Acute Myelogenous Leukemia & Benzene	Reed v. BP 21:23-25	Q. Is it still your position that benzene only causes AML. A. Yes.
	Thorpe 19:4-6	"AML is the only malignancy that I believe has been definitively linked with high dose chronic exposure to benzene."
	Detel v BP 12:18-24	Q. You are of the opinion that benzene can cause acute myelogenous leukemia, correct? A. I am of the opinion that, under certain exposure conditions, and at high enough concentrations and exposures, benzene has been linked to the development of AML, yes.
	2004 chapter p. 529	"Human toxicity associated with exposure to benzene has been observed for more than 100 years. Early reports in occupational settings clearly established that chronic high-dose exposure to benzene could result in aplastic anemia and other blood dyscrasias and the development of acute myelogenous leukemia.
	2004 chapter p. 529	"The causal relationship between benzene and acute myelogenous leukemia is unequivocal."
	2004 chapter p. 535	"Probably the best studied and most feared consequence of long-term benzene exposure is the development of myelodysplasia and acute myelogenous leukemia (AML).
	2004 chapter p. 535	"Pathologic evidence suggest that it is plausible (even likely) that myelodysplasia and AML represent different stages in a continuum of benzene induced hematopoietic damage."
	2004 chapter p. 542	"Chronic, high-dose exposure to benzene has been linked unequivocally to the development of AML, often with a preceding myelodysplastic period."
	Rose Decl ¶8	"It has been well established that AML has been associated with exposure to high-dose chemotherapy, radiation, and certain chemicals, including benzene."

Acute Myeloid Leukemia - Rare disease	Oakley 137:18-24	Q. Is leukemia considered a rare disease? A. Depends on which kind of leukemia. Q. AML. A. I don't know. I mean, I think it's rare, compared to breast cancer or lung cancer, but rare is kind of a relative term. There are 12 or 13,000 new cases of AML in the U.S. every year.
Acute Myeloid Leukemia - Secondary	Rose Decl. ¶ 8	"It is also clear that AML arising secondary to treatment with alkylating chemotherapeutic agents (or benzene) is a clinical entity that is distinct from AML arising de novo, or primary, which have no readily identifiable cause."
Acute Myeloid Leukemia Subtypes & Benzene	Oakley 145:22 - 146:3	Q. What subtypes of AML are causally related to benzene? A. Subtypes of AML? Q. Yes, sir. A. The only one that I think you could make a credible argument that would not be linked to benzene is an M3 or acute promyelocytic leukemia."
Ambient exposure & dose	Baker 35:15-21 Baker 36:8-12	"If you're exposed to 24 hours a day but it's ambient background one part per billion, that's going to get you nowhere close to what a gasoline service station would be exposed to during a 40-hour work week or someone in Pliofilm or the China study or any of these other big cohorts." "What I'm saying is just because a 24-hour exposure doesn't mean that it's going to be higher than someone in the occupational setting would experience. In fact, that almost always is not the case."
American Chemistry Council	Remboldt 40:17- Thorpe 116-117	ACC and others are funding this project He's done work for the ACC on formaldehyde. It was for the Formaldehyde Council, which is part of ACC. He was paid about $25,000 for this. He's done and is doing work re butadience for the Olefins panel.
American Journal of Industrial Medieince	Baker 41:6-12	Q. Now, that's a reputable publication? A. Yes.
American	Behymer v BP	He did a review of childhood leukemia

Petroleum Institute I	5:25-6:19 Thorpe 115:19-25	for API. It's done but not published. Paid $27,000 for it He was personally paid $20,000 for it; Summit Toxicology was paid about 50 or 60,000. The whole project was very expensive; he doesn't know what the whole budget was. The exposure piece was very expensive.
American Petroleum Institute II	Behymer v BP 6:20-7:14	He has a new contract with API to look at data from the Pliofilm study. He has a confidentiality clause as to what until he publishes it. For sure in 2008. He's been paid $24,000 for it.
American Petroleum Institute China study	Oakley 178	he discusses ethical concerns
American Petroleum Institute Toxicological Review of Benzene	Baker 52:11-14	Q. Are you aware of the American Petroleum Institute in 1948 stating that there is no safe threshold for benzene exposure? A. I've seen that document.
Animal Studies	Oakley 30-34	He discusses why animal studies are relevant and useful
Asbestos research	Stubbs 94:25-97:19	Did one research project re asbestos involving molecular differences associated with fiber type specificity
Auer rods	Barker 17:19	"Auer rods. These are little sites of plasmic inclusions that are diagnostic of an M2 AML and there is some literature supporting that they are not seen as frequently in secondary leukemia as often as they are seen in primary or de novo leukemias.
Baker study on topo II	Remboldt 105:14-108:4	This study of which he was an author was later shown to be incorrect
Benzene Consulting	Behymer v BP 9:14-10:9	He's consulting on a couple of benzene cases: 1 NHL, 1 AML
Benzene - Diseases	Behymer v BP 15:3-	Q. Are you still of the opinion that benzene only causes AML? A. No. Q. What other diseases do you think benzene can cause? A. I think under the appropriate exposure conditions it can cause aplastic anemia. I believe there's literature and biological evidence to support myelodysplasia, as well as a variety of peripheral hematological

		abnormalities. Q. What would those include? A. Cytopenias, pancytopenia, lymphocytopenia.
Benzene - Leukemias	Cantu v. Citgo 58:12-59:1	Q. Have you seen this document? A. Yes. Q. Says 2001 which is, I guess, four years or so after Hayes came out? A. Yes. This is the ACGIH, their TLV documentation. So when they come out with the TLV just like EPA provided that support of their regulatory position, ACGIH comes out with a report that says here is what we think and here is why we think it. Q. Has ACGIH evaluated dose response with regards to benzene exposure? A. Absolutely, the have. That's what they do. This was out of that. To date, the only unequivocal dose response relationship for benzene-induced human cancer are those for leukemias." (Not just AML)
	Thorpe 38:7-25	Q. What does a chemically induced leukemia look like? A. Well, that depends really on the chemical. Q. Well, what does a benzene induced leukemia look like? A. Well, we've learned that most, in my view, about benzene from looking at the chemotherapy and I think it seems to match with the alkylating chemotherapy the best. And in those cases you would have potentially a hypocellular marrow. You would have a preceding pancytopenia, perhaps a preceding diagnosis of myelodysplasia and there would be evidence of cytogenetic changes. Q. What cytogenetic changes? A. The alkylating chemotherapy, you would see potentially monosome 7 or deletions in chromosome 5 as well as others, but those would be - seem to correlate the best.
Benzene studies ongoing	Oakley 151:23 - 152:7	Q. What other ongoing benzene studies are there? [other than the petroleum industry China studies] A. Well, NCI has got things going on in China. There's at least one study in Brazil that I know of, a researcher named Rego. They are doing some things in Germany. Rob Snyder is doing some in vitro work. Martyn Smith continually does in vitro work. David Eastmond is doing in vitro work. Neal Obernoff is doing - I mean, there's - there's API in

	Oakley 154-163	conjunction with Australia and the UK are doing a pooled analysis of the case control study.... He discusses his own ongoing studies re benzene
Blood journal	Oakley 176:1	“Blood is a heck of a good journal.”
Bone marrow	2004 Chapter p. 533	“the bone marrow acts as a trap for benzene and its lipophilic metabolites. This can potentially create locally high concentrations, with some studies reporting that the benzene levels found in the bone marrow are 150 times the concentrations measured in the peripheral blood. This is particularly germane to any discussion of benzene toxicity, because the bone marrow is arguably the most important target organ.”
Case reports	2004 chapter p. 537 Henricksen 187:19-20	“Beginning as early as 1897, case reports appeared in the scientific and medical literature linking or attempting to link chronic occupational benzene exposure with leukemia, specifically AML. ... Collectively ... they provide compelling evidence in support of an association between benzene exposure and AML. “I don’t rely on case reports.”
Cases he’s testified in	Detel v BP 13:5-15:16	Wilkinson - NHL - for Chevron Stubbs - for Radiator Specialty Remboldt - NHL - for US Oil Barker - AML - for Keenan Transport
Causation testimony	Henricksen 203:25 204:4 Thorpe 18:12-21	Q. So across the board as far as your retention to consult or testify in benzene-related litigation cases, is it true that you have never opined that benzene was the cause of the disease? A. I have never opined that. Q. So with respect to these - well, to the six cases in which you have given depositions this year that involved hematologic malignancies, in every one of them, did you testify that benzene did not cause the hematologic malignancy? A. Yes. Q. And is that also your opinion in the cases that you are here to testify about today?

