CAUSE NO. 08-CV-0161

MARY ELLEN HALL, INDIVIDUALLY AND AS REPRESENTATIVE OF THE ESTATE OF JOHN NEAL HALL, DECEASED, JOHN EMERY HALL AND LISA ANN EASLEY, Plaintiff(s),	)	IN THE DISTRICT COURT OF
V.	)	GALVESTON COUNTY, TEXAS
RADIATOR SPECIALTY COMPANY, ET AL, Defendant(s).	)	56TH JUDICIAL DISTRICT

****

ORAL DEPOSITION OF

ETHAN A. NATELSON, M.D.

JANUARY 18, 2010

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ORAL DEPOSITION of ETHAN A. NATELSON, M.D., produced as a witness at the instance of the Plaintiffs, and duly sworn, was taken in the above-styled and numbered cause on the 18th of January, 2010, from 1:34 p.m. to 4:43 p.m., before Wendi Broberg, CSR in and for the State of Texas, reported by machine shorthand, at the Law Offices of Baker Botts, L.L.P., One Shell Plaza, 910 Louisiana, Suite 3200, Houston, Texas 77002, pursuant to the Texas Rules of Civil Procedure.

INDEX

INDEX OF EXHIBITS

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ETHAN A. NATELSON, M.D.,

having being first duly sworn, testified as follows:

EXAMINATION

BY MR. LUBEL:

Q Please state your full name.

A Ethan A. Natelson.

Q Have you been hired as a witness on behalf of the defendants in this lawsuit?

A Yes.

(Exhibit 2 marked)

Q (By Mr. Lubel) Is Exhibit No. 1 out of your file a copy of your report that contains your opinions in this lawsuit?

A Yes.

Q Does your report provide the support for your opinions?

A I think so.

Q Are you prepared to discuss that in detail today?

A Yes.

Q Are there any opinions that are not contained in your report that you intend to offer as you know it today?

A Well, I can't anticipate what you're going to ask me, but I feel that the references I have support

the opinions I give in the paper.

Q I guess what I'm trying to clarify is as you sit here right not knowing what I intend to ask you, are you aware of any opinions that are not addressed in your report that you intend to offer in the case?

A Well, it's the same answer. In other words, I -- if I'm asked something, I'll answer it to the best of my ability. It may be peripheral to the case or it may be central to the case and without knowing what it is I'm going to be asked, I can't tell you that every question you might ask is contained in that letter.

Q What caused Mr. O'Neal's death?

A He --

MR. HUMPHREY: Excuse me. Form. Mr. Hall's death. You said Mr. O'Neal.

Q (By Mr. Lubel) Oh, I meant -- sorry. John O'Neal Hall's death.

A Yes.

Q What caused Mr. Hall's death?

A He had an overwhelming pulmonary infection which got him into the intensive care unit, and he died.

Q And what caused his pulmonary infection?

A I don't know if a specific organism was identified. I'm certain that his infection was likely aggravated by his blood condition.

Q What blood condition?

A He had a condition called myelodysplasia.

Q Did his leukemia contribute to his death?

MR. HUMPHREY: Objection. Form.

A Well, the M.D. Anderson people in reviewing the slides felt he had acute myeloid leukemia, a monocytic type of leukemia. That opinion wasn't reached by the doctors at St. Luke's who originally looked at the marrow. I don't have any dispute as to whether he had acute myeloid leukemia or was still simply in a myelodysplasia phase, but I think his bone marrow disease contributed to his death.

Q (By Mr. Lubel) What bone marrow disease are you referring to?

A He had a diagnosis of myelodysplasia, and, as part of that diagnosis, he has abnormal white blood cells with abnormal white blood function. That makes him susceptible to infection.

Q And how would that contribute to cause his death?

A Well, if you have a person of his age and they get an overwhelming infection and they're not able to use their white cells properly to snuff out the infection, it can cause death.

Q But what is it about his blood disease that

affected his ability to fight off the infection?

A I would say defective white cell function.

Q What does that mean?

A Well, normally when you have to kill an organism, let's say, and you have a pneumonia and we proceed to give you antibiotics, some antibiotics are what we call bactericidal meaning that by contact they can kill a bacteria. Many antibiotics are what we call bacteriostatic. That means they slow down the growth of the bacteria. Well, the way we get rid of an infection with our antibiotics is we may slow down the growth of the bacteria but the normal white cells phagocytize or eat up the bacteria and based on the enzymes they have inside the white cells they can kill those bacteria. And when you have a person who has few normal white cells, it is very difficult to cure an infection with antibiotics alone. And that's not only bacterial infections but white blood cell -- but fungal infections.

(Pager interruption)

MR. LUBEL: Are you okay?

THE WITNESS: Yeah.

Q (By Mr. Lubel) What caused Mr. Hall's blood disease?

A I don't know.

Q Did any of the treating physicians in the medical records you were furnished -- and I take it you reviewed the medical records?

A Yes.

Q Did they tell us the cause of Mr. Hall's blood disease?

A Not that I saw.

Q So you do or do not have an opinion as to whether Mr. Hall, in fact, had leukemia?

A The short answer to that is I'm not totally certain, but I would wouldn't dispute it.

Q So if the experts on behalf of the plaintiffs come in and claim that or contend that Mr. Hall prior to his death had acute monocytic leukemia, you're not going to contest that point?

A That's correct.

Q Had you wanted to, what would you need to review to confirm whether, in fact, Mr. Hall had leukemia?

A Well, I think I can explain a little bit about what the confusion might be. I looked at the bone marrows that I was supplied. Now, M.D. Anderson when they looked at them had many more copies of slides than I had, and they had done special stains on the bone marrow. And I saw what they saw, at least on the few

slides that I looked at, that there were a number of immature cells in the bone marrow, and superficially it looked like an acute leukemia. I can't dispute that. But his treatment and his infection can complicate the morphology. In other words, prior to his getting the -- prior to his getting the pneumonia his doctor put him on a compound called Neupogen. It's a stimulus to white cells. And many people with myelodysplasia have large numbers of monocytes that are literally typical as part of their myelodysplasia. When you give them Neupogen, you can make their white count sometimes go up extremely high, 30, 40, 50,000, and the morphology of the cells looks very abnormal and it looks like an acute leukemia. But when you stop the Neupogen and the effects subside, the bone marrow goes back to just the way it started, and it's still a myelodysplasia.

So what happened is he was getting a lot of Neupogen stimulating his bone marrow and he had a bad infection stimulating his bone marrow and, as a consequence, his bone marrow was full of a lot of immature cells. And it may well have been an acute leukemia, but I would have liked to have seen a bone marrow, let's say, in a more steady state. He wasn't exactly in a steady state when that bone marrow was done.

Q What do you mean "steady state"?

A Well, in other words, not taking Neupogen or a colony-stimulating factor, not having an acute infection, because I can tell you over the years I've seen many people with myelodysplasia who get an intense proliferation of their bone marrow, sort of simulating a leukemia when they have an overwhelming illness. And if they get over that illness, the proliferative phase of the bone marrow can subside.

And you see what -- the M.D. Anderson doctors, they used a term that means different things to different people. They used a term called blast equivalents. When we see a leukemia and we're confident it's a leukemia, we see myeloblasts, extremely immature cells. But many people feel that in monocytic leukemias you can count cells that are somewhat differentiated. They're a little more mature than blasts. They may be promonocytes. And they call those blast equivalents, meaning they mean the same thing as a blast but they're not exactly a blast. Well, sometimes those blast equivalents can subside as the patient gets away from the acute illness.

So, as I say, looking at the material I got, I certainly saw a lot of immature cells, and it may well have been an acute myeloid leukemia. And I

certainly wouldn't dispute that diagnosis. The M.D. Anderson people are expert pathologists. But, as I say, I didn't have enough slides and the setting of what had happened was unusual and so I wouldn't be a hundred percent certain about that. That's basically the long version of my opinion.

Q What did the M.D. Anderson doctors diagnose him with?

A They diagnosed him as an acute monocytic leukemia.

Q Otherwise known as AML?

A AML, correct.

Q That's the short version of what you just said, right?

A Correct.

Q Today can we refer to it as AML?

A Absolutely.

Q How did they diagnose Mr. Hall with AML?

A What they did is they looked at the cells, and they saw a lot of immature cells. And, as I said, they call those blast equivalents or blast like. They stained the bone marrow with a number of stains. One of them was called a peroxidase stain which highlights myeloblasts. That stain was negative for the most part. Then they stained it for a stain called lysozyme, and

monocytes, both mature and immature, produce heavy amounts of lysozyme. And these cells lit up with that stain, so they were clearly in the monocytic cell line. So they saw a bone marrow full of immature-looking cells that took all the stains for monocytes, so it was clearly a myeloid illness.

Q As opposed to what?

A As opposed to a lymphoid illness. Excuse me. And because the stains that were positive were all myeloid stains. So it was either an overreactive myelodysplasia because of the setting, or it was an acute monocytic leukemia. From my standpoint and my opinion about the case, it doesn't make a difference which one it was, and I will accept a diagnosis of acute myeloid leukemia monocytic variety.

Q When did you look at the slides from M.D. Anderson?

A I think I took a picture of them actually. It's somewhere in the --

MR. HUMPHREY: Be in this folder.

A -- in the thing.

I asked for the slides, and I got very few of them. And I took a photograph just on a Xerox machine of the slides I was given. I think I was only given four slides or so. Let's see if I can find them.

They were in here because I ran across them a moment ago.

MR. HUMPHREY: That looks like it right there.

A This was on 12/1/09 and my notes say I was given six cassettes of slides, but only two of them had bone marrow. In other words, they were a mixture of slides, some from his autopsy, some from this and that. And the number of slides that were actually bone marrow slides were only a couple of them. And I looked at them and I could see what the M.D. Anderson people were talking about, that is to say there were a lot of immature cells, but these were not what we call films or smears. In other words, they were like biopsies, a chunk of material. So you couldn't see the cellular detail very well. And those were basically the sides I got.

Q (By Mr. Lubel) Who sent you these slides on December 1st of 2009?

MR. HUMPHREY: I did.

Q (By Mr. Lubel) Baker Botts?

MR. HUMPHREY: Yeah, those are the ones we got.

Q (By Mr. Lubel) Your lawyers did?

A Yeah, the Baker Botts people sent me that;

that's correct.

MR. HUMPHREY: We sent him everything we got from the record service.

A Right.

And they were not -- I mean, for describing a bone marrow disease, they were not ideal, but I got the picture that M.D. Anderson saw. And that's why I say I saw enough so that I don't really dispute their opinion. But -- but I would say if you ask me of what I have seen of the record am I a hundred percent confident of that, I would say no because of the setting that these cells were taken in.

Q (By Mr. Lubel) The reason I asked when you had seen the slides is because the report that you've given regarding your opinions in the case --

A Yes.

Q -- is November 22nd.

A Right.

Q So you saw the slides after that?

A Whatever the date is. That's -- yes.

Q December 1st.

A That's so. That's correct.

Q And so I didn't see a reference in your report of your opinions to the slides --

A That's correct.

Q -- which is why I was curious.

A I may -- let me just see this for a second, if I can. What did I say?

Yes. In fact, in my last paragraph I said, "I reserve the right to amend this report as new or additional materials such as further estimations of dose-exposure levels and additional clinical material concerning Mr. Hall, such as his bone marrow slides, become available." So I had no bone marrow slides when I wrote this letter.

Q Nor did you feel like the bone marrow slides were critical to you forming an opinion in this case?

A That's correct.

(Exhibit 2 marked)

Q (By Mr. Lubel) So Exhibit No. 2 is your correspondence with Baker Botts regarding these bone marrow slides, correct?

MR. HUMPHREY: Objection. Form.

A Well, I wouldn't say it's correspondence. It's simply a notation to myself as to what I looked at.

Q (By Mr. Lubel) All right. Do you have correspondence with the law firm that hired you regarding forwarding the information?

A No.

Q How did you get it?

A Oh, in other words, correspondence that accompanied the slides?

Q Yes, sir.

A Is that what we're talking about?

Q Yes, sir.

A I thought you meant correspondence I sent to them.

Well, it may have -- what's the date on that letter? I have all the letters in here and they came in a little box and I don't remember if there was a cover letter or not, but if there is it would be in here.

Q Let's see.

MR. HUMPHREY: It's quite possible there's no cover letter.

A Yeah, I don't remember seeing a cover letter.

MR. HUMPHREY: As I recall, we just couriered them over to Dr. Natelson after I told him over the phone that they were coming.

Q (By Mr. Lubel) Was there anything in Mr. Hall's medical history that you attributed to his death?

A Well, he had significant heart disease, he had had coronary artery disease and had an aortic valve replace, so his cardiac function was not normal. And

when you have an overwhelming infection and you have a bad heart, that's just another factor into why you may die from it.

