is what he said.

21 MR. WATSON: He only answered as to Matanoski.
22 Counsel then proceeded in his question to list several
23 others.

24 MR. SUGARMAN: Prior to that he testified as to the
25 other three.

1 JUDGE SMOLEN: I will sustain the objection. Ask
2 the next question.

3 BY MR. SUGARMAN:

4 Q. In the face of your testimony that you agree,
5 as you testified a few minutes ago, with respect to
6 Wertheimer, Savitz and Peters, and now as to Matanoski,
7 that these studies purport to find some association, is
8 it your testimony that all these studies should be
9 totally disregarded?

10 MR. WATSON: Objection, Your Honor. Again it's the
11 same thing you ruled on before. Counsel is
12 characterizing the prior testimony. He's doing so
13 incorrectly. This witness did not answer that question.
14 The witness answered a question only as to Matanoski, and
15 in those prior studies the purported relationship was
16 only with wire code and not with measured magnetic
17 fields.

18 MR. SUGARMAN: I didn't say measured.

19 MR. WATSON: I know you didn't.

20 MR. SUGARMAN: I said exposure to E/MF.

21 MR. WATSON: And it is not necessarily exposure to
22 E/MF, Your Honor.

23 JUDGE SMOLEN: I sustain the objection. Ask a
24 question --

25 MR. SUGARMAN: He can answer that. That is

1 cross-examination. He can explain.

2 JUDGE SMOLEN: I sustained the objection.

3 MR. SUGARMAN: What I am saying is he testified --
4 can we have his answer read back as to the other three
5 studies?

6 JUDGE SMOLEN: The other three? Not Matanoski.
7 Matanoski said it was a post-summary.

8 MR. SUGARMAN: Yes. But as to the other three.

9 JUDGE SMOLEN: You referred to it in your question
10 as his testimony as to the study.

11 MR. SUGARMAN: Well, isn't this a post whatever it
12 is document relating to a study?

13 JUDGE SMOLEN: Well, you have to ask that of the
14 witness.

15 MR. WATSON: I might be able to help in one sense.
16 I think there is a word misunderstanding. As I
17 understand it, it was a poster, p-o-s-t-e-r,
18 presentation --

19 JUDGE SMOLEN: That's what I thought the witness
20 said.

21 MR. WATSON: -- rather than a post-presentation,
22 which is if somebody puts a poster up on a board.

23 JUDGE SMOLEN: We are wasting time. Ask one
24 question at a time rather than lumping all the studies
25 together.

1 MR. SUGARMAN: But, Your Honor, my point is
2 precisely that the studies all have to be lumped, and the
3 question is when you lump all these studies can you just
4 blithely say I see nothing that bothers me.

5 JUDGE SMOLEN: Ask the question again.

6 MR. SUGARMAN: Okay.

7 BY MR. SUGARMAN:

8 Q. You agreed with me a few minutes ago, or you
9 corrected me and qualified your agreement with me a few
10 minutes ago as to the three studies and said they were
11 wire code and they were children, childhood cancer, and
12 they did show an association.

13 JUDGE SMOLEN: Wait a minute. Let him answer that
14 question. Let him answer that question.

15 Make that a question. Was that your prior
16 testimony?

17 THE WITNESS: I don't know if it was my prior
18 testimony or not but I will assert now that the three
19 other studies, that is, the original Wertheimer study,
20 the Savitz study and the Peters study, both contain
21 information relevant to a link between wire code and
22 either childhood cancer or more specifically leukemia,
23 and in each of those three studies there is some positive
24 result. There are also some negative results.

25 BY MR. SUGARMAN:

1 Q. Now, there are some positive results there.
2 There are some positive results in the Matanoski whatever
3 you call it. And my question to you is is it your
4 testimony that all four of those can be safely
5 disregarded?

6 A. Mr. Sugarman, in attempting to form an opinion
7 and continually update my opinion as to whether or not
8 electromagnetic fields cause cancer in human beings, I
9 have tried to rely on the totality of the epidemiologic
10 literature, which now numbers more than 50 studies. It
11 is not to me a question of whether or not we pick out
12 four studies and say we can ignore them or interpret them
13 one way or the other. The most important characteristic
14 of epidemiologic data is it is subject to error and
15 chance and the need to interpret it in a pattern. So I
16 neither dismiss these studies nor do I form my opinion
17 solely on the basis of them.

18 I want to point out, too, that every one of those
19 studies includes substantial negative information with
20 the exception of the original Wertheimer study. They are
21 not clear-cut positive. As a matter of fact, the Peters
22 study is in my judgement an overwhelmingly negative study.

23 Q. By overwhelmingly negative, you mean very few
24 of the exposed population show a manifestation?

25 A. No. I mean that the Peters study used

1 measurements of magnetic fields in the homes of the
2 subjects. It's the first study to have done so in any
3 substantial proportion of people. With regard to the
4 measurements, the Peters study is negative. There is no
5 link whatever between childhood leukemia and in-home
6 measurements of magnetic fields.

7 Furthermore, with regard to the wire code, the
8 study is substantially weaker than the Savitz study and
9 also much less consistent. A more subtle point in the
10 Savitz study -- in the Peters study -- with regard to
11 wire code is that all of the effect of the wire code is
12 restricted to the highest exposure category. And even is
13 there it undergoes a major reduction when it is corrected
14 for other factors that could influence risk.

15 Both of these things, that is, the fact that the
16 long-term time trend is down and the fact that correction
17 for extraneous factors reduces the effect, strongly
18 suggest that you are not dealing here with a true causal
19 factor. So it is negative as far as I am concerned with
20 regard to the wire code, at least at the interpretative
21 stage if not the literal data stage, and it is clearly
22 and unequivocally negative with regard to the magnetic
23 field measurements.

24 Q. The magnetic field measurements, were they of
25 proximity to a transmission line?

1 A. The measurements were made in the homes of the
2 children.

3 Q. Right. But was there a proximity to a
4 transmission line?

5 A. There may have been in some homes. In others I
6 would assume there was not.

7 Q. It's not reported, is that right?

8 A. The data are not provided for the location of
9 the measuring device and the location of all facilities
10 that might influence it for everyone of the nearly 500
11 children in the study. Statements are made to the effect
12 that the measurements were taken under some conditions
13 and those conditions are specified. For example, one of
14 the conditions was in the middle of the child's bedroom,
15 if at all possible.

16 Q. But you are saying that as far as you know
17 there were no transmission lines involved?

18 A. No, I didn't say that.

19 Q. Oh, okay.

20 A. What I did say was I don't know whether or not
21 there were any transmission lines involved.

22 Q. What were the magnetic field measurements, what
23 was the range of them?

24 A. The magnetic field measurement range is given
25 in the actual paper. May I consult it?

1 Q. Sure. I sorry. I thought you had a copy of
2 it?

3 A. I have it.

4 Q. Was it Table 3?

5 (Witness perusing document.)

6 A. Table 3 gives some measures of central
7 tendency, averages and the like, of the measurements made
8 in the home of the children with leukemia and the
9 children who don't have leukemia, and you see, for
10 example --

11 Q. But none of them are of a level sufficient to
12 qualify as being affected by transmission lines, is that
13 correct?

14 A. Well, if I could, I would like to finish my
15 answer to the previous question since you asked me to
16 characterize --

17 Q. No, I didn't. All I asked you was whether --

18 JUDGE SMOLEN: The witness can finish the answer to
19 the previous question.

20 MR. SUGARMAN: He wants to talk about the results
21 of the study and I am not into that at this point. I
22 just want to know if they show exposure to transmission
23 line milligauss --

24 JUDGE SMOLEN: I understand.

25 You can finish your previous answer.

1 A. Well, I will simply state, rather than reading
2 any numbers, that the information in Table 3 shows that
3 the cases and controls have essentially identical
4 readings with regard to their magnetic field exposures.

5 BY MR. SUGARMAN:

6 Q. And there is nothing in there that shows any
7 elevations that you would expect near a transmission
8 line, is that right?

9 A. Mr. Sugarman, I cannot answer that question.

10 Q. Okay. Fine. You can't answer it.

11 A. I don't know what level one would expect to see
12 in a home that was relatively close to a line of what
13 relative size operating under what relative load
14 conditions.

15 Q. So you don't know.

16 Then in your testimony at lines 36 to 42 on page 28
17 you discuss the exposure of people proximate to this
18 transmission line. Did you have any information as to
19 the elevated levels of milligauss associated with this
20 transmission line?

21 A. I don't have any information as to what the
22 readings would be at the edge of the right-of-way or
23 within the right-of-way of this line under various load
24 conditions, no.

25 Q. Do you think that might have anything to do

1 with an increased risk of cancer?

2 A. No, I don't.

3 Q. So it wouldn't matter to you?

4 A. It's not that it wouldn't matter. It's that
5 the 230 kV line is not going to produce enormous fields.
6 The fields are going to be similar to those produced by
7 other lines. And in --

8 Q. What kind of lines?

9 A. And in any case, if I may complete the answer,
10 there has been no link established between magnetic field
11 exposure and cancer. So unless you were going to talk
12 about something that was of a completely different nature
13 I will have to stay with my response.

14 Q. You say there has been no link established but
15 you acknowledge that studies have purported to find such
16 a link?

17 A. That's correct.

18 Q. But in your view they don't establish it?

19 A. Mr. Sugarman, I will contend that not only is
20 it true in my view that there is no established
21 cause-effect relationship between magnetic field exposure
22 and cancer, but I will further contend that it is not
23 true in anybody's view. At least I know of no one who
24 holds that view, and I think I know the opinions of quite
25 a few people.

1 Q. Your testimony is limited to the effect of
2 magnetic fields on cancer, is that correct?

3 A. Yes.

4 Q. It does not deal with any other health
5 conditions?

6 A. That's correct.

7 Q. Have you seen the studies -- I say studies --
8 the letters in Lancet -- are you familiar with Lancet?

9 A. I read The Lancet, yes.

10 Q. Have you seen the two letters in Lancet in
11 December, 1990, and March, 1991, relating to findings of
12 health effects of electromagnetic fields in Sweden?