	Thorpe 66:24-67:4	A. That the benzene exposures were insufficient to causes these diseases, yes. Q. Now, expanding the question beyond just this year, in all the cases in which you have testified where the plaintiff had AML, was it your opinion that the plaintiff had not been exposed to enough benzene to cause his AML? A. Yes.
Causative dose	Oakley 120:13-121:7	Q. What's the range, when you look at the literature, on the low end, starting with the low end, of exposures to benzene that can induce AML? A. That have been statistically significantly associated with an increased risk? Q. Start there. A. You don't - you know, there are broad ranges. So the Glass study suggests that in something greater than 8. Q. 8 what? A. Part per million years. It goes up to 60 part per million years is what they reported. Guenel. Q. Did you say "60" or "16"? A. From 8 to 60. Q. Okay. I just wanted to make sure I heard. A. Yeah. And Guenel, I think, is something greater than 17 part per million years up to infinity.... . . .
	Henricksen 212:5-13	Q. ... is there published, peer-reviewed literature that says there's an increased risk from AML from benzene exposure at 3 to 8 part per million years? A. You'll be able to find probably 3 - yeah, maybe, like I said, a quirky finding here or there where you could get to that." Q. Have you personally authored any letters to the editor as to those, quote, quirky findings to enlighten the scientific community that those findings are either illegitimate, don't pass the rad face test, or anything of that sort? A. No. ...
ChemRisk	Remboldt 30:22-31:21	Q. Have you spoken before attorney groups regarding anything other than benzene?

		A. I gave a presentation to a group of - it was a mixed group, but attorneys, scientists, and I think there were regulators in attendance as well on the molecular biology, if you will, of asbestos toxicity. Q. And what organizations sponsored this presentation? A. The organization that sponsored my participation was ChemRisk, a consulting company here in Cali-fornia. I'm assuming they got fund-ing from the attorneys that came. But I was asked by ChemRisk to come Q. Was this sponsored by the DRI, the Defense Research Institute? A. Not to my knowledge, no. Q. Was there any organization that sponsored it? A. No. Q. It was just a group of primarily asbestos defense attorneys? A. That's my understanding, yes. It was a small group and it wasn't publicly-you know. But you asked."
	Stubbs 77:16-24	Q. What group is doing the exposure piece [of the VCCEP]? A. That is ChemRisk. Q. And what is ChemRisk? A. It's a consulting company htat has offices several places. And one individual in that group is primarily doing the exposure piece on this project. Q. And who is that? A. Julie Panko is her name.
	Remboldt 41:16-42:1	He did an historical exposure assessment of individuals to vinyl chloride while working with Pamela Williams at ChemRisk for a trade association
	Remboldt 42:2-43:2	He did an expert consultation on the toxicology of MTBE for Pamela Williams at ChemRisk
	Remboldt 43:3-11	He did an exposure assessment and dose reconstruction for benzene exposure to workers on tankers for ChemRisk for colleagues at ChemRisk
	Remboldt 43:12-44:3	He was an expert witness for an occupational laryngeal cancer case from laboratory work for Chemrisk for a law firm in Los Angeles
	Remboldt 44:4-	Characterization of benzene exp. at manufactured gas plant -Williams
Childhood leukemia	Reed v. BP 6:10-8:7	He proposed this to industry at the Munich conference, and they agreed,

		but he was slow doing it and other reviews came out, “So I’ve had to kind of modify what I was doing so that it just wasn’t redundant of what had already been published.”
	Remboldt 37:25-38:16	“Then item No. 4 [on his CV], expert witness testimony and consultation on the alleged association between pesticide exposure and various forms of lymphoma. For whom have you done that work? A. The expert witness testimony part never occurred. The consultation was through ChemRisk.” ... Q. Do you understand that it was attorneys defending a case where it was claimed that pesticides caused lymphoma? A. That’s my understanding.”
Chromosome abnormalities - importance	Baker 11:18-12:7	Q. Doctor, why do you feel that cytogenetic analysis is important in AML cases? A. Why do I feel they’re important? Q. Yes. You stated that in your answer. A. Well, I think they’re important for understanding etiology. They’re important for understanding prognosis. They’re important for lots of reasons, understanding the biology, the underlying biology. At some point it might even be important with regard to treatment, but particularly from what I was doing in this case, **cytogenetics are very important in trying to understand the etiology of a specific case of AML.**
Chromosome abnormality	2004 chapter p. 541	“In secondary MDS, cytogenetic abnormalities occur in an overwhelming majority of cases (approximately 90%-95%), and the progression to AML is swift and usually unalterable (often within q year). Cytogenetic abnormalities seen in secondary MDS are often indistinguishable from those reported in secondary AML. The well-described propensity of secondary MDS to progress to AML also leads to the rational assump-tion that they may represent dif-ferent stages of the same disease.”
	Barker 22:16-23:3	Q. Would you agree with me that there are occasions where secondary leukemias present with no cytogenetic

		changes? A. Again, you are going to find examples in the literature of where that's true, but it is in the vast minority of cases. In some cases, some studies and series, the percentage of secondary leukemias that have some type of cytogenetic abnormality approaches a hundred percent, 90, 95 percent, so the myelodysplasia is less convincing. The lack of cytogenetic abnormalities in my view is fairly solid evidence that it did not have a chemical etiology.
	Barker report	"AML cases that develop secondary to chemical exposure (including chemotherapy) exhibit cytogenetic abnormalities often in excess of 90% of the cases."
	Oakley 203:17-25	Q. The majority of people that have benzene-induced malignancy have abnormal chromosomes, correct? A. Correct. Q. Why? A. Well, because it's probably part of the mechanism by which a cytotoxic chemical like benzene can transform a cell. It involves an alteration in the chromosomal structure."
Chromosome abnormality absence	Thorpe 38:3-6	"Well, Mr. Hazlehurst's AML was cytogenetically normal which, in my view, is not consistent with what we think a chemically induced leukemia would look like."
Chromsomes in benzene induced leukemia	Thorpe 38:7-25	Q. What does a chemically induced leukemia look like? A. Well, that depends really on the chemical. Q. Well, what does a benzene induced leukemia look like? A. Well, we've learned that most, in my view, about benzene from looking at the chemotherapy and I think it seems to match with the alkylating chemotherapy the best. And in those cases you would have potentially a hypocellular marrow. You would have a preceding pancytopenia, perhaps a preceding diagnosis of myelodysplasia and there would be evidence of cytogenetic changes. Q. What cytogenetic changes? A. The alkylating chemotherapy, you would see potentially monosome 7 or deletions in chromosome 5 as well as others, but those would be - seem to

		correlate the best.
Chromosomes 5 & 7 abnormality	2004 chapter p. 540	"One useful hallmark of secondary leukemia includes cytogenetic abnormalities that frequently involve the loss of part or all of chromosomes 7 or 5."
	Barker - Report	"AML arising secondary to treatment with alkylating chemotherapeutic agents is a clinical entity distinct from AML arising *de novo*. One hallmark of this type of secon-dary leukemia is the involvement of recognizable cytogenetic lesions, specifically the loss of part or all of chromosomes 7 and/or 5. Secondary AML following therapy with alkylating agents (e.g. melphalan, nitrogen mustard) is also frequently preceded by myelodysplasia or myelodysplastic syndrome (MDS) (~30-8-%) and typically has a latency period of 2-10 years. AML cases in workers occupationally exposed to benzene or chemical mixtures where benzene was the only recognized leukemogen, also appear to possess this same constellation of cytogenetic and morphological characteristics."
	Rose Decl ¶8	"It is also clear that AML arising secondary to treatment with alkylating chemotherapeutic agents (or benzene) is a clinical entity that is distinct from AML arising de novo, or primary, which have no readily identifiable cause." One hallmark of this type of secondary leukemia (sAML) is the involvement of recognizable cytogenetic lesions specifically the loss of part or all of chromosomes 7 and/or 5. It has been estimated that cytogenetic lesions involving chromosomes 7 and /or 5 occur 85-95% of the time in AML arising secondary to alkylating agents. In contrast, deletions of chromosome 5 and/or 7 occur much less frequently in primary AML. This disparity in the frequency of these specific cytogenetic abnor-malities serves as a useful bio-marker for discriminating between de novo and secondary leukemias, and as such, provides valuable insight into the issue of causation As the disease progresses, cytogen-etic abnormalities (5-/7- as well as others)are observed in virtually every case of sAML, providing evidence of the genotoxic mechanism

