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CAUSE NO. A-167,693

JAMES COWEY AND RUTH COWEY	)	IN THE DISTRICT COURT
PLAINTIFFS,	)	
VS.	)	JEFFERSON COUNTY, TEXAS
RADIATOR SPECIALTY COMPANY, ET AL	)	
DEFENDANTS.	)	58TH JUDICIAL DISTRICT

****

ORAL DEPOSITION OF
ETHAN A. NATELSON, MD
DECEMBER 3, 2003

****

ORAL DEPOSITION OF ETHAN A. NATELSON, MD, produced as a witness at the instance of the PLAINTIFFS, and duly sworn, was taken in the above-styled and numbered cause on the 3RD day of DECEMBER, 2003, from 2:09 p.m. to 3:32 p.m., before Mark A. Miller, CSR in and for the State of Texas, reported by machine shorthand, at the offices of the Stehlin Oncology Clinic, 1315 St. Joseph Parkway, Suite 1800, Houston, Texas, pursuant to the Texas Rules of Civil Procedure and the provisions stated on the record or attached hereto.

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A P P E A R A N C E S

FOR THE PLAINTIFFS:
MR. LANCE LUBEL
MR. J. ROBERT BLACK
HEARD, ROBINS, CLOUD, LUBEL & GREENWOOD
910 TRAVIS STREET, SUITE 2020

HOUSTON, TEXAS 77002

FOR THE DEFENDANTS UNITED STATES STEEL CORPORATION, ARISTECH CHEMICAL CORPORATION AND USX CORPORATION:
MR. STEPHEN C. DILLARD
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1301 MCKINNEY, SUITE 5100
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FOR THE DEFENDANT RADIATOR SPECIALTY COMPANY:
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HOUSTON, TEXAS 77002-6707

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EXHIBITS

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THE REPORTER: Would you like to read and sign your deposition?

THE WITNESS: Yes.

THE REPORTER: Would you like to take the deposition by the rules?

MR. LUBEL: Yes.

ETHAN A. NATELSON, M.D.,

having been first duly sworn, testified as follows:

MR. LUBEL: All right.

EXAMINATION

BY MR. LUBEL:

Q. Please state your full name.

A. Ethan A. Natelson.

Q. Where do you reside?

A. At 8707 Wateka Drive in Houston.

Q. How are you employed?

A. I have multiple employers. I'm employed in part by St. Joseph Hospital where I manage the -- one of the residency programs, so-called transitional internship program. I'm also employed by the Stehlin Oncology Clinic where I do private practice.

Q. Dr. Natelson, my name is Lance Lubel. You and I have never met before today, have we?

A. I don't think so.
Q. And are you familiar with my law firm?

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A. No.
Q. You don't recall ever being hired by my law firm to be an expert?
A. No.
Q. Who hired you in this case?
A. Well, originally I was contacted by an attorney by the name of Ricky Raven of Porter & Hedges. And then subsequently by attorneys from Ellis Carstarphen, and then subsequently by Mr. Dillard from Fulbright & Jaworski.
Q. Did you ever have any conversations with a lawyer by the name of Bob Scott?
A. I don't think so. I know who Bob Scott is, but I don't think I spoke to him in reference to this case.
Q. You've been hired by the defendants as an expert in these chemical exposure cases for a number of years; is that correct?
A. For several years, yes.
Q. How did you get introduced to this business of being an expert witness?
A. Actually, I think the first case -- I have a listing here of the cases that I've testified in since 1992. I think the first time I got involved in this was in 1992, and it had to do with a patient that was my

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patient, Mr. Mimms, who had chronic granulocytic leukemia. And he alleged that his illness was caused by benzene exposure. And I testified as a treating physician at a hearing in Galveston concerning that issue, and I think that was the first testimony I gave relative to, let's say, benzene.

MR. LUBEL: Let me go ahead and mark this as Exhibit Number 1.

(Exhibit 1 marked.)

Q. (BY MR. LUBEL) This is a list of testimony that you've given that you've provided to us at your deposition, correct?
A. Yes.
Q. Who was the defense lawyer that contacted you?

A. You're talking about which case, the original case that I mentioned?
Q. Correct.
A. I'm not certain. It may have been John Shoebotham, but I can't be certain about that.
Q. As I'm sure you're aware, Mr. Shoebotham is a partner with Ricky Raven?
A. Yes.
Q. How did you meet Mr. Dillard?
A. I really don't remember. It's been a long time ago. And it had to do with a case, but I can't

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tell you which case it would be.
Q. It's my understanding, and correct me if I'm wrong, you've testified on a number of occasions for Mr. Dillard and his law firm of Fulbright & Jaworski; is that correct?
A. That is correct.
Q. And you've had discussions with him prior to today to discuss what your testimony would be in Mr. Cowey's case, I take it, correct?
A. Yes.
Q. And Ms. Yates here, have you met her before?
A. Yes.
Q. How do you know her?
A. Again, it would be on another case, and I can't -- I can't tell you which case that might have been.
Q. Now, when you get hired as an expert witness, irrespective of what side hires you, do they have to go through the medical school or another organization to hire you?
A. No.
Q. They call you directly?
A. Yes.
Q. And I take it that any proceeds that you earn from your expert testimony work goes into your medical

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practice; is that correct?
A. Well, it would go into my pocket, however you want to describe that.
Q. Are you part of a partnership of doctors?
A. Loosely. We have a group, the Stehlin

Oncology Clinic, that consists of a couple of surgeons and a couple of internists, of which I'm one of the internists, and a dermatologist, and we all see patients up here in this setting.

Q. Great. It appears to me that you've brought with you all of your file materials regarding Mr. Cowey and issues that you deem important to the disease of myelodysplasia. Would that be a fair statement?

A. Yes.

Q. (BY MR. LUBEL) I'd like to mark as Exhibit Number 2, the October 13th, 2003 report that your attorneys at Fulbright & Jaworski forwarded me, and ask you if that is a true and correct copy of that report?

(Exhibit 2 marked.)

A. Yes, that would be a true and correct copy.

Q. (BY MR. LUBEL) Now, when you issued the report that we've marked as Exhibit Number 2, did you have some discussions with the lawyers that had hired you about generally what your report should cover?

A. Not what my report should cover, no.

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Q. When they hired you did they ask you or tell you the scope of your work, what issues they wanted you to look at?

A. No.

Q. How did you know what to do?

A. Well, what happens typically on a case of this nature, or any case, whether it's a malpractice case or benzene case, is, I'm asked if I would review a case. And I'm typically asked not to write anything down. And I review the case and then give my opinion to the attorney.

And at that point they may ask me to put pen to paper and write a report or they may not. And ultimately, if the case proceeds, I write a report. But that report is simply based on my style of a report. There is no outline sent to me or any specific memo on how that report should be structured.