Q Okay. So did his heart kill him?

A It would be hard to put a finger on what killed him. I would say his infection killed him.

Q What role did his heart play in his death?

A I couldn't put a percentage or a number on that.

Q Did the doctors that treated him discuss the role of his heart in his death?

A I don't recall. There's an autopsy report, and it has an opinion in there.

Q Does it claim that the heart caused his death?

A I don't recall. We can look at the autopsy report and see what their conclusion was.

Q Do you have that with you?

A Well, yeah, it's in the file of materials. Let's see if I can find it. I'm just trying to find the bottom line here at the end. I have their final diagnosis. Their diagnosis, I can read you what they say, but basically their diagnosis is respiratory failure. And it finally says in last sentence: The patient eventually developed respiratory failure, what we call ARDS, acute respiratory distress syndrome. The

family requested removal of support, and the patient expired. So you could say, well, he expired from hypoxemia, low oxygen levels, if you wanted to pick a number. In the final anatomic diagnosis they also look at his bone marrow and other things. They do not make a diagnosis of acute leukemia on his bone marrow. They make a diagnosis of myelodysplasia.

Q Myelodysplasia is a blood disease?

A Yes.

Q Is it a cancer?

A Not always.

Q Was his cancer?

A I would say in his case we would consider it cancer, yes.

Q What's different about his MDS that makes it cancer?

A Well, in some cases of myelodysplasia it's an autoimmune process, and if we have a patient with myelodysplasia and we're able to correct that with our antiimmune system drugs and their blood count comes back to normal, it wasn't a cancer. But that's perhaps 10 percent of people with myelodysplasia. Most people with myelodysplasia -- and I think he would be in that group -- it would be considered a cancer.

Q Did he have a form of myelodysplasia? And

today we'll call it MDS, is that okay?

A Yes.

Q Did he have a form of MDS that can be benzene induced under the right circumstances?

A Well, he had MDS, and I would say that based on what is in his bone marrow, yes, I think that his bone marrow could have been potentially induced by benzene.

Q The reason I ask you this -- and you and I have talked about this more than once, right?

A Yes.

Q You have opined, you've testified under oath, that there's a particular form of MDS called RARS, R-A-R-S, that you do not believe can be benzene induced, correct?

A Correct.

Q But you have testified that the other forms of MDS under the right circumstances can be caused by benzene exposure?

A That's true depending on what classification you're using because the original classifications had chronic myelomonocytic leukemia as a form of MDS. That's been moved out of that category.

Q Which classification system do you prefer to use?

A You can use the current classification system,

the WHO system.

Q As opposed to the FAB?

A Yes, I think that would be the -- has supplanted that.

Q And that's in your file materials, that classification, correct?

A Yes. Actually, I have a copy of the World Health Organization book that's got a copy of or a chart that shows that in there.

Q Now, there's more than one form or subtype of MDS, correct?

A Yes.

Q You've testified previously that the only form that you're convinced is not caused by benzene exposure is RARS, R-A-R-S, correct?

MR. HUMPHREY: Objection. Form.

A We're talking morphology forms, I think is what you're dealing with, and you get into a difference of what something looks like under the microscope or what it looks like in conjunction with other tests. In other words, I think there are forms of myelodysplasia such as one that the World Health Organization refers to as the 5q minus syndrome. That's a form of myelodysplasia. By looking at that bone marrow under the microscope, it could be caused by chemicals or benzene, but in clinical

usage there's no evidence of that. In other words, once we know that it's got this particular chromosome defect, that makes it unlikely to fit into a benzene-related disease.

Q (By Mr. Lubel) But you do acknowledge that Mr. Hall's particular form of MDS can be benzene induced?

A Yes.

Q And obviously for many years you've testified under agreement you've acknowledged that AML can be caused by benzene exposure?

A Yes, particularly certain forms of AML. There are some that are far less likely than others.

Q And the particular -- the only form that I've heard you testify that you did not believe was related to benzene exposure was the M3 form.

A I think that there is -- that is true. The M3 form is not thought -- or acute promyelocytic leukemia is not thought to be caused by benzene. I think the Inversion 16 AML there is no evidence that it can be caused by benzene.

Q Which form is that?

A Inversion 16? It's just referred to as Inversion 16 AML.

Q It's not one of the M1 through 9 --

A It can be either M4 or M2. It's not fixed into a particular M category.

Q As a hematologist is there a relationship between MDS and leukemia?

A Is there a relationship between them?

Q Correct.

A Well, a number of people who have MDS catch leukemia as the years go by.

Q Why is that?

A Well, because -- why? I don't always know why, but in some cases what happens is that if there's a chromosome defect it may be a single one and then over time it becomes multiple and the bone marrow function becomes worse and eventually it may evertuate in an AML. And if you looked at all comers with myelodysplasia, that probably happens 20 or 25 percent of the time. It happens less in certain types of myelodysplasia, perhaps more in others.

Q Are you saying that MDS predisposes someone to leukemia?

A Yes, it is preleukemic syndrome.

Q But it is not necessary that somebody have preceding MDS in order to have or develop a leukemia, correct?

A Certainly not.

Q I think you said 20 to 25 percent of the MDSs turn into leukemia?

A Yes.

Q Does that happen irrespective of whether they are treated with leukemogens like chemotherapy or radiation therapy? Is that in its natural course?

A That's in its natural course, yes.

Q So when you give the statistic 20 to 25 percent of MDSs turn into leukemia, you're not referring to those that are treated with chemotherapy?

A That's correct.

Q Do you know what percentages of the MDS that are treated with chemotherapy turn into leukemia?

A I couldn't put a number on that.

Q Just an estimate?

A I really couldn't put a number on it because, you see, typically you're treating them with chemotherapy when they're forming increasing numbers of blasts, not enough to make the diagnosis of AML but enough to suggest that they're on a slippery slope, and you now institute chemotherapy and chemotherapy may predispose to acute leukemia. So it's not always clear whether it's your chemotherapy or it's the natural history any longer.

Q (By Mr. Lubel) How does one tell?

A I don't know that one can tell.

Q So even if you have a patient that has MDS that is receiving chemotherapy treatment, respected hematologists can't tell whether their subsequent leukemia was, in fact, caused or contributed to be caused by the chemotherapy itself?

A I think that's a reasonable statement, yes.

Q Same with radiation therapy, same answer, or is it different?

A I think really it would be the same answer. You get into some unique situations. For example, I may have a person who has an acute leukemia and they are treated with chemotherapy and they get a bone marrow transplant. Their original acute leukemia has no chromosome defect. As a part of that transplant process they receive chemotherapy and radiation. A year or two or three elapses, and they catch leukemia again. This time the leukemia has minus 7 chromosome. They have a new form of leukemia. It may even look different than the one they started with. Well, in those circumstances you can be not hundred percent certain but you can be fairly certain that your therapy caused that second leukemia.

Q Can that happen in the absence of the therapy? Can the leukemia morphology change?

A Well, in the absence of therapy a person with leukemia would only live a few months, so you wouldn't be able to know that.

Q I didn't see in your report any criticisms of any of Mr. Hall's treating doctors, correct?

A That's correct.

Q I take it you're not critical of them?

A No.

Q But you've not formed the opinion that any of the doctors or hospitals that treated Mr. Hall committed malpractice, correct?

A That's correct.

Q Let's take a step back for a minute. I want to talk to you about methodology. You've heard that term at least since the time period you've been in these lawsuits, right?

A Yes.

Q Pretty important?

A Yes.

Q You have attempted to follow the same methodology in all of these cases, right?

A Yes.

Q What is it?

A What is it?

Q How would you lay out your methodology?

A My methodology. Okay. Let's take this case, for example. The first thing I would like to know is what is the primary diagnosis. And in this particular case the primary diagnosis, the primary hematologic diagnosis -- the patient had other medical conditions -- the primary hematologic diagnosis is myelodysplasia. That is a very general category of disease, and there are many different forms of myelodysplasia. And in many cases we don't know the cause for them. And over the years people have tried to separate them into smaller and smaller categories to try to determine a cause. So that's the first thing I would do, is to look at what's the primary disease. And then the second feature is what is the natural history of this disease.

Q What does that mean?

A Well, that means that if I have a patient who gets hepatitis C, over time part of the natural history is to develop cirrhosis of the liver from it and over more time to develop liver cancer in that cirrhotic liver. That's the natural history. So if I see someone with hepatitis C as a primary diagnosis and they've caught liver cancer, I'm not surprised. That's the natural history of the disease. In this particular circumstance Mr. Hall has myelodysplasia. It is the natural history of the disease to progress to acute

leukemia, so whether he does or doesn't have acute leukemia in my view is not an essential feature of his illness. And that's why I say I don't dispute it, but it doesn't carry a tremendous importance for me because the question here is what is his illness and why did he have it.

Q But he did not divert from the natural history of MDS in your opinion?

A No, he did not divert from the natural history of MDS.

Q And when you look at the primary diagnosis, the reason you're looking at that at the beginning is to evaluate whether, in fact, the patient has a disease that can be chemically induced in the first place?

A Well, that's one thing I look at. I look at what is the disease and then what are the causes of that disease.

Q And he met that?

A He met that he had the disease. He had myelodysplasia, yes.

Q In other words, he met a disease that can be benzene induced?

A Yes.

Q It followed the natural history of the disease as you see it --

A Yes.

Q -- or as you appreciate it, correct?

A Yes.

Q What was the third part of your methodology?

A The third part of the methodology is to attempt to look at causation.

Q That's pretty broad, though.

A That's a broad thing. But under causation would be how is it that we can -- are there ways that we can suggest causation because other than having somebody with an immune-based myelodysplasia, treating them and the myelodysplasia goes away, it's very difficult to prove causation. So in any individual case we can't prove causation with certainty. We can just say what's likely and what's more likely than not. And when we look at myelodysplasia as a general category, there is general agreement in the literature that the vast majority of cases are what we call de novo, or you like to say idiopathic, that we do not know the cause and we do not find evidence from the record that there was a cause, either radiation, chemical, chemotherapy, viral infection. We don't find an underlying illness that led to that diagnosis and is referred to as idiopathic or, in this case, de novo.

Q Those are synonymous in your opinion,

idiopathic and de novo?

A They're not exactly synonymous. Idiopathic simply means we don't know the cause. De novo, when it's taken in the context of myelodysplasia and AML, says we don't know the cause, but it is not chemotherapy, radiation therapy or benzene or immunosuppressor drugs, for example.

Q So you're saying de novo is more specific on the considerations that were taken into account on causation?

A I think so, yes.

Q And so in your opinion a reference to a disease such as MDS being de novo or referred to as de novo means that whoever was evaluating causation considered chemotherapy, radiation and chemicals such as benzene?

A Yes, whether that person was an attending physician at the time or me today. In other words, I'm looking at what I have from the record and would I put that in a pile that is likely to be de novo or a pile that is likely to be secondary to something and then to determine what that something might be.

Q Now, I take it with respect to Mr. Hall you saw no evidence of it having a secondary cause specifically from chemotherapy or radiation treatment?

A Correct.

Q He did not have a smoking history according to your report?

A According to his report I take it on faith that he was not a smoker. The medical records say he did not smoke.

Q And so you considered smoking as a potential cause of his MDS or leukemia?

A Yes.

Q You ruled that out?

A Yes.

Q As best you could in your methodology which is looking at the medical records?

A Yes.

Q You looked at or analyzed the medical records to see if he had had preexisting chemotherapy or radiation treatment, secondary causes, correct?

A Yes.

Q Didn't see any?

A I didn't see any.

Q What else did you consider in the causation category?

A In the causation category. Well, in this instance we're looking at benzene as a potential cause for this leukemia, and so I noticed that one of your experts modeled his benzene exposure and thought that

the cumulative amount over many years by inhaled route was around 6.7 part per million and a little slightly higher if he factored in skin or other sources. And one of the articles I referenced in here by the World Health authority, their chapter on myelodysplasia, indicates that while benzene may cause myelodysplasia it causes it at exposures well above the recommended levels. Well, those levels are not high enough, according to the World Health Organization fascicle, to cause benzene-induced myelodysplasia so that his exposure history would be weak if benzene was the cause. That would be one thing I looked at.

The second thing I looked at is the morphology. And while I've said that his morphology could be consistent with benzene-induced leukemia because there's not a hundred percent perfect concordance on what morphology looks like, the literature suggests that there is a strong pattern to the myelodysplasia we see from benzene, and that pattern is somewhat similar to what we see from chemotherapy in that generally the bone marrows have reduced numbers of cells, what we call hypocellularity. His bone marrow didn't have Hypocellularity.

Q You didn't see any reference in the medical records or the pathology?

A It said it specifically was normocellular, normal cells.

Q There's no reference to hypocellularity, correct?

