IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF TEXAS
MARSHALL DIVISION

CHARLES WILSON and LAURA WILSON,	)	
Plaintiffs	)	
vs.	)	CIVIL ACTION NO.2:06-CV-286
RYCOLINE PRODUCTS, INC.; et al.,	)	
Defendants	)	

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ORAL DEPOSITION OF
DR. WAYNE SNODGRASS
September 14, 2007

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ORAL DEPOSITION of DR. WAYNE SNODGRASS, produced as a witness at the instance of the Plaintiff, and duly sworn, was taken in the above-styled and numbered cause on September 14, 2007, from 9:47 a.m. to 1:07 p.m., before Brenda L. Kaplan, CSR in and for the State of Texas, reported by Stenographic method, at the Tremont House Hotel, 2300 Mechanic Street, Galveston, Texas 77550, pursuant to the Texas Rules of Civil Procedure and the provisions stated on the record or attached hereto. That the deposition shall be read and signed by any notary public.

A P P E A R A N C E S

FOR THE PLAINTIFF:

Mr. Keith Patton
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EXHIBITS

WAYNE R. SNODGRASS,

having been first duly sworn, testified as follows:

E X A M I N A T I O N

BY MR. PATTON:

Q. Please introduce yourself for the record.

A. All right. My name is Wayne Snodgrass.

Q. And, Dr. Snodgrass, what kind of doctor are you?

A. I'm a physician and a toxicologist.

Q. What kind of physician are you?

A. I'm a -- I have a sub-specialty practice in medical toxicology and clinical pharmacology.

Q. You were hired as an expert in this case by Ashland, correct?

A. Yes.

Q. What was your objective, or what was your job or purpose in serving as an expert in this case, as you understand it?

A. What I agreed to is just telling what I thought, just what my opinion was about the information.

Q. Opinions on what information?

A. Mr. Wilson's disease and his exposure to chemicals.

Q. Is it your opinion in this case that Mr. Wilson's disease -- well, first of all, do you

dispute the diagnosis or do you dispute that Mr. Wilson has myelodysplastic syndrome?

A. Yes. That is not an accurate diagnosis.

Q. What do you believe his diagnosis to be?

A. His diagnosis is refractory anemia with ringed sideroblasts, which is a distinct diagnostic entity. Myelodysplastic syndrome is just simply a collection of different diseases.

Q. So, his disease is RARS?

A. Yes.

Q. And do you consider that to be a type of myelodysplastic syndrome?

A. Not particularly, no. It's a -- it's a distinct disorder. It's classified with that many classifications. But that's all that MDS is; it's a listing of different types of diseases.

Q. Is it your opinion in this case and would you testify at trial in this case that benzene exposure does not cause RARS?

A. That's correct.

Q. Would you testify at trial in this case that Mr. Wilson's exposure to benzene, whatever that exposure might be, was in no way any type of cause of his disease?

A. That's also correct.

Q. Okay. So, just to ask it sort of one more time with a couple of different words: Is it your opinion that benzene played absolutely zero role as a causative factor of Mr. Wilson's disease?

A. Yes.

Q. Okay. What did cause Mr. Wilson's RARS?

A. Medicine, in 2007 does not know the exact molecular origin of many diseases, including RARS. It's a spontaneous event in the genetic control of his cell production.

Q. I want to make a list of all of the things that cause RARS.

A. Okay.

Q. What would we put on that list?

A. The list in the future, when we get such information, perhaps might include some of the following, but to my knowledge, there's no publication or data on these factors I'm about to list. They might include any disorganization or -- or disregulation of the precursor cells for the erthrocyte; they might include disregulation genetically of mitochondrial function that results in iron accumulation in mitochondria that then localize around the nucleus.

And it may include relationships to the genes that control aging and how aging occurs in the

relationships, in other words, what we call "linked loci," on that. And those are yet all to be determined as spontaneous causes.

Q. So, the real cause of RARS, which you believe will be figured out in the near future -- or some time in the future, is going to be some type of disregulation of the cells or some type of other genetic event that happens, correct?

A. Yes.

Q. Is it your belief, is it your opinion, that environmental factors play zero role in disturbing or disregulating cells or genes?

A. With regard to RAS, specifically?

Q. No, with regard to anything.

A. No.

Q. Okay. Where are we missing each other?

A. That's a very broad question you asked. So, does the environment have any influence on genes? Yes.

Q. Okay. Does the environment have any influence on cells?

A. Yes.

Q. Do chemicals have any influence on genes?

A. Just as a broad question, yes.

Q. Do chemicals, as a broad question, have any influence on cells?

A. Yes.
Q. Does benzene have any influence on genes?
A. At some sufficient dose, as a broad question, yes.
Q. Okay. So, benzene can affect the genes?
A. At some sufficient dose, yes.
Q. And there's a debate about what that dose might be?
A. It's above the circulating background that we all have currently in our body of benzene, which is 200 nanograms per ml of benzene in our serum.
Q. Can you repeat at that one more time, the measurement, again? 200 --
A. Nanograms per ml of benzene in our serum. We all have that as a background level.
Q. So, we're all walking around with a little bit of benzene in our body?
A. Yes.
Q. And you know this as a medical toxicologist and clinical pharmacologist?
A. Yes, sir.
Q. Can that little bit of benzene that floats around in all of our bodies, 200 nanograms or so, can that have an adverse effect on cells or genes?
A. Not that I'm aware of anybody's demonstrated in

in experimental data.

Q. How do we get that benzene in our body? That background 200 nanogram level, how do we get it inside of us?

A. There is benzene in the air from man-made sources. There's benzene in egg yolk. There's a variety of sources.

Q. What are some of the other sources? Egg yolk, man-made sources?

A. Cigarette smoke, even if it's passive.

Q. Second-hand cigarette smoke?

A. Second-hand smoke would be included.

Q. So, if I was sitting in a room where a lot of people were smoking, I would get additional -- I would inhale benzene into my body from that second-hand smoke, correct?

A. That's detectible, yes.

Q. Okay. Detectible, such that it would contribute, or maybe even raise that 200 nanogram level?

A. It -- in an individual, depending on where they start and depending on how much is in the room, perhaps it might raise in the body. It's, generally also reported as just part of your background exposure.

Q. Okay. What are the other sources of the

200 nanograms or so of benzene in a person's body?

A. It's a small amount -- it's not quantitated as to the contribution to the 200 number, but there's a small amount that we produce from other normal body metabolism.

Q. Are some people walking around with more than 200 nanograms of benzene in their body?

A. Yes.

Q. And what would cause a person to have more than that baseline -- can I call it a baseline level?

A. Yes.

Q. What would cause a person to have more than that 200 baseline level?

A. The most characterized that I'm aware of is cigarette smoking.

Q. Because if someone smokes cigarettes, they're essentially inhaling a little bit of benzene into them every day?

A. Yes.

Q. What if someone were, like, a bartender and they worked in a bar where somebody -- where people smoked all day and all evening, would that bartender then get an increased level by inhaling benzene daily in their work?

A. Compared to the 200 number?

Q. Yes.
A. I'm not aware of data that's looked at that question. If the dose were sufficiently high, perhaps it might be above 200.
Q. So, is it your testimony that someone being exposed to second-hand smoke doesn't necessarily have a higher level above the 200 nanogram baseline?
A. It would depend on the dose in the air.
Q. What if someone worked in a refinery where benzene was present, would they have a higher level than the 200 ppm -- or 200 nanograms?
A. Currently, most refineries, one would predict they would not, because refinery workers are less exposed than, let's say, distributors, for example.
Q. What do you mean by "distributors"?
A. Well, for example, the truck drivers that deliver gasoline or things like that.
Q. People who deliver gasoline for a living, they're going to be exposed to a little bit of benzene on a daily basis by working with or around that gasoline, correct?
A. Yes.
Q. So, they might have higher than a 200-baseline dose?
A. They might. I've never seen data.

Q. Okay. Well, whether or not you've seen data, is it your opinion -- would you expect someone working with gasoline to have higher than the 200-nanogram dose?

A. It would depend on the dose exposure, which depends in part on their handling characteristics.

Q. Stuff like if it's windy outside or how far their nose is from the source of the liquid?

A. And the actual benzene content of the gasoline, itself, yes.

Q. So, benzene content, proximity, ventilation, things of that nature, elements like that would go into the evaluation to determine whether or not that person might have a level of benzene above 200 nanograms. Fair statement?

A. Yes.

Q. What if somebody worked with a benzene-containing solvent on a daily basis at arm's length, indoors, in a room with less than ideal ventilation, would you expect them to have a level of benzene in their body above the 200 nanograms?

MR. SHOEBOTHAM: Objection, form.

A. And what is the concentration of benzene in the solvent?

Q. (BY MR. PATTON) Well, what is the

concentration of benzene in gasoline?

A. Nowadays, it's about half a percent or less.

Q. So, it's about 500 ppm or less?

MR. SHOEBOTHAM: Objection, form.

A. I'd have to go through the numbers to come up with it. However, you may be right; I haven't sat down to convert it.

Q. (BY MR. PATTON) I think it's .1 percent equals 1,000 ppm. That's my understanding.

A. Okay.

Q. Do you believe that to be correct?

A. Yes.

Q. Okay. So, if someone were working with a product that had half of 1 percent, that would be .5 or --

A. It would be approximately that, yes.

Q. Well, that would actually be 5000 ppm?

A. (No response.)

Q. Let's just get this straight real quick. .1 equals 1000 ppm, true? And then 1 percent would equal 10,000 ppm. So, .5 percent would equal 5000 ppm. I think that's correct.

A. I'll assume it is.

Q. All right.

A. I haven't done the calculation, but I assume it

is.

Q. So, if we presume that gasoline has 5000 ppm benzene in it, your testimony, as you just stated, is that a gasoline worker could, under certain conditions, as working as a gasoline-distribution worker, experience daily exposures to benzene via that liquid such that it would increase the amount of baseline benzene in his body above 200 nanograms?

MR. SHOEBOTHAM: Objection, form.

Q. (BY MR. PATTON) Is that a fair statement?

A. Not actually, because the only dose response data I can relate to this is that years ago when there was much more benzene in gasoline and individuals used to pump gas, it wasn't self-service stations -- so, 5 percent benzene or more even, '60s, '70s, even earlier, that the urinary metabolite of that -- one of the metabolites of benzene could be increased only when they were very closely working with that, spilling it, and in the area of a clearly high level of exposure.

So, that was considerably more than .5 in order to -- .5 percent in order to get a change in your urine metabolites. So, that's the only data I know, relevant to that assessment.

Q. So, your testimony is that besides that little piece of data that you're aware of, there's no evidence

to indicate that people working with benzene daily would have an increase in their 200-nanogram baseline rate?

A. To my knowledge -- there may be data, but I'm not aware of it. I'm not aware of data that has specifically been gathered to answer that question and published.

Q. How many studies in the literature are you aware of that examine benzene as a causative factor of RARS?

A. The Strom study is the only one I'm aware of.

Q. Strom 2005?

A. Yes.

Q. Okay. What about studies that deal with benzene as a causative factor of refractory anemia with excess blasts, or RAEB?

A. To my knowledge, the Strom study is the only one that addresses that.

Q. The Strom study looks at MDS sub-types, correct?

A. It looks at -- one way of describing it is as you did, is the -- MDS, again, is a list of different disorders. They've looked at the various types that are often listed in that description.

Q. Is it your professional opinion in this case, that the whole body of literature which discusses

benzene and MDS is totally irrelevant when examining and exploring benzene as a cause of RARS, the disease that my client has?

MR. SHOEBOTHAM: Objection, form.

A. It depends what you mean by "totally irrelevant." But in terms of specifying a particular disease, I -- the -- just the terminology "MDS" does not do that.

Q. (BY MR. PATTON) Do you believe it is a fair and prudent method to examine the body of literature, which discusses benzene and bone marrow disease, any bone marrow disease, when examining causation in the instance of Charles Wilson?

MR. SHOEBOTHAM: Objection, form.

A. If I understand your question correctly, "Is it useful" --

Q. (BY MR. PATTON) That would be a good question. Is it useful in some way, to examine -- let me start over.

When examining causation of Charles Wilson's illness and whether or not that can be connected to benzene exposure, is it useful to look at the world's literature which discusses benzene and bone marrow diseases generally?

MR. SHOEBOTHAM: Objection, form.

A. It may be useful to be able to -- to look at that and see what does or what does not appear to relate to Mr. Wilson.

Q. (BY MR. PATTON) Is it useful to look at the studies that deal with benzene and MDS -- outside of the Strom study, is it useful to look at those studies when examining benzene as a possible causative factor of Charles Wilson's disease?

MR. SHOEBOTHAM: Objection, form.

A. Unfortunately, it's not.

Q. (BY MR. PATTON) So, then, the only study that matters is Strom 2005?

A. Short answer is "yes."

Q. What's the long answer?

A. The long answer is that the other studies don't separate out the distinct disease entities.

Q. So, they don't matter?

MR. SHOEBOTHAM: Objection, form.

A. You can't -- you can't make any conclusions from them.

Q. (BY MR. PATTON) Are there -- if we agree -- well, first of all, do you agree with me that RARS is a sub-type or variation of MDS?