Q. And I didn't mean to imply anything sinister about it. All I -- really what I was driving at is when they called you and said, we want you to be an expert in the case, did they lay out generally what they wanted you to do; in other words, we want you to look at

Mr. Cowey's case and see if any chemicals caused his disease? Did they give you any general scope of duties in the case?

A. Well, I don't recall the exact conversation, but I would assume it went, we have a patient who alleges he has an illness, a hematologic illness, consequent to chemical exposure, would you look at this case. Something to that effect.

Q. So what you do is, is you take and you write a report based upon a complete and thorough review of the case?

A. Based on the information that I'm provided.

Q. And you don't permit the attorneys, irrespective of who hires you, to limit what you're able to do in the case as far as if they're going to ask you to give any opinions?

A. No.

Q. Is that true?

A. Yes, that's true.

Q. All right. So when you wrote Exhibit Number 2, your report in this case, you weren't limited by the lawyers that had hired you in what you could say?

A. That's correct.

Q. Or what authorities you could rely on?

A. That's correct.

Q. Or what information you could review; is that true?

A. That's correct.

Q. So would it be fair to say, at least when you issued the report in October of this year, you had laid out in general summary fashion all of your opinions that related to the case as of the date of the report?

A. Yes, based upon all of the information that I had at the time, and I recall, for example, in this particular case, I had requested, if available, to review the slides. Generally, if it's a case that has to do with leukemia or myelodysplasia, I like, not that I misbelieve the reports, but I like to simply observe the slides myself, but I didn't receive that, for example. So, sometimes -- so, I go with what I have, and then write a report.

Q. All right. So other than the slides, regarding Mr. Cowey, is there anything else that you've requested that you have not received?
A. No.
Q. Now, do you have any modifications or changes to your report?
A. No.
Q. Are there any addendums that you've made?
A. No.
Q. Have your opinions changed in substance?
A. No.
Q. Have the bases for your opinions changed?

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A. No.
Q. Thank you.
MR. LUBEL: Now I'm going to mark as Exhibit Number 3 a June 27th, 2003 report to a Mr. Dougherty that was in your file materials. Do you recognize that?
A. Yes.
(Exhibit 3 marked.)
Q. (BY MR. LUBEL) And essentially, the report that you gave to Mr. Dougherty, in approximately June of 2003, has the same opinions as the one that you offered to Mr. Dillard's firm in October of 2003?
A. Correct.
Q. There may be some minor differences, but the substance is the same, would that be fair?
A. That would be fair.
Q. Do you agree with the diagnosis of Mr. Cowey that's contained within his medical records?
A. Yes. He has myelodysplasia. Now, there are many classifications of myelodysplasia, and in the, what's called the French American British classification, he would be classified as a refractory anemia, or primary refractory anemia, whereas in the World Health Organization classification, which is a little bit newer, he's a refractory anemia with

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multilinear dysplasia. It's a simple subtle difference of classification. It certainly wouldn't change what he has or his prognosis.
Q. Since your report refers to his specific cell

type of myelodysplasia as primary refractory anemia, can we just use that term today?
A. Yes.
Q. And to the extent that I use MDS, as a substitute of trying to pronounce myelodysplastic syndrome, is that okay with you?
A. Yes.
Q. Can benzene exposures cause primary refractory anemia that Mr. Cowey has?
A. Yes.
Q. Is it a progressive disease?
A. Myelodysplasia is generally considered to be a progressive disease, yes.
Q. Is it a permanent disease?
A. Well, the only way to reverse the disease completely would be to do a bone marrow transplantation. Aside from that, we have treatments that may modify its course, but it is a progressive disease.
Q. And in Mr. Cowey's case, as I understand it from looking at your report, he was not a candidate for bone marrow transplant; is that true?

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A. Yes, I believe he would not be a candidate.
Q. What does it mean by that?
A. Well, in order to do a bone marrow transplant, the first hurdle to pass is you have to have a suitable donor. And the best donors, of course, are a brother or a sister match. And someone of his age is not likely to have a brother/sister match that could be a suitable donor. And in order to find a donor, then one would have to go into what's called the unmatched donor pool.

And there one might be able to find a donor that matched with him, at least superficially, even though it was an unrelated person, but those transplants are more difficult to carry out and harder on the patient and less successful. So that simply because of his age and the potential -- lack of potential for a donor, I think that makes things difficult.

Secondly, and more importantly, his age and underlying heart disease, there are a lot of stresses to go through during a bone marrow transplant. One must receive a lot of immunosuppressant therapy, and be able to sustain serious infections, and it would be rather

hazardous for someone of his age to do that.
Q. I take it, you're not critical of Mr. Cowey's treating physician for not putting him through that?
A. No, I would not think he's a good candidate

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for that.
Q. Do you know Dr. Youman, his treating physician in, Austin?
A. I don't think so. I may have met him many years ago. I can't place him.
Q. Were you aware that he did some work out at the Mayo Clinic?
A. No. But I don't know his background completely.
Q. Were you familiar that he worked, at least underneath -- for a short period of time, under a Dr. Wintrobe, that is at least of some reputation in your field of hematology?
A. Yes. I met Dr. Wintrobe many years ago, and I know Dr. Wintrobe.
Q. And is he in, at least in some regards, considered to be the father of hematology, that subspecialty?
A. Yes.
Q. I take it, that's one of the books that you have in your library as a resource?
A. Yes.
(Exhibit 4 marked.)
Q. (BY MR. LUBEL) Have you had a chance to look at Dr. Irons' testimony?

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A. Yes.
Q. Have you looked at -- I guess you hadn't seen Dr. Raabe's testimony or looked at his deposition?
A. No, I wouldn't have seen his deposition. There is a report, I believe, in here by him, but I have not seen his deposition.
Q. Generally, in your work as an expert in these cases, do you look at the other experts' reports and depositions?
A. Well, if they're available. Now, commonly I will not have those at my disposal when I write my report.

Q. I take it that Mr. Dillard has asked you to attend trial in Beaumont, Texas, correct?
A. Yes. He's let me know this case may come to trial, yes.
Q. What is your understanding of when that may happen?
A. I don't remember the trial date.
Q. Do you anticipate between today and the trial day, which I'll represent to you is from the last words we got from the court, as sometime around the 10th of December, do you anticipate looking at the other experts' deposition testimony to the extent that Fulbright & Jaworski sends you that information?

