IN THE UNITED STATES DISTRICT COURT

FOR THE NORTHERN DISTRICT OF OHIO EASTERN DIVISION

EDDIE A. FANNIN,

Plaintiff,

vs No. 5:93-CV-2594

NORFOLK & WESTERN RAILWAY COMPANY,

Defendant.

Deposition Of

OTTO WONG

Tuesday, December 16, 1997

REPORTED BY: DONNA LEE BECKETT, CSR #3450

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BE IT REMEMBERED that, pursuant to Notice of Taking Deposition and on Tuesday, the 16th day of December, 1997, commencing at the hour of 2:05 o'clock p.m. thereof, at the Law Offices of Burns, White & Hickton, 50 California Street, Suite 2310, San Francisco, California, before me, DONNA LEE BECKETT, a Certified Shorthand Reporter in the State of California, personally appeared OTTO WONG, Sc.D. F.A.C.E., herein, called as a witness by the Plaintiff, having been by me first duly sworn, was examined and testified as hereinafter set forth.

APPEARANCES OF COUNSEL

For the Plaintiff (Appearing telephonically)
HARTLEY & O'BRIEN
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Wheeling, West Virginia 26003
By: B. DEAN HARTLEY, Attorney at Law

For the Defendant
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By: QAVID A. DAMICO, Attorney at Law

EXAMINATION BY MR. HARTLEY
MR. HARTLEY: Q. Dr. Wong, as an epidemiologist, do you attempt to determine whether causality can be determined between Substance X and Disease Y?
A. That's true.
Q. Could you explain to me how you do that?
A. Basically we have what we call the. Hill criteria for causation. We look at the data on that relationship, exposure to Substance X and Disease Y in terms of what we call the strength of association, consistency of association, specificity of association, dose response relationship and so on. Those are the major criteria that we use in determining whether there is an association between exposure and the disease.
Q. Well, let's talk for a second about specificity. When you utilize the term specificity, what do you mean?
A. Well, in terms of specificity, we are talking about both components of the relationship. First we've got to be specific in terms of exposure. And we've got to be specific in terns of the disease outcome as well. For example, if 1 make the statement, chemicals cause canter, that is not very specific, but certainly that is true. But that's not very informative. We need to specify what kind of chemical and we also need to

specify what kind of cancer.
Q. Does specificity require you, when you're utilizing the Bradford Hill criteria, to exclude other types of cancer and only center on one specific type?
A. No, it does not because a chemical may be able to cause more than one type of cancer. But whatever cancer we are talking about, we need to have specific data on the relationship between that chemical and the specific kind of cancer that we're talking about.
Q. Are you talking about specific data meaning biological plausibility or something else?
A. I am talking about epidemiologic data.
Q. Would you agree with me that when you are doing your epidemiologic studies that if you look at particular types of cancers that are similar in nature they can be lumped together to determine statistical significance?
A. Well, when you say "similar," that's a big word. To some you may be similar. To others it may not be that similar. If I am reading your question right, I don't think multiple myeloma is similar to leukemia or Hodgkin's disease. That's what we're talking about.
Q. If we look at the various studies you would agree, would you not, that multiple myeloma is a

lymphatic disease?
A. It's one of the specific types of lymphocytic disease, yes.
Q. And it is classified on ICD9 as 203, correct?
A. Yes, sir.
Q. And in your paper in 1989, which you did with Dr. Raabe entitled -- R-a-a-b-a -- A Critical Review of Cancer Epidemiology in Studies of Petroleum Industry Employees with a Quantitative Meta-analysis by Cancer Site, didn't you suggest that you could combine cancer of other lymphatic tissues into one category for statistical analysis?
A. In 1989 we looked at some old papers when we do the review and the meta-analysis. At that time we said that some of the groups overlap in the old studies and some of them may be combined into different groups. I did say that. But that doesn't mean -
Q. Well, if -
A. Can I finish?
Q. Yes, sir. 1 thought you were. I'm sorry.
A I guess that's the problem of not being able to see face-to-face. I'm not quite done with the answer.
But when you look at more recent studies you've got to look at the biology of the disease, what

we understand at this point. Certainly, for multiple myeloma it's very distinct. Now we do understand that it's very distinct from leukemia and it's very distinct from non-Hodgkin's lymphoma.
Q. Well, what you are saying is that it has changed since 1989, in your opinion, whether you should compact the various diseases into one category?
A. In 1989 I didn't say that. You have to look at them as a combined group. I'm saying that it's open to question. We should look at that as a total group. We should also look at the specific groups as well. But now the hematologists tell us that multiple myeloma is a very distinct disease, very different from other categories in the lymphopoietic cancer broad category.
Q. How long, in your opinion, has multiple myeloma been considered distinct from lymphomas?
A. At least within the last, I would say, my understanding is seven or eight years. Maybe longer than that.
Q. And upon what do you base that opinion?
A. Based on -- well, I'm sure you have a copy of my report. The report dated November 25th, 1997.
Q. November 28th, Doctor?
A. No, 25. 1 have two reports. The first one was dated November 25th, 1997.

Q. I don't have that report. Was that to
Mr. Donahoe?
A. Yes.
Q. I don't have that report. I never received
that report.
MR. DAMICO: You never received the November 25th,
'97?
MR. HARTLEY: No, sir, I did not. I've got
November 28th of '97.
Q. Does November 25th, 1997 discuss paints and
solvents and everything else?
A. The report dated November 25th, 1997 talks
about benzene exposure and multiple myeloma.
Q. I don't have that report.
A. Well, that's kind of unfortunate, because it's
going to be difficult for me to refer you to specific.
references and so on.
MR. DAMICO: We'll put it in the fax machine to
you right now. What's your fax number? Off the
record.
1fliscussion off the record.)
MR. HARTLEY: Q. Doctor, the last question to you
was; I believe, what do you base your opinion on that
the lymphoma should be considered separate and distinct
from multiple myeloma?

A. Right. And I was going to refer you to my report which you would be receiving shortly. Anyway, let me point out where I talk about that in my report. That's on Page 3, the first full paragraph. And I do have two references there. One is dated 1981. The other one is dated 1985. Although those were published in 1981 and 1985, 1 was not aware of those references until much later. So as far as I'm concerned, 1 didn't know of that until much later.
Q. Could you tell me the title of the 1981 article?
A. The 1981 is actually a textbook on hematology. And the lead author of the textbook is Wintrobe. W-i-n-t-r-o-b-e.
Q. And the other one?
A. And the other one is an article actually written by someone at OSHA. Her name is Tsongas. T-s-o-n-g-a-s. 1985.
Q. And what does Ms. Tsongas say in her report in 1985?
A 'Basically Dr. Tsongas said that we should - when we analyze the data we should separate the disease entity out.
Q. Did she give a reason for that?
A. Basically, she cited some, I guess, hematology

1 papers, some epidemiologic papers and so on, to support
2 that.
3 Q. Is Dr. Tsongas a pathologist?
4 A. I don't know what she is.
5 . Q. So you just relied on her paper of 1995 that
6 said that you should separate those out?
7 A. Well, those are the two samples that indicate
8 that non-Hodgkin's leukemia and multiple myeloma are
9 different diseases.
10 Q. And you think that she is a doctor?
11 A. I think she has a Ph.D.
12 Q. And Ph.D. in what, Dr. Wong?
13 A. I don't know.
14 Q. Other than the Wintrobe book and the Tsongas
15 report of '85, was there any other pathological
16 evaluation that you reviewed that suggested that you
17 should not classify the lymphopoietic malignancies for
18 statistical purposes when you're doing an analysis?
19 A. There are quite a few. Those are just two
20 analysis.
21 Q 'When you testified before OSHA on the benzene
22 standards did you have an opportunity to talk to Dr.
23 Tsoingas?
24 A. No.
25 Q. She was not in the office at that time, the

OSHA office?
A. I didn't go to OSHA and testify. I testified in front of an administrative judge in a hotel in Los Angeles.
Q. Were there any OSHA representatives there?
A. As far as I know I recognized one person, Dr. Infante. I-n-f-a-n-t-e.
Q. And were you hired by OSHA to review and testify during the hearings on the benzene standard?
A. I was a consultant to OSHA for the hearing, yes.
Q. That means you were paid by them, right?
A. Not a whole lot of money. Yes, if you call that pay.
Q. You got some remuneration?
A. Right.
Q. Have you reviewed your '86 testimony before OSHA in the recent past?
A. Not really, but I remember roughly what I said.
Q: "Isn't it true that in response to the comments by Dr. MacMahon -- M-a-c, capital M, a-h-o-n -- wherein he recommended that maybe it was not appropriate to analyze the lymphomas and the leukemias together, you then broke down non-Hodgkin's lymphoma and included

multiple myeloma with non-lymphoma in your report to OSHA?
A. That's true. And that's because the software package that we used for the analysis at that time, we were not able to break it up into non-Hodgkin's lymphoma and multiple myeloma. We were limited by the software that we used at that time.
Q. How did the software prevent you from doing that?
A. Because basically the software grouped part of the non-Hodgkin's lymphoma and multiple myeloma as one group. And that's the way how we calculated the SMR at that time.
Q. In your testimony before OSHA, you never indicated in response to Dr. MacMahon's remarks that the computer program was the cause of the problem but rather you said that the difficulty in separating certain poietic cancers from each other is that a certain amount of overlap appeared to occur. And there's no comment about a computer program in that testimony, is that correct?
A. I don't think I talked about the computer program at that time. I think right now you're focusing on multiple myeloma. But at the OSHA hearing we were talking about different diseases within that

broad category in general. We were not focusing on multiple myeloma alone. And 1 can tell you that for some disease I still think it's appropriate to combine certain ICD groupings together. In fact, for non-Hodgkin's lymphoma we have to combine at least two ICD codes in order to get multiple myeloma. So up to today I still think that certain combinations is appropriate, but not across the board.

Q. Would it be appropriate to combine non-Hodgkin's lymphoma with multiple myeloma in your analysis of a cohort?

A. As -- well, it depends on what the question is. If the question is on multiple myeloma, specifically on multiple myeloma, no, it would not be appropriate to utilize the results combining multiple myeloma and non-Hodgkin's lymphoma together.

Q. But you combined those in your testimony before OSHA, didn't you? You combined ICD 8, 200, 202, and 203, which is multiple myeloma.

A. That's right, because, as I said, the software program would not allow us to do a separate analysis.

Q. Well, you also testified in 1986 in front of OSHA that the medical literature is replete with reports documenting the transition from certain lymphomas and multiple myelomas to leukemia. And

leukemia phase has long been recognized in some non-Hodgkin's lymphoma cases. And leukemic transformation of lymphoid cells is considered a part of the natural course of some non-Hodgkin's lymphoma and lymphoma in general.
A. When you say testified to that effect, I think it's slightly misleading. Because if you read. the statement, or if you read my report, my publication which appeared in 1987, basically I quoted a number of people saying what you just said, okay, those -- at least some thoughts in the community, in the scientific community at that time. And being an epidemiologist, I simply say that there are people who think this way. Then they think that we should combine some certain categories for analysis. But at the same time for certain categories we should also separate them.
Q. Now, then, was your computer program able to differentiate between leukemia and lymphoma?
A. Leukemia and lymphoma, yes.
Q. Why did you combine lymphoma with leukemia in your statistical analysis in '86 before OSHA?
A. Well, if you look at any epidemiologic studies, we have a hierarchy of causes of death. We started with all causes of death. And then we break it down into cancer deaths and non-cancer deaths. Among

cancer deaths we break that down by different body systems, respiratory system, gastrointestinal system and so on. So I don't see anything that's different from we combine colon cancer and stomach cancer in some analysis, but at the same time separate that in some other analysis.
Q. Didn't you do that in your publication of what is commonly referred to as Wong 1 and Wong 2?
A. I don't know what's commonly referred to. I published two papers in 1987. Part 1 and Part 2.
Q. Part 1 and Part 2.
A. I commonly refer to them as Wong 1 and Wong 2.
Q. You did that in those publications as well, did you not, combine the lymphatic malignancies along with the leukemias?
A. That's right. But, as I said, it's not any different from the situation of cancer of the digestive system. In the cancer of the digestive system we combine stomach cancer and colon cancer together. But that doesn't mean that the stomach is the same as colon.
Q. Well, even after Dr. MacMahon made the recommendations that you split it out, you in your testimony to OSHA justified the combination and then published articles combining the various categories of

disease that came from the cohort, didn't you?
A. That's not correct at all. In 1987, very specifically I have published results specific to leukemia by itself.
Q. Did you combine the lymphatic leukemias with lymphomas in your analysis?
A. In some analysis I did, but not in all. 1 present both sides at that time.
Q. And in '87 you found a statistically significant increase in the cohort you were studying for lymphatic leukemias, did you not?
A. Yes, I did. And I said that the increase was driven by the increase in leukemia.
Q. And what cell type of leukemia did you find in the cohort that you were reporting on in 1987?
A. In the 1987 paper I point out that I do not have enough cases to do any cell type specific analysis.
Q. What does your paper report as the cell type you found?
A: I find all kind of cell types.
Q. Well, let's look at them. Do you have your paper?
A. Yes, I do.
Q. In which one do we want to look at them, dose

response analysis or general results?
A. Hold on for just a second. I think we want to look at the second paper and Table 16.
Q. What paper is that, 1 or 2, Doctor? I'm sorry. I didn't hear you.
A. 2.
Q. Table 16?
A. Right.
Q. What is the title of Table 16?
A. Characteristics of 22 Deaths from Lymphatic and Hemopoietic Cancer.
Q. What cell types did you find in this cohort?
A. I have CML. I have CLL. I have some non-specified leukemia. So we don't know exactly what cell type we're talking about. I think we also have ALL.
Q. Were there any AMLs?
A. Not specifically, but we do have non-specified acute leukemia, I think. So that could have been AML.
Q. How many of those did you have non-specific acute leukemias?
A. I don't have the ICD code in front of me.
b. I do. What number do you think it would be? What is AML?
A. AML?

