IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF IOWA
CENTRAL DIVISION

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|---|---|---|
| MARGARET J. MIELKE, individually as the surviving spouse, as the Administrator of the Estate of and as the Wrongful Death Beneficiary of Walter J. Mielke, Deceased, | } | |
| Plaintiff, | } | No. 4:05-CV-88 |
| vs. | } | |
| ASHLAND, INC., et al., | } | |
| Defendants. | } | |

**********

ORAL DEPOSITION OF

ETHAN NATELSON, M.D.

November 27, 2006

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ORAL DEPOSITION OF ETHAN NATELSON, M.D., produced as a witness at the instance of the Plaintiff, and duly sworn, was taken in the above-styled and numbered cause on the 27th of November, 2006, from 1:12 p.m. to 4:29 p.m., before Janet G. Hoffman, CSR in and for the State of Texas, reported by machine shorthand, at Vinson & Elkins, 1001 Fannin Street, Suite 2500, Houston, Texas, pursuant to the Federal Rules of Civil Procedure and any provisions stated on the record or attached hereto.

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ETHAN NATELSON, M.D.,
having been first duly sworn, testified as follows:
EXAMINATION
BY MR. PATTON:

**Q. Please state your full name.**
A. Ethan Natelson.
**Q. Dr. Natelson, my name is Keith Patton. I represent Margaret Mielke in this case. Do you understand that?**
A. Yes.
**Q. If, at any time, during your deposition today you don't understand one of my questions, could you please ask me to rephrase it?**
A. Yes.
**Q. And can we have a mutual understanding that if you do answer my question, that's to say that you understood the question? Is that accurate?**
A. Yes.
**Q. Okay. What type of doctor are you?**
A. I'm what's known as a hematologist.
**Q. And are you practicing physician?**
A. Yes.
**Q. Do you practice here in Houston?**
A. Yes.
**Q. And how long have you been practicing as a hematologist in Houston?**
A. Since 1971.
**Q. Are you board certified in any specialties?**
A. Yes. Internal medicine and hematology.
**Q. And when did you become board certified in hematology?**
A. 1974, I believe.
**Q. What do you understand to be your role in this case?**
A. Well, I was asked to review the medical records and give an opinion about causation of acute myeloid leukemia.
**Q. Have you ever diagnosed anyone with benzene-related acute myeloid leukemia?**
A. Well, I've diagnosed a patient with what I thought was chemical-induced leukemia, and benzene was one of the chemicals the patient was exposed to. But I couldn't narrow it down for only benzene.
**Q. And is that only one patient that might fall in that category?**
A. Yes.
**Q. So is it fair to say that in your 30 or more years of hematology practice, you've only diagnosed one patient whose AML you would attribute to leukemia. Is that a fair statement?**
A. Whose AML I would attribute -- you said AML --
**Q. AML you would attribute to benzene or chemical exposure. Is that fair?**
A. Well, I wouldn't know about -- I have seen other patients who have had chemical exposures, but it wasn't defined what they were due to. In other words, I've seen another patient with myelodysplasia who worked for many years as a chemist; and I don't know what all he was exposed to. And it's possible his illness was chemical-related.
**Q. Besides it being possible that one or more patients that you've seen in your 30 years or more of practice may have AML which is chemically related, have you ever concluded that a given patient's AML was caused by benzene exposure?**
A. No.
**Q. Have you ever treated anyone with polycythemia vera -- which I'll refer to throughout your deposition as PV. Is that fair?**
A. Correct.
**Q. Have you ever treated anyone with PV?**
A. Yes.
**Q. How many patients in your 30 or more years, if you can estimate?**
A. I would say right now probably I'm currently

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seeing about 10 patients with that illness. Over the years, many, possibly as many as 100.

**Q. So over your 30 or more years of practice, you've -- again, I'm not holding you to this, but you'd estimate you've seen approximately 100 hundred or so patients with PV?**

A. Yes. Those are soft numbers, but it's an uncommon illness, but for hematologists, not all that uncommon.

**Q. Is it your opinion in this case that Walter Mielke's AML is not related to benzene exposure?**

A. Yes, that is correct.

**Q. Is it your opinion in this case that Walter Mielke's AML is not related to any benzene exposure?**

A. Yes.

**Q. If asked to testify at trial in this case, would your testimony be that benzene exposure played no role in Walter Mielke's AML?**

A. Yes.

**Q. Is it your opinion that Walter Mielke's exposure levels to benzene, whatever they were, were not enough to cause AML?**

A. Well, your expert, one of them, estimates his cumulative doses, I think, around 30-some-odd part per million, 32 or 33. I don't believe, if that level is

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correct, that that would be sufficient to cause AML.

**Q. Do you have any disagreement with the diagnosis of AML for Walter Mielke?**

A. No. He has a form of AML we call a granulocytic sarcoma, but it is considered to be AML, nonetheless.

**Q. Do you agree with me that there's ample scientific evidence showing that benzene causes AML?**

MR. CHILTON: Object to the form.

A. Yes.

**Q. Of all the scientific evidence available, do you believe that the strongest case of benzene exposure as a cause of blood disorder is strongest for the relationship between benzene and AML, as opposed to other blood disorders?**

MR. CHILTON: Object to the form again.

A. Well, the only, let's say, malignancy of the blood that benzene causes is acute myeloid leukemia. I would put myelodysplasia in with that illness because it's usually lumped together.

**Q. AML is potentially a deadly cancer. Is that correct?**

A. Yes.

**Q. Is there an average time between diagnosis of AML and death?**

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A. If you're talking about what we call typical de novo leukemia, where the AML starts abruptly, I would say untreated, most people would be, the older literature suggests, about eight to ten weeks.

**Q. Is there any other way to describe or classify the average time between diagnosis and death? For instance, someone with de novo AML who's not treated as compared to somebody with secondary AML that's treated, is there another way you can explain how you would describe that relationship between the time of AML diagnosis and death?**

A. Yes. I think -- in many people, or certainly a number of people, with secondary AML, it's not as explosive an illness as de novo AML. In other words, a person who gets a secondary AML may sort of get it through the back door. They have a slower increase in blast forms in the bone marrow over time, so that their illness, you might say, is not as aggressive in character; it still may be fatal, but it's not as explosive as many cases of de novo leukemia. So a secondary leukemia -- in fact, many times we'll even argue if it's leukemia or if it's myelodysplasia because we look at the blast count as an indicator of whether we call it leukemia. And there isn't universal agreement about how many blasts percentagewise it takes to make

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that diagnosis.

It used to be that one was required to have 30 percent blasts in the marrow to make the diagnosis of acute leukemia. Today that level has been lowered to 20 percent blasts in the marrow. In the case of MD Anderson Hospital, they often will treat people for acute leukemia at 10 percent blasts. So occasionally you'll see people who are clearly not normal, they have an increased numbers of blasts. And it's not always clear whether one wants to put that in the acute leukemia pile or the MDS pile.

And so that's why I say a secondary leukemia may be a little slower in its expression than a de novo leukemia. But a de novo leukemia untreated, survival is very short.

**Q. So I asked a bad question? It's hard to answer that?**

A. It's a difficult question to answer.

**Q. When we use the term -- or I should say when you use the term secondary leukemia, as you will probably throughout your deposition and as you reference in your report -- what do you mean by a secondary leukemia?**

A. Well, secondary leukemia -- and I think I may have put someone's definition in there -- secondary

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leukemia is a leukemia that has a background of it could be an illness that predisposes to leukemia, like myelodysplasia or myeloproliferative disease. It could be a leukemia induced by the chemotherapy drugs we commonly use today. It could be a leukemia induced by a radiation exposure. It could be a leukemia induced by environmental chemical exposure, such as, let's say, benzene today.

Those are typical. It could also be a leukemia that -- let's say there is some genetic predisposition, in other words, an unstable hereditary chromosome abnormality, something of that nature. In other words, we distinguish that from de novo, which is a category of leukemia in which we don't find any predisposing cause. And it has certain characteristics. In other words, de novo leukemia has a certain pattern to it. Secondary leukemia has a different pattern.

It's generally thought that about 85 percent of all AML is de novo, perhaps 15, maybe 20, percent are secondary. So a de novo is the more common form of AML.

**Q. What is the source or basis of your opinion for that 85 percent, slash, 15 or 20 percent?**

A. There are many, many articles in the literature that suggests a fellow by the name of Petters

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Bejeurgard (phonetic) has written that numerous times. There may be one in here that mentions that by Smith. That's just sort of a standard number that I could reference probably 25 articles that say that.

**Q. So you consider it to be generally accepted in the medical and scientific community that approximately 15 to 20 percent of all AMLs are secondary AMLs. Correct?**

A. Correct.

**Q. What exposure levels are necessary, in your opinion, for benzene to be a cause of AML?**

A. Well, the data isn't wonderful; but based on the pliofilm exposure is what the EPA generally uses. And Dr. Wong is the one who assayed that by looking specifically at the AML cases, comes up with numbers of in excess of 200 part per million years. People have argued about that, and the reason is is the data is not wonderful and the number of cases are small. But it is obviously very high.

**Q. So for you to conclude that benzene could be a possible cause of the secondary AML, you would need to see 200 PPM years of cumulative exposure before you would even consider benzene as a cause. Is that a fair statement?**

A. I would have to see a very high level. And

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there are, of course, other features of secondary leukemia that one would like to see.

Just as an example, in so-called de novo AML, it occurs very abruptly, so the blood counts are, let's say, normal, maybe even two weeks preceding the diagnosis. It's a very sudden onset.

In the case of secondary leukemia, probably two-thirds of the time you see a period of myelodysplasia, where the bone marrow is sick but not frankly, or floridly, leukemic. If I was making a diagnosis of secondary leukemia, I would like to see -- a helpful feature would be that there was some period of myelodysplasia before the onset of the AML. I'd like to see that there was a substantial exposure to benzene. I would like to see that the latency period from that chemical exposure to the onset of leukemia was within a certain window of time. I would like to see certain patterns of chromosome abnormalities or some chromosomal abnormality at all, because it's very rare to find a benefit-induced leukemia without an obvious chromosome abnormality.

So it's a constellation of things. But getting back to your original question, yes, the high dose, of course, is one of the things we look for.

**Q. I might have missed a couple of notes when I**

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**was listening to your answer; but just so I'm clear, for you to conclude that a person's secondary AML was caused by benzene exposure, you would need to see substantial exposure, which I have some questions about, a certain latency window, certain chromosomal abnormalities. And what else? What did I miss?**

A. A period of myelodysplasia. In other words, those are things that favor a diagnosis of secondary leukemia over a de novo leukemia.

**Q. When you say substantial exposure to benzene, what do you mean by substantial exposure?**

A. Well, I would say that we know, from many modern cohort studies, that for many years it's difficult to identify an increased incidence of AML among people who have had some benzene exposure but not high-grade exposure.

From one of these articles -- I referenced the Bloemen article, which is, I think, a Dow Chemical article -- they have a part-per-million cumulative estimate in their group of 40 part per million years cumulative dose, so they don't have any increased -- statistically significant increase in AML. So it's obviously higher than 40.

Wong's data says it's 200. Other articles say maybe it's even as high as 400 or 600. I really

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don't know how high it is, but I would like to see at least in the 100 to 200 part-per-million range, if they could be estimated.

**Q. Are you familiar with the surgeon general's report on smoking and AML?**

A. Generally.

**Q. And what do you understand to be the cumulative dose that's examined and discussed in that study, in that report?**

A. There is no cumulative dose because we don't know that benzene has anything to do with the cause of acute leukemia in smokers. We know that smokers are generally thought -- heavy smokers get about 3 to 5 part-per-million cumulative years, but that's too low to cause AML. So the general thinking in literature is that there's some other cause for the AML other than the benzene.

**Q. So you disagree with the surgeon general's report that 15 parts per billion as a cumulative dose of benzene exposure --**

MR. CHILTON: Object to the form.

**Q. -- is enough to cause AML in 50 percent of all smokers?**

A. If that's what he said, I would disagree with that. I thought that the surgeon general report simply

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says that a 20-pack-a-year smoker has essentially a doubling of the risk. I wasn't aware that the surgeon general report specifies the specific cause, other than the fact that there's a risk with heavy smoking.

**Q. I want to talk about latency a little later. But while we're still on the subject, if you had a patient who presented to your office with benzene exposure, chromosomal abnormalities, and an MDS period consistent with secondary AML, and that person handed you a report that showed he was exposed to 100 PPM years as a cumulative dose of benzene, would you conclude that benzene could then be a causative factor in that man's AML?**

A. He has a period of myelodysplasia; he has AML. It would depend on what chromosome abnormalities he has. There are certain ones that are very typical for de novo leukemia and others that are more classical for chemical-induced leukemia. So, for example, if a patient you're talking about -- if they had an AML, they had a period of myelodysplasia, they had a five or seven chromosome abnormality, and they had considerable exposure to benzene, I would have to say that could be a consideration, yes.