A Correct.

And in the subsequent references that I have in here, the bone marrows in benzene-induced disease are typically hypocellular.

Second, the bone marrows in benzene-induced suppression often have an increase in eosinophils. That's a type of a white cell that's got red granules in there. He didn't have that feature in his bone marrow. The bone marrows from people with benzene don't have any ring sideroblasts in them. He didn't have any ring sideroblasts. I think one report said he had one stray ring sideroblast, but we'll call it negative. So he didn't have ring sideroblasts. That doesn't help us, in other words, because benzene-induced disease is not associated with ring sideroblasts, but he didn't have ring sideroblasts. Had he had ring sideroblasts, that would, again, made it less likely for benzene, but he didn't. So he's got hypocell -- he doesn't have hypocellularity. He doesn't have eosinophilia.

The other feature of these marrows is that

what's called erythrophagocytosis. And I can explain that a bit better. Most bone marrows have scavenger cells in them, what we call large histiocytes, and in certain bone marrow diseases those scavenger cells become very active and they eat up normal cells or seminormal cells. And when you look at the bone marrow you see these histiocytes full of gobbled-up cells, and that's very typical in benzene-induced myelodysplasia. He didn't have that.

So, yes, he had myelodysplasia. He didn't have the typical form you see from benzene. That doesn't exclude benzene from causing it, but it was not the typical feature that we see.

Q Okay.

A Then --

Q Go ahead. I didn't mean to interrupt you.

A Then the next feature when you go from the morphology is the chromosome array. He had an interesting chromosome analysis, and his chromosome analysis showed that he had what's called deletion 20q. And what that is it's a variable loss of genetic material from one of the arms of the 20th chromosomes. It's referred to as an interstitial deletion, sort of part of the innards are missing. And that particular defect has been well studied and occurs in several bone

marrow diseases, including polycythemia vera where it was first seen, and some feel it can be seen in as many as 10 percent of people with polycythemia vera. It also occurs in my myeloproliferative diseases, and it can occur in myelodysplasia. And, generally speaking, about 5 percent of people with myelodysplasia have the 20q defect. To my knowledge it has never been described from benzene exposure, and most of the diseases it occurs in can't be caused by benzene. They're not benzene-related diseases.

Now, in the case of Otto Wong, who's been studying these interstitial deletions among the Chinese with heavy benzene exposure, he has found in the Chinese general population with myelodysplasia they, too, have the 20q minus phenomenon, and he found it in 4 percent of the Chinese patients he studied with what he thought was de novo myelodysplasia; that is to say, by questionnaire and examination, no history whatsoever of benzene exposure. Another author says it occurs as possibly as high as 9 percent in Chinese with de novo disease. So the bottom line is 20q occurs at least with about the same frequency in China as it does in the United States.

However, in his heavily benzene-exposed group, Dr. Irons, rather -- I think I may have said Wong

previously -- but what Dr. Irons found was that not a single patient had the 20q minus deletion. So the fact that he has the 20q minus deletion does not support benzene as a cause because it doesn't seem to appear either in the literature as a consequence of benzene or in a study, an epidemiologic study, if you will, of people with heavy benzene exposure. It's not a phenomenon that's been identified.

So, again, we're looking at things that are likely and to me he's got not an impossible morphology for benzene, but he isn't have the ideal morphology for benzene. He has a very low benzene exposure, lower than what the literature tells us can cause myelodysplasia. He has the wrong chromosome for a benzene-induced myelodysplasia having the 20q chromosome. And the general dogma in the literature is that when you're looking at benzene-induced hematologic disease, in general it's a very rare phenomenon. So were this disease to be from benzene, you would be talking about an extraordinarily rare manifestation of an extraordinarily rare illness in the United States. So, therefore, I would have to say in all medical probability this is not likely to be a benzene-induced illness and is likely to be de novo, granting the fact that we don't have a biochemical test that we can apply

at this time to people with myelodysplasia to prove or disprove a particular etiology.

Q Have you finished your answer on your methodology?

A That's my methodology, yes.

Q Let's go back --

A Okay.

Q -- because you gave us more than a mouthful, fair?

A Absolutely, yeah.

Q I think you said that roughly 80 to 85 percent of the MDSs are de novo?

A Yes.

Q Did I hear you correctly?

A Yes.

Q You brought with you a stack of studies?

A Yes.

Q Are there studies that you're relying upon that have determined the percentage of MDSs that are de novo versus secondary?

A Yes.

Q Can you pull those, please?

A Many of these articles touch on that, but I can pick some specifically. Let's just take this one, for instance. This is an article that's published in the

yearbook of the American Society of Hematology. So this is what the American Society of Hematology is educating people about, hematologists and oncologists around the country, and it's written by several experts in the field. And this article says: At present, 80 to 90 percent of patients with myelodysplasia or AML diagnosed at major centers are de novo cases where --

Q How did they determine that?

A Pardon me?

Q How did they determine that?

A By the skill of the doctors who examined and questioned these patients and taking into consideration all other aspects of the disease, the morphology, the chromosomes, their employment, the pace of the disease, many other things.

Q Let's mark that as Exhibit 3 just so you and I are having a clear record.

(Exhibit 3 marked)

Q (By Mr. Lubel) The article you're referring to I've put an Exhibit 3 sticker on it, correct?

A Yes.

Q And so is this a review article, or is this an actual study of a population?

A This is a review article by a person who has studied for many years many populations.

Q Are you able to tell us from looking at this review how the authors calculated the 80 to 90 percent?

A No.

Q Do they -- do they set forth their methodology and the population that they studied?

A No.

Q Do you have any studies that set out the methodology and provide us with details of how this 80 to 90 percent are de novo?

A I think the closest thing to that might be Dr. Irons' work because, as he explains it, what he did in his Chinese studies are that everything was blinded. In other words, one group received slides, had no clinical information, and they made a diagnosis based on the slides. Another group did the chromosome analysis, blind, and had no information about the clinical nature of the disease. Another group, blind, questioned the patients in detail about their occupation and their exposures and came to a conclusion whether they were or were not exposed. And then they put that material together in their population. And so he has a nonexposed or a de novo group, and he has what he thinks is a benzene-related group.

Q Can you pull out the literature you're referring to?

A Let's see where that might be. Let me find my report.

MR. HUMPHREY: Actually, I think Lance took your copy of the report.

THE WITNESS: Oh, he took my copy of the report. That's why -- eventually I can find it in here. Let's see. Let me just see what number that is.

A Yes, this is a preprint or it's an article in press, and I think it's one of several that Dr. Irons has written concerning this issue. I'll put that on there.

(Exhibit 4 marked)

Q (By Mr. Lubel) That's Exhibit 4, correct?

A That is Exhibit 4.

Q So how many patients with MDS was Dr. Irons studying?

A 649 cases. And he alleges that this is the largest number of cases ever studied from a single center.

Q What center was it?

A It's what's called the Fudan Clinical Pathogen Clinical and Molecular Research Center, Institutes of Biomedical Sciences, Fudan University, Shanghai, China.

Q Now, what information did Dr. Irons gather on the 600 something MDS people?

A Well, let's see what he says. He studied their bone marrow and applied the criteria we've talked about, the WHO 2008 criteria, to make a morphologic diagnosis. The subjects were administered questionnaires by a certain group and information on previous disease work histories and exposures to potential etiologic agents such as benzene, and a total of 80 of 649 cases were determined to have some benzene exposure. He then further subdivided that, and I think that's discussed in the paper, as to what their exposures were. Then he studied their cytogenetics, their chromosome analysis. And then, to be as specific as he could about what benzene did to the bone marrow, he took the cases who had the highest benzene exposures, and, as he described them to me, these were sometimes as much as 5,000 cumulative part per million exposures.

Q Can I stop you real quick?

A Yeah.

Q How many of the -- was it 649...

A Yes.

Q ...did you say had documented exposures to benzene?

A By questionnaire 80 of the 649 were determined to have some benzene exposure is what he says.

Q So what percentage is that roughly, 20 percent?

A No, 13.2 percent.

Q 13.2 percent?

A Uh-huh.

Q Now, how many of the 649 patients with MDS had previous exposure to chemotherapy treatment?

A I don't know that.

Q Does he say?

A He doesn't specifically say that in the article.

Q How many of those patients had had preexisting or previous exposure to radiation?

A I don't know that.

Q How many were smokers?

A I don't -- he states subjects were administered questionnaires and information on previous disease, work histories and exposures to potential etiologic agents he says such as benzene. I would have to assume that he's asking them other things, radiation, chemotherapy and smoking, but I don't know that.

Q Does he give us the data on how many are chemotherapy, how many are radiation, how many are smoking, et cetera?

A I don't see that in there.

Q I mean, Dr. Natelson, if one were to from that one population of patients try to evaluate and express

an opinion as to how many are secondary versus how many are de novo using your definitions --

A Yes.

Q -- wouldn't you need to know how many had preexisting or previous chemotherapy, radiation, smoking histories, benzene exposures, et cetera?

A Well, he's telling us there are benzene exposures. The purpose of this paper is to isolate the patients with benzene exposure, then grade their benzene exposure by degree and then isolate what are the features of that illness.

Q I'm asking you a different question, though. I'm not disagreeing with what you just said. My point is if you're trying to evaluate how many of the patients are de novo versus secondary, irrespective of what the secondary cause is, don't you need to know in the secondary category the data that backs up how many have had the exposures whether it be to chemotherapy or radiation or smoking?

A Yes, I understand that. But I don't know that he doesn't have that data. He's simply presenting a snapshot of this, and the paper is designed looking at specifically the characteristics of benzene exposure. He may tomorrow decide to publish a paper about what you're asking.

Q But what I'm asking --
A But I'm saying --
Q -- is not addressed.
A Yeah, it's not addressed.
Q Right?
A Correct, yeah.
Q I'm just saying the question I'm asking of doing the count or looking at the percentages of de novo versus secondary, you don't see the data in that paper? That's not to say it doesn't exist, you just don't have it there to do the math, correct?
A That's correct.
Q Do you have any studies that actually do the math versus just make the conclusion?
A I don't think that I have anything like that in this -- in this -- these groups of papers.
Q You understand what I'm asking? I'm saying there's -- review articles frequently make conclusions, right?
A Yes.
Q And then they're supposed to cite to support or they often do, correct?
A Yes.
Q This 80 to 90 percent figure that you've referred to for MDS patients being de novo, that solely

comes from review articles, correct?

MR. HUMPHREY: Objection. Form.

A Yes and no. In other words, there is one article I don't think I cite among these. There's one by Mauritzson which specifically was published in leukemia perhaps six or seven years ago in which he looks at something like 5,000 cases of AML and classifies them into de novo/secondary and then also the MDS into de novo/secondary, and he has the chromosome defects they have. In other words, it's an abstract of what's in the literature in terms of frequency based on real cases that have been reported.

Q (By Mr. Lubel) Well, do you have that article with you?

A No.

Q Okay. What's the name of it?

A It's called -- it's spelled by the last name M-a-u-r-i-t-z-e-n, that's close, and it's a well-known paper. I've cited it in previous articles, but I didn't -- I didn't choose to site every article I have in my file.

Q And is it your recollection that that article breaks down the methodology that was used to determine whether they were de novo or secondary?

A Oh, I see what you're saying. No, it doesn't

go into that. There are limitations in what one can put in an article. This is -- he's abstracting the literature based on cases that have been reported, and he's taking the word for the doctor as to how they made that determination.

Q It's a review, it's not a study?

A I would call it a study. He's got a large number of cases. He's breaking them down by many different features.

Q Does he have the cases, or is he looking at articles that talk about the cases?

A He's deriving the cases from published articles.

Q And so you're saying if we looked at the articles -- have you looked at the articles or provided --

A His primary source material, no.

Q Are you --

A See, some of these come from articles, some of these cases are taken from large leukemia groups like the old Southwest Cancer Chemotherapy Group that M.D. Anderson was a participant in, and there are many cancer groups around the country that abstract this data.

Q Go back to Dr. Irons' article. We've marked

that as Exhibit 4, correct?

A Yes.

Q Does Dr. Irons,.based upon the 649 MDS patients that he claims to have studied, right...

A Yes.

Q ...does he set forth the percentage of de novo versus secondary in there?

A No.

Q Does he do that analysis, as far as you can tell, from reading the article?

A I can't tell -- he doesn't address that analysis in this article.

Q Now, if you come to my office and examine and do questionnaires and look at medical records on, let's say, the 30 people I've got working for me and one has leukemia and they have no history of benzene exposure, they have no preexisting chemo, radiation, weren't a smoker, you can't find a secondary cause -- are you with me so far...

A Yes.

Q ...you're not going to be able to conclude from looking at that group that a hundred percent of people with leukemia are de novo, correct?