A. No. I would say it's a distinct, different disease.

Q. Okay.
A. It may be listed under -- MDS is simply a collection of listing of different disorders.
Q. What is AML?
A. AML is acute myelogenous leukemia.
Q. Is that one distinct disorder?
A. As we learn more about it, it may become sub-divided. Currently, most people consider it one disorder.
Q. But it has sub-types, doesn't it?
A. Yes.
Q. Like M6, M4?
A. There are sub-types beginning to be defined, yes.
Q. Let's talk about ML a little bit. When were AML sub-types first defined?
A. You know, I'm not a hematologist or oncologist, so I'm going to have to defer to Dr. Natelson or someone. I don't remember the time line.
Q. How many different sub-types of AML are there, approximately?
A. I don't know at the moment.
Q. Okay. Is there a range that you could estimate?
A. It would be better to ask someone like an

oncologist, hematologist.

Q. Have you ever testified as an expert in a case involving AML?

A. Yes.

Q. And benzene?

A. Yes.

Q. An AML/benzene case, you've been an expert in that type of case, right?

A. I think so.

Q. Did you look at the sub-type of AML when examining the evidence on causation in that case?

A. There was one case. The one I'm recalling, I think, was the -- it was a different sub-type than would be compatible with benzene, and I -- promyelocytic leukemia, as I recall.

Q. That's a sub-type of AML?

A. It used to be considered that. It's beginning to be classified more as its own distinct entity.

Q. Well, let's talk about MDS again, then. Let's say someone has RARS, as Charles Wilson. Okay? Mr. Wilson does not have any cytogenetic or chromosomal abnormalities, correct?

A. That's correct.

Q. Okay. So, he has RARS with no abnormalities, true?

A. True. He has a loss of a Y chromosome, but that's not relatable.

Q. Okay. Do some RARS patients have chromosomal abnormalities?

A. I don't know for certain. I think a possible -- a very few may.

Q. Okay. Well, let's say someone has RARS with chromosomal abnormalities, like a Inversion 16 or a deletion of the 5 or 7 chromosome. Okay? Let's just take that for example.

Would the Strom study be instructive on each of those two different types of RARs?

MR. SHOEBOTHAM: Objection, form.

A. No.

Q. (BY MR. PATTON) So, the Strom study wouldn't be useful for an RARS with chromosomal abnormalities?

A. It would be.

Q. It would be?

A. For that question, the answer is "yes."

Q. Okay. And would the Strom study be useful for examining a person alleging benzene exposure as a cause of RARS with no chromosomal abnormalities?

A. Yes.

Q. So, the Strom study is useful for both, with or without chromosomal abnormalities?

A. At this stage and this time in the history of science simply because it doesn't make that distinction. It includes both.

Q. Okay. Well, before 2005, the relevant literature on benzene in MDS, it didn't make the distinctions of sub-types such as RARS or RAEB, did it?

A. Typically, no.

Q. All right. So, at that stage, it was all relevant, wasn't it?

MR. SHOEBOTHAM: Objection, form.

A. No.

Q. (BY MR. PATTON) So, in 2004 -- let's say you were asked to look at this case, this Wilson case, in 2004 and there's no Strom study, would your opinion be, "Well, there's just no relevant literature to support benzene as a cause of RARS, because RARS has never been examined in the literature in relation to benzene exposure"? Would that be your testimony?

MR. SHOEBOTHAM: Objection, form.

A. No.

Q. (BY MR. PATTON) Well, your testimony today is that the Strom study is the only relevant study because it deals with RARS specifically, which you call a "distinct disease."

A. Yes.

Q. So, where -- what am I missing you?
A. You're missing the disconnect that occurs between amounts of benzene exposure in the general population and the demographics of MDS, if you want to, you know, call MDS as a -- just put all of those categories together, what is the demographics of MDS, and how does that fit with benzene, and there's a major disconnect in the general demographics.
Q. Okay.
A. There are isolated cases, yes, but in the general demographics, it's not connected, because MDS has characteristics such as age of onset and benzene characteristics such as what is the population exposure that could reasonably lead to anything like MDS or leukemia, and those frequencies are markedly different.
Q. Does benzene cause MDS?
MR. SHOEBOTHAM: Objection, form.
A. If you put all categories together, based on some older literature at some sufficiently significantly high dose, then, yes, in the general rubric of how you use the word "MDS."
Q. (BY MR. PATTON) We happen to have "Harrison's Principles of Internal Medicine" on the table with us here today, and there's a hematology textbook that Dr. Haas talked about, "Wintrobe's," I believe?

A. Yes.
Q. Wintrobe's hematology text talks about benzene is a causative factor of MDS. Do you disagree with Wintrobe?
A. Yes.
Q. So, Wintrobe's wrong?
A. Well, if you went to that author today and showed him this same information --
Q. Showed him Strom 2005?
A. Sure. Would he rewrite that chapter, sentence? Greater than a 51 percent chance he would, because it's new, better information.
Q. Are you aware of anyone who has written to Wintrobe and said, "Hey, here's the Strom 2005 study. Now this chapter in your 9th or 10th edition of your hematology/oncology text is wrong, you need to rewrite it"?
Has anybody brought that to his attention that you know of?
A. Not specifically that, no.
Q. Have you brought that to his attention?
A. No.
Q. Will you?
A. No. There's so many things to change in each edition, it would take a full-time job to do that.

Q. So, even though you admit that benzene can cause a disregulation of the cells and you admit that benzene can cause a disregulation of genes, you do not believe that benzene can cause RARS, true?

A. Well, let's extend that logic. If benzene at some sufficiently high dose caused some disregulation of some sort of gene function, why aren't all cancers due to benzene? Why should it just pick AML? Should be -- every cancer should be due to benzene, right?

Q. Do you think all cancers are related to benzene?

A. No.

Q. Do you believe Charles Wilson was exposed to benzene?

A. Yes.

Q. And what were the sources of his exposure to benzene?

A. Well, one that he's contending here has to do with the --

Q. I'm not asking you what he's contending. I'm asking you what you believe Charles Wilson's sources of exposure to benzene are, based on your review of the box of materials that you reviewed for this case?

A. Various solvents were used in the printing area.

Q. So, do you believe that some of those solvents contained benzene?

A. At some level, at some detectible level.

Q. Okay. And you believe that that benzene was a source of Charles Wilson's exposure, correct?

A. He had some exposure.

Q. Okay.

A. Probably.

Q. Okay. So, you agree with me that Charles Wilson had exposure to benzene via use of printing solvents?

A. It's -- even small amounts of benzene, perhaps, were still in those solvents that would be measurable at some analytical level, yes.

Q. Okay. All right. So, when we're making a list of Charles Wilson's exposure to benzene, he had exposure to benzene in printing solvents. What else was a source of exposure to benzene for Charles Wilson?

A. I don't recall information about other individuals smoking or in the break room. And I don't recall the -- to my recollection, he did not have a history, himself, of cigarette smoking; so, I don't recall that as a possible exposure source.

There may be in -- even today, in some plastics or some materials in construction and other

sources, electrical wiring and things, there may be tiny, tiny amounts of benzene even in the other aromatic solvents that off-gas in these kinds of construction or building materials that we'd all be exposed to.

Some benzene in foods, as I mentioned before.

Q. Like egg yolk?

A. Yes.

Q. How much benzene was in the Ashland printing solvents that Charles Wilson used?

MR. SHOEBOTHAM: Objection, form.

A. My estimate would be somewhere -- if you looked at Mr. Whitlock's information, somewhere in the approximately 2 and-a-half to 5 ppm is a possibility.

(Snodgrass Exhibit No. 1 marked.)

Q. (BY MR. PATTON) I'm marking as Exhibit 1, your report. Can you turn to Page 5, please -- rather, Page 3. Can you read that last sentence of the first paragraph?

A. "Unpublished information regarding Ashland Blend 3078 D and Blend 7650, indicate considerably lower benzene content compared to the assumptions of Dr. Nicas."

Q. Okay. Who gave you that information -- well, first of all, what unpublished information are you

referencing?

A. I'm referencing information that I requested from Morgan Gaskin.

Q. Okay.

A. And she checked with someone and called me back and gave me some information.

Q. What information did she give you -- Morgan Gaskin is a lawyer for Ashland, right?

A. Yes. Yes.

Q. What information did she give you?

A. In that it was in the -- somewhere in the approximate 5 or less part-per-million range.

Q. Well, hold on. What unpublished information did Morgan Gaskin identify?

A. I assumed she talked to somebody at Ashland.

Q. Did she identify for you the unpublished information?

A. No.

Q. When you say "considerably lower benzene content," what do you mean by that? Why did you choose those words?

A. If you go back to Dr. Nicas's report, he has -- it's actually confusing -- at least, it was to me. He talks about unit weight percent, and then he talks about weight per weight and equates them. And so, I -- in

going back over that report, my estimate is that he's talking about .00175 percent, which is 1,750 -- I'm sorry -- .175 percent, which is 1,750 ppm.

Q. Are you a chemist?

A. Not now.

Q. How much benzene did Morgan Gaskin tell you the unpublished information indicated was in the Ashland solvent blends?

A. Somewhere in the range of less than 5 ppm.

Q. Okay. Do you believe that's a sound scientific method, for you to put that sentence in your report based on what Ashland's lawyer tells you?

A. It doesn't reflect Hill criteria, but it does have relevance to the case.

Q. So, Ashland's lawyer telling you that there's 5 ppm or less benzene in these solvents, that has relevance to the case; the body of MDS literature, pre-2005, outside of Strom, doesn't have relevance to the case. Is that your testimony?

MR. SHOEBOTHAM: Objection, form.

A. The body of the literature prior to 2005 may have some relevance, but it depends on how you want to twist the data and interpret it.

Q. (BY MR. PATTON) You only like to use high-quality data when you examine the relevant benzene

literature; is that true?

A. That would be the preference.

Q. Okay. What does "high-quality data" mean?

A. It could be data that perhaps approximates Level 1-A or 1-D or 1-C or 1-B or 2-A or 2-B, 2-C or 3-A or --

Q. Slow down, Doctor, please.

A. All right.

Q. Let's start over. Okay?

A. It could be levels of --

Q. Hang on. I'm going to start -- ask a new question.

Okay. Page 6 of your report, do you see the last sentence before your Summary of Conclusions? You say, "There are no" -- can you read that for us?

A. "There are no published high-quality data that true MDS develops at lower cumulative dose exposures."

Q. Okay. I've written down on this piece of paper here -- and I'll mark this as Exhibit 2 to your deposition.

(Snodgrass Exhibit No. 2 marked.)

Q. (BY MR. PATTON) I've written on here: "High-quality data according to Dr. Snodgrass." And as it relates to your opinions on the data that my experts, or Plaintiffs' experts, rely on in their opinions, I'd

like you to make a list of what makes something high-quality data on Exhibit 2. And you can talk about it as you write.

A. Cochrane levels defined.

Q. Okay. What is a Cochrane level?

A. So, it is a -- they are defined as Levels 1, with Sub-Types A, B and C and D; and 2 A, B, C and D; 3 A, B, C and D; 4 A, B, C and D; and the various characteristics of scientists, physician/scientists, assessing published data as to its quality.

Q. Can you write that down?

A. Okay.

Q. "Cochrane levels defined." And you wrote "Characteristics of quality of data." Is that what you wrote?

A. Yes.

Q. Okay. Who is Cochrane?

A. Don't recall. It's a database nowadays. It may have been named after an individual. I don't recall who the individual was.

Q. Okay. What is a Cochrane level?

A. It's a way of categorizing the quality of information.

Q. Now, are there -- you said there are four different levels, one, two, three, and four?

A. Yes.
Q. And then, there's four divisions within each Cochrane level?
A. Yes.
Q. A, B, C and D?
A. Yes.
Q. So, it's essentially 16 different levels in evaluating the quality of data --
A. Yes.
Q. -- or the quality of a study?
A. Yes.
Q. Okay. So, that's to say that a scientist who would read up and learn about Cochrane levels would be able to take any study -- let's take the Strom study. Okay? He could evaluate the Strom study and give it a rating, so to speak, within the Cochrane levels, the 16 different Cochrane levels? Is that a fair statement?
A. Yes.
Q. Okay. So, high-quality data has a defined Cochrane level, whatever that level may be?
A. Yes.
Q. So, it's like a grade, 1 through 16?
A. Yes.
Q. How do we grade the Strom study -- well, before I ask that, is there a -- does each published study in

the scientific literature, whatever the subject may be, does it receive a grade, or a Cochrane level?

A. No.

Q. Why not?

A. It's not routine, working practice for the scientific community.

Q. So, evaluating and grading scientific literature under the Cochrane level guidelines is not common practice in the scientific community, correct?

A. It's common practice for those who deliberately set out to assess information. It's -- it's not as formally applied by reviewers and editors to put a number on it, but it's definitely the equivalent of it as applied by reviewers, who then tell an editor "yes" or "no" to accept an article for publication.

Q. The Strom study was published in The Leukemia Journal?

A. Yes.

Q. So, if I were to go to the editor of The Leukemia Journal, I would -- I could say, "Hey, Editor, you defined or you graded every study before you put it in your journal, and you gave each one a Cochrane level," and he'd say, "Yes, I did"?