Chromosome 5 & 7 abnormality	Henricksen 179:23 - 180:1 Powerpoint	"That constellation of cytogenetic changes is very consistent with an alkylating-induced AML and is consistent with a benzene-induced AML." "Based on Current Understanding ... Chemically induced AML cases appear more likely to contain cytogenetic abnormalities than idiopathic AMLs. Existing data is consistent with the involvement of chromosome 5 and 7 in benzene-induced AML. The presence of specific cytogenetic lesions does not demonstrate etiology."
Chromosomes - benzene	Rose Decl¶10	"There is a constellation of cytogenetic lesions that result from a benzene induced AML, including 5-, 51- or monosomy 7."
Chromosomes - topo II inhibitors	Remboldt 109:2-110:22	He's unaware of any chromosomal abnormalities induced by topo II inhibitors other than 11q23 and t(15;17). Not aware of any others. He's not aware that inv(16) is associated with topo II inhibition, "but I could be wrong."
Cigarette smoke	Stubbs 121-122	
Collins, James	Oakley 20:24-21:10	Q. Is he a good epidemiologist? A. He's an excellent epidemiologist. Q. Have you seen his opinions about the Chinese study that Dr. Ions is doing for benzene? A. I have not. Q. Would it surprise you if Dr. Collins has testified under oath that Dow Chemical would not participate in the funding of that study because he believed it was biased? A. I don't know anything about that.
Competitive inhibition	Saltonstall 57:7- 59	he discusses it
Consultation	Remboldt 36:8-15 Remboldt 36:19-24	His CV says: "Expert witness consultation and testimony in toxic tort litigation dealing with benzene and other chemical exposures and the alleged development of hematopoietic malignancies including various types of leukemia, multiple myeloma and non-Hodgkin's lymphoma." Q. How many matters have you consulted on in this topic during this period while you've been affiliated

		with Summit Toxicology? A. Both of the affidavits that we discussed were prior to Summit Toxicology, just so we're clear on that. Maybe three times, four times. Q. And going back to No. 1, the sources of your payment or funding for those activities are defense attorneys and insurance companies, true? A. That's correct.
	Remboldt 37:8-11	
Consultation	Henricksen 48:12-7	He has been retained by law firms representing defendants in benzene cases about 30 times - about 4-5 AML cases.
	Henricksen 49:16 - 50:11	Q. How many times, in those four or five AMLs, have you rendered the opinion in any format, to the lawyers, to the company, in a report, that the AML was secondary to chemical exposure? A. There's at least one case that I can think of where it seemed like it was a possibility. Q. Okay. Well, seemed like it was a possibility. What was your opinion was to whether or not the AML was secondary to chemical exposure? A. That it was a possibility. Q. Well, a possibility could mean, I mean, 1 percent possibility or it could mean 99 percent possibility. So in your opinion, was it more likely than not that that person's AML was from chemical exposure? A. I didn't get that far. Q. And why didn't you get that far? A. Just never went any further than that. My understanding is they settled, but you'd have to track that down. I don't - I don't know what happened.
Cytopenias	2004 chapter p. 535	"Chronic exposure to benzene can result in a depression in one or more blood cell lineages or cytopenias."
Dar study	83:19-85:20	He hasn't read the study, just read the abstract. It is of no significance to him, because it concerned a pseudolymphoma, which he says is benign hyperplasia of lymphocytes, not even technically a true lymphoma, it wasn't malignant. And the subject had a pre-existing skin condition. And people use

		hydroquinone all the time.
Defense Research Institute DRI	Henricksen 68-69	He has never presented at DRI, but he was asked to meet with a group of attorneys and discuss benzene toxicity at the last meeting in Phoenix. He gave a talk to about 10 attorneys in a side group regarding benzene toxicity.
Delzell study (Unocal)	Remboldt 82:1-14	He has read the study. There is no reason why he did not include it in Exhibit 4-his literature compendium
Dermal absorption of benzene	Oakley 92:9-12	Q. Other than Maibach and Paustenbach, do you know of anybody else that is considered to have an expertise in that area of dermal absorption? A. It's just not my field.
Dose - cumulative	Thorpe 20:18-22 Thorpe 20:24-21:14	Q. Is there any study that has conducted an analysis of different dose metrics and specifically determined that the dose metric that is most sensitive for predicting leukemia risk is cumulative dose? A. I am not sure that anyone has gone about addressing the question the way you posed it. Q. You told me that the benzene exposure has to be of a sufficient dose and of a sufficient duration. Are you able to tell me what the dose component of that has to be as separate from the duration? A. Well, I am not sure that you could really do that. Q. Can you tell me what the duration part has to be separate from the dose component? A. That is really the flip side of the same question. I mean – Q. What is your answer though? Q. I think that is the debate as to whether or not - if you simply look at cumulative exposure, that does not give you all the information that you probably need to understand the risk and the biology. I think that would be generally accepted by everyone in this field, so both components are important. Acute exposures do not increase one's risk of leukemia, so there has to be some kind of prolonged exposure in order to start seeing an increased risk. Really low levels of benzene, no matter how long you are exposed to them, does not increase one's risk of developing AML. So there you have to have some degree of

		intensity. When you combine those, you need to get somewhere around 50 to 500 part per million years before the scientific literature will support that you have an increased risk.
Dose cumulative ambient air	Thorpe 71:1-20	Q. Do you have an opinion whether the cumulative dose of benzene that we all receive from inhaling the air wherever we live is a cause of leukemia? A. It is my opinion that it is not a cause of leukemia. Q. And why do you consider that not to be a cause of leukemia? A. Because it takes a lot more benzene to give a person leukemia than what you are going to be exposed to from just background living in an urban environment. Q. Do you know what a lifetime cumulative dose of benzene is from inhaling the air in California? A. I don't off the top fo my head. It wouldn't be that difficult to figure out, but no, I don't. Q. Okay. IN any event, it is your opinion that the - even though we are constantly exposed to benzene by inhaling it in the air, that that low level exposure to benzene continuously throughout our life doesn't significantly increase the risk of leukemia? A. That is my opinion, yes.
Dose - intensity	Thorpe 21:21-24	Really low levels of benzene, no matter how long you are exposed to them, does not increase one's risk of developing AML. So there you have to have some degree of intensity.
Dose - intermittent	Thorpe 23:20-24:1	Q. Now, you mentioned the terms "peak" and "intermittent," so what do you mean by them? A. Well, I think peak is peak. Peak is how high it got to within a certain exposure duration. Intermittent means it happens intermittently. It comes - it is not a continuous metric. It is not a continuous exposure.
Dose - peak	Thorpe 23:20-23	Q. Now, you mentioned the terms "peak" and "intermittent," so what do you mean by them? A. Well, I think peak is peak. Peak is how high it got to within a certain exposure duration.
Dose - intermittent & peak	Thorpe 22:16-23:7	Q. And how many acute exposures of any dose does it take to cause leukemia - to cause AML?