**Q. And what would you consider to be, quote, unquote, considerable exposure?**

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A. Well, you said above 100 part per million. I would say if the latency were within reason, one still can't be 100 percent certain because in no case of leukemia can we specifically say this caused it or that caused it. We only make an estimate of that by looking at all of these features, and we come up with what's a reasonable conclusion based on frequencies.

**Q. So you would need to consider all available information, consider all factors, consider the exposure to benzene, consider other things like radiation, chemotherapy, genetic concerns, before you could draw a conclusion regarding causation of a secondary AML patient?**

A. All those things are important. For example, in the case of chromosomes, the what we call balance translocations, where a piece of one material moves from one chromosome to another and there's a reciprocal movement back and forth -- a classical one would be the 8 to 21 translocation -- we know that if we took 100 of those patients with that translocation, three would be secondary and 97 percent would be de novo. So that's one that you would very seldom see after a chemical. Could, but it's very seldom seen.

**Q. Is there a safe level of benzene exposure?**

A. Same from what?

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**Q. Safe from any adverse health effects?**

A. Allegedly, you can smell it at 4 parts per million. So anything -- and that's somewhat variable, but low doses might -- give an odor. We think acute leukemia, as I've said, is very high dose levels. We think aplastic anemia is probably even higher than acute leukemia in terms of dose levels. So I don't know -- I mean, it would have to be safe from what? If we said acute leukemia, you're probably talking about levels, as we said, in the hundreds of part-per-million years.

**Q. Do you consider benzene to be a threshold carcinogen?**

A. I believe that all chemicals are threshold carcinogens. So yes, I certainly do.

**Q. What is polycythemia vera, or PV?**

A. Polycythemia vera is a -- it's a relatively uncommon illness that occasionally is genetic but mostly acquired: The mean ages of onset is in the 60s. In general, we know of no cause. And it's an illness in which the bone marrow becomes autonomous from its usual controls and the bone marrow begins to overproduce normal-appearing blood cells and seems to break away from the normal controls that prevent those levels from getting high:

In recent years, it's been shown that many

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of these people have a peculiar mutation called the JAK, dash, 2, or JAK-2 mutation. And that is common to a number of illnesses, not just polycythemia vera; but most people with polycythemia vera have that. Other illnesses also have that and don't have polycythemia vera. So it's an association. But it's something that's being studied these days, and we often may use it as a diagnostic test today if we think a person has polycythemia vera.

**Q. What is the significance of JAK-2 -- I can't remember the rest of it.**

A. Well, none today. In other words, if I knew -- let's suppose you came and saw me and you had polycythemia vera and I measured your JAK2 and it was positive, I'd say that's interesting. If I measured your JAK2 and you said -- and it was negative, I would say that's interesting. And it has no, let's say, therapeutic significance right at the moment; but it's an observation that's being studied. And you see more and more publications on it, and it's a test we often employ. There are many other ways to make the diagnosis of polycythemia vera, but that's sort of the newest high tech way, you might say, to help that diagnosis.

**Q. Besides helping the diagnosis, is there anything of that JAK2 test which would indicate a cause**

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**of PV?**

A. No.

**Q. Okay. If I understand your testimony, there are two types of PV. There's genetic acquired. Correct?**

A. Yes.

**Q. And under the acquired category of PV, there's generally understood to be no known cause of PV. Is that accurate?**

A. That is correct.

**Q. Can you cite us to any literature which would give any clue to what could possibly cause PV?**

A. Well, some people in the literature suggests that there are genetic influences, which we mentioned. It seems to be more common in some populations than other populations, but the causation is obscure at this time.

**Q. PV is myeloproliferative disorder. Correct?**

A. Yes.

**Q. And what does that mean?**

A. Well, myeloproliferative -- myelo refers to bone; proliferative means growing. And so what it means is that we see a bone marrow that has too many cells in it. It's overly cellular. We sometimes call that hypercellular, H-Y-P-E-R, cellular. So if you look at a

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bone marrow report on someone with polycythemia vera, they'll say hypercellular marrow, overly proliferative marrow, pan-myelosis. All of those terms mean that there's too much bone marrow in there.

**Q. And there are other myeloproliferative disorders. Correct?**

A. Yes.

**Q. And what are those disorders?**

A. Well, initially, this was a concept promoted by Dr. Dameshek, who is a prominent hematologist and was the founder of the journal "Blood," which is the standard journal we use. He listed initially polycythemia vera, essential thrombocytosis; and some of those people may go on to develop polycythemia vera. Chronic myeloid leukemia, or CML, and idiopathic, or agnogenic myeloid metaplasia, sometimes called AMM.

And those four illnesses he put into the same category because they had clinical features that were very, very similar. And it was known that all of those had a propensity to go into AML but at vastly different rates.

**Q. So PV can develop into AML with or without therapy. Correct?**

A. Correct.

**Q. Thrombocytosis can develop into AML with or**

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**without therapy. Correct?**

A. Correct.

**Q. CML can develop into AML with or without therapy. Correct?**

A. Correct.

**Q. AMM can develop into AML with or without therapy. Correct?**

A. Correct.

**Q. And MDS, of course, can develop into AML with or without therapy. Correct?**

A. Correct.

**Q. Do you know what the incident rate of PV is?**

A. Well, the incident rate is generally given around -- I think it's about -- and I think some of the articles speak to that -- about 2.3, 2.5 cases per 100,000 people.

**Q. And what about the incident rate of AML?**

A. That's somewhat age-related. In other words, a person who's in their 70s has a rate of about 150 per million population. Someone who's, let's say, 30 might have four or five per million. The rate goes up astronomically with age. So it depends on the age of the individual. Overall, it's thought that we see about perhaps 12,000 AMLs in the United States each year.

**Q. Per 100,000?**

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A. No. Total cases of AMLs each year in the United States.
**Q. I'm sorry. That would be a lot.**
A. That would be a lot, yeah.
**Q. That would be horrible?**
A. That would be horrible, yeah. AML is -- it's not rare. And that's a good thing. It's more frequent than we like to see.
**Q. You did not review Walter Mielke's blood in this case. Correct?**
A. Correct.
**Q. You didn't review any slides. Correct?**
A. Correct.
**Q. You did not review his bone marrow. Correct?**
A. Correct.
**Q. Did you perform any tests on his tissue or blood?**
A. No.
**Q. Did you perform any calculations regarding his illness, such as incident rates, considering different factors specific to him? For instance, age, some of the PV treatment, other factors? Did you perform any type of mathematical calculation?**
A. Well, I looked at those features. I mean, there are articles that deal with numbers about

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frequency of AML development relative to no treatment or treatment with phlebotomy alone versus treatment with P-32 versus treatment with P-32 plus chemotherapy, as he had. There are some general findings of that. There's been a so-called polycythemia vera study group in force and effect for many years, and periodically you see reports on the outcome of some of those patients.
**Q. I guess what I'm asking is a more specific question. Did you perform any math specific to Walter Mielke, in light of some of the other information available to you and some of the stuff you just referenced?**
A. No, no mathematical calculation.
**Q. And if you testify at trial, would you -- or do you currently plan on performing any such calculations?**
A. No.
(Exhibits 1 and 2 marked.)
**Q. Do you have any notes, handwritten notes or typewritten notes, about your opinions in this case, other than your report, which I marked as Exhibit 2?**
A. No.
**Q. You have no notes. Correct?**
A. Other than, let's say, tabs that I've put on some of the pages.

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**Q. We'll go through some of those. Did Walter Mielke have any genetic or hereditary factors that may have caused his PV?**
A. None that we're aware of.
**Q. As we sit here today, do you have any opinion within reasonable medical probability as to what was the cause of Walter Mielke's PV?**
A. No.
**Q. In your opinion, is benzene exposure a possible cause of PV?**
A. No.
**Q. Is benzene exposure a possible cause of AMM?**
A. Well, that's a question. There are anecdotal reports that support that. There are no epidemiologic reports that suggest that. I don't know the answer to that question except to say that we have no definitive evidence that benzene can cause AMM.
**Q. Is benzene a possible cause of CML?**
A. No.
**Q. Why not?**
A. Why not? Because of the fact that if we look at the three -- there are three large studies that have to do with heavy exposure to benzene -- probably higher than we would ever see in the United States -- and hematologic diseases -- and none of them have shown a

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statistically increased incidence of CML.
**Q. What kind of incidence would you need to see for it to be statistically significantly, in your opinion?**
A. Well, I would like to see, A, that the diagnosis was established, and by proper diagnostic techniques; and that one saw consistently at least the doubling of the risk.
**Q. Would you need a tripling of the risk?**
A. Well, the higher the number, the better off one would be. But what you're looking for is at least a doubling of the risk and consistency among more than one study.
**Q. Can you can exclude benzene as a potential cause of Walter Mielke's PV?**
A. Yes, I think so.
**Q. On what basis?**
A. Well, because of the fact that the hallmark benzene's effect on the bone marrow is to reduce the cellularity. In other words, many studies have looked at chronically exposed patients to benzene, and their marrows show less than normal cellularity. These bone marrows are chocked full of cells, so it's exactly the opposite of what you would expect to see in some chronically exposed to benzene.

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Plus in these cohorts I've described, they don't find any evidence of polycythemia vera among these heavily exposed benzene patients. So I would have really no reason to think it's not the right type of bone marrow, and there's no epidemiologic background that suggests to me that polycythemia vera can be caused by benzene.

**Q. The cohorts that you referenced, do those -- did those cohorts' studies look for PV as a cause of AML?**

A. Yes, because in the case of Aksoy, for example, in one of the articles I have in here, they are looking at hematologic changes. In other words -- and a change could be up or down. They didn't say, Okay. We're only going to look at people who get anemic. They said, We're going to look at what the blood shows in people who have had benzene exposure.

**Q. Do you believe there to be any -- strike that. To your knowledge, is there any peer-reviewed scientific literature which supports benzene as a cause of increased cellularity?**

A. Not that I'm familiar with.

**Q. So you're not familiar with any studies -- peer-reviewed studies which conclude or relate benzene exposure with an increase in cellularity?**

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A. Well, certainly when a person develops acute leukemia, whatever the cause, at that point they have increased cellularity. But if you're talking, let's say, a chronic illness that, you know, is not acute leukemia, something like a myeloproliferative disease, no, I don't know of any evidence that that's caused by benzene.

**Q. When did Walter Mielke -- when was he diagnosed with PV?**

A. If I can look at some of my notes . . .

**Q. Please.**

A. Around August of 1992 he was found to have abnormal blood counts.

**Q. And what treatment did he receive for his PV?**

A. He initially received phlebotomy therapy.

**Q. And what is that?**

A. That means you take blood out of a person and throw it away.

**Q. Is that a reasonable and accepted method of treatment for PV?**

A. Yes.

**Q. What other treatment did he receive?**

A. Shortly thereafter, he received a radioactive phosphorus.

**Q. And can we refer to that through the rest of your deposition as P-32 treatment or just P-32?**

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A. Yes.

**Q. Have you ever had occasion to treat a patient with PV with P-32?**

A. Yes.

**Q. Has that been the case during all your 30 or more years -- I guess it's about 35 years, to give you full credit. Has that been the case throughout your 35 years of medical practice?**

A. No.

**Q. What is the change there? What is the difference?**

A. Well, I gave P-32 in around 1969 and probably early in 1970 because in those years I was training to be a hematologist. And if one wanted to give P-32 at that time, you had to demonstrate to the authorities that you had given it to somebody. It's sort of a circular argument. But my professor, who's now deceased, liked P-32; and so when I was in my training, I would give it so I could have a resume that showed I gave X number of people P-32. That would get me licensed to give it to others when I was practicing on my own.

However, I never really saw the need to give P-32 to most of these polycythemia vera patients.

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And so I chose not to continue to pursue that. And so I would say the last time I gave P-32 was probably around 1970.

**Q. Is P-32 treatment still a common treatment today?**

A. It's very uncommon in the United States.

**Q. Why is that?**

A. Well, it sort of went off the radar screen around 1981. Prior to 1981, it was known that it had the potential to cause acute leukemia in people with polycythemia vera. And then in 1981, the polycythemia vera study group published in the New England Journal an article, of which I have that here. They showed that the incidence was higher than most people had suspected.

And after that, most hematologists got cold feet about giving it in the United States; and its use dropped considerably. Then subsequent to that, it became difficult to get because the manufacturing of it dried up, as some of these other articles testify to, and the regulations to give it became more stringent. And so people like myself, practicing hematologists, couldn't give it any longer. And so in the United States it hasn't been used much since the 1980s.

