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IN THE CIRCUIT COURT OF KANAWHA COUNTY

WEST VIRGINIA

IN RE: ASBESTOS CASES (III)

CIVIL ACTION NO. 92-C-8888

ORIGINAL

DEPOSITION OF: RAYMOND D. HARBISON, M.D., Ph.D.

DATE: March 29, 1994

TIME: 1:10 P.M.

LOCATION: Radisson Hotel
Salon F
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Gainesville, Florida

TAKEN BY: Counsel for the Plaintiffs

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MR. PATRICK: Did you want to put something on the record before we started?

MR. HENDRICKSON: Yes. Just to maybe help you out a little bit, Dr. Harbison is being offered to testify about toxicology and pharmacology and their development and sort of as a demystifier of the use of asbestos as a mineral.

He has not reviewed the medical literature and will not be called to testify about the state of the art as far as medicine goes. He has not reviewed any Westinghouse documents and will not be called to comment upon anything that Westinghouse may or may not have done and will not be used at trial to testify about the O-I documents that he has been -- the Saranac Lake study primarily that he has testified about, I believe on two or three occasions.

Is that correct?

THE WITNESS: That's correct.

MR. HENDRICKSON: So I just thought I would put that on the record so that it may help you out.

MR. PATRICK: Okay. I think that may shorten the deposition a good bit, but there may be some questions that relate to some areas of prior testimony. I understand he is not being offered to opine about the Saranac documents as it relates to Owens-Illinois or Kaylo or whatever, but that may come up just in terms of his understanding of the medical literature as it pertains to asbestos, if that becomes an issue, and it may not become an issue in this case.

MR. HENDRICKSON: Okay, that's fine.

Thereupon, RAYMOND D. HARBISON, M.S, Ph.D., being first duly sworn, testified as follows:

DIRECT EXAMINATION

BY MR. PATRICK:

Q. Dr. Harbison, my name is Charles Patrick and I'm an attorney representing the plaintiffs, one of many attorneys representing the plaintiffs in this consolidated action in West Virginia, and it's my understanding that you are prepared to testify as an expert witness on behalf of Westinghouse in the West Virginia asbestos cases. Is that correct?

A. I've been asked to prepare to testify concerning pharmacology and toxicology with regard

to the general area of materials, including many different types of chemicals.

Q. Okay. Let me show you the last page of a witness disclosure that has your name.

MR. PATRICK: And if we could, let's mark that as Exhibit 1.

MR. HENDRICKSON: Do you want that as 2, the notice being Number 1?

MR. PATRICK: Yes. We'll have that Number 2, since the notice is Number 1.

Q. And if you would just look at it. And it states, "Raymond D. Harbison, M.S, Ph.D., practices pharmacology and toxicology. Dr. Harbison will testify about the evolution of toxicology and pharmacology as it relates to all substances, including asbestos."

Now, with respect to that portion of the witness disclosure, is that the understanding of your general testimony that's going to be offered in this trial?

A. Yes, sir.

Q. Let me stop right there. When were you first contacted about this particular proceeding?

A. I would estimate it was probably about a month and a half, two months ago.

Q. And who contacted you?

A. David Hendrickson.

Q. And I believe you have testified at the request of Mr. Hendrickson before?

A. I have.

Q. And that was in a proceeding in West Virginia?

A. Yes, sir.

Q. And when was that? He was nice enough to fax me the direct examination of your testimony in this case, but I don't have a date. Do you know when this was?

MR. HENDRICKSON: Can I answer it?

MR. PATRICK: Sure.

MR. HENDRICKSON: It was in March of last year, around March 15th or 16th.

THE WITNESS: Of '93.

MR. HENDRICKSON: '93, right. That might not be the exact date, but it was in the middle of March.

MR. PATRICK: That's okay. I don't have the cross, but I see Mr. Lipman, and I assume that's David Lipman from Miami, Florida.

MR. HENDRICKSON: The reason I didn't send that, Ann said send the direct and fax it.

I would be glad to give you the cross, too. I'll be glad to furnish it to you.

MR. PATRICK: That's fine, and I'm not complaining about not having the cross.

Q. But it was David Lipman who did the cross-examination?

A. I believe that's correct, yes, sir.

Q. And in that instance you were testifying on behalf of Owens-Illinois?

A. Let me say I don't testify on behalf of anybody, I am an independent practitioner of pharmacology and toxicology, but it was certainly at the request of Owens-Illinois.

Q. And at the request of Mr. Hendrickson and at the request of attorneys for Owens-Illinois I believe you have reviewed, at least reviewed prior to your testimony in this case, certain documents from the Saranac Laboratory as it related to tests concerning Kaylo?

A. Yes, sir.

Q. Now, Mr. Hendrickson stated for the record that you have not looked at any documents pertaining to Westinghouse. Is that correct?

A. Yes, sir.

Q. Have you looked at any documents other

than the Kaylo documents or the Saranac Laboratory documents that may pertain to asbestos generally?

A. I have not, other than scientific literature and other information that I am generally familiar with. Nothing specific.

Q. Okay. Let's go back to the witness disclosure. It says you will testify about the evolution of toxicology and pharmacology as it relates to all substances, including asbestos. And I know this is a general question, but could you explain sort of what that means? What is the expected thrust of your testimony in that area?

A. The science of pharmacology and toxicology as it relates to the evaluation of the effects of substances on the human system, as it relates to dose-response relationships and the evaluation of levels that are without effects on the living system, the evaluation of the potential harm and benefits of materials. Generally the concepts and principles that underlie the practice of pharmacology and toxicology.

Q. First of all, is asbestos a toxic substance?

A. Well, asbestos can be a toxic substance, as can all other substances. The level of exposure

will determine whether it's toxic or not toxic.

Q. And in what way is asbestos toxic?

A. Well, if there is excessive exposure to asbestos, it can affect the respiratory system resulting in a fibrosis or a pneumoconiosis, known as asbestosis. It may also lead to an increased risk of cancer of the respiratory system and also an increased risk of a mesothelioma, or a cancer of the lining of the pleural cavity, or of the cavity of the body.

Q. And when you say excessive levels of exposure can cause these diseases, what do you mean by excessive?

A. Well, there are guidelines for levels of exposure that don't cause those effects to occur. It would be levels that are considerably above that that may lead to those effects or those changes or that increased risk.

Q. Do you know what the presently existing threshold limit value or permissible exposure level is for asbestos?

A. It is .2 fibers per cc, or per milliliter.

Q. And who has set that standard?

A. Who has set it?

Q. Who has set that particular limit for exposure to asbestos, if you know?

A. The Occupational Safety and Health Administration or the American Conference of Governmental and Industrial Hygienists, which is a guideline, not a legal standard. OSHA is a legal standard.

Q. Is it your understanding at levels below .2 fibers per cc would not result in an increased risk for either asbestosis, lung cancer or mesothelioma?