A. No, he would not say that, because he didn't do it.

Q. Okay. Well, did somebody else do it and then tell the editor of The Journal of Leukemia?

A. No. No. The way the process works is that, for reviewing articles for peer-reviewed publications, the editor will receive the journal article being submitted by the authors and the editor will determine whatever he thinks or she thinks is -- who are expert in the area and send it to them.

Q. Peer reviewers?

A. It's called "peer review."

Q. Okay. So, the editor sends the paper to a reviewer?

A. Yes.

Q. And then what?

A. And then the peer reviewers either accept or reject or they accept with modifications and tell the editor that, and then the editor makes a decision.

Q. And then where does the -- where does the Cochrane level come into that?

A. In that process, not formally, no.

Q. Informally?

A. Informally, in the sense, perhaps -- they're not thinking of Cochrane, typically; but in the sense of they think they know what is quality information, and they just say, yes, it's good data or it's not good

data; or it's a good paper, or it's not; or they have other reasons for either accepting it or not accepting it.

Q. Have you ever been excluded from testifying in a civil lawsuit?

A. No.

Q. What is the Cochrane level grade of the Strom 2005 paper?

MR. SHOEBOTHAM: Objection, form.

A. I don't have all the 16 criteria right in front of me, so I don't have them memorized. This is not a prospective, randomized, controlled trial; so, therefore, it's not a 1-A, and probably not even a 1-C or D.

Q. (BY MR. PATTON) The ones that are the highest quality?

A. Yeah -- well, it can't be if it's not a -- it can't be a 1-A or perhaps a 1-B if it's not a prospective, randomized, controlled trial, which this is not, because it's an epidemiology study. By definition, epidemiology studies, even cohorts, are going to be a lower level.

Q. So, the Strom study is a low-quality study?

MR. SHOEBOTHAM: Objection, form.

A. Did I say that?

Q. (BY MR. PATTON) I'm asking you.
A. I'm asking you: Did I say that?
Q. No, you didn't.
My question is this: Is the Strom study a low-quality study?
A. No.
Q. It's a high-quality study?
A. Yes.
Q. Okay. What makes it a high-quality study?
A. Because they have appropriate controls and they have a specific -- if you want to call it "hypothesis," a specific goal in looking at the relationship of benzene to specific types of erythroid dysplasia.
Q. You said on Page 6 of your report: "There are no published high-quality data that true MDS develops at lower cumulative dose exposures."
Do you see that on Page 6 of your report?
A. Yes.
Q. Okay. Dr. Parent is one of Plaintiff's experts in this case, correct?
A. Yes.
Q. Dr. Infante is one of the plaintiff's experts in this case, correct?
A. Yes.
Q. You reviewed both of their reports, right?

A. Yes.
Q. You reviewed the citations of the authority that they used to support their opinions in this case, right?
A. Yes.
Q. You reviewed their depositions, right?
A. Yes.
Q. Is it your position that Plaintiff's experts rely upon no high-quality data in reaching their conclusion that Mr. Wilson's RARS form of MDS was caused by exposure to benzene?
A. They rely on data that in some cases is about as good a quality as it could be based on the way that study was designed and done, but it -- the interpretation and the ascribance of significant of that data is not appropriate.
Q. So, that makes it low-quality data?
A. No, it made -- the authors may have done as quality a job as they could do based on the characteristics of how they designed the study, but the interpretation and application to come and get a conclusion is not reasonably possible to make the conclusions that they did.
Q. I'm not talking about the conclusions. I'm talking about the data, itself, because you say: "There

are no published, high-quality data that true MDS develops" -- what is "true MDS"?

A. Well, it would be -- if you want to look at all the listings of -- within the categories of MDS that are listed here, I think there are five or six different sub-types.

Q. Is RARS a true MDS?

A. I think, no, in 2007 and onward, because it's going to become clear that this is a distinct disease.

Q. Your report is dated July 5th, 2007, right?

A. Yes.

Q. And you say: "There are no published, high-quality data that true MDS develops at lower cumulative dose exposure."

Why are you even talking about true MDS, when my client has RARS, which you don't believe to be MDS?

A. Simply to address the question.

Q. Is RARS true MDS?

A. No.

Q. So, your opinion has no relevance to this case, because you're talking about true MDS, not RARS, right?

MR. SHOEBOTHAM: Objection, form.

A. I would disagree.

Q. (BY MR. PATTON) Why would you disagree?

A. My opinion does have relevance. He has RARS, and that is not caused by benzene.

Q. Did you go through all or each of Dr. Parent's pieces of authority that he sites in support of his opinions, all 186 of them?

A. No.

Q. Did you evaluate -- did you evaluate any of them to give them a Cochrane level grade to -- so that you could determine whether or not they were high quality or low quality?

A. That's already been done in a different way, but no, I did not do it.

Q. Who did that in a different way?

A. The Shatner article.

Q. What does the Shatner article say?

A. Well, what the Shatner article does is to look at over 300-some published -- over 400 published articles and studies related to benzene, and AML particularly, and he is able to divide them out into studies that meet various quality criteria that I've listed in my report.

Q. At the -- and that's -- in Page -- on Page 5, on the bottom half of your report, right?

A. Yes, sir.

Q. Near the bottom of the second to last paragraph

on Page 5, starting "Highest quality," can you read that for us?

A. "Highest quality data show that cumulative benzene exposure 200 ppm-years is the threshold for increased risk of AML. There are no published high-quality data that true MDS that is caused by benzene exposure develops at lower cumulative benzene dose exposure."

Q. And that's the same sentence that you repeated in the next paragraph at the bottom, too, right?

A. Yes.

Q. So, because you give such credence to the Cochrane levels and to Shatner and to high-quality data, if there were an instance where someone has AML and they had 201 ppm-years of exposure, you would agree that the benzene caused that person's AML?

MR. SHOEBOTHAM: Objection, form.

Q. (BY MR. PATTON) True?

A. That's as a misconception of what the information means.

Q. The information would mean there's an increased risk, right?

A. Yes.

Q. Okay. So, if someone had 201 ppm-years exposure to benzene and they had AML, your opinion would

be, generally speaking, that that person had an increased risk of AML from exposure to the 201 ppm-years of benzene, true?

A. It might be a small risk, but it might -- but it would be increased compared to the general population, yes.

Q. Would you believe that the 201 years -- ppm-years of benzene exposure were a substantial causative factor in that person's AML?

MR. SHOEBOTHAM: Objection, form.

A. "Substantial causative factor at 201 ppm-years based on all the information available" -- from a lay perspective, no.

Q. (BY MR. PATTON) Are there any circumstances under which you could opine that a person's AML was caused by benzene exposure?

A. Yes.

Q. What are those circumstances?

A. It's a dose response phenomenon: As you increase the dose, the risk the likelihood that AML was caused by benzene becomes greater. So, certainly, at -- above 400 ppm-years, that risk begins to increase significantly.

Q. So, if I, as a plaintiff's lawyer, had a case in which I could show you evidence that someone was

exposed to 401 ppm-years of benzene exposure and they had AML, would you support my case that benzene exposure was a substantial causative factor of that person's AML?

MR. SHOEBOTHAM: Objection, form.

A. If the sub-typing of AML, in a sense, were the appropriate type, yes.

Q. (BY MR. PATTON) So, we've got to go back into sub-type again?

A. Likely, for AML, yes.

Q. Okay. And we would have to look, then, only to the studies of AML that examine AML and sub-type; is that your testimony?

A. That examine -- sub-types that examine cytogenetic abnormalities, the factors that are associated with benzene, and in the future that examine oxidoreductase quinone metabolism and allele's.

MR. PATTON: I'll object to the nonresponsive portion of your answer.

Q. (BY MR. PATTON) If I had a client that I could show you had 401 ppm-years of benzene exposure and they had AML, you would not be able to support causation in that case unless there were certain sub-types, true?

MR. SHOEBOTHAM: Objection, form.

A. Most likely, yes.

Q. (BY MR. PATTON) Okay. So, you would have to

take a look at that plaintiff's -- or that patient's sub-type and then do the analysis, and then looking -- you would only look to the relevant literature of sub-type, right?

MR. SHOEBOTHAM: Objection, form.

A. Let me ask you what -- to be more clear on that answer, if there were information about cytogenetics, that would be very helpful.

Q. (BY MR. PATTON) Let's say -- we'll call him "Plaintiff X." Okay?

A. Yes.

Q. Let's say Plaintiff X has AML-M4. Okay?

A. Yes.

Q. A non-smoker. 401 ppm-years of benzene exposure. Okay? No cytogenetic abnormalities. Could you support benzene as a cause of that disease?

MR. SHOEBOTHAM: Objection, form.

A. No cytogenetic abnormalities. So, what that does is that stratifies the risk differently.

Q. (BY MR. PATTON) What do you mean "stratifies"?

A. Okay. It makes --

Q. Because I don't think I've ever heard that word.

A. Okay. "Stratify" means you separate into groupings. So, if you showed me -- and let me make the

comparison here. I'm not even thinking about M1, M4, M6. I'd have to go back to the literature, and a hematologist can you tell you better than I on that.

But if you're thinking about what is known about benzene and its effects on the body or on cells, and if you have -- and it is well-understood and accepted that benzene tends to cause chromosome aberrations. Chromosome aberrations can be a gain of chromosomes, it can be a loss of chromosomes, it can be something we call polypolidy -- p-o-l-y-p-l-o-i-d-y -- and other differences.

So, if you find -- and I may not have this memorized correctly -- but if you find an addition or a deletion, like a 5-7 or a 17-18 or 17-19, those -- and that that were the case of the gentleman you were describing with AML and he had those, then -- and you have a 400 and -- I think it becomes, perhaps, more likely than not with that degree of benzene exposure.

Q. Tell me again what "stratify" means?

A. Stratify means to divide out or split out into various groupings.

Q. So, when something like a hematology text indicates that benzene causes AML, for you to give an opinion on benzene causing AML you would need to further stratify AML; you would need to put it in separate

groups?

A. Yes.

Q. Okay. And so, then, once you put it into the separate groups -- I wish I knew offhand how many sub-types there were, because -- I know there's about five in MDS -- but given your position on MDS, I think it's a better context of AML.

Do you know approximately how many sub --

MR. SHOEBOTHAM: Objection, form.

MR. PATTON: Forgive the sidebar.

Q. (BY MR. PATTON) Do you know approximately how many sub-types of AML there are? I'm not going to hold you to this.

A. All right.

Q. Just so we can talk about it.

A. I can't remember, actually. I'm sorry.

Q. Okay. But you, as an expert in examining causation, you would want to stratify AML? You would want to look at sub-type and then you would want to look at chromosomal aberrations within each sub-type, correct?

A. Yes.

Q. And then you would want to look at the specific chromosomal aberrations within each sub-type. So, you would further stratify it as far as you possibly could

in examining causation. That's the way you would work, right?

A. Yes.

Q. That is your methodology, to stratify to the lowest -- or most specific possible manner, right?

A. To -- my methodology is to base my approach on the best available data, which now, compared to 30 years ago, we have much more information on what these disorders are like.

Q. Page 7 in your report. It has references.

What does the Bjork reference stand for, generally.

A. These are last names of the first author of papers.

Q. Sure.

A. And I didn't have time to finish it, putting all the specific information in; so, I don't recall the Bjork paper without looking at it.

Q. Were you under a time crunch when you were writing this report?

A. Yes.

Q. When were you first given the Wilson case to review and opine on?

A. Plenty of time to write a report. I was under time issues from my other workload.

MR. PATTON: Objection, nonresponsive.

Q. (BY MR. PATTON) When were you given the --

A. Oh. I don't remember. I can find a -- I apologize. I can find the -- a letter that was sent to me, perhaps.

Q. I think it's back in the box. And it's all right. We can continue.

MR. PATTON: Let's take a break. Off the record.

(Short break.)

Q. (BY MR. PATTON) Doctor, we took a short break; and I want to finish out talking about Exhibit 2. When want made a list of what is "high-quality data," according to you, you talked about your deference, or respect, should I say, for the Cochrane levels, correct?

A. Yes.

Q. And what is the next part?

A. That within those 16 various categories, which I don't have here and I don't have memorized -- if we had those, we could list those as the way one applies this to individual studies.

Q. Is there a level within that 16 Cochrane grades, so to speak, that you make it a cutoff, that something is above the line and, therefore, high quality or below the line and, therefore, low quality, or how

does that work?

A. It's a continuum.

Q. Some are higher than others?

A. Yes.

Q. Okay. And then, what's this part you wrote here that I can't read (indicating)?

A. "Characteristics of quality of data."

Q. Okay. That's something that --

A. It's defined in each of the -- in the actual formal listings --

Q. Okay.

A. -- if we had a copy of it.

Q. So, "high-quality data," that phrase, for you is all about the Cochrane levels, right, and those things set forth within the Cochrane levels?

A. High-quality data is sort of a qualitative descriptive term. The Cochrane is an attempt to try to semi-quantitatively apply some numerics to that.

Q. Okay. Do you consider each of the references on Page 7 of your report to be high-quality data?

A. They have -- and I've not done this, but they would have varying levels of -- if you tried to write them by the Cochrane approach -- varying levels of Cochrane category. Some are higher -- depends on what you mean by "high quality."