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A. Yes.
Q. Is that generally what you do before you give testimony if it's available?
A. Yes.
Q. Exhibit 4, can you identify that for us?
A. That's a curriculum vitae on myself.
Q. That's your resume?
A. Yes.
Q. Is it current?
A. Yes.
Q. When was the last time it was updated?
A. Oh, I don't know. Probably several months ago. Probably several months ago. But I would say it's reasonably current.
Q. Dr. Natelson, what is your opinion as to whether or not, based upon reasonable medical probabilities, the MDS that Mr. Cowey has will ultimately result in his death?
A. Well, I would say if he lives long enough, he'll die of the MDS. In other words, when you have an older person who has MDS, they frequently will die with it, not necessarily of it. But it is a progressive disease and frequently is lethal.
Q. If Dr. Youman has testified that Mr. Cowey's life-span from his MDS will be between six months and

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roughly 18 months, do you have any qualms with that?
A. Well, it might be a little bit longer, but I have not examined him, and I haven't seen his current

blood film or recent bone marrow, and so I wouldn't be in a position to estimate that as accurately as the other oncologist would be. But it's a progressive illness, and it could be as short as six months, yes.

Q. And you would agree that Mr. Cowey's treating physician, at least at this point in time, is in the best position to determine what his life-span is based upon his current condition?

A. Yes.

Q. In the October 2003 report, you identify 13 sources of information that you actually cite in your reports; is that correct?

A. Let me see. Yes.

Q. How did you find those?

A. These are all articles that, in some cases, I've had for a number of years. I may have found these through other cases. I may have researched the literature for some of them. In a couple of instances, I subscribe to these journals, such as Blood, and so I would have the article in the normal course of events. Cancer also is one that we subscribe to in this office, and the New England Journal of Medicine. So, in some

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cases, they're journals that would come across my desk; in other cases, I've looked them up.

Q. Do you make any effort to assess whether or not the studies themselves are unbiased?

A. I'm not sure what you mean by that.

Q. You understand that a lot of these studies you use are what we call epidemiology studies?

A. Yes.

Q. And bias can impact or influence the validity of those studies?

A. Yes.

Q. Is that true?

A. Yes.

Q. And sometimes bias is not just from the face of the study sticking out at you, sticking out like a sore thumb, something you can readily see, would you agree with that?

A. That would be true.

Q. And I'm just curious as to whether or not you, in your practice as a hematologist and an expert in

these chemical exposure cases, undertake any independent effort to see if the studies that you're going to use, you're going to rely upon, are unbiased, or otherwise reliable?

A. The answer to that would be no. I accept what

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is written. Now, it's put in context of other articles on the same subject, and so I may have several articles that address the same issue and I choose to reference one, but I have many others that show the same phenomenon.

Q. So would it be fair to say that to the extent that you have an opinion about an area related to hematology, and you place a cite for it, generally speaking, you're not going to give that opinion if there has just been one article on it?

A. Well, I think you have to cite a specific case. In other words, if you're talking about an epidemiologic study, we generally would like to have multiple studies that tend to show the same thing. If you're talking about, say, a paper describing a new treatment for an illness that's effective, that single paper may be very useful.

Q. But I thought, and I may have misunderstood you, the proposition that you were given was, generally speaking, you may only site one article with respect to a proposition; however, you generally have more than one article on that opinion?

A. Yes, that would be true.

Q. And would it be a fair statement that at least with regard to the 13 specific studies that you

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reference, that you've cited, in your October 2003 report, that you believe those to contain reliable information?

A. Yes.

Q. You have no reason to think otherwise?

A. I have no reason to think otherwise.

Q. And many times do you recognize the names of the authors because that's not the only study they've authored?

A. Yes.

Q. And over time do sometimes these authors start

to develop either a good or a bad reputation in their field?
A. Well, I'm not sure I can really answer that question accurately. Some of the names become very familiar.
Q. Let me ask you this, at least with respect to the authors that are listed in the 13 references that you have in your report, would it be fair to say that they've either got good reputations in your mind, or you're not aware of their reputation, but have no reason to doubt what they have to say?
A. Yes, that would be a reasonable statement.
Q. In other words, you know who Dr. Wong is, don't you?

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A. I've never met him, but I know who he is.
Q. And you understand that he's an expert that is frequently hired by the defense?
A. Yes.
Q. And, in fact, he writes a lot of defense slanted articles?
MR. DILLARD: Object to the form.
MS. YATES: Same objection.
A. He writes frequently on benzene exposures and consequences thereof, and epidemiologic articles, yes.
Q. (BY MR. LUBEL) Were you aware that Dr. Wong's work has been funded by industry?
A. I've been told that numerous times. I haven't verified that. I have no reason to doubt it.
Q. Did Mr. Dillard or Mrs. Yates or any of the lawyers at their respective law firms, did they tell you that the studies that industry was running over in China right now that Dr. Irons was working on, that they were funded by industry, and that the main goal of those studies was to effect regulations, number one, and number two, lawsuits that they were in?
MR. DILLARD: Object to the form.
MS. YATES: Same objection.
A. I was told none of those things.
Q. (BY MR. LUBEL) Well, to the extent that the

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studies that Dr. Irons and others are working on in China, that are being funded in whole or in part by

industry, to the extent that, in fact, the purpose of those studies is to help defend them in lawsuits, and to effect regulations on their behalf to their benefit, in the United States, would you with agree those studies would not be unbiased?

MR. DILLARD: Object to the form.

MS. YATES: Objection, form.

A. Not necessarily. In other words, if these gentlemen are there to assess accuracy of data and to simply ensure a flow of good data, and an accurate interpretation of that data, that might not necessarily be biased toward one side or the other.

Q. (BY MR. LUBEL) But how can it be an accurate flow of reliable data if, in fact, their goal is to benefit them in lawsuits and to help them in regulatory matters?

MR. DILLARD: Object to the form.

A. Well, I don't know what their goal is. In other words, you're telling me that that's their goal. But it depends on who these gentlemen are and what their credentials are and what they hope to find there. And the fact that they're sponsored by a particular organization doesn't mean that they're going to alter

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their data to suit that organization.

Q. But does it make a difference in your mind if the purpose of the studies, irrespective of who sponsors it, the purpose of it, is to help industry in their lawsuits, would you agree that that would make a study biased?

MR. DILLARD: Object to the form.

MS. YATES: Object to the form.

A. Not necessarily.

If I could give just a simple example, many of our drug studies that we see today on new agents, in fact, an agent that this patient, Mr. Cowey, received, those studies are funded by industry. And they're looking for data.