**Q. In the course of your clinical practice, have you had occasion since 1981 to see any patients who have**

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**received P-32 as a treatment of their PV?**
A. No.
**Q. Did it surprise you to see that Walter Mielke received P-32 as a form of treatment for his PV?**
MR. CHILTON: Object to the form.
A. Very much so.
**Q. Some of these studies I might mark as individual exhibits; others I might not. I'll mark this as Exhibit 6. And can you identify this document, please?**
**(Exhibit 6 marked.)**
A. Yes. This is an article entitled, Increased Incidence of Acute Leukemia In Polycythemia Vera Associated Chlorambucil Therapy.
THE WITNESS: C-H-L-O-R-A-M-B-U-C-I-L.
A. It's published in February of 1981.
**Q. Is that the article you referenced a few minutes ago?**
A. Yes.
**Q. What type of therapy is administered or is the subject of that article?**
A. They had three limbs. One group of patients were treated by phlebotomy alone, simply taking blood out of them, and no other medical treatment. The second limb was a tablet called chlorambucil, or Leukeran is

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the brand name of that; and they used that to control the bone marrow proliferation. Leukeran poisons the bone marrow, if you will. And then the third limb was radioactive phosphorus.
**Q. P-32?**
A. P-32.
**Q. And that study examines of effect of those types of treatment as a causative factor of AML. Is that fair?**
A. Yes. Now, this was a relatively short-term study. It had only gone on for six and a half years. And of course, these patients are continuing to be followed. But the results were so striking, even at six and a half years, that this drug became essentially obsolete in the United States.
**Q. "This drug" being P-32?**
A. Yes. Leukeran, as well, I might say. Both those treatments.
**Q. How many patients were followed in that study?**
A. They started out with 431 previously untreated patients.
**Q. 131?**
A. 431.
**Q. How many of those 431 patients had occupational exposure to chemicals?**

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A. I have no idea.
**Q. Do you know how many of those patients were tire builders?**
A. I have no idea.
**Q. Any idea how many were rubber workers?**
A. No idea.
**Q. Any idea what part of the country those patients came from?**
A. Well, it tells you; this is a group that -- some of them didn't even come from this country; in other words, they came from the United States, France, Israel and Sweden.
**Q. U S, France, Israel, and Sweden?**
A. Yes.
**Q. How old are the patients? Is there a range or a median?**
A. I don't see age referred to in this particular article.
**Q. Is the word "benzene" mentioned anywhere in there?**
A. Not that I can see.
**Q. Are other causative factors of AML, besides phlebotomy, chlorambucil, and P-32, examined in that article?**
A. Now, say that again question.

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**Q. Are other causative factors of AML considered in that article, besides PV treatment with phlebotomy, chlorambucil, or P-32?**
A. Well, phlebotomy, we don't believe, causes AML. In other words, in phlebotomy patients -- there's no evidence from phlebotomizing people for other reasons -- we don't think phlebotomy causes anybody to have AML. So the idea is the phlebotomy alone group is looking at the natural history of leukemia in the polycythemia vera patient. The other two limbs suggest that those treatments increase the incidence of AML.
**Q. Does that study consider smoking as a factor in AML?**
A. No.
**Q. Is smoking a cause of AML?**
A. I think so, yes.
**Q. There are genetic factors which could cause AML. Correct?**
A. Yes.
**Q. Are genetic factors dealt with in that article?**
A. No.
**Q. Have you ever spoken to any of these authors about this article?**
A. Let me look at the list again. Many of these

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people are probably dead.
**Q. You probably do know a couple in here.**
A. Murray Silverstein is the third author that -- I knew him.
**Q. Let me ask a better question. Have you had any professional experience with any of the authors of this article specific to the subjects in this article?**
A. No.
**Q. Is this the strongest piece of literature discussing the relationship between P-32 as a cause of AML?**
A. No. This is an early study, as I pointed out. We have many follow-up articles, some of which I have here.
**Q. Okay. What would be the next article that you would rely on as demonstrating that P-32 causes AML? Do you refer to this one as the Berk study?**
A. Could be. I think he's the lead author.
**Q. The Berk 1981 --**
A. Yes.
**Q. -- study?**
A. This particular article that I have here is from 1996, so it's considerably more recent than that article; and it's continuing to look at these patients --

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**Q. Same patients?**
A. Same patients. It's in this PVSG protocol, which is this group's protocol. And they have 93 patients that they're looking at now who received P-32 alone as their treatment. And they give a graph. And I've highlighted the graph. And they have this broken out, and this is the -- these are the people treated only with P-32. And you can see what their incidence rate is of AML.
**Q. And what is that incidence rate?**
A. Well, it's close to 40 percent at 20 years.
**Q. And this is the Najean 1996 study. Correct?**
A. Yes.
**Q. And I marked this as Exhibit 7.**
**(Exhibit 7 marked.)**
**Q. Does this Najean 1996 study examine occupational history of the individuals followed?**
A. No.
**Q. Does it consider possible exposure to chemicals as a causative factor of AML?**
A. Only chemicals that deal with the treatment of polycythemia vera, chemotherapy chemicals.
**Q. Does not consider benzene. Correct?**
A. No.
**Q. Does it consider smoking?**

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A. No, I don't believe.
**Q. Does it consider genetic factors as possible causative factors of AML, besides P-32 treatment?**
A. No.
**Q. And these are 93 of the 431 people --**
A. I believe that's correct.
**Q. Just to finish my question for the record. Of the 431 people examined in the Berk 1981 study. Correct?**
A. Yes.
**Q. This Najean 1996 study deals with hydroxyurea also. Correct?**
A. Yes.
**Q. I guess I want to go through that Exhibit 7 in a little more detail. For instance, when we talked about the Berk study, we talked about phlebotomy, chlorambucil, P-32.**
**What are the agents that are explored in the Najean study?**
A. Well, hydroxyurea is also used in a compound called pipobroman that I've never used, and it was never widely used in the United States but apparently used in Europe.
**Q. What else?**
A. Those two are the two drugs that are mentioned

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in there.
**Q. Okay. So the Najean 1996 study does not examine P-32 as a possible cause of AML. Correct?**
A. It does. It's got people treated with P-32 alone.
**Q. And also has people with treated with hydroxyurea?**
A. Other limbs of the study -- yes -- received hydroxyurea, and in particular, people receiving both hydroxyurea and P-32. He says in his -- in his abstract here, The patients receiving P-32 and hydroxyurea as maintenance had an excess risk 29 percent at the tenth year. And others have commented that the combination of P-32 and hydroxyurea is much more leukemogenic than hydroxyurea alone.
**Q. As a medical professional, can you sit here today and conclude that each of these 93 individuals examined in the Najean study -- that each of their AML was caused by hydroxyurea or P-32 or a combination of both?**
A. Not in every case, because people who have -- we know that acute leukemia occurs in the general population at certain rates. And because a person had a grocery store or was an attorney, had P vera, that doesn't obviate their risk of catching acute leukemia

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because they get very old. In other words, there are certain statistical risks of AML based on age.

So when you do a study, you might -- if you were talking about the relative risks, you might want to factor in the baseline risk of acute leukemia in the general population. But I would have to say that when you're talking about rates like this, it's obvious that these risks are way in excess of what we see in the general population.

**Q. This shows us -- this Najean 1996 study shows us an increased risk, but it does not give us a definitive cause of each individual's AML. Is that accurate?**

A. No. I think it gives us a cause. In other words, it says that you've got a baseline rate of polycythemia vera going into AML of, let's say, arguably, one and a half percent; and now you've got maybe even up to 40 percent after 20 years in some of these people. And the only thing you did differently to them is to give them P-32. That tells me P-32 is bad; it gives people acute leukemia.

**Q. Well, doesn't benzene cause AML?**

A. Under certain conditions.

**Q. What if each of those 93 or so individuals examined in that study was exposed to 200 PPM years of**

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**benzene? Would that change your opinions?**

A. Well, it might, but this is a randomized study. In other words, you see they started out with a group of the 431 people. So that whatever risk factors there might have been sort of were -- whether smoking or where they worked, and all of those things, age, to some extent, those are was -- it would be hard to see how they were all in one pile. In other words, they should have been as a randomized trial, evenly distributed.

**Q. If we picked one person out of those 90 or so individuals examined in the Najean study from 1996 -- if we picked one person, you would need to evaluate their occupational exposure, their genetic factors, their smoking history, their use of P-32 and hydroxyurea, other possible causative factors, to reach a conclusion, within a reasonable degree of medical probability, that a given agent, be it the hydroxyurea or P-32 or a combination of the both, caused the AML. Correct?**

A. In general, whenever you see a person with AML, you take into account all of the history.

**Q. You would need to conduct a differential diagnosis to reach that conclusion that I just laid out for you?**

A. Yes.

**Q. That's a fair statement?**

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A. Yes.

**Q. What is a differential diagnosis?**

A. Well, in a differential diagnosis, you have a particular finding; and that finding could be anemia, it could be pneumonia. And you're looking to see what are the -- what is the -- what is the illness, what is the cause of it, and more specifically, what is the exact illness.

**Q. Did you perform a differential diagnosis as it concerns your opinions in this case of the cause of Walter Mielke's AML?**

A. Yes.

**Q. Can you walk us through your differential diagnosis in this case?**

A. Well, to me, I see that a person had AML. And in a way, just the type of AML was important because the type of AML this person had is very unusual, a so-called granulocytic sarcoma. And you'd say, well, who has granulocytic sarcomas? Because of the next hundred people with AML I might see, maybe none would have granulocytic sarcoma. It's very rare. And that's sometimes called a extramedullary myeloid tumor, extramedullary meaning outside the bone marrow. In other words, this is a leukemia -- normally, when we see acute leukemia, the bad cells, the blast cells, are

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inside the bone marrow or outside out circulating in the blood.

In this case, the bad cells are actually in the tissues forming lumps or masses, like you might see in a patient with lymphoma. And so it's unusual. It's referred to as extramedullary proliferation.

And this article is just on that subject. And it says, by definition, they arise outside the bone marrow but usually develop during the course of systemic myeloproliferative diseases. In other words, a granulocytic sarcoma is just the kind of leukemia you might see in someone with polycythemia vera. It would be a very rare, not unheard of, leukemia to see in someone who didn't have myeloproliferative disease.

So the type of leukemia, right off the bat, perks my ears up because it's an uncommon form of leukemia.

**Q. We were going through your differential diagnosis. Is that it?**

A. That's started out. So whenever you're looking at a differential diagnosis of acute leukemia, there are certain aspects of it you look at. First, the type of leukemia if there were specific chromosome abnormalities, which we don't have here, and the character of the cell -- there are different types of

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acute leukemia. So we say, Okay. First looking at the type of acute leukemia, this is an AML. It's an AML that's associated with extramedullary proliferation of cells.

Then we say what is the background of the patient. Well, it's a background of polycythemia vera. And we know that polycythemia vera by itself gives an increased incidence of AML. And so we then look with some detail at the treatment.

And here's a man who had his treatment of his AML radioactive phosphorus. And he just didn't have a little bit of radioactive phosphorus; he had a lot. If you look at some of the articles on how much you give -- and that's detailed in some of these articles -- typically, we're talking about giving one or two injections. There are some of the articles here that detail that. This man had, by my calculations, at least seven injections. So he didn't have the amount of P-32 that many of these patients had; he had much more P-32 than they had.

So I would say if one accepts P-32 as leukemogenic, he had an extra shot of it. So that is another thing I put in my differential diagnosis.

Then I see, Well, what else did he have besides radioactive phosphorus P-32? Well, he had a

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alkylating agent, and the alkylating agent was called myleran. And we know myleran is a drug that's rarely use anymore. It was used for chronic myeloid leukemia. And in the years that I used it, I would typically give someone two or four milligrams a day. In his case, he got ten milligrams a day. So he didn't just get a little bit of alkylating agent; he got a lot of alkylating agent. And we know alkylating agents cause AML, whether it's in people with polycythemia vera or people without polycythemia vera.

Then he also got hydroxyurea. And as many of these articles will tell us, the -- as this one says, hydroxyurea followed by P-32 is clearly not to be used since it is associated with an unusually high incidence of acute leukemia.

Well, he got hydroxyurea, and he didn't get trivial doses of that drug either. So here's a man who's got an illness that predisposes to just the kind of leukemia that he has, and he's been given high doses of three agents, all of which, on their own, could be leukemogenic; and he's had all three of them together.

In addition to that, he has had radiation therapy to his prostate. Now, if you look at people who have radiation therapy to their prostate alone as just a single therapy, particularly if they've had it by what's

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called IMRT, which is a very confined way of giving radiation therapy, it's thought that the incidence of acute leukemia is not increased. But here again, we're adding this radiation to other radiation that he has. So I think its addition may have played a role here.

And so when I look at that -- those things, it tells me that, far and away, the most likely cause of his leukemia was -- was the basic illness and its treatment. And I can't tell you what percent each element gave in terms of risk. Although I would think probably P-32 is highest on the list, any of those elements could have caused it.

Now, continuing the differential diagnosis, because this case has to do with benzene, I looked at that. And of course, I, myself, certainly would have no reason to dispute that he was exposed to benzene in the form of solvents. I saw your expert's -- I would have no way of estimating what he was exposed to myself, but your expert says 33 parts per million. Well, that generally is lower than most estimates of what it takes to cause AML. So he doesn't have, by your expert, enormously high exposure.

I look at the window of opportunity to catch AML from chemicals; and it's remarkably consistent, including benzene, that there's a window of

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about 2 to 10 or 12 years from the exposure during which the secondary leukemia may evolve. Well, he's far away from that window of exposure. So he doesn't have the right dose. He's got the wrong window of exposure.

