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

GERHARD K. RAABE, Dr.P.H., F.A.C.E.

DECEMBER 2, 2003

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ORAL DEPOSITION OF GERHARD K. RAABE, Dr.P.H., F.A.C.E., produced as a witness at the instance of the PLAINTIFFS, and duly sworn, was taken in the above-styled and numbered cause on the 2nd of December, 2003, from 8:57 a.m. to 2:19 p.m., before Kathy Miller, CSR in and for the State of Texas, reported by machine shorthand, at the law offices of Fulbright & Jaworski, 1301 McKinney, Suite 4400, Houston, Texas, pursuant to the Texas Rules of Civil Procedure and the provisions stated on the record or attached hereto.

A P P E A R A N C E S

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EXHIBITS

GERHARD K. RAABE, Dr.P.H., F.A.C.E.,
having been first duly sworn, testified as follows:

EXAMINATION

BY MR. LUBEL:

Q. Please state your full name.

A. Gerhard Karl Raabe.

Q. Where do you live?

A. 2215 Aquetong Road, New Hope, Pennsylvania, 18938.

Q. What is your profession?

A. I am an epidemiologist.

Q. Are you a medical doctor?

A. No, I'm not.

Q. Are you a toxicologist?

A. I have training in toxicology, but I don't hold myself out as an expert.

Q. All right. Are you an industrial hygienist?

A. Once again, I have training in industrial hygiene but don't hold myself out as an expert.

Q. Have you -- have you been retained in this case to offer opinions on either toxicology or industrial hygiene?

A. No.

Q. I take it that you understand I have had the chance recently to review the report that you issued, or

the affidavit, in this case, correct?

A. Yes.

Q. And as I appreciate it, what you have been asked to do in the Cowey case is to serve as an epidemiologist and to discuss causation with the jury; is that correct?

A. Yes.

Q. Do you agree with the diagnosis of Mr. Cowey of myelodysplastic syndrome?

A. I have no objective reason not to disagree.

Q. Okay. But as we sit here today, for the purposes of the opinions that you offer in this case, between now and trial, can we assume that you're going to take the position that, in fact, Mr. Cowey does have myelodysplastic syndrome?

A. Well, specifically, he has refractory cytopenia with multilineage dysplasia, which is a -- which is a myelodysplastic syndrome. One of many.

MR. HEARD: Lance, you need to clarify the question. He accidentally said he has no reason not to disagree.

MR. LUBEL: Okay.

THE WITNESS: I accept the diagnosis that -- that has been presented.

Q. (BY MR. LUBEL) All right. By his treating

physician?
A. Correct.
Q. So, today, when we talk about Mr. Cowey and myelodysplastic syndrome, is it -- is it okay that we refer to that as MDS?
A. I will at times qualify my answer based on specific forms of MDS.
Q. But -- but you do appreciate that he does have a form of MDS?
A. Yes.
Q. All right.
A. No question.
Q. What caused Mr. Cowey's MDS?
A. As far as I can view it at this stage, based on what I have seen, it's idiopathic. We don't know what caused it.
Q. What are the accepted causes of MDS?
A. Well, depending on specific forms, but generally speaking, would -- would be high, continuous exposure to benzene, elec -- ionizing radiation, and a variety of cytotropic drugs used in the treatment of various cancers.
Q. Are there any other causes, or at least recognized causes, of MDS other than the exposures to benzene, ionizing radiation, or drugs in the treatment

of cancer?

A. Well, this -- I'd say those are probably the -- the best established causes. Association with farming, pesticides, nonionizing radiation, all appear in literature with -- with varying degrees. MDS has only recently been categorized in a way that you'll start to see more studies coming forward. So, there's not a lot out there.

Q. Is it your position as an expert in this case that farming causes MDS?

A. No.

Q. Is it your position as an expert in this case that pesticide exposure causes MDS?

A. The data isn't sufficient to establish causation at this time.

Q. So, what you're saying is that one day in the future, pesticide exposures may in fact cause MDS or be recognized to cause MDS?

A. Well, there are not enough systematic studies on MDS, particularly subtypes of MDS, out there; so, you don't have a body of literature for which you can say this is showing up consistently.

Case series of MDS that have been used in some of these studies are not all defined in terms of their subtypes or their genotoxicity; and as a result, you're

going to get varying results depending on the mix of cases in the series that are on the study. So, more refined study is going to be needed before we're going to be able to pin down -- pin down to the causes -- potential environmental causes, as there are.

Q. But as you sit here today, you agree with Dr. Irons and Dr. Natelson that benzene exposures can cause MDS; is that true?

A. Certain forms, yes.

Q. What forms?

A. Well, I'm not sure if we understand that completely; but it's -- certainly -- the signature chromosomal aberrations of 5 and 7 are demonstrably there in -- in pretty much all forms of what we would consider secondary MDS, those -- in other words, MDS caused by some external factor.

Q. Can you tell us as you sit here what forms of MDS are caused by benzene exposures?

A. I don't think we know that very well. Certainly, if there are blasts -- blasts -- blasts present in the early stages, it's much more likely to progress to AML, which would suggest a closer association. And as I indicated, chromosomal aberrations in 5 and 7 -- in other sites as well but 5 and 7 most reproducibly.

Q. Can you write down for us on a piece of paper the forms of MDS that are not caused by benzene exposure?

A. No, I cannot.

Q. Can you write down for us on a piece of paper the forms of MDS that are caused by benzene exposure?

A. Well, I don't think we know enough about the various subtypes of MDS, other than what I described to you earlier, that provide a clear guidepost; so, the answer is no.

Q. And as you sit here today, you can't tell us what caused Mr. Cowey's MDS, correct?

A. Based on the data available, I don't see any known cause.

Q. As you sit here today, can you tell us the most likely causes of Mr. Cowey's MDS?

A. No.

Q. Do you understand the distinction I am drawing? I understand what you're saying is you can't tell us to a certainty what caused it. And I am trying to lower that threshold and say: Can you tell us, more likely than not, what you believe caused his MDS?

A. Well, I -- no, I can't, because it's -- the chromosomal aberrations are not suggestive of the secondary forms that are -- that are induced by the

known causes we discussed. The vast majority of MDS's are idiopathic.

Q. That's true with all cancer, isn't it?

A. Some more than others. I mean, some we have, you know, a lot -- lot more information on -- on causation.

Q. But there is a number of cancers for which we know of certain causes of it; but, by and large, most of what we know about it is idiopathic?

A. I think you would have to be very specific in which forms of cancer we're talking about.

Q. Well, like lung --

A. You don't --

Q. Talking about lung cancer.

A. You, you know, very rarely see a lung cancer in a nonsmoker or someone without enormous -- well, a nonsmoker for sure. Radionuclide exposures. I mean, it -- you don't see lung cancers frequently without some suggestion of an environmental cause out there somewhere. And by environmental, I mean life-style factor as well as occupational or ambient environmental.

Q. Can smoking cause lung cancer?

A. Yes.

Q. What type of cigarettes can cause lung cancer?

A. Pretty much any type of inhaled tobacco smoke

can cause lung cancer.

Q. What -- what is it about tobacco that causes lung cancer? What is in the tobacco?

A. Well, a number of materials that act as initiators, such as benzopyrene; polonium-210, which is an alpha emitter. There's a whole bunch of variety of tars. I mean, it's a -- you know, it's a list of, I don't know, 500,000 different materials, a significant number of which have been known to be initiators and a significant number of which have been known to be promoters.

Q. Now, does every cigarette have the same components?

A. No.

Q. And does it -- there's different makers of cigarettes. Different companies make them. You understand that?

A. I understand that.

Q. And my question is: Do each of those companies have the same components in their cigarettes?

A. I have no way of knowing. I mean, the answer is, they share a significant overlap on common components. Some cigarettes have additive materials, as well, that I am not really that familiar with.

Q. Do you believe that there is sufficient

evidence that all forms of cigarettes, made by whichever company, cause lung cancer?

A. Well, it's not as simple as that. I mean, there -- there is a significant element of dose in the -- in the mixture. I mean, you would theoretically, at least, if not practically, have to smoke way -- many more low tar cigarettes than -- than the older, unfiltered, high tar cigarettes. So, there is an element of dose in all of this. But intrinsically, the -- the intrinsic hazard components are probably there in all cigarette smoke.

Q. When you look at studies that -- that stand for the proposition that smoking can cause lung cancer, do the studies tell us which cigarette company's cigarettes they're studying?

A. I haven't looked at all the studies. Probably not. Most of the -- most of the time you see pack years, and that's it.

I don't know what you're looking for.

Q. Well, what I am trying to figure out is how scientists such as yourself make the leap that cigarette smoking for all brands, for all manufacturing brands, can cause lung cancer if the scientists don't know the differences between the brands, what -- what the components of the cigarettes are.

A. But cigarette smoke -- I mean, I -- I have not looked at this literature in a very long time; but cigarette smoke from a variety of different sources, filtered, unfiltered, different tobacco sources, which is generally known, all share intrin -- intrinsic carcinogenic materials of the same -- in the same class. Some more than others. Some less than others.

So -- and then, of course, when we're looking at long latency cancers like cigarette smoking, what you're talking about has not been as big of an issue as it might be going forward because the differences between the cigarettes was not that great in terms of trying to market ultra low and ultra lights. I mean, those are relatively recent events.

So, basically -- I mean, this -- what you're saying is not in -- what I think you're getting at, so I might as well just go into it for you, is when you have mixtures where you don't have a clear understanding of all the components, in an epidemiologic study sometimes you will get results that are unexpected, in the sense that you might -- and that does happen from time to time. You will see cigarette studies that don't show a clear dose response except for maybe at the very highest smoking edge. That could be for a number of reasons, but one of the reasons could be that the mix of pack --

individuals that make up that cohort and the pack years that are being calculated for each individual vary because some people smoked a particular brand of cigarette that may have been lower in tar than another brand.

So, studies -- that's why when -- when you study something called "organic solvents," you really can't establish any form of meaningful causation because all organic means, it's got a carbon atom in it. So, it's not very useful for specific causation. It's useful in establishing hypotheses for more refined studies.

Q. But is it a -- only a hypothesis that cigarette smoking causes lung cancer, or is it past that?

A. No, because -- way past that. I mean, we -- we -- there have been so many different studies done, all types of different ways, in different populations. So, you've got -- going back to what we use to -- to assess causation, Bradford Hill criteria: We have strong associations, statistical associations. We have many studies that show dose response, even though they were done in different fashions and different cohorts and different designs. We have consistency. We have pretty good specificity. I mean, cigarette smoking does

contribute to several other cancer sites, but it is very prominent in smoke -- in lung cancer.

So, you have a body of literature, well-established, demonstrating consistent relationships; and that's how you eventually come to a causation. And if you read the preamble to either the '64 Surgeon General report or the later one, which at the moment I'm blocking on the date, probably in the '80s somewhere, both of them sort of walk you through the literature in a sense of, "Here are all the -- array of all the studies here. There are statistical findings. Here are the dose-response relationships. Here is the level of consistency. Here is the level of specificity. Temporally, they all have, you know, latencies varying depending on dose but -- but reasonably consistent latency periods."

So, that's how you establish causation.

Q. You have to get to those criteria?

A. Yeah.

Q. And once you've met the Bradford Hill criteria in more than one competent, well-done study, then do you have causation?

A. Well, we're talking general causation, right?

Q. Right.

A. We -- yes, you would -- and when you say more

than one, that doesn't necessarily imply two; but some body of literature -- and, remember, that you also have -- and the Bradford Hill tends to be glossed over when we're talking about epidemiology, but consistent understanding of underlying biologic mechanism, concordance with studies done in animals. So, you have a body of literature where you have at least some notion of mode of action as well as the statistical and epidemiologic evidence to work from.

So, when you have gone through all -- all that and -- and the answer is still yes, this all hangs together, it makes sense, you have established general causation that this can, indeed, cause cancer in humans; but you're still on -- when you get to an individual, now you still have to talk about issues of dose and exposure.

Q. But the Bradford Hill criteria -- and I guess that's the point I am driving at -- they address general causation, not specific causation, correct?

A. Well, they were designed to assess a body of literature, yes.

Q. And a body of literature, by its very nature, includes more than one person?

A. Oh, yeah.

Q. It includes groups of people?

A. Sure.

Q. And that's your focus, is groups of people, as an epidemiologist?

A. Yeah. But, I mean, I'm also trained in risk assessment. I mean, my firm does risk assessments, as well; so, the body of epidem -- if you spoke in purely epidemiologic criteria, they're generally in groups; so, when you're looking at a specific causation, the issue becomes how well does this person line up against what you've learned from -- from -- from group -- from groups of similar people -- people with similar characteristics.

Q. But would you agree, Dr. Raabe, that the Bradford Hill criteria were designed to look at groups of people to determine whether an agent or substance was capable of causing a given outcome or disease?

A. Yes.

Q. And I take it that since you, like Dr. Irons and Dr. Natelson, do, in fact, believe that benzene exposures -- at least certain benzene exposures can cause MDS, that you-all do believe that, in fact, they have met the Bradford Hill criteria. Would that be an accurate statement, at least from your perspective?

A. Well, now you're talking generally MDS --

Q. Correct.

A. -- okay, not specific forms or specific --
Q. That's right.
A. -- chromosomal aberration groups?
Yes. Yeah. It's weak, though.
Well, actually, let me -- let me step back from that.
The -- we don't have -- I mean, we're really looking at causation, benzene and MDS, from an extrapolation of what we know about AML. There are no studies out there that demonstrate a dose response from benzene to MDS in any particular form.
Q. What about the Hayes and Yin 2000 study?
A. I discount trying to use the -- the Chinese studies in their -- in their current form because there are so many confounding issues on the way they -- the way they did the case identification. It's been heavily criticized. The cohort itself is really enormously heterogeneous in terms of industrial makeup. There is very little information on confounding. A lot of the leukemia cases in the earlier studies, in later studies become MDS cases.
I, and a bunch of others, including the U.S. E.P.A., both in -- in their large risk assessment update and in their most recent, I guess, 2000-2001 update in IRIS, say you can't use that study for dose response.

So, while it's sitting there, I don't -- and there is no question in -- because of the enormously high exposures to benzene in China that some of those MDS cases are related to benzene. I am not trying to discount what have already been found, but you can't use it for dose response. And that being the case, you know, I go back to my original statement. You don't -- you don't have dose response information for MDS.

So, in -- in the traditional sense -- I mean, I think -- when we say we believe that benzene can cause MDS, or at least certain -- under certain conditions, we're talking about more what we've learned from very large case series and what we know from -- from AML. So, in the purest sense, it hasn't met the Bradford Hill criteria.

Q. Why is it acceptable for scientists such as yourself to rely on the information that you have on AML to make the leap, if you will, that, in fact, benzene can cause MDS?

A. Well, there have been a number of -- let's put it this way -- enough case series -- you know, normally case series would not be acceptable to us as epidemiologists, but we -- we do have a large body of literature on AML, and we do have enough knowledge from case series that generally, for -- across all forms of

MDS, about 30 percent might progress to AML; and specifically, the -- the form that Mr. Cowey has, some percentage lower, 15, 16, 17 -- pick a number -- may progress to AML.

So, with -- with knowledge of the form and knowledge of the chromosomal aberrations that are also seen in AML, and the underlying biology, I think, you know, we're in a comfort zone where we don't have to have every piece of Bradford Hill checked off purely for MDS because, clearly, some MDS is on the road to AML. And if you go back in time, you know -- we all -- it's also consistent with what we know about AML in general, in that -- benzene-related AML in that in most cases for there to be increased risk for AML, you need a high enough exposure to induce some observable toxicity. We don't always observe it because you're not looking, but some type of bone marrow toxicity that -- that is a precondition.

So, I mean, if you just sort of make -- stretch the timelines out, some people, maybe, go right -- right through early bone marrow toxicity into AML; some go into an MDS phase, or preleukemic phase. You know, pick your historical jargon. I mean, that's been the problem with MDS, it just has not been well-defined as a disease up until, you know, into the

middle, late '90s, really.

So, you don't have a rich body of it by itself; but I think I am giving you a rationalization of why we're comfortable in -- on moving into the AML literature as the -- as the baseline.

Q. Do you remember the conclusion in the Hayes and Yin article of 2000 where they state that the -- the relative risk for people exposed at levels less than 10 parts per million was 3.1?

A. I remember that's -- that's one of -- one of the statements in the report, yes; but I -- as I mentioned earlier, I -- I and many others, including our U.S. Government, believe you can't rely on their dose -- on their dose-response information.

Q. Now, do you have with you today documentation that sets forth the criticisms of that article?

A. I didn't bring that because I actually keep that in a separate file; but if you have everything that was attached to my affidavit there, you will have -- you will have a paper by Travis, you'll have a paper -- I mean, I didn't cite all of that literature; but I cited some of it. You have got a paper by Wu. You have got a paper by Wong.

Q. Can we -- can I interrupt you real quick?

A. Sure.

Q. You -- your attorney handed me a stack of articles.

A. Yeah, I can --

Q. Can I just -- will you just pull out the ones that say that?

A. Sure. Certainly.

I did not cite in this report, and therefore wouldn't -- I didn't bring it with me, the E.P.A. documents; but here are three -- three references that deal with at least some of that criticism.

Q. What -- what is the E.P.A. document you're talking about? What's the title of them?

A. Okay. The title of them are -- actually, I might have the IRIS with me. We were talking about that with something else yesterday.

Q. Take your time.

A. Well, actually, if you go to the IRIS -- the cancer information retrieval system, it's the U.S. E.P.A. -- well, just look up IRIS, E.P.A. and IRIS, I-R-I-S. You will get their calculation -- you'll be sent to a web site that has their environmental risk assessments for ambient exposures out in the community for benzene. That -- the documentation of -- of -- in their -- the documentation of that number speaks to criticism about the Chinese studies.

The other document, I believe, is 1997; and it is the -- it's a rather thick document. I am sure it's on the web site for E.P.A. -- the Carcinogenic Risk Assessment Guideline document. It looks at multiple myeloma, leukemia, non-Hodgkin's lymphoma, and all the studies on benzene, and concludes, essentially, that leukemia is the site that we've got to look at, and makes conscious mention about all the Chinese studies and why they're currently unsuitable for -- for quantitative risk assessment.

Q. But I take it, since this 1997 E.P.A. document is dated before the -- the Hayes and Yin 2000 article, that it couldn't be --

A. No.

Q. -- be referring to that article in particular?

A. Well, that article was rereviewed in the 2001 IRIS document.

Q. So, specifically, when you talk about the -- a government agency, you're saying I need to go to the IRIS document?

A. Well, you're trying to pick out one specific study.

Q. Correct.

A. I mean, there's a whole long list of Chinese studies, starting with Yin in the late '80s, moving into

Hayes or Dosemeci. I mean, there is quite a long list of -- their -- coming out of that cohort. And the most recent -- if you're looking for something that criticizes them, the most recent one is after the -- the most recent Hayes paper, and that's in the IRIS.

But the -- the fundamental problem has been that there is not -- despite all these criticisms, and at sometime some attempt for rebuttal from some of the investigators, the underlying issues have never really been addressed. That's why there's a multimillion-dollar consortium in Shanghai now doing studies of benzene -- MDS, aplastic anemia, leukemia, and non-Hodgkin's lymphoma.

Q. I didn't -- I need to talk to you about that.

Tell me about this -- the Shanghai consortium.