Q. (BY MR. LUBEL) Funded by industry to protect them in lawsuits?

A. No, but to promote a product.

Q. Tell me about studies that you're aware of that you believe are legitimate where the purpose of the

study is to protect industry from lawsuits.
MR. DILLARD: Object to the form.
A. I don't know of any such studies.
Q. (BY MR. LUBEL) Tell me of any studies that you're aware of that are legitimate that you would rely upon where the purpose of the study was to lower what's

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permissible for benzene exposures as to make regulations less burdensome on industry?
MR. DILLARD: Object to the form.
A. Again, I don't know of any such studies designed with that intent in mind.
Q. (BY MR. LUBEL) Have you ever relied on any such studies?
A. Not that I'm aware of.
Q. If you would, if you would look at the citation or the reference list that you have in your October report and just tell me the authors that you're familiar with.
A. Well --
Q. By name.
A. By name. The first reference is Dr. Carl Aul, who has written many articles over the last several years on myelodysplasia, and I've not met him, but I recognize his name.
The next article, the first author is Heaney, I don't think I know him. I may -- if we look at the paper, there may be other authors whose name I recognize, but his was a review article from the New England Journal of Medicine on myelodysplasia.
The next one, I don't know that person either. That was simply a compilation of leukemia statistics in

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the United States that was published in the Journal of Cancer.
The next one, Maes, I don't know that doctor personally.
The next, Williams, I don't know that person. But the second author on that has published previously on the Pliofilm cohort, Paustenbach, or something of that nature.
Smith has published articles before on therapy-induced leukemia, I recognize that name.

Finkelstein, I don't know.
Hayes is a person who has published several articles on statistical analysis, particularly of benzene-related studies in China.
Q. What type of reputation does -- is it a man or a woman, Hayes?
A. I believe he's a man.
Q. What type of reputation does he have in that field?
A. I don't know anything bad or good about his reputation.
Van Leeuwen, this is a person I don't know. He wrote a nice treatise on secondary leukemias and myelodysplasia.
Dr. Wong, as I say, I've never met. I've

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certainly seen many of his papers that have to do with epidemiologic studies, disease causation.
Dr. Pedersen-Bjergaard is well published in the field of genetic defects consequent to chemotherapy drugs and therapy-related leukemia and myelodysplasia.
The same would be true of the next author, Mauritzson, has published a number of articles in this field.
Smith is from the University of Chicago group, I have listened to lectures by some in that group. I don't place this particular doctor.
Q. The Hayes study that you referred to, that's a study out of China, correct?
A. Yes.
Q. Have you seen his study in the year 2000?
MR. DILLARD: Object to form.
MS. YATES: Same objection.
A. You have to point at which study are we talking about.
Q. (BY MR. LUBEL) Hayes and Yin and others published a study, in the year 2000, kind of an update of the Chinese work, and I was wondering if you have seen that.
A. I don't recall that specific article. I may have it in my files, but I don't recall it.

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Q. To the extent that one of the conclusions that

that group reached -- and I take it, that you understand that the two groups that did the work that Hayes was involved with in China were the National Cancer Institute and the equivalent organization in China, were you aware of that?

A. Yes.

Q. To the extent that one of the conclusions that they reached in the updated 2000 study was that there was a relative risk of three for benzene exposures less than 10 parts per million leading to or linked to MDS, do you have any qualms with that, as you sit here, without seeing the study?

A. Well, I don't -- if you say that's what they found, that's what they claim. I know that there is great dispute about what the levels of exposure were during the Chinese study.

Q. I understand. But were you aware that they had -- in the 2000 study, they had actually gone back and gotten better exposure data?

A. I don't know that they got better data, but they -- there remains dispute about benzene exposures in China.

Q. Is that something that you have studied with any particularity or focus?

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A. Well, I've been in China for a couple of weeks, and I can understand some of the problems there from what I saw and the difficulty in obtaining good data, but -- and I can understand why in some of the articles -- before I went to China, I couldn't really understand why some of the articles would say to get follow-up on the patients, we asked their neighbors or checked down at the local police station, and I thought to myself, that's very bizarre, why would they say that. And now having been to China for a couple of weeks, I know why they would say that.

Q. Why is that?

A. Well, because their society is so utterly different from ours, and their factories, their universities are not on par with ours, by many orders of magnitude. And I can understand why data would be difficult to accurately estimate from recordkeeping.

Q. But without seeing the 2000 study, you don't

know whether you have any disagreements with it or not, correct?
MR. DILLARD: Object to the form.
A. Well, I wouldn't be able to, of my knowledge, know whether their estimates are correct or incorrect, in other words.
Q. (BY MR. LUBEL) Well, I mean, generally

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speaking, without you having the 2000 study, and having time to review it, and then you and I sit and visit and have a conversation about it, at this point, since you hadn't done that, you can't say whether you have any problems with that study or not?
MR. DILLARD: Object to the form.
A. Well, I can't comment about the study in detail because I haven't seen it. Or if I have seen it, I don't recall what it said.
Q. (BY MR. LUBEL) Fair enough. What independent efforts have you made to assess the conclusions in the pre2000 Chinese studies?
A. Well, in the pre2000 studies, there have been several, you might say, rebuttal reports from different people. Dr. Wong has written one. There is another one from a fellow named Budinsky. There is a third one, also by Wong, more recently, about those studies. And, of course, there is mention of those studies in some of the articles from OSHA and regulatory bodies that I have here are commenting about those studies.
Q. What do they say about that?
A. Well, they simply say that those studies -- I'm paraphrasing what they say -- that the relative risks for benzene exposure and leukemia must go back to the Pliofilm study because of discrepancies in these

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particular studies in China.
Q. Do you have that information here? Is it the I R I S?
A. That's one report that says that, yes.
Q. Is there anything else that you've got?
A. Actually, you've got it right there. This report.
Q. Sorry about that.
A. Yes. This report here, which is from the

National Center for Environmental Assessment.
(Exhibit 5 and 6 marked.)
Q. (BY MR. LUBEL) If you would, I'm just going to go ahead and hand you, and we'll just go off the record for a minute, Exhibit Number 5 and 6, which are both of the documents you just referred to, and if you can just open these reports to the sections that you're talking about.
A. Thanks.
(A break was taken from 2:45 to 2:46.)
Q. (BY MR. LUBEL) If you could just reference the exhibit number as you're talking about it as you do it.
A. All right. The first one is, Carcinogenic Effects of Benzene, an Update, and this is from the National Center for Environmental Assessment, and it was

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written in 1998.
Q. Is that Exhibit 6?
A. Yes. This is by Bayliss, I believe, is the lead author. And talking about the Chinese studies, "the second potential problem was the development of exposure estimates for the 74,282 workers, during the earliest period, only three percent of the exposure estimates were based on actual measurements."
And the sum and substance of what's in this article is that the reference study for consequences of benzene exposure in leukemia, as they put it, hence the Pliofilm workers are the preferred population for estimating the effects for exposure to benzene.
Q. What's the date of that article?
A. This is 1998.
Q. So that would have been before the 2000 Hayes study, correct?
MR. DILLARD: Object to form.
MS. YATES: Form.
A. Yes. This is written in 1998. And we called Mr. Bayliss to see if there was a more recent update of this, several months ago, and he said no.
Q. (BY MR. LUBEL) Great. And you've shared with us your comments about that particular article, Exhibit 6, as they relate to this case?