Q. Yes.
A. I think AML is 205.0.
Q. Acute is 205.0. How many 205.Os do you report?
A. As I told you, we don't have any.
Q. And how many non-specific acutes do you have? Do you need that number as well?
A. Yes. I think that's 207.0, is that right?
Q. Leukemia of unspecified type, 208.
A. 208?
Q. Yes.
A. What about 207?
Q. 207 is acute arrhythmia and aleukro leukemia. That's 207.
A. Yes. What revision are you looking at?
Q. I'm looking at the ninth.
A. The ninth. The one that I used, I think, is the eighth in the study. So there may be a slight difference there. 1 think 207.0, Case 12, Case No. 12.
Q. Was the non-specific acute?
A: Yes, the non-specific acute.
Q. How many multiple myeloma did you find?
A. I found three.
Q. Did you find an elevation of multiple myeloma within the cohort?

A. Based on my calculation I should expect 2 point -- it's in the report. You have a copy of the report now?
Q. Not yet. I'm looking at Page 393 of the second part of your 1987 paper.
A. As I said, in that paper I did not report the expected because the calculation wasn't done at that time based on that program. And also, the rates of multiple myeloma was not widely available in the '80s. But subsequently I have done a calculation on how many multiple myelomas I should expect. And for that study 1 should expect -- let me find a number here. I should expect 2.58. Okay. Roughly two and a half cases of multiple myeloma. That's on Page 3 of my report. When you get it you can take a look.
Q. What did you base your expected rate on?
A. The expected rate was based on the rates published by the National Cancer Institute.
Q. Is that the U.S. general population?
A. Yes, sir.
Q: The rate that you just gave me, what was it?
A. No, that was not the rate. That was the expected.
Q. What was your expected?
A. To 2.58.

Q. In your paper on Page 393, which is Wong 2,
you indicated that you're going to utilize 0.56 as your
expected.
A. Where are you?
Q. Under your chart you go eight lines down on
the left-hand column.
A. Okay. The difference is when I said. 2.58,
that was for the entire cohort, for the entire study.
The .56 refers to a much smaller group, what we call
the intermittent exposure.
Q. I understand that.
A. So that's the difference.
Q. So the expected for the intermittent exposure
group was still 0.56, correct?
A. It should be.
Q. And that means then that the SMR for the
intermittent multiple myeloma group was still 357.1,
correct?
A. Yes.
Q. And was that statistically significant?
A: 'No.
Q. What was the confidence interval? 46.2 to
1298?
A. Right. So that interval includes the norm of
100 percent. So, therefore, it's not statistically

significant.
Q. I understand that it's not statistically significant, but it shows at least an elevated risk, which could be by chance. But it does show an elevated risk, correct?
A. It's based on two cases, remember.
Q. I thought you had three.
A. 1 think you're confusing the two analyses.
Q. There were not three multiple myelomas in the intermittent(exposure group?
A. No, there were two in the intermittent exposure group. For the entire study we had three. So when you use the three as the observed, you have to use the expected of 2.58.
Q. I understand what you did.
A. Do you understand what I'm saying?
Q. Yes. Why didn't you use internal controls for your expected for the intermittent exposed multiple myeloma cases?
A. Because that group is far too small to be stable enough for internal comparison. I said very clearly in my OSHA testimony as well as in my papers. We started -- when we designed the study we have a much larger internal control group planned, but we end up with a very, very small group. And that is just not

stable enough at all.
Q. Could you use the overall industry as an expected or as a group from which to derive your expected multiple myeloma cases?
A. When you say "overall industry," what are you referring to?
Q. The chemical industry?
A. There is no such data. You can't just go to one place and say, okay. Here are the rates of multiple myeloma or here are the rates for leukemia for chemical workers. There's no such thing. No such data base.
Q. Explain to me why you didn't use your own industry controls. You had 6- or 7,000 people, didn't you, in this study?
A. Yes.
Q. What was the amount of your control group? How many were in your control group?
A. Out of the 7,000 or so I think 3,000 or so were not exposed.
Q:-'So you couldn't use the 3,000 people to determine your expected for multiple myeloma for intermittently exposed?
A. No, it's too small. Multiple myeloma is very rare. For comparison to be meaningful you have to have

a large group that when you use the age specific rates you should have at least a few deaths in each age category.
Q. Well, you didn't have any leukemia deaths, did you, in your controls?
A. No.
Q. So wouldn't that same analysis apply to your leukemia calculations?
A. I think you just misspoke. Can you repeat the question?
Q. Sure. If you assert that your control group was too small to determine what the expected would be for multiple myeloma, why wouldn't that also apply to your leukemia expected rates since you found zero expected in that group?
A. I said the same thing for leukemia, that the rates, based on internal comparison, was not stable.
Q. You didn't say that to OSHA. You told OSHA in response to Dr. MacMahon that you felt it was better to use internal controls from this group then to go out and use the general population?
A. No.
Q. Because -- let me finish -- because there was, according to you, deficits reported by various investigators of leukemia in various industrial

1 settings.
2 A. 1 said that. But I also said that using
3 internal control would have certain advantage, okay,
4 and that's what you just said. But at the same time I
5 also said that at the hearing, as well as in my papers,
6 that using internal comparison, especially when the
7 disease is rare and the comparison group is small, then
8 the rates would be very unstable. And if we don't see
9 any deaths in a comparison group, then the comparison
10 would not be appropriate.
11 What we are saying is if someone is not
12 exposed to benzene there would be no leukemia at all,
13 which is, of course, not true.
14 Q. But you still utilized that data. You had no
15 leukemias in your control group.
16 A. I utilized that data because in our contract
17 to the Chemical Manufacturers Association, we specified
18 that we have an internal control group. So, therefore,
19 1 analyzed the data both ways. But I spelled out both
20 the advantages and disadvantages of using internal
21 comparison.
22 Q. If you felt that the internal comparisons were
23 ineffective, for lack of a better term, for leukemia,
24 why did you. publish your data?
25 A. As I said, it has both advantages and

1 disadvantages. And we also have in our contract with
2 CMA that we would use an internal group as well as an
3 external group. And, therefore, I published both. And
4 I spell out the advantages and disadvantages of both
5 methods. I think it's very clear.
6 Q. I don't think it is, to be honest with you.
7 A. What is not clear?
8 Q. Because you testified to OSHA there's no
9 obvious reason to suspect that the comparison group was
10 any different from the exposed group. So why not use
11 internal controls for multiple myeloma as compared to
12 the general population?
13 A. Because we did not see any leukemia in the
14 comparison group, the internal comparison group. That
15 means zero. So anything compared to zero is an
16 elevation, is an excess, okay? That doesn't make
17 sense. Because what we are saying is in a general
18 population we should not expect any leukemia at all.
19 That's not true.
20 Q. That would be the same for multiple myeloma,
21 right? 'Would you not expect any multiple myeloma in
22 the general population? I don't understand the
23 distinction that you make.
24 It was permissible in your mind or acceptable
25 in your mind to utilize internal controls with no

1 leukemias for your comparison with leukemias, but it
2 was not acceptable to use internal controls for
3 multiple myeloma because it was a smaller group.
4 A. Okay. 1 think you misunderstood what I did in
5 the paper. In my paper, back in 1987, the focus was on
6 leukemia. Okay. There was some interest on multiple
7 myeloma, but not the main focus. The main focus was
8 leukemia, okay? And for leukemia, I analyzed the data
9 using both the internal comparison as well as the
10 external comparison.
11 And the reason I used the internal comparison
12 was, when we design a study we were hoping that we have
13 a much larger group. And hopefully, that would give us
14 some stable rate for us to do some comparison
15 internally. Because internal comparison has certain
16 advantages. But it turns out, by the time we finished
17 the study, the comparison group was very small. And we
18 end up with no leukemia death in the internal
19 comparison group at all.
20 And 1 point out both advantages as well as
21 disadvantages of using internal comparison group. But
22 since 1 specified that analysis in my contract, in my
23 proposal to the sponsor, I went ahead and do that.
24 Besides, I also think it's a very interesting question,
25 whether to use internal comparison or not. When and
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how to use internal comparison. And this case demonstrates -- my study demonstrates that theoretically internal comparison groups are good. But in reality it's very difficult to get a group large enough to provide you with stable rates. And that's the reason for doing that.

Q. Where in Part 1 or Part 2 of your 1987 publications did you explain that you were going to use both internal controls and U.S. general population and the explanation for why? Could you find that passage for me?

A. Yes. Hold on for one second. Okay. On the page in the first paper, on Page 378 -

Q. 368 or 378?

A. I said 378.

Q. The 1987 publication? .

A. Yes. Part 1.

Q. General results?

A. Yes.

Q. Okay. I don't have that page. But go ahead. Let me see if it's in backwards or something. Yes, I have it. I'm ready. It was in the wrong place.

A. On the right-hand side column the paragraph that starts with "The SMR procedure." Do you find that?

Q. Yes. "The SMR procedure is usually satisfactory"?
A. Yes. There I talk about the use of both internal and external comparison groups and also talk about the advantages and disadvantages of both.
Q. Did CMA suggest to you not to use internal controls?
A. No, the proposal was prepared by us and we basically said we would use both internal and external.
Q. Did you have any disagreements with CMA before you published your findings in '87 about publishing the findings on internal controls as opposed to utilizing the general population as your control group?
A. Well, when you say "CMA," it's kind of like CMA is one entity. Okay. But actually, it's not. We have a committee from the industry that I contact in terms of the study. And, of course, some people agree with the way I analyze the data and some people may not agree.
Q. And what particular individuals did not agree with your use of internal controls?
A. I don't even remember the names of the individuals. But, you know, that's what I have done. And that's how I would like to publish. Nobody would stop me on that.

Q. Why couldn't you have used your entire group as opposed to just intermittently exposed? Why couldn't you have used all of your exposed for your estimated for the multiple myeloma as opposed to the general population?
A. That doesn't make sense to me, your question.
Q. What did you base your controls on for the intermittent exposure group for multiple myeloma? Maybe my question is poor. I assume that you -- did you use your entire controls? You had 3,000 controls, didn't you?
A. Yes.
Q. Did you utilize that or attempt to utilize that to calculate your expected for multiple myeloma?
A. No, I used the U.S. general population.
Q. And the reason that you say you couldn't use all of your control group was because it was too small?
A. It's too small, right.
Q. How many people were in the intermittent exposed group?
A. Over a thousand, 1 think.
Q. And you could not have used 3,000 controls as a comparison for your 1,000 intermittent control group?
A. I think you're getting confused. In epidemiology, usually we have two groups. One is what

we call the exposed group. That's the group where we're interested in finding out if there is an increased risk in certain diseases. The way to find that out is to compare the rate of disease or mortality in the exposed group to rates of the same disease in a comparison group. Okay? Do you follow me so far?
Q. Yes, I do.
A. So I think you're mixing up the intermittent group and the comparison group. Because the intermittent-group is one of the exposed groups. In my study 1 have two groups of people exposed to benzene. One is what we call continuous exposed group. The other one is intermittent exposed group. So those are the two groups combined we call the exposed group. And then I have the third group called the comparison group. That group would not be exposed to benzene indirectly, but may be exposed to only a low level background. And I call that the comparison group. That is our internal comparison group. But in my study I also have the external comparison group, which is the U.S. general population.
Q. Why couldn't you calculate then your expected for your entire exposed population the three multiple myelomas versus the control group?
A. Because the control group is too small.

Q. Can you explain to me, Dr. Wong, how your '87 study failed to find any AMLs?
A. Well, there are number of reasons. No. 1, in that study we have only seven leukemia deaths. So it's not that many. There were a couple of them unspecified. So it could have been AML. And we don't know for sure. The other thing is the exposure may not be high enough to really give us more than one or two cases of benzene related AML.
In another report, which 1 published in 1995 using the NIOSH data, I find that in order to have an increased risk of AML you have to have at least 200 PPM a year, if not higher. That is pretty high exposure. We just don't see that in my study.
Q. I understand what you published in '95. But aren't AMLs the majority of leukemias in the adult population?
A. The majority, did you say?
Q. Yes, sir. They compose the majority of leukemias in the adult population?
A. It's not the majority. When you say the majority, you mean more than 50 percent, right? I don't think it's more than 50 percent.
Q. Aren't they the largest group of leukemias found in adults, AMLs?