13 A. Yes, I have.

14 Q. And do those relate to cancer?

15 A. Yes, they do.

16 Q. And do you regard them as epidemiological
17 information of value?

18 A. No, I don't.

19 Q. Why not?

20 A. Mr. Sugarman, it is my policy not to
21 incorporate into my overall assessment of a cause-effect
22 relationship information which is published in letter
23 form or which appears as posters at scientific meetings
24 unless there is something most exceptional about the
25 letter.

1 Q. What do you mean by most exceptional?

2 A. Something that is to the effect that it is so
3 lengthy and detailed that it represents what is in effect
4 a published study. That is quite rare.

5 Q. Are you aware that Einstein's theory of
6 relativity was first published as a letter?

7 A. I have heard it said that Einstein's theory of
8 relativity was published in every form conceivable to man
9 but I have never actually seen the document. So no, I
10 don't.

11 Q. Hawking's book has a picture of it.

12 MR. WATSON: Objection, Your Honor. Move to
13 strike. Counsel is testifying.

14 JUDGE SMOLEN: Sustained.

15 BY MR. SUGARMAN:

16 Q. Dr. Cole, have you done any epidemiological
17 studies related to electromagnetic fields?

18 A. I am just beginning a study at this time. I
19 have not completed any study in this area.

20 Q. What exposure are you going to use?

21 A. The study that I am going to be doing will be
22 based on a detailed occupational history.

23 Q. And how are you going to characterize exposure?

24 A. It will be described in terms of probable,
25 possible and likely, based on the occupational history.

1 Q. But to what? Exposure to -- I mean, we all
2 have exposure to E/MF in every house as the control in
3 the Peters study shows. How are you going to
4 characterize the exposure. Exposure to what?

5 A. It is to magnetic fields.

6 Q. You are not going to distinguish between people
7 who are exposed to it in every home in the country versus
8 people who are exposed to a high power transmission line?

9 A. My study is a case-control study which focuses
10 on the workplace.

11 Q. How are you going to characterize the
12 workplace?

13 A. By going through the records of each individual
14 human being who is incorporated into the study.

15 Q. But are you going to characterize the amount of
16 milligauss to which the person is exposed?

17 A. No. That information will not be available.

18 Q. You don't think it has any relevance?

19 A. I think it would be very relevant. I would be
20 delighted to have it. But I know of no way to obtain it.

21 Q. Didn't Matanoski obtain it for his New York
22 telephone study?

23 A. She obtained some information for some of the
24 workers through an extremely expensive approach, that is,
25 she tried to simulate circumstances that had existed 20

1 or 30 years ago. But I would like to point out that
2 Matanoski's study and mine are fundamentally different.
3 Her study is a follow-up study and my study is a
4 case-control study. It is as a practical matter
5 infeasible, maybe even impossible, to get exposure
6 assessments for my people.

7 Q. What kind of study was Peters' study?

8 A. The Peters study is a case-control study.

9 Q. What is the meaning of the exposure, Table 3?
10 You say it is almost impossible to get exposure levels.
11 Table 3, exposure, arithmetic mean, median, geometric
12 mean. Isn't that exposure?

13 A. This is exposure and it is a case-control
14 study.

15 Q. And it is?

16 A. It is indeed.

17 Q. So why are you saying it is impossible for you
18 to get it?

19 A. It's a completely different setting. These are
20 children and these are residential studies. Even so, he
21 failed to get measurements on the vast proportion, I
22 think 30 percent or so.

23 In my study it relates to the workplace some 10 to
24 30 years ago. It is just not practical. The workplace
25 is gone now, it is changed. In Peters' study he went to

1 the home in the present time and made the assumption that
2 it would be similar to the home of five years ago. It is
3 a somewhat different circumstance.

4 Q. You say you would like to have the information
5 as to the exposure levels. You would be delighted to
6 have it, I think was your phrase. How can you form an
7 opinion as to the dangers of this transmission line
8 without having the exposures?

9 A. I'm sorry, Mr. Sugarman. What transmission
10 line?

11 Q. The proposed Woodbourne-Heaton 230 kV
12 transmission line.

13 A. Oh. I have tried to arrive at an overall
14 position with regard to the likelihood that E/MF causes
15 cancer. I have arrived at the position that this is not
16 an established cause of human cancer. As a matter of
17 fact, I went a little bit further and said it is very
18 unlikely that it will ever been shown but that is perhaps
19 neither here nor there.

20 Given that I hold the position that E/MF is not an
21 established cause of cancer in human beings, the question
22 of this particular line, the Woodbourne-Heaton line, fits
23 right into the overall pattern that this line, like other
24 line, is not a cause of cancer.

25 Q. So let me ask you this, then. Do you have any

1 position of agreement or disagreement with PECO's
2 recommendation that people consider prudent avoidance of
3 exposure to E/MF?

4 A. The question of prudent avoidance, as I have
5 understood it, is either a question of individual
6 behavior or perhaps could be extended to public policy.
7 And this is an area where I just don't have any
8 expertise. I do restrict myself to the study of the
9 causes of cancer among human beings.

10 Q. I'm afraid I don't understand. If you were
11 giving this Commission an opinion as to whether they
12 should advise people to forget about prudent avoidance
13 you would say you have no opinion?

14 A. If I were asked by the Commission to say
15 whether or not people should consider or not consider
16 prudent avoidance -- is that the question?

17 Q. Yes.

18 A. My first response to them would be exactly what
19 I responded to you, that I am a cancer epidemiologist,
20 not a public health policymaker.

21 Q. Let's say we go one step further and we say we
22 are concerned about the -- we don't want people to be
23 exposed to any health risks. From your vantage point
24 should we encourage prudent avoidance or would we be
25 wasting our time and money?

1 MR. WATSON: Objection, Your Honor. He has already
2 testified it is beyond his field. It's not covered in
3 his testimony.

4 JUDGE SMOLEN: It's the third time, the same
5 question.

6 MR. SUGARMAN: Sustained?

7 JUDGE SMOLEN: Sustained, yes.

8 MR. SUGARMAN: No further questions.

9 I just wanted to make sure that I had not missed a
10 chance to get an answer by not asking the question
11 another way.

12 JUDGE SMOLEN: Well, you are practicing law the way
13 it is supposed to be practiced.

14 MR. WATSON: Your Honor, we have the pages of the
15 transcript that the OCA has asked for this morning.

16 (Document handed to Ms. Dusman.)

17 MS. DUSMAN: Thank you.

18 JUDGE SMOLEN: Do you want to break now to take a
19 look at those pages?

20 MS. DUSMAN: I would appreciate that, Your Honor.

21 JUDGE SMOLEN: Let's take a ten minute break. When
22 we come back on the record, without looking at that, do
23 you have any other questions based on your reservation
24 this morning?

25 MS. DUSMAN: I may, Your Honor, but it will be

1 brief if at all.

2 JUDGE SMOLEN: How about redirect? Are you going
3 to have any or not?

4 MR. WATSON: Unlikely, Your Honor.

5 JUDGE SMOLEN: All right. Ten minute break.

6 (Recess.)

7 JUDGE SMOLEN: Back on the record.

8 Further cross?

9 MS. DUSMAN: Just a couple of questions, Your
10 Honor.

11 JUDGE SMOLEN: Go ahead. Cross-examination.

12 FURTHER CROSS-EXAMINATION

13 BY MS. DUSMAN:

14 Q. Dr. Cole, having looked at what we have been
15 referring to as the Peters study, which was authored
16 primarily by London, is it accurate that you stated in
17 response to a question by Mr. Sugarman that there is no
18 information in this study as to distance to transmission
19 lines?

20 A. I think what I tried to say was that
21 information is not provided with regard to specific
22 individuals about the distance between, say, their home
23 and the transmission lines. It is true that if a home
24 was located near a transmission line that that would
25 influence the wire code that was assigned to the

1 particular residence.

2 So the people who made the wire code assignment
3 would use the fact of presence of a transmission line or
4 a distribution line to influence the wire code. But we
5 are not provided with that information in any way that we
6 can disentangle it from the wire code collectively.

7 MS. McCLOSKEY: May we have just a minute, Your
8 Honor?

9 JUDGE SMOLEN: Yes.

10 (Pause.)

11 BY MS. DUSMAN:

12 Q. Just so I understand your answer, Dr. Cole,
13 would you explain your understanding of what wire code
14 is?

15 A. Yes. First I will say that there are several
16 different wire codes that are in use. This particular
17 study used both the so-called Wertheimer-Leeper wire
18 code, which they refer to as the Denver-Wertheimer-Leeper
19 wire code, and they also the Caune code but they don't
20 present results for that. They say that the results were
21 less positive. So perhaps I should talk about the
22 Wertheimer-Leeper wire code.

23 As originally used by Wertheimer it was a four
24 point scale from very low current, to low current, to
25 high current, to very high current configuration, and she

1 would assign a home to one of these four categories
2 depending upon what she perceived as the service drop to
3 the home and any structures in the immediate area of the
4 home.

5 In the Savitz study this was refined to a five
6 category scale, and I believe that is what they have used
7 in this paper too, a five point scale.

8 Q. Is it accurate to state that this study
9 supports -- the study that we have been referring to,
10 which is the Peters study, primarily authored by London,
11 just published -- that it supports an association between
12 childhood leukemia risk and wiring configuration or
13 wiring code as you just described it?

14 A. If I don't --.

15 MS. DUSMAN: Your Honor, if the witness would
16 please answer yes or no first and then explain if
17 necessary.

18 JUDGE SMOLEN: Well, if he can answer yes or no.

19 THE WITNESS: This particular question I cannot
20 answer yes or no. I think both of those answers are
21 wrong.

22 BY MS. DUSMAN:

23 Q. Would you direct your attention, then, to the
24 -- you have the study before you?

25 A. Yes, Ma'am.

1 Q. Would you direct your attention to the final
2 sentence in the abstract on the first page of the article
3 that you provided us and read it into the record?