So to me, it isn't really a consideration because the amount of therapy he had for the polycythemia vera dwarfs any other potential cause of the illness.

**Q. All right. Let's take a short break.**

**(Break taken.)**

**(Exhibit 8 marked.)**

**Q. Dr. Natelson, I've marked as Exhibit 8 the Nappi, N-A-P-P-I, study that you reference. It has a lot of big words, so I'll let you pronounce them. Can you tell me again the title of this article?**

A. The title is Extramedullary Hematopoietic Proliferations, Extraosseous Plasmacytomas, and Ectopic Splenic Implants (Splenosis).

**Q. What is the significance of that study as it relates to your opinions in this case?**

A. Well, it is this: It feeds into some of these other articles. For example, if you look at the one article we already went over -- and that was the one that showed a graph of the frequency. This one over here -- you'll notice something that, at first sight,

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when the author talks about these patients in here, he's lumped in a few patients that had lymphoma. And you'd say to yourself, Well, why in the world has he done that? And that's explained in this article and other articles I have, is that there's another statement in here; he says, When this tumor develops, it's been confused with non-Hodgkins lymphoma. And otherwise -- again, he says, otherwise, the gross appearance, meaning how does it look, of extramedullary tumor is indistinguishable from that of non-Hodgkins lymphoma. So many patients who develop this proliferation of tumor cells outside the bone marrow in a case of leukemia, it's confused with lymphoma. And it was in Mr. Mielke's case, too, because it looked -- that was their first thought, that he had lymphoma.

And so I don't know how well those, quote, lymphoma cases were studied; but I think this author linked them all in because they likely were part and parcel of the same process. And that's why he put them into that group. I think there may have been four or so people in here that he thought had lymphoma or classified as lymphoma.

**Q. And the article you keep referring back to --**

A. That's this Najean's article from 1996. So I think these articles are complementary to each other in

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terms of what did Mr. Mielke had and the unusual type of leukemia.

**Q. So Exhibit 8, the Nappi study from 2003, essentially just examines -- talks about the type of extramedullary myeloid tumor that Walter Mielke experienced?**

A. Yes. It describes several different types of myeloid tumors, but one of them is the type that he had.

**Q. Does it talk about the cause of those tumors?**

A. No -- well, excuse me. I would say only in the fact that it said by cause he says they're more frequent in people with myeloproliferative diseases, but he doesn't discuss the cause of the myeloproliferative disease. So I guess it depends on how far you want to go trace the root of the cause.

**Q. So maybe it's just the big words in the title that confuse me, but I'm trying to get to the heart of the relevance of this study. And as I understand you, the Nappi 2003 study is significant, in your opinion, because it describes the type of tumor and the relationship of the type of tumors consistent with what Walter Mielke experienced in the context of PV. Correct?**

A. Yes.

**Q. Okay. Does it discuss treatment with P-32,**

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**the Nappi study?**

A. No.

**Q. Does the Nappi study discuss causation of AML as it's related to extramedullary myeloid tumors or PV or P-32?**

A. No.

**Q. Does the Nappi 2003 study discuss benzene at all?**

A. No.

**Q. Does it discuss occupational exposure?**

A. No.

**Q. Does it cover a certain category of -- does it study a specific group of people?**

A. Well, it's a specific group of people with these illnesses, but not as a cohort.

**Q. So the Nappi 2003 study tells me more about the type of tumors that Walter Mielke experienced, but it doesn't speak to issues of causation. Is that accurate?**

A. Correct.

**Q. Okay. What's the next study you have there?**

A. This is.

(Exhibit 9 marked.)

**Q. Wait, let me cut you off there. Sorry. I had another one that I had marked as Exhibit 9, and this is**

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**the Nathaniel Berlin 2000 article. Correct?**

A. Yes.

**Q. Let me ask a question just so we have it clear. What is the significance of the Berlin 2000 study as it relates to your opinions in this case?**

A. Well, there are several aspects of it. It's an article that describes in a little bit more detail how a person is treated with P-32 and a little bit of why its use has declined in the use of polycythemia vera. And it mentions the half-life of the drug. It mentions how it's delivered, the doses, the federal government regulations that are now in force and effect. And it discusses some early treatment of polycythemia vera with P-32 back as far as 1939.

It discusses even the use of arsonic to treat P-32. Talks about the response that people get to P-32 therapy. It points out that in 1948 was the first report that P-32 might cause AML. And it tells us about the fact that P-32 use, at least at the time this article was written, which was year 2000, is very unusual. And it also tells us that, as I think I quoted before, its use with hydroxyurea is followed by a high incidence of acute myeloid leukemia.

**Q. Can I see that article, please? As you just described -- and I don't mean to summarize your**

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**testimony, but this -- but I guess I will anyway, but this Berlin articles gives us a summary of P-32, its histories, its uses, its administration, et cetera. Correct?**
A. Yes.
**Q. And in the course of that, this Berlin 2000 article also speaks to the role of P-32 as a cause of AML. Correct?**
A. Yes.
**Q. And that little piece about causation is spoken to here on page 4 in the section you highlighted. Correct?**
A. Yes.
**Q. And that references a footnote, which is footnote 69, which appears to be the Murphy study from 1997. Is that accurate?**
A. Yes.
**Q. Have you reviewed the Murphy study? Is that in your materials also?**
A. It isn't in my materials. I have seen that many years ago. I'm not sure I have that paper in my files.
**Q. Does this article talk about -- or does it follow a group of patients who were treated with P-32 and examine how the relationship with P-32 as a**

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**causative factor of AML -- how that relationship might be explained?**
A. Well, in general terms. In other words, it is a review article. It's not a -- he comments on others' findings. But it's simply meant as a review article.
**Q. Okay. In that Murphy study from 1997 that's examined in footnote 69, which we don't have in front of us, in all fairness, do you know if that -- do you know how many people that study follows?**
A. No. It's been some time since I saw that article.
**Q. And you don't know if any of those people were exposed to benzene?**
A. No.
**Q. What does the Berlin 2000 article have to say about hydroxyurea as a cause of AML?**
A. Well, it simply points out that it's -- the author believes it causes AML at a low incidence rate, but when it's combined with these other agents, it's more than just additive. In other words, it seems to potentiate the leukemogenic properties.
**Q. And that conclusion is consistent with that in the Berk 1981 New England Journal of Medicine study and the Najean. Correct?**
A. Yes. This is a newer article -- this is

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2005 -- by Finazzi, who's written a number of articles on this subject called Risk-Adapted therapy in essential thrombocythemia and polycythemia vera. And he also discusses the use of hydroxyurea and says the use of hydroxyurea in patients already treating with alkylating agents or radiophosphorus also enhanced the leukemic risk, and he says hazard ratio 7.58 with a significant confidence limit. So he says the same thing that the other one says, is that hydroxyurea may have a low rate of leukemogenesis, but that when you combine it with these other treatments it becomes very significant.
**Q. May I see that, please?**
A. Yeah.
**Q. I will mark this the Finazzi 2005 study as Exhibit 10. How many patients are followed in this study?**
**(Exhibit 10 marked.)**
A. I'd have to look at that and see what he says in there. I don't know.
**Q. Can we do that, please?**
A. This is from, actually, a different study than the Berk study, and it's one they call ECLAP, E-C-L-A-P, or European Collaboration Study. And it talks about 1,638 patients from 12 European countries, and it talks about their follow-up.

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**Q. And each of those patients received hydroxyurea and/or P-32 treatment for their PV?**
A. I don't know that. In other words, in the body of the article, he may give some numbers. He says the 1638 patients prospectively enrolled in the ECLAP study with a median diseased ratio of 6.3 years represents a particularly appropriate population.
**Q. So did these 1600 or so patients have PV?**
A. They all had PV. And he tells us that if they got hydroxyurea alone, he did not think that they enhanced the risk of leukemia over and above the basic incidence with polycythemia vera, but that if you add them to people treated with alkylating agents or radioactive phosphorus, it did.
**Q. How much did it increase the risk?**
A. Well, he says, again, the use of hydroxyurea in patients already treated with alkylating agents or radioactive phosphorus enhance the leukemic risk as a ratio of 7.58.
**Q. Of the 1638 patients examined in that study, how many went on to develop AML?**
A. I don't know the answer to that.
**Q. Is that a function of the math of the --**
A. I don't think that we have those specific numbers in here. He simply also -- or he doesn't put

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the actual numbers in this article.
**Q. What kind of occupational history did the 1638 patients in that article have?**
A. That is not mentioned.
**Q. Is it mentioned whether or not any of the individuals in that article smoked?**
A. No.
**Q. Is it mentioned whether or not any of them were occupationally exposed to benzene?**
A. No.
**Q. Does it make any reference to benzene?**
A. No.
**Q. Does it make any reference to genetic factors as genetic considerations as also being possible causative factors of a given person's AML?**
A. No.
**Q. Does that article have anything to do with benzene exposure?**
A. No.
**Q. And again, what is the significance of this article in your opinion in this case?**
A. My objective is to tell the jury what the peer-reviewed medical literature says about the subject -- that's what this is -- and that there's a uniform opinion that there's particular incidence of AML

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with this illness and that that incidence is augmented by the use of these other drugs.
**Q. All right. Let me see that, please.**
A. This one is another general review article.
**Q. I'll mark this as Exhibit 11. This would be the Michiels -- M-I-C-H-I-E-L-S -- Netherlands Journal of Medicine article from 1999, and --**
MR. PATTON: Off the record.
(Discussion off the record.)
(Exhibit 11 marked.)
**Q. Dr. Natelson, the Michiels study, Exhibit 11, from 1999 -- I think I went right to the page regarding causation, but can you tell us again the significance of that study as it bears on your opinions in this case, besides just being a review of P-32?**
A. It's a review of P-32 and also hydroxyurea. And it, again, gives the same opinion that hydroxyurea is associated with, they say here, a 10 percent incidence of acute leukemia after long-term follow-up of about ten years.
**Q. How many people were followed in that study?**
A. Well, I'm not sure if he's talking about the same study; but in the next paragraph he says, A recent review of more than 1200 patients who had polycythemia vera indicated that the death rate in those receiving

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P-32, alkylating or non- alkylating myelosuppressive agents was three to four times higher than those who did not.
**Q. May I see that?**
A. (Witness complied.)
**Q. And the part that you just read is associated with footnote 46, which appears to be a Gruppo Italiano Studio Policitemia, Polycythemia vera: The natural history of 1213 patients, 1995.**
**Did I accurately recite that from footnote 46?**
A. Yes.
**Q. Did you review that article in coming up with your opinions in this case?**
A. No.
**Q. How did you come to draw upon the articles that we've talked about and that we're continuing to talk about in forming your opinions? How did you select the articles?**
A. Well, some of them I had in my file. For example, the original one by Berk, and maybe a few others, were in my file. And then I looked up on the PubMed to try to get recent information about P-32 to see if I could find any articles that commented on its use in recent years. Because, as I said earlier, I was

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very surprised that it was used in this case. And whereas I was familiar with it, I wasn't familiar with the fact that it was continuing to be used. And I wanted to see if that was the case, so I got some newer reviews on the subject.
**Q. Why didn't you review the Gruppo Italiano study from 1995?**
A. Well, no particular reason except that you could chase your tail trying to review every source material that an author looks at. When I read a review article, I accept that the review has been fair. And they're in peer-reviewed journals, so I assume he's not quoting too badly the source materials.
**Q. Do you know the occupations or the potential other causative factors that might be in place or were in place for the individuals studied, the 1200 or so people studied in that Gruppo Italiano 1995 study?**
A. No.
**Q. Do you know if any of those subjects were exposed to benzene?**
A. No.
**Q. Do you know the dose of hydroxyurea or -- scratch that. Do you know the dose of P-32 those subjects were administered?**
A. No.

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**Q. Do any of the articles we've talked about so far discuss the dose of P-32 administered to these patients in the context of P-32 as a causative factor of AML?**

A. Well, they discuss the dose of P-32 given to the patients, and then you subsequently see the results. In other words, Berlin's article deals with that. So does Berk's article deal with that, as to how much P-32 was given.

**Q. Which studies talk about that?**

A. I believe the one by Berlin, for example. And he's telling us how P-32 is used. And he says here that in the polycythemia vera group study, he's telling us that they -- by agreement, they gave what's called 5 millicuries at a shot, and no more each time. And that's in the ballpark of what Mr. Mielke got. I think sometimes he got 7.5 millicuries. But they adopted a maximum, he says, of 5 millicuries.

So they would give a first dose. He says a second dose of 2 to 3 millicuries was given in three months if the counts were still abnormal. And he says approximately half the patients who had a good response to the first dose didn't require a second dose. So in other words, a lot of these people just had a single dose. At follow-up three months after the second dose,

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an additional 30 percent no longer required further P-32 treatment. So in other words, he's telling us that by the time you got by two shots of this stuff, 80 percent of the people didn't need any more of it. That's that particular study group.