A. Yes.

MR. HENDRICKSON: Hold on just a second. Could you just separate them out one at a time?

Q. That's fair. Is it your opinion that if you abide by the permissible exposure limit -- let me rephrase the question.

Is it your opinion that an increased risk of asbestosis does not occur if exposures are less than a PEL of .2 fibers per cc?

A. That is correct.

Q. Is it your opinion that there is no increased risk for cancer, lung cancer specifically, if the exposure is less than .2 fibers per cc?

A. With regard to the occupational exposure,

yes.

Q. And the same question for mesothelioma, is it your opinion that if exposures to asbestos are below .2 fibers per cc that there is no increased risk for mesothelioma?

A. Again with regard to occupational exposure, the answer would be yes.

Q. Now, what do you base those opinions on, what specific document or materials or whatever?

A. Based upon my knowledge of the permissible exposure limit, the Code of Federal Regulation with regard to the permissible exposure limit and the use of the permissible exposure limit in the workplace for a level of allowable exposure that does not significantly increase risk for any disease, including cancer.

Q. Now, at what point would one have to be exposed above the permissible exposure level of .2 fibers per cc before there was an increased risk for asbestosis?

A. I haven't done that, and it would depend on the length of the exposure, it would depend on the fiber size, it would depend on the respirable characteristics of the fibers. All of those would be important considerations in evaluating that risk.

So to evaluate the risk, one would have to evaluate the exposure, which would be dependent upon the individual, where he worked, how long he worked and all of those other factors, to evaluate the exposure, which would ultimately determine the potential risk.

Q. Would your opinion be the same for asbestos-related lung cancer?

A. Yes, it would.

Q. How about asbestos-related mesothelioma?

A. Yes, it would.

Q. Are you aware of reports in the medical literature that have demonstrated cases of mesothelioma where exposures to asbestos were relatively brief or transient?

MR. HENDRICKSON: Once again just note my objection to the fact that he hasn't reviewed the medical literature, he is not going to testify about the medical literature, it's kind of outside his realm.

But if you have an answer, go ahead.

A. I really haven't evaluated that.

Q. In your prior testimony that you gave in 1993 I noticed that you talked about a number of substances, polyvinyl chloride, salt, I believe you

even talked about water, various substances that were capable of becoming toxic in excessive quantities. Do you recall the general thrust of that testimony?

A. I do.

Q. Have you attempted to establish a hierarchy, a level by which you can put either polyvinyl chloride or cyanide on top and asbestos somewhere below that or asbestos on top? Have you tried to do something like that?

A. For what?

Q. For any toxic chemicals or agents.

A. Well, it would depend on the effect. So one would have to look at the response in order to rank the effect or to evaluate the potency, so I haven't done that. It would depend on what one was looking at, whether it's mortality, morbidity. What the evaluation was would determine the hierarchy, that is, the potency.

Q. In your opinion is there a safe dose of asbestos?

A. Yes.

Q. What would that be?

A. The exposure below .2 fibers per milliliter and even above that, at some level above

that, and I don't have an opinion about what that is. It would again depend on the exposure.

Q. You wouldn't know what a dangerous level of exposure to asbestos would be then?

A. Sure, I would. I would have to determine that based upon the potential exposure, based upon what the individual was exposed to and how long that individual would be exposed.

Q. Have you done anything of that nature for this case?

A. I have not.

Q. Are you familiar with the OSHA regulations as they pertain to asbestos, and in particular the document that's dated June 20, 1986 concerning asbestos?

A. I don't have complete familiarity with it. I would have to look at some passage or some part of it. I don't have complete familiarity.

Q. Are you familiar with the fact that OSHA conducted a literature search, a review of the medical literature in an attempt to try to determine the extent to which asbestos was a dangerous or toxic substance?

A. I am not aware of that.

Q. Let me just read from Pages 22615, and

this is a document dated June 20, 1986, and it's the introductory paragraph for health effects. It states that, "OSHA is aware of no instance in which exposure to a toxic substance has more clearly demonstrated detrimental health effects on humans than has asbestos exposure."

My question is, do you agree or disagree with that particular statement?

A. I would have to look at the context in which that statement is made, whether that's with reference to an overexposure or in reference to an effective dose. I would have to look at the context. I don't know.

MR. HENDRICKSON: Do you mind showing it to him?

Q. Sure. (Hands document to witness.)

Well, Dr. Harbison --

A. I'm sorry. Can you tell me where that was?

Q. Yes. The third column, last area there, health effects.

A. (Peruses document.) Okay.

Q. My question was, do you agree or disagree with that statement?

A. Well, I would generally agree with the

statement that there are clear effects produced by asbestos. I wouldn't take one sentence out of context and cite that there is no instance in which it is more clearly demonstrated. I would certainly agree that for asbestos there are clearly demonstrated toxic effects that can occur as a result of some levels of exposure to asbestos.

Q. Doctor, in your opinion at the present time would the use of asbestos-containing industrial insulation create a hazardous condition?

MR. HENDRICKSON: I'm not trying to nitpick. Because in this case we have all kinds of different products, we have packing, we have pipe covering, are you talking about the gamut or are you just talking about pipe covering or --

MR. PATRICK: Well, let's just limit it at this point to pipe covering.

Q. In your opinion, doctor, would the use of asbestos-containing pipe covering create a hazardous condition?

A. It depends on the opportunity for exposure or the opportunity for contact. It depends on whether exposure could result from that use and whether that exposure would be an exposure that

could result in some adverse effect or increase the risk of some adverse effect occurring.

Q. Well, you're familiar with the Saranac documents, are you not?

A. I am.

Q. And you're familiar with at that time the threshold limit value for asbestos or asbestos-containing dust was 5 million particles per cubic foot of air. Correct?

A. Yes.

Q. In your opinion would exposure to asbestos in excess of that threshold limit value of 5 million particles per cubic foot of air create a hazard?

A. It's possible.

Q. Are you aware of instances in which insulation workers who had used asbestos-containing thermal insulation developed asbestosis?

A. I don't specifically recall that. I would have to look.

Q. Let me show you a document which is numbered 5742, and it's one of our master exhibits for the West Virginia proceeding, we'll have it marked as Exhibit Number 3 for this case. And this concerns cases of insulation workers who had made

claims against Owens-Corning Fiberglass for the inhalation of asbestos-containing Kaylo.

Have you seen this document before?

A. I don't believe so.

Q. Let me ask you to assume that Owens-Corning Fiberglass between 1953 and 1958 was a distributor for Owens-Illinois of Kaylo, and that after 1958 they manufactured Kaylo, which contained asbestos, and that between approximately 1955 and 1973 they received somewhere in the neighborhood of 75 claims from workers who alleged asbestosis as a result of the use of Kaylo.