A. The largest type, right, yes.
Q. And would you agree with me that your study of the various chemical plants found a statistically significant relationship between other cell types of leukemia and benzene exposure in '87?
A. There seems to be some indication, but the increase was not comparing to the general population. The increase was not statistically significant.
Q. What about comparing to your internal controls?
A. Well, comparing to the internal controls since we have zero, anything is significant compared to the internal group.
Q. What was your SMR when you compared it to the internal group?
A. We shouldn't use the word "SMR" if we use the internal group.
Q. It should be the RR?
A. Yes, relative risk.
MR. DAMICO: What disease process are you talking about?
MR. HARTLEY: Leukemia.
THE WITNESS: Hold on for one second. Well, for leukemia it's infinite because you divide something by zero it's infinite. It's not well defined.

MR. HARTLEY: Q. Did you find a dose response relationship for leukemia in your study of the chemical plants?
A. For leukemia, let me see. Yes. If you look at Table 14 on Page 391.
Q. Of Wong 2?
A. Of 2, yes.
Q. Table 14, Wong 2. 39 -- all right.
A. You will see that under the column of relative risk, it's undefined. Because again we divide something by zero. But we were able to calculate a chi-square, meaning that -- well, the chi-square is a statistic that we used to determine to see if there is a dose response relationship or not. And that turns out to be not significant because the P value -- do you see that?
Q. Yes.
A. The P value was 0.088. In order for it to be significant it has to be less than 0.05.
Q. I must be missing something. On Table 14, Page 3'91, entitled "Mantel-Haenzel Relative Risk and Extension Chi-square for Lymplates and Hematopoietic Cancer, Leukemia, non-Hodgkin's lymphoma, non-Hodgkin's Lymphopoietic Cancer by Maximum Peak Occupational Exposure to Benzene Adjusted for Age and Race."

That's the table you're talking about?
A. Yes.
Q. And the leukemia undefined chi-square is 6.46, right?
A. Are you looking at Table 14?
Q. That's what's published in the literature, 14, yes.
A. The chi-square is .91.
Q. No, different document then, Doctor. I have Page 391 of Wong 2.
A. That's interesting. Hold on for one second. I know what the problem is. Table 14, the one you looked at, must be a copy of the journal paper when it's published. For some reason they duplicate Table 12. Go back to the previous page and look at Table 12.
Q. Yes. Table 12 is the same as Table 14.
A. Right. The one I have is a reprint which they corrected Table 14. I'm sorry for the inconvenience. But your Table 14 is actually the same as Table 12.
Q. What does Table 12 tell me?
A: 'Table 12 tells me that the chi-square is 2.91. It's not quite significant. Looking at maximum P exposure. And you are correct that when we look at cumulative exposure it is significant. And that's going to Table 12.

Q. So when we look at cumulative exposure in Table 12 there is a significance to the P .01 4evel value?
A. Right. That is based on internal comparison. And as you can see next to non-exposed, under the column of observed deaths is zero. Do you see that?
Q. Yes, I do.
A. So that means we did not see any leukemia in the small group of non-exposed people.
Q. Did you see it for non-Hodgkin's lymphopoietic cancer?
A. Non-Hodgkin's lymphopoietic cancer, we have two cases.
Q. And what was the chi-square for that, 364?
A. 364, which was not quite significant.
Q. It would be that there would be a 94 percent chance that it would be -- within a six percent chance that it would be within chance?
A. Right.
Q. Six out of a hundred it could be by chance?
A. Yes.
Q. So it's one-tenth of a point off in being significant?
A. Yes. In the paper I also add that even for that non significant trend, upward trend, that was

driven by the trend in the leukemia. Because leukemia, if you see the ICD code after leukemia is 204 to 207.
Q. Yes, I do.
A. And that is part of the non-Hodgkin's lymphoma cancer as well. Basically, I have included all the lymphopoietic cancer in that group, minus 201. 201 being Hodgkin's disease.
A. Now, what was the cumulative exposure in parts per million? Do you see that, 180 to 719 cumulative exposure?
A. Yes.
Q. That would be under ICD 8, 200, 202 and 203, which would include multiple myeloma, correct?
A. That would include -- let's see.
Q. 203 is multiple myeloma, correct?
A. 200 is lymphosarcoma which is part of the non-Hodgkin's lymphoma. 202 is, again, non-Hodgkin's lymphoma. And 203 is multiple myeloma. So it's a combination of both non-Hodgkin's lymphoma, as well as multiple myeloma.
Q. So you combined them for statistical purposes in this analysis based on cumulative exposure, correct?
A. Yes, because 1 could not separate 202 from 203 in the analysis.
Q. What tables, Doctor, have your breakdowns of

diseases?
A. Well -
Q. Is that Page 358 or 368? Or where did you identify the cell types?
A. The cell types?
Q. Yes, sir.
A. That would be the last table, Table 16 on Page 393.
Q. For the cumulative exposure classed, group of 200, 202 and 203 of 180 to 719 parts per million months you had a relative risk of 2.23, correct?
A. What table are you looking at?
Q. I'm back on No. 12.
A. Okay. Can you repeat the question?
Q. Sure. For the class categories of 200, 202 and 203 with 180 to 719 parts per million months cumulative exposure you had a relative risk of 2.23?
A. Yes, sir.
Q. And you also had a chi-square for that trend of 14, correct?
A. 14 applies to the trend. The trend meaning that comparing all four categories of exposure, non=exposed, less than 180, 180 to 719 and 220 and over. That 0.14 applies to all four.
Q. All four groups?

A. Right. That's what we mean by trend, including all four categories. And that clearly says it's not significant at all.
Q. It was at .71. Where did you break out - what table do I need to look at for your SMR for 200, 202 and 203? Not the trend of exposure, but for the cause of death.
A. For the overall exposed group you may want to go to the first paper, Table 9.
Q. What page is that on?
A. On Page 374. If you look at the table, probably in the middle of the table, you will see the lymphatic and hemopoietic cancers.
Q. 200, 209?
A. Right. And underneath that the standard grouping are the following, the following four groups, lymphosarcoma, which is ICD Code 200. Do you see that?
Q. Yes.
A. And the next one is Hodgkin's disease, 201. And then the third group is leukemia from 204 to 207. Do you see that?
Q. Yes, sir.
A. And the last group is the residual, which is other lymphatic tissue cancer which would include ICD Code 202. By the way, that is a typo there. It should

be 202 and not 22. And then 203. And then 208 is really a very, very small group. For all practical purposes the fourth category, other lymphatic tissue cancer I would say would be 95 percent, would be either 202 or 203. And, as we said, 202 is non-Hodgkin's lymphoma. And 203 is multiple myeloma.

In the old terminology they break non-Hodgkin's lymphoma into two groups, lymphosarcoma being ICD Code 200. And then what they called "other lymphomas, which would be 202. And that's why I said even back in the '80s I said certain groups we should combine. And this is a very good example where we should combine. Because in looking at 200 or 202 separately you don't get a good picture. You should combine 200 and 202 in order to get a full picture of non-Hodgkin's lymphoma. By the same time, if you want to look at multiple myeloma, you've got to look at 203. But in the statistical tabulation back then is combined, 203 is combined with 202 in this group. So what I'm trying to say is that that's why in my analysis I was able to combine 200, 202 and 203 together, adding the first and the fourth category together.

Q. Out of 202, 203 and 208, what percentage is 203, in your opinion?

A. Forgetting about 208, I think 202 and 203 may be roughly about the same, percentage wise.
Q. 50-50?
A. Roughly, yes. Well, it depends on age group and so on. But roughly I would say about 50 and 50.
Q. What about in a younger population?
A. I really should look at the statistic before I answer the question.
Q. Isn't multiple myeloma generally considered a disease of older age?
A. Yes, older age. People in the older age group would have much higher rates.
Q. Statistically it occurs in the sixth decade of life?
A. Start ticking off in the 50s.
Q. That would be the sixth. decade of life. Zero to 9 would be the first decade. So if you had a population that was under 50, what would the percentage breakdown be, in your opinion, between 202 and 203?
A. I don't know. 1'd have to look at it.
Q. Doctor, you've done some studies on gasoline?
A. Yes.
Q. And did you present at the Gasoline Symposium in Canada?
A. I did not present any paper in Canada on

gasoline.
Q. Were you present during the presentations?
A. Did you say Canada?
, Q. Yes, sir.
A. No, I did not.
Q. Were you present?
A. In Canada?
Q. In Miami.
A. I just found my note. It's in Miami. Miami is not in Canada, for your information.
Q. Miami may be Canada if you live pretty south. But anyway.
A. 1 think what you're referring to is 1990. Early 1990s there was a symposium on gasoline. Yes, I did present some results of my gasoline study at that meeting, yes.
Q. Was there also a presentation about the development of multiple myeloma from gasoline at that symposium?
A. By whom?
Q I'm sorry?
A. By whom? By me?
Q. I can't remember what the guy's name was. 1 didn't bring my environmental health perspective with me.

A. You have to tell me the name because I didn't go to everybody's presentation, especially when the meeting was held in Miami.
Q. It's warm down there. How about a guy named Snadder?
A. Ralph Snadder from Exxon?
Q. Yes. Did he present some study on gasoline and multiple myeloma?
A. He present -- well, okay. I think why you get confused with Canada. He present the results of a Canadian study that he did back then. He present some preliminary results. And to be quite honest with you, I don't remember -- I do not remember exactly what he said at the meeting.
Q. Did you present at Benzene '95?
A. Oh, yes. We have -- actually, we have two presentations at Benzene '95. You are referring to the conference at the Rutger's University, right?
Q. Yes. Did you speak? Because I was there and 1 don't remember you speaking.
A. -As I said, we have two presentations. One presentation was on cell type specific leukemia in the - petroleum industry. My co-author actually made the oral presentation. His name is Jerry Raabe. And at the same time I also have a poster presentation on

multiple myeloma.
Q. Did you do that with Dr. Lamm or did you have your own poster?
A. I did not do any work with Dr. Lamm.
Q. Doctor, you presented in Miami on gasoline workers?
A. 1 present some preliminary analysis, yes.
Q. And what was the topic of your presentation? What were you looking for?
A. Well, I was looking for -- basically we present mortality analysis by different causes of death. Of course, the interest was leukemia. One of the main interest was leukemia, because in gasoline we have 2 to 3 percent of benzene. So the focus was on leukemia. But at the same time kidney cancer was another interest because of some animal studies.
Q. What population did you study, terminal workers or distribution workers or what?
A. I think they mean the same thing, terminal workers and distribution workers.
Q. As that who you studied?
A. Yes. 1 have two groups of people in the gasoline study. One is what we call land based terminal workers. The other one would be what we call marine workers. These are the people who are

responsible for the transportation of gasoline and other petroleum products on barges or on ocean tankers.
Q. Were there criticisms of your control group for having a small SMR for all causes in your distribution workers?
A. In the land base group we found a low SMR for all causes when we compared to the general population.
Q. What about your control group, did your control group have low SMR also for all causes?
A. That was our control group. The control group was the general population.
Q. The control group was from the United States?
A. Yes.
Q. Were there criticisms for utilizing that as a control group by Dr. Enterline?
A. I think Dr. Enterline raised a question. Actually, Dr. Enterline was one of the reviewers for our study all along. So he was looking at the same set of data over my shoulder for three, four years while we were doing the study. And he raised the question, you know. why we see such a deficit in our study. And we tried to explain that.
One of the reasons may be the people who work with gasoline may smoke less. Maybe that's one of the reasons. In fact, there was another person who was an

outside reviewer for the conference. I think that was Dr. McLaughlin at the National Cancer Institute at that time. And he gave us some very interesting insight. He said that the general population, sometimes you have people who have very high rate of heart disease, as well as high rate of non-malignant respiratory diseases and so on. And maybe that accounts for the deficit we saw in our study. But regardless of what the explanation is we also have the second group. The second group is the marine distribution workers. And certainly for that group we don't have that problem. We don't see an unusually low SMR.