**Q. What is the next article you have there?**

A. This is an article about the use of hydroxyurea alone, and these authors felt that high hydroxyurea is leukemogenic. And what they pointed out is that they not only had what they thought was an excess of cases of AML with the use of hydroxyurea, but that those that had gotten the hydroxyurea had a very specific chromosome defect, lending credence that perhaps the hydroxyurea was the specific cause of that.

**Q. What was the chromosome defect?**

A. It's a defect in the 17th chromosome, but they call it a 17-P deletion. And they allege that these results show the treatment with hydroxyurea alone is associated with leukemic risk of approximately 3.5 percent. And these are people being treated for essential thrombocytosis. They're not polycythemia vera patients, but they are myeloproliferative-diseased patients. And they thought hydroxyurea alone caused an increase in AML. That's the basis of this article.

**Q. And this is the Sterkers study from 1998 for**

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**blood. Correct?**

A. Yes.

**Q. I'm going to mark that as Exhibit 12.**

**(Exhibit 12 marked.)**

**Q. And as you stated, these results show the treatment with hydroxyurea alone is associated with the leukemic risk of approximately 3.5 percent. Correct?**

A. Yes.

**Q. So from this study, is it fair to conclude that if 100 patients were treated with hydroxyurea it could expected that 3 1/2 of them would then develop into AML?**

A. That's correct. As a single agent, treated as a single agent.

**Q. Is that statistically significant, in your opinion?**

A. I don't know the answer to that. I would say this: These are not polycythemia vera patients. These are essential thrombocytosis. And it's generally thought that the leukemia risk is lower in people with essential thrombocytosis than it is in polycythemia vera. So that would make these results all the more significant, because that's a disease that doesn't very often go into AML.

**Q. And if I'm reciting this correct, can you tell**

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**us how many individuals are examined and what they were treated with and followed -- how long they were followed?**

A. Between 1970 and 1991, we diagnosed essential thrombocytosis in 357 patients. One or several therapeutic agents had been administered to 326 of them. That included hydroxyurea in 251 of them and only hydroxyurea in 201. So they had 201 people that had only been treated with hydroxyurea.

**Q. And then there's also a reference to Busulfan and P-32. Correct?**

A. Yes. In some of the others, they got this pipobroman, and others they got Busulfan, which is the drug that Mr. Mielke got.

**Q. What was the relationship between Busulfan and AML?**

A. I'm not sure they tell us that because they're focusing on the hydroxyurea. Let's see if I can . . . Well, he says with median duration of 98 months, 17 patients, or four and a half percent of the total, had progressed to AML or myelodysplasia.

**Q. What was that number, again?**

A. He said 17 patients had progressed to AML or myelodysplasia. 14 of these patients had received hydroxyurea as sole treatment in seven cases -- 14 of

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these patients had received hydroxyurea, had sole treatment in seven cases. And then subsequently other -- some of these people got P-32. Some got Busulfan. It's a mixture of things. And so the thrust of their paper is really having to do with hydroxyurea. And they commented, as I think we discussed before, perhaps not with Busulfan, but these authors say after 1980, to avoid the leukemogenic affects of Busulfan and P-32, patients were generally treated with hydroxyurea.

And so that's why I say I was so surprised when I saw that this patient had been treated with not only P-32 but large amounts of P-32 and also large amounts of Busulfan.

**Q. What makes Walter Mielke's dose of P-32 high?**

A. The numbers of times he got it. In other words, what the PVC people said is we're going to give a maximum of 5 millicuries when we give this dose. And that was the maximum they gave at any one injection. They said we're going to give so much per weight of the individual with a maximum of 5 millicuries, so that nobody in that study got more than 5 millicuries at any one shot.

And as we've seen, in about 80 percent of those patients, they only got a couple of shots, maybe two shots. In this case, he's given doses ranging from

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5 to 7.5 of -- it should be millicuries. I wrote microcuries. And so he's getting higher than the recommended individual dose, and he's getting more total doses. So if you looked at the total millicuries of P-32 he got, he's gotten more than most. Let's put it that way.

**Q. Exhibit 12, the Sterkers study that we just talked about, does this study cover the occupational history of the patients?**

A. No.

**Q. Does it talk about other possible causative factors for AML besides the treatment covered that you just talked about?**

A. No.

**Q. Does it factor in smoking as a cause of AML?**

A. No.

**Q. Does it factor in benzene exposure as a cause of AML?**

A. No.

**Q. Does it factor in any other chemical or other carcinogenic exposure as a factor --**

A. Only the ones they have talked about in the articles.

**Q. We only have one left, but maybe after that, I would be able to correct that last part of my question**

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**that I keep asking. Can I see that last article, please?**

A. (Witness complied.)

**Q. This is the article about JAK. Correct?**

A. Yes. It's one of many articles. It's just an editorial about it.

**Q. What is the -- I'll mark this as Exhibit 13. And this is the Kaushansky editorial. Is that fair?**

**(Exhibit 13 marked.)**

A. Yes.

**Q. Entitled, "On the molecular origins of the chronic myeloproliferative disorders: it all makes sense." Can you explain to us the relevance or significance of this paper as it bears on your opinions in this case?**

A. Well, nothing in particular as it bears on my opinions about the causation of AML. It's simply a paper to show that Dr. Dameshek put these patients in a particular category on what we call clinical grounds, meaning that their illnesses looked to have some similarity. They had a lot of cellularity of the bone marrow; it was very hypercellular. They had often what we call pan-myelosis, meaning the same thing: that there were lots of cells in the bone marrow. And he reasoned they must have something in common with these

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illnesses.

And here many years later, we find that, yes, they do have something in common; he was correct. They have a particular abnormality. We don't know that that abnormality is causative, but we know that it links all of these different illnesses, because not all of them have it, but many of them, in all of the categories we know as myeloproliferative diseases.

As he points out here -- it's kind of interesting -- when this JAK2 was first identified, the reason it's called JAK2 was somebody just said, Oh, it's just another kinase. Kinases are common enzymes. They didn't attach much significance to it, so they called it J-A-K, just another kinase, in other words. And it turns out it was quite significant. Just a curious thing.

**Q. In the Sterkers 1998 study that we talked about, you mentioned that this study concluded that hydroxyurea resulted in a specific chromosomal defect, a 17-P deletion. Correct?**

A. Yes.

**Q. Is a 17-P deletion consistent with benzene-induced AML or any other secondary leukemias?**

A. Not that I'm aware of.

MR. PATTON: Let's go off the record.

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(Break taken.)
(Exhibit 14 marked.)
**Q. I've marked as Exhibit 14 the Morgan 2005 study from some entity I've never heard of. Can you tell me what this article is, where it comes from, and what the significance of it is as it bears on your opinions?**
A. Well, I'm not an expert on this journal; but it's a peer-reviewed medical journal that often publishes reviews. And this is a particular paper that is entitled "Benzene and the hemopoietic stem cell." It's an attempt to review the mechanism of leukemogenesis with benzene.
**Q. And what is that mechanism?**
A. Well, this author believes that the mechanism is similar, as do many authors, with the other modern chemotherapy drugs we use today.
**Q. So from a mechanistic point of view, benzene-induced leukemia looks a lot like chemotherapy-induced leukemia. Is that accurate?**
A. That is correct.
**Q. And how is that significant to your opinions?**
A. Well, as he comments in here -- and many other articles do that too -- there is a particular latency that we associate with all chemotherapy drugs that

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produce leukemia and with benzene, which is an obsolete chemotherapy drug. And as he points out in here in speaking about benzene and relating it to chemical-induced leukemia from other drugs, similar to alkylating-agent-associated AML -- alkylating agent would be Busulfan, for example -- similar to alkylating-agent-induced AML, hematotoxicity and myelodysplasia often precede AML associated with benzene exposure. The latency period between exposure and disease lies between four months and ten years. Metabolism of benzene seems to be necessary for the toxicity. Cytogenetic changes seen include monosomy of chromosomes 5 and 7 together with interstitial deletions of chromosomes 5 and 7 together with translocation into 21q22.
So what he's telling us here is that -- and many other articles say the same thing -- is that benzene is, to a hematologist like myself, simply an obsolete chemotherapy drug. It works the same way as our modern chemotherapy drugs. It's probably not as potent as our modern chemotherapy drugs. It can cause leukemia. When it causes leukemia, we see similar chromosome abnormalities and we see a similar latency. And so that helps me in this case, particularly with latency.

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**Q. So Mr. Mielke's secondary leukemia looks like a benzene-induced leukemia. Fair statement?**
A. No, because his latency period -- his benzene was many years preceding his AML.
**Q. So is it your opinion that the latency period with alkylating agents is more consistent with the period experienced by Walter Mielke?**
A. Yeah. In other words, his exposure to benzene, whatever that exposure might have been, is simply too remote to be a consideration here. And the exposures he had to the P-32 and these other agents are much more attractive as a causation.
**Q. Why was Walter Mielke's exposure to benzene too remote?**
A. Well, I believe that his last exposure in here was considerably more than the ten years, as commented in this article.
**Q. That's assuming his last exposure was approximately 1985. Correct?**
A. That's correct. If we have a last exposure in '85 and we have an acute leukemia in 2003, in January of 2003, that gives us about 15 -- 18 years latency, which is longer than was generally thought to be the case.
**Q. So you calculate the latency period for benzene-induced leukemia as the latency period**

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**beginning as of the date of last exposure?**
A. No. I think the latency is really like every other chemotherapy drug we have. The latency is when you've received the proper cumulative dose. That gets you in the game. In other words, when we give people chemotherapy, it has always been the case that if you look at the chemotherapy that you get, there is a cumulative dose that you find at which you're in the game to catch leukemia. Now, 95 percent of the time you won't catch it; but you don't get it from that first pill or the second pill or the third pill. You get it after you've accumulated a certain amount of dose.
So true latency would be from the point you accumulated the dose to the actual development of leukemia. Now, that's the beauty of our chemotherapy data. Because number one, we have thousands of cases as opposed to a handful of benzene. Number two, the time interval between the first administration of drug and the last administration of drug is very short, usually no more than four to six months.
So some articles on chemotherapy-induced latency will start with the last exposure; some with the first exposure. It doesn't make any difference because they're so close together. And so, therefore, that's why latency is so accurate from chemotherapy data. When

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you look at benzene data, we don't have that accuracy because we don't know when the cumulative dose was really achieved. What we do know, though, is that from the two larger studies -- the Aksoy study and the Chinese benzene study -- that those authors suggest the latency is quite similar to what we see from chemotherapy. And that's what Hayes says in his article, and that's what Aksoy says in his article. So even though the data is poor, it looks identical with our chemotherapy data.

And so I don't think you can take a person who has a work period of 20 years and really say his latency began with the first day of work or the very last day of work. We don't know when the latency occurred. That's very hard to do. So what's happened in all of these benzene studies is people have arbitrarily given a latency date. In the case of the Hayes benzene study, he said, "Okay, latency begins after you've worked here six months. That's when it starts." That's the way he defined latency.

In the case of the Aksoy study, he did things a little differently. He noticed that benzene, like our modern chemotherapy drugs, can knock down the blood counts. And he said, "Okay. I'm going to look and see when did their blood count crater? And even if

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it got better, that tells me that they had a substantial exposure." And when he looked at from the time that they got low blood counts to the time they got leukemia, that's when he says months to six years. So his latency is totally different than Hayes's latency but, again, comes to the same general conclusion.

I think the pliofilm data simply can't be used for latency. There are too few cases, and their time span so long during their employment that it simply isn't a useful study for latency. It may be a useful study for cumulative dose. But I just don't think you can use that study for latency. There are too few cases in there and too wide a period to work with.

**Q. What does the Chinese study say about latency?**

A. Well, we've got it here. That's right on the top there.

**Q. That's Hayes.**

A. And he says, This finding suggests that recent benzene exposure is predictive of subsequent risk for ANLL/MDS, analogous to the short latency and wavelike increase, then decrease, in risk seen for several forms of radiation-induced leukemia and for chemotherapy-related AML. In other words, he's saying, just as I'm saying, that it looks just like chemotherapy, modern chemotherapy.

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**Q. Because you talked about that article, I'll go ahead and mark it as Exhibit 15.**

**(Exhibit 15 marked.)**

**Q. This will be the Hayes '97 study, Exhibit 15. So when you talk about once a person gets the cumulative dose, they're in the game, so to speak?**

A. They're in the game.

**Q. What is the cumulative dose of P-32 necessary to get someone in the game?**

A. Well, the cumulative dose apparently is less than -- is 10 millicuries or less, because we're told that 80 percent of these people had 10 millicuries or less. So obviously it's somewhere between 0 and 10. It's in that interval. And he had a whole lot more than that dose.

**Q. And the normal dose is 5 to 7.5 --**

A. Well, he said the normal dose is -- they gave, I believe, 2.3 milligrams per some weight index or meter squared. I've forgotten which it was; but whatever it is, it topped off at 5 in the polycythemia study. And they said, "Okay. Here are our calculations. We calculate this guy should get 5.67 milligrams; we'll give him 5." In other words, the maximum dose they recommended giving at any one injection was 5 millicuries.