A. There's a consortium of researchers associated with the Fudan University, the National Cancer Institute of China, the Regional -- I might not have all these terms -- names correct -- the Regional Cancer Registry for Shanghai, which is a huge population of some 23, 24 million; and they have set up a large laboratory, and they're identifying very early cases of myeloproliferative diseases, including MDS. They have got an extensive biology program and an extensive industrial hygiene and epidemiology program appended to

that that will follow a bunch of cases for the next five to ten years; and I think its current price tag is somewhere in the 25, 27 million, not counting the Chinese contributions.

Q. The current --

A. Budget.

Q. -- consortium?

A. Uh-huh.

Q. And you think that consortium was, in part, put together to look at low-level benzene exposure?

A. I -- that consortium was, in part, put together for two reasons: China is one of the few places where you will have a range of benzene exposures going from moderate levels, probably in the 10 ppm range, all the way up to still 50 and 100 ppm daily time-weighted averages, unfortunately. So, it's one of the last places you can really study benzene systematically. It's a place that has a -- a very rigorous catchment process, but there is -- in other words, they can identify cases over a large population very effectively in a relatively short time; so, you can get good samples, good -- you know, good, fresh bone marrow, all the things that the biologists want to look at.

The workplaces are still in existence, so you

can get direct measurements. You'll be able to study the course of these diseases much better than has ever been done; and since the -- since the study is essentially prospective in -- the exposure assessments are going to be much more reliable than they had been in the original Chinese cohort.

Q. Are you involved in that study at all?

A. I helped try and get funding for it while I was still working for Mobil Oil when I was still -- when I was chairing a large A.P.I. committee. I'm not involved with it currently.

Q. And were you successful in getting funding?

A. Yeah, but -- although the price tag has grown radically from --

Q. Who did you get funding from?

A. A number of major U.S. petrochemical companies. I believe there's some government funding in the mix. I mean, it's a --

Q. I am talking about you in particular. Who did you go to and get funding for that study?

A. Oh, at the time I was chairman of the Health and Products Stewardship Committee of the American Petroleum Institute; so, I was the senior committee chair on the health side, so I gave several presentations to what would be either vice presidents or

directors of health, environment and safety for member companies.

Q. To oil companies?

A. Yeah, that was my audience. There were other folks who got -- trying to get money from other groups.

Q. And did you do a PowerPoint presentation, or how did you present it?

A. Yeah, it was a PowerPoint presentation.

Q. Do you still have it?

A. Yeah, I think so.

Q. Do you have any problem giving us a copy of that?

MR. KEMP: I prefer you direct your request to me, Lance; and I'll get back to you on that.

MR. LUBEL: I'm just asking him if he has a problem. I understand that you can make objections.

Q. (BY MR. LUBEL) I'm just asking you if you have a problem letting us see it.

A. As far as I understand, I'm under no confidentiality restriction on the presentation.

Q. And as I understand -- and correct me if I am wrong -- you were making presentations to members of the American Petroleum Institute, such as major oil companies, concerning why you -- the group needed to fund -- or help fund the studies over in China?

A. Right.

Q. And this is work that Dr. Irons is currently working on, I take it?

A. Yes, he manages the -- the diagnostic laboratory for them, and obviously is an active researcher in molecular biology of myeloproliferative diseases.

Q. That's the hardest words to say in this area.

What was Dr. Irons' involvement, if any, in the fundraising?

A. Well, it was a very long process. I don't think he had any direct -- well, he was -- he was asked -- there was advisory committees put together, and I don't remember who all was on them, but he was certainly one of -- one of the people asked to come up with research proposals. At that time they were skeleton proposals of the types of research questions that could be answered by such -- such an exercise.

And I don't know -- you would have to ask Dr. Irons whether, you know, he was filing grant applications simultaneously with that. I don't know. But -- but he wasn't part of the fundraising group. He was -- he was more one of the potential investigators.

Q. Would you take information from Dr. Irons and put it into your presentation?

A. No, I don't think I did that.
Q. Were there --
A. I mean, maybe in a general sense, the type -- you know...
Q. And what was your primary pitch, if you will, or presentation, as to why the major oil companies should participate in the studies in a funding sense?
A. Well, we -- I don't know what my major pitch was -- I mean, we're talking -- we're going back to '97, '98, and '99; so, I -- I don't even remember what's all in the PowerPoint presentation.

I mean, clearly, we were concerned that the investigations all based on the original Yin cohort would forever be tainted. In other words, there was -- because they lost most of the cohort. I mean, there's a very complex history on that -- on that cohort. The National Cancer Institute, Hayes, and the rest of them really don't know who the people are in that cohort because they were assembled in a very ad hoc way. Some of the materials they have well laid out, and some of the materials they have very -- very poor documentation for.

So, recognizing that over time we needed -- we needed a -- more study specifically focused on non-Hodgkin's lymphoma, which was one of my concerns,

personal concerns, I wanted to see more data, because I didn't feel comfortable that what was coming out of the China studies made sense in the rest of the body of literature. Folks like you and others seem to still periodically think multiple myeloma is related to benzene, despite all the evidence; and that the only thing we really had that's really good for dose-response analysis is the Pliofilm cohort, and even there we have three different, if you will, proposals -- published versions of what their exposures were. So, even there you get a range of dose response. They're all quite high.

So, there seemed, to me, to be value in -- in doing additional epidemiology and molecular biology using more current techniques than we had available in the '60s and '70s and '80s to help better specify dose-response issues, general causation issues. So, I mean, I think that's -- that was sort of my motivation.

Q. But I don't understand what the benefit to the oil companies would be. How did you convince them to help fund the studies?

A. Well, you may have noticed, but indirectly there are subparties in -- in litigation. There are parties in regulation. Every time somebody regulates benzene, it has an impact on gasoline production. It

has an impact on -- on a whole range of their activities. So, it's in their interest to get it right.

In other words, if you -- you know, if -- you know, in a very hypothetical sense, you know, does it make sense to keep benzene -- benzene levels in gasoline at 1 percent, which is where they are now in this country? They're not that everywhere around the world. It costs money to control the benzene content to those levels. You know, is that money well spent? Are there better things to do with the -- with the resources?

So, I mean, you know, there is a whole bunch of business reasons that getting a good scientific answer as to what -- what are the risks at low levels, what are the specific -- you know -- I mean, while we're convinced that AML is -- is the primary or only real hazard associated with high levels of benzene exposure, other than aplastic anemia, of course, that doesn't mean everybody else is. So, walking away from the science doesn't make a lot of sense.

Q. But what was it about the Chinese studies that kind of initiated the need, if you will, to go push for additional studies, get funding from oil companies and other parties? What conclusions, if you will, out of the Chinese studies generated the -- the energy for people to say, "Hey, we need to go fund some other

studies and find" --

A. Well, the one that bothered me was the suggestion that benzene had -- was related to non-Hodgkin's lymphoma. I mean, if there was a single driving force, that had never shown a -- you know, been demonstrated anywhere else. So, is there something about the Chinese genetics? Is there something about -- remember, part of the problem in the Chinese cohort, which I alluded to earlier, is the fact that, you know, we're not talking urban dwellers.

We're talking about a significant part of that cohort cooks their food with -- with brown coal in closed environments. They have subsistence farming and agriculture. Their pesticide exposures are dramatically different than ours. Their -- none of -- their benzene exposures are normally different from what -- probably what we ever had in this country, unless you go before 1950 when benzene wasn't really presumed to be all that toxic.

So, there are many, many factors in that -- that are not part of the Chinese cohort. The Chinese cohort was essentially, "Let's look at benzene." Dr. Yin, who I have worked with and been on committees with over the years, was trying desperately to convince the government that 50 ppm, which wasn't even a -- like

an OSHA regulation, it was the recommended guideline, so you didn't even have to actually follow it that well, was trying to convince the federal government that 50 was too high.

He would have been enormously comfortable with 10. But what ended up happening is as he was losing funding, N.C.I. was looking for more international partners; so, they took his original cohort, with its many, many limitations, and, you know, frankly ran with it. So, that's problematic to the body of science that we have around the world from other places. So, we need to understand it better.

Q. What was driving Dr. Yin, in your opinion?

A. Well, I think I just told you. I mean, you know, he and I were on an international program panel for -- international program for chemical safety panel. We wrote a monograph on benzene. And, you know, we're discussing in the monograph meeting, you know, recognizing that it's a worldwide recommendation, so you weren't, you know, trying to push the lowest technical levels but what was -- what did the science say seemed to be safe. So, we were sitting there debating, you know, is 1 too low? Is 5 okay? Should it be 3? Should it be 10?

But, you know, as you got down to the last

chapter in the monograph, and -- and poor Dr. Yin is trying to say, you know -- "You know, we have got folks well exposed over 50s." I mean, his motivation was, we knew 50 was wrong. We -- that you shouldn't be exposing people at those levels. I mean, your aplastic anemias -- they have a lot of cases of aplastic anemia in China.

Q. But, I mean, was there an external source, if you will, that you felt as though was driving Dr. Yin to -- to try and reach these conclusions, or did you feel like he honestly and genuinely thought this is where things ought to be?

A. Well, he was, you know, a physician/epidemiologist in China that had seen lots of aplastic anemia, looked at where these guys worked, recognized that they were almost -- you know, that it almost certainly was benzene related, initiated a very large cohort with internal funding, doing the best job you could in the period for him, which was way below our standards of quality control, to help demonstrate that. So, I mean, you have to -- you know, his English is not very good. So, you know -- but --

Q. What I --

A. -- but I think that's what his motivation --

Q. Was he biased, in your opinion?

I am not talking about from a scientific standpoint.

A. No, I -- well, I mean, biased only in the sense that going into the study he knew that they were exposed too high. Okay? But I -- I -- there is nothing that I -- there is nothing in his studies or demeanor that strikes me that, you know, the data was manipulated to show responses where there weren't any. I think it was just sort of by our standards, you know, moderate, inadequate quality work. He relied on the diagnoses that were coming out of these little regional clinics. Many of the diagnoses were wrong.

Q. Didn't the 2000 study that he and Dr. Hayes and others did, didn't they go back and look at that data again?

A. Well, they -- they redid a big chunk of the hematology that they could. They couldn't do it all. So, yeah, I mean, I am not saying improvements haven't been made where they can be made; but the -- but remember -- I remind you, again, they had no identification on these individuals. So, they could not go back and -- and really do good audits on exposure. They couldn't go get additional materials. I mean, if they had the materials -- they didn't -- if Yin had the materials, they had it. If he didn't have the

materials, they didn't. And that was sort of the end of it.

And that's why the numbers changed so dramatically too, you know, which that's not -- you know, if a study was done by me and -- and every time I reported out, the numbers changed dramatically, I'd be sitting in very long depositions when people wanted to use my study in their case.

So, you know, there's lots of problems that I don't think were, you know, intentional that are just -- you know, say that you need another Chinese series of studies using more modern techniques to help define, you know, what -- what was really going on.

MR. LUBEL: Do you mind if we take a short break?

THE WITNESS: Fine with me.

MR. LUBEL: Is that okay with you guys?

MR. KEMP: Yes.

MR. LUBEL: Thank you.

(A break was taken, 9:43 to 9:49 a.m.)

(Exhibits 1 and 2 marked.)

Q. (BY MR. LUBEL) Can you identify Exhibit Number 1?

A. Exhibit Number 1 is a paper authored by Dr. Otto Wong, published in 1999, titled "A critique of

exposure assessment in the epidemiologic study of benzene-exposed workers in China conducted by the Chinese Academy of Preventive Medicine and the US National Cancer Institute."

The second paper is also a paper by Dr. Wong, titled "Benzene and Dose-related Incidence of Hematopoietic [sic] Neoplasms in China."

And the third paper is by Weiai Wu, W-U, "Occupational Cancer Epidemiology in the People's Republic of China."

And these are just a sampling of -- from me.

MR. KEMP: Are these all Exhibit 1?

MR. LUBEL: Yes.

MR. KEMP: All three papers?

MR. LUBEL: Yeah.

Q. (BY MR. LUBEL) And what I asked you to do was to take out, out of the articles that your lawyer handed me, the ones that you felt criticized the Chinese studies that we have been talking about today, correct, and that's what you did?

A. Yeah, of the set -- I mean, I have additional ones; but of the ones I brought with me, that's -- those are the ones that I think relate to your question.

Q. And then you told us about an E.P.A. document that you said we could get on IRIS, I R I S? Is that

correct?

A. Yes.

Q. Can you put your hands on that and fax that to your attorneys?

A. Yes.

MR. LUBEL: Jeff, will you provide that to me?

MR. KEMP: Absolutely.

Q. (BY MR. LUBEL) I don't know that I'm computer intelligent enough to find it.

A. Would you like the earlier one, as well?

Q. Yes, sir, please.

And then, Exhibit Number 2 is a list of the -- is actually the articles that are referenced in your affidavit in this case, except for the three articles that you pulled out and marked as Exhibit Number 1; is that correct?

A. Umm --

Q. Sir?

A. -- this really should be part of Exhibit 1.

Q. Okay. What is it? And we will attach it.

A. It's an article titled "Hematopoietic Malignancies and Related Disorders Among Benzene-exposed Workers in China" by Lois Travis; and that really -- we are placing that in with Exhibit 1.

But the remainder are -- are the studies I attached to and used specifically for my affidavit.

Q. One of the authors in these studies is a Dr. Wong. Do you know him?

A. Yes, I do.

Q. And how do you know Dr. Wong?

A. We've worked together over the years and co-authored papers together over the years.

Q. Are you generally familiar with his involvement with the American Petroleum Institute and Chemical Manufacturers Association?

A. Yes.

Q. What do you know about it?

A. Well, it was -- he was contracted to conduct an epidemiologic study by the then-called Chemical Manufacturers Association. Actually, I believe he was working with -- started out working with Dr. Irving Kaybashore in that process. That's where I first met him. Mobil contributed data to those studies.

Over the years, he has conducted several very large cohort mortality studies at the behest of the American Petroleum Institute. Over the years, he and I have independently worked on projects together that I had an interest in, or specifically I and Mobil had an interest in; and we co-authored several papers together.

Q. And were you aware that oil companies, including Mobil, have hired Dr. Wong to testify in benzene exposure cases just like Mr. Cowey's?

A. I don't know that -- I mean, I know he's been hired to give testimony. I don't know specifics or whether any of them were anything like Mr. Cowey.

Q. Were you aware that Dr. Wong had been hired by Mobil, and other oil companies like Mobil, on benzene exposure cases?

A. I don't understand the question.

Q. Were you aware that Mr. Wong had been hired as an expert by Mobil in benzene exposure cases?

A. You mean toxic tort cases?

Q. Correct.

A. I think I am -- well, I am not aware that he was specifically hired by Mobil, but I am aware that he has testified for oil companies in such cases. I -- I never -- I never hired him; and so, I don't -- I don't know whether he was ever used -- retained specifically by Mobil in any of these litigation cases. I don't know that.

Q. Now, who did you hire when you were at Mobil to do this expert witness work?

A. I didn't. That was -- that would have been the law department. My -- my group is only scientific

in the medical department.

Q. Did you participate in helping the law department find experts to work on these cases?

A. No, I don't believe so. I don't recall doing that.

Q. Nobody ever asked you, as a scientist with Mobil Oil Company, what experts they should hire to defend these toxic tort cases?

A. I honestly don't recall participating in those types of discussions. I mean, certainly they knew of Dr. Wong and my association with Dr. Wrong; so, I am not saying that we hadn't discussed Dr. Wong. But I don't recall sitting down with the -- any of our environmental counsel and giving them a list of -- a list of potential witnesses. I -- I know I probably sat down with them in the course of various settings and listed epidemiologists that we had worked with that I felt were, you know, professionally very good; but I don't think it was in the context specific to the kind of question you're asking.

Q. Well, what context would it have been in?

A. Well, if -- if we were proposing -- well, environmental counsel would have probably sat in on large decision points where I was going to go ahead and do a study or contract a study, one or the other, and

either wanted -- was letting them know who we would bid it to or letting them know who we were considering for putting on an advisory panel, a reviewer panel; so, they would have heard those discussions.

Q. Why would the lawyers be involved in that?

A. Well, I don't know if it -- there was only one lawyer involved in that. He was environmental lawyer for the corporation, and he sat in on all executive briefings; so...

Q. Was he a scientist?

A. Yeah. I am trying to get money from management; so, I have got to explain to management. I mean, it wasn't a scientific meeting. By the time I -- you know, we -- we decided in the medical department what we wanted to do. Now -- now I would go to -- to the level of the organization that would free up a couple hundred thousand dollars so I could go ahead and do it; so, that would be general managers or vice presidents, depending on how -- how many hundred thousand dollars I needed.

Q. What I am trying to figure out is what role a lawyer would play in this whole process of you trying to hire people to perform studies.

A. I don't think -- I don't know if he played any role other than part of the information -- I mean, he

was not part of the decision process, if that's what you're getting at.

Q. Now, what studies have you worked at Mobil that you participated in?

A. Oh, many.

Q. Well, let's talk about benzene. What studies on benzene?

A. Benzene or benzene related?

Q. Benzene related.

A. Okay. I contracted initially in the early -- late '70s, early '80s for -- to reconstruct the employment cohort for -- and subsequently conducted mortality analysis for the Beaumont refinery, the Torrance refinery, the Paulsboro refinery, which were owned by Mobil at the time.

Q. Who asked you to do that?

A. I -- it was my recommendation that we do that because we had a P.M.R. study from Terry Thomas that suggested that there might be some excesses and that the best way to understand that was not with a P.M.R. study but with a cohort study.

Q. Who is Terry Thomas?

A. Terry Thomas was a -- at the time a master's level epidemiologist working for the National Cancer Institute that was working with partial union data. So,

we initiated those studies.

A few years later, after those studies were completed, we began -- initiated the process. By that time I'd built an in-house resource so we could update those studies internally; so, we went through a process and updated those studies.

As we gained more knowledge about the specificity of -- in the early days, it wasn't clear whether we were just -- most of the epi studies tended to just look at the large category of leukemia. As biologies got better understood, it became more important to look at cell types; so, we further updated those studies and specifically looked at cell type.

It was -- I determined that we didn't have enough cases inside Mobil and that it would be useful to try and pull results from a number of different cohorts, and I'd say Otto Wong and I worked together on it and published a paper on cell type specific leukemia in the petroleum industry.

Q. What did you call the -- how do we refer to the late '70s, early '80s study where you included the Beaumont refinery and others? What's -- what phrase can we use to refer to that?

A. Initial cohort mortality study.

Q. And who did you hire to work on that study

outside of Mobil?

A. Robert Morgan, Phil Enterline, Otto Wong. The Stanford Research Institute did a lot of the data collect -- assembled a lot of the data for us in Beaumont.

Q. But you weren't just looking at the Beaumont refinery, correct, you were looking at the other Mobil refineries?

A. The three largest.

Q. And how did you select those, other than size?

A. Well, the Joliet refinery was recently built; and I -- it was designed to be a cross-crafting refinery, so it would not have been -- well -- so, all the work population was relatively young, hired in the midwest, and were all cross-crafted in the sense that maintenance workers did not just single-job things. And at that period we were trying to get a sense of if there are any specific crafts or specific work groups that -- which might show an excess of disease.

So -- so, we went to the ones that were the oldest, had -- and we had done site visits and established that had record -- paper records that were adequate to reconstruct into the late '40s, starting with post-war years '46 and '47. Torrance, we could only go back to the -- to '60. So, it was really a --

part feasibility, part size.