Doctor, in your opinion wouldn't that type of information have demonstrated that the use of Kaylo may create a hazardous situation?

MR. HENDRICKSON: Just for the record, has this been admitted against OCF?

MR. PATRICK: I believe it has been.

A. I couldn't come to a conclusion about that without having reviewed the other potential exposures. I don't know what these individuals did, what kind of exposure to Kaylo they had, what kind of exposure to other asbestos-containing materials they had. It would be impossible from simply a list of names associated with asbestosis to determine

whether or not Kaylo had anything to do with the asbestosis. It would have to be evaluated.

Q. Well, let me just ask you to assume that there were instances where persons exposed to Kaylo developed asbestos-related diseases, and that was from exposures to Kaylo without using respiratory equipment.

If you make that assumption, would you have an opinion that those individuals should have been advised to use respiratory equipment or to handle the substance with care?

MR. HENDRICKSON: I'm going to interpose an objection just because I understand where you're going with this and I understand what you're trying to demonstrate, that these folks, evidently the document purports that they were Workers' Compensation claims initiated against OCF by workers handling insulation products. It doesn't necessarily mean that they were handling Kaylo, and also it doesn't necessarily indicate what levels of exposure they had prior to the year they filed their claim.

I think your hypothetical is okay, but you have to add a few more facts so that he can maybe answer it more directly because I think

you're leaving out some of the crucial facts that he may have to take into consideration to give a fair answer to your question. So I'm going to object to the question as it's stated. He can answer if he can. You can either get an answer that way or you can rephrase it, either way you want to.

Q. If you can answer the question.

A. I can't remember it.

Q. Let me see if I can rephrase the question.

Doctor, let's assume that 75 individuals over the course of years between '55 and '73 developed asbestosis and made Workers' Compensation claims against Owens-Corning Fiberglass. Doesn't the fact that those individuals, assuming they became sick from asbestosis, doesn't that demonstrate that either the TLV for asbestos that was in existence in that time was either being exceeded or the TLV was not safe?

MR. HENDRICKSON: Note my objection.

A. Can we take that question apart? With regard to whether it was exceeded, I don't know if it was exceeded or not, so you want me to assume that it was exceeded?

Q. Well, I just want you to assume that these individual workers using Kaylo got sick from asbestosis. Make that assumption.

A. And it's caused by Kaylo?

Q. And it's caused by Kaylo.

A. And that's been determined using appropriate scientific method and there's no question about that?

Q. That's correct.

A. Okay.

Q. So my question is if those individuals were getting sick using Kaylo, that either their exposures exceeded the TLV or that the TLV was unsafe.

A. Okay, I have to take that apart. With regard to exceeding the TLV, you want me to assume that it did exceed the TLV?

Q. Well, not exactly. I'm just asking if you take it apart, that one of the two instances must have occurred, that either the TLV was unsafe and that these people were getting asbestosis from exposures to asbestos below the TLV, or that it was safe and that the TLV had to be exceeded. Would you agree with that statement?

MR. HENDRICKSON: I would object to that.

Again, Charles, I don't think you can do it that way, not for guys on this list anyway, because the very dates -- I mean I don't think you can do it that way.

MR. PATRICK: Let me see if I can back up.

MR. HENDRICKSON: I mean maybe you can ask it that way. He can certainly give you an answer, but the answer I don't think is going to have all the facts in there necessary for him to answer it, again just because of the very nature of what these guys were doing and weren't doing. I really think the question may be a little bit unfair.

Q. If you can answer it. Can you answer that question?

A. I can answer it taking it as two separate questions. If they got asbestosis, does it mean that the TLV was not appropriate, I have to evaluate that. Not necessarily. It depends on whether or not the TLV was exceeded and by how much. All of those factors would have to be used in the evaluation of whether or not that TLV was safe or not safe.

And the second one is if it was exceeded

by some order of magnitude, is it possible that that exposure could result in an asbestosis. That's a possibility.

Q. Well, it's certainly a possibility as evidenced by that document. Wouldn't you agree?

MR. HENDRICKSON: I object to the question.

A. Well, that's not fair because you made me make all those assumptions. This document does not confirm that Kaylo causes asbestosis.

MR. HENDRICKSON: Did you want to mark this?

MR. PATRICK: Yes.

(Whereupon, Plaintiff's Exhibit Number 3 was marked for identification.)

BY MR. PATRICK:

Q. Doctor, let me show you another exhibit which has been admitted into evidence as our Exhibit 556, as to Owens-Corning Fiberglass. And I don't mean to dwell on this, but you have reviewed the Saranac documents previously. I'm trying to find some reference point in terms of asbestos. And this concerns Kaylo, it's dated September 17, 1963. I ask you if you have ever seen that document before?

MR. PATRICK: And after you have reviewed

it, let's have that marked as Exhibit Number 4.

A. Could I ask a question? I can't read the top. Whose memo is this?

Q. This is one from Owens-Corning Fiberglass.

A. I don't think I've ever seen this document.

Q. Doctor, let me just ask you, it's stated in the second sentence, "Asbestos (as found in Kaylo) when breathed into the lungs causes asbestosis, which often leads to lung cancer." Doctor, is that in conformance with your opinion after reviewing the Saranac Kaylo documents?

MR. HENDRICKSON: Well, once again, the Saranac Kaylo documents stop well before the date of this memo, which is 1963, so I would object to relating this document back to those studies, when the final Saranac Laboratory report was printed in 1955. This document is clearly eight years after that fact, so whatever the general state of the art was in 1963, it probably evolved since that time, and so I'm not sure it's fair to relate it -- I mean if you're relating back to the Saranac studies, I don't think that's fair. If you're

asking him if today his knowledge is embodied in that sentence, then maybe that's a fair question.

Or maybe even if you ask if that was his -- I don't know how old you were in 1963, I'm not trying to date you -- maybe if you ask him if that was his view in 1963, maybe that's fair. But I mean to relate them back I don't think is fair.

Q. Weren't you at Drake in 1963?

A. Yes.

Q. You were a student at that time, so I don't think that would be a fair question.

But I think the question would be fair: Doctor, have you come to that opinion today, in 1994? If asbestos as found in Kaylo were to be breathed into the lungs it would cause asbestosis, which would lead to lung cancer?

A. That's not what it says.

Q. I understand that.

A. But your question is different than what this sentence says.

Q. Well, let's read it verbatim. Would your opinion in 1994, today, be that, quote, "Asbestos (as found in Kaylo) when breathed into the lungs

causes asbestosis, which often leads to lung cancer"?

A. I don't have an opinion about that because I haven't evaluated that, so at the present time I don't have an opinion about that.

Q. So it would be fair to say that no one with Owens-Illinois, no attorney for Owens-Illinois or Westinghouse has shown you any documents pertaining to Kaylo as it may relate to human experience since documents dated 1952, '53, '54 as it may pertain to Kaylo at Saranac?