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And in the case of Mr. Mielke, he got 5 on several of these; but on a couple of them, I believe he got 7.5. So he got a little extra. And he got, according to what I've marked on there, seven separate injections.

**Q. So for P-32, essentially, one dose and someone is in the game, so to speak?**

A. That's what it would appear to be, yes.

**Q. And that's to say one dose and you have an increased risk of AML?**

A. Yes.

**Q. What about hydroxyurea? What's the cumulative dose necessary for someone to get in the game?**

A. I would say that would be unknown. We just don't have the kind of data to really tell us that.

**Q. What is the dosage of hydroxyurea?**

A. What is the dosage? Well, they come in 500 milligram capsules, and it depends on what you're treating. In general, in polycythemia vera -- and I have a number of people taking hydroxyurea -- most people take 500 milligrams to 1,000 milligrams a day. He was taking 1500 milligrams a day, which is not an enormous dose. But what happens with that drug is it's very nauseating, and the more you give, the less likely your patients are to really take it. And so I don't

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like to give more than about a gram of that because I never know for sure if they're really taking it or not.

**Q. Have you ever had occasion to give someone 1500 milligrams per day of hydroxyurea?**

A. Only on -- not on polycythemia vera. In some instances in acute leukemia when the white count is very, very high, we usually give IV chemotherapy. And you can supplement that by giving hydroxyurea by mouth, and it makes the white count come down faster, which is thought to be beneficial. And I've given more than 1500 milligrams a day in that circumstance.

**Q. Would you consider a single dose of hydroxyurea to be enough of a cumulative dose to increase the risk of AML?**

A. Highly unlikely.

**Q. But you don't know what that dose would be?**

A. I don't know what that dose would be.

**Q. Do the articles we talked about discuss that dose?**

A. I don't believe that they do.

**Q. Is that unlikely, given the context of it's just a pill that is taken for some extended period of time and therefore harder to determine the cumulative dose? Or why do you think that's not covered in the literature?**

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A. I don't know why it isn't covered. You know, in terms of oral doses, we have that data for alkylating agents. There actually are data on the number of milligrams taken by mouth that puts you at risk. So the fact that it was taken by mouth isn't the reason we don't have that data. Apparently, it's just not published anywhere that I've seen.

**Q. Myleran is a alkylating agent and, therefore, can create an increased risk of AML. Correct?**

A. Yes.

**Q. Walter Mielke received myleran. Correct?**

A. Yes.

**Q. Did you do any determination of the cumulative dose of myleran Mr. Mielke received?**

A. No.

**Q. Do any of the articles that we've talked about or any other literature perhaps that you may know of discuss the cumulative dose of myleran necessary to get in the game, so to speak, as a causative factor or for an increased risk for AML?**

A. Not that I'm aware.

**Q. How is myleran administered?**

A. It's a 2-milligram pill, and it is used for -- was primarily used for chronic myeloid leukemia and rarely for polycythemia vera.

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**Q. Radiation therapy for prostate cancer, you talked about that as a possible cumulative or causative factor of Walter Mielke's AML. Correct?**

A. Yes.

**Q. Is there a latency period associated with radiation therapy and AML?**

A. Generally, it's thought to be similar to alkylating agents. In other words, alkylating agents are often called radiomimetic because they mime what radiation does. And so they're generally short latency periods for AML. They may be long latency periods for other cancers.

MR. PATTON: Off the record.

(Break taken.)

**Q. Radiation therapy, is there a cumulative dose of radiation necessary for an increased risk of AML?**

A. I don't know the answer to that. You'd have to ask a radiation physicist that. There may be data on that. I'm not aware of it.

**Q. Okay. So you know of a threshold level of cumulative dose of P-32 to cause AML, and that would be somewhere between 0 and 10. Correct?**

A. That's correct.

**Q. So anywhere between 0 and 10 micrograms, is it?**

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A. It's called millicuries for Madam Curie. So it's MC -- it comes as millicuries; and I have to look it up and see whether what's administered is in micro or milli. Let's see if I can find one of those articles so I can be specific. The numbers is 5, is what's usually used.

**Q. Okay. So is it your opinion that there's no safe level of P-32 in the context of its characteristic as a possible causative factor -- let me make a better question.**

**Is it your opinion that there's no safe level of P-32 because any administration of P-32 would create an increased risk of AML? Is that a fair statement?**

A. No. Obviously, there must be a safe level; but what we can just say is that we find an increased risk of AML in people that have gotten 5 to 10 microcuries. And so that's enough. Now, whether 1 microcurie could do it, I have no idea.

**Q. And we don't know the cumulative dose necessary of myleran for myleran to be a causative factor of AML. Correct?**

A. I don't know that. I mean, it may well be that there is literature on that subject; but I personally don't know that.

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**Q. The literature we talked about today talks about myleran is a causative factor or is creating increased risk of AML; however, it doesn't discuss it in the context of a cumulative dose. Correct?**
A. That's correct.
**Q. Hydroxyurea is, likewise, addressed in the literature as increasing the risk -- or increasing the incidence of AML; however, it is not examined, at least in the literature that we talked about today, in context of the cumulative dose necessary for that to happen. Correct?**
A. Yes.
**Q. And same question for radiation therapy? What was the conclusion?**
A. Well, my conclusion is I don't know the answer to that. It may be that there is literature out there. But again, a radiation person would be the one that would have better access to that.
**Q. But benzene, you have to have 200 PPM years as a cumulative dose to create an increased risk of AML. Is that your testimony?**
A. Yes. That is a common estimate.
**Q. So what is the latency window for benzene as a cause of AML, in your opinion?**
A. Well, I think the latency -- there's no magic

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in this world, and I think benzene is simply another chemotherapy drug. And we have chemotherapy drugs of all variety and structures and different ways to administer them, and they all come out between about 2 and 10 to 12 years. I don't think there's any difference between benzene and any of these other drugs we have. And that's simply what the literature like this Morgan article supports. So I think what goes for general chemotherapy we use today goes for benzene.
So as this article says, I think that we would look at latencies around 2 to 12 years.
**Q. What is the pool of people whose information is drawn upon to reach the conclusion of 2 to 10/12 years as the benzene AML latency period? How many people are we talking about that were studied to reach us to that conclusion?**
A. Well, not many. Because you see, if you look at the Aksoy study, he had -- we talk about the benzene as though it was a very strong leukemogenic drug. It really isn't. He had 29,000 people, and he had 40 cases of AML.
**Q. How many people?**
A. 40 out of 29,000. If we look at the Chinese study, they had 74,000 people; and they had 28 cases of AML and MDS combined. Well, I mean, if we took 74,000

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people and gave them the kind of chemotherapy we give today, we'd expect about 5 percent of them to get AML, or that's about 3700 people. So benzene doesn't have the potency of our modern drugs, but it certainly, under the proper conditions, can cause AML.
**Q. And what are those proper conditions, again, for benzene to cause AML?**
A. We think it's a cumulative dose that, as we best know from limited data, is about 200 part per million years exposure or higher.
**Q. When you say we know, that's your opinions. Correct?**
A. That is my opinion based on what is in the literature.
**Q. Have you reviewed Dr. Brautbar's deposition in this case?**
A. Yes.
**Q. And do you have any criticisms of his methodology in arriving at his opinions?**
A. No. I don't know all of his methodology, so I have no particular criticism of it.
**Q. Well, as his opinions are set forth in his -- have you seen his deposition?**
A. Yes.
**Q. You've seen his report?**

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A. Yes.
**Q. Dr. Levy, you've seen his report and deposition. Correct?**
A. Yes.
**Q. Do you have any criticisms professionally of Dr. Brautbar's opinions as those opinions -- I'm sorry -- of his methodologies as those are explained in the course of his opinions?**
A. Not of any methodologies, no.
**Q. What about Dr. Levy? Any criticisms of his methodologies?**
A. No.
**Q. What criticisms do you have of Dr. Brautbar's opinions in this case?**
A. Well, if you can hand me his deposition, I made a couple of notes on there.
**Q. We are not going to mark this as an exhibit.**
A. Well, in one area he talks about latency. Can we go off the record a second?
**Q. Sure.**
**(Discussion off the record.)**
**Q. We're back on the record. Dr. Natelson, before we took a break, I asked you questions about your professional -- or your criticisms in this case of Dr. Brautbar's opinion. You have a few notes on there, if**

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**you could explain nose criticism first, please.**

A. Well, only a very few points. One is he talks about latency and asked about latency from benzene to acute leukemia. And he says years ago it used to be 20 years, then it came down to 12 to 15 years. And then suddenly he starts talking about, oh, it can be 50 years. And I don't know where that came from. In other words, I haven't seen any new literature that says it's 50 years. In fact, we just talked about a recent paper that says it's 2 to 10. So I don't see any basis for that statement.

And then he talks about -- let's see on page 95 --

**Q. Can I stop you for a minute?**

A. Yes.

**Q. On the subject of latency, your opinions of the latency windows for benzene being 2 through 10, slash, 12, and that being the product of limited information from the Hayes study and the other studies that you referenced, those numbers, is that a median 2, dash, 10, slash, 12?**

MR. CHILTON: Object to the form of the question.

A. No. Generally, what we find is that in chemotherapy data, which is exquisite data -- and that's

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data that articles all suggest benzene acts just like our modern chemotherapy data, so it should be the same -- in that data, the window is generally given from about 2 to 10 or 12 years with certain agents toward the shorter side and certain agents toward the longer side. In other words, alkylating agents, like the myleran-type drugs, are more like the 7 to 10 years.

Drugs that we call topoisomerase II inhibitors, which many people think benzene is like, they actually are closer to the two- to three-year period; but they're all within this two- to ten-year period. If you look at the large chemotherapy studies, the mean latency is around 62 months from whatever you want to choose. The last part of the chemotherapy or the first part of the therapy, it's in the 60 some-odd-month range. So it's short.

And you just don't see -- and that data comes from many large studies of patients with illnesses like Hodgkins disease and breast cancer, where they're going to get these drugs and then live out, essentially, a normal life expectancy, so you can follow them for many years. And so we have -- and we're talking thousands of cases. So we have exquisite data.

**Q. Okay. Continue with your criticisms.**

A. Okay. He says over here on -- we're talking

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on page 95 and talking about leukemia from P-32. I also have to admit it's very rare, as his own treating physician says. Now, I guess that's a relative term; but when I see an article that shows me 40 percent of people that get P-32 can catch leukemia, I don't see how anybody can say that's rare.

**Q. Which article are you speaking of?**

A. The one with the graph. Any of the articles, whether they say it's 10 percent or whether they say it's 20 percent or whether they say it's 30 percent -- and this one goes up to almost 40 percent -- all of those numbers are not rare. So I don't think that this statement is consistent with the literature.

Then on page 106, he says, I think what you're trying to do is separate MDS, which is myelodysplasia, from AML, which is not possible. The majority of the epidemiologic studies in the past have looked at MDS as AML because MDS usually will transform into AML.

Well, that would be false. In other words, number one, MDS is not a single illness; it's a group of illnesses. So it depends on what form of MDS you're talking about. For example, in the so-called cyteroblastic anemia form of MDS, the transformation rate into AML is only 14 percent. In the case of

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chronic myelomonocytic leukemia, it's maybe 20 percent.

So this would be incorrect that myelodysplasia will usually transform. It may. It certainly will transform into AML more than the general population. But I think "usually" is not a good word, and I think you always have to qualify it by what kind of myelodysplasia you're talking about.

Essentially, the other thing is this: On page 74, in talking about threshold, he says benzene is considered as a carcinogen without threshold.

Well, based on all that we know about chemical-induced leukemia, that's a ridiculous statement. We don't know of any other chemical that produces leukemia that has no threshold.

**Q. What about myleran or hydroxyurea?**

A. Well, that information is out there. With alkylating agents, in general, there is information on numbers of grams and milligrams. And in fact, there's a very recent paper that I haven't used for this case that actually gives some beautiful graphs; and it shows that with alkylating agents. So that information is out there. But I don't know of any drug that's a carcinogen chemical that we have that there's not thought to be a threshold dose. That's simply the way these drugs work.

And I would disagree with his statement

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that benzene is considered a carcinogen without threshold. He may believe that, but I don't believe that the majority of the peer-reviewed medical literature would go along with that statement.

Those are the things that I would notice in here that I would disagree with.

**Q. Where did we miss each other about 10 minutes or so ago when we were talking about myleran and hydroxyurea and not being able to identify cumulative dose?**

A. I think I said it at the time. If you'll go back, I said we do know it with certain of the alkylating agents, like Cyclophosphamide, which is the classical one; and we do know that with that class of drugs. Now, that information may well be out there with myleran. I don't know it. But I do know it with Cyclophosphamide. And as a general class, alkylating agents are known to be cumulative in terms of their causation of leukemia.

**Q. Any other criticisms of Dr. Brautbar's opinions in this case?**

A. No.

**Q. Have you testified in any other cases where Dr. Brautbar was a witness?**

A. I'm confident that I have. His name is very

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familiar to me, but I can't tell you which ones those might be.