If you mean overall, then maybe they're high quality with regard to what they had to work with -- excuse me -- and maybe they simply are unable to be higher quality because of the nature of the design of the study or the limitations in the data that was accessible.

Q. But isn't it true that all scientific literature is going to have varying levels of quality? You know, for instance, well, this is a real positive for the study or -- and at the same time, another issue is a real negative for the study?

It's kind of like the weather. Yes, it's sunny out, which is nice; but it's humid, which is unfavorable. I mean, can't each study be looked at to have some positive attributes or strengths and some weaknesses?

A. Within a range, perhaps.

Q. And then do some studies just fall out of that range such that they have so many weaknesses that they just don't pass muster for any type of appreciable quality?

A. Sometimes I have seen that occur.

Q. Well, for instance, today could be characterized as something of a high-quality day, because it's sunny out, it's warm, it's not raining,

it's generally pleasant, it's not too windy. Whereas, if you had a day where there's, like, a tropical storm coming through, as in a few days ago, most people would agree that is a -- an unfavorable day or a bad day weather-wise?

Do you understand my analogy?

A. Yes.

Q. Okay. Are there some studies that when you take a look at them, you can say, you know what, that's just a bad study. It's like a day where there's a tropical storm coming through; it's just not good, it's not favorable, and it's just below muster, it's below the line of something that anyone would appreciate or like?

MR. SHOEBOTHAM: Objection, form.

A. Well, then, it would be a dis -- "favorable" may be favorable to the case, in a legal setting. Whether it's good or not or bad or not in a quality sense, may be a different distinction of terms or what were their methods, what were the data sets that they had to work with.

Q. (BY MR. PATTON) What are your criticisms of Dr. Parent and Dr. Nicas's -- I'm sorry -- Dr. Parent and Dr. Infante's opinions as it speaks to their reliance upon data of whatever quality that may be?

A. My critiques are very similar to what would be in the Institute of Medicine Report that looks at that information. And that is that -- and also similar to Shatner's with regard to a different end point. And that is that your -- there's issues of -- very specific kinds of issues a variety of more than I can recall, but issues of misclassification, of confounding of the design of the study and how strong a conclusion one can make from that type of design or study; and so, that, they take a variety of studies and don't really talk about the limitations or weaknesses of those studies.

Q. Well, you cite -- you cite 18 references on Page 7 of your report.

A. Yes.

Q. In your report, do you discuss the strengths and weaknesses of each --

A. No.

Q. -- study?

A. I do not.

Q. Did you review each of the 186 references by Dr. Parent and sort of check them off, so to speak, and say, you know what, that one has too many weaknesses or that other one has too many weaknesses or that other one is just bad science or -- did you go through and individually critique each of their -- each of

Plaintiff's expert's references?

A. No.

Q. Nor did you go through and grade them, so to speak, per the Cochrane levels, true?

A. Correct.

Q. You just didn't do that, right?

A. That's correct.

Q. Okay. So, how do you reach the conclusion, then, that Plaintiff's experts, Dr. Parent and Dr. Infante, their opinions are based on no published high-quality data?

A. Because if you'd look at the kind of studies they look at, and you want to answer the question, "yes" or "no," does benzene cause RARS, they do not address the issue, specifically; they do not show dose response data; they do not describe dose response data; they do not address that even as a factor.

Q. Does the Strom 2005 discuss dose response?

A. No.

Q. What's the strength of the data in the Strom study?

A. Its strength is that it looks at RARS specifically.

Q. Okay. What are the weaknesses of the Strom study?

A. There -- part of what any kind of case control study would have -- or retrospective case control study cannot be as -- the same strength of evidence as, say, a prospective cohort study would be.

Q. So, you think prospective cohort studies are the best studies under which to examine benzene as a causative factor in a given type of bone marrow disease, true?

A. Well, that's just general epidemiology information, yes.

Q. Prospective cohort studies are the best?

A. Yes.

Q. Which prospective cohort studies are cited in your report?

A. To my knowledge, I don't recall any of them being prospective cohort studies.

Q. So, you don't cite any prospective cohort studies for your conclusion that benzene doesn't cause MDS and benzene was in no way a causative factor of Charles Wilson's MDS?

A. I'm not aware of any published prospective cohort studies that address benzene and RARS or MDS.

Q. Okay. So, then what's the next best type of study out there, highest quality?

A. What's available by design would be a case

control-type study.

Q. Okay. Case control studies, those are reliable -- that's a reliable methodology? That's a relevant piece of scientific literature that's respected in the scientific community?

A. It's regarded as to what it is.

Q. It's going to have strengths and weaknesses?

A. Yes.

Q. Each case control study will have strengths and weaknesses, right?

A. Often, yes.

Q. You cite some case controlled studies?

A. Yes.

Q. Okay. They all have strengths and weaknesses, don't they?

A. Yes.

Q. Some of them look at dose response, some don't?

A. Yes.

Q. Okay. What's the problem with -- or what are your criticisms of Plaintiff's experts in terms of the types of studies they use? Do they point to any prospective cohort studies?

A. No.

Q. All right. Do Plaintiff's experts rely on any case control studies?

A. Yes.
Q. And your position is the case control studies they rely on do not support benzene as a cause of RARS, true?
A. Correct.
Q. Do you believe the case control studies that Plaintiff's experts rely on support benzene as a possible causative factor in MDS, generally?
A. When you use that terminology, then the answer to that would be there's older literature at sufficiently high dose of exposure, years ago for benzene, that some AML cases are preceded by what would be then, in those days, termed "MDS." And I think it would be general agreement that at sufficiently high dose, a long enough period of time, that benzene can lead to AML, could, in some of those cases, have produced what is -- was then, in those days, called "MDS."
Q. Okay. Do you believe Dr. Infante misinterprets some studies in relying upon them in support of his opinions?
A. Yes.
Q. Which studies does he misinterpret?
A. If you look at Page 10 of Dr. Infante's report, on the bottom half of that page he cites a 1970 paper by

Girard and Revol, and this is under the section that is entitled "Epidemiological Studies Subsequently Provided Evidence That Benzene Caused AML and MDS."

And in that study is -- he states near the bottom of that paragraph that the -- he's indicated that the frequency of benzene exposure was statistically significantly elevated for patients admitted with acute leukemia and aplasia.

Q. What's aplasia?

A. Aplasia is the opposite of MDS. So, it would be a matter of whether you want to include that as something related to MDS or not, as to how you interpret that study -- or to use that study to relate it to MDS.

Q. What other criticisms do you have of Dr. Infante's opinions in this case, either expressed in his report or through his deposition?

A. On Page 13 of his report in the first full paragraph, which starts: "For dose response analyses" -- he's talking about Hays, et. al, in 1997 -- he makes the statement that reflects that he is not dividing MDS into the actual different disease categories that it is; and he states: "Simply because MDS is a precursor to AML..."

Some distinct disorders may be a precursor to AML, but not necessarily and not necessarily

specifically that.

Q. The Hays study didn't stratify sufficiently for your taste, correct?

A. It just didn't divide into RARS or other distinct disorders, no, it did not.

Q. How far -- if we look at Page 7 of your report, in your references, how far stratified, so to speak, are each of these studies that you cite, the 18 studies, or authority that you cite?

A. None, to the extent that Strom is.

Q. It's unusual for a -- for a benzene-type study or study that discusses benzene to stratify as far as Strom, isn't it?

A. Well, I will correct my response. It's -- the Wong studies have done that.

Q. How far did they stratify?

A. And what they did was not related to MDS. It was related to leukemia, AML.

Q. Did Wong look at all leukemia or did Wong look at only AML?

A. That's the point. He looked at AML specifically.

Q. Did he look at AML by sub-type?

A. No.

Q. So, you rely on the Wong study because it talks

about AML and the cumulative dose necessary, according to Dr. Wong's study, to increase the risk of AML from exposure to benzene; but the Wong study doesn't stratify beyond AML, correct?

A. Correct.

Q. And so -- remember when I was talking about "Plaintiff X" earlier, my potential client who had 401 ppm-years exposure to benzene?

A. Yes.

Q. Would the Wong study at all be helpful to you in opining in that type of case?

A. Yes.

Q. Even though he doesn't stratify?

A. Yes.

Q. Can you explain that?

A. Because -- because the hypothetical individual you've mentioned is -- I think you mentioned 401 -- that is above 400, which is a higher risk. And 200 is a very conservative estimate; it's above 200. So, it says that he would definitely have some risk of benzene being a cause of his AML if we don't know the sub-types.

Q. So, he's above 400. So, all AMLs are caused by benzene exposure above 400?

MR. SHOEBOTHAM: Objection, form.

A. No. We don't know that. In fact, we know the

opposite. We know to some extent that some types would not be because of the type of cytogenetic findings you have.

Q. (BY MR. PATTON) So, when Dr. Wong says that excess of 200 ppm-years exposure to benzene causes an increased risk of AML, it's not really an accurate statement; you would need to look, then, further to the sub-types of AML before you could say that 200 -- excess of 200 ppm-years exposure would cause a given sub-type, correct?

MR. SHOEBOTHAM: Objection, form.

A. Let me be sure I understand your question. Wong's study looked at the data available, and it did not have sub-type information for him to be able to make any kind of classification like that. So, all he could say, it was AML; so, that's the extent of what can be concluded from the data he had available.

Q. (BY MR. PATTON) Don't you think you're trying to have it both ways in this case? You're trying to say that benzene doesn't cause RARS, and here's one study that's ultra-stratified and that's the end of the inquiry.

Meanwhile, when you talk about benzene generally is a cause of other bone marrow diseases, you're willing to opine that it takes significantly

higher exposures than one might opine Mr. Wilson had; and the basis for that is a really low, if at all, stratified study like Wong.

MR. SHOEBOTHAM: Objection, form.

Q. (BY MR. PATTON) How do you reconcile that as a scientist?

A. There's no need to reconcile anything. They're all very consistent with the data that's available, and when you have dose response information, that's very relevant to making an assessment about an individual person's likelihood or not likelihood to have a disorder related to benzene or not related to benzene exposure.

Q. But the dose response data from Wong says, "Excess of 200 ppm-years equals increased risk of AML," right?

A. Yes. It starts as a threshold at that point.

Q. Okay. So, then once you're above that threshold, you would still need to further stratify or sub-divide a given patient's AML before you could then say, "Okay. Wong is right, because this patient has this certain sub-type that's shown in other literature"?

A. Wong's 1995 data did not address sub-types.

Q. Did Crump '94 examine sub-types?

A. Of AML, no. Of leukemia, yes.

Q. What types of leukemia did Crump look at?

A. So, lymphocytic leukemias versus myelocytic leukemia.

Q. What about Ward '96, does that look at -- how far does that stratify?

A. I would have to look at the study. I don't recall it without looking at the data.

Q. Can we look it up? I don't think it's in that box, to be honest.

A. I don't think I have it here.

Q. Okay. We can move on.

You work at a poison control center, right?

A. Yes.

Q. You're the medical director at the Texas Poison Center in Santa Fe?

A. No, it's the Texas Poison Center that serves Houston and Galveston, and it's located here in Galveston.

Q. Do you give opinions in your clinical work as to a specific substance causing a specific disease?

A. In a general sense, that is a question that sometimes is asked, yes.

Q. And do you go to a textbook or some other type of medical or toxicological authority that talks about, okay, substance "X" causes illness "Y," or do you just

take that for what it is or do you then dive into the literature and start stratifying to give the opinion that, yes, "X" caused "Y," or, no, "X" did not cause "Y"? How far do you dig when you give a causation opinion in your clinical practice?

A. Well, the context and setting is very different than here in a legal setting. So, much of the time, if not a far majority of the time, it's something that's relatively well-known or accepted; so, cause is sort-of already understood. The question is in that individual patient -- in a general sense, cause is understood. The question is in that individual patient is that what's doing it to this patient or are there other factors involved or, if it is causing it, how do we manage this and how do we treat it. So, the setting is actually rather different.

Q. I know I'm bouncing around a little this morning, Doctor; and I think I'll wrap it up at some point, but I want to next talk about the list of cases that you've testified in. Is that okay?

A. Yes.

(Snodgrass Exhibit No. 3 marked.)

Q. (BY MR. PATTON) I've marked as Exhibit 3, a list of testimony in the last four years. And this is a list that you've faxed to the lawyers at Thompson

Knight, correct?

A. Yes.

Q. Okay. How many of these cases did you testify on behalf of a plaintiff?

A. Approximately, I'd say, typically, around 40 percent.

Q. 40 percent of your work is with plaintiffs?

A. Yes, sir.

Q. How many of these cases involve benzene? And can you tell me which one, so I can write "benzene" next to it on my list?

A. Well, this is from memory. I know I've testified or at least had a deposition in a benzene case or -- one or more. I see a xylene case listed. The fourth case is Martinez. That was not benzene. I'm trying to recall, see if I can recall a benzene case.

I apologize. I know I have in the past; I just don't remember them.