A. That's not really a fair characterization of what I have done. I haven't looked at the human experience. The only thing I have looked at is the animal testing that was done at Saranac Lake and the industrial hygiene testing that was done at the Kaylo, or the Owens-Illinois plants. That's all I've looked at. I haven't looked at human experience or human epidemiology or any other human information other than that that I described.

Q. I believe in your prior testimony after reviewing the Saranac documents it was your opinion that if workers were exposed to asbestos below the then existing threshold limit value of 5 million particles per cubic foot, that would have been a

safe exposure. Is that correct?

A. I don't think that's a fair characterization of my testimony, either. I think it is put in the context of time. And was it reasonable based upon the testing, based upon the animal experimentation to come to a conclusion that that was a level that would be safe in the workplace, and my answer to that was yes, based upon that chronology, that time and that information.

Q. Well, let me ask you to assume that 11 years after the final report of Saranac to Owens-Illinois on Kaylo it was demonstrated that workers exposed to Kaylo would develop asbestosis. Wouldn't you agree that at that point in time the TLV was called into question?

MR. HENDRICKSON: He's not referring to that document now (indicates). Correct?

Q. Not in particular.

A. No, you are referring to this document, aren't you?

Q. Yes, 11 years after 1952. 1963.

A. I haven't evaluated that timeframe, so I don't know the answer to that question. I haven't been asked to look at that.

Q. Doctor, if it's demonstrated that

exposed to cyanide in small doses, we're exposed to salt, we're exposed to various substances that may be toxic, including asbestos; and that we have to keep all of this in context. Is that a fair statement of your testimony?

A. Well, I think it's an unfair way to ask that question because you started out by saying toxic substances. I don't think that's what my testimony was.

I think what my testimony was is that we're exposed to chemicals all the time, that chemicals are basically everything in your life other than light, radiation and sound waves. And if the exposure is high enough, resulting in a dose that is sufficient to cause an adverse effect, then virtually all substances can be toxic at some level of exposure. So chemicals you're exposed to, and if the exposure is high enough, it can result in toxicity.

Q. Would you agree that for a particular substance when used for the purpose for which it is intended that there is a reasonable likelihood that a hazardous dose would be exceeded, that that substance should either bear a warning label or should be regulated in some fashion?

MR. HENDRICKSON: Again I'm going to just interpose an objection.

Go ahead.

A. I'm not sure I understand that question.

Q. Well, I think one of the examples that was used in trial approximately a year ago is you're familiar with Tegrin shampoo?

A. Yes, I am.

Q. And Tegrin shampoo contains coal tar, correct?

A. That's correct.

Q. Coal tar has been implicated as being a carcinogen, isn't that correct?

A. Constituents of coal tar, some constituents, yes.

Q. In fact, if you look at the history of known carcinogens, the constituents of coal tar were some of the first implicated in causing cancer.

A. The constituents of coal tar, not coal tar, but soot from chimneys, the answer is yes.

Q. The chimney sweeps in England were described as having cancer of the scrotum as early as the 1700s, correct?

A. That's correct.

Q. But yet, if we look at Tegrin shampoo, we

can find that coal tar is an ingredient of Tegrin shampoo. Correct?

A. Let me correct the 1700 statement first. Not all chimney sweeps developed scrotal cancer. Some did. It depended on the exposure. And do we still use coal tar today in shampoo, is that the question?

Q. Yes.

A. That's correct.

Q. And I believe your testimony was that there is no warning label on Tegrin shampoo. Correct?

A. That's correct.

Q. Even though it's indicated as one of the ingredients on the side of the bottle, there is no specific warning label?

A. There is the no specific warning with regard to cancer, that's correct.

Q. And if you looked at the medical literature on Tegrin shampoo or if you looked at all of the reports to the Food and Drug Administration for Tegrin shampoo, I take it you would not find any adverse, or very few, if any, adverse consequences from the use of Tegrin shampoo or the FDA wouldn't allow its general use to the public. Correct?

MR. HENDRICKSON: I object to the form of the question.

You can answer.

A. Well, there are several questions in that question. First of all, is there a reporting database with regard to Tegrin shampoo? I don't know the answer to that. And does FDA regulate substances that are used by consumers? The answer is yes.

Q. But you are not aware that the FDA has a database whereby doctors or physicians or in this case maybe dermatologists may say we've had the case of someone using Tegrin shampoo and they have developed malignant melanoma from the use of it. Are you aware of such a database?

A. I haven't looked for such a database. I don't know that one does or does not exist.

Q. Well, as a general proposition wouldn't you agree with the hypothetical that if the use of Tegrin shampoo caused 10 percent of those people who used it to develop malignant melanoma, then there would be a hazard created such that either Tegrin shampoo should be removed from the market or that a warning be placed on it stating that there was a hazard created?

A. If Tegrin shampoo resulted in cancer in 10 percent of the people who used it, would it be removed from the market or would there be a warning placed on it?

Q. Correct. This is a general principle of public health policy.

A. I suspect it would probably be restricted in use or eliminated from use to either eliminate the exposure or prevent the adverse effect occurring as a result of that exposure.

MR. HENDRICKSON: You're referring to today's standard, right?

MR. PATRICK: Yes.

Q. All right. Now, are you familiar with the name of Dr. Irwin J. Selikoff?

A. I am.

Q. Are you familiar with his studies of insulation workers using asbestos-containing thermal insulation?

A. I haven't looked at those for some time. I'm not really familiar with those.

Q. Are you aware that Dr. Selikoff in some of his studies reported that 10 percent of asbestos insulation workers developed malignant mesothelioma?

A. I am not.

Q. Are you aware that the Environmental Protection Agency based upon the data from Dr. Selikoff as well as many other investigators recommended a ban on the use of asbestos in 1989?

A. I am generally familiar with the restrictions on the use of asbestos. I don't recall the legislative history or the record of decision with regard to that and how the EPA made that decision.

Q. As a toxicologist can you tell me, is it possible for you to tell me what the 10 most toxic substances would be that we are exposed to?

A. It depends on the use. It depends on what the exposure is. It depends on what the effect is.

Q. So there is no way for you to say just in the abstract, to list 10 toxic substances and say we've got cyanide, we've got polyvinyl chloride, we've got PCB, we have DDT and asbestos and say, all right, DDT is number one; is it possible for you to do that as a toxicologist?

A. Well, I could certainly use the methodology whereby, for example, I could go to the poison control center annual report. I think if you look in there, probably aspirin or Tylenol would

emerge as those substances that kill more children than any other in a year's time. It would probably also be related to furniture polish and other materials.

Now I'm giving you sort of a generality. I don't know what that actual ranking is, but I would have to go to the poison control center and I would look at the annual report of the poison control centers as to the mortality and morbidity that is reported as a result of inquiries or information provided to the poison control centers.