**Q. You also have in your file -- first of all, everything on the table here -- is this your complete file?**

A. Yes.

**Q. Do you have any other documents or notes or computer notes or anything else that has to do with your opinions in this case that we don't have with us today?**

A. No.

**Q. Okay. We have the disk of the transcript of Dr. Levy's deposition. Correct?**

A. Yes.

**Q. And let me go back and just read into the record from Dr. Brautbar's deposition. Per your notes, you have 52-8, 74-1, 84-11, 86-15, 116-6, 95-14, 96-13, 106-7.**

**We also have Dr. Levy's --**

A. I would comment that those are not all things that I disagree with. Some of these things -- dashes refer to things I do agree with, but they're all what I thought were interesting points.

**Q. But everything that you disagree with, did we talk about it?**

A. Yes.

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**Q. I'll next show you Dr. Levy's deposition. And can you explain to us what your notes are on the cover of that deposition and then what your opinions are -- are, rather, criticisms of Dr. Levy's opinions?**

A. One is a phone number, and I don't even remember who that's to. It may be to Mr. Chilton's office because it's got a 312 area code. I must have used this to write something down on. It has nothing to do with this specific deposition. Then I did write some things down. And let's see if I . . . This isn't 48. I was just interested in what he was saying about cohort studies. I don't have any particular criticism of that.

Page 102, if I can find this, it was just a comment. He was talking about the pliofilm study. I just made a note about that.

Page 103, I don't remember what I said here. And what I don't have here, because I wasn't really looking through it -- is that in the -- now, we can find the individual pages, but in the first part of this deposition when he's asked what the cause of the AML is, he says it was due to the polycythemia vera and its treatment. Then the second part of the deposition, when asked the same question, he says it's due to benzene. I couldn't understand it because I thought it was very clear what he was asked and what he answered in

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the first part of the deposition, but then it was like he changed gears in the second part of the deposition.

**Q. Any other criticisms?**

A. No.

**Q. We're not going to mark Dr. Levy's deposition. Also going through your time, you have Dr. Nicas's, Dr. Brautbar, Dr. Levy, and Dr. Rose's reports. Correct?**

A. Yes.

**Q. Do you have any criticisms of Dr. Nicas's opinions in this case?**

A. No.

**Q. You're not an industrial hygienist?**

A. No.

**Q. You don't perform any type of benzene exposure modeling and calculations, or anything of that nature?**

A. Correct.

**Q. And you're not giving any opinions on measurements of -- or estimations of Mr. Mielke's cumulative benzene exposure dose. Correct?**

A. Correct.

**Q. Your file also includes perhaps a duplicative copy of the expert witness disclosure?**

MR. CHILTON: Is one the supplemental one and one the original one?

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MR. PATTON: That's correct.
**Q. Your deposition also -- your file, rather, also includes a copy of Dr. Schrek's deposition. Correct?**
A. Yes.
**Q. There are a handful of red check marks in this transcript. Can you tell us the significance of those marks?**
A. Let's see. Well, the first one was a question of when did he start treating Mr. Mielke, and he says it was 1993. So that was just to help me as a frame of reference.
And then on the next check mark, which is on page 14, he says that people with polycythemia vera are more likely to come down with AML so that P vera is considered a risk factor for AML. And he agrees with that. He's asked what the cause of polycythemia vera is, and he says it's unknown.
In talking about the injection, it says it looked like there were four or five but that a detailed review of the records would be -- might be closer. And as I say, I've got pages marked in there that suggest it's seven.
And he says on page 18 that he's never seen anybody give more than six. In fact, they start

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taking the medicine down at about four treatments. Well, this guy got seven.
And then on page 19 he says the Busulfan is an alkylator, meaning an alkylating agent. And I think, without doubt, Busulfan probably increases the incidence of acute leukemia.
And then he talks about another alkylating agent called Melphalan. And then on page 32 he's asked, In your opinion, the AML was caused by the polycythemia vera? He answers yes. And then, again, to a reasonable degree of medical certainty, do you believe the AML was caused or resulted from the polycythemia? And his answer is the polycythemia and the treatment of the polycythemia together, yes.
Then again on page 25, he says, Even in the days before we had new treatment for polycythemia vera, it was known that these patients would get acute leukemia much more often than the average person.
And then again on causation, in his particular case on page 28, keep in mind that I think the major cause here was the treatment, the P-32.
And then he said makes a very correct statement on page 30 in terms of causation. The patients are very interested in Why did I get this? How did this happen to me? They have a natural tendency to

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want to blame their cancer for something. And that's a remarkably true statement.
It talks on page 32 about trying to keeping the blood counts down one way or another, and he mentions Winthrop's (phonetic) Textbook of Hematology. Actually, it's Wintrobe, W-E-I-N-T-R-O-B-E.
Then he tells us on page 33 that you can get AML from benzene when you swim in it. Then I thought this was a cute comment on page 37. And talking about why he was originally asked about causation, he says he had a wife that was a bulldog and she was convinced that his work was the cause of his leukemia and brought me volumes and volumes of information.
Then and he says something interesting, I thought, on page 38. He's asked whether smoking is a cause of AML, and he says no.
I thought this was interesting. On page 52 he's talking about Myelotarg, which is a new drug for acute leukemia. And it's very expensive, about 3 or $4,000 an injection. And I'm sure the company that makes this would like to know that it's an antibody, it's made by bacteria in a big vat in abandoned breweries around the country. They probably would dispute that, as to where this drug is made.
Let's see. That's the only thing I would

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have to comment about his deposition.
**Q. Okay. I'm not going to mark his deposition as an exhibit. Is there any industrial hygiene data upon which you are relying on in this case?**
A. No.
**Q. Are there any studies specific to tire builders upon which you are relying on in forming your opinions in this case?**
A. No.
**Q. Have you considered the Pasamonte (phonetic) study from 2003 in your opinions in this case?**
A. No.
**Q. Have you considered the Monson study from 1976 in your opinions in this case?**
A. No.
**Q. Have you considered the McMichael study from 1974 in your opinions in this case?**
A. No.
**Q. Have you Patalski (phonetic) and Kreger (phonetic) studies from 1973 or 1975 in your opinions in this case?**
A. No.
**Q. Did you look for any literature specific to tire builders, in forming your opinions and doing your literature review in this case?**

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A. Well, not specifically tire builders; but there are some of the articles -- and I may have referenced some -- that have to do with the tire industry, cohort studies.

**Q. Where are those at? Are they referenced in your report?**

A. Well, you said you didn't want to look at; but they're probably referenced in my report, whatever cohort studies I referenced. Let's see what I referenced in here. I take it back. I do not reference ones that are specific to tire production.

**Q. Have you reviewed any quantitative exposure data considering Walter Mielke's benzene exposure in forming your opinions in this case?**

A. Well, only the estimates that your expert has given.

**Q. Nothing else?**

A. Nothing else.

**Q. Are you relying on any qualitative information concerning Walter Mielke's benzene exposure, in forming your opinions in this case?**

A. You'll have to define that better for me. What qualitative information are you talking about?

**Q. For instance, there's testimony from Mrs. Mielke bearing on her observations and odor of her husband's clothes, things of that nature. There's also testimony by Ron Smiley and Delmar Huseman (phonetic) in an affidavit from Ron Smiley in this case which gives descriptions or other qualitative information bearing on benzene exposure. You did not review any of that information in forming your opinions. Correct?**

A. That's correct.

**Q. Have you had any conversations with any industrial hygienists as it relates to exposure levels that Walter Mielke would have had to benzene?**

A. No.

**Q. Do you know what Walter Mielke's job duties were?**

A. Not in any detail.

**Q. Do you have any other information regarding how much benzene he was exposed to in his work?**

A. No.

**Q. Do you have any opinions on the levels of exposure to benzene that he experienced in his work?**

MR. CHILTON: Just so the record is clear, other than what he's previously testified to from Dr. Nicas.

**Q. Besides your references to Dr. Nicas's report.**

A. No.

**Q. Do you have any opinions concerning the significance of the type of exposure a person has to benzene -- for instance, high intensity or peak exposure, as compared to consistent but low level exposure -- as that nature of exposure might bear on the likelihood of it as a cause of AML?**

MR. CHILTON: I'll just object as to form.

A. With all chemicals, it's generally considered that the cumulative dose makes the poison. Now, when you're getting very high peak levels, you can sometimes achieve that cumulative dose earlier. So for example, when we look at chemotherapy studies of people getting massive amounts of drug for the purposes of bone marrow transplants, sometimes you see them getting leukemia within a year following the treatment. In other words, you short of shift that curve because they get such an enormous cumulative dose. But it's still the cumulative dose. So my thinking is that the best evidence we have suggests that the cumulative dose, not the peak dose, is what's important.

**Q. Have you ever been to the Bridgestone Firestone tire-making facility in Des Moines, Iowa?**

A. No.

**Q. Have you ever been to any Firestone facility?**

A. Only a Firestone repair shop occasionally, but that would be the closest.

**Q. Have you ever been to any tire-building facility?**

A. No.

**Q. Have you ever had any conversations with any physicians that worked for Firestone?**

A. No.

**Q. Have you had any conversations ever with any physicians that worked for Shell?**

A. No.

**Q. Have you ever had any conversations with any physicians that worked for Ashland?**

A. No.

**Q. Have you ever performed any investigation as to how many workers at tire-making facilities have been diagnosed with AML?**

A. No.

**Q. What about diagnosed with other blood disorders?**

A. No.

**Q. Is Exhibit 2 the only draft of your report?**

A. Yes.

**Q. Did you draft that yourself?**

A. Yes.

**Q. Did you type it or dictate it?**

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A. Typed it.
**Q. Did anyone help you in this case in terms of reviewing the medical records, searching the literature, or anything else that might have contributed to the end product of your report?**
A. No.
**Q. Have you discussed this case with anyone besides the attorneys for Shell and Ashland?**
A. No.
**Q. I apologize if I asked this before, but how did you come up with the literature that we've talked about today and that is in this other pile?**
A. Well, most of these articles are articles that I have in my files. There are a couple of articles that I researched specifically for this study, and those would be the ones, sort of the more recent follow-up on the polycythemia vera study group work, and also reviews on P-32. Those would be the newer articles that I would have.
**Q. Can I see that pile, please?**
A. (Witness complied.)
**Q. I want to go through these articles very quickly just to see if they're cited in your report. Is the Berk New England Journal of Medicine article, Exhibit 6, cited in your report?**

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A. Yes.
**Q. The Najean study is?**
A. Yes.
**Q. The Nappi study?**
A. Nappi, yes.
**Q. Berlin?**
A. No, not -- the Berlin study is not.
**Q. Why not?**
A. I probably didn't have it at the time I wrote this report up. In other words -- or I had it and simply didn't include it, for one reason or another.
**Q. Was any of the literature that you're relying on in your opinions in this case provided by the attorneys?**
A. None whatsoever.
**Q. Exhibit 10, the Finazzi study?**
A. Yes, I have a Finazzi study in here. I'm not sure if that's the same one. Yeah, it's in here.
**Q. The Michiels, the Netherlands Journal, 1999?**
A. That's another one I don't have in here.
**Q. Why aren't those two articles cited in your report?**
A. It may be that I overlooked them from my file. Sometimes if I'm giving a deposition, I'll go back and look through my file and I may find something in there

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that's worth adding because I can't always predict what I'll be asked. So I try to anticipate some of the questions.
**Q. Morgan, 2005, I do not believe it's cited in your report.**
A. That's correct.
**Q. The Hayes study?**
A. That is cited, yeah.
**Q. Kaushansky, Exhibit 13?**
A. Yes.
**Q. Sterkers?**
A. Yes.
**Q. I don't want to talk about the rest of these articles, but I do want to cite them and mark them as exhibits.**
**(Exhibit 16 marked.)**
**Q. Exhibit 16 is the Smith study from 1996. Is that correct?**
A. Yes.
(Exhibit 17 marked.)
**Q. Exhibit 17 is the Eastmond study from 2003. Correct?**
A. Yes.
(Exhibit 18 marked.)
**Q. Exhibit 18 is the Whysner study from 2003.**

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**Correct?**
A. Yes.
**Q. Have you had occasion to work with Dr. Whysner before?**
A. No.
**Q. Are you aware that he's an expert in this case?**
A. I saw that somewhere. I didn't know that at the time I actually was looking at this paper, and then I was looking through something and I saw his name. And I don't know him. I've never spoken with him. I don't know anything about him.
**Q. Have you read his report in this case?**
A. No.
(Exhibit 19 marked.)
**Q. Exhibit 19 is the Larson study from 1996?**
A. Yes.
(Exhibit 20 marked.)
**Q. Exhibit 20 is the Larson study from 2005?**
A. Yes.
(Exhibit 21 marked.)
**Q. Exhibit 21 is the -- is this the Yin study from 1996?**
A. Linet study, yeah.
(Exhibit 22 marked.)

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**Q. 22 is the Aksoy study from 1979 -- or 1980, rather.**

**(Exhibit 23 marked.)**

**Q. 23 is the Aul, A-U-L?**

A. Yes, that's in here.

**Q. Study from some unknown year?**

A. No. It's 1998.

(Exhibit 24 marked.)