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A. I don't know. I -- let's see if I referenced that.
Q. It may not have been on your reference list.
A. I don't believe it's on my reference list.
Q. Well, then, have you at least shared with me your comments with regard to the questions I asked you about the Chinese exposure levels, that are referenced in Exhibit 6?
A. Yes, to the extent that they're discussed in there.
Then --
Q. Just for the record, you're looking at Exhibit Number 5 right now, correct, Dr. Natelson?
A. That's correct. And from this article, on page 10 of 16, the Pliofilm workers study provides the best published set of data to date for evaluating human cancer risks from exposure to benzene.
Although the ongoing Chinese cohort studies perhaps may provide information in the future to better characterize risk of cancer at low dose exposure, their use in the derivation of risk estimates remains problematic at present for the reasons cited in section 2, A2.
So, essentially, these two articles are telling us the same thing, that we need to rely on the

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Pliofilm cohort of workers for assessment of risk concerning benzene and leukemia.
Q. Now, this was last revised, at least according to the document, Exhibit 5, on January 19th of 2000. Did you see that?
A. I don't recall the date on it. Yes.
Q. So, it wouldn't appear that if the updated Hayes article, in 2000, was released after that date, whether they had the opportunity to review the updated information?
A. I don't know that they did.
Q. What caused Mr. Cowey's MDS?
A. Well, I think that as in most cases of MDS, the cause would be unknown.
Q. Have you considered the generally accepted known risk factors of MDS as the potential cause of his disease?

A. Yes.
Q. And what are the generally accepted known risk factors for his disease?
A. Well, the primary risk factor would simply be age. We know that the incidents of both acute leukemia and acute myeloid leukemia and myelodysplasia goes up strikingly with age, and so that the vast majority of patients you see with these diseases are older than 60,

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frequently older than 70.
Q. Were there any other risk factors?
A. Well, certainly risk factors are prior chemotherapy or, let's say, chemical treatments, chemical-related treatments, or radiation, may predispose to these illnesses.
Q. Are you aware that benzene exposure was considered a risk factor for MDS?
A. Yes.
Q. And is that generally accepted within the knowledgeable scientific and medical community?
A. Yes.
Q. That's textbook medicine, correct?
A. It's commonly thought to be the case.
Q. What I mean by textbook medicine is, is that we're not talking about just one article that somebody publishes somewhere that says we think benzene causes MDS, it's gotten past that stage to where there is multiple articles that lend credence to it that allows for textbooks to put it in there, correct?
A. Well, we have general acceptance that benzene causes acute myeloid leukemia. And many textbooks point that out.
Now, myelodysplasia, that term, is a relatively new term. But in general, we believe that

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anything that causes AML can cause myelodysplasia.
So, by inference, even if you couldn't find an article that said benzene causes myelodysplasia, if we say, benzene causes acute myeloid leukemia, it tells us it can cause myelodysplasia.
Q. And is that an appropriate way to look at it from a scientific and medical standpoint?
A. I think so.

Q. And is it, likewise, appropriate for scientists and medical people, such as yourself, to consider the literature that's been written on acute myeloid leukemia in order to assess MDS?
A. Yes.
Q. What other names has MDS gone by?
A. Well, for many years we simply used the term called preleukemia for certain of the myelodysplasia syndromes.

Now, other illnesses -- myelodysplasia encompasses several different syndromes. And we knew some of those syndromes by different names; for example, idiopathic sideroblastic anemia, or refractory sideroblastic anemia, is a classification of myelodysplasia, but that has been a stand-alone disease for many years and has its own ICD-9 Code.

Chronic myelocytic leukemia, again had been

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known for many years, has its own ICD-9 Code as a stand-alone disease. Those two particular diseases were lumped under the general category of myelodysplasia, I would guess probably around 1982, and into the classification system.

But prior to that, we used a simple term, preleukemia, to describe an illness like Mr. Cowey's.
Q. In order for you to link up a person's MDS to benzene exposure, what evidence must you have?
A. Well, we -- it's my belief, and I believe well substantiated by the literature, that what we call chemical-related leukemia, or secondary leukemia, is what benzene is.

In other words, benzene is an obsolete chemotherapy drug, and its mechanisms -- potential mechanisms leukemogenesis are identical to our modern chemotherapy drugs. So what we say for modern chemotherapy agents should go for benzene.

Now, there is a big difference between modern literature and benzene literature. In the case of benzene literature, the problem is you have a handful of cases and often very poor data on exposure.

In the case of therapy-related leukemia and myelodysplasia, we have exquisite data. If a person didn't smell something bad, he got injected with a gram

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of chemical IV, so we know exactly what they got and when they got it.

We also, because we commonly keep statistics on cancer patients, if they were to develop a secondary leukemia, or myelodysplasia, we know exactly what the latency period would be, and whether it differs for different chemotherapy drugs. And we also know what type of leukemia or myelodysplasia occurs following chemotherapy. We also know the results of treatment and life-span in people in those circumstances.

So, some of the articles I have here relate to that, so we can say that there are certain features of chemotherapy-induced leukemia. And those features might be that intensity of dose is very, very important. That a trivial exposure is not likely to cause an illness, it needs to be of a large magnitude. And exposures that are extremely large magnitude may accelerate that latent phase, and so you may get AMLs developing fairly early in people who get, let's say, industrial strength chemotherapy, as we use for bone marrow transplantation and other treatments.

We also know that certain types of leukemia have different chromosome patterns, and so certain types of therapy-related leukemia occur very early on to the final chemical exposure.

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But in general, what we know is, that one needs a very large exposure, that the illness that one gets is acute myeloid leukemia and its associate of myelodysplasia being one of them. That the latency period after that can be as short as a year, but generally, most people feel averages out about five years, and that typically comes back to baseline about 10 to 12 years after the last exposure. So that the latency period is very well defined for chemotherapy-induced leukemia.

We also know that there are very characteristic chromosome changes in therapy-induced leukemia. For example, the common ones that are seen are abnormalities of chromosomes five and seven, and in most series, what you find is that in a therapy-related leukemia setting, 70 to 75 percent of the patients that

have chromosome abnormalities will have them of chromosomes five and seven. It's certainly not exclusive to those chromosomes, but that is a very common pattern.

We also would say that now that we know what therapy-related leukemia is, and how it's defined by thousands of cases, we can now go back and take the few benzene cases we have and see if it fits into that paradigm. And it does very nicely, because, for

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example, in the Hayes articles, he says that all his cases of leukemia and myelodysplasia occurred in the first 10 years from the last exposure. That fits exactly with what we know from our benzene exposure.