	Thorpe 23:8-19	A. Acute exposures don't cause leukemia. Q. Not at all? A. Not in my view. Now, if you start doing it more than once, then it's no longer acute. Now you are starting to talk about intermittent peak exposures over some prolonged period of time. And there you can quantitate what those exposures would end up being and you are going to be comparing those to the quantitative epi literature. If you are just looking at intensity, pure intensity, there are studies that have tried to associate risk of leukemia with intensity of exposure and those are consistent with the cumulative. They have to be pretty high. Q. How many peak and intermittent exposures to benzene does it take to cause AML? A. I guess it depends on what you mean by peak and how high they are. The only time that has ever been quantitated in the epidemiologic literature that I'm aware of is the study on the Dow workers published by Ireland where they saw an increased risk, bit it was not statistically significant. But nonetheless they saw an increased risk with peaks over 00 part per million the number of days that they had a peak of 100 part per million correlated with AML risk.
Dose - peak intermittent cumulative	Thorpe 24:12-25	Q. Do you consider peak and intermittent benzene exposures to be important in assessing leukemia risk? A. In terms of the - how they contribute to the cumulative exposure, yes. Q. But not otherwise? A. Well, how can they not contribute to the cumulative exposure? Q. I am just asking, you don't consider peak and intermittent benzene exposures important in assessing leukemia risk in and of themselves, is that true? A. I don't know how you could do that. I don't know how you could assess them in and of themselves except for how they contribute to the cumulative overall exposure with the exception of the Ireland paper.
Dose - intermittent	Thorpe	Q. Is intermittent or continuous exposure to a particular dose, which

v continuous	25:17-26:5	of those present the greater risk of leukemia? A. Oh, I see what you are saying. Probably the only what that they have been compared in a way to allow an answer to that is in the animal studies. And in the animal studies that literature is pretty conflicted. There are some studies that indicate that high dose intermittent exposure damages the marrow more than that same dose over a continual basis. But then there are other studies by Cronkite that are exactly the opposite, so I don't think that is totally known.
Dose - risk increased	Rose Decl¶14	"I am not aware of any reliable evidence to indicate that exposure to benzene in air at concentrations of 10 ppm or lower results in any increased risk of AML, nor have levels of benzene below 10 ppm been shown to be toxic to the bone marrow."
Dose metrics	Solis 14:16- Oakley 181-182	Q. What dose metric or metrics do you consider to be most relevant to benzene-induced immunotoxicity? A. That's a good question. I'm not sure. Q. What does metrics do you consider to be most relevant to benzene-induced genotoxicity? A. I think that's going to be an absolute concentration phenomenon. Q. So that would not be a cumulative-dose situation? A. No. ... He discusses the issue
Dose metrics - grams and micrograms	Baker 28:17-20	"When you put it into millions of micrograms, well, I have no idea why anyone would do that. You certainly cannot compare that with the epidemiological data in existence."
Education - Hem/Onc	Reed v. BP 25:15-	Q.... Have you had an, in your postgraduate work, training in hematology and oncology? A. Yes. ... I took the standard medical school pathology series that went through that organ system and looked at all of the various hematological diseases. And then we had an organ specific toxicology series, which also went through, from a more of a toxicological point of view, bone marrow effects and the effects on the hemato-poietic system. . . .

		W.
EPA Consulting	Behymer v BP 11:5-12:7	He consulted for EPA in 1990-1991, he did routine environmental work, mostly on the Rocky Mountain Arsenal, a big Superfund site in Denver, EPA's Region 8.
Epidemiology	Behymer v BP 26:17-27:8 Stubbs 113:13-114:22...117	He's now teaching a course in epidemiology at the University of Colorado in Denver. Title is Environmental Epidemiology. To grad students. Started Spring '08. He uses Gordis Fundamentals of Epi He doesn't hold an advanced degree in epidemiology. He's never worked as an epidemiologist for OSHA, but he uses epidemiological data as a toxicologist all the time. He's never conducted epidemiological research.
Epidemiology limits	Detel v BP 47:19-23 Saltonstall 45:4-10 Thorpe 37:15-20	Q. ... isn't the best epidemiology can give us, is whether or not there's an association between a chemical and an illness? A. I would say that's true. Q. Isn't it true that epidemiology is too insensitive an analytical methodology to determine whether there's a threshold below which an exposure to benzene cannot cause AML? A. I wouldn't disagree with the fact that epidemiology is a pretty imprecise analytical tool." Q. Well, isn't epidemiology too insensitive an analytical technique to determine increased risks for low level exposures? A. Well, I have heard that argument. Q. Isn't it true? A. I'm nor sure whether it's true or not.
Erythro-leukemia	2004 chapter p. 540	"Erythroleukemia (M6) is a frequently reported subtype of AML in benzene-induced leukemias."
Experiments	Stubbs 60:17-61:7	last did experiments re effects of benzene metabolities in 2000/2001
Expertise	Stubbs 48:2-15	Q. What do you consider yourself an expert in? A. Well, Im' a toxicologist. Q. ... as a toxicologist, what are you an expert in? A. Well, through my training and my

		research, I specifically investigated the molecular, biochemical alterations in hematopoietic cells in immunological cells following exposure to various chemicals, one of which ... was hydroquinone, which is an active metabolite of benzene. So I've done basic research on the effects of benzene on the specific cell types of interest in this matter." Q. Well, let me approach it another way and ask you, in what specialties or fields of toxicology do you consider yourself to be an expert? A. General toxicology, I consider myself to have expertise in. Hematotoxicology, toxicology associated with hematopoietic system and the immunological system, benzene toxicology, heavy metal toxicology, molecular toxicology.
	Remboldt 124:22-125:8	
Expertise, lack of	Reed v BP	He's not an industrial hygienist or a chemical engineer. 26:11-16
	Cantu v Citgo 62:25-65:5	He's not a medical toxicologist, a medical doctor, he's not board certified by the Board of Toxicology (he hasn't taken the test yet), not an epidemiologist, a statistician, a certified industrial hygienist, his expertise in IH is limited, he doesn't do exposure assessments
	Cantu v Citgo 67:6-24	He can't legally diagnose and illness using pathology or histology
	68:5-15	He's not an expert in ICD codes
	Stubbs 44:9-14	Q. Morphologically, chronic lymphocytic leukemia is a small cell lymphoma, correct? A. Well, I'm not really qualified to answer that. I think you would need to address that to a pathologist."
	Stubbs 59:17-	He's not an oncologist hematologist, hematopathologist,
	Stubbs 78:3-11	A. I'm really not the right person to be asking that question, because I'm not that good with exposure assessments. Q. You're not an expert in industrial hygiene? A. No, ma'am. Q. Have you ever personally conducted any type of exposure assessment to benzene?

		A. No.
	Remboldt 123:6-20	Not an epidemiologist, doesn't consider himself to be an expert in occupational medicine, hematology, hematopathology, cytogenetics, industrial hygiene,
	Remboldt 123:21-124:2	re immunology: "I know more than some, but no, I'm not a card carrying immunologist."
	Remboldt 124:9-14	Doesn't consider himself to be an expert in ecotoxicology, forensic toxicology, clinical toxicology
	Remboldt 125:9-11	Not an expert in pathology
	Henricksen 79	Not an IH, a pathologist, oncolo-gist, hematologist, epidemiologist
Exponent	Stubbs 49:20-50:2	His CV says he was a Senior Toxicologist with Exponent from May 2001 to October 2002
	Stubbs 99:18-25	Brett Finley was the practice director
	Barker 8:3-10:8	Q... you worked for Exponent out of Boulder, Colorado. Is that correct? A. That was my first consulting job and I did work in the Boulder office part-time for about a year. Q. Did you have any interaction with Dennis Paustenbach back during that time period? A. Yes. Q. And did you do work for Dr. Paustenbach? A. ... I'm pretty sure when I was with Exponent there was one benzene and multiple myeloma case that I believe you were involved in. Cronise was one, if I'm recalling it correctly, and I did help Dr. Paustenbach understand some of the multiple myeloma literature in that case, but my role was fairly peripheral. Q. Since that time with Exponent, have you done any work with Dr. Paustenbach? A. Well, Dr. Paustenbach and myself, our career paths have been parallel for a while, but I never really actually worked with him. When I left Exponent, I went back to the university. I had another grant funded and I was working there, which I did for about a year, maybe a little less, and then Dennis, Dr. Paustenbach