Q. Can you estimate approximately how many benzene cases you've testified in?

A. I'd say at least two and maybe three or four.

Q. Two to four benzene cases?

A. I would estimate that number at this point.

Q. How many cases have you testified in on behalf of Ashland?

A. To my recollection, none.
Q. How many cases have you testified in involving Mr. Shoebotham or his firm?
A. Two, or perhaps three or four.
Q. So, each of your benzene cases were with his law firm, Thompson & Knight?
A. I'm not certain. I -- there may have been one or two. Perhaps, it was with different attorneys.
Q. Which cases on here involved Mr. Shoebotham and his law firm?
A. None of them.
MR. PATTON: Mr. Shoebotham, just for the --
MR. SHOEBOTHAM: I --
MR. PATTON: -- ease of clarification --
MR. SHOEBOTHAM: I can --
MR. PATTON: -- do you have any input on this?
MR. SHOEBOTHAM: I can tell you one from memory that I think may have just been omitted from the list. It was a case called Scott versus Pharmacia in which Mr. Snodgrass testified, and I was one of the lawyers that retained him.
And I don't see that on the listing. So, I would ask Dr. Snodgrass to supplement his list orally

with that deposition. That deposition took place in either late 2005 or early 2006.

MR. PATTON: Who was the plaintiff's lawyer?

MR. SHOEBOTHAM: Mike Kesky.

MR. PATTON: That was that Scott case?

MR. SHOEBOTHAM: That's the only one that springs to mind, just looking at the list.

Q. (BY MR. PATTON) You've never testified on behalf of a plaintiff in a benzene case, correct?

A. I don't believe I have been asked -- or seen data that was sufficient for causation. I -- correct, I've not.

Q. Have you ever reviewed cases involving benzene exposure for a plaintiff's attorney?

A. I think I may have been asked, yes, but I am trying to remember. It's been more than -- probably more than three or four years ago.

Q. How much are you being paid for your time in this case?

A. $350 per hour for review.

Q. And how much for deposition or trial testimony?

A. It's 750 per hour.

Q. How much time did you spend reviewing materials in this case?

A. I spent 44 hours.
Q. And you know that from memory?
A. Yes.
Q. You don't have any time cards or time sheets or invoices with you today?
A. No.
Q. Have you created any type of time sheet or invoice?
A. No.
Q. How do you know it's 44 hours?
A. I keep track in my mind.
Q. Is that an effective way of getting paid? I'm joking, partially.
A. It's just what I do.
Q. $350 an hour, and you don't write down your time spent at that rate?
A. It's 44 hours.
Q. Okay. And it's not written down anywhere?
A. Correct.
Q. Okay. What's 44 times 350? You went to school longer than I did, not that that automatically makes you more qualified. $15,400?
A. Yes. Yes.
Q. For the record, I got it before you did.
A. That's correct.

MR. SHOEBOTHAM: Also for the record, no one had a calculator.

Q. (BY MR. PATTON) Okay. Have you been asked to testify at trial in this case?

A. Yes.

Q. Do you know when the trial is scheduled?

A. In a relatively shorter period of time, in the next several weeks.

Q. Do you know where trial is at?

A. I don't know specifically.

Q. Is there any other information that you would like to review in forming or revisiting or confirming your opinions in this case that you have not been provided?

A. I can't think of any information at this point in time.

Q. What about the benzene content of the Ashland solvent blends? Remember how in your report you said there's unpublished information?

A. Yeah.

Q. Would you like to see published information on benzene content?

A. I -- if there was some information available, that would be of interest to see, yes.

Q. And I'm talking about that line on Page 3 of

your report.

A. Yes.

Q. Tell me again about the basis for your opinion -- or statement, rather, that unpublished information indicates considerably lower benzene content compared to the assumptions of Dr. Nicas. Was it just that one phone conference with Morgan Gaskin?

A. Yes. And then, of course, subsequently, it's Dr. Whitlock's deposition.

Q. Does it bother you that the -- that this case involves benzene exposure, involves benzene alleged as a constituent or contaminant in the Ashland solvent blends? You're working for Ashland in this case, but Ashland can't tell you how much benzene is in that product. Does that bother you?

MR. SHOEBOTHAM: Objection, form.

A. Well, I think that Dr. Whitlock would have an accurate idea of what was in there.

Q. (BY MR. PATTON) But don't you think Ashland -- wouldn't you expect Ashland to have documents supporting Mr. Whitlock's assertions?

MR. SHOEBOTHAM: Objection, form.

A. You know, an industrial hygienist can answer that question. I -- I don't know about those issues, actually.

Q. (BY MR. PATTON) Do you always take your client's word for it, or do you ask for documents or other proof to back up their statements?

A. Well, I often ask for other information if it's available.

Q. And did you ask for other information in this case?

A. Yes.

Q. Because you wanted to see more than just Whitlock's statements, true?

A. Well, I wanted to see if there was other information, yes.

Q. And continuing on Page 3, you talk about approximately one-half of the amount of benzene inhaled is absorbed into the body. Approximately one-half of absorbed benzene is excreted, unchanged, via the lungs over a 36-hour period, depending on exercise level and amount of body fat. So, is that to say that in a given day if a person were to inhale one unit of benzene, okay, half that unit would go right back out within 36 hours?

A. Yes.

Q. Can you explain that more for me? I guess I should try to get it in your words, rather than me reading your sentence.

Tell us about your opinions and your understanding of how benzene comes in the body and then leaves the body and what the bases of those opinions are.

A. It's actually very simple, that benzene has a -- what we call a "vapor pressure" so that a solution of benzene in a container, an open container, will have a little bit of vapor of benzene above it going into the air. So, if you inhale some air that has benzene in it, then your body will absorb about a half of that. The other half comes right back out in the next breath.

And then of the one-half that you have now absorbed, over 36 hours half of that comes back out of the body if you're no longer exposed, being exhaled through the lungs.

Q. So, 25 percent of what was originally inhaled stays in?

A. Approximately, yes.

Q. Okay. And then does that ever go back out?

A. Yes. It's handled by the body. It's excreted by the kidneys. It's excreted in the bowel movement. It's metabolized in metabolites, and go out the kidney.

Q. All humans metabolize things differently, right?

A. Within range, yes.

Q. I could go to McDonald's and eat a Big Mac and you can eat -- go to McDonald's and sit next to me and eat a Big Mac, and our bodies would metabolize the substances in that Big Mac differently, right?

A. We might generate different amounts of body heat from that fuel source, that's correct.

Q. And then, as far as the amount of time that it would take for all the ingredients or substances in that Big Mac to come back out of our body or to stay or whether it goes to the liver or the kidneys, my body would handle that differently than your body would, correct?

A. Within a somewhat narrow range, yes.

Q. Okay. And the point that I'm trying to segue into here is: All individuals handle and metabolize products or substances a little bit differently, correct?

A. That's true. That's why the genetics becomes important.

Q. Have you testified in any cases involving, like, alcohol or drugs?

A. Yes.

Q. Okay. And so, different people respond to alcohol and drugs differently than others, right?

A. Again, within a somewhat narrow range, yes.

Q. I could drink a bottle of whiskey here today and you drink a bottle of whiskey today, and our bodies would react differently to that?

A. Depending on what reaction is measured, it might be somewhat different --

Q. Okay.

A. -- possibly.

Q. Some people are more susceptible to feel the effects of a given substance, so to speak, true?

A. Within a range, yes.

Q. Okay. And just as some people are allergic to cats and dogs, other people are not allergic to cats and dogs, true?

A. That's allergy, which is not really dose response.

Q. Okay.

A. That's a different process.

Q. When we talk about dose response, though, let's -- coming back to Plaintiff X, who had 401 ppm-years of benzene exposure. If you had two different people, Person 1 and Person 2, and let's say that they -- each of them worked side by side their whole life with benzene and one had 2000 ppm-years of benzene cumulative dose and the other had 2000 ppm-years cumulative dose, one person might get AML -- even though

both are at an increased risk, one person might get AML, one person might not get AML, true?

A. That's true.

Q. Okay. Why the individual difference?

A. It's genetics.

Q. Some people are more genetically susceptible to feel the effects of a dose response relationship than others, true?

A. Within a range, yes.

Q. Do the studies that talk about benzene as a causative factor of bone marrow diseases -- do they talk genetic susceptibility?

A. Only the more recent ones begin to address that somewhat.

Q. Do any of the studies cited by your report talk about individual genetic susceptibility?

A. Tunca and Zhang approach that, yes.

Q. What do they say about it, generally? I mean, we could all go read the report, but what are your opinions on what they say or --

A. Well, what we've talked about before, which is the chromosome aberrations, whether you can have a chromosome gain or chromosome loss within cells, or polyploidy, those types of phenomenon.

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

(Short break.)

MR. PATTON: Back on the record.

Q. (BY MR. PATTON) We were talking on the break about the list of cases that you've testified in, Dr. Snodgrass, and it's my understanding there's some discrepancy between Exhibit 3 and what you've actually testified in. Could you or your attorney explain that for us?

A. Yes. There is a case called "Scott" that involved benzene, and also a case entitled "Boren," B-o-r-e-n, that involved benzene.

Q. And you were working with Ashland's law firm in this case on both of those cases, right?

A. Yes.

Q. All right. And in each of those cases was it your testimony that benzene was not a causative factor in the plaintiffs' diseases?

A. Yes.

Q. Do you know what the diseases at issue in each of those cases were?

A. I don't, actually, at the moment.

Q. But in any event, it was your testimony that benzene played absolutely no role in causing the individuals -- workers, in those two cases?

A. That's correct.

Q. And you have never testified in a case that benzene played any causative role in a person's bone marrow disease, true?

A. I have not been presented a case that way, yes, correct.

Q. In your opinion, does the Strom study tell us definitively that benzene exposure does not cause RARS?

MR. SHOEBOTHAM: Objection, form.

A. It -- the data would -- I would interpret it to say it's highly -- highly likely, highly likely that it does not cause RARS.

Q. (BY MR. PATTON) Okay. And which part of the study tells you that or do you interpret that way?

A. Well, it's an overall result from the study, or you could find some of the -- for example, Table 6 has data that's -- lists that information.

Q. Okay. Which part is it?

A. Right there (indicating).

Q. Okay.

A. So, on Table 6, you'll see a section of benzene or solvents or gasoline exposure; and then the -- they're looking at what we call a -- "an odds ratio." And the odds ratio could be significant. The lower limit of the confidence interval has to be above 1.0 --

Q. Okay.

A. -- otherwise, it's not significant.

And so that what you see is that -- for the RARS group, that none of the odds ratios for having that, either refractory anemia or RARS -- first of all, none of those odd ratios are above 2, which is generally accepted to be a more significant odds ratio; and secondly, the confidence intervals are -- you know, the lower limit is always below 1.

And then, the trend for the significance of the test in the case of the refractory anemia with excessive blasts, which can go on to become leukemia in a greater percentage -- perhaps maybe 20 percent or more of those, perhaps, can go on to become, for example, a leukemia; whereas, with the RARS, much of the data says less than 1 percent -- the trend for the test is not significant. There's a number here of .34. Whereas, for the excessive blasts, it's significant, which is a value less than .001. That's a statistical test.

Q. In Table 6 here, they examine -- well, first of all, how many people in this RARS category are investigated or explored?

A. There are -- there were a total of 354 cases of all types together, and there were 67 that have refractory anemia and there were 38 that had RARS.

Q. Okay. And so, this column -- this first column

here in Table 6 at the bottom where you're talking about benzene, that's exploring 67 people with RA, 38 people with RARS, right?

A. Yes.

Q. And they asked those people what kind of benzene solvent or gasoline exposure they had, None, Low/Medium, or High, correct?

A. Yes.

Q. And how much benzene solvent or gasoline exposure is meant in the "None" category?

A. There was something called the "job exposure matrix" -- they list it as "JEM" -- which is developed by the National Cancer Institute, and it assigns a level of intensity of exposure for job titles. And it's None or Low or Medium or High, and it was -- it was asked to them in a questionnaire.

Q. So, how much benzene were the people in the Low/Medium category exposed to?

A. They do not have actual air level data or milligram-amount exposure information.

Q. So, on Table 6, we don't know what dose of benzene/solvent/gasoline any of the 67 RA people or 38 RARS people were exposed to, true?

A. You actually have an estimate of the dose, you don't have a specific measurement of it.

Q. Okay. Do we have an estimate -- do we know what the estimated dose is for each of these categories?
A. It's not specified in the paper, no.
Q. Okay. Do you know how they went about estimating dose?
A. They estimated it based on the --
Q. On the job category?
A. Yes, in that reference. I would have to go back and show you to explain it. It's the Dosemeci in 1994.
Q. What footnote is that?
A. That's Reference 16 in the -- to that Strom paper.
Q. Okay. So, what does it mean when the result of Table 6 here is not statistically significant? What does that mean to us?
A. It means it's not related.
Q. Okay. And then what's the problem with the odds ratio here?
A. It just shows it's not related.
Q. Okay. So, this tells us that benzene/solvent/gasoline exposure is not related, statistically, significantly to RARS for the 67 people who filled out a questionnaire and happened to be at M.D. Anderson, as well as the 38 people with RARS who

filled out the questionnaire and happened to be at M.D. Anderson at this time frame when the study was conducted, true?

A. Yes.

Q. Okay. So, putting aside the -- well, how many years of literature do you think there are out there that talk about benzene and leukemia and bone marrow diseases? 20, 30, 40 years of literature out there?