And so what that tells me, for example, is if I find a patient, for example, and they have had chemotherapy 30 years ago, and now they catch leukemia, it's not very likely that that leukemia is from that chemotherapy because the latency period is wrong.

If we look at chromosome data, there are certain chromosome abnormalities that can't prove therapy-related disease, but are very suggestive of it.

On the other hand, there are certain chromosome abnormalities that are much more suggestive or de novo for off-the-street leukemia and myelodysplasia.

And so by looking at the intensity of the exposure to the alleged chemical, by looking at the type of illness, what kind of leukemia are we talking about, or myelodysplasia are we talking about, what latency period we have, what chromosome abnormalities we have, when we put all of that together, I think we can make a good estimation as to whether or not it's reasonable or likely to consider this a therapy-related leukemia or unreasonable to consider it a therapy-related leukemia.

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Q. In order for you to link a person's benzene exposure, assuming it was high enough, to acute leukemia, or MDS, do you require that they have chromosome abnormalities at five and/or seven?

A. No.

Q. Why not?

A. Because of the fact that we know that you can

see people with de novo leukemia, who have never had any association with chemicals, have a five and seven abnormality. So having a five and seven abnormality doesn't prove therapy related, it just simply makes it more likely.

Q. And the reason, I take it, that you think it's more likely is because you see more of those chromosome abnormalities in the exposed people that have the disease?

A. Correct.

Q. But there is no study that you're aware of that concludes that in order for a person to have a benzene-induced MDS, they must have those chromosome abnormalities?

A. No. We generally say that if we're looking at leukemia that's de novo, let's say, off-the-street leukemia, probably as many as 40 percent have no chromosome abnormalities at all, yet they have leukemia.

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We generally think that if a person has therapy-related leukemia, or myelodysplasia, that actually almost all of them will have a chromosome abnormality, probably upwards of 90 percent.

So that usually if it's therapy related, there is a chromosome defect, and the common ones that are seen are five and seven, but that's not exclusive.

Q. How much exposure is necessary for a person to have in order for you to link their benzene exposures to the disease MDS?

A. Well, I would have to consider it to be similar to what we see for acute myeloid leukemia. And it's generally claimed that the cumulative part per million exposure is most closely related to the development of AML. And there is argument about how much that cumulative exposure is, but most people say at least 100 to 200 part per million years exposure. Some would say higher.

Q. And where do you fall within that exposure debate?

A. I would say at least a hundred part per million years.

Q. Now we've talked about exposure, we've talked about the chromosome issue, and I believe you told me

earlier that his cell type of MDS was consistent with
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benzene exposure, correct?
MR. DILLARD: Object to the form.
A. Yes.
Q. (BY MR. LUBEL) You mentioned treatment. Does that play a part? I was kind of making a list as you were going through.
A. Oh, I see. How do you -- you mean treatment for the myelodysplasia?
Q. As far as --
A. I think I understand what you're referring to. Generally speaking, what we say is that if we have a leukemia, an AML, there are certain things that let us know that this leukemia might be very responsive to treatment. One of them would be to not have any chromosome abnormality. And we certainly see that in de novo leukemia. That's a good prognostic sign.
On the other hand, if we know that the leukemia is therapy related, and, for example, we saw a a five and seven chromosome abnormality, that would tell us the prognosis is terrible.
And the same chemotherapy that might cure someone with an absent chromosomal abnormality will very likely fail in someone who's got therapy-related leukemia. It's considered to be a worse disease.
Q. Do you agree with Dr. Youman when he said --
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and I know you haven't been furnished his testimony, but assume with me he said that Mr. Cowey's chromosome abnormality of Trisomy 8 was consistent with benzene exposure?
A. Well, it isn't the classical one for benzene exposure -- actually, I have read his deposition.
Q. Oh, okay.
A. And I'm aware of what he said. And what I can say is that if you look at patients with therapy-related myelodysplasia, there are reports of Trisomy 8 among such patients.
So it is not unheard of for a person with therapy-related myelodysplasia to have Trisomy 8, but it is not the typical chromosome abnormality for that group.

Q. Do you know what percentage of the therapy-related AMLs or MDSs have Trisomy 8 abnormality?
A. Yes, it's in one of these articles.
Q. Can you find that for us, please?
A. The best article would be the Mauritzson article. There are really two articles that I have here that bear on that. The first is the -- are these two articles. The first is the Smith study from the University of Chicago, and these are 306 patients with therapy-related myelodysplasia and AML. And of this

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group none have Trisomy 8.

Now, the second one by Mauritzson is a much larger study, it has to do with 5,098 patients. In this article, specifically under myelodysplasia, what they find is that in off-the-street myelodysplasia, de novo myelodysplasia, they had a 13 percent incidence of Trisomy 8, and in therapy-induced myelodysplasia, they had a two percent incidence of Trisomy 8.

So that Trisomy 8 was, as they put it in their abstract, abnormalities significantly more common in de novo disease were Trisomy 8 as a sole abnormality. But that's from their table.

Q. (BY MR. LUBEL) I'm going to ask the court reporter to make copies of these and make them Exhibit 7 and return them to you today. Is that okay?
A. Fine.
MR. LUBEL: We'll mark both of those articles as Exhibit Number 7.
MR. DILLARD: Why don't you hold off and get him to mark one so that we're not marking on his original.
(Discussion held off the record.)
(Exhibit 7 marked.)
Q. (BY MR. LUBEL) Are both the studies that you just referenced for us and we're going to mark as

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Exhibit 7, are they listed in your references?
A. Yes.
Q. Are those articles that you've used before?
A. Yes.
Q. You didn't find them specifically for this case?

A. No. Neither one of those are specific to this case.
Q. Did any of the literature that you've referenced in your report require you, for this case, to go seek more information, or did you have it from previous cases?
A. I had it from previous cases, and also from my general files. In other words, I have a large file on myelodysplasia with many articles that have not been used in any particular action.
Q. Now, I've had a chance to read your testimony in the Ward case.
A. Yes.
Q. That you gave to -- Mr. Hyde took your deposition?
A. Yes.
Q. And I know from that case that you do not believe that all cell types of MDS are causally related to benzene.

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A. That's true.
Q. Do it either way, you can tell us the ones that you think that are or the ones that aren't, whichever is shorter.
A. Well, it's easier, I think, to do it, to say the ones that I don't think, because it's primarily the one illness we call idiopathic sideroblastic anemia, or refractory anemia with ring sideroblasts. And that's an illness that's been well studied for many, many years and has never been related to benzene exposure.
And in the Hayes study, for example, of the 74,000 people, none of those patients had refractory anemia with ring sideroblasts. And as I said in questioning, in selecting out large numbers of people with refractory sideroblastic anemia, and querying them as to whether or not they've had any benzene exposure, the answer is no.
So there would be no reason, no epidemiologic study, nothing to suggest that that particular illness is benzene related.
Q. As you sit here today, would it be fair to say that that particular cell type, at least at this point in time, is the only one that you're willing to come out

and say is not causally related to benzene?
A. Yes.