4 A. "Our results support an association between
5 childhood leukemia risk and wiring configuration but not
6 direct measurements of electric and magnetic fields."

7 MS. DUSMAN: Nothing further, Your Honor.

8 JUDGE SMOLEN: All right.

9 MR. SUGARMAN: I just have one question.

10 FURTHER CROSS-EXAMINATION

11 BY MR. SUGARMAN:

12 Q. As you said, they have a category or several
13 categories, one of which is very high. Page 929, the
14 first paragraph, and then Table 7. Is that right?

15 (Pause.)

16 JUDGE SMOLEN: Do we have a question?

17 MR. SUGARMAN: Yes.

18 BY MR. SUGARMAN:

19 Q. Is that right? Am I reading it right?

20 A. Well, Table 7 does include reference to the
21 wire code and I think you said 929, first paragraph?

22 Q. Right.

23 A. It makes a reference to the wire code, yes.

24 Q. On page 928 they say they sketched overhead
25 electric transmission facilities. The last paragraph.

1 A. Yes, that's correct.

2 Q. On 929, in the right-hand column, the second
3 paragraph, it says, "Three highly influential 24 hour
4 magnetic field measurements (two cases and one control)
5 were excluded from all analyses involving continuous
6 scale data but were included in analyses involving
7 ordered categories." Do you understand what that means?

8 A. Yes, sir.

9 Q. What does it mean?

10 A. It means this: there were three readings, as it
11 says, two for cases and one for control, which were
12 extremely high. And that is what they mean by highly
13 influential. That is, the inclusion of those individual
14 measurements would pull up the average in and of
15 themselves.

16 So they they excluded those measurements when they
17 made individual assessments of means and averages and the
18 like so that the measure of central tendency would not be
19 pulled inordinately by any one or two measurements.
20 However, when they use categorization of the field
21 strength like low, medium and high, then they put them
22 into the high category.

23 That's what it means.

24 Q. Isn't that apples and oranges, putting them in
25 and taking them out?

1 A. No.

2 Q. Shouldn't they have been consistent and
3 excluded them altogether or included them altogether?

4 A. What they have done is commonly done and widely
5 accepted as long as it is pointed out that it is done,
6 and it is a common practice in what they call continuous
7 scale or what I might call ratio scale variables to
8 exclude what are sometimes referred to as outliers at
9 either end. It is a very common practice.

10 Q. And is it true that in Table 7, if you believe
11 it, that the higher the exposure category the more -- the
12 exposure category in terms of exposure based on the wire
13 configuration classification -- that the higher the
14 cancer risk or leukemia risk?

15 A. Again, Mr. Sugarman, it is true and it is not
16 true. That is, there is a trend in the direction that
17 you have just said.

18 Q. Right.

19 A. There is a trend towards increased risk with
20 higher wire code.

21 Q. Right.

22 A. But as I mentioned in response to a prior
23 question, there are two important factors that have to be
24 borne in mind before one attempts to interpret that and
25 come to the interpretation that there is such a

1 relationship.

2 I think that that interpretation is best shown in
3 Table 9, in the bottom half of it, where we have now a
4 much more sophisticated presentation of the same
5 information. That is, Table 7 represents what we might
6 term the crude data and Table 9 the refined data. And
7 what we see as we look at Table 9 is that in the second
8 category of wire code the risk is actually estimated to
9 be 20 percent lower than it is in the lowest. So we
10 don't have what we could call here a consistent dose
11 response.

12 The second thing is the comparison of the 2.15 with
13 the 1.73. This is quite important because it is only in
14 that category, that is, the very high category, that
15 there is really any meaningful association. So the
16 question becomes what is the interpretation to be placed
17 on that change, that is, from 2.15 to 1.73. The answer
18 is that when they have corrected for other factors that
19 could influence leukemia risk the apparent association
20 with very high current configuration wiring went down by
21 37 percent.

22 I point out --

23 Q. In each category --

24 JUDGE SMOLEN: Let him finish the answer.

25 MR. SUGARMAN: I'm sorry.

1 A. Please let me finish. I am speaking only of
2 the category very high. And I want to point out that the
3 change from 2.15 to 1.73, while it may look trivial, has
4 to be appreciated to be a 37 percent reduction, the
5 reason being that you subtract out the one, which is
6 baseline.

7 Now, the control that they have influenced to bring
8 about this change is imperfect. It is limited by the
9 data that they have and statistical devices. It implies,
10 though, such a large change, that if they had better data
11 the reduction would be even greater.

12 In my judgement, then, while these data set could
13 be described as saying that they do show some
14 relationship between wire code and leukemia risk, they
15 are very unimpressive to that effect.

16 BY MR. SUGARMAN:

17 Q. I understand.

18 Just so we are clear, the category very high
19 relates to what level of exposure?

20 A. I believe that comparison is actually given in
21 the paper.

22 Q. I think so.

23 A. There is a table somewhere in here that shows
24 the average milligauss reading in the homes in any
25 particular wire configuration category. At least that is

1 my recollection.

2 (Witness perusing document.)

3 A. Yes, here we are. Table 4 says that the mean
4 reading, that is the average, an arithmetic average, in
5 the very high current configuration homes is 1.15
6 milligauss.

7 MR. SUGARMAN: Thank you.

8 I don't have any further questions. Thank you very
9 much.

10 JUDGE SMOLEN: Anything?

11 MR. WATSON: No, Your Honor.

12 JUDGE SMOLEN: No further questions, no redirect.

13 The witness is excused. Thank you very much for
14 appearing and testifying, sir.

15 (Witness excused.)

16 JUDGE SMOLEN: That apparently concludes today's
17 session and we will reconvene on Friday morning.

18 The hearing is adjourned.

19 (Whereupon, at 12:06 p.m., the hearing was
20 adjourned, to be reconvened at 10:00 a.m., on Friday,
21 November 22, 1991, in Philadelphia, Pennsylvania.)
22
23
24
25

C E R T I F I C A T E

1
2 I hereby certify, as the stenographic reporter,
3 that the foregoing proceedings were taken
4 stenographically by me and thereafter reduced to
5 typewriting by me or under my direction; and that this
6 transcript is a true and accurate record to the best of
7 my ability.
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Philadelphia Electric Company Rebuttal Statement No. 2

A-110550F055

11/20/91

Phila. PD RGS

BEFORE THE
PENNSYLVANIA PUBLIC UTILITY COMMISSION

REBUTTAL TESTIMONY
OF
DR. PHILIP COLE

RECEIVED

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Information Control Division

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REBUTTAL TESTIMONY OF DR. PHILIP COLE

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- 1 I Background and Qualifications
2
3 Q. Please state your name and business address.
4
5 A. Philip Cole, Department of Epidemiology, School of Public Health, University
6 of Alabama, Room 203 Tidwell Hall, University Station, Birmingham, AL
7 35294.
8
9
10 Q. What is your occupation?
11
12 A. I am a professor and researcher in epidemiology and a medical doctor.
13
14
15 Q. Dr. Cole, what is epidemiology?
16
17 A. Epidemiology is the scientific observation of human beings for the purpose of
18 identifying the environmental causes of their diseases.
19
20
21 Q. Where are you employed?
22
23 A. I am Professor of Epidemiology and Chairman of the Department of
24 Epidemiology at the School of Public Health of the University of Alabama in
25 Birmingham.
26
27
28 Q. Do you hold any other positions at the University of Alabama?
29
30 A. Yes, I am the Associate Director for Epidemiology at the Comprehensive
31 Cancer Center at the University.
32
33
34 Q. Are you testifying today on behalf of the University of Alabama or any other
35 medical or scientific institution?
36
37 A. No. I am testifying in my personal capacity as a researcher in epidemiology
38 and a medical doctor.
39
40
41 Q. Dr. Cole, would you describe your educational background for us?
42

- 1 A. I received a Bachelor's degree from Michigan State University in 1960. My
2 medical degree, which I received in 1965, is from the University of Vermont.
3 After receiving my medical degree, I entered Harvard University, where I
4 received a Master's degree and a Doctor of Public Health (Dr. P.H.) in
5 Epidemiology.
6
7
- 8 Q. As a medical doctor, are you board certified in any medical specialty?
9
- 10 A. Yes, I am board certified in Preventive Medicine.
11
12
- 13 Q. Are you also licensed to practice medicine?
14
- 15 A. Yes, in the state of Alabama.
16
17
- 18 Q. Dr. Cole, could you give us a brief description of your professional career after
19 you received your Dr. P.H. from Harvard?
20
- 21 A. After receiving my Dr. P.H. from Harvard, I taught in the Department of
22 Epidemiology at that institution and conducted epidemiologic research. I
23 remained on the faculty at Harvard for nearly a decade and became a full
24 professor. I then left Harvard to chair the Department of Epidemiology at the
25 University of Alabama's School of Public Health, where I have remained as
26 Chairman and Professor.
27
28
- 29 Q. Could you give us some examples of epidemiologic courses you taught at
30 Harvard and the University of Alabama?
31
- 32 A. Examples of epidemiologic courses I have taught include the Epidemiology of
33 Cancer, Advanced Epidemiologic Methods, Principles of Epidemiologic
34 Research, and Epidemiology of Neoplastic Diseases.
35
36
- 37 Q. In addition to Harvard and the University of Alabama, have you taught in
38 any other programs?
39
- 40 A. Yes, I have taught in several other graduate programs, as well as at the
41 International Agency for Research on Cancer ("IARC").
42