Q. So you would have to look at the number of people affected by the use of a substance, would that be an appropriate piece of information that you would use in coming to a determination of what is the most hazardous substance?

A. Well, again you have to look at the definition of a hazard. A hazard is determined by use, so the number of people that you look at, they would have to be the people who are relevant to that particular use. It would certainly be important to look at a number of people, that would certainly be one factor in evaluating the potential hazard.

Q. There are approximately 250 million people in the United States. Do you know?

A. That's probably about right for 1994.

Q. And the likelihood is that most people, if not all people, at some point in their life have taken aspirin. Correct?

A. I don't know the answer to that, but I expect most probably have.

Q. So you have somewhere in the neighborhood of, somewhere between 240- to 250 million people who have been exposed or who have taken aspirin. And if you look at the number of people exposed -- let me withdraw that question.

One aspirin a day is not going to hurt you. Would you agree with that?

A. I would agree with that.

Q. And I believe it's your prior testimony that two aspirins a day may relieve a headache.

A. And one may prevent a second heart attack.

Q. And 30 may relieve someone's rheumatitis -- or rheumatological problems. Correct?

A. Correct.

Q. But 90 a day is going to kill you.

A. That's correct.

Q. So in that instance you have the

dose-response relationship. Correct?

A. Correct.

Q. And if you have 250 million people who are taking aspirin, just by the sheer number of people who are exposed the likelihood is that you'll have a large number of people who have had an adverse reaction or who have died from taking aspirin. Correct?

A. No, I don't come to that conclusion at all.

Q. What conclusion do you come to?

A. With regard to what?

Q. With regard to aspirin and in your prior testimony. You in your prior testimony were talking and using aspirin as a substance that we were exposed to in our normal, ordinary lives and trying to put that into context with exposure to other chemicals and other substances. And I'm trying to just discern the point that you were trying to make then, and which you may make in West Virginia if you are asked to testify.

A. The point is that there is a dose-response relationship and that if you're exposed to enough aspirin, it can be harmful. It can in fact be lethal. And if you're exposed to

lesser concentrations, it either has no effect or in some cases can have a beneficial effect. So it's a dose-response relationship.

Q. If you take that same analogy, or take the analogy of aspirin and use it with asbestos, wouldn't you agree that exposures below the threshold limit value for asbestos, and take a year, 1963, 5 million particles per cubic foot were still capable of causing malignant mesothelioma?

A. No, I wouldn't agree with that.

Q. You would not agree with that?

A. No.

Q. Are you familiar with the study by Dr. Christopher Wagner in 1960 among workers or persons exposed to asbestos in South Africa who developed malignant mesothelioma from living next to an asbestos mine?

A. I don't particularly recall that, no.

Q. Have you testified at trial during the year 1994?

A. Yes.

Q. Where have you testified?

MR. HENDRICKSON: You mean in an asbestos case or just in general?

Q. Any litigation, any trial testimony.

A. I'm trying to recall the most recent with regard to asbestos. I can't remember. Oh, I'm sorry, it was Louisville, Kentucky.

Q. And when was this?

A. Probably about a month ago.

Q. And this was an asbestos case?

A. Yes, sir.

Q. And it was on behalf of attorneys representing Owens-Illinois?

A. Yes, sir.

Q. Do you recall the name of the defense lawyer?

A. I do.

Q. Who was the defense lawyer?

A. Peggy Whipple.

Q. Is this the only time that you've testified in an asbestos-related disease case in 1994 in trial?

A. Yes, sir, I believe so.

Q. Now, have you testified on any other issue in court in 1994?

A. I have.

Q. And when was this?

A. I'm not sure I can give you the exact dates. It would have been earlier this year in Lake

Charles, Louisiana, concerning phenyl propanolamine.

Q. Okay. I think you may have to spell that, if you can.

A. I think I can. P-H-E-N-Y-L P-R-O-P-A-N-O-L-A-M-I-N-E.

Q. And what is phenyl propanomaline?

A. I'm sorry, that's not what I said, but it's close.

MR. HENDRICKSON: Whatever you said, what is it?

Q. Whatever you said, what is it? That's correct.

A. It's a drug.

Q. And who were you testifying on behalf of in that case?

A. Well, again I don't testify on behalf of anybody. It was a pharmacist who had dispensed a remedy that contained phenyl propanolamine as well as other materials that caused someone allegedly to become sick and to have a seizure as a result of that exposure.

Q. And you were testifying on behalf of the attorneys who were representing the pharmacist in that case?

A. Again, I reviewed the pharmacology of

that material and was asked to describe the pharmacology and whether that material or materials could cause a seizure as a result of the use of those materials.

Q. Who was the defense lawyer in that case?

A. James Nieset.

Q. Do you remember the name of the plaintiff's attorney?

A. I do not.

Q. Does the name Billy Baggett have any -- refresh your recollection at all?

A. No.

Q. Were you in State Court or in Federal Court?

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

Q. Do you remember the name of the judge?

A. I do not.

Q. Any other times that you have testified in court on any issue this year?

A. Those are the ones I can recall.

Q. In your prior testimony you had indicated that you consult with or have testified for -- you have consulted for chemical companies or organizations concerned with chemicals. Is that correct?

A. I have consulted with chemical companies and with some organizations that deal with chemicals, that's correct.

Q. During the year 1993 were you called to testify or asked to testify in trial on behalf of any chemical company?

A. I'm sure I was.

Q. Do you recall?

A. I can't recall 1993. I just don't recall.

Q. Have you ever testified for a plaintiff in any type of case alleging injury from exposure to a toxic substance?

A. Well, you keep using that term toxic substance. I'm going to keep having a problem with it.

Q. I don't know how else to phrase it.

A. Well, with regard to exposure to a substance that has caused some harm. The answer is yes, I have.

Q. And on how many occasions have you testified on behalf of a plaintiff in one of these cases?

A. Again I don't see myself as testifying on behalf of a plaintiff or a defendant. I have

certainly evaluated exposures, conditions for plaintiffs, I could probably recall a half dozen times.

Q. When was the last time that you testified, and I'm sorry, I don't know how else to phrase it, at the request of an attorney representing a plaintiff who alleged an injury from a substance that may have become toxic?

A. Substances only become toxic if the dose is high enough. I will estimate 1992, thereabouts.

Q. Do you recall the circumstances of that particular case?

A. That was exposure to mineral spirits.

Q. Did someone drink mineral spirits?

A. Someone used mineral spirits in the workplace.

Q. What was the alleged injury?

A. A skin injury, an injury to the skin.

Q. In the asbestos context have you ever testified for a plaintiff alleging injury from exposure to asbestos?

A. I don't recall any specific testimony with regard to a specific injury in an individual with regard to asbestos.

Q. Have you written anything concerning

asbestos from a medical standpoint, a published medical article?