	Remboldt 52:11- Henricksen 42-46	approached me with his new company, ChemRisk, and asked if I would help start a Boulder office for ChemRisk, which I did, so I worked full-time for the new, the second version of ChemRisk here in Boulder for about a year and then I left that firm and went out on my own. His CV lists nine projects with Exponent: CCA, beryllium, latex allergy, dose reconstruction for benzene in methane production plants, brominated and chlorinated methanes, etc. He got job at Exponent through Sean Hays who was there when he applied. He worked on some benzene cases there.
Funding sources	Henricksen 80-87	API, Formaldehyde Council, EPA, Health Canada, CDC, State of Colorado The EPA hasn't asked him to conduct research on benzene. He actually hasn't been provided money by EPA or CDC.
Garabrant	Oakley 154 Henricksen 9-12	He's working with Garabrant on a reanalysis of the Pliofilm data based on Williams and Paustenbach's exposure estimates to see if the slope factor for benzene changes. He called Garabrant to ask him whether the later negative Health Watch studies negated the case-control study
General Acceptance	Henricksen 97:13-14	"I guess I don't know what you mean by general acceptance in the scientific community."
Glass	Oakley 120:13-121:1	Q. What's the range, when you look at the literature, on the low end, starting with the low end, of exposures to benzene that can induce AML? A. That have been statistically significantly associated with an increased risk? Q. Start there. A. You don't - you know, there are broad ranges. So the Glass study suggests that in something greater than 8. Q. 8 what? A. Part per million years.

Gordis	Behymer v BP 27:7-14	He uses Gordis' Fundamentals of Epidemiology in teaching. He believes it is generally trustworthy and reliable on environmental epidemiology.
Guenel	Oakley 126-128	he discusses the Guenel study
Harris Martin	Henricksen 73-74	He did a conference in New Orleans about 2005 and in California in the Spring of 2006 with Metzger, and another one. 3 conferences total.
Hematologic maligancnies	2004 chapter p. 542	"A variety of other hematopoeitic malignancies (Table 3) have been published in sporadic case reports and studies with the authors attributing benzene as an etiologic factor." Table 3: CML, CLL, NHL, APL, MM
Hill, Sir AB "Criteria"	Rose Decl.¶5 Remboldt 111:17- Remboldt 114:1-23	"The accepted methodology in the scientific, medical, and legal communities for rendering an opinion on the chemical cause of disease consists of faithful application of the Hill criteria." He's actually read Hill's article. He doesn't remember whether Hill used the word "criteria." Q. ... is it your opinion that all of these - what you're calling criteria, have to be satisfied in order to establish causality? A. I think if causality is present, then they all will be satisfied. Q. So is the answer yes, they all have to be satisfied? A. I think if there's an actual - if there's truly a causal relationship, then they will be. So yes." Q. Isn't it true that Bradford Hill in his article stated that they don't all have to be satisfied? A. ... I don't recall him making that - ... But even if that is what he says, in my view, that is too liberal."
Hourly rate	Stubbs Reed v BP	$220 per hour (2006) $260 per hour (2007)
Hypocellular bone marrow	Henricksen 14-16	He thinks a hypocellular bone marrow is indicative of a chemically induced leukemia; he got confirmation of this from Natelson
IARC	Saltonstall 59:10-18	Isn't it true that IARC has classified the occupation of painting as a carcinogenic occupation or an occupation that results in human

		contact with cancer? A. It's not based on leukemia. Q. I didn't ask that. A. Well, relative - Q. Has IARC classified painting as a carcinogenic occupation? Q. Based on lung cancer, yes.
Idiopathic	Baker 64	Q. And idiopathic typically is unknown cause? A. Well, that it's spontaneous, that it just occurred. De novo means it's new, it's not secondary to any kind of chemical or occupational or clinical exposures.
Immunotoxicity - benzene	Remboldt 63:17-64:6	Q. Have you in any way investigated the immunotoxicity of benzene? A. Not benzene per se. The metabolites of benzene, yes. Q. Which metabolites of benzene have you investigated for immunotoxicity? A. Hydroquinone. Q. Any others? A. No. Q. Did you publish that? A. Yes. Q. Which article is that? A. Hydroquinone, reactive metabolite of benzene inhibits NF-kB in primary human CD4+ lymphocytes and another on his CV Q. Incidentally, have you ever researched the literature regarding immunotoxicity of benzene? A. Yes. Q. And what do you conclude from that literature and your review of it? A. For animals, for humans? Q. Start with animals and go to humans, sure. A. I think the literature is pretty clear that high-dose exposure to benzene is an immunotoxic chemical in animal experiments. It is predominantly blocks of B cell lymphopoiesis. And there is literature that suggests occupational exposures to benzene of certain concentrations can result in subtle cytopenias in the peripheral blood including lymphocytopenias. ... The best ... is the Rothman study from 1996. ... At 7.6 ppm for a duration of six years, there was a statistically significant decrease in lymphocytes, the absolute lymphocyte count was the end point. No aromatic compound other than benzene is immunotoxic. No aliphatic

	Remboldt 6y8:1-15	or naphthenic compound is immunotoxic, to his knowledge.
Income - litigation	Thorpe 68:7-19	Q. And this litigation work that you do which compromises about 25 - you try to keep about 25 to 30 percent of your time, what percentage of your income does that generate? A. That is a good question. Probably more. Q. More than what? A. Probably would be more than 25 or 30 percent because a lot of my time - like this past fall I spent quite a bit of time teaching a toxicology class, but the university pays me very little. So if you average it all out, then the litigation might be 40 or 50 percent of my, you know, total income."
Individual susceptibility	Detel v BP 19:7-22:4 Saltonstall 60:10-12	he discusses the issue Q. Is it true that some individuals are more susceptible to toxins resulting in a cancer than others? A. I think that's yes.
Industrial hygiene principles	Cantu v Citgo 65:20-24	Q. The principles of industrial hygiene are to identify [] evaluate a[nd] control potential hazards in the workplace. That's not your job, is it? A. I did not realize that were the principles of industrial hygiene...
Industry Funding	Stubbs 82:6-10	API, CMA, and Formaldehyde Council
Industry work	Barker 11-13	He's only testified for industry, never for a plaintiff. He's never written a report supporting a claimant or plaintiff in his or her claim for compensation. He's only done litigation work for industry.
Infante Meta-analysis	Behymer v BP 37:19-44:25	His criticisms of Infante's meta-analysis
Intermittent exposure	Saltonstall 67:22- 68:11	Q. What if during those 40 years he quit smoking for a year at a time, a year here, maybe two years there, would that affect the amount - the dose of the smoking and therefore his risk of developing AML? A. It would definitely affect his

		dose. I don't think anyone can say definitively how that would affect his risk. Perhaps it would make his risk higher. Q. By smoking less? A. No. By smoking more intermittently the way you are describing. I'm not saying that's the case. I'm saying I don't, nor do you, predict how that's going to affect his AML risk. The dose will definitely go down. Q. If the dose goes down, doesn't the risk go down? A. That's not always the case.
Irons, Richard	Cantu v. Citgo 100:2-23	Q. Your mentor is Richard Irons? A. Mentor, yes. Q. He obviously – Dr. Irons has done quite a bit of work for those trade associations, hasn't he? (Referring to API and ACC) A. That's my understanding, yes.
	Stubbs 62:11-62:22	When he was a research associate from 1996 to 1997 Richard Irons supervised his research. When he conducted and supervised toxicology studies, examining biological alterations associated with the development of blood disorders following exposure to benzene, Richard Irons supervised him in this experiment. "Richard Irons held a grant that was paying for part of my salary and for the experiments."
	Barker 7:21	Q. You have a few publications with Dr. Richard Irons? A. That's correct. Q. Were you associated with Dr. Iron's laboratory at any point in time or were you just simply a co-author? A. No. Dr. Irons was my thesis advisor through my graduate training. Q. So you would have been working in his lab? A. That's correct. Q. When were you with Dr. Irons? A. I believe I started graduated school in 1990 and then I moved into Dr. Irons' lab, started doing experiments as a summer rotation the first summer and then the way our program was set up after two years of didactic classroom train-ing, they you settled into a lab to work on your thesis, so that would have probably