- 1 Q. What is the International Agency for Research on Cancer?
2
- 3 A. IARC is an arm of the World Health Organization dedicated to research on
4 cancer issues. I taught a course in Cancer Epidemiology at IARC.
5
6
- 7 Q. Are you currently involved in any epidemiologic research?
8
- 9 A. Yes, I am currently conducting several research projects in cancer
10 epidemiology. Two of these research projects are funded by the National
11 Cancer Institute. One is a study of the occupational causes of cancer,
12 including breast cancer. The other is a study of the causes of cancer of
13 esophagus in mainland China.
14
15
- 16 Q. Does your epidemiologic research have a particular area of emphasis?
17
- 18 A. Yes, my research for the past 25 years has focused on the epidemiology of
19 cancer and on the relevant methodology for conducting such research.
20
21
- 22 Q. Have you published any of your research in scientific journals?
23
- 24 A. Yes, I have published over 130 articles on epidemiology, nearly all of which
25 involved cancer epidemiology and the methodology of cancer epidemiology.
26
27
- 28 Q. What are some of the scientific journals in which your epidemiologic
29 research has been published?
30
- 31 A. The scientific journals in which my work has been published include the
32 *American Journal of Epidemiology*, the *New England Journal of Medicine*,
33 the *Journal of the National Cancer Institute*, the *American Journal of Public*
34 *Health*, and *Cancer*.
35
36
- 37 Q. Have you served as a scientific reviewer or editor for any scientific journals?
38
- 39 A. Yes. I have been an editor or on the editorial boards of several journals,
40 including the *American Journal of Epidemiology* and *Cancer Research*.
41
42

1 Q. Have you been involved with any other professional activities that involve
2 the review of epidemiologic or other research?
3

4 A. Yes. For example, I served on the Board of Scientific Counselors for the
5 Division of Cancer Cause and Prevention at the National Cancer Institute. I
6 also served as Subcommittee Chairman in connection with the National
7 Cancer Institute's National Planning Effort for Cancer Prevention and
8 Control, the purpose of which was to identify all known causes of cancer as
9 the first step to reducing cancer by 50 percent by the year 2000.
10

11
12 Q. Have you received any honors or awards in your professional career?
13

14
15 A. Yes. I was the John Rankin Visiting Professor of Occupational and
16 Preventive Medicine at the University of Wisconsin at Madison, and the
17 Eleanor Leader Memorial Lecturer at the University of Toronto. Among the
18 awards I have received are the American Cancer Society Faculty Research
19 award, the Kammer Merit in Authorship award from the American
20 Occupational Medical Association, and the French Cancer Society's Grand Prix
21 Lacassagne for cancer research.
22

23
24 Q. What is the Grand Prix Lacassagne?
25

26 A. The Grand Prix Lacassagne is an award given by the French Cancer Society for
27 excellence in cancer research. I was jointly given this award with co-
28 researchers in Boston, Greece, and Australia for research on the causes of
29 breast cancer.
30

31
32 Q. Have you ever been asked to provide advice on cancer epidemiology matters
33 to a governmental body or institution?
34

35 A. In addition to my work with the National Cancer Institute, I testified before a
36 Committee of the United States House of Representatives in 1987 on electric
37 and magnetic field ("EMF") health issues, focusing particularly on the EMF
38 cancer epidemiology research. I have also advised the State of Maine and the
39 State of Vermont on electric and magnetic field health issues.
40

41
42 Q. Dr. Cole, what were you asked to do in connection with this case?

1
2 A. I was asked to review the epidemiologic literature concerning power
3 frequency electric and/or magnetic fields and to prepare testimony evaluating
4 this literature. I was also asked to offer my professional opinion as to
5 whether the electric and/or magnetic fields associated with the Woodbourne-
6 Heaton 230 kV line will cause cancer.
7

8
9 II. Background on the Science of Epidemiology
10

11
12 Q. Before discussing the epidemiological studies on EMF and cancer, please
13 describe the major strength and limitation of epidemiology as a science.
14

15 A. Epidemiology is the study of humans in their everyday environments, and
16 the major strength of epidemiology is that it pertains directly to human
17 beings. As such, facts established through epidemiologic research have direct
18 implications for public health strategies, unlike most laboratory research. The
19 great limitation of epidemiology is that it is a non-experimental science and,
20 consequently, is highly susceptible to error. Both the strengths and
21 limitations of epidemiology must be recognized when conducting an
22 evaluation of epidemiologic studies.
23

24
25 Q. What is the importance of the limitation on epidemiology you described?
26

27 A. The history of cancer epidemiology has taught us that, with rare exception,
28 only epidemiologic investigations of the highest quality have proven to be of
29 value.
30

31
32 Q. What kinds of conclusions can be reached from a body of epidemiologic
33 literature?
34

35 A. In recognition of the imprecise and error-prone nature of the science,
36 epidemiologists generally draw causal inferences only when there is a
37 reasonably large number of independent studies with consistent findings.
38 Even then, epidemiologists do not place substantial confidence in findings of
39 weak associations where the magnitude of the reported "risk" may be less
40 than epidemiologic studies can correctly identify.
41
42

- 1 Q. What is meant by the term "causal inference"?
- 2
- 3 A. Generally, the question asked in an epidemiological study is whether
- 4 exposure to some agent causes disease. "Cause" is used in a very practical
- 5 sense in epidemiology. The term causal inference means that the
- 6 epidemiologic data is sufficiently strong and consistent that it is widely
- 7 believed to be true that exposure to the agent of interest increases the risk of
- 8 disease.
- 9
- 10
- 11 Q. Can epidemiologic data establish a cause-effect relationship?
- 12
- 13 A. Yes. Epidemiology is a non-experimental science, and the researcher cannot
- 14 exercise strict controls over the study population. It is thus sometimes
- 15 perceived that epidemiology can never establish the existence of a cause-effect
- 16 relationship. As a practical matter, however, epidemiological research is a
- 17 powerful -- perhaps the most powerful -- approach that we have for
- 18 establishing the causes of disease, including cancer, in man.
- 19
- 20
- 21 Q. Can you give an example of a cause-effect relationship that was established
- 22 with epidemiological data?
- 23
- 24 A. Two commonly known cause-effect relationships that were established with
- 25 epidemiologic data are the relationship between cigarette smoking and lung
- 26 cancer, and the relationship between certain types of asbestos and
- 27 mesothelioma. These relationships were eventually confirmed by laboratory
- 28 evidence.
- 29
- 30
- 31 Q. What kind of epidemiological data is necessary to establish a cause-effect
- 32 relationship?
- 33
- 34 A. Unlike laboratory research, it is the nature of epidemiologic research to
- 35 produce a pattern of mixed results (positive and negative results) even where
- 36 there is no cause and effect relationship between the disease at issue and the
- 37 agent under investigation. In fact, in the absence of a cause-effect
- 38 relationship, epidemiologists would expect to find a large proportion of
- 39 reported positive findings due to random chance alone. This is due to the
- 40 non-experimental nature of epidemiologic research, and it underscores the
- 41 need for consistent findings in drawing causal inferences.
- 42

1 This need for consistency has been well documented in cancer epidemiology.
2 Agents that have been accepted as genuine causes of human cancer share one
3 feature in common -- from the early epidemiologic research forward there
4 was a high degree of consistency of results among independent studies and
5 evidence of relatively strong associations between exposure and cancer risk.
6 In contrast, epidemiologic studies of agents which have not been and may
7 never be accepted as causal have been characterized by inconsistent findings
8 both within and among studies and, at best, weak statistical associations.
9

10
11 Q. How do epidemiologists evaluate a body of cancer epidemiologic research?
12

13 A. Application of the principles of epidemiology to assess the carcinogenic
14 potential of an agent involves a three-step process. First, individual studies
15 are examined to assess the quality, reliability and validity of the research
16 results. Second, the studies are examined collectively, giving appropriate
17 weight to individual studies, to assess consistency and other important
18 indicia of causality. Third, the epidemiologic studies are considered in
19 context with basic science research and other available information to assess
20 the coherence or biologic credibility of causal inferences.
21

22
23 Q. You have used the term "exposure" to an agent -- what does exposure mean
24 in epidemiology?
25

26 A. Exposure means contact with the environmental agent in question.
27

28
29 Q. How is exposure determined?
30

31 A. The ideal method is direct measurement, but that is often difficult to achieve.
32

33
34 Q. When direct measures of exposure are not available, how is exposure
35 determined?
36

37 A. If exposure cannot be measured directly, then epidemiologists use some
38 substitute (sometimes called a surrogate or a proxy) as an indicator of
39 exposure.
40

41
42 Q. Could you give us some examples of surrogate measures of exposure?