Q. But there were other Mobil plants at the time that you decided to run these studies that had benzene in them, correct?

A. Well, we had a very small refinery in the State of Washington, and we had the refinery in Joliet; and I think we still had but were in the process of -- I don't remember if we commissioned it or sold it or what, a refinery in Augusta -- it wasn't Georgia -- Augusta -- what -- I forgot what state it's in, but it's not -- it's not Georgia. It was another Augusta.

And that was it. We -- those were the total -- sum and substance of refineries in the United States.

Q. Did they have benzene at those other refineries?

A. No. The -- the only refinery that manufactured -- well, yes. I mean, obviously you have benzene in a lot of refinery streams everywhere; but only -- in that time period, the only refinery that had a petrochemical operation specific to benzene was in Beaumont. In other words, manufactured benzene was in Beaumont.

Q. Are you familiar with the -- the stream dripolene? You ever heard of dripolene?

A. I have heard it. I honestly don't remember what it means -- what it stands for right now.

Q. I mean, you understand in some of these refineries and chemical plants that there are products that are not -- that have -- that have benzene as a component of them?

A. Oh, certainly. Sure.

Q. And wouldn't those be relevant to study if you have got plants that have benzene as a component of a product?

A. See, you asked me what studies had benzene relationships. We didn't study those plants because of benzene.

I mean, we -- we're in an era where our concern was what -- it was basic descriptive epidemiology. This is the plant. These are the -- what -- what they manufacture, and these are the populations. Are there any unusual patterns of disease?

I mean, we've had a medical department since the '50s; but, you know, there -- there was nothing coming out of clinical observations to suggest that there was any specific problems. So, this was a more traditional cohort mortality study looking for patterns of disease that we would then subsequently study in more detail, as warranted. So, benzene was not, you know,

particularly --

Q. Wasn't the focus?

A. No. It was recognized that it was at these plants, but it -- it wasn't -- it wasn't a benzene study.

Q. And Mr. Morgan, Mr. Wong, Mr. Enterline, and the Stanford research group are the people that were involved in the study that were not employed by Mobil?

A. Well, they had staffs -- additional staffs. I mean Bob -- Bob Morgan actually brought Otto Wong in. Bob was principal investigator at Beaumont. Phillip Enterline was principal investigator in Torrance. And -- and Morgan was also principal investigator in Paulsboro.

Q. Now, did you know these men before you hired them to conduct the study?

A. I had met Bob Morgan. He was brought in as an epidemiologic consultant at some meeting or another of the American Petroleum Institute. That's where I first met him.

Q. The A.P.I.?

A. Uh-huh.

Q. Is that a yes?

A. Yes. I'm sorry.

Q. And Mr. Morgan brought Wong into these

studies, correct?

A. Yes.

Q. And how did Mr. Enterline get involved in them?

A. Phil Enterline is a -- an internationally known epidemiologist, as well. He was at University of Pittsburgh. I knew him. He had familiarity with the petroleum industry. And I don't actually remember where I first met him.

And so -- I mean...

Q. Why do you say he had familiarity with the petroleum industry?

A. Phil had done -- by that time, published a number of studies already on some petroleum-related exposures and had done some work on epichlorohydrin and a few other chemicals. I mean, he was an occupational epidemiologist of -- of high regard.

Q. Had he done work with the A.P.I. or the Chemical Manufacturing Association in the past?

A. I think he may have done some work for Shell, specifically; but I -- but to my knowledge, at that time he had not done any work for A.P.I.

Q. Now, did you -- did you enter into a contract with these men to perform this study?

A. Yeah, that's one of the reasons why the lawyer

sits in --

Q. All right.

A. -- because at the end of the day, I have got to write a conclusion -- write a contract.

Q. So, you and the lawyer for Mobil wrote the contract?

A. Well, they gave me the boilerplate version. I added a section on -- the boilerplate contracts didn't address publications and things like that; and so, I made some modifications to them and -- and sent them in to the contractor.

Q. And was there more than one contract for these men for this study?

A. Separate contracts for each refinery.

Q. Why is that?

A. Well, under different profit centers, the accounting is done that way.

Q. But was the form of the contract itself the same?

A. I think so. Yes.

Q. And did -- did Mobil retain editorial rights over the study?

A. No. We had a three-month window of review, and we could offer -- offer comments. And then the -- the investigator, I think, had one month to issue a

final report; and then he could do anything he wanted with it.

Q. Who was the investigator?

A. Well, Dr. Morgan for -- the ones we just discussed, the principal investigators.

Q. Was there a confidentiality clause or agreement regarding this study?

A. Well, there's -- there was a medical -- a medical confidentiality clause. They couldn't just -- could not disclose identity of any of the individuals in the study. I mean, medical -- it's sort of a standard medical confidentiality. And they could not discuss results of the study outside -- outside their organization or with -- until such time as the time report was issued; so --

Q. In other words --

A. -- no.

Q. -- what would happen if -- if Mobil didn't agree with the final report?

A. That's happened in other settings. Nothing happens. I mean, we offer our comments. They take them; they don't take them. The report is the report. And --

Q. Were the investigators free to issue a report at their discretion, irrespective of Mobil comments?

A. Yes. Oh, absolutely.

Q. And that would be contained in the agreement, correct?

A. Yes. It was very explicitly contained in the agreement.

Q. And I take it that you're -- the comments that Mobil had were put in writing?

A. You know, I don't remember. They may not have been. We may have -- may have been just invited to come in, and we sat down and sort of walked through -- walked through it.

Q. Now, did you have anybody look at the draft report done by the investigator before you met with the investigators to discuss your comments?

A. Well, it would have been myself; I think by that time I had a master's level person I had hired; and the -- probably the domestic medical director and the medical director probably would have read it. I don't recall at the -- at the draft stage whether any counsel had read it. They may have, but I don't remember. I'm not sure if they would have been normally in the loop.

Q. Did -- did you know from the outset that the results of the study would be published?

A. I assumed so. We weren't paying the contractors extra money to write a publication version

of the report. They were only being paid -- compensated to write the -- you know, the thicker internal -- if you're familiar with epidemi -- epidemiology, there is a tendency to have, in an unpublished version, more tables, more information than you could ever get published in a scientific journal. So, our contracts only asked for the -- and those early contracts -- a -- a report.

Q. What do you call that? What's the difference between the short, published report, and the -- the report that's got all the tables and raw data referenced in it?

A. I -- I don't think we have any jargon for that. I will say, in later years, when Otto and I worked together, we started going directly to a publication version of the report because it just added a lot of unnecessary time to, you know, have a -- one report and then another report.

But I think the term was report -- well, the jargon would be issue a written report versus issue a written report suitable for publication.

Q. All right. Now, I take it that in your field of epidemiology, you prefer to see the long report that's got all the tables and data, as opposed to the -- the published report that's got less information?

A. Well, certainly in that era where -- where to a large extent you're just trying to look for patterns; so, you would have more tables in some of the unpublished reports than you would see in -- in...

After we've updated the cohort several times, there was even -- not even a reason to rerun some of those tables over and over again because, you know, you knew they weren't going to provide any additional information; so...

Q. Now, did the workers at the plant sign anything to be a part of the study?

A. In that time frame, we notified the union that -- that we were doing the exercise; but since no -- anything coming out of the medical records, they would have already signed such a release. The Mobil release in that time period in the medical department said that you -- the results of the -- the examinations could be used for statistical reasons, statistical purposes. But in those studies there wasn't any -- I think we looked for Death Certificates in the medical records, but at that time there was no medical data being used in those records. So, the basic answer is no, they were not asked.

Q. How did you notify the Mobil workers that they were being studied at the various plants?

A. You said plaintiffs?
Q. Plants.
A. Oh.
Q. Sorry.
A. I -- how did we do that? I think there was -- there's a -- back then there was a bulletin board. I mean, the -- only the active workers would have known that -- that we were doing a study. The -- there wasn't -- I don't think there was any separate correspondence going out to retirees, and there certainly would not be to terminees because we wouldn't have -- we wouldn't have --
Q. Were you studying the retirees or the current workers?
A. We were studying anyone who worked a year or more from essentially 1946 to -- I forget the cutoff date right now, '80-something -- no, '70-something -- '79, probably, worked a year or more at the Beaumont refinery. So, that would include active workers, terminated workers, and retirees.
Q. What motivated the study? What initiated it?
A. Well, we were looking -- as I said, we were looking for patterns -- specifically cancer mortality, looking for patterns of potential for excess cancer mortality of the various cancer sites.

Q. Right. But, I mean, what --

A. You needed -- you needed the terminees and you needed the retirees because you -- cancer is a long-term process. So, to get a large population, you would want a large -- as large a population as you could establish; and you would want to have long -- you know, be able to look at duration of employment, and that included long-term workers.

Q. I guess my question is: Why in the late '70s did Mobil become interested in studying cancer and whether there was any pattern of cancer in the Beaumont refinery?

A. Well, let me just -- Mobil did a -- a study -- the medical department did an epi study on -- on their own in 1964 across the entire population of -- of Mobil. Now, that tended to be just active and retirees because it was -- everything was administered through one insurance plan.

Mobil's had a medical department since 1950. They've always been very interested in -- in the patterns of disease in the population, making sure that workers weren't harming themselves.

Q. Was the '64 Mobil study published?

A. No. That was in an era where, you know, public -- publication of things wasn't on any of these

radar.

Q. Have you seen the '64 study?

A. Yeah, it's been surrendered in some cases over the years. Actually, fairly recently only. I actually did not come across that report until I was cleaning out files in the Mobil-to-Exxon transition. So, I probably saw it when I first was hired and forgot about it because, I mean, by the -- by the standards of -- it doesn't show specific groups except in very broad classes; so, you know, office personnel, executive personnel, distribution personnel. So, it wasn't as informative as --

Q. And when was that surrendered in litigation, to your knowledge?

A. Oh, probably sometime this year somewhere.

Q. Do you remember who that was surrendered to? Was that Provost and Umphrey or Herschel Hobson or -- do you know these names I'm talking about?

A. Yeah, I know the names. I knew Herschel when he was a real -- a real professional.

Q. I'll tell him you said that.

A. You certainly can. Herschel knows me well.

It -- it may have been a Provost and Umphrey case. It wasn't a Herschel case, I'm pretty sure; but I don't remember.

Q. But did you have that data from the 1964 Mobil study to review, as you were, you know, an epidemiologist for Mobil?

A. Well, as I say, I'm pretty -- pretty sure -- when I found it, I recognized that I had seen it. Okay? But -- and -- and, of course, realized that -- you know, that it had some important import in today's -- today's world; but when I -- rereading it over again, I -- it reminded me why I probably forgot that I -- that it ever existed, because it -- it doesn't inform on any of the issues that -- I mean, it talks -- it -- it talks about the issues of cardiovascular disease and encourages Mobil to -- the medical department to continue its efforts for early diagnosis and treatment of those kinds of things. It talks about cancer, loosely talks about smoking, but didn't find any unusual distributions or patterns; so -- and it didn't look -- it didn't -- other than, I think, lung cancer, there was no other site-specific cancers. I mean, it was either all cancer, lung cancer, cardiovascular disease, accidents. I think that was the -- sort of the -- the focus.

Q. Do you know if Dr. Morgan and the group that performed the -- this initial cohort mortality study, whether they had access to the 1964 Mobil study?

A. No, they probably never saw it. They probably

never saw it.

Q. Where is the raw data from the '64 Mobil study?

A. I --

Q. The long report, as you call it, that's got the tables and all of that stuff.

A. Well, I mean, I have the whole report. Okay? I mean, that's what we're talking about. When you say the data, I don't -- I mean, I -- that's an era of mag tapes and IBM cards. I am sure it doesn't exist.

Q. Other than the -- this late '70s, the early '80s Beaumont refinery study that I think you have called the initial cohort mortality study, and this 1964 study that you just said was surrendered recently, what other benzene-related studies has Mobil either conducted or asked others to conduct on their behalf?

A. Well, after some toxicologic information on unleaded gasoline, there was a concern about kidney cancer specifically and exposure to gasoline; and -- and we conducted -- no individual company had enough distribution workers to do it, and -- on their own; so we, through A.P.I., conducted a very large study of the distribution of gasoline in the United States and the marine component of the distribution of gasoline. So, it's terminals, tankers, and -- and marine barging.

That study was conducted in the late '80s.

Q. Who conducted that for Mobil?

A. A number of investigators. Tom Smith, who is now at Harvard, handled the exposure component. Environmental Health Associates and Dr. Wong handled the -- the statistical and the report, the basic epidemiology reporting. We had Phil Enterline, Wegman, and -- and -- and -- I am blocking out his name -- a very famous industrial hygienist, I think from Harvard -- on a very active oversight panel that met several times a year.

And I guess that's the key players.

Q. Was that study published?

A. Yes.

Q. What's it called?

A. It's public -- there is quite a few publications out on it. There's an Environmental Health Perspectives monograph. I can actually tell you the publication date because I have a paper in it. It's peer-reviewed, but it's one whole issue. The 1996 Environmental Health Perspectives monograph, Volume 104, Supplement 6. It is entirely devoted to gasoline.

And then there are two subsequent publications by Dr. Wong in later years. I don't remember the dates. But if you do a search on unleaded gasoline and

epidemiology, you'll find them.

Q. Did --

A. And then --

Q. Oh, I'm sorry, go ahead. I didn't mean to interrupt you.

A. Well, you -- I'm back to your original question.

Additionally, in the late '90s -- well, in the '80s, I had been tagged to represent the U.S. at a World Health Organization IARC monograph on exposures in petroleum refining and gasoline; and, with Otto's assistance, we conducted a pooled analysis, sometimes called a meta analysis, of the major cancer sites coming out of not just our studies but the whole published literature. That was -- we published that.

And then in the '90s, Otto and I published a series of papers on multiple myeloma, non-Hodgkin's lymphoma, and solid tumors in petroleum-exposed groups.

Q. Were you doing that on Mobil's behalf?

A. Mobil let me use my time, and -- and I raised some money from A.P.I. to pay -- to pay Otto for his time.

Q. But I take it that you were still an employee of Mobil at the time those studies were --

A. Yeah.

Q. -- done?
A. Yes, that's correct.
And then we also updated the refineries one more time and published all of those.
So, all of this -- at least all the ones that my names are on is on the back of my C.V.; so...
Q. At any time have you published a study that relates to benzene where you were not employed in some capacity by a refinery or chemical company?
A. No, I didn't -- when I was with the State of New York, I didn't -- we didn't to that much occupational, since we were focused on New York City.
No.
Q. Do you know John Spencer?
A. Yes.
Q. Did he do any work for you when he was at -- when you were at Mobil?
A. You know, I actually don't know. He did not for me specifically, and my group was epidemiology and health risk assessment.
Whether he did any contract work for other parts of Mobil, I don't know.
Q. As you sit here today, you don't ever recall hiring John Spencer to do work on behalf of Mobil. Is that true?

A. Well, I know I never -- I know I never did.
Q. Do you know if John Spencer has ever been hired on any of the studies that you have done on -- related to benzene?
A. I -- I don't think so, but I -- but, you know, I -- I wouldn't know all the subcontractors that may have been used by some of these other investigators. So, I honestly -- I guess my better -- the best answer is I don't know, but I don't think so.
Q. How did you first meet?
A. I think when I first met John Spencer was when I had been asked to look at -- conduct some health risk assessment work for a silica-exposed foundry plant. That's where I first met John.
Q. For silica exposures?
A. Silica, and I think they had -- also had some asbestos found.
Q. Do you remember which plant?
A. Yeah.
Q. Was it Tyler Pipe?
A. Yes.
Q. It's a real shocker, isn't it?
A. It's interesting.
Q. Were you hired on behalf of Tyler Pipe or some product manufacturer?

A. Some product manufacturer.
Q. And were you -- was your role having something to do with cancer?
A. Yes.
Q. And did you understand Mr. -- Mr. Spencer's role to have something to do with exposure?
A. Yes. I had seen his name previously on -- on -- on exposure or assessment reports. I sort of knew him, but when -- you asked me when I first met him.
Q. Do you recall who you were working for in the Tyler Pipe case, the foundry case?
A. No, I don't remember the law firm or the -- or who they were specifically retained for.
Q. Was that just one case that you worked on?
A. Yeah, I worked on a gastrointestinal cancer case.
Q. What other cases or involvement have you had with Mr. Spencer in his role as an industrial hygienist, other than that one litigation case we have talked about?
A. I mean, he -- I read his reports in some other litigation cases; and I have spoken with him on his exposure reconstruction and the exposure monitoring he did on Liquid Wrench.
Q. You talked to him?

A. Yes.
Q. What -- go ahead.
A. But I don't -- I mean, I -- I have a local industrial hygienist that I use when I -- when I need somebody to help me out in -- you know, on a project.
Q. Who is that?
A. Wayne Skopypec.
Q. When did you talk to Mr. Spencer about this particular case?
A. Saturday? Saturday, I think.
Q. Which Saturday?
A. This --
Q. Any Saturday?
A. Any Saturday. Last Saturday.
Q. And who set that call up?
A. I think it was Saturday. It may have been Sunday, but I think it's Saturday.
I did. I initiated the call.
Q. So, it's been a couple of days ago, essentially?
A. Yeah.
Q. And why did you call Mr. Spencer?
A. Well, two reasons: one is, after reading Vernon Rose's report, I thought he made a misstatement, flat out incorrect, on the issue of what would happen if

you spiked benzene into an over-the-counter product like a current version of Liquid Wrench; and so, I wanted to review that with him. As I said, I'm trained; but I -- I always like to check my opinions.

And, also, I have been aware previously, although never seen the work product, that he had been doing some exposure reconstruction work on Liquid Wrench. And in this case I did see a work product and wanted to review with him, make sure I understood what Table 1 was versus Table 2 and 3 and, you know, how we did the analysis, how the monitoring was done, how long the samples were taken for, that kind of stuff.

Q. And do you have notes of that conversation?

A. I don't have notes, but I have a very clear recollection of it because it's only a couple of days ago.

Q. Now, had you visited with Mr. Spencer about these two issues before you issued your sworn affidavit in this case?

A. If I understand the question, no.

Q. Let me be more specific.

You issued an affidavit on approximately October 30th of the year 2003 specifically about Mr. Cowey's case, correct?

A. Correct.

Q. Had you talked to Mr. Spencer about any matters related to Mr. Cowey's case before you issued that particular affidavit?

A. Not specific to Mr. Cowey. I mean, I -- from previous discussions going back, I don't know, six, eight months ago, I -- I knew he had done Liquid Wrench reconstruction work; and I had a vague recollection of what it was. And so, I assumed that what I was seeing in this case was a product from that exercise. I don't remember actually when he did it, but -- but I knew it was done; and I knew -- knew vaguely what the results were.

Q. Well, I take it that you would not have spoken to Mr. Spencer this past Saturday about his exposure reconstruction work in this particular case if you had felt like the work you had seen six to eight months ago was adequate for your needs. Is that true?

A. No. I felt it was adequate -- well, I mean, it wasn't -- I didn't need it for my opinion to begin with; but -- but as long as I wanted to check -- knowing that I was going to be asked questions today, as long as I was going to call him, I -- you know, re -- you know, rather than -- I guess what you would say is I reconfirmed, you know, that this was sampling that they had done in -- in the generic sense and not specific for

Mr. Cowey and that Table 1 was actually monitored results and they were essentially two-hour -- two hours extrapolated data out of time-weighted average concentrations. What I knew previously, it reconfirmed that, you know, it was standard NIOSH-OSHA protocol, that they did it in a -- I guess the -- I got a few more specifics than I had previously described.