A. Yes.

Q. So, in forming your opinions in this case, you essentially chose to put aside the 20, 30, 40 years of literature and focus on one study from 2005 examining 67 refractory anemia patients and 38 with refractory anemia with ringed sideroblasts patients who happened to be at M.D. Anderson at some point in the mid 2000s and filled out a questionnaire which asked them how much benzene/solvent/gasoline they were exposed to, and determined that there was no statistically significant relationship? That's your opinion?

MR. SHOEBOTHAM: Objection, form.

A. No.

Q. (BY MR. PATTON) Where am I wrong?

A. I looked at enormous amounts of literature going back many years.

Q. And all that literature tells you that there's

just no way benzene was a causative factor in Charles Wilson's MDS/RARS, true?

MR. SHOEBOTHAM: Objection, form.

A. That's correct.

Q. (BY MR. PATTON) Have you reviewed any quantitative exposure data concerning Charles Wilson's benzene exposure in forming your opinions in this case?

A. I have reviewed the testimony of the chemist, but nothing further.

Q. Do you have any opinions as we sit here today as to what level of exposure to benzene Charles Wilson experienced by use of the Ashland solvents and other printing solvents in his 26 years of work as a printer?

A. I think the industrial hygienist will address that better than I for -- for an air level. I -- I don't recall his numbers at the moment. I did look at some of that.

It's a low air level exposure. So, it would be -- if you look at how Dr. Nicas calculated his 18.1, that -- this would be considerably below that.

Q. So, do you have a number that you're relying upon that you consider to be Charles Wilson's level of exposure to benzene in this case?

A. I think the best number will come from Mr. Spencer. I would defer to him to give you that

information, but I think it's going to be a much, much lower level than the 18.

Q. What information -- I don't want to know what information he gives me. What information did he give you?

A. I'll have to look at his material.

Q. Well, let me back up. In forming your opinions prior to your report, what did you understand to be the level of exposure that Charles Wilson experienced, less than 18.1 and that was it?

A. It would be that -- less than 5, say, 2-and-half parts per million in the benzene content of the solvent; therefore, that would translate into a low air level exposure.

Q. What if someone came out with study tomorrow and it -- we'll call it the "Jones study." Okay? Let's say, the Jones study -- well, let's call it "Strom 2." Okay?

Let's say a second Strom study came out and it had this same Table 6. What number would have to be in these benzene/solvent/gasoline categories for you to feel that there was a positive correlation or a statistically significant causal relationship between benzene/solvent/gasoline exposure and RARS for you to give the opinion that benzene exposure can be related to

RARS?

A. What you would want to see would be data that's statistically significant and, therefore, relatable.

Q. Okay.

A. So, the odds ratio would be above 2, and the confidence interval would be -- the lower limit of the confidence interval would be greater than 1.0.

Q. So, let's say the Strom 2 study comes out tomorrow and it has an OR above 2 and a CI with the lower limit above 1.0 --

A. Yes.

Q. That would be enough for you to say, "I, Dr. Snodgrass, believe there is a positive correlation between benzene exposure and RARS and that benzene exposure is a possible causative factor of RARS"?

MR. SHOEBOTHAM: Objection, form.

A. Yes.

Q. (BY MR. PATTON) Okay. So, how would you reconcile having the Strom 1 study which says no correlation and the Strom 2 study which has a correlation? Why do you pick Strom 2?

MR. SHOEBOTHAM: Objection, form.

A. Well, I might not have picked it, if it's weak enough. If it's -- I would look at these various characteristics, some of which are included in the

Cochrane kind of criteria: What are the numbers of persons; what are the other characteristics of information that are available for them; what are the confounders; and probably try to look for additional information -- perhaps in the second paper there might be more additional information about the -- something related to the dose exposure; and put all that together and say, "Which is the better quality paper?"

Q. (BY MR. PATTON) What if it's the same, same number of people, same information or lack of information on dose, same confounders?

A. Right. Then -- and the conservative approach would be to say there's a risk.

Q. So, then you -- then you could opine, yes, there is a risk of an increased chance?

A. Yes. Biologically, that's not likely to happen, but I understand your question.

MR. PATTON: I'll object to that last part as nonresponsive.

Q. (BY MR. PATTON) Have you had any conversations with any industrial hygienists as it relates to exposure levels Charles Wilson would have experienced of benzene?

A. No.

Q. You didn't talk to John Spencer, correct?

A. That's correct.

Q. Did you talk to Buddy Whitlock?
A. No.
Q. Did you talk to Tom Keenan?
A. No.
Q. Did you talk to anyone at Ashland?
A. No.
Q. Did you talk to anyone besides Ashland attorneys in forming your opinions in this case?
A. No.
Q. Did you talk to any of your colleagues about this case?
A. No.
Q. Did you talk to any hematologists about this case?
A. No.
Q. Did you talk to any oncologists about this case?
A. No.
Q. Did you ask to be put in touch with someone from Ashland to discuss this case and the chemicals at issue?
A. No. I just asked for that information we discussed earlier.
Q. Through Ashland's attorney?
A. Yes.

Q. The citations on Page 7 of your report.
A. Yes.
Q. Did Ashland provide any of those studies to you?
A. No.
Q. Did Ashland provide you with any literature that discusses what Ashland believes to be the relationship between benzene exposure and bone marrow diseases?
A. I don't recall any, no.
Q. They didn't give you any studies or any other authority on the subject, true?
A. That's true.
Q. The studies you rely upon in forming your opinions, those come from your own library and your own experience, right?
A. Yes.
Q. Did you ask Ashland what additional studies or what additional literature they might have that perhaps would talk about these specific solvent blends and the incidence of leukemia or the incidence of bone marrow diseases in relation to the use of those solvent blends?
MR. SHOEBOTHAM: Objection, form.
A. I had the MSDS sheets. No, I did not ask Ashland.

Q. (BY MR. PATTON) Have you ever been to a printing facility?

A. Yes.

Q. Tell us about that.

A. It's been quite a few years ago. I visited a printing facility for one of the large newspapers in Washington, D.C.

Q. Have you ever been to any Ashland facility?

A. Not that I know of, no.

Q. Outside of the context of this case, which I understand you said you didn't talk to anyone from Ashland, have you ever otherwise spoken to anyone from Ashland?

A. Not that I know of, no.

Q. In the course of your clinical work, have you ever encountered a printer who was having some type of ill effects from exposure to solvents?

A. That is possible. I don't recall a specific case, but I get a lot of referrals from physicians. So, it's possible, but I don't recall a case.

Q. What kind of -- can you tell us about the type of referrals that come to mind that you were thinking of there?

A. Well, there are numbers of times where physicians, family practitioners or internists, are

seeing a person who is having some sort of symptoms or health complaints and the patient may believe it's related to some exposure at work or non-work; and because it's involving a chemical and -- though, I'm not trained in that area, they will refer the patient for me to evaluate.

Q. Did anyone else help you in forming your opinions or writing your opinions or writing your report or performing your research, anything of that nature?

A. No.

Q. You did this all by yourself?

A. Yes.

Q. Do you have a typist or someone, or do you type it yourself?

A. No, I type it.

Q. You have ten fingers that type as well as mine do?

The other cases that you've testified in as reflected on Exhibit 3, subject to the verbal amendment that we've had here today, did you have more than one piece of authority supporting your opinions in those other cases?

A. Sometimes.

Q. I mean, is it a fair statement that you're relying very heavily on the Strom study in supporting

your opinion that no causation of Charles Wilson's MDS/RARS by benzene exposure? You're relying very heavily on the Strom study, true?

A. Well, yes, it's true I -- I'm using that study because that's the most relevant to his diagnosis.

Q. And when you worked as an expert in these other litigation cases, did you rely in a similar fashion heavily on a given -- on one single given study because it was so specific to that person's diagnosis?

A. Most of those other chemicals or drugs involved have a lot more published data that was relevant to the question of the individual. Whereas, in this case, we -- in 2007, I'm only aware of this one paper that's this specific.

Q. This Strom study, it says here on the front page cases where patients registered at M.D. Anderson Cancer Center between 1999 and 2003 with no restrictions on age, gender, or ethnicity, right?

A. Yes.

Q. Okay. So, this is a four-year window of patients they took a look at, correct?

A. Yes.

Q. Did they go through and pull every RA patient and every RARS patient and then do the questionnaire or find the other information with each such RARS patient

during that four-year time period?

A. Yes.

Q. That's your understanding of the study?

A. Yes.

Q. Okay. You would agree with me, as a scientist, that if M.D. Anderson were to use a different four-year window for this study, let's say '95 to 1999, they would probably have different results, wouldn't they? They wouldn't have the exact numbers in Table 6, you wouldn't be inclined to believe, would you?

A. They might not have those same numbers, but the statistics would be likely the same.

Q. Why do you think the statistics would likely be the same?

A. That's why you do statistics, because now, once you've got this set of data, even if you don't take into account the biology, the likelihood that another set being not reproducible to this is very low, much less -- much less than 5 percent. So, it's a 95 percent or greater likelihood that the next set of patients will give you similar odds ratios and confidence interval information.

Q. Well, what if the sampling had -- instead of 38 RARS patients, what if it had 380 RARS patients?

A. Then you would get even tighter intervals

likely. So that the confidence interval might be -- probably not quite up to the 2.3 and the 3.3 you see there, but would probably be down in the low 2s, for example, you know, at the upper limit of the confidence interval. And so, that's -- that would be the effective numbers.

Q. But if there were 380 RARS patients examined in another study during a different four-year time period, the other -- the new 300, so to speak, they could possibly answer differently or inconsistent with the data that led to this table, right?

MR. SHOEBOTHAM: Objection, form.

A. That turns out not to be correct, because if you look at the statistical trend analysis here, the .001 for the RAEV -- so, it's not even 95 -- it's more than even 95 percent likely you'll get the same data. It's probably 99-something percent likely you'll get the same data and odds ratios, in that it is not significantly associated.

So, as you add more numbers, this will tighten up to even more significance that there is not an association.

Q. So, you believe this sampling of 38 RARS patients plus, combined with 67 RA patients, gives us a pretty reliable baseline on what would be found in any

other study?

A. Yes, sir. For this type of statistics. I had a minor in statistics when I did my Ph.D. And for this type of statistics, when you've got an "N" of 30 or greater, you're getting into the 95 percent confidence for your data, often.

Q. Even though they only took a snapshot of a little over 100 people?

A. That's right. And so, the likelihood of that being repeated on different time windows is quite high.

Q. Have you reviewed Dr. Haas's deposition in this case?

A. Yes.

Q. And what opinions or -- opinions and/or criticisms do you have of what Dr. Haas had to say about Mr. Wilson's disease?

MR. SHOEBOTHAM: Objection, form.

MR. PATTON: How can I fix that question?

MR. SHOEBOTHAM: It's really broad. I don't know. The answer is: I don't know how to fix the question, but I think it's overly broad.

MR. PATTON: You just think it's a bad question?

MR. SHOEBOTHAM: I think it's overly broad and --

Q. (BY MR. PATTON) How about I ask a couple of more specific questions and that might help us get through.

Did you review Dr. Haas's deposition?

A. Yes, I did.

Q. When did you review it?

A. Yesterday.

Q. When did you receive it?

A. Day before that.

Q. Okay. And did anything stand out -- I mean, I see you flipping through the pages. And first of all, you didn't take any notes on his deposition --

A. No.

Q. -- did you?

A. I did not.

Q. Did anything stand out?

MR. SHOEBOTHAM: Objection, form.

A. I agree with him about there's probably going to be a reclassification in RARS as a distinct entity and that type of thing. What I'm looking for -- there was some part of this about the relationship to benzene, as I recall. He thought it was related to benzene, and I would disagree with that.

Q. (BY MR. PATTON) Because you disagree with Wintrobe, who Haas -- Dr. Haas calls the "godfather of

hematology/oncology," correct?

MR. SHOEBOTHAM: Objection, form.

A. Yes.

Q. (BY MR. PATTON) You disagree with the godfather of hematology and oncology?

MR. SHOEBOTHAM: Objection, form.

A. On that one sentence, the way it's stated, yes.

Q. (BY MR. PATTON) Because you think the godfather of hematology and oncology should be given a copy of the Strom study and revise his paragraph on benzene as a causative factor for MDS, true?

MR. SHOEBOTHAM: Objection, form.

A. Well, let's just say Dr. Wintrobe is no longer living, but he would be the first to look at new data and evaluate it carefully.

Q. (BY MR. PATTON) And he would take a look at this new data in the Strom study and the 105 people who were examined with the combined category of RA/RARS and he would say, "That's enough. Time to revise the textbook"? Is that your position?

MR. SHOEBOTHAM: Objection, form.

A. It's the best data we have, yes.

Q. (BY MR. PATTON) It's a small amount of data, isn't it? Isn't the Strom study a small amount of data, because it speaks to benzene as a cause of MDS and

benzene as a cause of the various sub-types?

A. It's a well-designed study, so it provides a lot of information. The numbers are not that small, necessarily. You would like to see larger numbers, but you also, in science, want to see reproducibility, and that will come with the next study.

Q. Okay. Other criticisms or opinions of Dr. Haas's testimony in this case?

A. Well, the one I'm recalling was the issue of relating it, benzene, as a cause of RARS, and I disagreed with that. That's all I recall.