- 1
2 A. Some commonly used surrogates are information from questionnaires, from
3 employment records, and from death certificates.
4
5
6 Q. What exposure data can be derived from surrogate sources?
7
8 A. Using surrogate data sources, inferences about the population's actual
9 exposure are made based upon some supposed exposure.
10
11
12 Q. What would be an example of this type of inference?
13
14 A. For example, using occupational information from a death certificate, one
15 might infer that an individual who was listed as having worked as a service
16 station employee had been exposed to gasoline fumes.
17
18
19 Q. Are there any problems with surrogate measures of exposure?
20
21 A. Yes, there can be. With surrogate measures it is often difficult to know if the
22 surrogate is a reliable predictor of actual exposure. In the example above, the
23 service station employee may only have worked at the cash register, and may
24 never have been exposed to gasoline fumes. This is one of the typical
25 problems of surrogate measures of exposure.
26
27
28 Q. Is it important to have accurate measures of exposure in epidemiologic
29 studies?
30
31 A. I think its fair to say that epidemiologic studies are only as good as their
32 assessment of exposure.
33
34
35 Q. What is the consequence of not having reliable exposure data?
36
37 A. A lack of good data on exposure can throw the results of a study into question.
38 If we are not confident about the actual exposure, it's unlikely we can have
39 confidence in the results of the study.
40
41
42

1 Q. Is evaluation of the surrogate a factor that you consider when analyzing
2 individual studies?
3

4 A. Evaluation of the surrogate is an important factor in the evaluation of
5 epidemiological studies, both individually and as a group.
6
7

8 Q. After completing an evaluation of one individual epidemiologic study, is an
9 epidemiologist in a position to say whether or not a particular agent has an
10 adverse effect on health?
11

12 A. No. Conclusions concerning the relationship of a agent to a disease rarely if
13 ever can be based on a single study.
14
15

16 Q. What else is necessary to reach a conclusion from epidemiologic research?
17

18 A. Conclusions need to take into account the results of all the relevant studies.
19 It is thus necessary to evaluate not only the strengths and weaknesses of each
20 study on its own merits, but also the strengths and weaknesses of each
21 individual study in the context of the entire body of literature.
22
23

24 Q. Do you ever completely dismiss the results of an epidemiologic study when
25 conducting this evaluation?
26

27 A. Occassionally, a study is so deficient that that it provides no useful
28 information and can be completely dismissed. More commonly, the analysis
29 of the individual study reveals that it has both strengths and limitations.
30 Some studies are so limited, either in design or in conduct, that they are
31 properly termed "hypothesis-generating" – that is, they raise a question
32 without providing useful or persuasive information about the answer to that
33 question. Such studies are given relatively little weight in the overall
34 epidemiologic analysis.
35

36 This is far different from dismissal of the study. An epidemiologic
37 evaluation that did not include analysis of substantially all of the available
38 data would be of questionable utility, and the utility of the evaluation would
39 decrease as larger amounts of data were dismissed or otherwise excluded.

1 III. Epidemiologic Research on EMF and Cancer
2
3

4 Q. Dr. Cole, how many epidemiologic studies have been published regarding
5 EMF and cancer?
6

7 A. Approximately 50 epidemiologic studies have been published which contain
8 data on EMF and cancer. These studies are known as the "primary" literature.
9

10
11 Q. Have you personally reviewed each of these studies as part of your analysis of
12 the epidemiologic literature?
13

14 A. Yes. As I stated earlier, review of the strengths and limitations of each
15 individual study is a necessary step in analysis of a body of epidemiologic
16 literature. I have therefore carefully reviewed the data, design, and
17 conclusions of each of the epidemiological studies in the primary literature.
18

19
20 Q. Did you review any literature other than the primary literature?
21

22 A. Yes. The epidemiologic literature in this area also includes commentaries on
23 the literature, review articles, and abstracts. I have reviewed all of this
24 secondary literature of which I am aware, including the Office of Technology
25 Assessment ("OTA") review, and the draft Environmental Protection Agency
26 ("EPA") report. It should be emphasized, however, that these secondary
27 sources do not contain original data, and are by and large interpretive in
28 nature. Thus, while the secondary literature can be helpful in understanding
29 the state of the scientific debate, they should not be given the same weight as
30 the primary literature.
31

32
33 Q. What kinds of epidemiological studies have been conducted regarding EMF
34 and cancer?
35

36 A. Several designs and methods have been used to study a variety of cancers and
37 measures of exposure to magnetic fields. For childhood cancer, the most
38 important studies are the residential case-control studies. Adult cancers also
39 have been examined in residential studies, using both the case-control design
40 and the retrospective follow-up ("RFU") design. There have also been
41 numerous studies of adult cancers in the occupational setting. The

1 occupational studies are case case-control studies, proportionate mortality
2 ratio ("PMR") studies, and retrospective follow-up studies.

3
4 A. The Research on EMF and Childhood Cancer

5
6 Q. Dr. Cole, please describe the "case-control" design of epidemiology studies.

7
8 A. In the case-control method, one identifies a group of people who have
9 developed a disease, such as cancer. These people are known as the "cases."
10 A second group of people, known as the "controls," is then identified. This
11 group consists of people who have not developed the disease of interest, but
12 who in other ways are similar to the cases. For example, the cases and
13 controls should be of the same age group.

14
15 Having identified these two groups, one then seeks to describe the pattern of
16 exposure to the agent of interest in each group, looking for similarities and
17 differences in that exposure.

18
19
20 Q. Does the case-control method have any particular strengths?

21
22 A. Yes. For example, case-control studies generally allow for good control over
23 confounding. Not every case-control study, however, controls adequately for
24 confounding.

25
26
27 Q. What is confounding?

28
29 A. "Confounding" means that the factor under study is not an actual cause of the
30 disease but rather is only a marker of some true cause. For example, some
31 early studies seemed to show an association between coffee drinking and
32 bladder cancer. It is likely that coffee drinking does not cause bladder cancer.
33 Rather, these studies were probably reflecting the the fact that heavy coffee
34 drinkers are, on average, heavy smokers. Smoking is a well established cause
35 of bladder cancer.

36
37
38 Q. Does the case-control study have any particular weaknesses?

39
40 A. Yes, the design is difficult to conduct correctly and is prone to bias.

41
42

1 Q. What is bias?

2

3 A. In epidemiology the word "bias" is not used in its everyday sense of
4 "prejudice." Instead, it means "in error."

5

6

7 Q. Please describe the residential case-control childhood cancer studies related to
8 EMF.

9

10 A. To date there are five published residential case-control studies regarding
11 EMF and childhood cancer: the studies by Wertheimer and Leeper (1979),
12 Savitz (1987, 1988), Fulton (1980), Tomenius (1986), and Myers (1988).
13 Generally, these studies identified childhood cancer victims in a specific
14 geographic area and time frame, as well as a group of controls in the same
15 geographic area and time frame. The residences of these cases and controls
16 were identified, and data was collected for each residence related to magnetic
17 fields or some surrogate for magnetic fields. It would be worthwhile to
18 separately discuss each of these childhood cancer studies to help provide an
19 understanding of some of the epidemiologic issues involved in this area.

20

21

Wertheimer and Leeper (1979)

22

23 Q. What was the first epidemiologic study on EMF and childhood cancer?

24

25 A. The Wertheimer and Leeper study, which was conducted in Denver, was
26 published in 1979. It was the first published epidemiology study in this area
27 and initially raised the question of whether exposure to magnetic fields is
28 associated with an increased cancer risk.

29

30

31 Q. Did Wertheimer and Leeper study actual magnetic field levels, or did they use
32 a surrogate?

33

34 Q. Wertheimer and Leeper used only a surrogate for magnetic fields known as
35 the "wiring code." The "wiring code" is a surrogate measure of exposure in
36 which the size, number, arrangement, and distance of electric lines are used
37 to predict residential exposure to magnetic fields. These data are gathered
38 visually. For example, the relative width of the electric lines might be
39 estimated by simply looking at the wires from the street.

40

41

42 Q. What results did Wertheimer and Leeper report in their 1979 study?

1 A. They reported that the wiring code was associated with childhood cancers,
2 including childhood leukemia.

3
4
5 Q. What is your evaluation of the Wertheimer and Leeper study?
6

7 A. As I stated earlier, the Wertheimer and Leeper study was the first
8 epidemiological study conducted regarding EMF and cancer. As is often the
9 case with such exploratory studies, there were numerous problems in the
10 design, conduct, and analyses of the study.
11

12
13 Q. Could you give an example of a limitation of the Wertheimer and Leeper
14 study?
15

16 A. Yes, I can give several examples of major shortcomings with this study. First,
17 the cases and controls are of dubious comparability. If the case and controls
18 are not truly comparable, the results of any comparisons between the two
19 groups are also questionable.
20

21 Second, the data results show a pattern that I can only term as "inordinately
22 consistent." The paper contains about 30 comparisons between the wiring
23 code surrogate and various types of cancer. The results of all but one of these
24 comparisons fall within a narrow range of positive associations. This pattern
25 has not been observed in subsequent studies and, based upon my experience
26 reviewing epidemiologic data on other agents, the inordinate consistency of
27 this pattern stretches credulity. It is more likely that this inordinate
28 consistency resulted from a systematic bias in the study design or conduct.
29

30 Third, the researchers did not use "blind" procedures when they assigned
31 wire code classifications to the residences involved in the study. In other
32 words, the person who assigned the wiring code for each residence knew
33 whether or not it was the residence of a cancer case. Unblinded procedures
34 such as this can introduce bias into a study. Additional shortcomings could
35 be enumerated. These limitations of the Wertheimer and Leeper study
36 render it non-persuasive, and I view it merely as an early exploratory study
37 that raised the question of whether exposure to EMF is associated to cancer,
38 but that contributed little if any to answering that question.
39
40
41
42

Fulton (1980)

1
2
3 Q. Please describe the Fulton study.

4
5 A. Fulton attempted to replicate the findings of Wertheimer & Leeper
6 concerning childhood leukemia by using the wiring code surrogate in a study
7 of childhood leukemia in Rhode Island.
8
9

10 Q. What results did Fulton report?

11
12 A. The conclusions of Fulton were in conflict with those of Wertheimer &
13 Leeper. Fulton found no relationship between childhood leukemia and
14 wiring codes. The Fulton study also is an early study that has been subject to
15 criticism. The findings and interpretation of the study are unclear, and I
16 consider it to be a negative, but non-persuasive, study.
17

18 Myers (1988)

19
20
21 Q. Please describe the study by Myers.

22
23 A. The Myers study was conducted in the United Kingdom. Rather than using
24 the wiring code surrogate, the Myers study used distance from power lines as
25 a surrogate for magnetic field exposure.
26
27

28 Q. What were the results of the Myers study?

29
30 A. The Myers study, which was conducted in the United Kingdom, reported a
31 slight increase in cancer risk for those living near a power line, but also
32 reported a slight inverse association between estimated magnetic field
33 strength and cancer risk. In other words, residences close to a power line were
34 associated with a slightly smaller risk of cancer than those further away from
35 a power line. The Myers study does not support the hypothesis that magnetic
36 fields are related to childhood cancer. Because of certain limitations in the
37 study, including its small size, the study also does not provide persuasive
38 refutation of the hypothesis.
39
40
41
42

Tomenius (1986)

1
2
3
4 Q. Please describe the residential childhood cancer study by Tomenius.