Q. What did Dr. Infante think of your control group?

A. The general population?,

Q. Yes.

A. I don't know what he thinks of that.

Q. Did he explain that to you when you were doing the study, what his thoughts on your control group were?

A. He was not a reviewer of our study. I did not ask him for consultation or advice.

Q. How about at the meeting on the gasoline symposium, did he comment on that meeting on the

control group?
A. He commented on a whole bunch of papers. And I don't remember exactly what he said on our paper.
Q. Now, we got into all this because of specificity and Bradford Hill. Would you agree that specificity must not be overemphasized in determining the causation of a particular substance to a particular disease?
A. I don't quite understand exactly what you mean by "cannot be overemphasized."
Q. Bradford Hill says in his 1965 article that established the criteria, "We must not, however, overemphasize the importance of the characteristic." That being specificity. Do you agree that some epidemiologists overemphasize the role of specificity in determining a causal relationship?
A. I don't think so. I think when Bradford Hill used the term "specificity" in that content he meant that a chemical may be able to cause more than one disease. We cannot say that simply because Chemical X cause& Disease Y that would imply that Chemical X does not or is not capable of causing another disease. I think that's what he meant. Okay?
And I totally agree with that. Certainly smoking can cause more than lung cancer. It can cause

other disease as well. But the evidence has to come from specific data, specifically related to death disease. Okay? If you show me that benzene can cause multiple myeloma specifically, you have data on that, then I will buy it.
Q. That also goes right to Bradford Hill's other point. And that is that you should not be totally wedded to statistical significance. If there's an elevated risk that is not statistically significant on a recurring basis then there should be some consideration of an association and causality. Would you agree with that?
A. Absolutely. And that's why I've been doing this meta-analysis. And that is when you have a small study there may be an elevation. But the excess or the elevation may not be statistically significant. And that can be due to two things. One is the sample size is not enough, not big enough to pick up that difference. Or it can be that indeed there is no relationship.
And I totally agree with Bradford Hill that if you do see an elevation from study to study, then there is something. Now, meta-analysis would be able to handle that because if you see some consistent elevation when you combine all the studies into the

meta-analysis, you will certainly see a statistically significant increase because you increase the sample size by doing a meta-analysis.
Q. Do you also agree with Bradford Hill's statement that, "No formal test of significance can answer those questions of causation. Such tests can and should remind us of the effects that the play of chance can create and they will instruct us in the lengthy magnitude of those effects. Beyond that they contribute nothing to the proof of our hypothesis." Do you agree with that statement?
A. I agree that when you do a statistical test by and in itself is not the whole thing. You've got to look at the whole picture. And that's the reason why in 19- --I believe 1965, Bradford Hill formalized the criteria. So you should not just look at one statistical study. You really have to look at what kind of chemicals are we talking about: how specific exposure was in the study; how long the observation period is, and how many studies reporting an increase or a lalc of an increase. You have to take all that into consideration.
a. Which goes back to the idea that if you have elevated non-statistical risk for a substance on a recurring basis and you have biological plausibility as

well as a temporal relationship and you have strength from a few studies, or a great deal of studies, that you can make a determination even though there is no statistical significance of any epidemiologic study that Substance X caused Disease Y, correct?
A. I agree completely. It's unfortunate that you did not get my November 25th report earlier because that's exactly what 1 did. I analyzed the literature, including some of my own studies on tissue of benzene exposure and multiple myeloma and basically applied the criteria. And the bottom line is when you look at all the studies taking into strength of association, consistency of association and so on, those response, et cetera, the conclusion is there is no connection between benzene exposure and multiple myeloma.
Q. I understand your conclusion.
A. I just want to explain to you how you get there.
Q. We'll get to that in a little bit. How many of the Bradford Hill criteria do you feel as an epidemiologist are needed to evaluate and establish the likelihood that Substance X caused Disease Y? Do we need all of them?
A. I don't think we can specify we need four out of six or five out of six or all six. I don't think we

can do that. I think we really have to use our professional judgment to see whether there is a consistent picture, whether the risk is high enough for us to rule out confounding, whether there is any dose relationship and so on. I don't think we can quantify that.
Q. Is a dose response relationship enough to establish causality?
A. It's one of the criteria that we should use.
Q. Is that in and of itself enough? If we have a temporal relationship is a dose response relationship enough to establish causality?
A. I think you also need consistency from different studies.
Q. How many studies do we need to be consistent on a dose response basis with a temporal relationship before we can say there's causality?
A. Again, 1 don't think we can quantify that. And it's somewhat meaningless to ask how many studies. How big are the studies? Are we talking about small studies? If we're talking about small studies, we need a lot of them. If they are good, well-designed large studies, maybe we don't need as many. I think it all comes down to when you combine the studies together what does the mete-analysis tell you. How big is the

95 percent competency.
Q. Can one study negate another study by itself?
If we have one positive study and we have one negative study, does the negative study tell us that the positive study was just by chance so there's no relationship?
A. If the two studies are about the same size, the same quality, then I would say we have inconsistent findings and we don't know what's what.
Q. Which would require the person doing the evaluation to utilize their own professional judgment and come to a conclusion as to whether Substance X caused Disease Y, correct?
A. I'm not so sure. If you have two studies and assuming that the two studies are the same in terms of quality and sample size and so on, if you have conflicting information, 1 don't see how one can come to a conclusion either way.
Q. But that would be the professional judgment of the evaluator, would it not? You may say you cannot come to a conclusion. But someone else may utilize their own professional judgment and say, "I think one - study is either flawed. I think one study is better than another one." Et cetera. For whatever reason they may utilize that same data and their own

professional judgment and come to a different conclusion than you would, correct?
A. I think we're changing the question slightly now. Because when I said we cannot come to a conclusion, that's based on the assumption that the two studies are similar in terms of quality and sample size. That's what I said. That's why I gave you that preamble. Now, if you say that one study is clearly better than the other, of course, we would put more weight on the better study. That's just logical.
Q. Were you criticized by Dr. Savidz from North Carolina as a result of your 1995 publication in Occupational and Environmental Medicine entitled "Risk of Acute Myeloid Leukemia, Multiple Myeloma and Workers Exposed to Benzene in Regard to Specificity"?
A. I do not consider that as criticism. Basically he said that I only looked at AML in my analysis. And he said that if I add up all the other leukemia cell types then I would also find a statistically significant increase in cell types other than AML.
And 1 have responded to his inquiry, I think, quite adequately in my response published alongside with his letter. And that is it's meaningless to combine CML, ALL, and -- what was the other one -- the

other three kinds. ALL, CLL and CML together. Okay?
His whole approach is if we add all those numbers
together we would have enough cases to justify the
analysis from the statistical point of view.
And my response to him was a medical question,
a health related question is more than just statistics.
It would be meaningless to combine them because CML is
not the same as ALL. ALL is basically a childhood
leukemia. CLL is not. And they have different risk
factors and so on. Okay? And I point out to him that
1 did not say that my analysis proved that there is no
relationship between benzene exposure and CML. I
simply say that the data in that study did not provide
us with enough data, sufficient data to show either
way.
In order to look at the relationship between
benzene exposure and CML or other cell types we have to
go to other studies. But I disagree with him that an
analysis is based on statistics only. An analysis must
be able to make biological sense. And I think he
failed. in that.
Q. Now, when he wanted to combine all the
leukemia, as you did in 1987 and as you did before OSHA
from the Pliofilm cohort, all those leukemias
originated from the same stem line, didn't they, or

1 don't they?
2 A. They don't originate from the same, what we
3 call stem cells. That is one of the confusions. And 1
4 sympathize with you because I had a tough time trying
5. to understand that in the early days.
6 In the early 1980's, when people referred to
7 stem cell, most people thought that there is only one
8 kind of stem cell. But it turns out that later on, at
9 least I find out later, that there are different kinds
10 of stem cells. And some are more committed to a
11 specific cell line than the other. So when you say all
12 the leukemia cells coming from the same stem cell, it's
13 not quite true. They are coming from different cell
14 lines. And Professor Irons, at the University of
15 Colorado, certainly has written a lot about that. He
16 would be the expert to talk about that.
17 . Q. Would you agree with me that CML and AML come
18 from the same fluid-potent stem cells?
19 A. I'm not so sure I would agree with you. I
20 think specifically Richard Irons wrote a paper, or more
21 than the paper, saying that the metabolites of benzene
22 would only have an effect on AML, but not on CML.
23. Q. What does the metabolite of benzene, the
24 hydroquinone, have to do with CML? Don't other
25 investigators believe that there are other avenues that

benzene utilizes to attack the stem cells in creating the malignancy?
A. I don't know about that, whether there are some people who believe in that or not. Certainly I think Richard Irons is the authority on stem cell. And he doesn't think that benzene can cause other cell types besides AML. And from the epidemiologic point of view, if indeed benzene can cause CML, then we should be able to see that in epidemiologic studies. I have yet to see a study linking benzene directly to CML.
Q. Didn't you have some CMLs in your study, Doctor?
A. I think you misunderstand or you may not be able to interpret epidemiologic study.
Q. That may be true, because I'm just a country lawyer, Doctor. I have no idea what I'm talking about, to be honest with you. I'm just wondering, didn't you have some CMLs in your study?
A. Clearly if you're implying that if I have CMLs in my study then, therefore, benzene must be able to cause CML.
Q. I'm not asking you that. First of all, did you have CML in your study?
A. Yes, I have CML in my study. And that is quite to be expected because whenever you look at a

group of 3,000, 4,000 individuals, let's say, lawyers.
If I have 3,000 lawyers and I observe them over 40
years I can tell you for sure I would expect to see
some CMLs.
Q. How many CMI-s did you have?
A. I don't remember. Maybe I have two. Not that
many cases.
Q. What number is CMI, what 1CD 8 number?
A. I think it's 205.1.
Q. If it's 205.1 it looks like you had two of
them.
A. That maybe right.
Q. Now, then, weren't CMLs found in the Chinese
cohort?
A. It depends on which study, which paper you're
talking about.
Q. What about the most recent update with the
lead author being Hayes?
A. I don't have that paper in front of me, so I
won't be able to tell you the numbers.
Q. Do you have the Quinn or the Travis update in
front of you?
A. No, I don't.
Q. Do you have any of the Chinese literature in
front of you?

A. I have one, which is Yin, 1996. By the way, in that paper, Yin certainly analyzed multiple myeloma as a separate group. And he didn't find a single group of multiple myeloma in a cohort of 70,000 workers exposed to benzene.
Q. Multiple myeloma is a very, very, very rare disease in China, is it not, Doctor?
A. Yes, but at the same time if benzene can cause multiple myeloma certainly you would see at least one or two cases.
Q. What if the Chinese do not develop multiple myeloma generally because of genetic differences, you would not see a rise regardless of exposure, would you?
A. Are you saying that we don't see multiple myeloma at all in Chinese?
Q. No, I don't think you can say that. I think you can say, though, that it is very, very rare.
A. Rare to the extent that we don't see a single case in 70,000 people over many years?
Q. Doctor, how many multiple myeloma are in the controls?
A. In what study?
'Q. In any of the Chinese study?
A. I'm not so sure they list that.
Q. He had to list it if he made a comment that he

1 didn't have any multiple myeloma in his exposed, didn't
2 he?
3 A. No, he did not list that.
4 Q. Did he just make the comment he didn't find
5 any multiple myeloma in his control group?
6 A. Yes, in the table he simply said it is zero.
7 Q. Did he list the expected?
8 A. He didn't list the expected.
9 Q. In the body of the report?
10 A. 1 don't see that.
11 Q. At least not in the '96 body?
12 A. Not in the paper that I have in front of me.
13 MR. DAMICO: The court reporter needs like a
14 two-minute break.
15 MR. HARTLEY: Okay.
16 (Brief recess.)
17 MR. HARTLEY: Q. Doctor, in your abstract to Part
18 2 of the 1987 publication you indicate there is a dose
19 response relationship between cumulative exposure and
20 non-Hodgkin's lymphopoietic cancer was a borderline
21 significant P equaling to point zero P6.
22 A. We looked at that before.
23 Q. We talked about that before?
24 A. Yes.
25 Q. You also mentioned -- I want to get one thing

clear. Are you saying that AML and CML do not originate from the same stem cells, as far as you understand?
A. I better not answer that question because 1 really should. go back and look at some of the Richard Irons papers. What I said before was that the metabolite of benzene only picked on, according to Richard Irons, only picked on AML, not CML.
Q. I'm not concerned with what the metabolite does. I'm concerned about what the stem cell AML and CML originate from.
A. I don't know if they originate from the same committed stem cell or not. That's what I don't know.
Q. How about the primitive stem cell, are you familiar with whether AML and CML derive from the primitive uncommitted stem cell, the same one?
A. Well, when you use the term "uncommitted primitive stem cell" then everything has to come from that.
Q. So everything comes from the primitive uncommitted stem cell, does it not?
A. Right. But that theory is really out of date now.
Q. It is out of what?
A. Out of date. That we should look at committed

Q. Are you going to testify that benzene does not cause non-Hodgkin's lymphoma?
A. In this case?
Q. Yes, in this case. If that question is put to you by me during cross-examination, would you say that benzene does not cause non-Hodgkin's lymphoma?
MR. DAMICO: Well, I'll object on relevance.
MR. HARTLEY: Q. That means you can answer the question during deposition.
A. I have not done a complete search on non-Hodgkin's lymphoma and benzene exposure. My assignment coming here today was to look at benzene exposure and multiple myeloma and that's what I have done. If you can convince the lawyers to authorize me to do work on non-Hodgkin's lymphoma and benzene exposure, if I have time I'll look into that.
Q. What would you charge Mr. Damico by the hour to do that, Doctor?
A. I charge $380 an hour.
Q. I kind of doubt he's going to want you to do that.
MR. HARTLEY: But, Dave, if you do, let me know.
MR. DAMICO: I'll send you half the bill.
THE WITNESS: Maybe if you are interested in finding out the truth, maybe you can pay me to do that.