A. I don't think so.

Q. Have you done any studies that may have looked at the adverse effects of asbestos?

A. Studies in the laboratory?

Q. In the laboratory, correct.

A. I have not.

Q. And have you reviewed the medical literature concerning the health effects of asbestos?

A. I have.

Q. Have you reviewed that in any detail?

A. I have.

Q. Have you reviewed the epidemiological studies that may exist concerning asbestos?

A. I have, yes.

Q. Would you consider yourself an expert in that area?

A. I would consider myself to have expertise in the area of asbestos, yes.

Q. Other than reviewing the medical literature concerning asbestos, what other information have you gained that would allow you to be offered as an expert witness in that area?

A. I don't understand.

MR. HENDRICKSON: We're not offering him --

Q. Okay. I'm sorry, you're not being offered as an expert witness with respect to asbestos.

In preparation for your deposition today did you review any materials?

A. Anything specifically for today?

Q. Yes.

A. I did not.

Q. Did you bring with you anything today?

A. I did.

Q. What did you bring?

A. I brought a copy of my curriculum vitae.

Q. May I see that, please? Doctor, in looking at your CV under "Summary of Experience," you have from 1993 to the present Academy of Toxicological Sciences, Board of Directors. What is the Academy of Toxicological Sciences?

A. It's a board-certifying academy that certifies toxicologists.

Q. Is that a national organization?

A. It is.

Q. And from 1991 until the present you are

listed as a science advisory board consultant, Environmental Health Committee, United States Environmental Protection Agency. What are your duties in that position as a consultant?

A. To provide advice and counsel to EPA concerning various issues dealing with environmental health and safety.

Q. When was the last time you were asked to provide consultation to that committee?

A. Probably six months ago.

Q. And do you recall what the advice was, what it concerned?

A. I do not.

Q. Doctor, are you familiar with a toxicologist by the name of Myron Mellman?

A. I am.

Q. How do you know Dr. Mellman?

A. I have seen books of which he is an editor.

Q. Have you ever met Dr. Mellman?

A. I don't think so.

Q. Do you have any opinion on his expertise in the area of toxicology?

A. I do not.

Q. Do you recall the name of the book that

you have seen by him?

A. It wasn't by him. It was a book edited by Dr. Mellman. I don't know.

Q. Now, on your CV under the category of industrial experience you list a number of organizations beginning with Occidental Oil, and you state "Worker safety in oil shale production." What do you mean by that?

A. I evaluated oil shale production and the effects that result from exposure to workers in oil shale production, made recommendations as to changes in that exposure to prevent adverse effects occurring as a result of that exposure.

Q. And with Shell Development Corporation, pesticide use and safety, what did you do there?

A. Presented a series of seminars to show with regard to research from my laboratory that -- discovered that cholinesterase inhibitors could result in growth promotion and the effects of exposure to these materials in utero and the enhanced body size or body weight that could result from that in-utero exposure.

Q. What would be the exposure that one would have to those substances or chemicals in everyday, ordinary life?

A. I'm not sure I understand your question. It was to develop a food additive to enhance livestock production. Cholinesterases are used as pesticides around your home. There's malathion, parathion.

Q. Petrolite Corporation, health assessment of waste incineration methods, what was that about?

A. For the secretary of the Department of Environmental Regulation I reviewed the use of waste fuels for the production of energy sort of as a co-generation facility and the consequences of exposure to the gases and other materials that would be released into the environment as a result of the burning of those wastes.

Q. I'm going to skip down a little bit to sort of about the middle of the page, Texaco toxicology consultant. Are you still a consultant for Texaco?

A. I am.

Q. And when was the last time you gave advice to Texaco on any matter?

A. Probably six months ago.

Q. What is the general area of your consulting activities with Texaco?

A. Toxicity testing, risk assessment,

materials safety data sheets. Those would be the general areas.

Q. Do you consider yourself an expert in risk assessment?

A. I do.

Q. Have you read any medical or other type of literature concerning a risk assessment pertaining to asbestos specifically?

A. Ever?

Q. Yes.

A. Yes.

Q. Have you ever done a risk assessment for asbestos?

A. I've done a lot of risk assessments. I don't know if I've ever done one for asbestos or not. I don't specifically recall one for asbestos.

Q. Are you familiar with the risk assessment data that was presented to the Environmental Protection Agency for the use of asbestos in buildings?

A. And when was that?

Q. It's been ongoing, but probably five or six years ago now.

A. I certainly recall seeing information about that. I don't know if we're talking about the

same thing or not.

Q. Something along the lines that if you're exposed to asbestos in a building at certain concentrations you may have 20 cancers per million, as opposed to being exposed to some other substance and developing 50 cancers per million. Have you seen data along those lines?

A. I have seen data like that, but I don't think it's what you say it is. It is an estimate of the upper bound of risk. It doesn't mean that there will be cancer. It's the use of a cancer potency factor to evaluate the upper bound. It may be zero. So yes, there is certainly data like that and EPA does use that methodology for estimating risks for exposure to asbestos as well as other materials.

Q. Are you familiar with that data as it was used by the Environmental Protection Agency for asbestos?

A. For what?

Q. For asbestos.

A. But for what reason, for remediation or --

Q. For establishing regulations on the use or removal of asbestos in public buildings.

A. Yes.

Q. You are also listed as having industrial experience with the Chemical Manufacturers Association, technical review of health effects of PCBs. What is that?

A. I participated in the evaluation of the human health effects of PCBs for evaluating the risks associated with exposure in the environment, and specifically for the evaluation of risks associated with exposure to soil and surface levels of PCBs.

Q. And then you are listed as having experience with the American Petroleum Institute, comments to EPA concerning Resource Conservation Recovery Act. What was that?

A. I reviewed the recommendations for the characterization of wastes in the hazardous category and provided comment about the characteristics that were used to evaluate the hazard for the hazardous constituents in the Resource Conservation Recovery Act.

Q. I take it from this type of experience you were retained by the API to make comments to the EPA. Is that correct?

A. That's correct.

Q. Do you have a contract with the API?

A. I do not.
Q. Did you have a contract at that time?
A. No, I did not.
Q. Was this for compensation?
A. It was.
Q. Do you recall how much you received for that particular consultative role?
A. It was about $1,000.
Q. You have a private practice of toxicology, do you not?
A. I do.
Q. Is this a corporation or a partnership or how is this practice set up?
A. It's me as an individual.
Q. You are not incorporated?
A. I am not.

MR. PATRICK: Why don't we take a break?

(Brief recess.)

MR. PATRICK: All right. Let's go back on the record, if he could.

BY MR. PATRICK:

Q. Doctor, I'm going to make this CV an exhibit to the deposition, make it the next number.

If I turn to the end, Page 36, it's under Books/Monographs/Reports, and you have here

"toxicant profile" on various substances. First of all, what is a toxicant profile?