5
6 A. Tomenius conducted his study in Sweden. He also used a "distance"
7 surrogate. He categorized a residence as "exposed" to magnetic fields if the
8 residence was within 150 meters of an electrical installation, including power
9 substations, transformers, electric railroads, high voltage wires, and subways.
10 In addition, Tomenius collected data on measured magnetic fields.

11
12
13 Q. What were the results of the Tomenius study?

14
15 A. There were two major findings of importance to interpreting the Tomenius
16 study. First, with regard to childhood leukemia, the Tomenius study found
17 no association either for the distance surrogate or for measured magnetic
18 fields. This was inconsistent with the Wertheimer and Leeper results, which
19 had found an increased risk of childhood leukemia associated with the wiring
20 code. Second, Tomenius' data shows an inverse trend between distance of a
21 dwelling from the installation and cancer risk. Tomenius also found an
22 inverse relationship between cancer risk and measured magnetic fields. In
23 short, the Tomenius study reported that, among those exposed to magnetic
24 fields (whether exposure was measured by the surrogate of distance or with
25 actual magnetic field measurements), increased exposure resulted in lower
26 cancer risk. Due in part to the inverse trend apparent in the Tomenius data, I
27 conclude that the Tomenius study provides no support for the hypothesis
28 that EMF's are related to an increased risk of childhood cancer. In fact, it
29 provides some evidence against that hypothesis.

30
31 Savitz (1988)

32
33 Q. Please describe the residential case-control study of childhood cancer
34 conducted by Savitz.

35
36 A. The Savitz study, which was conducted in Denver, collected data on the
37 wiring code surrogate and childhood cancer. Savitz also collected data on
38 actual measured magnetic fields and childhood cancer. In addition, Savitz
39 collected data unrelated to magnetic fields, such as parental smoking, breast
40 feeding history, and traffic density on nearby streets.

- 1 Q. What is your overall evaluation of the Savitz study?
2
3 A. The Savitz study showed a marked improvement in design, conduct, and
4 analysis over previous childhood cancer studies. This study also has
5 limitations, several of which are pointed out by Dr. Savitz himself in his
6 published paper. To date, however, this study should be considered the best
7 done of the published studies on childhood cancer and EMF.
8
9
- 10 Q. What were the results of the Savitz study?
11
12 A. The Savitz group has reported data in several publications. In summary
13 form, Savitz' data show no significant associations between cancer and
14 measured magnetic fields, but they did show a few weak, irregular statistically
15 significant associations between childhood cancer and the wiring code
16 surrogate. Savitz also has reported data from this study population showing
17 similar weak associations between childhood cancer and failure to breast feed
18 (Davis and Savitz, 1988), between childhood cancer and paternal smoking
19 (John and Savitz, 1991), and between childhood cancer and high traffic density
20 on nearby streets (Savitz and Feingold, 1989).
21
22
- 23 Q. Is it unusual to find a few irregular statistically significant associations in a
24 case-control study?
25
26 A. No, in fact they are to be expected even when nature is in a null state -- that is,
27 even when there is no true association between the exposure (or the
28 surrogate) and cancer. Random chance alone would produce some
29 statistically significant associations in a large group of calculations such as
30 conducted in the Savitz study.
31
32
- 33 Q. What is your interpretation of the results of the Savitz study?
34
35 A. The Savitz study contains a large amount of data, and many comments on
36 this study are possible. For this testimony, I would like to make three
37 comments. The first deals with the data on measured magnetic fields and
38 the wiring code surrogate. The pattern of data, with no statistically significant
39 associations between measured fields and cancer, and a few weak irregular
40 associations between the wiring code and cancer, is consistent with nullness --
41 that is, with a lack of effect of exposure to magnetic fields.
42

1 The second level of interpretation attempts to place the magnetic field data in
2 the context of the larger body of data that has been reported from the Savitz
3 study on breast feeding, traffic density, and exposures. The pattern of weakly
4 positive results found in this larger body of data suggests that none of the
5 exposures is itself a cause of childhood cancer. Rather, I interpret this pattern
6 as suggesting a profile or risk factors that characterize children at greater risk
7 of leukemia: parents who smoke, absence of breast feeding, residence near
8 busy streets, etc. Viewed in this larger context, I would hesitate to accept any
9 of the weak reported associations, including the weak irregular associations
10 with wiring code, as descriptive of a cause-effect relationship.
11

12 Finally, I would note that the positive associations with the wiring code that
13 Savitz did report are weaker than the associations reported by Wertheimer
14 and Leeper. In epidemiology, we expect that if a reported statistical
15 association in one study is truly descriptive of an association that is occurring
16 in the real world, that association will become stronger, not weaker, as the
17 design and conduct of subsequent studies improves. The fact that the
18 reported association is weaker in the relatively well-done Savitz study
19 suggests that there is no true association between EMF and cancer underlying
20 these statistical reports.
21

22 Peters

23
24
25 Q. Are you familiar with the childhood cancer study in California by Peters?

26
27 A. Yes. The Peters study is residential case-control study of childhood cancer
28 conducted in California.
29

30
31 Q. What is the basis of your familiarity with the Peters study?

32
33 A. I was a member of the advisory committee which sponsored the Peters study,
34 and was present at the advisory committee meeting at which Dr. Peters
35 presented his preliminary results. Since the results were preliminary, they
36 are subject to change as analysis and review of the data continue. At present,
37 the Peters' results have not appeared in the published literature. The only
38 public announcement regarding the Peters' study of which I am aware is a
39 press release summarizing some of his preliminary findings.
40

41
42 Q. What is your evaluation of the Peters study?

1 A. Not enough design information and data are available from the Peters study
2 to conduct a detailed epidemiologic evaluation of the study. As an
3 epidemiologist, I therefore cannot rely upon the Peters study until
4 information is available to conduct such an evaluation.
5
6

7 Q. Are you able to reach any conclusions about the Peters study?
8

9 A. With the limitations just expressed, I can state my preliminary opinions
10 about the Peters study. The Peters study examined spot measurements of
11 magnetic fields, 24-hour measurements of magnetic fields, and the wiring
12 code. Peters found no association between either of the measures of magnetic
13 fields and childhood cancer. He found an association between the wiring
14 code and childhood cancer that is weaker still than the association seen by
15 Savitz. With regard to the findings on measured magnetic fields, my
16 preliminary evaluation is that this study confirms the consistent findings in
17 the published literature that there is no association between measured
18 magnetic fields and cancer. With regard to the findings on wiring code, my
19 preliminary conclusion is that the weaker association between the wiring
20 code and cancers continues the pattern of better-done studies reporting
21 smaller associations, which again suggests that there is no true association
22 between EMF and cancer underlying these statistical reports.
23

24 Additional Issues Related to the Childhood Cancer Studies

25
26
27 Q. Dr. Cole, are there any further issues that you would like to address with
28 regard to the childhood cancer studies?
29

30 A. Yes. I would like to mention three further issues: consistency of results,
31 biological plausibility, and an ecologic comparison.
32

33
34 Q. What is the importance of consistency in the childhood studies?
35

36 A. As noted previously, consistency is an important factor in accepting a cause-
37 effect relationship. If a cause-effect relationship truly existed between an
38 exposure and a disease, one would expect that relationship to appear
39 consistently in any one study, and also to appear consistently between studies.
40

41
42 Q. Do the childhood cancer studies show consistent results?

- 1 A. The data from the wiring code and other surrogates is inconsistent. Some
2 studies report a weak association, while others fail to report an association.
3 The data from the measured fields consistently shows no statistically
4 significant association between measured fields and cancer risk.
5
6
- 7 Q. What is meant by "biological plausibility"?
- 8
- 9 A. Biological plausibility refers to the question of whether there is a credible
10 biological mechanism that supports the hypothesis that exposure to the agent
11 causes cancer. Biological plausibility can be established either through
12 mechanistic research, or through animal research that shows that exposure to
13 the agent causes cancer in experimental animals.
14
15
- 16 Q. What is the importance of biological plausibility?
- 17
- 18 A. The importance of biological plausibility varies depending upon the strength
19 and consistency of the epidemiological evidence. If the epidemiological data
20 consistently shows high risks of exposure, as is the case with cigarette
21 smoking and lung cancer, we can accept the epidemiological data even
22 without supporting evidence of biological plausibility. When the
23 epidemiological data is weak and inconsistent, biological plausibility is more
24 important. If the experimental data is supportive of biological plausibility in
25 such a situation, we can place more confidence in the epidemiologic results; if
26 biological plausibility is missing, we place less confidence in the
27 epidemiological results.
28
29
- 30 Q. Does the experimental data on EMF support biological plausibility?
- 31
- 32 A. No. While biological effects have been reported by some researchers, those
33 effects are not related to the processes of carcinogenesis and do not provide
34 biologically plausible support for the epidemiological data.
35
36
- 37 Q. Do you consider it to be "non-scientific" to include biological plausibility in
38 your evaluation of the epidemiologic data?
- 39
- 40 A. No. Biological plausibility is a standard factor that is considered in the
41 evaluation of most, if not all, bodies of epidemiologic literature. Professor
42 Branford Hill (1965), in his widely quoted paper published in the *Proceedings*

1 of the Royal Society of Medicine, listed biological plausibility as one the
2 factors that should be considered in an epidemiological evaluation.
3
4

5 Q. What is an ecological comparison?
6

7 A. In an ecological comparison, one collects data on the general population,
8 rather than on individuals within that population. For example, in this
9 century cigarette smoking and lung cancer rose sharply during the same time
10 frame, a fact that provides persuasive evidence that there is a causal
11 relationship between the two events. This type of ecological comparison,
12 while subject to limitations, can provide important information as to a
13 suggested relationship between an agent and a disease.
14