1 Just kidding you. You can ask the next question.
2 MR. HARTLEY: a. Thank you. I was going to
3 respond with a smart remark but I elected not to.
4 Does not multiple myeloma come from the same
5. cell line as non-Hodgkin's lymphoma? That was the
6 basis for the first question.
7 A. I don't know whether they're coming from the
8 cell line, the same, and what stem. I think we should
9 really go back and look at that. And again, I'm not an
10 expert in the area. So I would not be the one who
11 would even do that research. I think, again, someone
12 in the area of hematology or toxicology would be able
13 to answer that question and not me.
14 Q. How do you know what disease category each of
15 the various malignancies should be grouped in when you
16 do your analysis? Do you just go through and say this
17 is 2 and this is 203 so we can put them together or do
18 you have some understanding of that process?
19 A. I have some understanding based on the review
20 papers done by people like Richard Irons and some other
21 hematologists. As I point out to you in my report,
22 there's some textbooks on hematology, as well as review
23- articles, indicating that multiple myeloma,
24 non-Hodgkin's lymphoma and leukemia are different
25 diseases. And when 1 look at the publications by the

National Cancer Institute, multiple myeloma is a distinct entity. They don't put multiple myeloma and non-Hodgkin's lymphoma together as one analysis.
Q. One final question about the Chinese study and then we'll move on. When you are doing a study of a population to determine whether there's an elevated risk of a particular disease you certainly want to look at a population that develops that particular disease entity before determining whether or not that would be the logical cohort to study, don't you?
A. I don't follow your question. It's kind of long, Can you repeat that?
Q. Sure. When the study of the Chinese cohort was made going in, if we know that multiple myeloma is a very rare disease among the Chinese, it would not make one surprised to learn that there is either a deficit or no increase in multiple myeloma for the benzene exposed population, would it?
A. When you say "rare," I don't know how rare that is. Because they did not report -- at least based on the article I have in front of me -- we don't know what the expected would be. 1 guess the proper question to ask is in the Chinese cohorts of 70-plus thousand workers exposed to benzene, that's what they claim, I would like to know how many multiple myeloma

1 they expect to see in that cohort. Okay? If they only
2 expect one case or two cases and they don't see any,
3 then I would say that's reasonable. But on the other
4 hand, if they expect to see five or six or even more
5, cases and they don't see any, then I would say why is
6 that? I want to look into that.
7 Q. Is it better to study populations with a
8 greater disease frequency or one that has less of a
9 frequency of the disease you're studying?
10 MR. DAMICO: That's a difficult question. Did you
11 say "better"?
12 MR. HARTLEY: Q. Yes. Would you study a group of
13 30-year-old males for prostate cancer?
14 A. I think I understand what you're asking. If
15 you study a population in which the rate of disease is
16 rare, then you need a large population in order to come
17 up with something statistically meaningful. Is that
18 what you're asking?
19 Q. Yes.
20 A. By the same time -- well, I shouldn't say
21 that. On the other hand, if you study something that's
22 very common in the population, most likely that implies
23. theft disease in that population would have many, many
24 different risk factors. And that would create a
25 different set of problems.

Q. What about smoking, is smoking an example of that?
A. Well, it depends on what disease you're talking about. Certainly for tobacco-related diseases that would be a problem.
Q. For lung cancer. There are many risk factors for lung cancer other than smoking?
A. Was that a question or did you just comment on that?
Q. That's true, right?
A. What was true?
Q. That there are other risk factors for lung cancer other than smoking?
A. Oh, absolutely.
Q. And that would be an example of what you're talking about?
A. Yes.
Q. Are there risk factors for multiple myeloma?
A. Yes, I think so.
Q. Could you delineate those for me?
A.' I think I might have listed a number of them as an example in my report, dated -
Q. I didn't get that report yet. I'll have to disconnect and call down.
A. On Page 3.

Q. Of which report, Doctor?
A. The report dated November 25th, 1997.
Q. Why don't you list the risk factors for me.
A. Based on a number of studies, at least some risk factors has been identified in those studies. And that would be virus, use of laxatives, a history of diabetes, and, well, talking about cigarette smoking, including cigarette smoking. Those would be examples of risk factors of multiple myelorna.
Q. How does cigarette smoking relate to multiple myeloma? I believe you testified back in '95 that based upon your review of the literature at that point in time you did not think that there was a sufficient connection between smoking and multiple myeloma for someone to opine that there was a relationship or a risk factor.
A. I don't know where -- I just list them as risk factors that have been identified by some people in their study. I did not say that the relationship is definitive. Again, the lawyers have not asked me to do a complete research on those issues. And 1 didn't spend much time on it. In fact, those are the studies that I come across when I look at benzene and multiple myeloma. They also talk about other risk factors. I just list them as an example that multiple myeloma may

A. When you talk about studies, the only thing I can think of would be the study published by Rinsky - R-i-n-s-k-y. Rinsky in 1987. But, as you know, subsequently, I have updated the study and published in 1985. And basically we found that there was no relationship. Even in the 1987 Rinsky paper he did not find a dose response relationship between benzene exposure and multiple myeloma.
Q. But he nevertheless found an elevated - didn't he find as SMR of three point something or four point something that was statistically significant?
A. Close to four.
Q. And that cohort indicated or that study demonstrated that there was an elevated risk of multiple myeloma in this cohort, did it not, in '87?
A. On the surface it does. But at the same time, if you go back to the Hill criteria, then the Rinsky 1987 paper only meets the first criteria. And that is a significance of association. It does not meet consistency. Because when you look at other studies you don't find that. It does not meet dose response, because even Rinsky himself admits that there was no dose response relationship.
Q. Well,, you can't make the dose response relationship on four cases, can you?

A. Well, but you based the findings on four cases, the same four cases. You said that was a significant increase.
Q. Is there enough statistical, Doctor, for four cases to provide you with a dose response relationship if you're breaking up the doses as Rinsky did?
A. Well, actually there are two questions. One is indeed you see an upward trend, but the trend may not be statistically significant. And that may be because your study is too small. It does not provide you with enough cases. So that you can say that there is a significant trend. But there was no trend at all. I'm not talking about whether it's significant or not. Three out of the four cases were short-term workers. One guy worked there for like four days.
Q. 1 understand that. But when you made your comments on the study to Gulf Canada in 1983, didn't you indicate that the cohort definition appeared adequate?
A. The cohort definition of what study?
Q Of the Pliofilm study.
A. Yes.
Q. Didn't you indicate that the cohort definition appeared adequate and the cohort definition included anyone who worked on the job site for one day? That's

what you told Gulf Canada, that that definition was adequate.
A. Was inadequate, right?
Q. No, adequate. A-d without an I-n. Adequate. Wasn't that your statement to Gulf Canada in 1983?
A. You have to show me the document in 1983. I want to read the whole paragraph. I don't want you to pick out one sentence and read that to me. I don't know what content that would be.
Q. Do you remember making comments on the NIOSH study of leukemia and benzene studies to Gulf Limited in August of 1983?
A. I remember making some comments. I don't remember the date. It was sometime ago. But certainly, if you want me to explain to you, you've got to show me the document.
Q. It's hard when I'm on the East Coast and you're on the West Coast. But the first sentence of your second paragraph says, "The cohort definition appeared adequate." At that point in time you did not make a comment or contest the adequacy of the cohort definitions. Now, you did go on to point out though, and I will be fair to you, that, yes, there were some people who were only on the job site for a very, very short period of time. But nevertheless, you indicated

1 that the definition of the cohort appeared adequate.
2 Do you recall that?
3 A. Yes, but that has nothing to do with the dose
4 response relationship. What I'm trying to point out to
5 you in terms of the multiple myeloma result is that
6 three out of the four cases were short-term workers.
7 Certainly that doesn't give us any indication of those
8 responses at all, significant or otherwise.
9 Q. If three out of the four were short-term
10 workers, when you reviewed the results for Gulf Canada
11 why didn't you say that the cohort definition is
12 inadequate because it over includes and has the
13 possibility of including people who were only on the
14 job site for one day?
15 A. No. No. No. I think you're mixing up two
16 things. One is the cohort of definition. The cohort
17 of definition, you can define it any way you want.
18 Most people would define it as anyone who worked at the
19 site for at least three months, six months or a year.
20 But NIOSH decided to use one day. Okay? And assuming
21 that indeed, you know, those people worked there for at
22 least one day, that is a correct definition. That's
23 okay. But that means they have to include a whole
24 bunch of short-term workers.
25 I would not design a study as such because you

are wasting your money and effort on a lot of people with short-term exposure at that particular site. But those people may have a lot of other exposures from elsewhere that we don't know, exposure to other chemicals. What I'm saying is in terms of the multiple myeloma finding there are only four cases. But three of them work at that site for a very short period of time. How can we say that those three multiple myeloma deaths were related to the exposure at the site when they were short-term workers, when they spent most of their time, their career elsewhere? That's a distinction between a cohort definition and a causation analysis.

Q. I understand that. But since that was the definition of the cohort we're stuck with those one- or two-day workers and their development of multiple myeloma, are we not?

A. Yes. But the interpretation has to be appropriate. If in the Rinsky study they see all four cases among people who worked there for 20-some years with a lot of exposure, then I say maybe there is something between benzene exposure and leukemia. But that's not the case. It's the opposite.

Q. Do you think that no one considers benzene to be a risk factor for multiple myeloma?

A. Certainly you do. Certainly the experts you hired do. I can't say that statement. That doesn't make sense, that question.
Q. Well, do you believe that it is recognized within the scientific community that benzene is not a risk factor for multiple myeloma?
A. Certainly not based on properly done epidemiologic studies.
Q. Do you know who Bernie Goldstein is?
A. Yes, .I know who he is.
Q. Are you familiar with his work?
A. Basically he's a toxicologist. I don't think he's an epidemiologist. I really don't know his work that well.
Q. Would you agree that he is well-known in the area of benzene related diseases?
A. He is well-known in terms of toxicology related to benzene, not on benzene epidemiology.
Q. And he has expressed his opinion, has he not, in, I believe, the early 1990s, that benzene more probably than not causes multiple myeloma? Has he or has he not done that first? And then you can explain.
A. I have seen some of his editorial comments. And basically I think he relies on a couple of case reports to support his opinion. And he may even rely

on the Rinsky 1987 paper. But that is incomplete literature review. And most of that is based on case reports. And I don't think case reports are appropriate for causation analysis.
Q. He looked at the biological possibility, did he not, as a toxicologist?
A. Yes, it's kind of interesting. When you look at the biological possibility certainly if you find something, then you look at whether it is possible or not. But saying something that's possible is no proof of causation. The bottom line is if indeed you do have a relationship between benzene exposure and multiple myeloma you should be able to see that in epidemiologic studies.
Q. But isn't it more rare than leukemia?
A. It may be rarer than leukemia, but, still, we have so many studies. When you have some time, read my report. You will see that the studies indicate that there is no increased risk. It is not just based on one or two studies.
Q. "We were talking about the fact of the scientific community, would there be anyone who would think it would be related. And naturally you pointed to me, as the plaintiff's counsel, and the experts that I have hired.

Goldstein is not hired by either side and was in fact a consultant to API at one time. And he has concluded that multiple myeloma is probably caused or is more likely than not to be caused by benzene exposure.
A. I don't think that statement is completely true. Dr. Goldstein certainly has some project from API in the past. He might have been a consultant to API. But at the same time, you know, a lot of us have been consultants to OSHA as well. I don't think how much that can buy us. The part that you said Dr. Goldstein was not hired by -
Q. Either side.
A. -- either side, I don't think that's true. I think at least in one case I ran into him. He was hired by the plaintiff attorneys.
Q. Have you ever testified for the plaintiff in a benzene case, Dr. Wong?
A. No.
Q. Have you ever been asked to?
A. Some people asked for my opinion and I indicated to them. And I think that that was the end of the conversation.
Q. Have you ever advised plaintiffs counsel that have asked you that there would be a conflict of

interest in light of all the work you do for industry?
A. I don't see that as a conflict of interest.
Basically I just report on the findings that I have done, as well as interpret similar studies done by other people.
MR. HARTLEY: I'm going to stop for five or ten minutes here. I'm going to stop and see where the report is because they haven't brought it up.
MR. DAMICO: Let's go off the record.
(Discussion off the record.)
(Brief recess.)
MR. HARTLEY: Q. When you do your meta-analysis and you combine various epidemiologic studies, do you review those studies to note whether they are of equal value?
A. When I combine those studies, for example, refinery studies, the cohort definition would be quite similar. And the exposure would be quite similar, as well. I don't combine refinery studies with paper mill studies, for example. I don't combine refinery studies with truck drivers, so to speak. So at least there is some common exposure across the studies.
Q. What about is latency important in those various studies if you're looking at them when you have a younger cohort as opposed to an older cohort in the