Q. Okay. Do you believe Dr. Infante uses a sound methodology in formulating his opinions in this case?

A. Not entirely, no.

Q. What are your complaints in that regard or criticisms in that regard?

A. My criticisms are that he disregards other information regarding the strengths of -- and related to the quality of the information he's got and how you interpret that information; and in interpreting or incorporating, as an example, perhaps one or two cases of lymphocytic leukemia as saying that that's now relevant to benzene, when the majority of your scientists disagree with doing that.

Q. Couldn't some similar criticisms be made of

your opinions and your methodologies, though, in sort of boxing out much of the world of literature and focusing so strongly and heavily on Strom? Someone could make a similar criticism of you in that regard, couldn't they?

MR. SHOEBOTHAM: Objection, form.

A. No, because this is very focused on the specific diagnosis and it's separating out a dose-response relationship.

Q. (BY MR. PATTON) What's the dose in the Strom study?

A. They classified it as Low or Medium or High.

Q. How many ppm-years is Low, Medium, or High?

A. They don't have that information listed there.

Q. So, they don't really classify out the dose, do they?

A. It's not given a number, that's correct.

Q. Do you consider yourself a public health professional?

A. In a broad sense, yes.

Q. Do you agree that workers who have been exposed to benzene have a right to know about the hazards associated with that exposure?

A. I'm not an expert on right-to-know laws, but as a general rule, yes.

Q. And as a general rule, you would also agree

with me that a responsible chemical company should know how much benzene is in the products it sells, true?

MR. SHOEBOTHAM: Objection, form.

A. I'm not an expert in all that. Again, I think Mr. Spencer or others can answer that question better than I.

Q. (BY MR. PATTON) If someone were to come in your poison control center and, say, they had a bottle, okay, and the bottle came from "Company Z," and it was a beverage, okay, and they told you they were feeling sick and they showed symptoms of -- whatever disease, but acute, serious symptoms that required emergency medical attention, okay, would you want to know what chemicals or what ingredients were in the bottle that that person drank from Company Z?

A. We might, yes.

Q. Would you believe that you could go to Company Z and say, "Hey, I have a person here in our clinic who is sick; and he drank your product, and we think it might be related. Tell us what's in your product that he drank" -- you would expect a responsible product manufacturer to be able to give you that type of information, wouldn't you?

A. This was something made for drinking?

Q. Sure.

A. They would have some idea of what was in their product, yes.

Q. What if someone came into your clinic and they were exposed acutely to some type of solvent and they were having severe ill effects that required emergency medical treatment, would you want to know what was in the solvent they were working with?

A. For acute care, it often doesn't really matter if general solvents, but it would be useful information to have.

Q. Okay. And who would you -- where would you go to get that useful information? Where would be the first person you would go to?

A. If the source of the exact material were available and the manufacturer were known and one could contact the manufacturer, yes.

Q. That would be the first place you'd go, ask the manufacturer, because it's his product, right?

A. Unless it's a generally-recognized product, it might have other information that's published, generally, yes.

Q. But in any event, one of the first places you would look would be the manufacturer of that product, because you would believe that a reasonable, prudent, and safe manufacturer of chemicals would know what's in

that product, true?

MR. SHOEBOTHAM: Objection, form.

A. Depending on the percentages and all that, within range, yes.

Q. (BY MR. PATTON) You know there's benzene in stoddard solvent, right?

MR. SHOEBOTHAM: Objection, form.

Q. (BY MR. PATTON) You know that as a scientist, don't you?

MR. SHOEBOTHAM: Objection, form.

A. In the older formulations, yes.

Q. (BY MR. PATTON) Do you know that there's benzene in xylene, as a scientist?

MR. SHOEBOTHAM: Objection, form.

A. Well, again, Mr. Spencer can tell you more about that. That's very much dependent on the -- just to say there's some there, well, you know, is it a very, very small amount, or a very large amount, it's going to depend on the processes and how it's made.

Q. (BY MR. PATTON) So, like, for stoddard solvent, the benzene content would depend on -- the benzene content of a given sample of stoddard solvent would depend on a variety of factors: Where it's made, how it's treated, how it's blended, its reaction with other chemicals, those sort of things, right?

MR. SHOEBOTHAM: Objection, form.

A. Right. And you'd have to know those things in the time frame of the manufacturer.

Q. (BY MR. PATTON) Are you kind of skeptical of Buddy Whitlock's testimony that there's less than 10 ppm benzene in some of the chemicals at issue in this case?

A. No.

Q. But you acknowledge that that benzene range would differ, depending on refining methods, location, years, things of that nature, right?

MR. SHOEBOTHAM: Objection, form.

A. Correct.

MR. PATTON: Let's take another short break.

(Short break.)

Q. (BY MR. PATTON) Dr. Snodgrass, we've -- I've marked all the documents that I want to use as exhibits -- attached as exhibits to your deposition; and I want to go through those with you, quickly, for the court reporter's sake.

Exhibit 1, that is your report in this case, dated July 5, 2007, correct?

A. Yes.

Q. Exhibit 2, this was the list you and I were making of high-quality data.

And I had a follow-up question on that: Are there any publications that -- within which Plaintiff's experts cite papers as authority which you feel to be unreliable publications? For instance, there's citations to the American Journal of Industrial Hygiene or Journal of Environmental Medicine or Leukemia or Blood or other journals like that.

Did you make any type of review of Dr. -- of the references cited by Dr. Parent or Dr. Infante and their accompanying journals and say to yourself, "You know what, that journal is just unreliable," or "They'll publish anything," or "They publish low-quality data," things like that?

A. I don't recall seeing any journals of that type.

Q. Would you agree with me that the journals that contain the articles that Dr. Infante relies on and Dr. Parent relies on, they generally are respected, peer-reviewed scientific journals or other authority, the authority speaks for itself, that if they're publishing a report, that report is at least of some high enough quality, that it is relevant to the pertinent scientific community? Would you agree with that?

MR. SHOEBOTHAM: Objection, form.

A. Not as worded. The peer-reviewed literature is as good as the peer reviewer so that you have a range on that. So, I think it's -- I haven't sat down to do just that, what you've mentioned, which is to go through their references and look at the journals as -- because I probably would come up with, say, there's some journals here that are recognized, generally, not to have as generally high a quality papers just by common knowledge among scientists, but it doesn't mean that it's not peer reviewed.

Q. Some of the journals are like Ivy-League caliber journals, whereas, some might be lesser journals; is that a fair statements?

A. It's much more -- yes, it's much more difficult to get a paper accepted for publication in the journal called Science than it would be for, maybe, the International Archives of Occupational Medicine, for example.

Q. So, when you conclude that much of the data that Plaintiff's experts rely on is not high-quality data, you're not necessarily faulting the journal, correct?

A. Correct. It has nothing to do with the journal. It has to do with the paper itself and the data.

Q. Exhibit 3 -- well, first of all, is there anything else we should include on our list of what high-quality data means to you besides the Cochrane levels and their characteristics of quality data?

A. There are other categorization schemes, as well as the Cochrane, that list quality; but -- levels of evidence. There are those types of listings. But the Cochrane is one, as well. Cochrane and Oxford is one, as well, that's known.

Q. When you use the term "high-quality data," as you did a couple of times in your report, is that a phrase that you commonly use when opining in similar cases?

A. I don't know how common it is, but I perhaps have used it, yes.

Q. I mean, when you look at any given case-controlled study or other piece of scientific authority for the relevant field of benzene and blood or bone marrow disorders, it's real easy to go through and talk about the strengths and the weaknesses of each study. Isn't it pretty easy to just go through and make a blanket statement like, "The plaintiff's experts don't rely on high-quality, relevant data"?

MR. SHOEBOTHAM: Objection, form.

Q. (BY MR. PATTON) Isn't that a pretty easy

criticism to throw out there?

MR. SHOEBOTHAM: Objection, form.

A. It's a short, few words. I think, for me, it means that -- what is the strength of that data; what's the consistency of that data; what's the data source; what -- how does that relate to how we understand the biology and the dose issues; and, you know, how good is that study.

Q. (BY MR. PATTON) But you did not look at or review each and every study cited by Dr. Parent and Dr. Infante and review the individual strengths and weaknesses and quality, so to speak, according to Cochrane and/or others, and reach a conclusion each one of those was low quality, therefore, Plaintiff's experts failed to rely on high-quality data; is that a fair statement?

A. No. What I did was I realized that there was very few data on RARS regarding benzene and limited data, very limited data, on the whole grouping of various diseases called "MDS" and benzene compared to the enormous amount of data on benzene and AML; and therefore, when I used the information that Dr. Shatner's paper did, which was to go through -- and that paper went through over 400 articles -- and they -- and I spell that out in my report, is that they listed

their criteria for what would be essentially high-quality information regarding AML.

And you can apply similar kinds of specifics, as listed in my report, where either the MDS type of category or RARS and benzene, and that's what I attempted to do.

Q. Okay. And the only RARS/benzene study is Strom?

A. That's selective and specific for the RARS, itself, yes.

Q. Exhibit 3 is a true and correct copy of your list of testimony, subject to the verbal modifications today, right?

A. Yes.

(Snodgrass Exhibit No. 4 through 11 marked.)

Q. (BY MR. PATTON) Exhibit 4 is your Deposition Notice, and I included a subpoena duces tecum asking you to bring everything essentially bearing on your opinions and what you relied on and reviewed in this case. Have you done that?

A. Yes.

Q. You don't have any handwritten notes?

A. No.

Q. No summaries of any information?

A. No.
Q. Is there any other literature that you will rely on in expressing your opinions to a jury in this case, other than what you've brought with you today to your deposition or what you've cited in your report?
A. Not that I think of, unless a different type of question triggers a different type of memory and some other issue comes up, no.
Q. Doctor, I'm generally familiar with the 18 citations on Page 7 of your report -- come to think of it, I might not be positive as to which any of these are. If I were to convey a question to your attorney as to, you know, "What is the Rathman study," because that just doesn't ring a bell for me, today, right now, as we sit here, would you be happy and willing to provide that information so that it could be conveyed to me with a citation?
A. Yes.
Q. Okay. Exhibit 5 is your curriculum vitae, correct?
A. Yes.
Q. And this was in July 2007, but it's still current as we sit here today?
A. Yes.
Q. Have you published anything on benzene?

A. No.
Q. Have you ever made any presentations specific to benzene?
A. I don't recall any at this time.
Q. Have you authored any journal articles on benzene?
A. No.
Q. And you are a reviewer for journals, correct?
A. Yes.
Q. Have you reviewed, in the course of your peer review duties, any articles relating to benzene?
A. Not in terms of peer review. The prior question about any articles, I don't recall if we've published the data. It was -- I was specifically involved in animal work in the 1970s at the National Institutes of Health.

One of the chemicals we studied, we did a fair bit of work on, was benzene and bone marrow in an animal model and looking at some of the bio-chemical effects. And I just don't recall if we ever got that published. So, I -- but that's not -- it would not -- so, the answer to your question would be: There were no publications, I can recall.

Q. Who funded the study that you just talked about?

A. The U.S. Government.

Q. Has the chemical industry or any chemical company ever funded any of your work besides expert work in litigation?

A. I had one drug study for a medication funded partially by a drug company years ago.

Q. For instance, the Chemical Manufacturers Association or Ashland or Dow or any company like that, or organization of that nature has never funded any of your work besides litigation, correct?

A. Correct.

Q. What percentage of your income is derived from testifying as an expert in litigation?

A. 10 percent, 15 percent.

Q. What percent of your professional time is spent working on litigation matters such as this one?

A. I'd say around 10 to 15 percent.

Q. I marked as Exhibit 6, "Harrison's Principles of Internal Medicine, 16th Edition." I found this in your box. What is this document and its relevance in relation to your opinions in this case?

A. This is a couple of pages from a textbook of general internal medicine. In those pages, they discuss myelodysplasia, and it lists the various categories under the WHO -- World Heath Organization's

classification of myelodysplastic syndromes. And it simply list those, and including in that list would be RARS.

Q. Okay. Do you believe Harrison's Principles of Internal Medicine to be a reliable piece of authority in matters of medicine?

A. It has good information, generally.

Q. And just so that all of us lawyers have 16 copies of the Strom study, I went ahead and marked that as Exhibit 7 to your deposition, too. Okay?

A. Yes.

Q. And Exhibit 8 -- well, I also marked it, because we talked about it so much.

A. Yes.

Q. I marked as Exhibit 8 to your deposition, Evolving Classifications of the Myelodysplastic Syndromes. This is an article offered by Komrokji -- K-o-m-r-o-k-j-i -- and this appears to be from the Current Opinion in Hematology Journal, 2007; is that right?

A. Yes.

Q. How did you come across this article?

A. I -- like I did for the rest of it. I searched for articles relevant to this diagnosis.

Q. What is the value of this article in your

opinions in this case?

A. It -- the value is simply that it's -- it says, as we know now, that these are becoming -- as we get more information, that diagnoses like RARS are a distinct category, distinct diseases.

Q. Is it possible and even perhaps probable that the myelodysplastic syndromes could be reclassified in a different way in the next, say, five years such that the Strom study would be difficult to interpret or view as instructive on issues of benzene causation specific to the different MDSs or MDS sub-types?

A. No. What -- in the next five years -- it would just be my, of course, estimation of what might change -- but you may get some bio-markers that may correlate with the diagnosis, and then you could begin to apply some of those as blood tests.