A. It is a profile of the physical-chemical properties, the toxicity, the general knowledge of a toxicant.

Q. The various problems that may be caused by exposure to this toxic substance?

A. That's one aspect. The occurrence, where it is, what it does, where it goes, its physical-chemical properties, whether it's a gas, liquid, solid, water solubility.

Q. You have one here for trichloroethylene. Is that commonly known as TCE?

A. Yes, sir.

Q. In your opinion is TCE a carcinogen?

A. A human carcinogen?

Q. Human carcinogen.

A. In my opinion trichloroethylene is not a human carcinogen.

Q. Has it been demonstrated to be an animal carcinogen?

A. It has been demonstrated to be an animal carcinogen, but the present knowledge regarding its mechanism of action indicates that it is an animal carcinogen in mice, for example, because it causes

peroxisome proliferation. And because of the high levels that are used, it produces a liver tumor and the consensus would be that that is a unique species response, not extrapolatable to people, and perhaps not even extrapolatable from one species to another, that is, from mice to rats, for example, or mice to other species.

Q. Well, you have here profiles on alkyl benzenes, carbon tetrachloride, methylene chloride, tetrachloroethane, trichloroethylene, vinyl chloride. In your opinion are any one of these substances or chemicals carcinogenic? You told us about TCE.

A. In animals or people?

Q. In humans.

A. Well, could we go down the list?

Q. Sure.

A. Vinyl chloride is a designated a human cancer-causing agent.

Q. Let me stop you right there. Designated by whom?

A. By the Environmental Protection Agency. Under some circumstances of exposure it can increase the risk of cancer in humans and under some circumstances of exposure it may be able to produce

a very specific form of cancer, an angiosarcoma.

The other substances, methylene chloride, tetrachloroethane,alkyl benzenes -- I'm sorry, did I get them all?

Q. Methylene chloride.

A. -- methylene chloride, are not designated human cancer-causing agents. Some of them are cancer causing in laboratory animals but, for example, methylene chloride again has a unique metabolic response in the animals in which it's tested and it is not likely a human carcinogen and probably not a carcinogen in other species based upon the metabolism of methylene chloride, that is, its much more rapid metabolism leading to reactive intermediates that are not readily detoxified that leads to the response in some species.

So none of the others are designated human cancer-causing agents and some of them would be suspect as to the relevance of that response to a risk in humans.

Q. How many substances have been designated by the Environmental Protection Agency as being able to cause cancer in human beings?

A. As Class A designated human carcinogens?

Q. Class A.

A. I don't know the answer to that. It's probably 20, 30. That's the range. I don't know the exact number.

Q. Do you know how the EPA goes about classifying these agents as carcinogens and then putting them in these categories such as Class A and whatever classes may exist?

A. I do.

Q. How do they do that?

A. For Class A you have to have sufficient evidence in humans that it can cause cancer. For the other classes, there is varying confidence or varying amounts of information, and in animals that determines whether it's a B1, a B2 or a C.

Q. Asbestos is a Class A carcinogen as designated by the EPA, is it not?

A. It is.

Q. And you may have told me this already. Is vinyl chloride designated as a Class A carcinogen?

A. It is.

Q. I see you've written some papers on the health effects of cocaine.

A. I have.

Q. And, of course, you're always asked this:

You were involved in the autopsy of Elvis Presley?

A. Yes, sir.

Q. Was cocaine -- am I allowed to ask these questions? I don't know, is that subject to -- is it a matter of public record?

A. Some of it is.

Q. Was cocaine one of the agents identified in his body?

A. I would have to go back and look. I don't remember cocaine as one of the specific drugs. But there were certainly lots of others.

Q. You testified in some type of proceeding related to the autopsy of Elvis Presley, correct?

A. Yes, sir.

Q. What was that?

A. It was before the Medical Examiner's Board of the State of Tennessee as to the usual course of medical practice with regard to the use of drugs and the pharmacological properties of those drugs.

Q. Did you testify concerning the cause of his death or concerning whether or not some other individual may have contributed to his death?

A. I testified concerning the cause of his death.

Q. Wasn't there some question subsequently as to whether or not an individual may have prescribed substances that may have contributed to his death?

A. Yes, there was.

Q. Did you participate in that at all?

A. I did.

Q. Is that public, a matter of public record?

A. It is.

Q. Was that a physician for him?

A. It was.

Q. Do you remember his name?

A. Dr. Nikapolous.

Q. What was the result of that hearing or trial with respect to that individual?

A. I'm not sure I remember all of the results of the trial. I do know that some of his privileges to practice, certainly with regard to dispensing controlled substances, was withdrawn.

Q. And you were asked to testify by the State of Tennessee?

A. I'm sorry?

Q. Were you asked to testify by the State of Tennessee?

A. Yes, sir.

Q. I mean you were not asked to testify on behalf of anyone associated with that physician, I take it.

A. No, sir.

Q. Doctor, I don't have too much more. Let me just ask you a few other things. You have as part of your experience an American Bar Association short course concerning the role of expert testimony in environmental litigations. When was that approximately?

A. It was the 50th anniversary of the American Bar Association meeting. I think that was in Atlanta, probably about 1981.

Q. Was it that long ago?

A. Maybe it wasn't that long ago.

Q. It was in Atlanta, though?

A. It was in Atlanta, yes. Maybe it was the middle '80s.

Q. I think Chief Justice Berger was still on the Supreme Court, because I believe he spoke. I don't recall. But do you recall Justice Berger being there?

A. I do not.

Q. Do you still maintain an outline of that,

of your talk or presentation at that particular seminar?

A. I do not.

Q. Do you recall what was discussed at that seminar?

A. Yes.

Q. Can you tell me?

A. Yes, and I will. I'm trying to recall. It was with regard to the exposure to some polychlorinated hydrocarbons and the evaluation of that exposure and potential risks associated with environmental exposure to those substances.

Q. Did you speak about how an expert should present his testimony or how an attorney should put an expert on the stand in a case?

A. I did not.

Q. In your prior testimony I believe you stated that for consultative purposes and for trial I believe you charge $175 an hour?

A. Yes, sir.

Q. Is that still the same rate today?

A. Yes, sir.

Q. And what percentage of your time professionally is spent with private consultations, private testimony, as opposed to your work with the

University of Florida?

A. I have to sort of separate that question out.

Q. Answer it any way you feel that you can.

A. With regard to evaluating or testifying or involved with lawyers it probably varies, 15, 20 percent, 25 percent depending on the year. The remainder of that time up to about 40 or 50 percent would be in consultation for health and safety issues at the Environmental Protection Agency, emergency response responsibilities, providing consultation to emergency rooms. Those would be the general broad areas of consultation.