A I think somehow you lost me in the questioning. You've got one patient in your office who's got AML, and

they have no history of any potential preceding cause?

Q Correct.

A And then I got lost as to what you're asking.

Q So they're de novo, right?

A If I have a patient with an acute leukemia and I've questioned them and they have all the features of de novo leukemia, it is de novo leukemia.

Q A hundred percent --

A Well, in my judgment --

Q -- of that population? A hundred percent of that population?

A Of that single patient?

Q Right.

A Yes.

Q And so the population or the group that's being studied is pretty important, right, as far as extrapolating from that statistic and saying this applies all places for all times, true?

MR. HUMPHREY: Objection. Form.

A It depends on the study. In other words, this is a study of a large number of people referred because they have a diagnosis.

Q (By Mr. Lubel) Whose study are you referring to?

A Dr. Irons in this case. I thought you were

asking me about this one.

Q I'm talking about generally now, see, because you've already agreed that Dr. Irons doesn't set forth specifically the breakdown between de novos versus secondaries.

A He doesn't --

MR. HUMPHREY: Objection. Form.

A -- record that in this article.

Q (By Mr. Lubel) I'm not saying it wasn't done, it's just we can't tell?

A Correct.

MR. HUMPHREY: Objection. Form.

Q (By Mr. Lubel) You can't look at it and give me an answer to that question, right?

A Correct.

Q Likewise, you don't see backup data for you to do the math yourself in the article?

A Correct.

Q In other words, you can't tell me how many of those patients had preceding or previous chemotherapy treatment?

A That's correct.

Q Without that type of information, you can't do a percentage or mathematical breakdown of de novo versus secondary, correct?

A Correct.

Q Not to say Dr. Irons hadn't done it, it's just we don't have the information to know whether it's been done or not, correct?

A Correct.

Q You don't have any articles with you that set forth that level of detail, correct?

MR. HUMPHREY: Objection. Form.

A I'd have to look, but I think I have. In other words, when you have an expert giving a number like that who has published numerous papers on this subject abstracting such patients, I don't have the primary source material, but I have the answer.

Q (By Mr. Lubel) Okay. Well, so when -- you're referring to Exhibit 3?

A I'm referring to Exhibit 3, yes.

Q All right. So how many of these author's patients --

A Yes.

Q -- with MDS --

A Yes.

Q -- have had benzene exposure?

A Well, certainly the -- since he's fully aware that benzene causes AML, when he tells us that 80 to 90 percent are de novo cases, those are cases that he

doesn't feel they had any significant benzene exposure because if he did he'd call them secondary.

Q How many? How many of his patients have had benzene exposure?

A Of what degree?

Q Occupational setting.

A I don't know.

Q Does he say?

A He doesn't say.

Q How many of his patients have had chemotherapy or radiation treatment before MDS was diagnosed?

A It would be some of the 10 to 20 percent.

Q How many?

A Somewhere between -- he says, whereas, 10 to 20 percent represent therapy related. So somewhere between 10 and 20 percent of his overall numbers are people who have had chemotherapy.

Q What's his overall number of people with MDS? Does he say? Did the author say in this study?

A Oh, he's not giving a number, no, in that study.

Q Does he give it anywhere how many patients he's treated with MDS or seen?

MR. HUMPHREY: Objection. Form.

A Not in the article.

Q (By Mr. Lubel) Do we know the raw numbers as to the number of patients he's seen that have had previous chemotherapy or radiation treatment?

A Well, he has some 44 references to what he says, and if one were to abstract these 44 papers, it's likely that you would find some of that information at least in some of these papers. He's giving a summary of his many years of experience in this field.

Q Okay. And so how many patients have these authors treated that had occupational settings in benzene?

A I don't know.

Q Do you know anything about the type of patients he treats --

MR. HUMPHREY: Objection. Form.

A No.

Q (By Mr. Lubel) -- as far as their occupations?

A I don't know.

Q Is it reported in the study?

A No.

MR. HUMPHREY: When you get to a stopping place, Lance --

MR. LUBEL: Yes.

MR. HUMPHREY: -- can we just get up and stretch our legs for a minute.

MR. LUBEL: Yeah, I need to get some water.

(Recess from 2:37 p.m. to 2:42 p.m.)

Q (By Mr. Lubel) Earlier in your deposition you told us that there's typically a pattern that is seen in the morphology of benzene-induced disease, correct?

A Yes.

Q I take it you're referring to either MDS or leukemia?

A MDS.

Q MDS. The first one that I wrote down was hypocellularity.

A Yes.

Q Can you do your best and describe that in layman's terms?

A Yes. Normally when you look at a bone marrow and you look at the bone marrow biopsy, would be the best way to tell the cellularity, what you see is about anywhere from, say, 50 to 60 percent cells, and the rest is fat. So that would be a 60 percent cellular bone marrow. In a patient who has a florid leukemia, the fat may be totally driven out, and we have a hundred percent cellular bone marrow which is referred to as a packed marrow. In most people with myelodysplasia, their cellularity is normal, so they have about a 40 to, say,

60 percent cellular marrow. In the case of a hypocellular marrow, it's typically less than 20 percent. And so --

Q Less than 20 percent what?

A Less than 20 percent cells and 80 percent fat, if you will. So that would be a hypocellular marrow, a reduction in the number of cells per square inch.

Q Well, let's take the Dr. Irons' article that you've referenced.

A Yes.

Q I think it's Exhibit 4.

A Yes.

Q For the 649 MDS patients that he was studying, how many of those were hypocellular using your definition?

A He doesn't tell us that information.

Q Does he in any -- strike that.

Do any of the literature that you have with you address that as far as examining percentages for hypocellularty, looking at patients and determining how many are hypocellular versus hyper?

A Well, there are discussions in here. I don't know that there's anybody who looks at a percentage. There are several papers in here that point out that hypocellularty is common among chemical-induced

leukemias and less common among -- I mean chemical-induced myelodysplasia and less common among de novo myelodysplasia.

And the other aspect of that is that we talked about classification. And one of the classification of myelodysplasia is myelodysplasia-U, meaning we can't fit it into any nice box. And in chemical-induced myelodysplasia, from our chemotherapy drugs and from benzene, they often fit into the U category. In other words, they don't neatly lend themselves to the classification we use for de novo disease.

Q I thought you told us earlier that in Dr. Irons' article that we marked as Exhibit 4 that approximately 80 of the 649 patients with MDS had some exposure to benzene?

A Yes.

Q How many of the 80 had hypocellularty using your definition?

A I don't know that because he graded the benzene exposure from trivial numbers to higher numbers, and he focused on the people with unequivocal, what he called his index cases, who had unequivocal extremely high levels in myelodysplasia.

Q How many people fit into that category of

unequivocal high levels of exposure to benzene?

A I think he has 23 patients in here.

Q And out of the 23, how many of them were hypocellular?

A Let's see what he says. Let's see if I can find his tables here.

MR. LUBEL: We're going to go off the record while he finds it.

(Recess from 2:47 p.m. to 2:47 p.m.)

A He says that he's got 14, or 61 percent of them, that are hypocellular.

Q (By Mr. Lubel) I misunderstood.

A 14 or 61 percent that are hypocellular, he's got 7 that are 30 -- that makes up 30 percent that are normal in cellularity, and he has 2, or 8.6 percent, that have an increase in cellularity. So that what he is telling us I think is what I said, that more frequently in a chemical-induced bone marrow or a benzene bone marrow you see hypocellularty. It's not a universal feature, it's a common feature.

Q So out of the 23 benzene-induced MDSs in Dr. Irons' article, approximately 61 percent were hypocellular using your definition?

A Yes.

Q Seven were normal?

A Yes.
Q Two had an increase. Is that hyper --
A Yes.
Q -- cellular?
A You have an increase, yes.
Q Instead of hypo it's hypercellular?
A Hyper, that's correct.
Q And that gives us 23 if we do the math, 14 plus 7 plus 2, correct?
A Yes.
Q How many had an increase in the eosinophils?
A He has 21 of 29 or 72 percent.
Q 21 of 29 of the benzene-induced MDSs?
A Yes.
Q 72 percent?
A Yes.
Q And are we able to match up of those 21 of 29 how many of those also were hypocellular?
A He doesn't show that in the -- I don't --
Q You understand I'm looking for the overlap?
A I understand you're thinking looking to see who had both features. I don't -- let's see if I can figure that out.
Q We'll go off record while you look at it.
A I don't see that in these tables that he has

this. I mean, he's this compared to his controls, but he doesn't give the -- he doesn't give a table of who had both.

Q Now, how many had ringed sideroblast?

A None. Oh, among the patients, none.

Q Among the benzene-induced MDSs, none?

A Yes, none.

Q I wrote down scavengers because the word you used was so complicated to spell. What do we call it?

A Let's see if he puts this in here.

Q What was that area? Just refresh my recollection.

A Histiocytes. Let's see how he describes it in here.

MR. LUBEL: Off the record.

A He says, "The predominant pattern of bone marrow pathology in benzene exposed MDS cases; namely, multilineage dysplasia, meaning that the cells look funny in all three cell lines, the red cells, the white cells and the platelets; abnormal eosinophils, hematophagocytosis. That's h-e-m-a-t-o-p-h-a-g-o-c-y-t-o-s-i-s.

Q (By Mr. Lubel) Does that last term refer to the scavengers you were talking about earlier?

A Yeah, they're phagocytizing or eating, good

cells eating the bad cells. You could put it that way.

Q They're like the infantry in the immune system, correct?

A Yeah.

Q First line of defense?

A Well, yeah, in some respects, yes.

Q So how many of the benzene-induced MDS patients that are referred to by Dr. Irons in Exhibit 4, that article, have the phagocytes?

A Phagocytes. He doesn't say. He doesn't give that in his table.

Q How many had abnormal chromosomes?

A He does have that in here. He has -- let's see how many. He has -- that was the interesting feature of his article because he didn't have large numbers of them with abnormal chromosomes. The frequency of clonal cytogenetic abnormalities -- let's see. Wait a minute here. 30 percent. Let's see. Oh, no. Benzene, excuse me, it was 24 percent.

Q Of the benzene-induced MDS?

A Yes.

Q 24 percent had abnormal chromosomes?

A Yes. He says the frequency of clonal cytogenetic abnormalities in all MDS was 30 percent, the most common being Trisomy 8. The next common most was

this deletion 20q, and in the analogous frequency in benzene exposed cases was 24 percent. So I take it that he found 24 percent of his heavily benzene exposed patients had chromosome aberrations.

Q Does he set out which particular chromosome defects or abnormalities are present in that 24 percent?

A Yes. I think he says Trisomy 8 is the most common and the next is minus seven.

Q Does he have any other listing of what particular chromosome abnormalities he found? In other words, does he give you the raw data as opposed to just the conclusion?

A Yes. Whoops. Well, that's the over -- that's the overall group, the 649. Let's see if we can find benzene exposed group.

MR. LUBEL: We're off the record.

(Discussion off the record)

MR. LUBEL: Back on the record.

Q (By Mr. Lubel) From looking at both Exhibit 4, that Irons article from 2009 and the 2005 version that you brought with you, you did not see any data that reflects a breakdown of out of the MDSs that were thought to be benzene induced, what their particular chromosome defects were, correct?

A That's correct.

Q Do you have any literature with you today that you're relying upon that sets forth the particular chromosome defects that are necessary in order for an MDS to be benzene-induced?

A Well, I don't know that there are chromosome aberrations that would be necessary.

Q And, in fact, you've testified previously that it's not a prerequisite to be benzene-induced MDS for there to, in fact, have been any chromosome abnormality, correct?

A That's correct.

Q It's one of the clues that you're looking for?

A It's simply a clue. You're trying to make a judgment on what is probable and what's not probable, as we do in many diseases. And the chromosomes are one aspect of that.

Q And you've previously told us that having chromosome abnormalities puts it in a more likely category, everything else being equal, correct, versus not having it?

A Versus not having them. Well, I would say that having them, many people as they get older they have them. So having them certainly doesn't prove that one has it from benzene. It's almost in the reverse. Not having them is probably more significant than having

them, in a way.

Q Which way does it cut, not having them?

A In other words, if we say that -- if the articles are correct here -- that 80 to 90 percent of people that have a chemical-induced leukemia or myelodysplasia have a chromosome defect, then not having one becomes very significant because that's a one in ten phenomenon or so. But having one can be -- and it also depends on what it is. Having one can either be seen in de novo disease or benzene-induced disease.

Q And just from looking at Exhibit 4, that 2009 article by Dr. Irons, the fact that greater than 75 percent of the benzene-induced MDS did not have any chromosome abnormality tells us something, correct?

A It does, yes.

Q Those statistics are exactly the opposite of what you've seen in other literature where they say in benzene-induced MDS or AML you expect to see most of them having some chromosome abnormality, correct?