Q. Like what?

A. Well, it described that they had a closed-in like -- I guess mini room with sheeting -- he mentioned that there is actually videotape on that. I have not seen those. And described how they used two different concentrations.

I asked him why -- why the concen -- the level -- why they used so much Liquid Wrench because, from my own personal experience, you don't need that much.

But, you know, basically just reconfirmed what I -- what I think -- what I -- my impressions of, of what the whole exercise was; and I figured getting it firsthand wouldn't hurt.

But I called him -- I mean, I wanted to know about the -- the spiking issue. I mean, Levy suggested -- not Levy -- Dr. Rose suggested that you'd end up with -- with variable results, actually implying

that you would end up with lower numbers; and based on what I knew about -- about the chemistry of mixtures, that, if anything, the number -- the benzene -- evaporative emissions off of the current raffinate would be higher and faster than they would have been -- not off the raffinate, excuse me -- the emissions -- the vapor emissions of benzene would actually be potentially higher when mixing it in with the current version of Liquid Wrench as opposed to a raffinate mixture of Liquid Wrench.

So, that's really what I, you know, started going in to get clarification on.

Q. Have you seen the videotape?

A. No, I have not.

Q. Did he talk to you about that?

A. He described the room and said that they taped it, but I have not had an opportunity to see it. I don't know if I need to see it.

Q. You don't know?

A. Well, I mean, it -- it would be nice to see; but it is not going to change anything in my -- in my view. I mean, it's just...

Q. The product that Mr. Spencer tested, was it the product that Mr. Cowey used back in the 1950s, '60s, and '70s?

A. No, it was a -- it was a current version of Liquid Wrench with the benzene content adjusted to two different levels --

Q. What was --

A. -- or three different. It might have been three different levels.

Q. What was the difference between the version of Liquid Wrench that Mr. Spencer tested and the version of Liquid Wrench that Mr. Cowey used back in the '50s, '60s and '70s?

A. Well, first off --

MR. KEMP: Objection, form.

A. -- first off, it's unclear from any of the testimony of Mr. Cowey which version of Liquid Wrench he used; but presuming that some -- you know, if we accept that in some time period he may have used a raffinate version, it would have been a -- a version that contained an unknown quantity of benzene in a mix with a -- with a waste oil or some -- a drip oil of some ilk in this raffinate mix. Its composition would have been different from what is currently used today because, after 1978, benzene was no longer a component of the material.

So, Mr. Spencer spiked the -- what is probably a lighter molecular weight material -- total molecular

weight material than the earlier raffinate containing Liquid Wrench and then simulated, with no air flow, some exposure scenarios, directly measuring some and calculating using near -- Near Field model, calculating some others.

Q. All -- my question simply is: Can you tell us the -- the make-up difference, the component -- the raw ingredient difference between the Liquid Wrench version that Mr. Spencer tested, which you've called the current Liquid Wrench version, and the Liquid Wrench version that Mr. Cowey used back in the '50s, '60s, and '70s?

MR. KEMP: Objection, form.

A. I -- I am not sure if I have a -- a material specification for exactly what John Spencer used, but I do have material specifications provided as part of disclosure on raffinate-containing and nonraffinate-containing mixtures. So, I think I can, although it's not my area of expertise to start playing that exercise.

Q. (BY MR. LUBEL) In other words, was cat -- was kerosene in either version?

A. I don't recall.

Q. Does the mixture of the Liquid Wrench version -- in other words, what's in it -- does it have any effect on how much a person's benzene exposure is

going to be?

A. Yeah. I mean, benzene in a higher molecular -- higher molecular weight set of materials would have a different -- would evaporate differently than benzene in a lighter molecular weight set of materials. But, as I say, the -- the -- I mean, that's my knowledge base of the exercise; and that's why when Dr. Rose suggested -- and I think I knew -- well, I'm pretty sure that I knew that the current version would have been a lighter version.

So, it struck me that, if anything, what Dr. Spencer did was likely to result in a conservatively high number, not a number that would -- in other words, if it -- it would not only probably be as accurate as using, if you could ever find a sample of 7 percent or -- or if -- or, if it ever existed, a 30 percent version of raffinate, that what Mr. Spencer did would give you numbers that would be potentially higher, but certainly adequate to give you a ballpark estimate of what kind of exposure you were looking at.

Q. Well, you said that Mr. Spencer spiked the current version of Liquid Wrench, correct?

A. Yeah, I think that's correct.

Q. Is this what he told you?

A. Well, I think that's what his report states as

well. I didn't -- I didn't ask him again on that.

Q. And what were the other components of the current Liquid Wrench version that Mr. Spencer spiked?

A. You should ask him this evening. I did not --

Q. I am asking you what you think -- what you know about that.

A. Well, I think it was probably -- I don't know what the 19 -- well, I guess the 2000-plus component mixture of Liquid Wrench is. I presume it's, you know, a lighter cut. It's either a kerosene or a stoddard solvent type material or a naphtha kind of cut, but it would be a low aromatic -- probably lower aromatic content than the raffinate was; so, I mean, that's generally what I -- what I would expect it to be, based on what -- what looked to be where they were heading on a formulation basis.

Q. But as you sit here today, you're not familiar with what the components were of the current version of Liquid Wrench that Mr. Spencer tested, correct?

A. Other than the benzene content.

Q. That he spiked?

A. Yes. But you asked me...

Q. But take aside the benzene that he -- that he added to it. Do you know any of the other components?

A. I have not seen any material chemistry on it;

so, I don't know.

THE REPORTER: I need to change paper.

MR. KEMP: Take a quick break, Lance?

MR. LUBEL: Yes, you sure can.

MR. KEMP: Thanks.

(A break was taken, 10:43 to 10:48 a.m.)

Q. (BY MR. LUBEL) You and I, before we took a break, were talking about John Spencer and your conversation with him, correct?

A. Right.

Q. And you told us that on this last Saturday, you talked to him about two particular issues: one, Dr. Rose's comment with regard to spiking the benzene; and Number 2, the exposure reconstruction work and, in particular, the tables?

A. Correct.

Q. Is there anything else that you talked to him about?

A. Yeah. He -- he referred me to a paper that he knew that I probably had to support -- to support the statement that spike -- you know, that what he did would -- would actually be -- yield predictable results.

Q. Do you have that with you?

A. No, I don't.

Q. What's the report?

A. It's the Elkins, Pagnato report.
Q. The 1963 study?
A. Uh-huh.
Q. Is that a yes?
A. Yes. I'm sorry.
Q. And, in fact, I take it that you have looked at that study?
A. I don't have it here.
Q. In the past?
A. Yes.
Q. At some point, you have looked at it?
A. Yeah.
Q. Did you recognize from looking at the 1963 Elkins study that the content of benzene --
A. I -- I did take notes.
Q. Okay. Great.
A. Up --
Q. We will come back to that.
A. That's all I have.
Q. In looking at the Elkins study of 1963, did you appreciate that the content of benzene in the petroleum naphtha that they were studying was less than 2 percent?
A. You know, it's -- I don't recall that level of detail.

Q. Well, tell me what Spencer told you about that -- the Elkins study.

A. Well, he told me that the -- Vernon Rose actually misread that study and -- and I probably should have been taking notes, but I figured I would not be expert enough to redo the calculation; but he referred me to a section of that report where he says, you know -- he said that, you know, if -- if you read it and understood it, you would see what I did was, you know, perfectly reasonable.

Q. Is the Elkins study applicable to Mr. Cowey's case?

A. I think it's -- no. I think it -- it -- only in the sense that -- that I believe Vernon Rose has used that as a reference -- as a criticism of what Mr. Spencer did. I mean, I wouldn't...

Q. Do you think that's the reason he used the Elkins' study?

A. Well, that and the fact that you can get, under some conditions, exposures of -- you know, moderately high exposures from mixtures containing benzene; but, I mean, that's not a -- a world-renown secret, either. So...

Q. So, I take it that you don't disagree with the -- the proposition that even low content benzene

mixtures, such as the naphtha that was studied in the 1963 Elkins study, can give off pretty high exposures in the 30 part per million range?

A. I -- I am not -- I've not reviewed the Elkins; so, I'm not going to agree with that piece of your statement. What I will agree with is that benzene-containing mixtures, under the right conditions of use, can give you exposures above the current -- current accepted T.L.V.s.

Q. And what information do you need to know with your experience in industrial hygiene to determine whether the mixtures itself that contained benzene, under the right conditions, can get you levels in excess of the permissible exposure limits?

A. Well, a big piece -- big piece of this is a quantity issue and conditions of use. I mean, we're talking -- we're talking Liquid Wrench exposures; and I -- I suspect, if you're at all a handyman, which I was in my first life when I was poor and had to do everything myself, you know, I have used Liquid Wrench on numerous occasions. I know that you don't need copious quantities of the material. Use -- you know, not even -- I mean, the levels of material that Mr. Spencer uses are, in my judgment, much higher than you would typically use; so, I considered what he is

doing sort of the worst case scenario type stuff.

Mr. Cowey, in his testimony, said he'd put it -- he'd put it on the -- on the piece and go away. And, in fact, I think he specifically said he'd go away for 24 hours, which is probably unusual in my mind, you know, to wait that long for it to work; and that Mr. Cowey tended to use it in ventilated areas.

But you're still talking, you know, half ounce, ounce kinds of quantities of a material that contained, you know, 1 to 14 percent, depending on -- on time period. You know, we don't know precisely what the exposure was.

But even in Mr. Spencer's simulation of -- of 30 percent -- and I say simulation. Not that he made it up. I mean, he directly applied that quantity to -- to a -- to a fitting and then measured evaporative emissions over a two-hour period. I mean, that -- that is not what Mr. Cowey says he routinely did; but it certainly gives you an estimated ballpark that confirms my own belief that the exposures were -- were relatively minor.

And remember, you know, I have done a lot of studies of acute myelogenous leukemia. You need dramatically high, repeated exposures to -- to initiate that disease process. So, they just aren't there in

Mr. Cowey's case.

MR. LUBEL: Will you read back my question?

(Requested Question read back.)

Q. (BY MR. LUBEL) I am not asking you about Mr. Cowey's case. I am talking about generally --

A. Okay. Generally?

Q. -- what you need.

A. Well, first I'd want to know what permissible exposure limits are you talking about? Are we talking about permissible exposure limit in the 1960, 1970, 1980 or 1990? So, that goes a long way in -- in the amount of detail and historical information I would need, I would -- if we have a material that has the intrinsic hazard to cause a disease. So, we have benzene and we have leukemia, acute myelogenous leukemia -- I generally would like to know as much about the quantitative nature of the exposure as I can get.

As an epidemiologist, I would not routinely be the one to generate that information; but if there isn't quantitative information specific to reconstruct it for the individual, I rely on what I know from petroleum refinery workers. We've studied them extensively. They -- we know that benzene is in 1 to 30 percent -- 1 percent to 30 percent in the streams all over the

refinery; so, it's not unusual to have refinery workers exposed to lows levels. I know that we have not seen excesses of leukemias in refinery workers hired after the 1950s when the T.L.V. went to 25.

So, I sort of would work through the logic of how likely is this individual to be exposed to concentration levels or time-weighted averages substantially above 25 ppm, how long that would have occurred, and when that occurred relative to when his disease was diagnosed.

So, that's the mental process I would go through.

Q. But how would you, with your experience in industrial hygiene, look at a mixture and determine what the exposures to benzene were from that mixture? Generally speaking.

A. I wouldn't. I mean -- I mean, while I may have had the academic training to do it, unless you do those things regularly and with precision, you know, I am not qualified to do that. So, I mean, I -- I would go back to whoever has asked me to do a risk assessment on an individual and say tell -- you know, "Do we have a -- an estimate of his exposure that's quantifiable?"

Q. You would hire somebody else to do it, is what you're saying?

A. Yeah. Well -- but not in all cases do you get one. So -- but, you know -- yeah, I mean, I -- I'd recommend that you go get one if you can. But you need a certain amount of information to -- to get -- to get a -- let me restart.

For purposes of a specific risk assessment, depending on the disease, you need varying levels of precision on the exposure estimate, if there was any reason to believe that the exposures were high during certain periods of time, that would suggest that you would be approaching cumulative time-weighted averages of 200 ppm, where the risk for AML goes up dramatically.

So, you -- I would look for more precision in the estimates when there are higher exposures. If the exposures are essentially trivial or diminimus, I need less precision in the estimate because I can see very simply that if -- if its concentration of exposures or time-weighted average s are, you know, under a ppm or under a few ppm, he's just not going to get a dose suitable. So, I can work from an exclusionary place, not -- not necessarily an inclusionary place.

Q. Let me ask you this: Does the content, the amount of benzene, in a mixture influence or have any impact on the person's exposure to it?

A. No, because that's intrinsic -- that's an

intrinsic property of the material. You have to look at conditions of use before it starts to make any sense.

Q. Conditions of use affect the exposures, correct?

A. Correct.

Q. But as a general proposition, the more benzene in the product, does that mean more likely than not there is going to be more exposure, if it's a mixture?

A. Only if exposure -- if the material is being used in -- in a condition that results in exposure. I can have 100 percent benzene sitting on my shelf for 30 years and come down with MDS, and that benzene did not cause -- cause it --

Q. Well --

A. -- because I don't have exposure to it.

Q. -- let's put it this way --

A. I am just trying to get you to separate --

Q. Yeah, but --

A. -- in my mind, a very --

Q. -- but you're talking about causation. I'm not talking about causation.

A. Yeah, but I'm talking very specifically about intrinsic hazard of a material versus the -- what the risk could be from that material.

Q. But I am not asking you about risk.

A. Okay. Then I misunderstood.
Q. I'm asking you about industrial hygiene.
A. The question.
Q. Let me ask it again.
If a person is working with a mixture, a product that contains benzene, would you agree with the general statement that the more benzene that is in the product would mean that exposures would be higher, assuming that the use condition -- or the conditions of use, there's going to be some exposure?
A. Yes. I mean -- I mean, that's kind of, in one sense, obvious. I mean, the -- even -- you get very dramatic differences, looking at the --
Q. I am not asking you about this case. I am just asking you in general.
A. No, but I'm saying, obviously, I mean, if you have the same amount of -- if you're exposed to the same quantity of the material and conditions of use, the higher the benzene content of that material is going to affect the total benzene exposure. I mean --
Q. Now, from an industrial hygiene standpoint, in order to estimate what the benzene exposures are from the mixture, assuming you know how much benzene is in the mixture, do you also need to know what the raw ingredients or components are of the rest of the

mixture, other than benzene?

A. We're rapidly getting to the edge of where I am comfortable offering opinion on industrial hygiene matters, but I will -- will go so far as to say it depends on the importance of the precision of the result that you're looking for. If -- if you are looking for high levels of precision and you're not testing the material directly, yeah, the more -- you will need more information.

Q. Okay. So, would it be appropriate for you, as an epidemiologist, assuming you didn't have any quantitative data, for instance, to rely on the Elkins' study if that study stands for the proposition that under different use conditions a mixture that contains as little as 1.6 to 2 percent benzene can result in some pretty high part-per-million exposures?

A. You're misunderstanding, and I think now you are going back to the Elkins' report. I think you are misunderstanding an intrin -- a key element of -- of this whole exercise, and that key element is it is substantially -- the volume of material that is being evaporated, it plays a big role in what the total ppm content in a -- in a relative space is going to be.

And you're -- you're trying to oversimplify the issue of the quantity of material that is -- that is

evaporating. And here we're talking half ounce, ounce of materials. We're not talking an industrial exposure where, you know, there's 2 gallons of -- of 3 percent material spilled in a confined space. Different beasts.

Q. Are you -- is it your understanding that the Elkins' exposure data came from confined spaces?

A. No. I am just giving you my -- trying to give you my -- my analogy as it -- as it applies generally to this case.

Q. But tell me how the Elkins' --

A. I don't remember the Elkins' report.

Q. -- data -- okay.

A. We've covered that already.

Q. All right.

A. If you want to get into that, I will sit here and read it and go over it; but I think it was a pretty detailed industrial hygiene report.

Q. Can you point to me or -- what were Mr. Cowey's exposures?

A. To --

MR. KEMP: Objection, form.

A. -- what?

Q. (BY MR. LUBEL) To benzene. How often did he use Liquid Wrench?

A. Oh, potentially -- well, we --

MR. KEMP: Objection, form.

A. From my recollection of reading his deposition, we don't have specific information on how frequently he used it. Based on the fact that during the period of time when Liquid Wrench contained benzene, he worked on approximately eight or nine houses -- I mean, most of the houses he worked on were in later years -- and his description of using it to free up pipes, that -- that doesn't -- I don't believe from any of the testimony that he gave that he was replacing every pipe in the bloody house. So, you know, if he used it periodically when -- when he was doing the plumbing piece of his fixing -- house fixing up, he'd have intermittent, periodic exposure to whatever level of benzene it contained; but he also describes that he would just apply it and then leave the area and come -- come back 24 hours later.

So, unless you're postulating instantaneous exposure, he may -- even if it did contain benzene -- have trivial exposure.

Q. (BY MR. LUBEL) How often --

A. He would have had other exposures to a variety of materials going back into those time periods for which I have not seen data on, but presumptively other materials, paints, or -- I don't know if he did

furniture stripping. It wasn't quite clear what all he was doing in some of these houses. You would have had other solvent material -- containing materials that -- that may have had benzene levels.

Q. How often did Mr. Cowey use Liquid Wrench, or do you know?

A. I -- I don't think -- my reading doesn't permit me to offer a quantitative estimate.

Q. Do you recall there being a specific question asked that detailed to Mr. Cowey?

A. I remember questions on how much Liquid Wrench he may have used in the course of a year, and that seemed -- struck me as quite a large volume; but I think it was -- he did offer a volume. I don't recall how frequently he said he used it on a daily -- I don't know -- I don't recall anything that says daily or weekly or...

Q. You don't remember that line of questioning --

A. No.

Q. -- correct?

How do you recall the testimony where there is an indication that he used approximately 6 gallons a year?

MR. KEMP: Objection, form.

A. I recall that -- that his original statement

was like three to five, maybe six, kind of range of -- so, I don't know exactly whether it was six or three or something less than that; but that's a lot of Liquid Wrench, if you've ever used Liquid Wrench.

Q. (BY MR. LUBEL) And it's in particular a lot of Liquid Wrench over 26 years with benzene in it, is it not?

A. Well, we don't --

MR. RILEY: Objection, form.

MR. KEMP: Objection, form.

A. I don't recall any statement saying that "I used 6 gallons a year up until '78"; so, we -- we really -- we don't know two things: we don't know which Liquid Wrench he used, and we don't know the -- the time period you're laying out. It did not contain benzene the whole time period.

Q. (BY MR. LUBEL) Really?

A. Well, which -- maybe I misheard your -- the time period.

Q. Let me ask you this: If Mr. Cowey used approximately 6 gallons of Liquid Wrench that contained benzene between 1952 and 1979, would you agree that that's a lot of Liquid Wrench containing benzene?

MR. KEMP: Objection, form.

A. It's -- I -- I don't know if it contained any

benzene --

Q. (BY MR. LUBEL) I am saying assuming.

A. -- what he bought.

Q. I said assuming.

MR. KEMP: Same objection.