		been ’93 perhaps. Q. Did you stay there after completing your PhD in toxicology? A. I did. I did a post-doctoral research fellowship...with Dr Irons Q. That looks like from ‘96 to ‘97
Kirkeleit paper	Behymer v BP 45:21-47:13	His comments on it
Laitinen	Oakley 94:16-25	Q. Do you know if mechanics typically have more dermal exposures than they do the other routes? No opinions? A. I recall one paper, Laitinen, I think was the author, and that was the group of workers they looked at. And in that case he estimated - she; I don’t know whether it’s a her or she - but that author estimated that dermal represented, I think, somewhere between 1 and 80 percent, with the - the best guess was 60 percent of that person’s cumulative exposure.”
Latency - benzene	Oakley 61-62	“There have been really good analysis of three, probably, of the most important cohorts of benzene-exposed workers: The Health Watch, NCI, and the Pliofilm. And all three of those have had analysis done to try to understand the association between the timing of exposure and the risk of disease. And they’re all consistent. And they all say the same thing; nad that is, that it’s only exposures that occur within 10 to 15 years of diagnosis that matter. Those are the only ones that influence the risk of AML to any appreciable degree. Anything that occurred earlier than 15 years does not affect the risk estimate. So that time frame, that time window, is the most important in terms of understanding a person’s risk. It’s consistent upon those three cohorts. It’s consistent with radiation, with chemotherapy. I mean, it fits in with the biology of our understanding of the chemically-induced disease.”
Latency - etiologic window	Saltonstall 79:8-17	“What I[‘m saying is if you had exposures in 1980 and you weren’t diagnosed with AML until 2003, there’s very good evidence in the scientific literature to support the fact that those expsoures in 1980 did not increase your risk of developing disease, leukemia, in 2003. ... That window of opportunity, etiological window seems to be somewhere between

		ten, maybe 15 years. Q. So you are talking ti could be too long of a latency period for one to be related to the other? A. For benzene and AML, absolutely
Latency -topo II	2004 chapter p. 541 Oakley 199:13-14	“Leukemia secondary to topoisomerase II inhibition has a short latency period (6-36 months) and the absence of a preceding MDS. “Sometimes you can see leukemia associated with topo drugs in six months.”
Lindsay on benzoquinone	Remboldt 105:25-106:1	He hasn’t read the article by Lindsay re benzoquinone as a topo II poison
Lindquist study	Saltonstall 70-71	“It’s a positive study.” “Positive that painting as a profession is linked with an increased risk of AML.” “This one is over–the-top positive .... Something with a relative risk of 13, that’s huge.”
Litigation work	Baker 15:3-4	“I try to limit my litigation to about 30 percent of my professional time.... Sometimes it depends on how short a time frame you look at. It might be more than 40, 30 percent if I’m real busy on a case or two, but over the course of a year that’s what I try to do.
Litigation work re benzene	Oakley 229-233	he’s done Liquid Wrench cases, Craig case in 2003, Stephenson (not designated as an expert), Sugar Creek refinery cases, and for Chevron, Flint Hills NHL case in which he testified
Lymphocytes	2004 chapter p. 536 Remboldt 93:13-94:3	“It also has been reported that lymphocytes were highly, perhaps uniquely, sensitive to benzene toxicity. Suppression of absolute lymphocyte count is considered by the EPA to be the most sensitive endpoint in humans, and as such, forms the basis for their regulatory non-cancer toxic endpoint (reference concentration) for benzene inhalation.” Q. Isn’t it true that the lymphocyte is the most sensitive cell to benzene hematotoxicity? A. - he didn’t directly answer
Lymphoma, secondary	Remboldt 128:9-12	Q. Isn’t it true that large B cell lymphoma is the most common subtype of secondary non-Hodgkin’s lymphoma reported in that [the chemotherapy] literature? A. That, I do not know

Mechanics	Oakley 94:16-25	Q. Do you know if mechanics typically have more dermal exposures than they do the other routes? No opinions? A. I recall one paper, Laitinen, I think was the author, and that was the group of workers they looked at. And in that case he estimated - she; I don't know whether it's a her or she - but that author estimated that dermal represented, I think, somewhere between 1 and 80 percent, with the - the best guess was 60 percent of that person's cumulative exposure."
Meta-analyses	Behymer v BP 32:4-17	He reviews meta-analyses but doesn't rely on them because "you can sway the results substantially by what you include and what you exclude."
Multiple myeloma	Behymer v BP 33:19-23	Q. All right. Do you know why it is in your paper, under multiple myeloma, athat you stated that in the follow-up of this cohort there were not additional cases of multiple myeloma? A. I don't. That was a mistake.
MDS	2004 chapter p. 540 Henricksen 17-21	"Many of the blood dyscrasias reported in the past to be caused by benzene exposure would be classified according to modern diagnostic criteria as MDS. This would include all cases of "preleukemia," cases of AA with "paradoxical hypercellular" bone marrow, some erythroleukemia cases, and even multilineage cytopenias." He discusses diagnostic and pathologic features of MDS
MDS - secondary	2004 chapter p. 541	
MDS subtypes	Oakley 173-175	He discusses relationship between MDS and AML and the subtypes that are caused by benzene and those that aren't: MDS with ringed sideroblasts and CMML.
Myelodysplastic phase - MDS precedent	2004 chapter p. 540 Barker 19:16- 21:25	"Another distinguishing character-istic of secondary AML is that it is often, perhaps invariably, preceded by myelodysplasia." Q. Are you saying for a secondary leukemia, one that is the result of chemical exopsure, to be causally related to that exposure, the individual must develop myelodys-plastic syndrome before the

		frank leukemia? A. No.
	Barker 126:3-19	Q. "... just focusing on the cytogenic and pathological characteristics of secondary leukemia, where does the literature come down on one side or the other as to whether or not those types of characteristics have to be present in order to relate an AML diagnosis with chemical exposure? A. I don't really know the answer to that. I don't think that it does come down on that side."
	Henricksen 162-163	Asked whether that is still his opinion: "I think the 50 percent, maybe more, is a fair estimate."
	Henricksen 26:8-12	"That is my opinion, that you have to have a myelodysplasia that is distinct and diagnosed prior to the diagnosis of AML for it to fit into the category of a chemically induced leukemia."
	Henricksen 131:4-18	Q. Across the board, though, taking into account all of the studies, do you have an opinion on the percentage of secondary AMLs that also present with MDS first? A. ... I think across the board, you would probably see something on the order of, I don't know, 50, 75 percent, I don't know, because it is so variable. But the reason why it's so variable is there's two distinct classes of chemical leuke-mogens that are used in the clinic.
	Henricksen 132:25 - 133:25 Henricksen 178:24-179:4	He doesn't know of any diagnostic criteria requiring precedent MDS for a person to have secondary AML Q. Is there a source of the literature that you could refer me to that says myelodysplasia that occurs a few days before or really in conjunction with the diagnosis of AML is not indicative of a secondary exposure, but one that occurs six months before is? A. No.
Myelofibrosis	Stromberg 12	He's never published in the area of myelofibrosis or done any resarch in this area.
Natelson	Henricksen 14-	He had a discussion with Natelson regarding the clinical presentation of what a chemically induced leukemia might look like
Nested case control studies	Behymer v BP 29:15-30:8	"There's been a series of nested case control studies in refinery workers, which I think are also very good, and collectively that may end up being