various studies you're going to attempt to combine?
A. Certainly that creates somewhat of a problem when you combine the studies because different studies use different cut points for latency or for exposure, length of employment, length of exposure and so on. And that being the case, it would be impossible to combine different studies by length of exposure and length of latency or by any other exposure measurements. That is somewhat of a problem. But to the extent that when you do a meta-analysis you also need to look at individual studies. And we did that. And we do some, what we call stratified meta-analysis. In other words, we look at the refinery workers as one group. We look at the distribution workers as another group and so on and see where there are any major differences between different groups.
Q. How do you account for short follow-up time in the various cohorts?
A. We tried to do that. In fact, I don't know whether - 1 assume you may not have a copy of our multiple myeloma meta-analysis paper which was published last month in the Journal of Regulatory Toxicology and Pharmacology. In that paper, actually, we tried to group the studies of short-term follow up,

well, shorter follow up. Let's see. That's Table 6 in that paper.
We grouped the studies with length of follow up less than 20 years as one group. And we grouped studies whose length of follow up is 20 years or more as another group. And in that table I present the SMR for both groups of studies and they are about the same. In one group the SMR was 0.99. In the other group the SMR was 0.92. So they are very comparable.
Q. When you are looking for confounders in a particular study; suppose we're doing a study for the relationship, for simple terms, of cigarette smoking and lung cancer. What types of confounders would you be looking for when you are actually doing the study?
A. Well, if the interest is lung cancer, then you have to find out what else do we know about lung cancer. What other risk factors we have for lung cancer and whether those risk factors would be more or less the same, similar, between the exposed group, the group who smoked, and the group who do not smoke.
Q. Now, this may be a stupid question, but what's the risk factor?
A. The risk factor for lung cancer?
Q. No, a risk factor in general.
A. Risk factor meaning that if you do have that

risk that risk will be increased to a certain disease as a result of that. We would say that smoking would be a very strong risk factor for lung cancer.
Q. To be a risk factor does there have to be a determination of causality?
A. In a way it does because without that you don't know that that is a risk factor.
Q. Who determines risk factors, the author or the scientific literature?
A. I don't think it's black and white. The authors present--- usually the authors present the data, present the analysis and make some kind of interpretation of the data and may or may not come to a definitive conclusion in terms of causation. But other epidemiologists looking at the same data can either agree or disagree with the author.
Q. As to the confounders or as to the causation analysis that he's trying to either prove or disprove?
A. All of the above. I mean, you know, other people looking at a specific study may disagree with how the data should have been collected, how the data should have been analyzed or the interpretation was not supported by the analysis. The criticism, the agreement or disagreement can be throughout the entire research process.

Q. When you're looking at risk factors for a particular disease process you only utilize the risk factors that have been determined to be causally related to the particular substance under investigation?
A. I'm not so sure I understand your question. But when you do a study you want to include some of the confounders. You want the control for some of the confounders. You would like to have information on the confounders so that you can adjust for that in the analysis or control for that in the design. One simple confounder that everyone of us would agree would be age on the disease process. For example, if I have a group of workers exposed to benzene and I can calculate the rates of leukemia in that group. I want to compare that to the general population. But, of course, 1 need to adjust for age because we know that leukemia rarely changes as we get older. So age is a confounder to that extent. And we need to adjust for that in the analysis. And that's why we have the procedure called standardized mortality ratio, SMR. In the SMR the expected age is adjusted. That means that we have adjusted for that.
Q. Are confounders and risk factors synonymous?
A. In most situations I would say. yes because

basically a risk factor is more general than confounder. Confounder is a risk factor whose effect we don't want to study, per se. But we need to be aware of that so we can adjust for that. For example, if I look at a study of, again, between benzene and leukemia, age is a risk factor for leukemia. But in this study, in this study that I conducted, age is also a confounder because I'm not interested in the age effect on leukemia. I'm only interested in adjusting for or controlling the confounding effect into age.

Q. Is age a risk factor for multiple myeloma?

A. Oh, certainly because depending on what your age is your risk of multiple myeloma would be different.

Q. Did you look at any statistics to see what the risk of developing multiple myeloma at the age of 39 would be?

A. I have not -- I can't give you a number. But certainly that's something very easy to look up.

Q. Do you think it would be higher or lower than the age at 60?

A. I think it would be lower.

Q. Would you agree that developing multiple myeloma at the age of 39 is certainly statistically improbable?

A. No.
Q. Would you agree that it would be unlikely to occur absent some environmental agent?
A. No.
Q. Is it more likely than not to occur at 39 without some environmental agent being the causative agent?
A. No. Regardless of age most multiple myeloma cases that we report in this country are not related to any specific exposure.
Q. Even at the age of 39?
A. That's true.
Q. And we talked a little bit earlier about statistically multiple myelomas normally occur in the sixth decade of life, correct?
A. No. We say that that is the age group we start seeing more multiple myelomas than the other age groups.
Q. Did you review Mr. Fannin's background, if you will, one, his social background, to determine if there were any risk factors in his background for the development of multiple myeloma?
I don't know what you mean by any -- I guess documents related to his social background. The documents that I have reviewed specific to the

plaintiff in this case are all listed on my November 25th report on the first page. Basically they include answers to interrogatories, as well as deposition transcripts and also medical records. That would be the extent of my information.
Q. In your opinion, Doctor, is there any known cause of multiple myeloma?
A. I don't know whether ionizing radiation would be one. I don't know whether ionizing radiation is a known -- known, I assume you mean well-accepted by everybody. I don't know whether that's well-accepted or not.
Q. Did you find any risk factors in your review of this case for Mr. Fannin's development of multiple myeloma?
A. I have not done that. 1 have not been asked by the attorneys to look into other risk factors.
Q. Do you know whether there's an association between metals and multiple myeloma?
A. Metals?
Q Metals generally.
A. I'm not aware of any.
Q. How about stainless steel and multiple myeloma?
A. I'm not so sure 1 understand.

Q. Working with stainless steel.
A. Working with stainless steel itself?
Q. Yes, sir.
A. It's kind of difficult to answer that question. Because when somebody works with stainless steel I assume he or she is also exposed to other things in relation to working with stainless steel. And I don't know what that is. That person may be a welder or that person may be -- I don't know. I don't know what other exposure would be. But I don't know of any direct relationship between stainless steel itself and multiple myeloma.
Q. What about working with aluminum?
A. 1 have not done anything on -- 1 have not looked into that. I can't answer the question.
Q. Propane, working with propane or working around propane?
A. I'm not aware of any.
Q. What about working around coal or coal dust?
A. That I don't know.
Q is rheumatic fever related in any way to multiple myeloma?
A. I think there may be one or two studies or may be even two or three studies reporting a non-significant increase for that. _But, again, I don't

think I would be in a position today to make a definitive statement on that, because I have not looked at that issue specifically.
Q. Well, you had to rule out other potential risk factors, didn't you, for Mr. Fannin, or did you just assume that since benzene doesn't cause multiple myeloma I don't need to look at anything else that he's been exposed to?
A. I'm not sure I understand the question. But my assignment in this case was to look at whether there is any relationship, causal relationship between benzene exposure and multiple myeloma. I have not been asked to look at stainless steel or aluminum and so on.
Q. So when it comes time for trial you won't come into the courthouse and tell the jury that Mr. Fannin's exposure to metals or stainless steel or aluminum or propane or coal or coal dust was the cause of his multiple myeloma, will you?
A. At this point I don't have non-Hodgkin's leukemia to say that. But on the other hand, if the attorneys decide to ask me to look into that, then I may be able to offer an opinion when I testify. But , I'm sure if they asked me to do that we would tell you what the opinion is on that.
Q. What articles do you recall that indicate that

rheumatic fever is a risk factor for multiple myeloma?
A. I think it's one of the papers that I have reference to in my first report, the report dated November 25th. I won't be able to find the paper for you now, but one or two papers may have talked about that.
Q. Would you be kind enough to advise Mr. Damico sometime about what papers it is?
A. Okay.
Q. Has it been proven causally that rheumatic fever is associated with multiple myeloma?
A. I cannot answer the question. As I said, I have not done a thorough research on that that 1 feel comfortable to answer your question today. And if the lawyers ask me to do that, I will be happy to do it.
Q. So as we sit here today under your own definition of a risk factor, that it needs to be causally established as a substance that would cause multiple myeloma, you can't say that rheumatic fever is a risk factor?
A. I cannot say that it is or is not today.
Q. I understand that. Now, then, are there certain occupations that the literature indicates have a higher risk of developing multiple myeloms based upon your review "of the literature?

A. Well, again, 1 said that my literature review was focused on the benzene as a potential risk factor. 1 did not look at occupations, per se.
Q. Well, you looked at diesel exposed workers, - diesel exposed workers and creosote workers. And in those articles they talk about various risk factors for various occupations, don't they?
A. Yes, sometimes they talk about occupations. Yes.
Q. And isn't it true that a few of the articles that you yourself cited indicate there's an elevated risk for railroad workers for the development of multiple myeloma?
A. Well, the problem is when you talk about a railroad worker we don't know exactly what they mean in terms of exposure. Some studies indicate that railroad workers might have an increased risk of multiple myeloma, but other studies indicate that there is no increased risk. So I don't think you would be, at least as far as my assignment goes, talking about benzene, creosote and diesel emission, I don't think 1 should rely on the job title, the occupation railroad workers. I should rely on the specific chemicals.
Q. Has it been reported in the literature that being a railroad worker is a risk factor for the

development of multiple myeloma?
A. I told you that a couple of studies indicate that there is an increased risk. But at the same time there are also studies indicating there are no increased risk.
Q. Can you tell me a study that indicates that there is not an increased risk for being a railroad worker for the development of multiple myeloma?
A. If you're patient, I can see if I can find the paper. Okay. If you turn to the references I have.
Q. In your 25th report or the 28th report?
A. It may appear in both reports. Yes, in fact, it appears in both reports. It's the study by Erickson. Do you see that?
Q. Wait a minute. Erickson and Carlson?
A. Yes. Do you have a copy of that paper?
Q. I do by happenstance.
A. If you turn to Page 98.
Q. I'm there. Table 2.
A. Wonderful.
Q. Railroad workers, the relative risk was 1.14. Wait a second. 90 percent.
A. Wait. Are we looking at the same paper?
Q. Maybe. Erickson.
A. Erickson and Carlson, Table 2.

Q. Yes. Erickson and Carlson.
A. The relative risk for a railroad worker is 0.5. The very first -- the one on top.
Q. Table 1 ?
A. Table 2.
Q. Railroad worker. 0.5.
A. Yes.
Q. All right. I looked at road workers and misconstrued that.
A. Road workers are not the same as railroad workers, I would assume.
Q. I would assume they're not either. Do you have -- I can never pronounce his name. Bopheta. Do you have Sopheta '88 in front of you?
A. Absolutely.
Q. Look at Bopheta '88.
A. Okay.
Q. Didn't they determine that there was a relative risk or an odds ratio for the development of multiple myeloma?
A. •What table are you on?
Q. I'm on Table 4.
A. Table 4.
Q. Page 556.
A. Yes, I think you gave me the wrong reference.

That's Bopheta 1989. Did you say '88?
Q. '89. I'm sorry. I meant '89. I said '88.
Didn't they determine that the odds ratio for railroad workers and the instance of multiple myeloma was 6.0 , statistically significant to the .05 level?
A. Yes, I'm looking at that. That's why 1 say you cannot rely on the occupation, because railroad workers can mean different things to different people. And certainly you have a big discrepancy between the two studies. One thing you do want to look at is when the two studies give you different results you may want to look at the sample size. If you look at the Bopheta paper, you will see that there are three cases. Do you see the three slash two? That means there's three cases of multiple myeloma in railroad workers and there were two railroad workers in the control.
So the important thing is to look at those three cases, the cases of multiple myeloma in railroad workers. Whereas, if you look at the Erickson paper - let me find my Erickson paper again. If you look at the Erickson paper, 1 look at Table 2 on Page 98. You see that you have seven cases of multiple myeloma.
23. Railroad workers.
So everything being equal, then the Erickson study should be more accurate and more reliable than

the Bopheta study because the sample size is much bigger.
Q. Well, just because a study is smaller, Doctor, doesn't necessarily mean that you can't find a relationship, does it? I mean, really, what you're concerned with is the Beta error, aren't you, not being able to find a relationship with a smaller study?
A. Well, we are concerned with both. If, again, if you think that they define railroad worker in an accurate way in both studies, and everything being equal in those two studies, then sample size would be something to be considered.
Q. Why would sample size be something to be considered when you found a relationship in the smaller study?
A. Well, because a misclassification of one single case would change the result completely. After all, it's -- the whole thing depends on three cases.
Q. That happens all the time, doesn't it?
A. If the definition of railroad worker is not accurate, if somebody answered the question railroad worker incorrectly, if it happened in only one case, the whole thing would go away.
Q. Isn't that accounted for in the statistical tests, though?

A. Yes. If you take out one case, the 95 percent confidence level will be different.
Q. But it's accounted for when you do the statistical analysis, isn't it?
A. Assuming all three cases were indeed railroad workers. The statistical test does not account for misclassification. .
Q. Do you have Heinman?
A. I do.
Q. Would you find that for me? It's Table 1.
Did you find it, Doctor?
A. I'm still looking. What page are you on?
Q. It looks like it's Page 558.
A. Okay.
Q. At the bottom, transportation and material handling, Table 1, the continuation of Table 1 from the previous page.
A. Right.
Q. Road and railroad workers. The odds ratio is
Although not statistically significant because it includes 1, it nevertheless is elevated?
A. Yes. But it also includes road workers as well. So it's not just railroad workers, but also road workers.
Q. Turn to Table 2.