MR. HUMPHREY: Objection. Form.

A That's true.

Q (By Mr. Lubel) So Irons data, if you will, is opposite other data that you've seen, true?

A Yes.

Q And by "opposite," I mean it kind of reverses

the percentages the other way?

A It's a bit of a paradigm shift, you might say, in thinking, but one has to keep in mind that this is in a purely Chinese population. And another article I have referencing here suggests that chromosome aberrations among the Chinese are somewhat different than they are among what we would see in a Western civilization.

Q But given your many years of opinions on the record that there is not a prerequisite chromosome abnormality that is required in order for a cancer to be benzene induced, it wouldn't surprise you that Irons data that greater than 75 percent of them had no chromosome abnormality whatsoever?

A It surprised me based on the sum and substance of our medical literature, and Dr. Irons says it surprised him. He didn't feel that he was going to find that, but that's what he found and that's what he's reporting. And it leads him to think that benzene-induced leukemia is an unusual phenomenon and that it may be, in part, an inflammatory illness.

Q Which means what?

A Well, which means what? Which means this that --

Q Is that why you see different patterns?

A Well, I don't know. In other words, one theory

about this -- and many people have said the same thing is so, and we know it actually is so -- is that given a patient who comes in with myelodysplasia and no chromosomal abnormalities doesn't mean that they won't beget those chromosomal abnormalities as you continue to follow them over time. And the question is the when did you catch the disease? And what Irons is suggesting, I think, in part that he may see a person with an inflammatory type of bone marrow from benzene, and as that patient is followed over a number of years, they may develop chromosome aberrations suggesting that the chromosomal aberrations are not the cause of the disease. They're a secondary phenomenon. And that's an interesting thought, but actually borne out because even in Chromosome 20 disease, we see some patients that didn't have it and then they develop it as the myelodysplasia is followed. So it may well be that the chromosome in all cases isn't associated with the causation. It's a secondary phenomenon.

Q That means we just don't know right now?

A Well, he's opened up a paradigm shift and if what he finds can be reproduced. And one of the difficulties there is there aren't very many people who are swimming in vats of benzene these days. There certainly are in China. So he has a very unique

population to study there, and some of this work, unless the Chinese decide to do it or other investigators do, is really going to be difficult to, let's say, duplicate in this country, for example.

Q What are the main populations that have studied for exposures to benzene? We have Pliofilm, correct?

A Pliofilm is one. That's a series of about 1800 of people or so.

Q We've got the population that Hayes and Yin are studying over in China?

A Yes.

Q We've got Dr. Irons study population in China?

A Yes.

Q We have Aksoy's Turkish shoe workers, correct?

A Yes.

Q Who else do we have?

A Well, the ones that you're talking about that you mentioned are people who have had very heavy, presumably very heavy benzene exposures. There are several. I've got references here to several articles of highly exposed populations, a more modern population of shoe workers, some chemical called caprolactam or something close to that that's been studied. There are other populations out there that have been studied with high dose benzene.

Q Let's go through each of them, and let's talk about the pattern that you say is typically seen. We've already talked about Dr. Irons?

A Yes.

Q He's China group, right?

A Yes.

Q Let's talk about the Hayes and Yin population?

A Okay.

Q Do you recall whether any of their studies refer to the pattern or the details of the pattern, such as hypocellularity, the chromosome defects, things of that nature?

A Well, the cellularity, yes. Dr. Richard Larson has commented on that. And if I can find a reference list. This is my Reference No. 58, which is by the Hayes group, and they make a comment here, "Overall findings for benzene-exposed workers with myelodysplastic syndromes included bone marrow hypocellularity and marked dyserythropoietic features such as multinuclearity, megaloblastoid changes, impaired hemoglobinization, abnormal nuclear shape." And so he also comments about the hypocellularity aspect in the based benzene study.

Q Is that morphology pattern identical to what you've described today?

A I think so, yes. That's what Irons is saying that there's marked dyserythropoiesis. And, in fact, he shows pictures of abnormal red blood cell precursors.

Q And so what does that article tell us about the chromosome abnormalities?

A This particular one -- relativity few of these people had chromosome analyses done, and in the Hayes study, so that I don't know that it tells us a lot about that issue. The same would be true in the Aksoy studies and the Pliofilm studies.

Q They didn't break down the chromosome abnormalities that were found in the people with benzene-induced malignancies.

A No, because in the case of the Aksoy studies and the Pliofilm studies, these were in an era where chromosome analysis with what's called banding technique, which was an accurate technique, had not been developed. And so I would suspect that very few of those patients had chromosome analysis.

Q Do you know as you sit here if any of them did?

A I don't know that any of them had it in the Pliofilm study and Dr. Aksoy's work was mainly in the Seventies and banding, I don't believe, was developed into the Eighties. So he couldn't have had accurate chromosome studies. I don't -- and he doesn't in any of

his articles discuss their chromosomes.

Q Did any the literature -- for instance, the Larson article that you referred to --

A Yes.

Q -- that's part of the Hayes and Yin --

A Yes.

Q -- series of articles on China?

A Yes.

Q And benzene?

A Yes.

Q Do they give us the raw data on hypocellularity?

A No, because they only have a small number of people with myelodysplasia. I think that they claimed about maybe perhaps seven patients, and I'm not certain that they had bone marrows on all seven. So you're looking at a small number of patients, not as many patients as Irons study.

Q Do they tell us whether there's an increase in the eosinophils?

A They do not.

Q Do they make a reference to whether there's ringed sideroblast or not?

A They do not.

Q How about the histiocytes or the scavengers, do

they make reference to that?

A No.

Q Have you brought with you the literature that you're relying upon regarding this pattern of morphology that you've talked about today?

A Yes.

Q Okay. Have you discussed it with us today, or do you have more in your stack?

A No, I have more.

Q If you would just pull that out for us.

A This is an article, my Reference 59.

Q If you would just pull out the articles you're relying upon, and we'll mark them.

A I think those three articles. We have discussed those two.

Q We'll mark those three articles as Exhibit 5.

(Exhibit 5 marked)

Q (By Mr. Lubel) In all three of these articles that we've marked as Exhibit 5, are they discussing MDS patients?

A I think so. The Ruiz article, I don't know that he uses the term MDS, but these are people who had pancytopenias and abnormal blood counts from chronic exposure to benzene. And so he's showing us what the bone marrow looks like in people who have chronic

benzene exposure. And he had a large number of cases, but he did bone marrows on 33 of them. So he had a substantial number of people that he studied, and in what he tells us is that eosinophils were increased above 3 percent in 21 patients or 63 percent. So he's finding things similar to what Irons is claiming. And he says in none of the patients ringed sideroblasts were observed, which is the same as what Irons find. And he says global hypoplasia, meaning that there was reduced cells, was observed in 82 percent of the cases in various degrees. So he is finding pretty much the same thing as the other people find.

He also points out that they have abnormal morphology among the red cell and other precursors. So he's describing myelodysplasia without putting the name on it.

Q But are you suggesting that all three of those articles have identical findings from a morphology standpoint?

A Very similar findings.

Q Are they identical?

A Well, they're identical in that they find an absence of ringed sideroblast. They're identical in they find a strong frequency of hypocellularity. They're identical in that they find an unusual increase

in eosinophilia. And they're all identical in that they find abnormal morphology among the myeloid cells.

Q How are they different?

A They aren't different.

Q So they're identical?

A Well, I mean, they're done under different conditions. In other words, they're done by different investigators, different conditions, but they're considerably similar, I would say. The percentages are not identical. They're similar.

Q Well, we know that Dr. Irons, the subset of the population that he's referring of MDS, he believes are benzene induced, correct?

A Yes.

Q Is that also true for Ruiz and Linet? I thought you called it Larson earlier but...

A Well, he's the guy, I think, that looked at all the cells.

Q Okay.

A He's the hemology who's in that group from the University of Chicago.

Q Are all three of the articles focused on MDS patients that they believe are benzene induced?

A Yes, in different ways. In other words, in Irons' case and in Ruiz' case, they feel that they know

their patients were benzene induced because they have the evidence. In the case of Ruiz, there was a broken pipe in this factory, and all these people were sniffing giant doses of benzene every day for months and months. And so those are pretty clear that they were benzene induced, and he had a lot of them.

In the case of the Linet study or the Hayes study, this is a group that they feel are benzene exposed and -- but, you know, like the Pliofilm group, if you're taking a collection of people and saying I've got an increase in leukemia, there is some background, in other words. And it's hard to tell, if they have seven patients with myelodysplasia, to tell which one was the de novo one, which one was the benzene one. They're inferring that among that group, there's an increased incidence of myelodysplasia from benzene, but they can't put a one-on-one designation for every one of those samples that they looked at, where in the case of Ruiz you can because they all were poisoned. So Ruiz' and Irons' I think are a little more specific than the Hayes studies, but in general they have similar findings.

Q What difference does the level of exposures have on the morphological patterns?

A I don't know the answer to that. In other

words, what's being suggested here is whatever that level exposure was, it was high enough to cause considerable change in the normal marrow, and presumably they were very high levels. Dr. Irons suggests his people had enormous levels. Dr. Ruiz' people probably had enormous levels. A lot of people dispute what levels there were in the Hayes study, but they were likely high.

Q I mean, have you seen -- we know that not all of the benzene-induced malignancies have a chromosome defect?

A Correct.

Q And out of the ones that do have any chromosome defect, they're not necessarily the same defect?

A Correct.

Q And so we're not seeing a pattern that is identical? In other words, there's not a fingerprint that tells us each and every time?

A That's correct.

Q And so what I'm trying to figure out is whether you've seen evidence that the exposure level changes the morphology?

A Well, presumably these 33 people he studied were perfectly normal before they got their exposure. I mean, these were all healthy factory workers who got

poisoned. And there were many more than 33, but he picked 33 who had the worst blood counts to do bone marrows on.

Q Do we know whether the exposure level can affect the outcome of whether somebody has a chromosome defect versus whether they don't?

A Ah, I see what you're saying. I would say no, we don't know that.

Q Do we know whether -- it's the same line of questions -- whether the benzene exposure can affect whether it's hypocellular or the degree of hypocellularity?

MR. HUMPHREY: Form.

A Well, presumably very high levels can cause hypocellularity. More subtle levels, we don't know. In other words, we just know what the extremes are.

Q (By Mr. Lubel) Okay. So we haven't seen these studies on the lower levels and the affect on hypocellularity?

A No, not of what I have referenced here.

Q But have you seen such studies?

A No, I've seen studies looking at the blood cell counts, the peripheral blood cell counts of patients alleged to be exposed, but not bone marrow analysis.

Q Do you have an opinion as to what exposure

level it takes to cause hypocellularity?

A Not a number. I couldn't put a number on it that.

Q How about an increase in eosinophils?

A The same thing.

Q Does the exposure level, if you know, determine whether there's ringed sideroblasts or not?

A Well, all we can say is that the -- none of the exposed patients have ever had any ringed sideroblasts, regardless of what their levels are. And so we would have to say there's no evidence that benzene exposure can cause ringed sideroblast.

Q You're saying there's been studies done as to whether benzene exposure can cause ringed sideroblast?

A Yeah. Ruiz did it. He specifically looked for them. In the case of Aksoy, he did iron stains on all of his patients. He was a well-trained hematologist, and he could recognize a ringed sideroblast. And his professor Dr. Dameshek wrote many types about ringed sideroblasts. And certainly Richard Larson and the people at University of Chicago know what a ringed sideroblast is. And there's no publication that suggests that benzene can cause a ringed sideroblast.

Q My question is: Were any of the studies that you brought with you looking specifically or studying

specifically whether benzene exposures can cause ringed sideroblast?

A Absolutely.

Q Okay.

A They were specifically looked for by Ruiz. They were specifically looked for by Irons.

Q Do you understand there's a difference between a finding or an observation and designing a study to determine an outcome?

A Yes, the studies were designed to look for that phenomenon.

Q You're saying these three studies were designed to look for ringed sideroblast?

A Yes.

Q And determine whether, in fact, benzene exposures could cause ringed sideroblast.

A That was what -- they did iron stains on the bone marrows, and the iron stains light up the ringed sideroblasts, if they're present. So if they weren't looking for that, they wouldn't have done iron stains. And Ruiz specifically comments on it. Iron specifically comments on it. So certainly they were looking for them.

Q Looking for them and studying that as an outcome are two different things, correct?

MR. HUMPHREY: Objection. Form.

A I think in the context of these studies, they're identical.

Q (By Mr. Lubel) Do they set forth in these studies a statistical analysis --

A No.

Q -- on the ringed sideroblast?

A No.

Q How rare are ringed sideroblasts?

A They don't occur in a normal bone marrow.

Q How rare are they in MDS?

A They're not --

Q Just in the general population?