		better quantitative information than what the pliofilm provides us. Q. Quantitative you mean in terms of quantifying exposure? A. Quantifying exposure, but more precisely identifying what the dose/response relationship is going to look like.
Neurotoxicity symptoms	Baker 45:9	“Well, I think high-dose exposure to any type of hydrocarbon can have those nondescript, systemic kinds of effects, dizziness and euphoria, almost feeling drunk, headaches, yes.”
Oil Com-panies he’s worked for	Detel v BP 69:7-70:2 101:12-25 Cantu v. Citgo 98:-20-24	BP, Shell, US Oil, Chevron. His VCCEP work was sponsored by BP, Chevron, Dow, Dupont, Exxon, Shell, Sunoco Citgo and Valero
Pancytopenia	Henricksen 20-24	He discusses pancytopenia as a consequence of AML, MDS, and aplastic anemia
Pancytopenia precedent	Oakley 65:5-20	Q. But pancytopenia does not need to precede AML for there to be benzene causation, correct? ... A. . . . There are definitely examples of where the AML just appeared as frank disease. Q. But as you sit here today, based upon the current body of scientific and medical literature, do you have an opinion as to whether pancytopenia must preceded AML for there to be causation? A. No, I would not say that it must preceded. No.”
Patel paper	Behymer v BP 50:19-53:19	his comments about it
Parts washing	Oakley 99:6-19	“Q. And do you have an opinion as to whether a parts washer using a benzene-containing product would have predominantly more inhalation exposures or skin exposures or do you know? A. I don’t think there is any way to answer that question. Q. You need more info? A. Sure. Q. And typically, you’re going to rely on industrial hygienists for that -

		A. Yes. Q. – right? A. Yes. Q. Because that's kind of outside of your field. A. You bet."
Paustenbach, Dennis	2004 chapter p. 546	"I would like to offer thanks to my colleagues and friends for their suggestions and helpful critiques: Dr. Pamela Williams, Dr. Dennis Paustenbach, Mr. Sean Hays, Ms. Susan Flack, MS. Kathryn Robinson, and Mr. Patrick Kerzic."
Paustenbach - corrupting literature	Henricksen 204:20 - 205:9	He has read about allegations that Paustenbach corrupted literature re hexavalent chromium and asbestos. He recalls reading something from the Envrionmental Working Group, a letter in the Wall Street Journal.
Paustenbach - his work with him	Henricksen 205:10-20	Q. Have you personally worked closely with Dennis Paustenbach on any project? A. Yeah, one. I mean, one I would say we worked reasonably close together. Q. And what project was that? A. This was a benzene case when I was with Exponent and he was with Exponent, and it was multiple myeloma. And it was Dean Hartley and Dennis was the testifying expert. And he just had me kind of as a consultant based on my understanding of the disease, the literature, benzene, et cetera."
Peak exposures	Barker 60-61	He makes some concessions re peak exposures from epi studies Q. But would you agree with me that peak exposures play some role in benzene's ability to induce AML? A. I don't know exactly what role it plays and I would reiterate that in my view the literature is more consistent with cumulative exposure but having caveated it with those two, yes, I would agree. I don't think that cumulative exposure tells us everything that we necessarily need to know about how benzene is acting."
Power calculation	Oakley 135:25 - 136:15	Q. What was the power calculation? [for the Pliofilm study] A. You mean whether or not it could detect? Q. Yes. Exactly. A. I don't know. Q. Does he report it?

		A. No. No. I think others might have gone there. I don't know the answer to that. Q. Why don't they do it? A. A lot of them do. Q. A lot of them don't, too? A. Right. I don't know. Q. Does it help you? A. Well, I mean, I think if you read enough epidemiological studies and you're in this business very long, I think that's almost a given that studies have limited capacity to detect small changes....
Refinery visits	Stubbs 166-167	He visited the Shell refinery in Long Beach. He toured the laboratory where they conducted quality control experiments on their products
Rego	Oakley 152-154	discusses Antonio Rego's work in Brazil and his discussion with him re benzene and non-hodgkin's lymphoma
Regulatory Toxicology Pharmacol.	Solis 82:2-	He's written 3 abstracts regarding the relationship between hemato-toxicity and chemically-induced AML and article on this is 90% done. Q. So are you now going to put all that together in an article and submit it? A. Yes. Q. Has the article been written yet? A. Define "written." 80 percent of it is probably done. 90 percent of it. Q. To whom do you intend to submit that? A. I don't know the answer to that. It won't be to Regulatory Tox and Pharmacology. Q. Is that because of what I said today? A. Yes.
Route of Exposure	Oakley 46:16-21	Q. Let me ask you: This discussion we're having about whether it's skin, ingestion, or inhalation, once it gets into the bloodstream, there is no reason to think that the - the effects will be any different? Is that generally accepted in your field? A. There's no reason to think for benzene.
Safe dose	Detel v. BP 85:5-14	Shown 1948 API tox review, that the only absolutely safe concentration for benzene is zero, he was asked" Q.Do you agree with that statement? A. I would have to disagree with that statement? Q. Why?

		A. Because I believe there are concentrations that are safe. Q. What concentrations are safe? A. One part per billion is safe.
	Henricksen 98:11-14 99:4-5	“One part per billion is safe.” “And so are many other exposures higher than that.” “I think the evidence supports that the PEL is safe.”
Smoking - benzene-AML	Barker 30:3-10	Q. ... if in fact it is the benzene in the cigarettes that’s causing the increase of AML, the levels that have been postulated from the epidemiological literature may in fact be too high? A. If that set of facts and circumstances is accurate, then I think that’s right, but I’m not - I would not agree that that’s the case.”
	Remboldt 128:22-129:4	He hasn’t read the IARC 2004 monograph on smoking or the portion of it re smoking and AML, or the surgeon general’s report from 2004.
	Saltonstall 63:10-14	Q. Benzene is contained in cigarettes, correct? A. Yes. Q. Is it the benzene in cigarettes that’s cuasative in AML, in cigarette smoke? A. It doesn’t appear to be the case.
Smoking - Chromosome abnormality	Barker 25:7-15	Q. When cigarette smoking is a cause of AML, does one see cytogenetic changes? A. ... I think a lot of studies that I can recall reviewing did not evaluate that. There were some studies that did evaluate that and, yes, there were cytogenetic changes, but a lot of them, and I have a pile of them here and we can go through them, I don’t believe they actually reported one way or the other.
	Barker 157:12-158:6	Q. Let me ask it to you this way. You are of the opinion that, you know, benzene did not cause this particular man’s AML, but you want to say that smoking as a secondary cause may have caused it, correct? A. Well, what I’m saying is that the gasoline literature in my view does not support an increased risk of AML and the literature in my view does support that cigarette smoking increases one’s risk of AML.

		Q. Both of which are secondary leukemias, both of which you indicated could not have been caused because of the presentment, correct? A. When I said it was a secondary leukemia, just by semantics if it's secondary to something like cigarette smoking, but the presentation, cytogenetic or otherwise, of cigarette smoking induced AML, I don't know what they look like. It is not established and it is not reported in the literature.
Smoking - Latency	Henricksen 149:2-9	Cigarette smoking is an established risk factor for AML . . . . But, in my view, there is a pretty well-defined etiological window or latency when exposures affect the risk of disease. And his cigarette smoking was too long - too early prior to his diagnosis to, in my view, have anything to do with the development."
Smoking - Surgeon General Rpt	Barker 25:16-	Q. Has the Surgeon General found that benzene is the strongest known chemical leukemogen in cigarette smoke? A. I don't know the answer to that. Q. Have you reviewed the Surgeon General's report on smoking? A. I have not.
Sonoda paper	Behymer v BP 47:14-49:8	his comments about it
Statistical power	Remboldt 130:9-18	Q. The size of the study is important in the first place in determining whether the study has sufficient statistical power to detect an effect, true? A. I think that's generally true for occupational epidemiology studies. Q. And if a cohort study does not detect an effect, that doesn't mean that there is no effect. It may mean that the study lacks sufficient statistical power to detect it, true? A. I think as a general matter that could be true.... Q. Isn't it true that a statistical power calculation must take into account the specific disease? A. That's outside of my area of expertise.
Strom paper	Oakley 175	he discusses the Strom paper published in Blood in 2005