A. I have already said it's a lot of Liquid Wrench to use annually; so, I mean -- I don't know. I mean what -- what...

Q. (BY MR. LUBEL) That's a lot of --

A. We all -- but we already know that he -- that he also used -- states he uses it when -- wherever possible, in well-ventilated areas. He used a fan. And he put it on and would leave the area. So, I don't know if it's a lot of benzene exposure. It's a lot of Liquid Wrench. That's all I can tell you.

Q. You don't have enough information to assess his benzene exposure, do you?

A. Quantitatively?

Q. Correct.

A. No.

Q. Have you seen Dr. Samimi's report?

A. Yes.

Q. Okay. When were you furnished that?

A. I read it briefly yesterday.

Q. And did you talk to Dr. Spencer about

Dr. Samimi's conclusions?

A. No, I didn't.

Q. Do you have any criticisms of his report?

A. Dr. Samimi's?

Q. Yes.

A. It's ridiculous. I mean, it's...

Q. What are the criticisms?

A. Well, I -- I didn't go over it -- I didn't go over any of the calculations, per se; but it is ridiculous to presume that -- one of the scenarios is 4 ounces per application. And then the next scenario is using it twice a day, eight -- five days a week, 50 weeks a year. I did do a quick calculation that that's roughly 14 gallons of the material a year.

I -- I don't think refineries use that much material. It just -- it is just so improbable and so out of -- inconsistent with both the -- the testimony of Cowey. It doesn't even give me a -- an intelligent ballpark. I just found the whole thing sort of silly. And that's -- I stopped reading it, and I threw it back across the table.

Q. Well, how much is -- is a teacup?

A. Well, it depends what kind of teacup you have; but I will tell you the teacups that -- that my mother used to have were, I believe, 6 ounces.

Q. So, a half a teacup would be approximately 3 ounces?

A. Yes. But I don't know of people who go around putting Liquid Wrench in teacups and then applying it to pipes; so, the -- the speculation, what was that, from Mrs. Cowey, that -- that he used teacup -- half a teacup quantities, you can't -- you can't use that as a basis to try and quantitatively estimate how many ounces would be used. It would be way more logical to -- to look realistically at the -- at the material. I mean, I've done cast iron pipes. I've done nuts and bolts with it. You -- you apply, you know, enough material to, you know, saturate the joint that you're trying to loosen; and --

Q. How much is it?

A. I -- well, in my personal experience, it's probably somewhere between a few drops and a -- and a half ounce, because I know my can -- my 8-ounce can, or whatever can size it is, lasts for years. Now, I grant you, I'm not, you know, doing -- running bolts every time; but it's not a material you need copious quantities of. It would drip right off and make quite a mess.

It's just -- it's just -- and -- and nowhere -- I mean, using it twice a day, he had the

individual exposed, I think, for an entire hour period in his calculation, maybe longer, or maybe -- no, I recall, the 70 percent of the total volume -- sufficient to evaporate; so, there was no actual time calculation in there.

It just -- in my judgment, it was just too artificial to be of any utility for -- for anything.

Q. Have you covered all of the criticisms regarding Dr. Samimi's report?

A. Well, I said I have not attempted -- it's not an easy report to read, and I think I spent 15 minutes on it. So, I did not attempt to actually check to see whether his calculations are correct or other elements of his estimates made any sense.

Q. Would you agree that if Dr. Samimi -- any of the scenarios that he gave are reliable exposure levels for Mr. Cowey, that, in fact, he would have had enough exposure to benzene over a 26-year period to develop MDS? If his information is accurate.

A. I didn't actually follow it all the way through. It just struck me as so ludicrous on the volumes and the frequency that I didn't -- I don't even remember what -- what final numbers he came up with. They were all STELs; so, there was no -- I believe they were STELs. So, it's -- one would need to reconvert

them back to time-weighted averages for me to understand it better.

I mean, yes, I saw some numbers that would have been over the -- today's STEL, but a number of the numbers would have been under the time-weighted averages of the '60s -- of 1950 and -- well, certainly 1950 and 1940s, the numbers would have been acceptable T.L.V.s. So, I didn't attempt to go through the report at that level of detail. I leave that to others.

Q. What is the safe level of benzene exposure?

A. Safe is a political statement. A scientific statement is that there is no evidence of increased risk of disease until you accumulate 200 parts per million person years, and typically the concentrations would have to be over -- at least some of the concentrations would have to be over 50 ppm concentration levels. So, it's not just purely person years.

Q. So, if a person is exposed to less than 200-part-per-million years of benzene, they're at no risk of developing a benzene-related disorder or disease, correct?

A. They would be at no measurable risk.

Q. What do you mean by measurable?

A. Well, only that there is no evidence to say that they're at increased risk. I mean --

Q. So, they would be safe?
A. Yeah. It would be safer if they were exposed to substantially less than that, as well. I mean, any -- any number -- substantial -- you know, that's the level where we've got evidence that it's bad. So, you pick a safe level below that.
Q. So, I take it that you disagree with the statement that the American Petroleum Institute made in 1948 that there is absolutely no safe level of benzene exposure?
A. But that's a generic toxicity statement. I mean, no exposure to a toxic material cannot -- is the safest level you can achieve. I mean, at -- it's a semantic statement. It's not a -- a statement of -- of reality. Zero will be the safest exposure on any toxic material.
Q. Do you know what the A.C.G.I.H., the American Conference of Governmental Industrial Hygienists, recommended exposure level is to benzene right now?
A. Yeah. I believe it's .5 -- .5 MTLV.
Q. A half of a part per million?
A. Uh-huh.
Q. Over a working career?
A. Well, they presume a 40-year working career.
Q. That would be about 20-part-per-million years,

correct? Forty years at 25 --

A. Right.

Q. -- would be 20-part-per-million years, true?

A. Correct.

Q. That's a lot less than 200-part-per-million years?

A. That's correct.

Q. So, I take it that you disagree with the American Conference of Governmental Industrial Hygienists that exposure levels in excess of 20 part per million years are unacceptable?

MR. KEMP: Objection, form.

A. No. They -- they have chosen a wide margin of safety in their calculation. I mean, you're -- and you're talking something that they came up with in '92 or so. Up until that point it was 10 for many years. Actually, maybe -- it may have gone down to 1 before they went there.

But, as I say, setting of safe limits -- and I don't want to use political -- I mean social -- social politics is a -- is an -- is a different proposition than estimating where -- what -- what the risk levels are for demonstrable disease. So, I have no problem with the level. I did comment to them in that process that it was unnecessarily low.

Q. (BY MR. LUBEL) Do you have with you the studies that -- that show us that people that are exposed to -- or that conclude, actually -- do you have studies with you that conclude that workers exposed to less than 200-part-per-million years are safe from benzene?

A. Yeah, we have lots of -- I mean, the way you have just stated it, it would be any study that didn't show an excess of leukemia; and there -- are you interested in reports that demonstrate the 200 ppm as the point of departure where the risk goes up?

Q. I am interested in a study --

A. Or are you interested in studies that show that there is no risk in workers exposed to levels below that?

Q. I am interested in a study that concludes that workers exposed to less than 200-part-per-million of benzene are safe. Can you find that study or those studies that conclude that specifically?

A. Well, you don't write studies that way; but if you look at the petroleum refinery workers studies that are summarized in one of the papers that I have attached there, that demonstrates that since 1950 -- do you want me to --

Q. If you can just pull the studies --

A. Okay.
Q. -- because I am going to ask you to find the conclusion that says this.
A. Well, the conclusions don't -- you don't write conclusions that way.
Q. Well, get me the closest conclusion that says it.
A. I mean, what the study will say is that there was some evidence of increased risk in workers hired before 1950. There isn't in workers hired after 1950. Here's the array of acute myelogenous leukemia across all the studies. There will be discussion of what the exposure -- the published exposure numbers that have been described in various literature for benzene in petroleum refinery workers, and there will be a conclusion that there does not appear to be a risk at these levels. That's it. You don't work off of a threshold.
Q. Can you find the study that you believe is closest to stating the conclusion that workers at exposure levels to benzene below 200 parts per million are safe, or are not at risk of getting disease?
A. It appeared -- there are two references in the pile that you've got here. One is the -- the -- one is the -- is the short letter by Dr. Wong, 19 -- 1998, in

the Journal of National Cancer Institute. The other is the discussion of acute myelogenous leukemia in the paper -- and there are a couple of versions of the published paper. The ones -- the one that is included here is a paper by Raabe and Wong entitled "Leukemia Mortality by Cell Type in Petroleum Workers with Potential Exposure to Benzene."

Q. That's by who?

A. Raabe and Wong. Me, myself, and Dr. Wong.

Q. Okay. Now, let's go -- let's first talk about your study.

A. Okay.

Q. Can you find for me the conclusion -- or the closest thing you can find to a conclusion that people that are exposed to less than 200-part-per-million are not at risk of some type of benzene-related disorder?

A. Yeah, it's -- under the word, "Conclusion," there is a paragraph that resummarizes what I just said to you before. And if you go into the "Result" section and the "Discussion," section you will see more background on -- on that.

Q. Can you just -- will you highlight for us the -- the sentence that says it?

A. It's not a sentence. It's a paragraph.

Q. Can you highlight the paragraph? Or circle

the paragraph.

A. I mean, you're asking me for a conclusionary statement. That's the closest to -- to that. There is more discussion on -- about benzene exposures under the section called "acute myelogenous leukemia," which goes on for several pages.

Q. How do you deal with the -- the Australian Petroleum Institute study that ExxonMobil sponsored that says that lower levels than what you're suggesting causing benzene-related disorders can, in fact, cause benzene disease?

A. Well, you're talking about the Health Watch studies, most -- the first -- the first partial of that was published just this year. There is a whole -- excuse me. Are we -- are we done with this?

Q. Right now I'm talking about the A.P.I.

A. Okay.

Q. I mean, not the A -- the Australian Petroleum Institute?

A. The Australian?

That study has just come out. The unpublished version of the report clearly demonstrates that however they reconstructed the exposures, they came out with exposures substantially lower than either the U.K. study, the Canadian study, or any of the studies that

we've seen so far.

That suggests to me there's something wrong because it's hard for me to believe that the Australian Petroleum Institute was so different from the rest during that period. So, while they may -- you do understand enough statistics that a dose-response curve can slide anywhere on an axis. If I multiply all exposures by 10, I'll get the exact same shape of the curve. I have just moved the -- where -- where it falls in the various levels on a -- on the risk exposure profile.

So, I think they've got it wrong. It's -- and it only speaks to AML. I mean, they found no relationship to multiple myeloma and non-Hodgkin's lymphoma; so, it suggests to me that they may have the right shape, they just have the dosimetry wrong.

Q. Well, they did say there was a statistically significant excess of CLLs. You read the same study I did.

A. Well, I'm not sure if it's still significant in the published study.

Q. What happened from the unpublished to the published study?

A. Well, the numbers are different from the unpublished to the published, which is one of the things

that is going to have to get resolved in the scientific community over the next couple months or years.

Q. Well, why would the --

A. The numbers are different.

Q. Why would it be different?

MR. KEMP: Objection, form.

A. I'd just -- I'd be speculating. I can tell you what happens in studies that could account for that, but I wouldn't know why they're different. The numbers are different, though, between the published and the unpublished. And not all the tables. Some of the tables.

Q. (BY MR. LUBEL) Have you talked to anybody from ExxonMobil about the study?

A. I spoke with Rob Schnatter when the unpublished version came out.

Q. What did you say to him?

A. That this thing is a bear to read, and it looks like they got -- you know, why -- I mean, they were just a sponsor; so, I mean, they're sort of stuck with the same report that I am. They had difficulty understanding what's going on because a lot of the analyses you would like to see at cell type levels are not done at cell type levels. So, as you move through the unpublished version of the report, there's some

interesting stuff in one analysis, but they never do that analysis again when they're looking at other parts. Are you familiar with -- this is a 300-and-something page report with lots and lots of tables.

Q. So, they just missed it --

A. Well, I don't know what -- I mean, I -- I think there is reason to believe that there are errors in -- errors in either the exposure assessment or the way they calculated the dose response.

We already know from reading the unpublished one that the tumor registry from which they're generating their expecteds did not identify all the cases in their own cohort. So, you do have a potential problem where they used best evidence to get the cases; and -- and in the actual cohort piece of the study, the expecteds are coming from a somewhat different set.

So, there are a number of technical methodologic problems. The study's just recently out. Clearly has a different exposure profile for the industry than we have seen elsewhere. So, I -- you know, I think it's too early to tell whether it's -- you know, has any utility or not. I mean, I talk about it in my report briefly.

Q. And the -- the conclusion reached by Hayes and Yin and others in the 2000 Chinese study article, if you

will, that talks about excess risk of MDS at exposure levels below 10 parts per million, you disagree with that, too, correct?

A. I don't know if it's right or wrong. I am telling -- I am telling you that the -- the analyses of the -- of that series of cohort studies that come out of it are presumed to have what are potentially fatal flaws in exposure reconstruction, and that's -- I mean, I am not going to try and adopt a -- a result that even E.P.A. says can't be used.

Q. All right. The second article that you referenced as evidence that exposure levels to benzene in excess of 200 parts per million were necessary to cause benzene-related disorders was another Wong article, correct?

A. Yeah.

Q. Is it Exhibit -- in Exhibit Number 1?

A. Yeah.

Q. Is it the first article?

A. No. Well, I don't think he discusses that. No, it's -- it is this one.

Q. Can you highlight the section --

A. Sure.

Q. -- or circle it?

A. (Witness complies.) Well, this -- this --

there's -- there is another paper that I thought I also included in here, but I didn't. This -- this one you would have to pretty much read the -- I mean, it's only two typed columns. You would have to read the whole shebang to get the -- the essence of what I have -- what we were just discussing out of it because it tends to focus more on -- it just briefly focuses on leukemia.

There is one additional paper that I didn't cite here -- that is not cited, but it's also published by Wong, and I think it's probably 1996; but it's not in the -- the material is rereviewed in a number of different papers, but the -- the first paper in that series is not -- the first Wong paper in that series is not here.

Q. What other authors, in addition to Wong, of which you have articles, have reached this conclusion in the published paper?

Let me restate. Do you have an article here before you, or one that's referenced in your affidavit, that states or concludes the same thing that we have been talking about but was authored by somebody other than Wong or yourself?

A. There's a paper by Kenny Crump that does an analysis specifically looking at AMLs and nonAML leukemias.

Q. Do you have that with you?
A. No, I don't. But -- but Kenny Crump used different cut points. His interest was more a regulatory modeling interest; so, his cut points were 45 ppm years versus -- I don't know what the next higher category was. But you get -- it's the same -- exact same data set; it's just cut differently. And you get the same interpretations with just cut points that don't quite come up to the 200. I mean, what -- what Otto and I have done in various papers is essentially go back it up.

The other paper that you would want to look at, if you're interested in that subject, is a paper by Robert Schnatter -- not Robert -- Rob is -- that first name is Rob, R O B, Schnatter did using the -- the same --

Q. Is it Schnatter or Schattner?
A. Schnatter.

Schnatter took a different approach than Otto and I. He actually -- because our paper was already out, he's of the opinion that concentration is perhaps a more -- a more important metric than the actual cumulative exposure.

Q. What do you mean by that?
A. It's not -- and I don't disagree -- disagree

with him.

There are two ways to look at exposure metrics, or dose: dose rate -- in other words, how much -- they both have elements of time, but one is how much dose -- how much exposure is actually delivered in the period of time, as opposed to cumulative dose, which is how much exposure is accrued over time.

Q. Is he the fellow that thinks that the STELs, the short-term exposure limits, are more important than the long-term exposures?

A. Well, there are -- no. That's a different set of folks. Rob -- Rob is an epidemiologist. Rob -- what he thinks about the STELs, I have never discussed it with him.

But Rob -- Rob's analysis suggests that -- which is not inconsistent with -- with everything else -- that you really need exposures substantially above 25, probably over 60, ppm concentration over some period of time.

Q. What period of time?

A. He shows in excess at 60, AML, excess at 60 for six years. I think I am quoting it correctly.

Q. Sixty over what duration?

A. Six years.

Q. Every day?

A. Yeah. Well, time-weighted averages, the -- he did several analyses with different exposure metrics. One analysis says they have to be over 25 -- 20, 25 for six years; the other says 60 for six years, which is not inconsistent with what -- with what I have in my report.

Once -- once the T.L.V.s went down below 50 -- in other words, once we went down 25 ppm time-weighted averages -- we no longer saw AML excesses in any of these populations. I mean, that's an indirect way of saying the same thing.

What -- what -- what Rob was doing in his analysis was looking at -- he cut the concentration -- remember from the Pliofilm cohort, we have T -- we have time-weighted averages annually for these workers. So, he'd look at all the workers who never had time-weighted averages above X, and then another cut above X plus 10; so, in essence, you end up with a dose-response curve that is really focusing only on concentration. And you don't see any increased risk for AML until you get a long-term exposure at concentrations above 60, which was the average metric.

Q. So what he found was if you have high enough exposure levels, which you say is above 60 parts per million --

A. Well, I didn't say. I mean, it's --

Q. You said that's what the study says?
A. Right.
Q. -- for six years, then you can have an excess risk of leukemia?
A. Right.
Q. You disagree with that?
A. But that gives you -- you know, in the same -- you're in the same person years -- you're in relatively high person year levels at that point; so...
Q. I am just asking you if you disagree with his conclusions.
A. No, I don't disagree with it. It's just a different way of looking at it. I mean, what it all says is that we go back to the underlying biology. You need a certain threshold of dose that is going to initiate toxicity in the bone marrow that's going to -- and you need to sustain that over some period of time before you're going to increase the risk of -- of AML. And that's, in essence, why the T.L.V. is dropped -- I mean -- and why they left it at 25 for such a long time, because they were trying to reduce toxicity; and once you reduce toxicity to very low levels, the AML risk goes away.
Q. And does that risk vary depending on individual susceptibility?

A. Oh, sure. I mean, individual susceptibility, which is something we can't measure, is -- is -- I mean, otherwise everybody exposed, you know, would be -- would come down with the disease.

Q. In other words, not everybody that smokes a pack of cigarettes a day for 30 years gets lung cancer, correct?

A. Correct.

Q. There is variation, not only in groups but in individuals, correct?

A. Yes. But we just -- I mean, we don't -- I mean, that's why we're doing more and more molecular biology and molecular epidemiology where you're not only spitting -- splitting people by their external exposures but you're -- you're looking at whether there is different dose-response curves in people with different genetic phenotypes.

Q. Right.

Now we're getting ready to take a lunch break we agreed to, but I want to ask you just a few questions on the area before we take a break. And that is, you made reference to Mr. Cowey being exposed to other products other than Liquid Wrench. Do you recall that?

A. Yes. He states that he used a variety of different --

Q. Paints, thinners, et cetera?
A. Yeah.
Q. Are you of the opinion that any of those products or any components in those products have caused or contributed -- contributed to cause his MDS?
A. I don't have enough compositional information to -- to render -- to draw an opinion on that. I -- I am just stating that he had a number of other materials that if we had more information about -- I mean, unlike Liquid Wrench, there is not even a large discussion of -- about those other materials. The detailed discussion seems to be a little confined to Liquid Wrench. So, I just say that -- state that in the time periods that he was at those exposures, some of those materials would have contained some levels of benzene.
Q. But as you sit here today, are you of the opinion that any of those other products contributed to cause his MDS, given the information you have?
A. His chromosomal analysis is not of a pattern that's -- that is consistent with a benzene or secondary exposure MDS. I mean, he does have a trisomy 8, but he doesn't have the 8 that is -- is very frequently associated, and that's 5, 7 deletion.
Q. But as you sit here today, yes or no, did the other products that he used over his career cause or

contribute to cause his MDS?