A Just that general population of people with -- well, I would say that possibly, let's say, 10 percent of people will have RARS; that is to say, they will have huge numbers of ringed sideroblast. Others may have small numbers of ringed sideroblast that don't meet the category for RARS. I wouldn't say it's rare. I would say -- pick a number, I would say you could find ringed sideroblast in possibly 20, 25 percent of people with MDS that you studied. So you would certainly expect to see ringed sideroblasts in many people with MDS.

Q Same with AML?

A No, it would be less in AML. You would be less

likely to find ringed sideroblast in AML. But the bottom line is: So when you don't find any in benzene-induced myelodysplasia, that suggests strongly that benzene doesn't create that phenomenon.

Q Mr. Hall had a chromosome defect, correct?

A Yes.

Q How do you refer to his?

A It's called 20q minus.

Q How was it determined?

A By a standard chromosome analysis.

Q Do you have it with you, the medical record that did it or that revealed it?

A I'm sure it is in there. Right here.

Q This was done by St. Luke's Hospital?

A Yes.

Q Do you know the doctor that did the test?

A Well, Dr. Escudier, who I know, did the bone marrow mechanical aspiration. This was likely sent to a reference laboratory. I don't know if they do them on site in St. Luke's or they send them to M.D. Anderson or they send them to Baylor. But they likely send them to either Baylor or M.D. Anderson.

Q Have there been any other cytogenetic studies done other than this one?

A I don't think so.

Q Do you agree with the interpretation, second sentence it says, "Deletions of the long arm of chromosome 20 have been reported in myelodysplastic syndromes, especially RARS, as well as polycythemia vera and AML, including all subgroups except for M3"?

A Yes, that's a correct statement. Let's just see if we can who did this. It's got a medical director. It doesn't say what she's the medical director of. I don't know. You asked where this was done, and it's got the medical director's name, but I don't know what she's the director of.

Q What is the purpose of this test? Why are they doing it?

A Why are they doing it? Well, I think that when you're making a diagnosis of myelodysplasia if you see a chromosome defect, No. 1, it's a little bit more confirmatory of the diagnosis. You can certainly make the diagnosis clinically on the basic morphology, but it's comforting to know that there's an abnormal chromosome. That lends credence to what you see with the eyes.

Secondly, the nature of the chromosome defect often determines the clinical cause, and it determines the therapy, for example -- and the prognosis. Many people with 20q, for example, live many

years. It's considered a, quote, "good chromosome aberration." It often responds to treatment. The same with the 5q minus syndrome. And other aberrations sometimes portend a very bad prognosis. And so you do this to try to classify the illness better.

Q Are they looking at treatment options?

A Yes, both classification and treatment. In the case of the 5q minus, the best treatment is a drug called Lenalidomide. In the case of the minus 20, they often respond to erythropoietin injections.

Q It says, "Consider sending at the appropriate interval a follow-up bone marrow for chromosome studies should the clinical course persist or deteriorate." Does that make sense to you?

A Yes. I mean, it's arguing with apple pie. In other words, it's like the radiology report saying here's an abnormality, I suggest you repeat that in two months to see if it went away or got worse. And I think most clinicians making a diagnosis of myelodysplasia, unless there was a major change in the blood counts or the circumstances, wouldn't repeat that marrow any time soon.

Q Well, how could the chromosome abnormality go away?

A It does go away. That's the interesting thing

about it. If you take people who have minus five, for example, so-called the 5q minus and you treat them with Lenalidomide, in something like 60 percent of the time it will disappear and the bone marrow comes back and looks like normal. Now, it may not stay gone, but it does disappear. It's quite remarkable.

Q But without treatment?

A That is treatment. Lenalidomide is an immunosuppressant drug.

Q But if you have no treatment?

A Oh, yes. Yes, it can disappear if you have no treatment.

Q How?

A Well, I can just give you a typical example. I wrote a paper on a nice lady who got poisoned, and she got myelodysplasia. And she had what's called a Trisomy of the 15th chromosome, and it was present in about 60, 70 percent of her cells. And her husband was an attorney and very nice people, and I couldn't decide whether she needed a marrow transplant or observation. And so I decided I would observe her. And over the years I periodically repeated her bone marrow, and the frequency of that chromosomal aberration that was originally in about 60 or 70 percent of her cells went down to about 50 percent of the cells, went down to

about 20 percent of her cells and her blood counts improved with no treatment whatsoever over about an 18-year period. I've never treated her. She's still living.

And I didn't know what to make out of that until -- and, in fact, I published her case in the literature. And then some additional articles came out, and they found the same thing. And they said that with the particular chromosome aberration she had sometimes you can outgrow it. In other words, the normal cells will grow better -- something caused that defect to occur. And somehow the normal cells will gradually over time overwhelm it. And they say it's most likely to happen if you have 20 percent of the cells or less with the aberration. When you have 80 percent of the cells with the aberration from the getgo, it usually goes off into leukemia or MDS. And this lady I had was sort of on the borderline, and she lucked out. And the normal cells have gradually taking over her blood marrow.

Q But are you limiting the chromosome defects disappearing to that particular chromosome abnormality?

A No. As I say, that's well described with that particular one. It's well described with 5q minus, particularly when they're treated with Lenalidomide. In most other cases, they don't go away and generally get

worse with time.

Q Meaning you can take a picture and see a particular chromosome defect, and it can change over time?

A It can change, but usually for the worse, but occasionally for the better.

Q But even the type of chromosome abnormalities can change, depending on when the picture is taken, right?

A That's correct. They may -- Inversion 16 leukemia is like that. They may have the Inversion 16, and as much as 40 percent of the time they may develop other chromosome aberrations.

Q I've not heard you say, but I want to make sure the record is clear. You're not claiming that the particular chromosome abnormality that Mr. Hall had cannot be induced by benzene exposure, correct?

A Well, I don't know that it cannot be induced by benzene exposure. All I can tell you is that if you pick up one of any of number of papers on that aberration -- and I have several here -- it's not been described with benzene exposure, and none of the authors attribute it to benzene exposure. I can't prove to you that benzene exposure can't cause it. In Irons study he didn't find it in the benzene exposed people, in

contrast to the fact that he did find it in the benzene unexposed people.

Q He said he didn't find it?

A He didn't find it in the benzene exposed. He did find it in the unexposed.

Q He said he did not?

A Did find it in the unexposed.

Q Oh. In the exposed, you're saying that Irons said he did not find it?

A Irons said that he has -- and he showed slides at a meeting I had with him several days ago, more than are in this paper of his because he's getting ready to publish other aspects of that study. But he found not a single patient in the benzene exposed group with a 5q minus and not a single patient with a 20q minus.

Q That's what his article says, what you're saying?

A Well, this article, I think, talks about the 20. I'm not sure it says about the 5. But he says that he is unable to find that in a benzene -- those two aberrations in a benzene-exposed population; whereas, he does find them in his unexposed myelodysplasia at the same rate that everybody else finds them in the general population.

Q So you met with him recently?

A Yes.

Q Regarding?

A It was another case that Bob Scott had and Richard Irons was in town and I was invited over to sit in on his discussion. He was presenting some of what's in this article. And --

Q Who all was present?

A Bob Scott was, Stan Perry was. I'm trying to think who else was there. Mr. Gray was there. Richard Irons, myself, Ms. Randolph who works for Bob Scott. Mike Baker was there. I think that's it was at that meeting. And he -- I was invited over there.

I had met him many years ago, probably 18 years ago was the only time I've actually met him; although, I've talked with him over the phone a couple of times in the interim. And he was in town and presenting his stuff, and so I listened in on it.

Q What stuff was he presenting?

A This study that we're talking about that he's published here, his results.

Q Exhibit 4?

A Pardon me.

Q The Exhibit 4 study?

A Yes.

Q Was he showing you something that's not in this

published study that you've marked as Exhibit 4?

A He showed a number of slides. I don't think all of them were in that. He showed -- some of the ones that were, I remember distinctly the blood cell pictures. He showed those and he showed a table of chromosome aberrations and I think he had data in there that was not in this article.

Q Okay. Do you have it?

A Do I? No. No, I was just --

Q You didn't get a copy of it?

A No, I was just --

Q Why not?

A Because he was showing a PowerPoint of it.

Q Did you ask him for it?

A No. He was just giving a little you might off-the-cuff lecture of what he found.

Q How long did that lecture last?

A They were talking when I got there, and they were talking when I left. I was there probably an hour.

Q And it was for a case?

A Well, I don't know. I assume so. You know, it was -- I don't know if it was structured for a case particularly. I think he is an expert witness on a case that they have, but I don't know if the purpose of the meeting was solely that case or to hear what he had to

say about this study. From my interest in it, I was interested in what he presented.

Q Who invited you?

A Bob Scott did.

Q Do you work on cases with Bob Scott?

A Yes.

Q Which cases right now?

A I'm terrible on names. I have a case with him. And hang on, maybe I can think about it a little bit. It's going -- I can't remember cases.

Q Of all the lawyers you referenced that were at that meeting --

A Yes.

Q -- that attended, you work for them?

A From time to time, yes.

Q Tim Gray?

A Yes.

Q Mike Baker?

A Yes.

Q Bob Scott?

A Yes.

Q Steve Dillard?

A Well, he wasn't at that meeting.

Q But you do work for him?

A I have. Not for some time, but I have.

Q Stan Perry?

A Stan Perry, yes.

Q And, in fact, one or more of those lawyers have reviewed your articles that address benzene before they've been published, correct?

A I would just say one. Steve Dillard reviewed one of my articles as probably the eighth or ninth reviewer before I sent it in, yes.

Q Who were the other reviewers?

A Two of my professors from medical school Ed Lynch and Herb Fred, a couple of the other hematologists in my office, my wife. Attorneywise he was the only one who reviewed it.

Q And do you have the commentary he made?

A He didn't make any commentary. I sent it to him for his comments, and he said he liked it. And he didn't have any particular comment.

Q No changes?

A No, he didn't change anything.

Q No suggestive changes?

A No. He said he liked it, and he enjoyed it. I was interested to see if it was understandable to a person who has a layperson as opposed to an M.D. but had a substantial knowledge of the field, and I just wanted to understand if it was readable.

Q So what data did Irons present to you in the meeting with the lawyers over at Mr. Scott's office in the last couple of days that's not in his published article?

A The one feature that I remember -- and I can't even quote the numbers -- but he had something to the effect that in the Hayes article and the Richardson article and some of the others, they talk about the fact that recent exposures to benzene causes the problem. He had some data on that and essentially claimed the same thing, that where he saw aberrations from benzene, they were basically the consequence of recent exposures, not late exposures.

Q That's not in his article?

A That's not even mentioned in his article.

Q What data did he have to support it?

A I don't remember. He made that statement, and he was showing a slide. I don't remember exactly what was on the slide. But he made the statement that that was what he found, that recent exposures were most important.

Q Did you ask him why he didn't put that in his article?

A No.

Q He did not show you the underlying data that

supported the statement?

A I don't think so. He had some slides up and they were up when I came in and he was showing more stuff when I left. But the key things I got about it, about that meeting was the morphology. He showed all the pictures of that and the chromosome aberrations, and he talked about the fact that he didn't look vigorously, but didn't find these so-called interstitial defects like minus 5 and minus 20.

Q What does that mean?

A Well, it just means that you got a piece of a chromosome, and you're not losing the end of it, you're losing something in the middle. It's sort of interstitial. That's what a minus -- you're losing a piece. That's the minus. And the Q, that stands for bands that are in that chromosome. And the 20q minus is not always the same in every person. In other words, they may lose a large piece or a smaller piece or a different piece, but it's a piece from the long arm of that chromosome.

Q How long did you look at slides, Dr. Irons slides?

A I was there about an hour, maybe 15 minutes or so of it --

Q Were slides?

A Were slides, yeah.

Q What did you do the other 45 minutes?

A We just talked in general. I'm trying to remember if there was any particular subject. It had to do with his studies, and I'm trying to recall what, if anything, I asked. Oh, I asked him in detail about how these guys got their exposure to benzene. I was interested to see in terms of the eosinophilia was there a way that they were getting their benzene. In other words, was it tire manufacturing. What was it, to know if there was something else that was causing that eosinophilia, some other chemical. And he said, for example, that what they commonly do is he'll go to a factory. They will have an oaken cask, a gigantic cask with a wooden top. It's totally full of pure benzene, hundreds of gallons of it, and they dissolve raw rubber in it and get gigantic exposures in that room that they're doing it. And he said so some of those people are from the rubber industry. And he said no one in the world any longer dissolves rubber like that, but benzene is the best solvent for rubber. And so he mentioned that.