Summit Toxicology	Stromberg 6-7	It's a small toxicology consulting firm. Him, Sean Hays, and Lesa Alwyard. 20% of its business is litigation. Only he does this. It's all defense; he's never worked for the plaintiffs.
Susceptibility	Oakley 147:12-17	Q. Why do some individuals succumb to a benzene-induced malignancy at different exposures than others? Do we have an answer to that? A. Well, I mean, I think the - the short answer is there's going to be genetic susceptibility issues with - with individuals.
	Henricksen 74-76	The definition of individual susceptibility is that within a given population you are going to have variability in response.
	Henricksen 106:2-9	Q. Is it your testimony that if someone gets a disease, they are susceptible to the disease? A. Yes. Q. Okay. So everybody who gets a disease is the susceptible population, right? . . . A. Yes."
TERA	Stubbs 76-77	He submits the VCCEP to TERA for external review
Texas Department of Environmental Quality	Henricksen 215:8 - 216:	In April 2008, he did work as a consultant for the Texas Department of Environmental Quality re mobile air standards for formaldehyde, butadiene, and benzene. Stuart Kagan, a toxicologist for Shell, got him involved. Kagan met him at the SOT meeting in 2007 when Pyatt was giving poster presentation. He wasn't paid for this consultation.
Threshold	2004 chapter p. 546	"Additional studies of cohorts with good exposure data might allow the scientific and regulatory communities to definitively determine if a threshold exists for benzene-induced leukemia."
	2004 chapter p. 538	"Both sides of the threshold argument have staunch supporters, and currently the issue is hotly contested."
	Reed v BP 20:11-	Q. You wrote in your 2004 paper that additional studies were needed to be done to definitively determine if a threshold exists for benzene-induced leukemia. Is that still your opinion

		today in 2007? A. Well, my opinion today, as my opinion was in 2004, is that a threshold clearly exists for chemically-induced leukemia, including benzene-induced AML. Q. But you didn't write that in 2004, did you? A. Well, I think you're taking that out of context.
	Detel v BP 17:25-	Whether there is a threshold for exposure to benzene is debated in the scientific community. He disputes that it's a hotly con-tested issue although he wrote that
	Barker 43:13-	"I don't remember writing it, but that doesn't sound - I mean, I agree that that's what is in there. Q. And that was in 2004, correct? A. When I wrote that paper? Yes. Q. So in 2004 you wrote in the peer reviewed literature that we need additional studies to determine definitively that a threshold exists for benzene induced leukemia. Correct? ... Q. I'm simply trying to understand now that we are in 2006 why we have the ability to assert a threshold definitively when in 2004 Dr. Pyatt wrote that we needed additional studies to allow us to make a definitive determination of a threshold and he has asserted that the dose response curve needs to be studied, et cetera, and I'm just trying to understand why that position has changed since 2004. A. It has not changed since 2004."
Threshold - agency recognition	Remboldt 97:2-	he can't identify a governmental agency which recognizes a threshold for benzene-induced leukemia or carcinogenesis. He thinks OSHA has language in their document.
	Saltonstall 45:11-117	Q. Can you identify any governmental agency which recognizes a threshold for benzene-induced AML? A. Not worded the way you worded it, not. Q. Isn't it true that OSHA has taken a position in its publications that no threshold for benzene-induced leukemia can be established? A. I seem to recall something to that effect.

Threshold dose	Solis 10:24-11:12	Q. So is it your opinion that exposure, a dose of benzene below 40 part per million years, does not present a risk of leukemia to people? A. To AML? Q. Yes, for AML. A. Well, I don’tt hink you can ever draw a line in the sand and absolutely say that, but I think that is what the epidemiology would support. Q. You are aware of epidemiologic studies that have reported significant increases in AML at doses below 40 part per million years, are you not? A. Yes. Q. Do you think they are wrong? A. Well, they are not all. ...
Threshold - epidemiology	Saltonstall 45:5-10	Q. Isn’t it true that epidemiology is too insensitive an analytical methodology to determine whether there’s a threshold below which an exposure to benzene cannot cause AML? A. I wouldn’t disagree with the fact that epidemiology is a pretty imprecise analytical tool.”
Threshold - proof	Saltonstall 46:5-9	Q. Can you identify, then, for me any published peer-reviewed article which states that the authors of the article have proved that there’s a threshold for benzene-induced leukemia or AML? A. I don’t know how you would ever prove it.
Toluene	Stromberg 12 Stromberg 33	He’s never done bench-type research in the lab on toluene. He’s never published regarding toluene exposure. He assumes the benzene content of toluene during the 1990s was 1.-.2%. “It’s going to be the same amount approximately that’s in all toluene over the last decade or so.”
Topoisomerase II	Barker 47:1-	Q. Do you accept his theory that benzene acts similarly to topo inhibitors in inducing leukemia? A. I have a hard time with that theory. Q. Why is that? A. Well, the cytogenetic lesion that’s associated with topoisomerase inhibition, the 11q23 has not been recorded in any benzene cohort. The sub type of leukemia, which is sometimes seen with topo inhibitors, acute promyelocytic leukemia, I believe the literature supports a separate etiology for that. I did

	Remboldt 104:14-25	some work with Dr. Irons in his lab with David Crowell and that has demonstrated that there are lots of ways of inhibiting topoisomerase and if it acts as a poison to the enzyme, then it will not form cleavable complexes. Therefore, it will not cleave the DNA and will not result in that type of genetic alteration. ... Q. Do you have an opinion whether benzene metabolites are topoisomerase 2 inhibitors? A. ... I think it's debated. Whether specific metabolites of benzene can inhibit that enzyme in vitro, I think currently there is more evidence that supports that that could occur than there is evidence that it doesn't. So yes.
Tox review	Stubbs 153:14-17	"Now, this was a superficial, almost Readers Digest, type review. I was not going to get into the subtleties of the literature in this book chapter."
Trisomy 8	Saltonstall 80-82	
Turakulov	Remboldt 85:21-	he hasn't read it.
University of Colorado	Remboldt 46:12- Baker 13:19-20	He worked as an assistant research professor of toxicology at U. Colo. He was not a member of the tenure-track faculty. As an Assistant Research Professor he paid his own salary w/ money from grants he got. He's not a tenured track assistant professor.
VCCEP risk assessment	Remboldt 25 Reed v BP 8:8-12:5	Q. And what was the funding that you receive for this project? A. Me personally? Q. You or your company. A. I don't know. Q. Can you estimate it? A. 50,000 Q. And is that the entire funding for all phases and aspects of your work on this project? A. For mine, for Summit Toxicology, yes. But that is a ballpark. It could be higher. I just don't remember." This was funded by API and the American Chemistry Council. This originated with Exponent when he was part-time

	Detel v. BP 72:2-	there and then Sean Hays left Exponent and went to another company and took the project with him. And because it was for children, there was a piece of that project for someone with expertise in leukemia, in blood disorders. So he was always peripherally involved, but his role increased when the project left Exponent. He was paid $27,000 for the child sensitivity paper plus $10,000-$15,000 helping Sean with the risk assessment, writing up the human health hazard portion of the risk assessment. Sean did a lot of other work for it; he doesn't know what Sean was paid for this. Sean and he are partners in Summit. He thinks Sean's pay was more than his, that Sean got close to $50,000 for the risk assessment portion He describes the program and its sponsors/funders
Williams, Pamela	2004 chapter p. 546	"I would like to offer thanks to my colleagues and friends for their suggestions and helpful critiques: Dr. Pamela Williams, Dr. Dennis Paustenbach, Mr. Sean Hays, Ms. Susan Flack, MS. Kathryn Robinson, and Mr. Patrick Kerzic."
Zhang re hydroquinone lymphoma	Remboldt 87:20-88:24	He's read much of her work but hasn't seen an indication of chromosomal abnormalities associated with lymphomas.