MR. KEMP: Objection, form.

A. I don't know.

Q. (BY MR. LUBEL) But do you have an opinion that it did?

MR. KEMP: Objection to form.

A. My opinion is I don't know.

Q. (BY MR. LUBEL) What information do you need?

A. That means I have no opinion.

Q. Okay. What information do you need?

A. Com -- at least some composition -- well, I'll give you an illustration. Can we do that? If -- if we're talking about the paints, if we had compositional information on those paints, the -- the nature of use of painting an interior room and the time it takes to do that would have afforded him a much greater opportunity for an exposure to benzene if benzene was in those paints. I have no knowledge that benzene was in those paints; so, I can't offer an opinion whether paint contributed to his -- to anything.

Q. But you do know that benzene was a component of paints before the 1980s, correct?

A. I mean, I've seen the statements. I have not seen any analytic quantification as to what kinds of paints contained what levels of benzene.

Q. You can't say that the other products caused his disease, correct?

A. I don't have enough information to make that statement.

Q. Have you asked the lawyers that hired you to go assemble that information?

A. I asked the lawyers if they had more information on where -- were there -- was there another deposition that dealt with specifics on some of the other materials that he used at the same level of detail and did we know anything about -- with any level of detail the paints that he used, and the answer I received is no, they did not. So, I don't know if they looked, didn't look; but my question is was there more detail, and the answer was no.

Q. Based upon scientific probabilities, you can't relate any of the other products that Mr. Cowey used to his disease, correct, with the information you have?

A. No. I can just list them as potential sources of exposures that could be related to MDS generally, and I don't think they may have had the role with his MDS specifically because I think his karyotype is not the typical profile of a secondary MDS.

Q. And if I hear you correctly, what you're saying is that if you had a list of the products that he

used, including paints, as your example, and you knew the components of those products, then you would be in a better position to render an opinion as to whether any of those other products may have contributed to cause his disease.

MR. RILEY: Objection, form.

Q. (BY MR. LUBEL) Is that true?

A. That's true, Part A, yes.

Q. What's part B?

A. Part B is I would like to have had -- had the same types of questioning as to how frequently he used them, where he used them, and -- and what time periods.

Q. And other than that, what else, if anything, would you need?

A. Well, I mean, if we had that kind of information, then I -- then I would have gone back and said, you know, "It strikes me that a significant -- that this man was getting a significant benzene exposure from painting, and is there any way that someone could, you know, give me some ballpark quantitative estimates?"

That would have been my next question. But --

Q. And then from there you would render an opinion?

A. Well, I probably could have rendered -- I mean, it depends on how much benzene was in the paints

and what I knew about its application. I probably could have rendered an opinion without the quantitative material, certainly a differential opinion.

Q. What do you mean by that? I've got to stop you there. What do you mean a differential opinion?

A. Well, for example, even if we could confirm that he actually used a benzene-containing Liquid Wrench, okay, I -- I'd be able to say that the painting was so dramatically -- I am making this all up. We are all understanding that, right?

Differential opinion could be that the nature of the exposure from this source is so dramatic that, you know, the fact that he occasionally smoked and that he occasionally used Liquid Wrench and that he occasionally went to self-service gas stations, none of those contributed materially to his -- to any benzene exposure risk because it was swamped by this class of exposure. That's what I mean, a differential.

MR. LUBEL: Okay. Let's take our lunch break, and then we will come back.

(Lunch recess, 11:45 a.m. to 12:42 p.m.)

(Exhibit 3 marked.)

Q. (BY MR. LUBEL) What is Exhibit Number 3 that I have marked?

A. Exhibit Number 3 are the materials that I

received, less two depositions from Fulbright & Jaworski concerning Mr. Cowey.

Q. And do Exhibits Number 1, 2, and 3 constitute your file materials in this case?

A. Materials that I used in direct preparation for this. I have more -- more on myelofibrosis and a lot more on benzene and AML.

Q. But as far as materials that you used specifically for this case, does it constitute those materials?

A. Yes.

Q. Can you pull out the study that you brought with you that stands for the proposition that to have a ben -- benzene-induced disease, you need the chromosome defects 5 and 7?

A. I may not have brought all the -- cited directly all the cytogenetic articles, but I think that's discussed in at least one of these. Well, actually, you just overstated what I stated earlier. What I stated was secondary MDSs most frequently have, as the key representation, a 5, 7, plus other issues.

Q. Well, can a person that has a benzene -- benzene-induced MDS not have chromosome defects of 5 and 7?

A. I don't know. I am not an expert in that

area. I am just recounting you my recollection of the literature. I wasn't asked to specifically opine in that area.

Well, some of that is discussed in Ciccone. I think it's discussed in Rodella, "Cytogenetics and Occupational Exposures in Acute Nonlymphocytic Leukemia and Myelodysplastic Syndrome."

Q. Can you just -- as you're going through them, if you can just highlight those pages and the sections that deal with that.

A. All right. That will take a little longer.

The second paper is Rodella, 1993; and -- the whole Rodella paper is, frankly, related to that subject matter; but there is a specific table that speaks to the chromosomal aberrations that perhaps summarizes some of it, and that's in Table 2 of Rodella.

Q. Are there any of these studies that stand for the proposition that chromosome defects 5 and 7 need to be present in a person for their MDS disease to be related to benzene exposures?

A. I've not reviewed the literature from that perspective exclusively. There have been several studies specific to AML looking at long -- large numbers of cases -- I didn't bring those papers. I am not even sure if I have them all -- and they speak to the issue

more that -- that it is a frequent, very high percentage marker in cases that are either treatment therapeutic or benzene-related; and that's sort of where the proposition comes from.

I think the two I mentioned are the only two that I -- that I have with me here that specifically speak to the frequencies of cytogenic abnormality.

Q. Right. Do you mind handing me those?

A. Yeah.

Q. Oh, I just wanted to focus in on those two studies.

A. Well, as I say, it's not the complete literature by any regard, because I wasn't specifically rendering an opinion in that area.

I am pulling these out from my set -- from your set.

Q. Yes, sir.

So, the three studies that you've pulled are "The Myelodysplastic Syndromes" by David P. Steensma, et al?

A. Uh-huh.

Q. Is that true?

A. Yeah.

Q. And myelo -- "Myeloid Leukemias and Myelodysplastic Syndromes" by -- how do you pronounce

the first name? Ciccone?

A. Well, that's how I pronounce it.

Q. Okay. And then the third study is by Rodella, and it's "Cytogenetics and Occupational Exposures in Acute Nonlymphocytic Leukemia and MDS" --

A. Right.

Q. -- correct?

And have you highlighted for us those sections that deal with the chromosome abnormalities?

A. Yeah. As I say, this is not the best literature set for -- for those statements; but, yes, I did.

Q. Okay.

A. At least -- I probably didn't in Steensma. I did not highlight anything in Steensma. I mean --

Q. All right.

A. -- those are sections I found. There may be other sections in those papers.

Q. We are going to mark those three studies --

A. Can I mark them on your papers?

Q. Well, I have got mine as a whole group. I will make sure that the court reporter, or Jeff, copies them and gives them back to you before you take off. Is that fair?

A. Yeah. I -- just annoying because I made a

whole set for you just to prevent this from taking place.

Q. Well, I've had you highlight the set you brought with you.

A. Well, on three papers. I can do that in two seconds on those.

Q. Would you prefer to do that?

A. Yes.

Q. Okay. No problem.

A. They don't come back as fast --

Q. No, I know.

A. You imply they do.

Q. Of course, we've got the world's greatest court reporter here today. That's the only thing Dillard and I agree on.

A. Now I'm getting confused. All right. We will go along with your exercise, only because I don't see one of them in here. So, we will mark these three as -- separately, and please return them as expeditiously as possible.

Q. Yes, sir.

MR. LUBEL: So, we have marked these as Exhibit Number 4; and I am going to go ahead and put a clip on them.

(Exhibit 4 marked.)

Q. (BY MR. LUBEL) And Exhibit Number 4 are the three studies that you have currently in your possession at this deposition that address the chromosome defect issue; and you have highlighted the most significant areas, correct?

A. Yes. I think -- I think they were the most significant areas.

Q. The presentation/PowerPoint that we talked about earlier that you did to fundraise --

A. Uh-huh.

Q. -- how long will it take you to find that?

A. Probably by -- by the weekend, end of the week.

Q. In your materials --

A. And there were two other items that you requested as well, the two E.P.A. documents --

Q. Correct.

A. -- but I will send those to Mr. Kemp.

Q. Great.

Now, will you do your best to locate the John Spencer report that you have in your file materials?

A. I have got it here.

Q. You have got it in front of you?

A. Right. Well, you pulled it out.

Q. Great.

A. Do you want to mark it? This was sent to me from e-mail; so, I know I have a copy.

Q. I'll go ahead and mark it as Exhibit Number 5. Is that okay?

A. Fine.

(Exhibit 5 marked.)

Q. (BY MR. LUBEL) Where in his report are the -- the monitoring results he did from the spiked, new version of Liquid Wrench?

A. The actual results that were measured are in Table 1; and from table -- from the underlying data, there are near-field models of different ventilation rates in Tables 2 and 3. This is monitored results. This is model extrapolation.

Q. Now, during the monitoring test that was done, what was the worker doing?

A. As I understand it, I don't remember specifically what piece of equipment they -- they had, but they had a piece of equipment sitting on a -- on a -- on a counter that he applied different quantities of Liquid Wrench to; and they -- he had standard monitor near his breathing zone, and they took 15-minute and 2-hour samples for benzene analysis.

Q. Okay. Where in Mr. Spencer's report, if you can find the -- the sentence that -- that talks about

there was a piece of equipment on a counter when they ran the test?

A. That's just my recollection from earlier conversations with him, that they had something that they applied -- I don't recall what it was, whether it was a pipe joint or -- or -- it was a piece of metal. It was some -- something metallic that they put the various quantities on and then measured the airborne concentrations.

Q. Does the piece of equipment on the counter, is that referenced in the report?

A. No. I know that from prior discussions with Mr. Spencer.

Q. Now, who was the person that they used to test?

A. I don't know the individual's name. I believe it was one of -- I am presuming it was one of his employees or technicians.

Q. Does it say in that report?

A. No.

Q. Now, how close was the person to this piece of equipment on the counter when the Liquid Wrench in the current version was being applied?

A. I don't know. I presume that's contained in the videotape. It's specifics that I am -- I'm not

aware of. I mean --

Q. What does the report tell us about that?

A. It doesn't provide specifics about the methodology. At least, this one doesn't. I don't know.

Q. What was the breathing rate of the person that they tested?

A. I don't presume that they had him running in place; so, I presume it was a sedentary breathing rate, which is probably 18 per minute or something.

Q. Does the report tell you that?

A. No.

Q. What type of monitor were they using?

A. Total -- I actually don't know if they used the passive dosimeter or charcoal pump; but it was an approved NIOSH-OSHA procedure that he indicated to me was used.

Q. What does the report tell us about that?

A. It doesn't speak to the specifics of the -- of the methodology.

Q. Does the report tell you the -- where the test was run?

A. Where the analysis was run or where the sample was taken?

Q. Where the sample was taken.

A. No, it does not.

Q. Does the report tell you the -- the city that the test was done in?
A. No, it does not.
Q. Does it tell you who was present when the test was run?
A. No, it does not.
Q. Does it tell you when the test was done?
A. No, it does not.
Q. Do you know the answers to any of those questions?
MR. KEMP: Objection, form.
A. I have already answered the -- the questions individually, so...
Q. (BY MR. LUBEL) But from your discussions with Mr. Spencer, do you know the answers to those questions?
MR. KEMP: Objection, form.
A. Some of them.
Q. (BY MR. LUBEL) Which ones?
A. These -- I didn't ask him what city it was in, because I didn't think it relevant; but I -- I know it was done in a corner of a -- a warehouse that they had erected a plastic enclosure around. I don't recall the specific dimensions of that enclosure. And I know it had a work space like a table or platform in it for which the -- the material was applied to something that

was sitting on that work -- or table. That's the extent of the questions that you asked me that I have specific answers to.

Q. And that was information that you got from Mr. Spencer approximately six to eight months ago from a telephone discussion with him, correct?

A. I don't -- I said six to eight months. It could have been three months. It could have been eight months. I -- it was some time ago, totally unrelated to Mr. Cowey.

Q. What was the temperature in this warehouse when this test was being done?

A. I think at one time he told me, but -- but I don't -- I don't recall it now.

Q. What does the report tell you about temperature?

A. It -- the report just summarizes some previous work that Mr. Spencer did. It's -- it's not the analytical report that would back up the numbers that you're talking about.

Q. But does the report tell you what the temperature was when the test was run?

A. No, it doesn't.

Q. What difference does --

A. Well, I don't think it does. I didn't see it.

Q. What difference can temperature have on the results?

A. Well, the higher the temperature, the faster the -- the material would evaporate.

Q. And what difference does that make in a worker's exposures?

A. Only wrote -- most relevant to a short-term exposure. I mean, it's -- most industrial hygiene samples are -- you know, are calculated at the ambient temperature of where -- when the sample was -- was taken. So, I mean, this was obviously an indoor, no air movement; so, you know, it was probably 65- to 75-degree range. But that's all the information it tells you, or can surmise from it.

Q. What happens if the temperature is 92?

A. If the temperature were 92, the 15-minute STEL, which is not reported in this -- in this report, might be higher.

Q. Why is that?

A. Because you -- you'd get a -- materials don't evaporate instantaneously. They evaporate on -- on a curve. And don't ask me what that curve is or looks like; but, you know, the hotter it would be, the faster evaporation would take place. So, in a short-term measurement, you would tend to get a higher number.

Q. So, the hotter it is, you'd expect the exposures to be higher, at least on a short-term basis, correct?

A. Well, this -- this is a very specific scenario. I mean, you have no way of predicting what would happen, you know, standing outside doing this or in a -- or in a garage or workroom with a fan blowing. I mean, there is lots of variables that -- that have potential impact. I mean, what I liked about the Table 1 was the fact that it was essentially no air movement in a -- what you would consider a confined space.

So, by definition, it would be -- you know, at least at the -- and as I say, the numbers in the table are from the 2-hour sample, which probably would not have changed whether it was 90 degrees or 60 degrees; so...

Q. But would you agree on a short-term basis, like a 15-minute interval, that the hotter it is, the higher the exposures would be?

A. If the exposure was on a surface that was the same temperature as the air, I am not an expert, but I would believe that to be the case.

Q. And you --

A. It seems logical to me.

Q. -- you said that this test, to your knowledge, was done in a confined space. Does the report tell you that?

A. I don't see in this report where it specifically states that, although it does speak to the issue of no air movement, which implies some containment, because there's substantially more air movement here than just in this room than what would have been reflected in these numbers.

Q. And what did you say the containment was?

A. They created a structure out of, you know, some -- some contained room out of plastic sheeted -- sheeting.

Q. Did it have a roof on that structure?

A. I don't remember. I mean, I want to say yes; but I actually don't remember.

Q. Does the report tell you?

A. No, it doesn't.

Q. And we don't know how big that -- that room is, correct, from the report?

A. No, not from the report.

Q. Does the report provide any data on what Mr. Cowey would have been exposed to over two hours in a crawl space underneath the house using Liquid Wrench?

A. Well, as I said, I don't -- I don't recall

that was his common usage; but, no, it doesn't speak to that, in and of itself. But if he's in the crawl space putting on this ounce of material and then leaves and comes back the next day, these numbers are still going to be higher than what he would have been exposed to.

Q. I noticed in your report that you had reports from Dr. Irons and Dr. Natelson; is that true?

A. Yes.

Q. Do your opinions differ from theirs?

MR. KEMP: Objection, form.

A. I don't believe they do substantively.

Q. (BY MR. LUBEL) In general, would it be fair to say that you agree with the opinions given by Dr. Irons and Dr. Natelson, at least as they're contained in their reports?

A. I have one question on one of Dr. Irons' numbers that I think he may -- I don't know where he got it from. It's not -- it doesn't match up with the reference he gave for it -- where he speaks about 60 ppm years; but the rest of the material, at least in my reading -- cursory reading of those two reports, I have no argument with their conclusions.

Q. In general, would it be fair to say that the conclusions that you've reached in this case are the same as theirs?

MR. KEMP: Objection, form.

A. I didn't see their -- I didn't see their reports until quite recently; but -- yeah, I think we probably come at it in very different ways, but I think the bottom line is there's -- they're consistent with each other.

Q. (BY MR. LUBEL) How do you come at it different ways?

A. Well, Dr. Irons is, you know, a molecular biologist with a great deal of both diagnostic and study experience on this specific disease; so, his -- his focus tends to come at more of the various case series that he sees. So, you know, in the molecular genetic level and areas like that, he's much more of an expert than I.

I think from the epidemiologic perspective, we're both looking at the same set of literature and, therefore, are consistent; and the same with Dr. Natelson.

Dr. Natelson is a, you know, clinical toxicologist; so, you know, his perspective is also slightly different. But, you know, there's only one body of scientific literature that we're all working from. It's just a matter of which pieces you emphasize relative to your disciplinary expertise.

Q. Irrespective of what your different perspectives and the pieces that you emphasize, would it be fair to say that you-all's conclusions, in general, are the same, with regard to Mr. Cowey?

A. Yeah. I don't think any of us see the possibility of any exposure that would be high enough over sustained periods of time to increase his risk. If that's the basic conclusion, yeah, we -- we agree on that.

Q. Can you pull out for us the studies that you've brought with you that reflect that the Bradford Hill criteria require -- requires a relative risk of greater than 2.0 for a study to be statistically significant?

A. Well, both those statements are -- one -- you're misinterpreting two pieces. The Bradford Hill criteria do not require 2.0. The -- the relative -- the relative risk of standardized mortality ratio of greater than 2.0 is not inherent in the Bradford Hill criteria. Bradford Hill criterion is more generic in that it speaks to strong statistical associations. It does imply that statistical significance of those associations is -- is important.

And the issue of what -- whether the relative risk is 2.0, 3.0, 1.5, deals more with the issue of

relative risks above 2.0 are less likely, or unlikely, to be impacted by simply -- simple confounded exposures. And most of the 2.0 tends to come from academic textbooks and in litigation-related issues, Superior Court opinions; but 2.0, itself, is not a magic number in either epidemiology or in Bradford Hill.

Q. It doesn't come from science, does it?

A. Well, it comes from science to the extent that if you read classic textbooks like Monson's Occupational Epidemiology, you'll see a discussion -- in fact, Monson says 3.0 -- that once -- if you have consistent, relative risks of 2.0 or 3.0 or greater that are statistically significant across studies, even if the studies themselves had not -- for example, if you're looking at lung cancer and there isn't a special handling of the smoking issue, it's unlikely that difference in smoking rates, unless they're enormous, are going to impact that relative risk as a confounder. In other words, on a variable that you have not measured.

So, there is a large body of epidemiologic methodologic report -- reports and textbooks that -- that speak to high relative risks. But that's strength of association. That's just one element in the Bradford Hill criterion.

Q. Have you brought with you any authority that stands for the proposition that you need a 2.0 or greater relative risk for SMR?