He also mentioned the fact that -- I said, do these people ever get promoted? And he said, no. He said, the way it operates in China is that you're a

cauldron stirrer, you keep being a cauldron stirrer. There's very little room for advancement. You have that job and you keep that job, unless you get sick and leave. So we talked about the demographics of what he found a little bit in terms of the job work. Those are the things that come to mind that I can recall.

Q That's all you can remember?

A Yeah. I mean, as I say, I wasn't -- the meeting hadn't started actually. I must have gotten there, I'm guessing, like 1:00 o'clock in the afternoon, and I think he got there like 10:00 o'clock in the morning. And so the meeting had long been going on when I got there, and I just stayed in there for a while. I had to get back to work, and I left. I was there maybe an hour.

Q Do you know which of the oil companies paid Dr. Irons for that meeting?

MR. HUMPHREY: Objection. Form.

A No.

Q (By Mr. Lubel) You realize that his work has been funded by a number of oil companies?

A Yes.

Q For instance, Exxon?

A I don't know who all funds his -- funded his study. He told me when I spoke with him on the phone

some months ago a little bit about the study, that his funding was running out. And so what he was doing is this laboratory he had developed was sort of being converted to like a for-profit laboratory for all the hospitals in the area and that it's going to have to stand on its own as a profit-making institution.

Q Is he done with his work?

A I don't know the answer to that.

Q The article that you've brought with you --

A Yes.

Q -- that we've marked as Exhibit 4, is that the final piece of publication to come from Dr. Irons?

A I don't think so because he suggested that he was -- that he was doing something other with this group and I think there's going to be at least another forthcoming article; although, he didn't show it at the discussion there.

Q What is the topic?

A I don't know. But, I mean, it would have to do with benzene.

But I think this article is because -- I'm guessing it might have something to do with the latency because he brought up the business about the recent versus the late exposures, and none of that was in this article. And so I assume he's working on some other

stuff.

Q Have you seen the articles from Dr. Irons where he addresses the exposure levels?

A Well, he says something to that effect in this article. He has, like, four or five levels of exposure. That's in this article.

Q So they do a -- is it like a case control analysis where they look at each disease status patient and see what their exposures were?

A I can't tell you in that detail. In this article he gives sort of level ranges, as I recall, and in terms of what their exposure is. He's got maybe four or five different levels of exposure.

Q And so do you recall whether the article tells you where there's an increase in risk?

A No, it doesn't get into that. I think he's working with the people with the highest exposures.

Q So to your knowledge, Dr. Irons is not evaluating what exposure levels can cause disease?

A As far as I know, that would be true.

Q In any of your conversations or meetings with him has he suggested otherwise?

A No.

MR. LUBEL: Let's take a short break.

(Recess from 3:51 p.m. to 3:56 p.m.)

Q (By Mr. Lubel) Have you brought any literature with you today that concludes that the type of chromosome abnormality that Mr. Hall had cannot be induced by benzene exposure?

A No.

Q Have you seen any?

A No.

Q You make reference to a Surgeon General report on smoking?

A Yes.

Q Do you have that with you?

A Yes. It's an excerpt from that report.

Q Is there more to it than this?

A Yes, but I don't -- I mean, it's a book, basically. But that's just a synopsis of part of it.

Q Have you seen the book?

A I used to have some -- a disk of the book. I don't know if I still have it or not, but I may.

Q Have you studied the underlying data, if you will, or studies that they refer to?

A No.

Q When you look at the leukemia studies that involve smokers --

A Yes.

Q -- what do they tell you about the morphology

pattern that you've referred to earlier, hypocellularity increase in eosinophils, ringed sideroblasts, histiocytes, chromosome defects, et cetera?

A There is no information about that because it's very difficult to isolate a person's leukemia as purely being caused by cigarettes. So there are some suggestions in some of these articles about certain chromosome aberrations, but there really is no data on what the bone marrow would look like as opposed to any other person's bone marrow with these diseases.

Q It's not been studied, to your knowledge?

A Well, it's difficult to study because it's hard to isolate smoking from other things a person might do. And I've not seen a report that says this is what a bone marrow looks like in a smoker's leukemia.

Q To your knowledge, it has not been studied specifically?

A Correct, I've never seen such a study.

Q Now, there's other secondary causes other than benzene that can cause MDS or AML, correct?

A Yes.

Q Like we've talked about, chemotherapy, radiation, things like that?

A Yes, autoimmune diseases.

Q Let's talk about chemotherapy for a minute.

A Yes.

Q What is the morphology pattern to chemotherapy?

A Well, it's variable, but in general hypocellularity is prominent. It's not a universal finding, but hypocellularity is often seen in very abnormal morphology. The red cell precursors are the ones that are usually most evident.

Q How about radiation?

A Well, radiation can also obliterate a bone marrow and make it hypocellular.

Q Does it typically?

A Frequently, yes.

Q Frequently or typically?

A I'd say --

MR. HUMPHREY: Form.

A -- frequently.

And, again, it's looking at the timing. In other words, after an area of marrow gets radiated, it's very hypocellular. It may recover as time passes to a certain degree.

Q (By Mr. Lubel) How about the increase in eosinophils?

A I've never seen that with radiation, and I'm not aware of that with chemotherapy. I don't think I've ever seen that. Sometimes that happens with

Inversion 16 leukemia and a few other leukemias, but it's not a common event.

Q Are ring sideroblasts common from either chemotherapy or radiation?

A Not in the numbers that we see with refractory anemia and ring sideroblasts. That would be very unusual. It's been reported as a transitory phenomenon with chemotherapy. I think I reference that in my article. And you occasionally can see a few ring sideroblasts in many sick bone marrows, but to see the numbers that you see in what we call R-A-R-S would be very unusual.

Q For both chemotherapy and radiation?

A Yes.

Q How about the histiocytes, the scavengers?

A I think I've seen that and not paid much attention to that in other bone marrows. I don't think that's a specific finding.

Q So it sounds like in your opinion the benzene-induced leukemia or MDS the morphological pattern is different than what you would expect for chemotherapy or radiation?

A I think that there is a subtle difference. The eosinophilia I don't attribute to our standard chemotherapy or radiation. The hypocellularty could

certainly occur with chemotherapy or radiation, and both instances -- all three instances could cause chromosome aberrations of different types. There would be similarities.

Q But there's not identical overlap?

A There's not an identical overlap, correct.

Q And you wouldn't expect there to be given that they're different substances, correct?

A That's true.

Q Even within the chemotherapy category we're talking about multiple different types of substances?

A There are multiple different types of chemotherapy drugs, yes.

Q And they don't all do the same thing, right?

A They don't, that's correct.

Q Some treat particular forms better than others, right?

A Yes.

Q That's why they're developed?

A Yes.

Q The time course between onset of chemo -- you know, between chemotherapy treatment and onset of MDS or AML has been viewed differently or the observations have been different depending on the type of chemotherapy agents, correct?

A That is correct.

Q Even within categories of chemotherapeutic agents?

A Well, in general the so-called topoisomerase inhibitors, of which there are several, they generally have a short latency and the alkylating agents generally have a little longer latency, but it's quite variable.

Q Variable in what?

A Well, if you give somebody alkylating agents you might see a leukemia in three years or you might see it in eight years, but if you're talking topoisomerase inhibitors, most of them they occur, I'd say, within the first three years after you've accumulated a significant does. They're very early on.

Q But at least one of the explanations between the difference in the alkylating agents and the topoisomerase agents is the fact that they're different chemicals?

A They're definitely different chemicals, yes.

Q They have a different structure?

A Different structure.

Q And the benzene is different than both the chemotherapeutic agents, correct?

A Correct.

Q Your opinions about the dose necessary to cause

AML or MDS, that has not changed, has it?

A That's correct, it does not change.

Q And I've heard you say before that you'd, under the right circumstances, agree to as low as 40 ppm years, correct?

A Yes, because I think that the measurements are very inaccurate, and a 40 might really be an 80 or a hundred or higher. I would just say it's very high doses and certainly much higher than we were told in this case.

Q And if we look at the Lichtman article which you've cited -- do you have it?

A Yes.

Q I think I turned it sideways.

A I have to consult my list again and see --

Q Take your time.

A -- where that was.

Q I think I turned it --

A Well, there are two articles. There's one that has to do specifically with smoking and then this other one.

Q I think it's in '08.

A The smoking one is an editorial.

(Exhibit 6 marked)

Q (By Mr. Lubel) Before we get off the smoking

issue, Exhibit 6 that I've marked comes from your file. It's your Surgeon General reference, correct?

A Yes.

Q You're using this for part of your opinions, correct?

A Yes, in general that cigarettes are associated with MDS and acute leukemia, although that's specific to acute leukemia.

Q In other words, this particular reference is not specific to MDS, but you're using it for that, correct?

A Well, I'm just using it because smoke -- we generally think that if something causes leukemia it's likely to cause MDS, and that is discussing leukemia.

Q And it's entirely appropriate, is it not, for you or others like you to use the leukemia literature in an MDS analysis, correct?

A It is. They are certainly not the same illnesses, but they have similarities.

Q They're similar enough that you use that base of literature in your MDS cases?

A Yes.

Q Including lawsuits, not just patients?

A Yes.

Q Now, let's go back to the Lichtman article.

It's a -- looks like 2008. You're familiar with it, correct?

A Yes.

Q In fact, it cites yours, doesn't it?

A It does, yes, correct. I was quite surprised.

Q It says --

A I don't know the author personally. I've never met him.

Q It says: The major subtype of leukemia can be a result of long-standing cigarette smoking. Do you agree with that?

A That cigarette smoking may cause leukemia, yes.

Q Or relatively high level prolonged exposure to benzene greater than 25 to 40 ppm years. Do you agree with that?

A Well, I think it's higher than 40 and so -- but it's high.

Q What does it say there? It's the sentence right underneath your highlights.

A It says: Prolonged exposure to benzene greater than 25 to 40.

Q PPM years?

A It does. But, of course, the greater than 25 doesn't tell you how much greater. In other words, it's more than 25.

Q It cites your article?

A But my article doesn't say greater than 25 to 40. My article says greater than 40.

Q Okay. So is he wrong?

A Well, I don't know that he's wrong. He's citing my article. But I didn't say greater than 25 to 40. I said -- we can look at my article, but I think I said greater than 40.

Q We wouldn't know that he's misquoted you without going to your article, correct?

A That's true.

Q Is there anything else in here that we know he's wrong about?

A I don't know. You'd have to look -- it's a long article. We'd have to go through it.

Q That's one of the difficulties inherent in review articles, right?

MR. HUMPHREY: Objection. Form.

A Well, a review article is what it is, and you you're looking to see what the general opinion is about a particular illness.

Q (By Mr. Lubel) Treating doctors don't have the time to go through and look at all the citations in a review article, do they?

A That's correct.

Q Time does not permit treating doctors, whether they be hematologists or oncologists, in that field or other fields, to not only review review articles pertinent to their field but also go back and look at all the citations to see if what was stated was accurate?

A That's correct.

Q The standard of care does not require medical doctors to check the literature to see if what the author is stating is accurate?

A That's true.

Q You say most patients with MDS do not progress to AML in your report?

A Yes, that's true.

Q But you also say that an AML that has preceding MDS is more likely to be benzene induced everything else being equal?

A Well, what I said -- we can see what I said. But basically a chemical-induced leukemia is more likely to have a myelodysplastic phase. About two thirds of the time if you have a chemical-induced leukemia, there's a phase of myelodysplasia before the AML becomes apparent.

Q Well, for a leukemia case does having MDS break in favor of it being benzene induced or against it?

A Well, it would be a point that could be used in favor of it, yes.

Q It looks like, just as we've briefly touched on earlier, that you're using certain leukemia or AML literature on exposure levels for MDS, correct?

A Yes.

Q In your opinion that falls within the standard of care to do such, correct?

A I think so. That's the World Health Organization suggesting that that's the case, and I think that's a general opinion. In other words, we accept the fact that benzene may cause MDS, and we generally have no reason to think its levels are any different than that of AML.

Q And that's why you've relied upon particular literature on AML that addresses exposure levels?

A Yes.

Q There's a paragraph...

MR. LUBEL: Does he have your copy?

Q (By Mr. Lubel) It's on Page 5, Dr. Natelson.

A Page 5. Okay.

Q It's in the center. Do you see it?

A Yes.

Q Are you addressing the concepts of epidemiology there.

A In a sense. I'm not an epidemiologist. These are general statements.

Q Well, you're pretty clear that you're saying there should be a consistency of an association among the studies, correct?

A That's what you would like to see. In other words, if one study is very aberrant, it could be correct, but you would like to see some consistency.

Q And do you agree with the Union Carbide epidemiologist, Dr. Jane Teta, that statistical significance is not required in order for the studies to provide meaning in the epidemiological sense?

MR. HUMPHREY: Objection. Form.

A That would be beyond my expertise to comment about.

Q (By Mr. Lubel) Have you seen Dr. Teta's deposition?