15
16 Q. Have you conducted an ecological comparison for EMF and childhood
17 cancer?
18

19 A. Yes. Using power consumption as a surrogate for EMF exposure, data show
20 that power consumption has increased approximately 10-fold since 1950. If
21 exposure to EMF was associated with, for example, childhood leukemia, we
22 would expect to see a similar sharp rise in childhood leukemia rates over that
23 same time period. Yet childhood leukemia rates rose approximately 20%
24 from 1950-1960, and it is probably true that most of that increase was not
25 attributable to an actual increase in leukemia, but rather to improved
26 diagnostic procedures. Childhood leukemia rates have remained essentially
27 stable since 1960. This ecologic comparison further supports the conclusion
28 that exposure to EMF is not causally associated with childhood cancer.
29
30

31 Conclusion Regarding the Childhood Cancer Studies 32

33 Q. Dr. Cole, what is your overall conclusion concerning the childhood cancer
34 studies?
35

36 A. Looking at the childhood cancer studies as a whole and recognizing the
37 degree of validity of individual studies within that group, I conclude that
38 there is no demonstrated increased risk of childhood cancer when actual
39 electric or magnetic fields were measured. When surrogates such as the
40 wiring code have been used in the childhood studies, the data have shown a
41 pattern of mixed results between weak positive and negative outcomes. This
42 is a pattern of random association in a non-experimental science, not the

1 pattern of cause and effect. Long term time trends of EMF exposure and
2 leukemia in children in this country are not consistent with a cause-effect
3 relationship. Finally, neither theoretical considerations nor animal studies
4 are available to support a cause-effect hypothesis. I conclude that the
5 epidemiologic literature on EMF, when viewed as a whole, provides no
6 persuasive scientific support for the hypothesis that EMF causes cancer in
7 children.

8
9 **B. The Draft Report of the Environmental Protection Agency**

10
11 Q. Dr. Cole, are you familiar with the draft report of the Environmental
12 Protection Agency on cancer and EMF?

13
14 A. Yes. As I stated earlier in my testimony, I have reviewed the draft EPA report.

15
16
17 Q. Does the EPA report discuss the epidemiologic literature on EMF and
18 childhood cancer?

19
20 A. Yes, there is a separate chapter on the epidemiologic literature.

21
22
23 Q. Did you rely upon the draft EPA report in forming your opinion about the
24 epidemiologic literature and childhood cancer?

25
26 A. As I stated previously, secondary sources such as the draft EPA report can
27 provide useful context for the scientific debate, but they do not provide
28 primary data or design information. I therefore do not give substantial
29 weight to secondary sources. The draft EPA report also is a somewhat special
30 case even amongst the secondary sources.

31
32
33 Q. Why do you refer to the draft EPA report as a "special case"?

34
35 A. Normally, literature reviews and other secondary sources are published in
36 the peer-reviewed literature. These publications have thus been critiqued by
37 expert reviewers before they are published. The draft EPA report, while it has
38 been made public, is still undergoing its "peer-review" process. This fact is
39 clearly indicated on the cover of the draft EPA report. For instance, it states:
40 "Draft: Do Not Quote or Cite." In addition, the comments that have been
41 generated as part of the review process suggest substantial revision of the
42 draft EPA report may be forthcoming.

1 Q. Could you briefly describe the ongoing review process for the draft EPA
2 report?
3

4 A. The draft EPA report was authored by internal staff at the EPA. The EPA has
5 convened a panel of outside experts from academic and research institutions,
6 known as the Non-Ionizing Electromagnetic Fields Subcommittee (the
7 "NIEMFS" panel), to review and comment on the document. This panel held
8 several public meetings to receive comment on the EPA report and conducted
9 its own review of the draft report. Its final comments, which were recently
10 forwarded to the Radiation Advisory Committee of the Science Advisory
11 Board of the EPA, state that "currently available information is insufficient to
12 conclude that the electric and magnetic fields are carcinogenic" and that "the
13 EPA document has serious deficiencies and needs to be rewritten." The
14 NIEMFS panel's recommendations have been adopted by the Radiation
15 Advisory Committee and forwarded to an Executive Committee of the
16 Science Advisory Board.
17

18 In addition, the Committee on Interagency Radiation Research and Policy
19 Coordination ("CIRRPC") has also submitted comments on the draft EPA
20 report. CIRRPC is comprised of representatives from several agencies of the
21 executive branch of the federal government. Many of those agencies have
22 submitted comments on the draft EPA report. CIRRPC has compiled those
23 comments and forwarded them to the EPA. Some of those comments have
24 been supportive of the report. Others were not supportive. For instance, the
25 National Cancer Institute stated in its comments that: "Four staff members,
26 all well-versed in epidemiologic studies, reviewed the above document and
27 written comments are provided. All reviewers agreed that the human
28 epidemiologic data to date did not support the sweeping conclusions made in
29 the EPA document. In our judgment the conclusions presented remain
30 scientifically unsound and unnecessarily alarming."
31

32 It is not possible to predict what changes will ultimately be made to the draft
33 EPA report at the conclusion of this review process. In my opinion, however,
34 the draft EPA report should be treated as a secondary source that is still in the
35 process of review, and I give it little if any weight in my evaluation of the
36 epidemicologic data.
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1 C The Research on EMF and Adult Cancer

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3 Q. Dr. Cole, have you also reviewed the epidemiologic data on adult cancers?

4
5 A. Yes. The adult cancer studies include residential studies similar in design to
6 the childhood studies, as well as occupational studies.
7

8
9
10 The Adult Residential Studies

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12 Q. How many adult residential studies have been conducted?

13
14 A. Four adult residential studies have been conducted: Wertheimer and Leeper
15 (1982), Severson (1988), Coleman (1985, 1989), and McDowall (1986). The first
16 three of these are case-control studies, while the McDowall study is a
17 retrospective follow-up study. (I will discuss the retrospective follow-up
18 design in my discussion of occupational studies.)
19

20
21 Q. What were the results of the adult residential studies?

22
23 A. Wertheimer and Leeper conducted a residential case-control study of adult
24 cancer in Denver very similar in design to their childhood study, and with
25 many of the limitations of that study. They reported a strong and consistent
26 relationship between the wiring code and adult cancers. The remaining adult
27 residential studies, including the case-control study by Severson in
28 Washington State, the Coleman study in London and the RFU study by
29 McDowall in East Anglia, England, reported no association between wiring
30 code, distance from power lines, or measured fields and adult cancer.
31

32
33 Q. What do you conclude with regard to the residential studies of adult cancer?

34
35 A. With the exception of the early, flawed report by Wertheimer and Leeper, the
36 residential studies of adult cancer have been consistently negative for both
37 actual measured fields and for magnetic field surrogates. I conclude that these
38 studies provide no support for the hypothesis that EMF is associated with an
39 increased cancer risk in adults.
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The Occupational Studies

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Q. Please describe the occupational studies of adult cancer and EMF.

A. The occupational studies use some form of occupational information, such as a job title, as a surrogate for magnetic field exposure. For example, an occupational study might categorize all electricians as "exposed" to EMF.

Q. What types of study designs have been used in the occupational studies?

A. As I noted earlier in my testimony, the categories are: (1) case control studies, (2) proportionate mortality ratio ("PMR") studies, and (3) retrospective follow-up ("RFU") studies.

Q. Before discussing the individual study designs, are there any comments you would like to make generally about the occupational studies?

A. Yes. The approach of using occupational information such as job titles has limitations. As noted, these studies might categorize all electricians as "exposed" to EMF. However, not all electricians will have exposure to EMF. Those who do have exposure to EMF may also have exposure to industrial chemicals, welding solvents, or other potentially carcinogenic agents. This latter factor is an example of "confounding." Potential confounding is difficult to control in occupational studies, and is a major limitation of those studies.

Q. Does failure to control for confounders effect your evaluation of the studies?

A. Yes. it effects the evaluation of the individual studies, and the body of literature as a whole. If an individual study does not control for confounders, less confidence should be placed in any associations that are reported, because the researchers were not able to exclude other possible causes of the cancer.

The failure to control for confounders also effects the evaluation of the body of literature as a whole. The associations with cancers that have been reported in the occupational literature are not consistent. For example, one study might report an increase in leukemia, but not in brain cancer, while another study might report an increase in brain cancer, but not in leukemia.

1 It is possible that the workers in the first study had increased exposure to
2 benzene, which is known to cause leukemia in adults, while the workers in
3 the second study had exposure to some agent associated with brain cancer.
4 The inconsistent pattern of results in these studies is thus consistent with
5 multiple confounders operating in the various studies.
6
7

8 Q. Have any of the occupational studies controlled for confounders?
9

10 A. Yes. Despite the difficulty of controlling for confounders in an occupational
11 setting, Thomas (1987), in a study of brain cancer, was able to distinguish
12 electrical workers who were exposed to only electric and/or magnetic fields
13 from electrical workers who had additional exposures. Thomas found no
14 increased risk of brain cancer in workers exposed only to electric and/or
15 magnetic fields, suggesting that exposure to EMF is not a cause of brain cancer.
16 The Thomas results suggest that confounding should be considered a
17 substantial factor in the evaluation of the occupational studies.
18
19

20 The Occupational Case-Control Studies 21

22
23 Q. Please describe the occupational case-control studies.
24

25 A. Similar in design to the residential studies, the occupational case-control
26 studies compare exposure histories of subjects with and without cancer to
27 determine the relative risk associated with the surrogate for EMF exposure
28 used in each study.
29

30
31 Q. What were the results of the occupational case-control studies?
32

33 A. There have been 19 non-residential case-control studies which have
34 examined various indices of possible EMF exposure. These studies are
35 useful, but it must be borne in mind that the case-control design is a difficult
36 type of investigation to conduct. All of these studies have limitations and
37 many are flawed. One major limitation is that none of the studies included
38 measurements of EMF exposure. In addition, the men in these studies most
39 likely were exposed to known or suspected carcinogens. Nonetheless,
40 fourteen of these studies have reported some positive association between a
41 surrogate of EMF exposure and cancer. It should be noted that the Thomas
42 study, which did control for confounders, was a case-control study.