Q. So, you could further stratify, as you would say?

A. Yes. Or support the stratification with a bio-marker, for example.

Q. What is the -- well, first of all, I'll identify, for the record, Exhibit 9 is the Institute of Medicine article entitled Gulf War and Health, dated 2003; and this is from Volume 2, Insecticides and Solvents, correct?

A. Yes.

Q. What is the value of this article on your opinions in this case?

A. The value is simply that a literature search was done and a review was done by an independent committee of scientists regarding benzene and its causation, including myelodysplastic syndromes. And they looked at many of the same studies that Dr. Infante and others looked at, and that one of their conclusions is, as stated here: "The committee concludes from its assessment of the epidemiologic literature, that there is inadequate/insufficient evidence to determine whether an association exists between chronic exposure to benzene and myelodysplastic syndromes."

Q. And that's on Page 330 of Exhibit 9?

A. Yes.

Q. What is the importance of Exhibit 10 in your opinions?

A. This was simply -- I went to the American Cancer Society website to see what they would say, and one of their comments about MDS is they -- similar to the past edition of Wintrobe's textbook, they have a statement about: Long-term workplace exposure to benzene and certain chemicals used in the petroleum and rubber industries can increase your risk of developing

MDS.

So, it's a general -- they used just a general wording of "MDS."

Q. And you don't like that general wording of "MDS"?

A. I think if it were more accurate, it would -- it would separate out the different diseases.

Q. Exhibit 11, what is the value of this in your opinions?

A. This is the Surgeon General -- part of a Surgeon General's report from three years ago, and it's -- it really relates mostly to AML leukemia, not to RARS or any of the other myelodysplastic-type listings.

Q. Is this the article that attributes the benzene from cigarette smoke to increased leukemia, or am I thinking of a different Surgeon General article?

A. I think that may have occurred earlier, but this one does go into that data, yes, it does.

Q. I want to talk about this other stack of materials, and I'm not going to mark this as exhibits; but you had in your file an Ashland document called Health Hazard Determination Guidelines?

A. Yes.

Q. And this appeared to be Bates No. Ashland-Wilson 3530-3594. Did you review this

document?

A. Yes.

Q. And how did that bear on your opinions or what opinions, if any, do you have on that?

A. This is just general information about chemicals, and some of it's just general chemical information about toxicology and how you do rating systems, for example. It's just sort of general toxicology background; so, there's a lot of different kind of information in here, but it's not necessarily only selective for benzene.

Q. It talks about the hazard determination that they use for all their solvents, correct?

A. Yes.

Q. Did you see a lot of documents from Ashland evidencing compliance with those guidelines?

A. Probably Mr. Spencer is in a better position to answer that. I saw MDS sheets -- MSDS sheets.

Q. Okay. Did you review the --

A. Yes.

Q. -- report letter by Dr. Kopstein?

A. Yes.

Q. What opinions, if any, do you have on that?

A. I -- I simply disagree with his -- his overall conclusion. He's relating his support of the estimates

from Dr. Nicas.

Q. Doesn't he also point out some of the shortcomings or inconsistencies of Ashland's position, per the testimony of Whitlock, as it comports with the peer-reviewed literature on benzene content for these products?

A. I'm going to leave that up to Mr. Spencer or Dr. Whitlock to really address those issues.

Q. Okay. The Toxicological Review of Benzene, this is just something from your general benzene library?

A. Yes, it is.

Q. And this is from September 1998, CAS No. 71-43-2?

A. Yes.

Q. What bearing does this have on your opinions, other than -- well, what bearing does it have?

A. It provides a lot of the background information on benzene.

Q. And the next one you have, can you read that for us?

A. The carcinogenic Effects of Benzene and Update. This is April of 1998. This is from the EPA.

Q. And then what's the next one?

A. This is the -- this next one is the

Toxicological Profile for Benzene, 1997. This is by the ATSDR, which is part of the Centers for Disease Control.

Q. And those are documents that you rely on, generally, in your opinions as they relate to benzene?

A. They contain a lot of useful toxicology information about benzene that's independently written, yes.

Q. Okay. Do you have any other opinions in this case or do you intend to testify at trial about anything else besides what we've talked about today?

A. Not that I can anticipate at this time.

Q. Thank you for your time.

MR. PATTON: That's all I have for now.

MR. SHOEBOTHAM: I have a few questions about Exhibit 5, which we'll define as the "CV."

First of all, before I ask the questions, can we just agree that there's a box of documents that he brought with him that haven't been marked as exhibits?

MR. PATTON: Right. Yes.

MR. SHOEBOTHAM: All right.

E X A M I N A T I O N

BY MR. SHOEBOTHAM:

Q. Okay. All right. Dr. Snodgrass, I just want to ask you some questions about Exhibit 5. What is

that?

A. That's my curriculum vitae.

Q. And what is a curriculum vitae?

A. It's like a resume. It just lists your past employment and, in my case, publications and teaching and research and patient care activities.

Q. Is that a true and correct copy of your current curriculum vitae?

A. Yes.

Q. And does this truly and accurately summarize your professional background and experience?

A. Yes.

Q. I'd like to ask you, if you would, could you tell the jury about your educational background?

A. I have a bachelor of science degree in chemistry from Butler University in Indianapolis, 1968; I have a masters in pharmacology from Indiana University, 1971; received my M.D. from a medical school in Indiana in 1972; and I have a Ph.D. in pharmacology/toxicology in 1976.

Q. Okay. Are you a medical doctor?

A. Yes.

Q. And where are you licensed?

A. In the State of Texas and the State of Indiana.

Q. And is your medical license current and on file

with the appropriate authorities?

A. Yes.

Q. In addition to being a medical doctor, do you have qualifications in the area of pharmacology?

A. Yes.

Q. What is pharmacology?

A. Pharmacology is the study of substances or drugs or medications that have a beneficial effect for treating diseases or illnesses.

Q. Would you describe for the jury your qualifications in the area of pharmacology?

A. I have a Ph.D. and a master's degree in pharmacology, and I'm also board certified by the sub-board called the American Board of Clinical Pharmacology.

Q. Do you have qualifications in the area of toxicology?

A. Yes.

Q. What is the science of toxicology?

A. Toxicology is the area that deals with the adverse or toxic effects of chemicals or drugs.

Q. And would you describe for the jury your qualifications and experience in the area of toxicology?

A. I have a Ph.D. that's in toxicology/pharmacology, and I also have -- am board

certified by the American Board of Medical Toxicology, and I'm the past president of the American Academy of Clinical Toxicology.

I'm currently the medical director for one of the 60 certified regional poison control centers in the United States.

Q. Right. Now, tell the jury, if you would, about that activity. What is your current position with regard to the Houston/Galveston poison control center?

A. I'm the medical director for the Texas Poison Center that serves Houston and Galveston and surrounding 5 million population.

Q. And what are your duties in connection with that position?

A. That's a 24-hour-a-day, seven-day-a-week service. I have 12 nurses and pharmacists who rotate shifts to answer the telephone from the public, as well as from physicians and nurses and hospitals, regarding either acute, or even chronic, exposures or concerns about exposures to substances. And so, I provide that information. When the -- it's beyond the level of a nurse or pharmacist to talk with either a person calling there or a physician, I will help provide information or management for helping to manage patients who are ill due to these substances.

Q. And are these patients people who are calling into the Poison Control because of concerns about some possible exposure to a drug or a chemical?

A. Yes.

Q. And so, that's something that you deal with on a daily basis in your position as the medical director of the Houston/Galveston poison control center?

A. Yes.

Q. Do you hold any medical board certifications?

A. Yes.

Q. What are those?

A. I am -- my subspecialty -- the two sub-specialties were the American Board of Medical Toxicology and the American Board of Clinical Pharmacology, and then my primary board specialty is the American Board of Pediatrics.

Q. Do you have any position with the University of Texas?

A. Yes.

Q. What is your position with the University of Texas?

A. I'm a professor of pharmacology-toxicology, professor of pediatrics, and a professor of obstetrics and gynecology at the medical school.

Q. And where is that medical school located?

A. In Galveston.

Q. And do you have any teaching responsibilities at the University of Texas?

A. Yes.

Q. Would you describe those for the jury?

A. My teaching includes teaching resident physicians who are in training; teaching medical students; providing seminars or teaching to CME, continuing medical education, meetings for physicians in practice; teaching graduate students, who are in training for their Ph.D. in pharmacology and toxicology.

Q. Do you have any positions as a reviewer for any medical journals?

A. Yes.

Q. Would you describe for the jury some of the medical journals that you are a reviewer for?

A. I've been a reviewer for the New England Journal of Medicine; the Journal of Toxicology, Clinical Toxicology; the Drug Metabolism Disposition Journal; Journal of Pediatrics; Toxicology & Applied Pharmacology Journal; Developmental Pharmacology Therapeutics. The Poisondex Information System, I've been a reviewing editor.

I've been an invited reviewer for the United States Environmental Protection Agency for

particular issues, such as "Pesticides", in the past. Those are an example of some of the reviewer's activities.

Q. Do you currently hold any positions in which you have been requested by the United States Government, to serve on committees or be available to serve on committees, if necessary?

A. Yes. I'm an immediate past voting member for the Nonprescription Drug Advisory Committee to the FDA, and an immediate past temporary voting member for the Anesthetics & Life Support Drugs Committee for the Advisory Committee to the FDA.

I'm a current member of the Advisory Committee for Lead Poisoning and Prevention in Childhood for the Centers For Disease Control.

Q. Dr. Snodgrass, have you formed an opinion within reasonable medical probability as to whether the chemical, benzene, can cause the illness, refractory anemia with ringed sideroblasts, the disease that Mr. Wilson has?

A. Yes.

Q. What is that opinion?

A. Benzene is not a cause of RARS.

Q. And what is the basis for that opinion?

A. The basis for that opinion is -- includes the

Institute of Medicine Review, it also includes data published by Strom and others, showing no relationship.

MR. SHOEBOTHAM: I'll pass the witness.

R E - E X A M I N A T I O N

BY MR. PATTON:

Q. If we didn't have the Strom 2005 study with us today, you would not be able to give the opinion that you just gave, that you just -- what you just said to Mr. Shoebotham, correct?

A. There would not be that specificity of RARS versus just a general listing of all the types of substances -- disorders that are MDS, yes.

Q. In the course of your years of professional work in the field of toxicology, have you encountered Plaintiff's expert, Dr. Parent, before?

A. I've heard the name, but I don't know him.

Q. Have you ever spoken to him?

A. No.

Q. Do you have any criticism -- did you review Dr. Parent's curriculum vitae that was attached to the Plaintiff's expert disclosures?

A. Yes.

Q. Do you have any criticisms of Dr. Parent's qualifications to serve as an expert in this case to the same subjects that you're serving as an expert on?

MR. SHOEBOTHAM: Objection, form.

A. No.

Q. (BY MR. PATTON) Okay.

MR. PATTON: Mr. Shoebotham, can we have the agreement that I'll take the exhibits and make copies of them and send them back to you, and then you can make copies and return to your expert?

MR. SHOEBOTHAM: I agree.

MR. PATTON: Okay.

MR. SHOEBOTHAM: If that's all right with you, Dr. Snodgrass.

THE WITNESS: That's fine, yes.

MR. PATTON: That's all I have.

MR. SHOEBOTHAM: That's all I have.

(At the hour of 1:07 p.m., the deposition was concluded.)

* * *SIGNATURE REQUIRED* * *

CHANGES AND SIGNATURE

WAYNE R. SNODGRASS 9-14-07

PAGE	LINE	CHANGE	REASON

_____

JURAT

I, WAYNE R. SNODGRASS, have read the foregoing deposition and hereby affix my signature that same is true and correct, except as noted above.

_____

WAYNE R. SNODGRASS

THE STATE OF TEXAS)
COUNTY OF HARRIS)

Before me,_____, on this day personally appeared WAYNE R. SNODGRASS, known to me (or proved to me under oath or through _____) (description, 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_____

IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF TEXAS
MARSHALL DIVISION

CHARLES WILSON and LAURA WILSON,	)	
Plaintiffs	)	
vs.	)	CIVIL ACTION NO.2:06-CV-286
RYCOLINE PRODUCTS,	)	
Defendants	)	

REPORTER'S CERTIFICATION
DEPOSITION OF WAYNE R. SNODGRASS
September 14, 2007

I, Brenda L. Kaplan, Certified Shorthand Reporter in and for the State of Texas, hereby certify that the facts stated by me in the caption hereto are true; that the foregoing deposition transcript of WAYNE R. SNODGRASS, the witness hereinbefore named, was at the time mentioned taken by me in stenograph, the said witness having been by me first duly cautioned and sworn upon his oath to tell the truth, and later transcribed from stenograph under my supervision.

I further certify that I am neither attorney or counsel for, nor related to or employed by any of the parties to the action in which this

deposition is taken, and further that I am not a relative or employee of any attorney or counsel employed by the parties hereto, or financially interested in the action.

I further certify that the above and foregoing deposition transcript as set forth in typewriting is a full, true and correct transcript of the proceedings had at the time of taking said deposition.

WITNESS MY HAND, this the _____ day of _____, _____.

_____

BRENDA L. KAPLAN, Texas CSR 6977
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