A. And if you go back to Erickson, because you make a mistake of looking at the road workers, the road workers have a risk twice as high as railroad workers in the Erickson study. So when you combine the two, maybe the increase comes from the road workers and not the railroad workers.
Q. Would you turn to Table 2 for me?,
A. Yes.
Q. At the bottom, railroad, that would be transportation. Underneath that, railroads?
A. Yes.
Q. The OR is 1.4, although not statistically significant, because the lower end of the range is below one and still is an elevated risk for railroad workers to develop multiple myeloma, correct?
A. Correct.
Q. Going back for a second to -- let me find my note. Bopheta, '89. You indicated that if any one of these three cases were mischaracterized or their employment was mischaracterized in any way, that that would.6hange the odds ratio.
A. Yes.
Q. Do you have any evidence that any of those three cases were mischaracterized by anyone?
A. I'm not saying there is evidence. All I'm

saying is that being a very small study, and the result depends on only three cases, that we have to interpret that very carefully. That's all I'm saying.
Q. I just want to make sure you didn't have something that I was unaware of. Let's skip for a second to engine exhaust, Dr. Wong. In your opinion, is engine exhaust a risk factor for multiple myeloma7
A. Well, again, that's not very specific. 1 tried to include that because sometimes people include engine exhaust as both gasoline exhaust as well as diesel exhaust. And let me look at my report, what I said. Well, there are two studies from Sweden. The first study was done by, I think it's pronounced Flodin.
Q. I think you're correct.
A. 1987. And Flodin found a marginally significant increase relative risk of 2.1 for engine exhaust. And the 95 percent confidence interval was from 1.2 to 3.9. So that was very significant. But the authors themselves point out that there may be some confounding in their study. And certainly they would like to see some additional studies, preferably with some adjustment or control over potential confounding. And the second study was done a few years later published by Erickson and Carlson in 1992 from

Sweden, as well, the same country. And that study was twice as large as the first study. And they did not find a significant increase for engine exhaust.
Q. But they nevertheless found an elevated relative risk of 1.38. It might not have been significantly significant, but it was elevated.
A. Well, the 1.38 was without any adjustment for potential confounding. They presented two analyses, one without adjusting for confounding. The other one, adjusting for confounding, the relative risk was reduced to 1.1.
Q. Going back to the Flodin study, you indicated in your report on Page 3, the third line down the sentence that begins, The authors further remarked.
A. Right.
Q. That no study on multiple myeloma as related to engine exhaust has, to our knowledge, been published.
Then you indicated up above that there was the confounding factors that may have been involved that the authors pointed out.
A. Right.
Q. Do you accept Flodin's confounding factors as potential risk factors for the development of multiple myeloma?

A. Based on the Erickson study, certainly that seems to be the case. Because without adjustment for confounding the risk ratio was 1.38. After adjustment the risk ratio was reduced to 1.1. So there is some indication of that.
Q. Let me find this article for a second, Doctor. Doctor, look at Flodin for a second. Are you with me?
A. I have to find the paper.
Q. Okay. I thought maybe I lost you.
A. Okay. I have the paper.
Q. What are the confounders that the author in the Flodin study were concerned about?
A. I'm trying to find the paragraph. Well, in their study they find an increase related to engine exhaust, creosote and fresh wood. And some of the occupations would have exposure to all three. I think that may be one of the confusing parts.
Q. In your opinion, then, would creosote, engine exhaust and fresh wood be rich factors for the development of multiple myeloma?
A. Well, in my report I talk about two of the three things you mentioned. I think, looking at all the studies we have today on diesel exhaust and multiple myeloma, the literature is not completely consistent. But certainly I would think that the

1 weight of evidence would say that there is no
2 relationship between diesel exhaust and multiple
3 myeloma. And that's for diesel exhaust.
4 In terms of creosote, there are not a whole
5 lot of studies on the subject. There are only two or
6 three studies. And the findings from these two or
7 three studies are not consistent. So I would say at
8 this point we would not be able to say one way or
9 another in terms of creosote.
10 Q. If they're not risk factors then, Doctor, why
11 would you be concerned with the confounding effects of
12 those in any study?
13 A. I did not say that creosote is not. I just
14 don't know if it is or is not at this point.
15 Q. What about diesel exhaust, are you saying that
16 it is not a risk factor for multiple myeloma?
17 A. Based on other studies, 1 don't think it is.
18 Q. Now, when you made your analysis of whether
19 diesel exhaust was or was not a risk factor for
20 multiple myeloma, did you go through the Bradford Hill
21 exercise?
22 A. Yes. One thing is we have quite a few studies
23 saying there is no connection and studies that say yes.
24 Q. Well, the studies that say there are no
25 connections are simply not statistically significant,

isn't that true?
A. No, some actually report lower than unity, you know, the risk ratio.
Q. So is it based on the lack of consistency among the studies that you are unable to conclude a relationship between diesel exhaust and multiple myeloma?
A. More than that. We are also looking at the quality of the study and how big the studies are. The studies that are reporting an increase are based on only a few cases.
For example, if you look at one of the larger studies on people exposed to diesel emissions, which would be the study done by NIOSH, okay, the first author of that study is by the name Stern. S-t-e-r-n. And I have put the citation in the report. They looked at close to 5,000 cancer deaths among construction operating engineers exposed to diesel. And their result was they observed 73 cases of multiple myeloma and expect 81 cases. And the risk ratio was 0.90. That was a very large study. So I have to put more weight on that study.
4 Q. But didn't the authors themselves say, Doctor, that it's possible that malignant neoplasms were underestimated because workers that left before

retirement or death were not included in the analysis?
A. That may have an effect for either direction of multiple myeloma, depending on if indeed there was some cancer deaths missing, depending on what it is.
Q. Didn't the multiple myeloma have a longer latency period than the normal neoplastic disease?
A. I'm not so sure that's true for all other cancers.
Q. Would you agree with me that a longer latency period regardless of whether it's longer than the other neoplastics or not, if someone left the union and their vital status was not accounted for in the cohort, that you could be missing, as the authors suggest, neoplasms that have a long latency period?
A. Yes. But at the same time if they also missed, for example, prostate cancer, which also has a long latent survival, then you would actually make the statistic go the other direction. So, 1 mean, we can criticize the study for missing -- potentially missing some deaths. But we don't know the direction of bias, if indeed, that's the case.
Q. Well, the authors criticized it themselves saying that the malignant neoplasm -- it's possible that malignant neoplasms were underestimated in this study since workers who left the union before

retirement or death were not included in the analysis.
So they're basically saying themselves when they wrote this that there's a possibility of underestimation of malignant neoplasms, correct?
A. Well, right now the risk ratio is 0.9. Even if they underestimate by some extent it's going to be a long way before it would be elevated.
Q. Did you notice in the written materials that Mr. Fannin cleaned his paintbrushes with gasoline and he cleaned his hands with gasoline?
A. I read the deposition, yes. But what is your question?
Q. That was my question. Did you notice that in the deposition? That was my first question.
A. Yes.
Q. And did you review the literature on gasoline and multiple myeloma?
A. I did. I thought that was the focus of my November 25th report.
Q. What gasoline studies did you cite?
A. •The gasoline study that I have done.
Q. Which is petroleum workers, terminal workers exposed to gasoline, the statistical reports submitted to API?
A. One paper was published. One was reported to

API. Yes. There are two reports.
Q. Did you rely on the technical report that you
did for API
A. Both of them, yes.
Q. in forming your opinions?
A. Yes.
Q. I'd like to have a copy of the technical
report that you submitted to API on gasoline, since
you're relying on that to state that gasoline does not
cause multiple myeloma.
A. I have to ask the lawyer to get it from API.
I'm not sure I can Xerox a copy of that and send it to
you.
MR. DAMICO: We'll check into it and see what we
can do.
MR. HARTLEY: Under the rules you know if he
relied on it I'm entitled to see it.
MR. DAMICO: I understand. He's telling me now
that he may not be able to give it to me without
approval.
THE WITNESS: I cannot make a copy. I think that
the report is copyright. So I don't want to just make
a copy.
MR. HARTLEY: a. Do you normally give out your
articles that are in press to other people to review,

Doctor?
A. Yes, I do.
Q. And did you give out your reports in press to
Dr. Bergosagel?
A. Yes. Some things are accepted. I don't mind
giving it out. Before acceptance by the Journal, then
I would not.
Q. When was your study on multiple myeloma and
benzene exposure accepted by the Journal of Regulatory
Toxicology and Pharmacology?
A. I think it was quite a while ago.
Q. How long is quite a while ago?
A. It must be accepted around June or July.
Q. Did you give a copy of that to Dr. Bergosagel?
A. I might have given a copy to him when I went
up to Canada to see him.
Q. You met with him?
A. Yes.
Q. What did you and he discuss?
A. Basically we talked about the potential of
writing paper, a position paper together, because he
is an oncologist and I'm an epidemiologist. We were
thinking about how to combine the two disciplines and
write a review article. And I told him about my paper
on the multiple myeloma paper based on epidemiologic

1 studies. And I told him to come up with some
2 oncological aspects of multiple myeloma so that we can
3 write a paper together.
4 Q. You're not telling me that out of the clear
5 blue sky you went to Ontario to meet with Dr.
6 Bergosagel to discuss whether the two of you were going
7 to write an article together?
8 A. No. My mother lives in Toronto and I was up
9 there to see my mother. And it just happens that he
10 10lives in Toronto. In fact, I made the plan of going up
11 to see my mother a long time ago.
12 Q. How did you know of Dr. Bergosagel?
13 A. I have seen some of his writings on multiple
14 myeloma.
15 Q. Did you see him on the street corner and say,
16 "Hey, can we talk about writing an article together?"
17 A. No. No. No. I have seen his writings. He
18 has published on multiple myeloma.
19 Q. Did you discuss this case with Dr. Bergosagel?
20 A. No, I did not at that time. In fact, when I
21 went up to see him I don't think I was hired at all in
22 this case. That was before I got involved in this
23 case.
24 Q. When were you hired in this case?
25 A. That goes back to the first time was sometime

1 in September, I think.
2 Q. Doctor, do you have your CV in front of you?
3 A. Yes.
4 Q. I want to look at a few things with you on
5 that.
6 A. Okay.
7 Q. On your CV you list 121 articles.
8 A. Yes.
9 Q. At least the one I have. Those articles, and
10 I'm concerned with the more recent ones, which ones
11 were rejected by the various journals?
12 A. Which ones were rejected?
13 Q. Yes. Which ones did you have to submit to
14 more than one journal to get published? Did you have
15 to submit your study on Risk of Acute Myeloid Leukemia
16 and Multiple Myeloma in Workers Exposed to Benzene to
17 more than the Occupational and Environmental Journal?
18 A. Well, that paper was stimulated by a paper
19 written by Paxton. P-a-x-t-o-n. Do you know who I'm
20 talking about?
21 Q. She works for API, doesn't she?
22 A. She used to work for API. She doesn't work
23 for API anymore.
24 Q. She works for petroleum companies now.
25 A. I don't know about that. I don't know about

that. You can make any comments you want. She wrote a paper on the updated analysis of the Pliofilm study and published that in a journal called Risk Analysis. That's really a journal for risk assessment. I wrote a paper -- basically I pointed out that we should separate AML from other leukemias for analysis. And I believe they sent the paper out to different people to review. And 1 remember 1 received comments from three reviewers, one reviewer saying that the data is too limited to talk about a dose response analysis. And one paper said they had done analysis similar to that. I think that person, based on what he said, I think that was Kenny Crumb. Because he said he had already done some similar analysis and reached similar conclusions using a different approach. And one analysis said, yes, that's the right way to look at it.

But the editor came back and said that my reanalysis separating the leukemia into different groups would not offer sufficient new information and suggested that I may want to reduce it to a letter or something as comments. Which I don't want to do. So I resubmit to the Occupational and Environmental Medicine.

Q. So it was rejected by at least one journal?

A. Well, I didn't go by what they suggest.
Q. That's not my question. Can you answer my question? Was it rejected by a journal when you submitted it for publication?
A. It was not accepted at the current form. They wanted me to reduce that and I don't want to.
Q. How many journals rejected it or refused to accept it in its current form?
A. I didn't send it to any other journal. The second journal I sent it to was Occupational and Environmental Medicine and it was accepted without any changes.
Q. Were any of your other more recent articles not accepted as written by other journals? Was the reference No. 112, Cell-type Specific Leukemia Analyses in a Cohort of 208,000 Petroleum Workers not accepted in its original form by any journal?
A. No, we submitted it to the Regulatory Toxicology. We didn't send it to anyplace else.
Q. It was published in Epidemiology?
A. What?
Q. It was published in Epidemiology, No. 112?
A. Oh, 112. Yes, 112 is an abstract of a presentation we make. It's not a full paper. It is an abstract. It is the same paper that we published 105.