Q. Based on current information?
A. Based on current information, yes.
MR. LUBEL: Do you mind if we take about a three-minute break?
THE WITNESS: No.
MR. LUBEL: I'm almost done.
(A break was taken from 3:12 to 3:17.)
Q. (BY MR. LUBEL) What does the term "latency period" mean?
A. Well, latency period would be, in the case of AML, or myelodysplasia, a period between which a chemical produces some sort of damage in the organism, or in this case, DNA, and the time at which the consequences of that become evident, usually in the range of low blood counts or anemia or something of that nature.
Q. Why is it when people have cancer there is generally a number of years between the time that the damage was done and the consequences are noticed?
MR. DILLARD: Object to the form.
MS. YATES: Form.
A. There are many theories on that, but one thought is that damage begets damage, and what may happen is that an event occurs, such that a chromosome is damaged. The body has tremendous reparative

mechanisms to try to fix that, but sometimes it's not fixed, and the defect is carried over from cell division to cell division to cell division, and eventually the defect may cause a second defect to occur, and then a third, and finally, you have enough of a problem where it becomes evident in the cell counts, and that may take a certain amount of time.
And that's probably the reason that we have a window of latency, because, in other words, it's like -- it's as though the body has a certain period of time to fix something, and if it doesn't get it fixed, something bad is going to happen. And if it goes through such a long period of time, in some instances, it's too late for the bad thing to happen. The body has finally fixed

it.
And so I think that's why in the case of acute leukemia, we have a window why we don't have leukemia just occurring randomly after these exposures to chemicals.
Q. At what point in time do you start to calculate the beginning point of a latency period?
A. Well, I generally calculate that from the last chemical exposure.
Q. Is there a study or textbook that tells you that that's the way you calculate it?

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A. No, I don't think I've seen that. But the reason that that's done is because most of the time with cancer chemotherapy, we're talking about an onset of treatment and a completion of treatment that are not too far distant from each other. In other words, we don't often give people chemotherapy day after day, year after year. We give it for a finite period of time. So if you looked at the first exposure and you looked at the last exposure, it wouldn't be a vast difference, in terms of latency.
And so, generally, most of the authors will calculate it from when the treatment was completed to the time the illness appeared.
Q. So most people calculate it from the last day of the chemical exposure as opposed to the first day, would that be a fair statement?
A. I think that's true.
Q. But when you look at chemotherapy treatments by and large, it doesn't make a difference what study you use?
A. Because of the fact that within reason, they are -- they are spaced close together. Now, in some instances, what you'll find is a person will be treated, few years will go by, and they relapse, now get a second set of treatment, and so there, there is a big disparity

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between the first treatment and the last treatment. But most will take the last treatment as the point in time.
Q. What is the range of latency periods for AML?
A. We generally say, and many of these articles bear on that, and comment on that, we generally say that

it is -- the upper limits are 10 to 12 years.
Q. The upper limits?
A. Yeah. In other words, that most leukemias occurring from secondary causes occur from, let's say, year three to year seven, and under certain circumstances, they could occur as early as year one, or might occur as late as year 10 to 12.
Q. What affects that, whether it happens in year one or year 10 or 12?
A. In some instances, it's the intensity of the treatment. It appears as though you may get leukemia earlier with very intensive regimens.
Q. You're talking about chemotherapy treatment?
A. Yes. I'm talking about chemotherapy treatment.
Q. Let's digress for a minute and talk about benzene exposures. Do any of your articles tell us what the outer limits are for benzene exposures outside of the chemotherapy arena?
A. Yes. In other words, the Hayes articles, as I

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mentioned, says that. If we could find that article. He says, the temporal pattern of benzene exposure appears to be important in determining the risk of developing a specific disease. And then he says that, in our study, ANLL/MDS was linked to recent benzene exposure, and additional distant exposure did not appear to further increase risk. This finding suggests that recent benzene exposure is predictive of substantive risks for ANLL/MDS analogous to the short latency and wave-like increase, then decrease in risks seen for several forms of radiation-induced leukemia and for chemotherapy-related AML.

In other words, as I read this, he is saying that benzene is exactly identical to what we see for therapy-related leukemia.

Q. When was Mr. Cowey's last exposure?
A. Well --
Q. Or do we know?
A. I don't think we know that. Now, he alleges the use of Liquid Wrench, and having seen many experts describe Liquid Wrench, it is my understanding that after 1978, it did not contain benzene. And so one

would say that were he using Liquid Wrench, and did it contain benzene at the time he was using it, the last potential exposure would have been around 1978.

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Q. To that product?
A. To that particular product, yes.
Q. But to the extent that he had exposures to benzene in other products, that wouldn't surprise you, would it?
A. I don't know what his other exposures were, really. I've read his deposition. But I don't know what the -- what his potential would be for other benzene exposures in products. He talked about doing house painting and a number of other projects.
Q. You recognize that there is benzene, albeit in small amounts, in paints and paint thinners and things of that nature?
A. Yes. Benzene would be a contaminant in many solvents.
Q. It's even in gasoline?
A. Yes, it is in gasoline.
Q. So, it wouldn't surprise you if he had other exposures after 1978?
A. No, it would not.
Q. And are you prepared at this point in time to say when you believe Mr. Cowey's latency period should start to run for calculation purposes?
MR. DILLARD: Object to the form.
MS. YATES: Form.

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A. Well, I basically have his deposition to go on as to what he did. And of the materials he mentions, the only one that would have a potential for substantial amount of benzene in it would be the Liquid Wrench.
In other words, that is a compound that might have as much as five percent benzene in it, whereas you certainly wouldn't find five percent benzene in house paint, or anything close to that.
So that if I looked at what did he use that had the potential for, from his description, significant benzene, that would be the most prominent compound.
Q. But I thought benzene was a cumulative disease? Benzene exposure causes a cumulative disease?