**Q. Okay. 24 is the Leone study from 1999?**

A. Yes.

(Exhibit 25 marked.)

**Q. 25 is the Bergsagel and Wong study, B-E-R-G-S-A-G-E-L, studies from 1999?**

A. Yes.

(Exhibit 26 marked.)

**Q. 26 is the Irons study, 2005. Correct?**

A. Yes.

(Exhibit 27 marked.)

**Q. 27 is the R.T.Gun study from 2006. Correct?**

A. Yes.

(Exhibit 28 marked.)

**Q. Exhibit 28 is the Bloemen, B-L-O-E-M-E-N, study from 2004. Correct?**

A. Yes.

(Exhibit 29 marked.)

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**Q. Exhibit 29 is the Satin and Wong study from 1995. Correct?**

A. Yes.

(Exhibit 30 marked.)

**Q. Exhibit 30 is the Divine 1998 study. Correct?**

A. Yes.

(Exhibit 31 marked.)

**Q. Exhibit 31 is the Lewis study from 2003. Correct?**

A. Yes.

(Exhibit 32 marked.)

**Q. Exhibit 32 is the Lynge, L-Y-N-G-E, study from 1997. Correct?**

A. Yes.

(Exhibit 33 marked.)

**Q. Exhibit 33 is the Sorahan study from 2002. Correct?**

A. Yeah.

(Exhibit 34 marked.)

**Q. Exhibit 34 is the Wong study from 2001. Correct?**

A. Yes.

(Exhibit 35 marked.)

**Q. Exhibit 35 is the Satin study from 2001. Correct?**

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A. Yes.

(Exhibit 36 marked.)

**Q. Exhibit 36 another Aksoy study from 1971. Correct?**

A. Yes.

(Exhibit 37 marked.)

**Q. Exhibit 37 is the Wong study from 1995. Correct?**

A. Yes.

(Exhibit 38 marked.)

**Q. Exhibit 38 is the Smith study from July 2003 from Blood. Correct?**

A. Yes.

(Exhibit 39 marked.)

**Q. Exhibit 39 is the Wong study from 1995. Correct?**

A. Yes.

MR. PATTON: Off the record.

(Discussion off the record.

**Q. Dr. Natelson, we just we went off the record and talked about the significance of your tabs on this approximately 10 inches or so of medical records. Can you explain to us the significance of these tabs and the stars?**

A. Yes. Each of those tabs reflects a page that

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has some useful medical information for me. That might be a history and physical; it may be might be a date of something occurring; it might be a blood count. And in particular, some of those tabs have double stars. And those have information that have to do with the administration of the P-32.

MR. PATTON: Okay. Off the record.

(Discussion off the record.

**Q. Dr. Natelson, as we discussed off the record, I just reviewed the 10 or so inches of medical records that you have, and I pulled out every one with a flag or star on it and I attempted to pull out -- where there were two pages of a report, I attempted to pull out those pages too. Is that your understanding of what we just did?**

A. Yes.

(Exhibit 40 marked.)

**Q. I'm going to mark this collectively as Exhibit 40. Who hired you in this case?**

A. Mr. Chilton originally talked to me about this case casually, and then I think Mr. Shilliday sent me all the records. And so I guess I don't know the answer to that except to say that I think this gentleman was the one who first mentioned the case to me.

**Q. Have you had occasion to work with Mr. Chilton**

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**or his firm on prior occasions?**
A. Yes.
**Q. Approximately how many times have you served as an expert in a case that Mr. Chilton was working on?**
A. Well, one I know of is a case called Lebron, L-E-B-R-O-N. And I reviewed a case for him that did not come to -- that I provided a report that had to do with aplastic anemia. I've long forgotten the name of that case. And possibly one or two others. I really can't remember.
**Q. Have you ever had an occasion to give an opinion in a benzene case involving a tire builder or other worker at a tire-making facility?**
A. I can't recall.
**Q. Have you ever served as an expert in a case involving PV?**
A. Not that I recall.
(Exhibits 3, 4, and 5 marked.)
**Q. Exhibit 5 is -- this is an invoice dated November 27, 2006, reflecting your time and payment in this case. Correct?**
A. Yes.
**Q. What is your hourly rate?**
A. $250 an hour to review records and $400 an hour for deposition or anything else.

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**Q. What is your rate for trial testimony?**
A. The same: $400 an hour.
**Q. Have you been asked to testify in the trial of this case?**
A. I likely have, and I likely said yes. I don't remember the specific conversation; but generally if I take a case to review, it's generally agreed that I will testify in that case.
**Q. Do you know when the trial in this case is scheduled for?**
A. No.
**Q. Do you know where this case is pending at?**
A. No.
**Q. Does this invoice, Exhibit 5, accurately reflect your time in this case?**
A. Yes.
**Q. Exhibit 4, this is a list of testimony given by you at trial or by deposition since 1992. Correct?**
A. Yes.
**Q. Did you testify in the Minnehan case?**
A. I had that case. I'm sure I must have given a deposition. It's probably listed in there.
**Q. It's listed as No. 36. Do you remember your opinion in the Minnehan case? Do you remember that --**
A. Well, Mr. Minnehan had a chromosome defect

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called inversion 16, as I recall. That's a chromosome defect that has never in the world's literature been described from benzene. And he alleged it was from benzene, or that was the just [sic] of the case. And it's a chromosome defect that is a poster child for de novo leukemia. So in my opinion -- and I don't remember all the details in the case in terms of the latency and everything else -- but it was my opinion that that was a case of de novo AML.
**Q. Simply based on the chromosomal abnormality?**
A. That was one aspect of the case. I don't remember the rest of it. He didn't have any period of myelodysplasia, and he had all the features of a classic de novo AML.
**Q. Have you ever given testimony on behalf of plaintiffs in any case?**
A. Yes. Some of those are plaintiffs' cases, not benzene cases, but they are plaintiffs' cases, nonetheless.
**Q. Let me ask a better question. Have you ever testified for the plaintiff in a case involving benzene exposure as a cause of AML?**
A. No.
**Q. Have you ever given testimony in a case that benzene caused AML?**

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A. No.
**Q. Have you ever given testimony in a case that benzene caused any other blood disorder?**
A. No.
**Q. In all cases that you've testified in involving benzene, your testimony has been that the benzene did not cause the disease in question. Is that accurate?**
A. Yes.
**Q. Has your testimony ever been excluded by any court?**
A. No.
**Q. Has your license ever been suspended or revoked?**
A. No.
**Q. How many cases are you currently serving as an expert in?**
A. I don't know the answer to that because what will happen sometimes is I'll get a case and three years will pass and I haven't heard a peep about it and then I have to call the attorney and say, Can I throw these records away, or what's going on? But many of the attorneys I've dealt with are not very prompt about telling me whether one is active or not active, and I simply can't give you an estimate even. I do know I

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have several boxes piled in my garage, but I couldn't even tell you what the names of the cases were.

**Q. Have you ever published any paper in any peer-reviewed journal concerning any hematological condition or malignancies?**

A. I'm sure I must have. I'd have to look at my CV. I remember publishing one paper about AML, a patient with spondylitis who had radiation therapy through his spine. I've published a paper that had to do with carcinoma of the pancreas, the treatment of it. I'd have to look through and see.

**Q. Have you ever published -- let's look at your CV for a minute. Page 11 --**

A. Yes.

**Q. -- item 74 --**

A. Yes.

**Q. -- what is that?**

A. That's an article that I've submitted about two or three months ago to a journal, and I haven't heard anything -- well, I've heard that it's -- when you submit an article to a journal, it's usually reviewed by the internal editorial staff. And then if it passes muster there, it's referred out to other reviewers outside the journal. And it's been sent out to other reviewers, but I haven't heard whether it's been

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accepted. It's highly unlikely it would be accepted without revisions.

**Q. What is your article about?**

A. It has to do with just what it says, in other words, how a clinician looks at the issue -- clinician being someone who takes care of patients -- how a clinician looks at the de novo and secondary leukemia in particular as to the influence benzene may or may not play in that illness.

**Q. Do you have a copy of your paper with you today?**

A. No.

**Q. Could I get a copy of it?**

MR. CHILTON: I'll object for the same reasons that your experts have given with respect to articles have been submitted for peer review and are not available during the peer-review process.

MR. PATTON: Did you ask my experts for articles? I don't remember that.

MR. CHILTON: Yes, indeed.

MR. PATTON: Maybe we should just get all the articles and see what they say.

**Q. Have you ever published any article concerning exposure to P-32?**

A. No.

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**Q. Have you ever published any article concerning a worker exposed to benzene, besides item 74?**

A. Not that I recall. I don't think so.

**Q. You are not an epidemiologist. Correct?**

A. Correct.

**Q. You are not an expert in industrial hygiene. Correct?**

A. Correct.

**Q. You are not an occupational physician. Correct?**

A. Correct.

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

MR. CHILTON: Object to the form.

A. Yes. In general terms, yes.

**Q. Would that be the right thing to do, to warn users of products containing benzene and the dangers of benzene?**

MR. CHILTON: Objection. Form.

A. Yes.

**Q. As a medical professional, do you believe companies selling benzene-containing solvents have a responsibility to warn people who are working with benzene of its danger?**

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MR. CHILTON: Object to the form.

A. Yes.

**Q. If Shell and Ashland in this case didn't warn people using their benzene-containing solvents concerning the dangers of benzene, that would be unreasonable, wouldn't it?**

MR. CHILTON: Objection. Form.

A. That's probably so, yes.

**Q. Do you know what year hematologists first reported an association between benzene and blood-related problems?**

A. Well, it was many years ago, before the -- actually in the late 1800s, I believe. But in terms of acute leukemia, it was really Aksoy in the '70s that brought this to the forefront.

**Q. As a clinician, if a person came to you and said, Dr. Natelson, I've been hired at a job, and in the course of that work, I will be exposed to one PPM a year of benzene and I intend to work at that job for 30 years, would you believe it would be safe for that person to do that work?**

MR. CHILTON: Object to the form.

A. Yes.

**Q. So is it fair to say that you believe 30 or so PPM years of cumulative benzene exposure is completely**

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**safe?**
MR. CHILTON: Same objection.
A. It's safe in terms of there's no evidence it can cause AML.
**Q. What about the evidence cited by Dr. Levy and Dr. Brautbar?**
MR. CHILTON: Object to the form.
A. I would say that what you do is you have to look at each individual study and you have to look at the concensus. You might say, What about the study by Bloemen where they show 40? It doesn't give acute leukemia. All studies are useful, but you have to look at the total weight of the evidence.
**Q. In your opinion, the total weight of the evidence doesn't support benzene as a cause of AML at levels below 200 PPM cumulative years?**
A. That's generally true, yes.
**Q. What's the exception to the "generally true"?**
A. Well, as I say, that's a level that's commonly used; and I think it's not -- it's not as precise as we would like. Some people think it's a little higher; some people think it's a little lower. I think it's a reasonable number to put out there. It's a level that's not likely to be achieved in the modern era.
**Q. When you testified earlier about P-32,**

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**myleran, hydroxyurea, and radiation therapy all as causative factors in Walter Mielke's AML, is it fair to say that each of those incidents of treatment with those four agents is a cumulative factor in his AML?**
A. I think they're additive in some form or fashion. I can't tell you the formula that would be used to say how additive, but I think that, to start out with an illness that has an increased incidence of AML and you start piling on treatments that are known to augment that incidence, what you end up with is someone who is highly likely to catch AML.
**Q. And in your opinion, benzene in no way factors into that cumulative analysis?**
A. That's correct.
MR. PATTON: Let's go off the record.
(Discussion off the record.)
MR. PATTON: Dr. Natelson, I thank you for your time; and I have no other questions.
MR. CHILTON: We'll reserve signature.
(Proceedings adjourned at 4:29 p.m.)

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Changes/Signature to Deposition of ETHAN NATELSON, M.D.
Deposition Date: November 27, 2006

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

_____
ETHAN NATELSON, M.D.

THE STATE OF _____)
COUNTY OF _____)

Before me, _____, on this day personally appeared ETHAN NATELSON, M.D., known to me (or proved to me under oath or through _____) to be the person whose name is subscribed to the foregoing instrument and acknowledged to me that they executed the same for the purposes and consideration therein expressed.
Given under my hand and seal of office this _____ day of _____, _____.

_____
Notary Public In and For
The State Of _____
Commission Expires: _____

CERTIFICATE

STATE OF TEXAS )
)
COUNTY OF HARRIS )

I, Janet G. Hoffman, Certified Shorthand Reporter in and for the State of Texas, duly commissioned and qualified, do hereby certify that the foregoing is a true, correct, and complete transcript of the proceedings in the foregoing captioned matter taken by me and transcribed from my stenographic notes.

IN WITNESS WHEREOF, I have hereunto set my hand and affixed my seal of office at Houston, Texas, this 11th day of December, 2006.

_____

Janet G. Hoffman
Texas CSR No. 4802
Expiration Date: 12/31/07
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