A No.

Q Would it surprise you if she said you don't need statistical significance in the studies, what you're actually looking for is a trend of consistency among the studies?

MR. HUMPHREY: Objection. Form.

A I don't know that that would surprise me.

Q (By Mr. Lubel) Would you defer to an

epidemiologist or toxicologist in that area?

A To analyze that comment, yes.

Q Well, it doesn't look to me like for this particular case that you've pulled all of the studies that address lower level benzene exposures in AML or MDS, correct?

MR. HUMPHREY: Objection. Form.

A Well, I mean, I -- that's probably a true statement. I've looked at some modern studies.

Q (By Mr. Lubel) In other words, do you have the -- do you have the studies that Dr. Gardner lists in his chart?

A I --

Q Have you seen those recently?

A Not recently.

Q Did you get Dr. Gardner's report?

A It's got to be in there. I'm sure it is in there someplace, or maybe it isn't. I have his deposition. I don't know if I have his report. Let me see. Maybe this is it. Let's see.

Yes, I have an affidavit of Dr. Gardner.

Q Do you see that chart? What page is that?

A This is -- there's no page number. Appendix 4.

Q Do you recognize that?

A Well, I have these articles. I've referenced

Bloemen. I've referenced the Glass study. I have Collins. I have Rushton. I don't -- the Glass study doesn't suggest to me that low dose benzene causes myelodysplasia. In fact, she doesn't even discuss myelodysplasia. And the Bloemen article has to do with 40 part per million exposure causing AML as statistically significant. So I would doubt that these articles show consistent statistically significant evidence that low dose benzene causes myelodysplasia.

Q Well, I thought you said earlier it was appropriate to look at the AML literature?

MR. HUMPHREY: Objection. Form.

A Yes, it is. Glass is an AML, and she shows a deficit of leukemia in her study. So she shows that low-dose benzene doesn't cause AML, and she didn't study MDS. She shows nothing.

Q (By Mr. Lubel) She shows nothing?

A There's nothing in her study that suggests she has a deficit of leukemia in the study that she has, the most recent edition. She has like 11 cases with 12 and a half or so expected in a group with a mean level of benzene around 4.9 part per million years. So she shows no evidence that low-dose benzene in that group causes AML.

Q She's never said that before?

A She has said that. She did a subset analysis --

Q Oh.

A -- many years ago of, like, 79. There are 18,000 people in the group, so she picked out 79 and did a questionnaire study, which, of course, would be full of recall bias. And she came to what I consider erroneous results because the purpose of this study was not to cut out little subsets but to look at 18,000 people studied over time, and when you look at that, there's nothing there.

Q What's your criticism of the questionnaire part of it?

A Because I think some of these people were dead when that questionnaire was issued, and so it was -- and the questionnaire was answered by -- not even by the patient. But I think she said there were four different sets of questionnaires. My understanding was that some of those were answered by the family or coworkers. There were no specific measurements. And so you're talking recall bias on top of recall bias, and that's why you come up with number like that. And now she has to recant because she can find nothing. She finds no increased incident no matter what site they worked there, no matter how long they worked there. Looking at

the whole group, there's nothing there.

Q She recanted is what you're saying?

A That's the way I read in her latest report. She says, and I quote it in my article, that there's no increased incident with any site, with any duration of employment, and that the studies dealing specifically with AML are too small to do anything statistically. And that's what she says in her article.

Q Do you know the difference between a cohort study and a case control study?

A I wouldn't speak to that expertly. I have general knowledge, yeah.

Q Do you have an understanding as to whether the studies you're referring to are two different types that she did, one was a case control and one was a cohort?

A Oh, they may be a different study, but she's taking -- she's doing a subset analysis. She's taking a small group of 79 people from a group of 18,000 and trying to make sense out of that and when you do that you introduce all kinds of bias and your control group may not really be a good control group. And that's not the purpose of that study. The study was put together to determine what happened to these 18,000 people just like what happened to the 1800 people in the Pliofilm study. And as this study has gone along, we found out

that there's nothing there. There is no increased incidence of AML, and MDS, the illness here, isn't even discussed.

Q So do you have the same criticisms of Dr. Irons' work where he takes a subset of the MDS?

MR. HUMPHREY: Objection. Form.

A Because he's got a subset that has specific measured exposures to benzene, that's a whole lot different than taking a questionnaire to a person's relative. I think there's a subtle difference there.

Q (By Mr. Lubel) Is that your only difference in criticism?

A No, I think that Irons' study he's got people with major exposures, and he's finding a consistent result. In the case of the Glass study it's I think a subset analysis that when you look at the total picture of that study over time it's become clear that that subset analysis was inaccurate.

Q Why did the authors intend to do -- why did they do a subset analysis?

A I don't know why they did that.

Q Well, did they say why?

A I'd have to look again at the article to say why they even attempted that but they did do a subset analysis, yes, and that's what that was based on.

Q Is a subset analysis called a case control study, or do you know?

A A subset analysis could be. I think that's apples and oranges. A subset analysis is you're taking a large group and looking at a portion of it to determine if you can make some analysis. In other words, you might have people with heart failure you're studying and you want to know the people with heart failure who like to climb stairs and so you're going to select those out and see if maybe climbing stairs influenced their heart failure. It's a subset analysis. It could be matching it up with controls from another series, yes.

Q In other words, was what you were referring to as the Glass subset analysis, do you know if that was, in fact, a case control study?

A I don't know exactly how she structured that, but she did have controls, yes.

Q But you don't know if it was a case control study?

A The technical jargon is not my field.

Q Do you know if it was a cohort study?

A Well, no, I can't tell you that.

Q Where's that chart you had?

A What did we say? Right here, yeah.

Q So are you saying that Appendix 4 that the data it presents is inaccurate?

A We'd have to pull out each article and look at them one by one but I don't -- but I'm familiar with the Glass studies, and to me that doesn't support the fact that -- that benzene can cause, in low doses, myelodysplasia. And some of these other studies, I have looked at Rushton's, and I'm not certain that some of these articles are statistically significant in terms of their standard mortality rates. I'd have to look at each article individually to see.

Q You haven't done so, right?

A No, I haven't done so. I have looked at many of these articles. I have had probably most of them in my files, but I wasn't impressed that they showed anything. Bloemen, for example, I know that there's no statistical significance below 40 part per million years. I have that article here.

Q What does Dr. Gardner say?

A He claims that they have a high SMR, but he's not talking about statistical significance.

Q What does a high SMR mean?

A Standard mortality rate. So, in other words, if what's in the general population is 1 and your SMR is 2, you've got twice as much risk.

Q So you think he's reporting something wrong?

A Well, let's look at the paper and see. Let's see what number Bloemen is. That's a good one to analyze here. Let's see what number that is. That's No. 28. Okay. Well, let's see. It says in the title SMRs for Acute Nonlymphocytic Leukemia. Chronic Lymphocytic Leukemia NonHodgkin's Lymphoma were 1.1. So, in other words, for ANLL that would be 1.1, and that doesn't sound like it's got any statistical significance. What does he say it was?

MR. LUBEL: Objection. Nonresponsive.

Q (By Mr. Lubel) If you look at the chart --

A Yes.

Q -- he reports that between 28.3 and 79.1 ppm years the SMR for ANLL is 1.47.

A Well, as the way I'm reading this, he says SMRs for Acute Nonlymphocytic Leukemia, ANLL, and he's putting them in sequence, the first one is 1.1. That's the SMR for acute nonlymphocytic leukemia or AML.

Q It's not listing an exposure level, though. Possibly the article deeper inside of it provides a table or chart that gives exposure levels?

A Results for ANLL are particularly difficult to interpret due to the small numbers of deaths. He simply says, our risk estimates for ANLL are considerably lower

than those seen in the study on Chinese workers, and a more recent meta-analysis showed an SMR of 0.96 for AML. Average benzene exposures estimated to be below 1 part per million for refinery workers. Now he's talking about other people's studies in here.

But I don't see that he shows any table in here to suggest that what Dr. Gardner says is so, and I don't see that he has anything that would look statistically significant for a risk of ANLL.

MR. LUBEL: Objection. Nonresponsive.

A And he tells us that it's too small for analysis.

MR. LUBEL: Objection. Nonresponsive.

Q (By Mr. Lubel) You have not seen, at least in the Bloemen article which we specifically looked at, the SMRs that Dr. Gardner refers to in Appendix 4 of his report, correct?

A That's correct.

MR. LUBEL: I tell you what, let's take a short break. I'm going to go through this file real quick.

(Recess from 4:33 p.m. to 4:40 p.m.)

(Exhibit 7 marked)

Q (By Mr. Lubel) Exhibit 7 is your correspondence with the defense lawyers in the case,

correct?

A Yes.

Q Have we covered your opinions between what you've said today and your report?

A Yes, sir.

Q Is there any testimony that you've given today that needs to be changed or modified in order to make your testimony accurate --

A No.

Q -- that you can think of?

A That I can think of.

Q You know you're going to have an opportunity to review it?

A Yes.

Q Have I been polite to you?

A Absolutely.

Q Can you think of a time where I have been anything other than just a gentleman with you?

A Never other than a gentleman.

Q Okay. Thank you for your time.

MR. HUMPHREY: I have two quick follow-ups.

MR. LUBEL: Okay.

*

*

EXAMINATION

BY MR. HUMPHREY:

Q Dr. Natelson, Mr. Lubel asked you some questions about a meeting that you attended where Dr. Irons made a presentation. Do you recall those questions?

A Yes.

Q Approximately when did that meeting take place?

A I would say about a week ago.

Q Was that after you had written your report that summarizes your opinions in this case?

A Yes.

Q Was there anything in your November 22nd report, any of those opinions based in any way what you learned solely at that meeting where Dr. Irons gave this presentation?

A No.

Q Is there anything that you learned in Dr. Irons' presentation that in any way conflicts with your opinions as presented in your Noverber 22nd report?

A No.

MR. HUMPHREY: That's all I have. We'll reserve our questions for trial.

*

*

FURTHER EXAMINATION

BY MR. LUBEL:

Q In fact, if I understood you correctly, there's nothing in particular you are relying upon from that presentation by Dr. Irons with all those defense lawyers for this particular case?

A That's correct.

Q Correct?

A Yes.

Q You didn't go there for this case?

A No, I didn't go there for this case.

Q You went there for another case, and it just so happens that you heard things that involved information that's not in the articles that he has published?

A That is correct.

Q But you're not specifically relying upon that document -- pardon me -- that information for your opinions in this case?

A Correct.

MR. LUBEL: Okay.

(Proceedings concluded at 4:43 p.m.)

CHANGES AND SIGNATURE

WITNESS: ETHAN A. NATELSON, M.D. DATE: JANUARY 18, 2010

PAGE	LINE	CHANGE	REASON

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

_____
ETHAN A. NATELSON, M.D.

THE STATE OF _____)

COUNTY OF _____)

Before me, _____, on this day personally appeared ETHAN A. NATELSON, M.D., 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 _____

CAUSE NO. 08-CV-0161

MARY ELLEN HALL, INDIVIDUALLY AND AS REPRESENTATIVE OF THE ESTATE OF JOHN NEAL HALL, DECEASED, JOHN EMERY HALL AND LISA ANN EASLEY, Plaintiff(s),	)	IN THE DISTRICT COURT OF
V.	)	GALVESTON COUNTY, TEXAS
RADIATOR SPECIALTY COMPANY, ET AL, Defendant(s).	)	56TH JUDICIAL DISTRICT

REPORTER'S CERTIFICATION
ORAL DEPOSITION OF ETHAN A. NATELSON, M.D.
JANUARY 18, 2010

I, Wendi Broberg, Certified Shorthand Reporter in and for the State of Texas, hereby certify to the following:

That the witness, ETHAN A. NATELSON, M.D., 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 H. LUBEL 02:45
MR. SCOTT R. HUMPHREY 00:01

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:

Mr. Lance H. Lubel, Attorney for Plaintiffs

Mr. Scott R. Humphrey, Attorney for Defendant Radiator Specialty Company

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 22nd day of January, 2010.

WENDI BROBERG, Texas CSR 7091
Expiration Date: 12/31/11
Stratos Legal Services, L.P.
1001 West Loop South
Suite 809
Houston, Texas 77027
Ph. (713) 481-2180

FURTHER CERTIFICATION UNDER RULE 203 TRCP

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

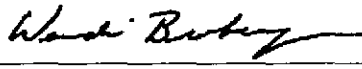
If returned, the attached Changes and Signature page contains any changes and the reasons therefor;

If returned, the original deposition was delivered to Mr. Lance H. Lubel, Custodial Attorney;

That $_____ is the deposition officer's charges to Mr. Lance H. Lubel, Counsel for Plaintiffs, 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 _____, _____.



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