1 Do you see 105?
2 Q. Yes.
3 A. That is the full paper. That is the full
4 paper.
5 Q. Was that accepted originally by Regulatory
6 Toxicology and Pharmacology?
7 A. Yes
8 Q. Did you submit that to any other journal?
9 A. No.
10 Q. What about An Updated Cohort Mortality Study
11 of Workers at Northeastern United States Petroleum
12 Refinery, was that originally rejected or not accepted
13 by any journal?
14 A. I believe that was the original place that we
15 submit to.
16 Q. What about The Application of Meta-analysis in
17 Reviewing Occupational Epidemiologic Studies?
18 A. Oh, that one is a special article. That was
19 what they call metalogical papers. That was actually
20 at the invitation of the editor. So that being the
21 case,. i don't think they would invite us to write an
22 article and then reject it.
23 Q. What about 104, Risk of Acute Myeloid Leukemia
24 and Multiple Myelome in Workers Exposed to Benzene,
25 Occupational and Environmental Medicine. Was that

originally accepted or rejected by any reviewer?
A. 1 think that was the one that we talked about before, that I told you that paper was stimulated by the paper written by Paxton. Her paper was published in Risk Analysis and I submit to that and I got three reviewers' comments.
One was, you know, critical in terms of the sample size, which I cannot do anything about because that was based on the NIOSH cohort. And one paper says that, well, that analysis had been looked at differently and we come to the same conclusion. Therefore, that reviewer doesn't think the information is that new. And the third one thinks it was quite favorable. And the editor said, "if you shorten that maybe we'll consider it." But I didn't want to do that.
Q. In your review of the materials in this case, did you determine whether Mr. Fannin was exposed to petroleum products? Do you understand that term?
A. I understand that term. But that is not my area of expertise. I am not in a position to determine what he was or was not exposed to. Basically, I was asked to look at epidemiologic data between certain exposure and multiple myeloma. And that is my focus.
Q. Well, then, did you look at Linet's study of

the hundred patients in the Baltimore hospital which found a relative risk of 3.3 for those exposed to petroleum products?

A. Yes. Petroleum product can mean anything. 1 was not asked to look at petroleum products in general. 1 was asked to look at benzene in specific.

Q. Did you look at the Linet study?

A. Yes, I did.

Q. Did you include it in your report?

A. Yes, I did.

Q. Where?

A. Look at the reference.

Q. Which one?

MR. DAMICO: It's in the November 25th report, the first page of the bibliography, the third from the last article.

MR. HARTLEY: Q. Did you comment on Linet in your paper that I just received today? That's why I'm. asking, because I haven't had a chance to look at it completely.

A. Hold on for one second.

MR. DAMICO: I know you're in a hurry. I'll shut up if you want me to. But Page 3 he mentions it, the second full paragraph. I don't know if it's mentioned anywhere else.

1 THE WITNESS: Yes. 1 mentioned Linet in the 1987
2 paper as one of the papers that suggests a number of
3 risk factors for multiple myeloma.
4 MR. HARTLEY: Q. Didn't Linet report a 3.3
5 . relative risk for petroleum products? And you did not
6 mention petroleum products in your potential risk
7 factors for multiple myeloma. My question is, why not?
8 A. Why not? Because those are lifestyles. The
9 things that I put down there are all lifestyle risk
10 factors. If you look at Page 3. None of that is
11 related to occupational exposure.
12 Q. You didn't put down any occupational risk
13 factors, did you, in any report?
14 A. Yes, on Page 6.
15 Q. Which one?
16 A. Under the subheading of the first report.
17 November 26th on Page 6 under the subheading on case
18 control studies of multiple myeloma, the very first
19 study is Linet 1987. She reports a risk ratio of 1.1
20 for benzene exposure.
21 Q. But she also reported a 3.3 relative risk for
22 petroleum products and you didn't mention that anywhere
23 in here?
24 A. Sir, 1 was asked to look at benzene exposure,
25 not petroleum products in general. If you want to make

a comment, make a comment.
Q. Which comment would that be? I didn't know I made a comment.
A. No. If you want to make a comment on petroleum products, you go ahead. That's not my focus.
Q. 1 understand. You didn't focus on paints and solvents either, did you?
A. I have not been asked to do that.
Q. Did you do any calculations of Fannin's cumulative exposure to anything that he was exposed to that you have reviewed or that you had gleaned from your review of the materials?
A. I have not.
Q. Are you going to testify at trial, Dr. Wong, about solvents or paints and whether there's a relationship between those two substances and multiple myeloma?
A. No, I have not done any work on that for this case. And I'm not in a position to testify on that.
Q. What about polycyclic aromatic hydrocarbons, are your in a position to testify regarding whether they are associated with multiple myeloma?
A. Well, let me specify or emphasize again. The three things I looked at in this case are benzene, creosote and diesel exhaust. Those are the three

1 things I will talk about, unless the lawyers ask me to
2 look into something else. If that's the case, I'm sure
3 they will let you know.
4 Q. Did you forward any written correspondence to
5 Dr. Bergosagel concerning this case?
6 A. No, I have never talked to him about this case
7 at all.
8 Q. Doctor, do you have an understanding of
9 whether there is benzene in solvents?
10 A. It depends on what kind of solvents you're
11 talking about.
12 Q. How about mineral spirits?
13 A. If there is, it may be a very small percentage
14 as contaminants. You have to look at the specific
15 product.
16 Q. Your study on -- back on your CV, No. 58, Wong
17 and Ragland.
18 A. Yes.
19 Q. I'm looking at publication No. 88. Was it
20 ever published?
21 A. That is so old I lost track of that. I guess
22 we never changed the word processor. We didn't change
23 that.
24 Q. Cooper and Wong, No. 52, A Study of Mortality
25 in a Population of Nickel Miners and Refinery Workers

submitted in '88. Was that ever published?
A. To be honest with you, I don't know what Dr. Cooper did with that paper. He's retired now. So I don't know exactly what he did with that paper.
Q. No. 62, Epidemiology, Toxicology and Quantitative Risk Assessment, An Integrated Approach, submitted for publication. Was it ever published?
A. We submitted it and then things changed so rapidly in terms of risk assessment that I decided to kind of take it back and maybe revise that.
Q. Did you ever revise it?
A. No, because risk assessment is really within the last five, ten years, become very, very broad.
Q. Did the journal reject it as it was written?
A. No, basically I just retracted it. There's no point in publishing a paper that's not complete.
Q. 63, Wong, Whorton, Morgan and Gordon, Cause Specific Mortality and Morbidity Among Paper Mill Employees, submitted for publication. Was it ever published?
A That may be a preliminary analysis of a paper that we subsequently published later. Let me see. We did publish a paper on paper mills. I think -- let me try to -- do you have Publication 116?
Q. Yes

A. That may be the same paper, except with more data, with more paper mills added to the project.
Q. Do you know whether it is or not?
A. I'm not definitely sure because the first one was sometime ago.
Q. What about No. 88, Wong and Foliart, Assessment of Lung Cancer Risk by Histologic Category in Workers Exposed to Chlorinated Chemicals, submitted for publication. Was that ever published?
A. That was too short. And I decided - we decided to just leave it as a technical paper. I think we submitted a technical paper to the Chemical Manufacturers Association.
Q. 119, that was published, right, A Nested Case Control Study of Leukemia.
A. 119? Okay. I have a more updated CV, so the numbers may be slightly different. What was the title of the paper?
Q. A Nested Case Control Study of Leukemia and Multiple Myeloma in a Cohort of Land-based Terminal Workers Exposed to Gasoline in the Petroleum Industry.
A. No, actually we were going to submit it, but we needed to do a little more work.
Q. It said it was submitted for publication. Did you withdraw it?

A. No. No. No. We thought we were going to submit it, but we have to do more work. So we have not submitted it at this point.
Q. Doctor, who pays for your time to write all these papers?
A. Writing the papers themselves?
Q. Yes, sir.
A. No one.
Q. You just go out and write them yourself? They're not funded by any agency or any association?
A. Sometimes if the paper is based on a project, of course, the project most likely would be funded by some group. And we would, you know, as a result of the project we would have a technical report submitted to the sponsor. And a lot of times we would, for lack of a better term, cut and paste the longer technical -report into a sort of manuscript and submit that for publication. But the sponsors would not pay for writing of the manuscript itself.
Q. Are you ever funded by a university or a college? Have you ever been funded by a university or college?
A. I don't think universities or colleges are in the business of funding studies.
Q. Have you ever been, absent your time at

Georgetown, after you left Georgetown, have you ever been asked to be on the faculty of any university and obtain grants to provide for your remuneration as a professor or a visiting professor?
A. Well, I'm on the adjunct faculty of Tulane University and I'm also a visiting professor at one of the medical schools in Taiwan. But not being there full-time it would be very difficult for me to apply for research -- apply for research funding as a university executive member.
Q. Does the adjunct professorship bestow any form of money to you? Do you get paid for doing that?
A. They pay for my traveling expenses and living expenses and so on. But basically 1 contribute my time to the university.
Q. Where does your general income come from then as Applied Health Sciences?
A. Well, the income, the majority would be coming from doing research, doing projects.
Q. Well, for whom?
A For mainly the industry.
Q. Can you give me some examples?
A. Well, certainly. 1 have done work for API. I have done work for paper product companies. I'm doing a study for the National Stone Association. I'm doing

work for the International Lead and Zinc Research Organization. 1 have done work for Mobil Oil Company. I have done work for Chevron.
Q. Would you agree with me, Doctor, that if the industry is not happy with the results of your reports that they wouldn't ask you to come back again and do another report for them?
A. You have to ask the industry.
Q. How much time have you spent in this case?
A. As I said, I was contacted the first time in September -- 1 think in September they sent me a package of materials to review. And 1 have identified the material in my November 25th letter. You see that on the first page. Basically I spent about a couple of days looking through, reviewing the package. And then in November they asked that I should write a report for this case. And I think I spent maybe two to three days doing some review and also writing the report.
Q. So how many hours do you think you have in this, 40?
A. Well, roughly two days in October. Probably about two to three days in November.
Q. About eight-hour days?
A. Yes, I'm talking about eight-hour days.
Q. Have you billed them for your time yet?

A. Yes, I have.
Q. Have you been paid?
A. I don't want to embarrass the attorney sitting across from me at the table. They have not paid me.
MR. DAMICO: I was unaware of this glaring error until early this morning or late this morning, 1 should say.
MR. HARTLEY: Doctor, I don't believe I have anything further.
MR. DAMICO: We'll read and sign.
MR.-HARTLEY: I'd like to get a copy of his technical report to API.
MR. DAMICO: We'll get it.
MR. HARTLEY: Thank you.
(Whereupon, the deposition concluded at 5:45 o'clock p.m.)

OTTO WONG, Sc.D. F.A.C.E.

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r,

1 STATE OF CALIFORNIA)
)
2 COUNTY OF SAN FRANCISCO)
3 I, DONNA LEE BECKETT, a Certified Shorthand
4 Reporter of the State of California, duly authorized to
5 administer oaths pursuant to Section 2025 of the
6 California Code of Civil Procedure, do hereby certify
7 that
8 OTTO WONG, Sc.D. F.A.C.E.,
9 the witness in the foregoing deposition, was by me duly
10 sworn to testify the truth, the whole truth and nothing
11 but the truth in the within-entitled cause; that said
12 testimony of said witness was reported by me, a
13 disinterested person, and was thereafter transcribed
_ 14 under my direction into typewriting and is a true and
t - 15 correct transcription of said proceedings.
16 I further certify that I am not of counsel or
17 attorney for either or any of the parties in the
18 foregoing deposition and caption named, nor in any way
19 interested in the outcome of the cause named in said
20 caption.
21 Dated the 30th day of December, 1997.
22
234
E.I:.V
24 DONNA LEE BECKETT
CSR No. 3450 (California)
25
L 118
c < NOGARA REPORTING SERVICE > >,

1
Otto Wong, Sc.D. F.A.C.E.
2 181 Second Avenue, Ste. 628
San Mateo, CA 94401
3
- Date: Tuesday, December 30, 1997
4 Re: Fannin vs. Norfolk & Western Railway
Deposition Date: Tuesday, December 16, 1997
5
Dear Dr. Wong,
6
Please be advised the original transcript of your
7 deposition is ready for your review.
8 You have 35 days from the date of this letter to read,
correct if necessary, and sign your transcript. It
9 will then be sealed and sent to the examining attorney
pursuant to the applicable law. You are not required
' • 10 by law to read and sign your deposition.
11 You may either come to our office to read and sign the
. original transcript, or you may contact your attorney
12 or the attorney who arranged for you to be present at
your deposition. If they have ordered a copy of the
13 transcript, you may review their copy and make
corrections by submitting, signing and returning the
14 attached form. If you choose to review your transcript
at our office, please call first to make an
15 appointment. Should you have any question regarding
these instructions, please call.
16
Sincerely,
17
18 NOGARA REPORTING SERVICE
130 Battery Street, Suite 580
19 San Francisco, California 94111
(415) 398-1889
20
21 cc: Aft counsel
Original deposition
22
23.

24

25 .