A. Benzene is cumulative, yes.
Q. So, each and every exposure can contribute to the disease process?
A. Ah, I see what you're driving at. Yes, that is correct.
Q. So, to the extent that he had exposures pre'78, and then he continued to have some exposures post'78, although, in your opinion, less significant, or smaller, they would still -- his last exposure in time for latency purposes would still be after 1978, would they not?
A. Well, I don't know when his last exposure

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would have been.
Q. That's fair enough.
Now, have you read Dr. Irons' testimony?
A. Yes.
Q. He says in here that he believes that the outside time limit for the latency period is 15 years. Do you remember seeing that?
A. Yes.
Q. In terms of quantitative numbers. And then I asked him could he pull the peer reviewed studies that state that opinion, in order for a malignancy to be related to benzene, the malignancy must manifest itself within 15 years of last exposure, and he said there was no literature that specifically was going to say that. Do you agree with that statement?
A. I'm not aware of such literature other than comments such as we see in the Hayes article, for example.
Q. You're not saying that the Hayes article stands for the conclusion that for a person to have a benzene-induced malignancy, such as AML or MDS, that it must manifest itself within 10 years of the last date of exposure, are you?
A. Not within 10 years, no. I think it could be a little longer than that. In other words, if you had

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exposure to benzene, and it was of the magnitude to cause leukemia, and then that exposure ceased, you might go 10 to 12 years before you caught the leukemia. But I don't think much longer than that, based on what we know

from chemotherapy data.
Q. And it's your opinion that the reason there is a range between one year to 10 to 12 years, or whatever it is, that, generally speaking, that range seems to be dependent upon the size of the exposures, or I believe you said intensity?
A. Well, that is one factor. It's the intensity of the exposure. In some cases, with chemotherapy, it's the specific drug used. Certain drugs tend to promote the leukemia to occur earlier than others. The concomitant use of radiation therapy. There would be other confounding factors. But there is a distribution curve when that leukemia occurs.
Q. Did smoking cause Mr. Cowey's MDS?
A. Well, there would be no way to know. We -- there are articles that suggest that myelodysplasia, like acute leukemia, is at slightly increased risk in smokers.
Q. What's your opinion based on his smoking history?
A. Well, he ceased smoking sometime earlier

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before this illness. I've forgotten -- I'd have to review what I said about his smoking history. I don't remember how many years he smoked.
Q. In your report you don't make any statement that smoking contributed to cause his disease. That's why I'm asking. I want to make sure.
A. I see. No. Well, it's generally thought that a person who smokes for many years, let's say 20 or 25 years, can accumulate as much as four to fix part per million years of exposure. That's well below what we think causes AML. But it is benzene exposure. And it is well known that a smoker generally is exposed to 10 times the amount of benzene that a nonsmoker is. But do I think myelodysplasia caused his illness -- do I think that smoking caused his illness of myelodysplasia, I would have no reason to think that.
Q. How about, do you think that chemotherapy agents or drugs caused his MDS?
A. We have no record that he received any such drugs.
Q. So the answer, I take it, based upon what you

have before you is that it didn't?
A. Yes. We don't have any evidence.
Q. Same question for radiation treatment, or ionizing radiation.

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A. Yes. There was no evidence he received radiation. His treatment for the prostate cancer was surgical.
Q. And I take it from what I've heard you say today, you do not believe that benzene contributed to cause this disease?
A. His disease of myelodysplasia we're talking about?
Q. Correct.
A. No, I don't think that benzene contributed to his disease.
Q. And that's based upon what?
A. His deposition; in other words, my reading of his deposition would be that he is not comparable to someone working in, let's say a benzene-related industry, that his exposures were casual, as most handymen, including myself, would have over the years and could not have been of the magnitude to give him a hundred part per million years exposure.
Q. Are you relying on any independent basis for that other than your own opinion, based upon your own work?
A. No. That's just my opinion.
Q. In other words, you're not relying on any exposure data outside of what you've seen from

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Mr. Cowey's own testimony?
A. That's correct.
Q. Did you use Liquid Wrench?
A. No, I never used Liquid Wrench.
MR. LUBEL: Enjoyed meeting you. Thanks for your time.
MR. DILLARD: We'll reserve ours. Thanks.
MS. YATES: Reserve ours.

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CHANGES AND SIGNATURE.

PAGE	LINE	CHANGE	REASON

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I, ETHAN A. NATELSON, MD, have read the foregoing deposition and hereby affix my signature that same is true and correct, except as noted above.

_____
ETHAN A. NATELSON, MD

THE STATE OF _____)
COUNTY OF _____)

Before me, _____, on this day personally appeared ETHAN A. NATELSON, MD, known to me (or proved to me under oath or through _____) (description of identity card or other document) to be the person whose name is subscribed to the foregoing instrument and acknowledged to me that they executed the same for the purposes and consideration therein expressed.

Given under my hand and seal of office this _____ day of _____, _____.

_____
NOTARY PUBLIC IN AND FOR
THE STATE OF _____
COMMISSION EXPIRES: _____

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CAUSE NO. A-167,693

JAMES COWEY AND RUTH COWEY	) IN THE DISTRICT COURT
PLAINTIFFS,	)
VS.	) JEFFERSON COUNTY, TEXAS
RADIATOR SPECIALTY COMPANY, ET AL	)

)
DEFENDANTS.)
) 58TH JUDICIAL DISTRICT

REPORTER'S CERTIFICATION
DEPOSITION OF ETHAN A. NATELSON, MD
DECEMBER 3, 2003

I, Mark A. Miller, Certified Shorthand Reporter in and for the State of Texas, hereby certify to the following:

That the witness, ETHAN A. NATELSON, MD, was duly sworn by the officer and that the transcript of the oral deposition is a true record of the testimony given by the witness;

That the deposition transcript was submitted on _____ to the witness or to the attorney for the witness for examination, signature and return to me by _____;

That the amount of time used by each party at the deposition is as follows:

MR. LUBEL.....1 HOURS:17 MINUTE(S)

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That pursuant to information given to the deposition officer at the time said testimony was taken, the following includes counsel for all parties of record:

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I further certify that I am neither counsel for, related to, nor employed by any of the parties or attorneys in the action in which this proceeding was taken, and further that I am not financially or otherwise interested in the outcome of the action.

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Further certification requirements pursuant to Rule 203 of TRCP will be certified to after they have occurred.

Certified to by me this 5TH of DECEMBER, 2003.

_____

Mark A. Miller
Texas CSR No. 1190
Expiration Date: 12/31/04

Nell McCallum & Associates
Firm Registration No. 243

0065

FURTHER CERTIFICATION UNDER RULE 203 TRCP

The original deposition was/was not returned to the deposition officer on _____;

If returned, the attached Changes and Signature page contains any changes and the reasons therefor;

If returned, the original deposition was delivered to _____, Custodial Attorney;

That $_____ is the deposition officer's charges to the Plaintiff for preparing the original deposition transcript and any copies of exhibits;

That the deposition was delivered in accordance with Rule 203.3, and that a copy of this certificate was served on all parties shown herein on and filed with the Clerk.

Certified to by me this _____ day of _____, 2003.

_____

Mark A. Miller
Texas CSR No. 1190
Expiration Date: 12/31/04

Nell McCallum & Associates
Firm Registration No. 243