A. No. I just told you that's not, in and of itself, a condition of Bradford Hill. There are --

Q. The reason --

A. I have such literature, but it's irrelevant to my report, as far as I'm concerned.

Q. But you don't have it with you today?

A. No.

Q. Is it cited in your report at all, that literature?

A. No. I think I said I -- the Bradford -- one of Bradford Hill's speeches -- and I believe that -- well, that my not be included because it's such a standard piece that you guys all have copies of; so, I tend not to carry all of it around with me -- that talks about strength of association and may give examples, but it doesn't say 2.0 is a -- it doesn't say 2.0 is a magic number. At least, I don't recall it saying that.

Q. In your report you say, "Cancer induction is a multistep process whose clinical onset is typically measured in years, if not decades, from the first time of exposure."

A. As a general statement, yes.

Q. What do you mean by that?

A. Well, traditional thinking -- and I think it's still current thinking -- is that you -- it's a multistep process. You need -- if we're -- if we're talking about -- well, it doesn't matter if we're talking about occupational or nonoccupational cancer. You need a step that is an initiating step, some sets of repeated -- the initiating step is typically thought to be a stochastic process.

That means repeated -- the body is constantly being bombarded with things that will interact with DNA, through diet, everywhere, constantly. The body normally repairs those, and everything's fine. If you have material that is constantly bombarded -- bombarding a target tissue and causing DNA disruption, at some point in time you could -- you would not get the appropriate level of repair and you have an initiating, potentially carcinogenic effect.

What you then need to have happen is -- fortunately the body is a redundant system. Many times those would just -- just be cleaned up by other parts of the organ system. But in -- sometimes they don't; and, as a result, you start to get what might be considered clonal expansion. It starts replicating. This defected cell starts making copies of itself that continue to

survive.

And then you have -- and along the way, there are, depending on which cancer you're talking about, lots of different chemical interactions that are either attempting to control that or promoting that; so, you have an initiation and promotion. And that process takes, depending on the cancer, a fair amount of time; but it's very cancer specific.

Q. You're talking about years?

A. Yeah, typically. Yeah. I mean, I don't think we're seeing -- certainly for environmental cancers, you know, it's very -- other than cancers that are induced through the use of chemotherapeutic agents, you're typically looking at, you know, a minimum of seven to ten years; and then depending on the cancer, 15 or 20.

Q. It's usually decades, is what your report says?

A. Yeah, as a general rule; but that's -- that's -- that's a motherhood and apple pie statement, not a -- not a -- an AML-specific statement.

Q. How -- how does benzene exposure cause MDS?

A. I don't think anybody knows exactly how it causes MDS. I mean, benzene is metabolized first in the liver, and then the various metabolites are further metabolized in -- in the -- in the bone marrow by a

variety of processes. There are several hypothesized metabolites of benzene that are thought to be key components.

The presumption is, is that there is a toxic event, a disruption of the homeostatic process in the bone marrow; and if that process is not abated, you have a -- that cell line is clonally expanded, and you end up with some type of myeloproliferative or lymphoproliferative disorder.

For AML, you -- it's a -- and you really should speak to Rich Irons on this subject. He's published a lot more than anyone else. There are a whole bunch of proteins that are in -- in mol -- and very complex molecular chemistry that support that clonal expansion. But you're looking at -- you know, a decade, and not much more over a decade for the whole process to cycle through once you have bone marrow toxicity going on to AML. I mean, you're looking at 11 years, outside 15 years, based on what we've seen in defined benzene exposed populations where you can actually make those types of calculations.

Q. And is MDS a deadly disease?

A. Depending on which specific form you have, it doesn't have a great prognosis.

Q. What about the form Mr. Cowey has?

A. Yeah, it's -- it typically has survivorship that doesn't go much past five years. I mean --

Q. But how does it kill?

A. -- I don't --

Well, most myelodysplastic or myeloproliferative diseases essentially don't kill you outright. You die of the fact that your body can't defend off other types of infections or disease. So, it kills in a variety of ways because the hematopoietic system isn't operating correctly; and depending on -- on the specifics of that, there is a whole cast -- range of cascading events that can be associated with it.

Q. Do you believe that Mr. Cowey's MDS will ultimately kill him?

A. You know, I don't -- I've not had enough clinical reports available to me to -- it's not something I would normally do anyway; but -- but I don't even have enough material to -- to know what -- what his recent blood counts look like or what his health status currently is.

Q. If his treating doctor, Dr. Youman, says that his life expectancy from his MDS is about six months to 18 months, would you agree or disagree with that?

A. From his current status?

Q. Correct.

A. Because, I mean, he's obviously lived longer than that since his diagnosis.

No, I would have no reason to dispute that. I have no data to dispute it with.

Q. Now, the Bradford Hill criteria require -- or one of the criteria of Bradford Hill is dose response between the agent and the disease; is that true?

A. Yes. That's why I said MDS in particular really doesn't, in the formal sense, satisfy the Bradford Hill criteria. We don't have studies that really deal with MDS or any of the subtypes in that regard.

Q. But you agree that benzene exposure can lead to MDS?

A. Yes.

Q. Can you tell us why it is that your affidavit that you gave says that benzene exposure does not cause MDS?

MR. KEMP: Objection, form.

A. It doesn't say that; so, that's -- I don't know where it is. That's a missreading. It doesn't say that.

What I say when I am discussing causality is that -- has been repeated AML -- a causal association between benzene and MDS has not -- has not -- and I use

Cole as the reference, what I'm -- what I'm specifically speaking to is general causation under the Bradford Hill I don't dispute, nor -- nor meant to imply, that -- that -- that benzene doesn't cause.

Q. (BY MR. LUBEL) But what does the affidavit say in that regard, if you can read that sentence for us?

A. Well, it -- essentially literally paraphrasing, I might even be quoting, Phil Cole's 1993 formal review of the Hill criteria as they apply to -- to a causal association between benzene and MDS has not -- has not, and then I cite Cole.

Q. Has not what? Just read that sentence for us.

A. (Reading) While a causal relationship has been established between repeated high doses of benzene and AML, a causal association between benzene and MDS has not, in the general sense of the Bradford Hill criteria.

And we talked about that earlier. I don't -- you -- you don't -- you can't show me a study where we have dose-response data specific for MDS in a group of -- in a set of -- in a study that we would all agree is suitable for risk assessment. That's what that says.

Q. That's what you mean? I mean, it doesn't say that.

A. Well, that's what it -- that's what it means.

I mean, it then goes on to discuss if -- if benzene, indeed, does cause MDS, this is how we have to look at it, by looking at the leukemia literature.

Q. You make a statement that "Scientific thinking today believes that bone marrow toxicity is a precursor event necessary for initiation of the AML process."

A. Yes. That's -- I do. That -- I think you just read the statement.

Q. Do you have a study with you that states that?

A. No, but I -- I didn't -- I didn't put a reference on it because I think there's a -- a fairly general consensus that you need some hematotoxicity as part of the initiating process. There are references out there. I don't -- I have not gone back and dragged that material out because most people generally accept that there is some truth to it.

Actually, what you would want to look at is some of Snyder's work, Bob Snyder from Rutgers, and probably -- well, I know for sure, although I don't remember the papers per se, I believe Rich Irons has also published in that area.

But most -- most of the folks that are doing molecular biology of -- of -- of aplastic anemia and leukemia recognize that there is a high probability, if

not a demonstrated requirement, for some toxicity. If there wasn't, there would be no point in doing medical surveillance for benzene-exposed workers.

Q. When was there published information available to companies that were interested in learning about the potential health hazards of benzene exposures concerning potential adverse health effects from it?

A. Well, you can go quite far back in time. I mean, it's -- the -- you know, the companies would tend to look at National Safety Council records and the American Conference of Governmental Industrial Hygienists, both of which have been reporting on benzene toxicity since the late '40s; and there's literature earlier than that, but when you talk about what companies would have reason to be knowledgeable about, the A.C.G.H. [sic] has a -- you know, put out a T.L.V., in '46 or so.

Q. 1946?

A. Sometime in that time range.

Q. And by benzene toxicity, are you referring to damage to the blood or blood-forming organs?

A. Well, the big concern up until the '70s was aplastic anemia. All the focus of toxicity concern was related to -- to bone marrow suppression and overt aplastic anemia, which is a -- a very serious,

frequently fatal, disease.

You'll also find -- I mean, at one point in the -- in the late '20s and early '30s, benzene was used to treat leukemia, because if you gave somebody -- injected them with a high -- a high dose of benzene, you killed the bone marrow. So, the thought was that if we could induce an aplastic state, we -- we would kill -- we'd reset the clock, so to speak. I mean, obviously they died -- many died of aplastic anemia as a consequence of the treatment; but...

But, no, the -- the toxicity has been known for a very long time; and that's what -- and if you look at the history of how the T.L.V.s have changed, like -- and I believe it was -- it was '40 -- it might have been '49, but '40 -- in that time range -- it was 100, then they lowered it to 50, then they lowered it to 35, and then sat with it at 25 for a very long time.

Q. My question is: When you referred to bone marrow toxicity, were you including damage to the blood-forming organs?

A. Well, it was mostly -- I mean, that's what aplastic anemia is, damage to the blood-forming organs. I mean, in its most severe state. So -- so, the -- so -- so that the -- that early literature was always looking at what -- what are levels that don't induce

hematotoxicity, however you want to measure that.

But the cancer issue, you know, the leukemia issue, didn't -- didn't really surface until -- I mean, you started seeing -- seeing dribs and drabs in the early '70s. I don't think anyone accepted general causation -- I mean, the general causation that benzene could cause leukemia probably didn't really arise until -- or get broad acceptance until the mid '70s or a little later.

Q. Were you aware that the American Petroleum Institute study that was authored in 1948 referenced leukemia?

A. The A.P.I. never did a study in 1948.

Q. You weren't aware of Dr. Drinker's study that the A.P.I. sponsored?

A. Are you talking about his -- his -- his 1948 summary of -- of the toxicity of Benzene? That's not a study. That's a review article.

Q. Are you familiar with that article?

A. Sure.

Q. Were you aware that that article discussed leukemia and benzene?

A. I am -- I mean, there have been case reports out there; but no -- no one accepted benzene as a cause of leukemia. I mean, if you go to the NIOSH 1974

document, it states quite clearly that while the -- there have been reports in the literature, the possibility of benzene causing leukemia needs to be further studied.

So, I mean, you will find reports in literature of people who had insights, based on either clinical experience or case -- case experience; but I know it wasn't -- I know that was not a generally accepted scientific assessment.

Q. Well, my question simply was: Were you aware that the A.P.I. 1948 article referenced benzene and leukemia?

A. I know it made some mention of leukemia, but I -- but I believe the primary discussion in the later -- I mean, the document also says 50 ppm of benzene is acceptable. And then I think there is probably a 1960 document of a very similar writing style that says if you take an A.P.I. document that says if you -- written by another outside expert that says if you take 60 milligrams of vitamin C daily, you will protect from benzene toxicity. I mean these are state-of-the-art documents that are not, by our thinking today, correct. So...

Q. Well, was it firmly established in the late 1940s that benzene exposures could cause blood-forming

disorders?

A. Toxicity. Always toxicity.

Q. They don't refer to the blood?

A. Yeah. I said hematotoxicity. Tox -- benzene killed stem cells and bone marrow cells, resulting in disruption of the blood-forming organs. But -- but the disease leukemia was not presumed to be directly caused by benzene. It just wasn't the case. I mean, yes, there were case reports. I am not saying that there weren't individual reports in the literature of individuals with leukemia who had benzene exposure; but there were lots of reports -- case reports of different things that never panned out, ever, either.

So, what I am speaking to are, you know, things like the National Safety Council reports of '50 and others that are speaking to hematotoxicity; and that's -- and if you read the A.C.G.I.H. documentation, which is probably a pretty good historical account of what a group of folks dedicated to looking at safe exposure limits, if you look at that over time -- and I think that I -- did I include that in this report or not? But that speaks quite clearly to the issue that leukemia is not -- is not even on the radar screen until you're well into the '70s.

Q. I am not asking you about leukemia. I am just

asking you if in the late '40s it was firmly established that benzene exposures could damage the blood-forming organs?

MR. KEMP: Objection, form.

A. I've said yes to that several times. That's what aplastic anemia is, and that's what anemia is -- toxic anemia is, damage to the blood-forming organs and the results therefrom.

Q. (BY MR. LUBEL) Do you have an opinion as to whether Mr. Cowey's smoking caused his MDS?

A. Based on my -- what he says in his deposition, his implication is that he did not smoke very much at all; and while smoking is strongly associated with acute myelogenous leukemia, it tends to be in heavy smokers. So, I don't see it as a risk factor for Mr. Cowey.

Q. So, smoking didn't cause his MDS?

A. If he -- if he's accurately reporting his smoking history, I don't believe so.

Q. Did ionizing radiation cause his MDS?

A. I attempted to get as much detail on his early treatment of -- for his prostate cancer. Best as I can judge, he did not have any either radionuclide or radiographic or radiation treatments. So, my conclusion is he did not have any exposures that could be associated with it, based on the data I have seen.

Q. Did any drugs cause Mr. Cowey's MDS, to your knowledge?

A. Well, that's a good question. But I don't have any drug history for Mr. Cowey; so, I can't make this -- I can't draw any opinion on that.

Q. Did any chemotherapy treatments cause his MDS?

A. Similar to the radiation issue, I could not locate any objective evidence that said he had chemotherapy for treatment of his cancer; so, I can't offer any opinion one way or the other.

Q. Did hair dyes cause his MDS?

A. I don't think hair dyes are -- have been established causally uniformly, but there is no evidence in the -- in the record to suggest that he dyed his hair regularly; so, I -- I mean, in my discussion of what -- what's been thrown out there, I list some of those things; but certainly I don't -- in my conclusionary remarks, I don't reiterate that I think they have any role.

Q. Did -- did pesticides cause Mr. Cowey's MDS?

A. That's an interesting possibility, because although we have no specific information on what pesticides were being used, I do recall at least in a couple of places that he was present during what would be very heavy exposures through air distribution of

pesticides; but I don't have any objective information that would permit me to draw even a partial inference on that, but it's certainly a possibility.

Q. As you sit here today, you can't say that pesticides played any role in his MDS?

A. No. I don't have enough information.

Q. Did farming cause his MDS?

A. Once again, similar to the pesticide story. It's -- it's an actively -- it's a research objective that's actively being researched. Right now there is not sufficient information to point to a specific form of farming or specific exposures within farmers; so, I can't, based on current evidence, derive a conclusion there.

Q. Will you agree that Mr. Cowey used benzene-containing Liquid Wrench over 26 years?

A. No, I would not agree with that. I don't see that in the record being established.

Q. If that is established, could that change your opinions one way or the other?

A. No, because, actually, my -- my opinion probably goes to the -- goes to the -- even if that were a true scenario, using an ounce of it periodically, particularly under the conditions that Mr. Cowey did, because it sounded like he actually was a responsible

user of the products he used, there would be no way he could get -- get an exposure high enough.

Q. But you're basing that on John Spencer's exposure data, correct?

A. No. No, not at all. I mean, I know enough about Liquid Wrench -- I mean, John Spencer's stuff is sort of like icing on the cake. It's nice to have because it sort of confirms what I already believed. But, you know, we've -- I am very familiar with the material he's using; and I am very familiar on how it was used. It's been used widely in -- you know, inside the petroleum industry for decades. We've -- you know, maintenance workers have been measured for benzene routinely; and Liquid Wrench, although, you know, it's an exposure that we would have, you know, reduced, and did reduce as soon as we were aware of it, there is no evidence that you could get exposures high enough to warrant -- to induce a disease from it.

Q. So, you don't need quantitative data on Mr. Cowey's benzene exposures to reach your conclusions, correct?

A. I'd very much like to have it. And as we discussed at some length earlier, I can render an opinion on an exclusionary argument that there's just no reasonable expectation that you could reach an exposure

threshold. I am not saying that he wasn't exposed to benzene. I am just saying that I can't see any scenario that would have given him, from Liquid Wrench, a high enough exposure.

Q. But you didn't need quantitative data to reach that conclusion?

A. Well, I'm very familiar with benzene and benzene literature and what types of workers are exposed; and, I mean, I -- I am not a novice about petroleum -- you know, petroleum refinery workers and the opportunities for benzene exposure. I -- I can see in this particular case that this infrequent and, in my estimation, still probably relatively trivial household use just doesn't get there. So, I mean, I don't need a number to -- to confirm it.

As I said, if we were closer to the 200 p -- if I could envision a scenario that gave -- you know, gave me higher concentrations regularly, then I would probably, you know, be more adamant about wanting a quantitative exposure assessment.

Q. How regularly?

A. Well, I mean, he's not even -- you know, he's not doing this daily. That's for sure. He's not. There's just no possibility in my mind that -- that this man has been daily undoing rusted -- rusted pipes and

bolts. I mean, it just doesn't have prime fascia face validity of any truth to it. I mean, this was not his occupation. He was not a mechanic in a petrochemical plant. And even there, I am not at all convinced that there is even any daily use. So, I mean, it just -- just doesn't cut it for me.

And, as I say, I mean -- you know, it is nice to have Spencer's stuff because it sort of confirms what I already believed.

Q. Have the lawyers for United States Steel Corporation provided you with the Mobil documents where they tested Liquid Wrench and the benzene content of it?

A. Oh, yes. As a former Mobil employee, they really made sure I saw those.

Q. Did you know the employees that were in those documents?

A. Yes.

Q. Were they good hands?

A. Well, one -- one said -- came from a refinery. I mean, there was the refinery environmental or safety manager -- I don't exactly recall his title -- that did -- that did the original -- or at least sponsored the original analysis somewhere in the refinery. I don't know where it was done. And -- and I know he was a well-meaning guy. He was doing exactly what he was

supposed to be doing.

The -- the replication of the work, as you see in the other Mobil document, which unfortunately is not an exact replication -- well, the same sample wasn't tested in both labs. One sample was tested down in Beaumont, and then another sample acquired and tested in Princeton -- I think it was Princeton, New Jersey; and that one only showed 7 percent. So, I mean, there -- there's still, in my mind, some reasonable belief that there may have been some error in the 30.

But fundamentally, even at the 7 percent, you know, if we had suitable alternative, you know, products, that those documents outline very clearly what we were doing in that time period.

Q. Do you have any question of the results reached by the Mobil employees that apparently you knew that authored these documents on Liquid Wrench?

MR. KEMP: Objection, form.

A. Well, they did not -- just to clarify, those gentlemen were manager -- you know, business managers. They did not do the analysis. They were just reporting analysis. So, I don't -- I don't know anything about who did the analysis. I know that we had -- Mobil technical services in Paulsboro and Princeton had superb analytic capability to measure benzene in a mixture.

So, the confirmatory -- what I would call confirmatory results are ones that I believe quite strongly. I mean, obviously the sample they had at 7 percent.

And the 30, I have no way of knowing if that's an accurate analysis or not. I mean, it doesn't comport with any of the internal documents that -- that either U.S. Steel in what they were sending out in their raffinates would suggest. So, I have no way of knowing if that 30 is correct. I am suspicious that it's not.

Q. (BY MR. LUBEL) And have you considered this Mobil --

A. But let me also go further --

Q. -- data --

A. -- and say that if it contained 30, it would have fallen into a whole different labeling requirement. I mean, it would have had probably a skull and crossbones, or something else, on it as well. And I -- I just don't think it had 30.

MR. KEMP: Objection, nonresponsive.