1 Q. Are you familiar with an occupational study by Matanoski?
2

3 A. Yes. I was a member of the advisory panel for the Matanoski work. There are
4 actually two studies by Matanoski, neither of which has appeared in the peer-
5 reviewed literature. The first, and larger, of the studies is a nationwide case-
6 control study of mortality in telephone line workers. The second is a
7 retrospective follow-up study of cancer incidence in telephone line workers,
8 limited to data from the state of New York.
9

10 Q. What is your evaluation of the Matanoski study?
11

12 A. As with the Peters study, not enough information is available about the data
13 and design of the Matanoski to conduct an epidemiologic evaluation. The
14 preliminary results of which I am aware, however, suggest that Dr. Matanoski
15 observed 2-3 more cases than expected of male breast cancer amongst the New
16 York state linemen. I should emphasize that an individual study must be
17 evaluated in the context of the entire body of epidemiologic literature. When
18 evaluated in the context of the larger body of epidemiologic literature, I view
19 these preliminary results from the Matanoski study as positive, but non-
20 persuasive.
21

22 The Proportionate Mortality Ratio Studies 23

24
25 Q. Please describe the proportionate mortality ratio studies.
26

27 A. The PMR occupational studies have evaluated the proportion of workers
28 presumptively exposed to EMF who die from cancer. As noted above,
29 proportionate mortality studies generally relate to occupational titles found
30 on the death certificates of persons employed in occupations where the
31 researchers assume (without testing) that there was more exposure to EMF
32 than is experienced by the general population. A major limitation of PMR
33 occupational studies is that spuriously high PMR's can emerge if the exposed
34 workers have an overall mortality rate that is lower than that of the general
35 population. This is typically observed in occupational settings (i.e., the
36 "healthy worker" effect) and may yield the appearance of risk that is, in
37 reality, artificially inflated.
38

39
40 Q. What were the results of the PMR studies?
41
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1 A. There are eight such studies and they are evenly divided between positive
2 and negative. The PMR studies, however, do not provide a reliable basis to
3 infer causality, principally because of the inherent limitations of such studies.
4 In this type of studies, slightly elevated cancer mortality rates are to be
5 expected due to the deficit of mortality that is typically observed among active
6 workers. The results of these studies are consistent with this expected
7 phenomenon and, as such, I do not believe that the results of the
8 proportionate mortality studies are helpful in answering the question of
9 whether occupational EMF exposure is associated with cancer.

10 The Retrospective Follow-Up Studies

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13
14 Q. Please describe the retrospective follow-up studies.

15
16 A. In the RFU occupational studies, a group of subjects who presumably
17 sustained moderate to heavy exposure to EMF over a long period of time (e.g.,
18 5 - 25 years) are traced up to a recent point in time. The number of deaths
19 within the group from specific diseases of interest are then compared with the
20 number to be expected if they had died at the same rate as the general
21 population. In general, the principal advantage of RFU studies is that they
22 usually are not biased to a large degree. The principal disadvantage is that
23 they are typically prone to confounding and may be imprecise given the
24 relatively few deaths from any one particular cause.

25
26 While the retrospective follow-up studies are not without their problems and
27 limitations, they generally are considered to be the most valid form of
28 epidemiological research that has been conducted to address the question of
29 whether cancer is linked to EMF exposure. One of the limitations of the RFU
30 studies is that they evaluate EMF exposure using job titles. In most other
31 ways (i.e., absolute measures of disease risk; superior, less arbitrary control
32 groups), however, they are more reliable than are proportionate mortality
33 ratio or case-control studies.

34
35
36 Q. What were the results of the RFU studies?

37 A. There have been 10 RFU studies and only one has reported positive results. It
38 is striking that virtually all of these studies are negative, a fact which provides
39 strong support against the hypothesis that EMF exposure causes cancer.
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1 **Conclusion Regarding the Adult Cancer Studies**
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3

4 Q. What do you conclude about the epidemiologic studies involving power
5 frequency electric and/or magnetic fields and adult cancer?
6

7 A. The studies involving power frequency electric and/or magnetic fields and
8 cancer show weakly positive to zero or negative associations. The studies that
9 use the most acceptable design consistently have found no association
10 between EMF and cancer. I conclude that this body of studies does not
11 support the hypothesis that EMF is associated with adult cancer, and that the
12 best-designed and conducted studies provide strong support against that
13 hypothesis.
14

15
16 D. **Conclusions Regarding the Epidemiologic Research on EMF and**
17 **Cancer**
18

19
20 Q. Dr. Cole, do you have an opinion on the relationship between power
21 frequency electric and/or magnetic fields and cancer in humans?
22

23 A. Yes, I do.
24

25
26 Q. What is that opinion?
27

28 A. Taken together, the epidemiologic reports fail to demonstrate any strong or
29 consistent pattern of association between EMF and cancer among human
30 beings. Indeed, even among the positive studies, findings are not supported
31 by the internal characteristics of the data. The summation can only be that, to
32 date, there is no demonstrated relationship between EMF and cancer in
33 human beings.
34

35
36 Q. In your opinion, will persons residing near or otherwise proximate to the
37 Woodbourne-Heaton 230 kV transmission line have an increased risk of
38 cancer from the electric and/or magnetic fields associated with that line?
39

40 A. Since I am of the opinion that EMF's have not been demonstrated to be
41 carcinogenic, I believe that the 230 kV transmission line poses no threat of
42 cancer to persons in its vicinity.

1 Q. Does this conclude your testimony?

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3 A. Yes.

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**BEFORE THE
PENNSYLVANIA PUBLIC UTILITY COMMISSION**

**EXHIBIT PC-1
OF
DR. PHILIP COLE**

**ON BEHALF OF
PHILADELPHIA ELECTRIC COMPANY**

November 1991

CURRICULUM VITAE

Name: Philip Cole **Born:** August 22, 1938; Boston MA

Position: Professor and Chairman
 Department of Epidemiology
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Education:	Michigan State University	B.A.	1960
	University of Vermont	M.D.	1965
	Harvard University	M.P.H.	1967
	Harvard University	Dr.P.H.	1970

Previous positions:

Department of Epidemiology Harvard School of Public Health Assistant and Associate Professor	1969-78 1978-79
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Consultant in Epidemiology and Biostatistics Unit of Epidemiology International Agency for Research on Cancer	1977-78
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Surgical Intern, Royal Victoria Hospital Montreal	1965-66
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Certification and Professional Societies:

Licensed, Alabama Medical Licensure Commission	1981
Member, American Epidemiologic Society	1973
Certified, American Board of Preventive Medicine	1971
Licensed, Board of Registration in Medicine, Commonwealth of Massachusetts	1966-1980
Diplomate, National Board of Medical Examiners	1966

Honors: American Cancer Society, Faculty Research Award 1973-78
Merck Lecturer, Montreal Cancer Institute 1977
Gordon Richards Memorial Lecturer,
Ontario Cancer Treatment and Research Foundation 1979
John Whittick Memorial Lecturer
Canadian Cancer Society 1980
Kammer Merit in Authorship Award
American Occupational Medical Association 1982
John Rankin Visiting Professor
of Occupational and Preventive Medicine
University of Wisconsin, Madison 1983
Eleanor Leader Memorial Lecturer
University of Toronto, Toronto 1985
Grand Prix Lacassagne du La Ligue
Nationale Francaise contre le Cancer
(with B. MacMahon, J. Brown and D. Trichopoulos) 1986

Major Committees:

Scientific Advisory Committee
Division of Cancer Cause and Prevention
National Cancer Institute 1978-80
Epidemiology and Disease Control Study Section
National Institutes of Health 1973-77
Clinical and Epidemiological Research
Advisory Group
National Cancer Institute of Canada 1973
Committee on Epidemiology and Prevention (Chairman)
National Bladder Cancer Project 1971-73
General Motors-United Auto Workers
Occupational Health Advisory Board 1982-87
Mott Prize Selection Committee
General Motors Cancer Research Foundation 1985
Prevention, Cancer Control (Chairman)
Steering Committee for the National Planning Effort
National Cancer Institute 1984-85

Major Committees (continued):

	Board of Scientific Counselors Division of Cancer Prevention and Control National Cancer Institute	1986-1990
	Scientific Advisory Committee Pittsburgh Cancer Institute	1987-1989
Teaching:	Harvard School of Public Health	
	The epidemiology of chronic diseases	1969-72
	The epidemiology of neoplastic diseases	1973-77
	Epidemiologic methods	1976
	Principles of epidemiology	1978-79
	University of Minnesota - Graduate Summer Session	
	The epidemiology of cancer	1971, 74-80
	Principles of epidemiologic research	1985
	Fundamentals of epidemiology	1986, 87
	International Agency for Research on Cancer	
	Cancer epidemiology	1974, 76, 78, 80
	University of Massachusetts-Graduate Summer Session	
	Principles of epidemiology	1981-84
	Cancer epidemiology	1982
	Tufts University - Graduate Summer Session	
	Epidemiologic bases of public health policy and law	1986, 87
	University of Alabama at Birmingham	
	Epidemiology of cancer	1980
	Principles of epidemiologic research	1980-83
	Advanced epidemiologic methods	1981
	Doctoral seminar	1981-

Research Interests:

The epidemiology of breast cancer, Hodgkin's disease and bladder cancer
Health effects of exogenous hormones
Occupational and chemical carcinogenesis

Editorships:

Associate Editor, Cancer Research	1982-85
Associate Editor, American Journal of Epidemiology	1982-88
Editorial Board, International Journal of Breast and Mammary Pathology	1984-
Editorial Board, Fundamental and Applied Toxicology	1984-
Editorial Board, Southern Medical Journal	1990-

Publications: See attached list.

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