Q. (BY MR. LUBEL) Have you considered the Mobil data in your analysis in this case?

A. Well, clearly, I -- in trying to come up with my worst case scenarios, I was willing to accept that 30 could be a possibility. I mean, I have no reason --

THE REPORTER: Could you excuse me for

just a second?

(Brief Pause.)

(Requested testimony read back.)

A. I mean, I have no objective reason to throw the 30 away, only the extent that it -- the analytic chemistry coming out of the -- the raw material going in never seemed to show a number that high. So -- so, I used, in my mind, the 30 as the worst case scenario. And still, you know, even if it were pure benzene, you know, what I would consider what you would use, the, quite honestly, few drops, not ounces, that you use when you apply Liquid Wrench, I mean, that's why they sold it in, what, 4- or 8-ounce containers typically. So, I mean...

Q. (BY MR. LUBEL) Did you --

A. I think I answered your question.

Q. Did you, based upon considering the Mobil data, assume that the most likely benzene content of the Liquid Wrench was 7 percent as opposed to 30 percent?

A. Well, remember, the approach I was taking was an exclusionary approach. So, it -- if I couldn't come up with a high exposure scenario at 30, obviously it was going to be lower at 7; and obviously, you know -- so -- I mean, the -- the raffinate documents say 1 to 14, average 3. Some other documents said 7. The Mobil

documents said 7. I mean, you have quite a range of potentially -- of benzene in the raffinate. That gets cut back in the actual formulation a little bit because there are other oils added in the final product; but in my mind, if I couldn't come up with a scenario that made it particularly hazardous at 30, it didn't matter to me whether it was 7, 6, 5, 29, 28, 24.

Q. But you took the Mobil information into account when you assessed your opinions?

A. Absolutely, because I would have never considered 30 as a possibility. I mean, I would have gone by the analytic chemistry coming from the supplier.

Q. Who were the companies that funded the approximately 26 million-dollar Shanghai consortium studies?

A. I don't know who all are in there. I know probably the top five petroleum companies are part of the group -- Mobil, Exxon, Chevron, Shell, BP -- if I had to guess. I don't know exactly. But I don't know what share -- you know, how much of that total comes from them. Well, it -- first of all, it doesn't all come in -- I mean, that's over the whole -- I think they're anticipating it to be an eight- to ten-year study -- set of studies. So, it's not a single check being written today.

Q. When you went and made your PowerPoint presentation to the companies, which companies do you remember talking to?

A. Well, when I was trying to go ahead -- go -- the budget estimates were way more -- were much more modest. I mean, I think we were looking at, you know, outside numbers of 6 to 8 million; and at the time I was trying to get the American Petroleum Institute to put it as a budget line item, in which case every petroleum company in the United States would have paid their dues -- proportional dues share. So, in my era, it -- it was really to try -- try to sell it to get a vote at A.P.I. to -- to budget the project.

As I understand it, that didn't -- they were unwilling -- I mean, some of this happened after I departed; so, it's hearsay -- A.P.I. elected not to put it as a budget line item, and individual companies were -- I think it became a subscription type of activity.

Q. What were you doing for Mobil back during the time period in 1977 when they were looking at the benzene content of Liquid Wrench?

A. October -- I came on board October, '77. I was -- which is right when the emergency temporary standard came out. I was part of the medical department. I was the medical department representative

to all of the various committees trying to figure out what do we do with this emergency temporary standard. So, I sat in on lots of meetings. My role then was advisory, as -- as an adviser, not as a -- not as a decision maker.

Q. Did you participate in Mobil's decision to take Liquid Wrench out of their complexes?

A. No, not directly. I mean, I -- I -- I actually don't remember the dates. I am pretty sure I was aware of -- I mean, looking at those documents, I have awareness of the -- of those kinds of issues. So, I probably sat in on some of the discussion around that; but --

Q. Do you recall offering any opinions or providing any input in the -- Mobil's decision to not let their workers use Liquid Wrench?

A. Well, as I recall, it was a recommendation. I don't actually -- I don't actually recall seeing any formal directives. We have a tendency to put things in directives when -- when it's a "thou shalt" issue. But it was normal practice for us to -- if we became aware of toxicity -- of a toxicity issue, that at -- that we didn't have firsthand knowledge of or could directly do a risk assessment on, if we had substitute materials readily available, they'd swap those out and then sort

it out over time. In other words, if it turned out that, you know, this is much ado about nothing, then, you know, sort of back off of the early procedures.

Q. Did Mobil continue to use Liquid Wrench after 1977 when they were looking at the benzene content of Liquid Wrench?

A. I don't know. I don't know operational details at that level routinely. I was at headquarters.

Q. You said you sat in on some meetings on it, though.

A. Yeah. They would have -- they would have been -- the environmental toxicology and medical department met quite regularly to keep each other informed of what's surfacing on each other's plate; so, we would have been aware of it and -- and would have not weighed in heavily, for -- I mean, you know, sitting in the medical department in New York, if the substance was Liquid Wrench, I'd use it. It's not exactly rocket science from a decision point of view.

Q. What did Mobil ultimately decide to do about Liquid Wrench that contained benzene?

A. I don't know. I think I already answered that question. At the operational level, I don't know what the -- what the final resolution was.

Q. When you refer to "alkylating agents," are you

referring to chemotherapy treatments?

A. Generally, yeah, in the context we were discussing here.

Q. And you're not saying that any alkylating --

A. No.

Q. -- agents caused his MDS, are you?

A. No. No.

Q. And then there is another word that you use in your report, and I can't pronounce it. It's epipodophyllotoxins, or something like that.

A. Yeah, I found that quite interesting. That -- I did not spend much time trying to go to primary references, but that comes out of Cancer Society summaries of the literature. It was something that -- it's on my list of interesting things to find out more about, but --

Q. What are they?

A. I don't know. That's why I wanted to spend more time and find out.

Q. How do you pronounce it?

A. Oh, I don't know.

Q. Nobody knows?

A. Nobody knows. Nobody knows.

Q. Well, you're not taking the position that that caused Mr. Cowey's MDS, are you?

A. No. I mean, I routinely, in my reports, list -- list materials of that type that -- that are recognized by others as having strong, if not causally, associations; and the reason I do that is sometimes subs -- as much to inform other parties because, as they're going through additional materials or new discovery, you know, if you never mentioned it, nobody will know that it's important; so...

If I thought it was important specific to Mr. Cowey, it would be -- my report style would be that it would be rediscussed in my conclusionary remarks as being potentially associated.

Q. Have you -- have you told us about all of your opinions regarding Mr. Cowey's case?

A. Yes, I believe.

MR. KEMP: Objection, form.

Q. (BY MR. LUBEL) Does your report -- between your report and what you've told us today cover your opinions in this case?

A. Unless I am given additional information, I think it summarizes, certainly, the current status of my thinking.

MR. LUBEL: Can we take a short break? And I think I am getting close.

MR. KEMP: Yes, sir.

(A break was taken, 1:54 to 2:00 p.m.)

(Exhibit 6 marked.)

Q. (BY MR. LUBEL) Can you identify Exhibit 6 for us?

A. Exhibit 6 is correspondence between Fulbright & Jaworski and myself, and it is -- it's complete except for, I guess, one bill that I sent in that doesn't seem to be here; but that's --

Q. What was that bill for?

A. For writing the report.

Q. How much did that cost?

A. I actually don't remember. I don't know, 8, $10,000, something like that.

Q. Can you go ahead and provide to Mr. Kemp all of your invoices?

A. Well, he has the invoice. He hasn't paid it.

MR. KEMP: We paid the retainer, and we take some time to pay the invoice.

Q. (BY MR. LUBEL) Who hired you in this case?

MR. KEMP: We're good for it.

A. Mr. Kemp. Mr. Kemp, I believe, made the initial contact.

Q. (BY MR. LUBEL) On behalf of United States Steel Corporation?

A. Yes, I believe that's correct. I normally

don't pay attention to those kind of things.

Q. In this case, have you talked to anybody other than Mr. Kemp?

A. Yes. I -- I had a few conversations with Mr. Dillard.

Q. Anybody else?

A. Mr. Epps. Carl Epps.

Q. What does Mr. Epps have to do with it?

A. I believe he is associated with either Radiator or U.S. Steel. I mean, I am -- I actually am not 100 percent sure at times.

Q. When did you learn that you were also hired as an expert for Radiator Specialty Company?

A. I don't -- I get hired by a law firm; and, quite frankly, I am not -- I don't pay a whole heck of a lot of attention to who all else -- who all is paying the tab, because I am looking at risk assessments and evaluation of the epidemiology on a particular case. So, when you ask me a date like that, it makes it sound like that was sometime after Mr. Kemp retained me.

Q. Were you aware that Radiator Specialty Company is paying half your bill in this case?

A. Now that you say it, I think I probably am; but I don't recall if I got two checks on the retainer or not.

Q. Well, irrespective of who sent you the first check, as you sit here today, do you understand that you're an expert for both United States Steel Corporation and Radiator Specialty Company?

A. As it applies to the epidemiology we're talking about here? Yes, I guess so.

Q. Well, the reason I ask you this is because in the correspondence between you and United States Steel Corporation's attorneys, there's a reference to half of your bill will be paid by counsel for Defendant Radiator Specialty.

A. Then, I don't -- I just don't remember that. But -- I mean, I don't know -- to me, all that means is that they're paying half the bill; so...

Q. Well, have you talked to Mr. Riley at all?

A. No. I mean, spoken with Mr. Riley regarding this case?

Q. Right.

A. This is the first time I met Mr. Riley. Today.

Q. Was that unusual for you to receive half your payment from a company when you hadn't met their lawyer or talked to them?

A. No, not at all. I mean, there are -- I don't need, nor do I particularly want to know, how you guys

run your business. But there have been occasions where there's either been, quote/unquote, joint defense funds, and there have been cases -- in which case I get a check written from some artificially created fund; and there are times when I send in a bill and I get six -- you know, one-sixth checks coming back and in no particular logical time sequence to cover that invoice.

So, while it might be important to you, it's not an important issue to me. I mean, I am retained by a law firm to evaluate a case. How they pay me is an issue for them, not for me.

Q. So, nobody from Radiator Specialty Company, to your knowledge, has contacted you to discuss this case as it applies to them?

A. Well, in the course of conversations with Mr. Epps, I mean, I think, you know, we have covered a lot of Radiator documents; but -- and, you know, that -- this is not the first, quote/unquote, Liquid Wrench-related case I have ever worked on. But I don't make a big -- I don't distinguish -- no, I -- to my knowledge, I have not spoken with anybody specifically from -- from Radiator.

Q. But you have got a written contract with Jeff Kemp at Fulbright & Jaworski, the attorney for United States Steel Corporation, specifically for the Cowey

case, correct?

A. Well, you -- I mean, I now know that it is for -- for United States Steel Company; but if you will notice, my agreement is between me and Fulbright & Jaworski.

Q. Right.

Why don't you have an agreement with Radiator Specialty Company?

A. Because they've agreed to -- they -- they've retained me to look at this case, and how they get their money back is not a concern to me.

Q. But they don't say they're going to get their money back from Radiator. They say that half the money is going to --

A. Well, that's what he's telling me; but the agreement that they signed says that they're going to pay me. Okay?

Q. Who is "they"?

A. Well --

Q. Fulbright?

A. -- Fulbright & Jaworski is going to see that I get paid at this rate for my work on this case. I don't care if they solicit on the corner for people who want to contribute to the payment fund.

Q. Well, where is the -- your check --

A. You're making me want to know a lot more about this than I historically ever wanted to know, but --

Q. Where is your -- the correspondence that reflects checks you've received for the other half of the monies from counsel for Radiator Specialty Company?

A. I don't even recall if I got two checks. I don't know that for a fact. Oh, I guess -- I guess I must have, if that's what he said. So, I must have received a half check...

I didn't bring any of this material -- I mean, other than a couple e-mails that I actually had in my possession, I did not bring my billing records for this case. So, I -- my set of records is not here; so, I must have something back in the office that has -- I mean, I don't even know whether there was correspondence attached to it. I do recall that the retainer has been paid; so, I presume that I have received two checks for $1400. But that's sort of the extent of my interest in the -- in the matter.

Q. Well, as you sit here today, do you know whether or not you have received any money from Radiator Specialty Company or their lawyers for this case?

A. I know the retainer has been paid; so, I have obviously received two checks for $1400. I don't recall whether -- I mean, very often I get a check that just

hasn't -- has a -- a name on it, has no correspondence; so I am -- I am not trying to be evasive. I just -- I just don't spend any time on that subject matter.

Q. Well, who owns Health Risk Services, Incorporated?

A. Health Risk Sciences.

Q. Who owns that?

A. I do.

Q. How long have you owned it?

A. Since its -- since it was created in 1999.

Q. Are there any co-owners?

A. No. Well, I don't know if my wife -- I don't remember how --

Q. Other than your spouse?

A. I don't know how that works, but --

Q. Other than your spouse?

A. No.

Q. And what is the business of that company?

A. Health Risk Sciences is a -- a -- specializes in the conduct of epidemiology and integrating epidemiology with industrial hygiene toxicology in conducting health risk assessments; so, we do a -- a variety of things. I mean, some litigation-related work, some cluster investigation, some epidemiology, some general consulting.

Q. And what percentage of your work is done on behalf of plaintiffs?

A. That varies dramatically year to year. This year -- oh, plaintiff -- you said plaintiffs?

Q. I did.

A. Good catch. I have not been retained by a plaintiff. For some reason, they don't knock on my door.

Q. So, would it be fair to say that 100 percent of your income, at least in the litigation context, is -- comes from defendants?

A. In the litigation context, to date, that's correct.

Q. And what percentage of the total income that your company makes comes from oil companies, chemical companies, industry of that sort?

A. That's what I was starting to answer. I mean, it varies dramatically year to year. Probably 80 to 90 percent comes from the petrochemical industry; and then there's a -- a mix of trade associations. I have occasionally done some work for health departments. I mean, it's a -- the rest is a mix of different -- different groups.

Q. Which trade associations?

A. The only trade association I am currently

working for is the asphalt industry. As -- as -- Asphalt Institute, I guess it's called.

Q. Have you done any work for the American Petroleum Institute?

A. Not since I retired.

Q. How about the Chemical Manufacturers Association?

A. Not since I retired.

Q. What are you doing for the Asphalt Institute or organization?

A. They're interested in conducting a large U.S. epidemiology study, and they have -- and it will be done in conjunction with the American Pavers Association, which is another association of the Pavers, as opposed to the manufacturer; and they've asked me to sit on -- in on all their planning meetings to help guide them to make sure that whatever they do turns out to be a good piece of science.

Q. What are they trying to learn from this study?

A. There was a -- some preliminary reports coming out of Europe where coal-tar pitch is -- is -- was a -- a high component of asphalt for many years, suggesting that there may be a lung cancer excess in pavers in Europe.

And so, they have spent a fair amount of time

on toxicity and on related chemistry of the U.S. paving industry and are looking now to reconstruct historical chemistry and formulation information. We're working with some folks from Harvard and a few other groups to look at the feasibility of could we do a -- in this country could you do a good paving study -- a study of asphalt pavers.

Q. Why isn't the study from Europe valid for the United States?

A. Because the formulation history, at least it's commonly viewed in the U.S., used much less coal-tar; and there is common agreement that the coal-tar pitch is the car -- the high carcinogenic component in asphalt. So, if -- if the U.S. chemistry history is correct, which you could confirm in an epidemiology study, you would not expect to see even a hint of lung -- I mean, there is still ongoing studies in Europe; so, I mean it's not that it's been established yet, but --

Q. What about bladder and kidney cancers?

A. No, not really. The -- World Health Organization/IARC just released a five-country cohort study, and the only questionable cancer site is -- is lung. Kidney and bladder are fine. The -- you don't get enough -- the PNA exposures just aren't high enough. I mean, it's not like coal -- you know, it's not like

coke oven workers or any of those areas where you might expect to see those high exposures.

Q. And would benzene -- benzo(a)pyrene be the component of concern in the coal-tar pitch?

A. It's probably a leading component. I mean, there is a whole range of PAHs and a few nitroso-PA compounds that have similar action. Coal-tar pitch is a pretty dirty dimish. They typically use benzo(a)pyrene as an indicator marker for it. But, yeah, it certainly is at the top of the list; but it's not the only PAH.

Q. Where did you retire from?

A. I retired from Mobil when Exxon acquired Mobil Corporation.

Q. And why is that?

A. Well, Mobil had a -- a retirement plan that you could actually retire at age 50. At the time I was 52. Because Exxon was taking over Mobil, Mobil had adopted a poison pill for its executives; and I was -- I was director of health risk assessment at that point, and I was high enough to be able to get a several -- several-year severance on top of an early retirement, on top of all the options being fully -- fully vested. So, when you sat down and ran the numbers out, unless I really wanted to work for Exxon, there was no good reason to stay.

Q. Was that in 1999 that you retired?

A. I actually stayed on into the early part of 2000 to do what we call "transition" work. We have a lot of confidential epidemiology medical records that had to be transitioned legally -- you know, I had to -- I had signed confidential documents for various state and health departments; so, we had to transition all that stuff so that I could transfer those documents, because I sure as heck didn't want to take them home.

Q. So, you started your company, Health -- Health Risk Sciences, Incorporated, before you left Mobil?

A. Yeah, because I had -- actually didn't know how long they -- how long the transition was going to go. I mean, it didn't -- it didn't -- when we say created the company, I mean, it was a set of legal activities. It was not the -- that there was a shingle hung on the door.

Q. And have you had a stable group of petrochemical companies that have been clients of yours since you started?

A. Well, I mean, you can see from my C.V., I'm widely published in certain areas of petroleum-related exposures; so, companies come to me when they have -- think they might have a problem. I mean, I have published on how to look at cluster investigations, a

variety -- wide variety of stuff. So, if they think there might be a problem, they will come to me and ask for advice.

I have done some work in Kazakhstan in 2000 through 2002, looking at environmental epidemiology where there -- they had set aside a large amount of money to build infrastructure in some of these rural Kazak villages and needed help to try to figure out how -- what was the best place to put the money kind of thing. So, it's quite a variety.

They tend to be petrochemical companies, but they're not -- it's not like -- I am not on a retainer for any company. They come to me on not as -- on an ad hoc basis when they have a problem.

MR. LUBEL: That's all I have. Thank you for your time.

THE WITNESS: Thank you.

MR. KEMP: I'll reserve my questions until the time of trial.

MR. RILEY: Same.

CHANGES AND SIGNATURE

PAGELINE	CHANGE	REASON

I, GERHARD K. RAABE, Dr.P.H., F.A.C.E., have read the foregoing deposition and hereby affix my signature that same is true and correct, except as noted above.

_____
GERHARD K. RAABE, Dr.P.H., F.A.C.E.

THE STATE OF _____)
COUNTY OF _____)

Before me, _____, on this day personally appeared GERHARD K. RAABE, Dr.P.H., F.A.C.E., 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: _____

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 GERHARD K. RAABE, Dr.P.H., F.A.C.E.

DECEMBER 2, 2003

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

That the witness, GERHARD K. RAABE, Dr.P.H., F.A.C.E., 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. LANCE LUBEL.....04 HOURS:17 MINUTE(S)

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.

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.

_____

Kathy Miller
Texas CSR No. 739
Expiration Date: 12/31/04

Nell McCallum & Associates
Firm Registration No. 243

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.

_____

Kathy Miller
Texas CSR No. 739
Expiration Date: 12/31/04

Nell McCallum & Associates
Firm Registration No. 243