Q. Are you familiar with a substance known as chlordimaform?

A. I am not.

Q. Doctor, can you give us your definition of a threshold limit value?

A. Threshold limit value is that concentration that you can be exposed to for eight hours a day, five days a week for an entire working life-time.

Q. Without adverse effect?

A. Without adverse effects.

Q. In your definition of a TLV would

exposures below the TLV ensure safety against adverse effects from a particular substance?

A. It would.

Q. Do you know if your opinion is stated in the definition of a threshold limit value by the ACGIH?

A. I don't know the specific answer to that. I am more familiar with the permissible exposure limit, and it certainly is within the permissible exposure limit. I would have to review the threshold limit value.

Q. In other words, in your opinion the TLV, or let's say permissible exposure limit, would be a, quote, "safe level of exposure" to a particular substance?

A. Yes, sir.

Q. Would you agree that if there was a substantial likelihood that a person was being exposed to a substance at levels above, and significantly above the threshold limit value, that that person should be advised of his or her exposure?

A. It would depend for how long, how high above the threshold limit value or the permissible exposure limit. It would depend on the

circumstances of exposure.

Q. Do you have any experience in industrial hygiene?

A. I do.

Q. Do you know whether or not if we were to have in this room a level of asbestos, ambient asbestos in the air, at the level of 5 million particles per cubic foot whether or not that level could be visible by the naked eye?

A. 5 million particles of asbestos?

Q. Per cubic foot.

A. And what would be the size of those particles?

Q. Well, somewhere between 5 and 20 microns in length and approximately a half a micron in width.

A. With that description I can't answer the question. I would have to look it up. I don't know at the present time.

Q. If you were to make reference to any body of work on asbestos as being authoritative, in other words, if you were going to try to get a question answered concerning the health effects of asbestos, where would you go to look personally?

A. I wouldn't have any single authoritative

source. I would go to a variety of places. I would go to the scientific literature, I would go to textbooks, I would go to electronic databases.

Q. Any particular authority in the field of asbestos-related diseases that you find authoritative?

A. No, sir.

Q. Is there any particular textbook or treatise that you might find authoritative?

A. Again I would have to look at the specific information and review it individually and independently.

Q. When were you first contacted about consultation for Owens-Illinois?

A. I don't know the answer to that. I would have to go back and look at my records. It's probably a couple years ago.

Q. Well, I believe you testified in preparation for the Baltimore consolidation in 1991, so maybe three or four years ago?

A. That sounds about right.

Q. And who made the first contact with you, if you recall?

A. I do. Gardner Duval.

Q. And who is Gardner Duval?

A. He's an attorney.
Q. Where, where does he practice?
A. I think Baltimore.
Q. Do you know an individual by the name of Bruce Shaw?
A. I do not.
Q. David Gray?
A. I do not.
MR. HENDRICKSON: You've met Bruce.
A. Oh, I'm sorry, I have met Bruce. He's the fellow from North Carolina?
Q. South Carolina.
A. South Carolina. I'm sorry, I have met him.
Q. How about David Gray? Does that refresh your recollection, that name?
A. I don't recall meeting David Gray.
Q. From Toledo, Ohio. Have you met any house counsel from Toledo, Ohio, for Owens-Illinois? If you recall.
A. The only person I recall is Peggy Whipple I think was in Toledo at one time. That's the only person I recall.
Q. Do you recall on how many occasions you met with attorneys representing Owens-Illinois for

purposes of discussing or reviewing the Saranac documents prior to the time of your first deposition in asbestos litigation?

A. Twice.

Q. Are you familiar with a doctor by the name of Harry Demopoulos?

A. I am not.

Q. Have you ever seen the testimony by a doctor by the name of Harry Demopoulos?

A. Not that I recall. I'm sorry, where is Harry Demopoulos?

Q. He is in Scarsdale, New York.

A. He was in a movie.

Q. That's correct.

A. That's how I know him.

Q. How do you know that?

A. Because I was watching this movie and I saw "Harry Demopoulos, M.D."

Q. Surely someone must have told you that. Were you watching the credits of the movie?

A. Sure. I don't remember the movie, but I remember him being in it.

Q. He was in several movies.

A. Okay. I only know one.

Q. Which one do you remember?

A. I can't remember the name of it.
Q. Sudden Impact?
A. Is that a murder mystery?
Q. It's the name of a Clint Eastwood film.
A. I think that's it. It's been -- 1979?
Q. It may have been. Like I say, he's been in several movies, so you may have seen him. Do you recall the role that he played?
A. No, I don't.
Q. Do you know if Harry Demopoulos has ever testified on the significance of the Saranac documents?
A. I do not.
Q. As I understand, the only understanding you have of Harry Demopoulos is as an actor in a movie?
A. Correct.
Q. Are you familiar with an expert by the name of Simino, a Dr. Simino, also from New York? I'm sorry, I can't remember his first name.
A. I'm sorry, I don't recall such an individual. I don't know him.
Q. How about a Dr. Keith Morgan?
A. No, sir.
Q. In your consultations with attorneys for

Owens-Illinois, were you ever provided transcripts of physicians or doctors who have reviewed the Saranac documents so you would see what their opinions may be as well?

A. No, sir.

Q. Have you ever read the deposition of a Dr. Garrett Schepers?

A. No, sir.

Q. Are you familiar with a Dr. Schepers?

A. Only from reviewing the Saranac documents and the publication.

Q. And I believe you reviewed the deposition of Dr. Willis Hazard.

A. I don't think he was a doctor, but yes, of Willis Hazard.

MR. PATRICK: I believe those are all the questions I have.

MR. HENDRICKSON: Do you have anything up in Pittsburgh?

MR. CARTER: Yes, just a couple.

CROSS-EXAMINATION

BY MR. CARTER:

Q. Other than Owens-Illinois and Westinghouse, what other companies have you consulted with regarding asbestos?

A. None.

Q. And I may have missed this because of the connection. Can you tell me how a substance makes it on the A list by the EPA in terms of human carcinogens?

A. I'm sorry, could we go back to that other question? The question you were asking me is with regard to any litigation, is that correct?

Q. No, it was broader than just litigation. It was just regarding asbestos.

A. I have certainly provided consultation about asbestos to companies other than Owens-Illinois and Westinghouse.

Q. Okay. Well, let me narrow that to manufacturers of asbestos-containing products, either present or past.

A. I'm sorry, I don't know who "other manufacturers" are. I'm not sure I can answer that.

Q. I guess if they didn't tell you they were making something or had made something, that would kind of answer the question.

A. Okay. I don't specifically recall having made anything specific.

Q. Okay. Now returning to the question I asked after that regarding the A list by the EPA for

human carcinogens, can you tell me how a substance makes the A list?

A. Yes. It is a review of the available scientific and medical information, and the review considers the human evidence and whether there is sufficient human evidence to designate a substance as a carcinogenic material.

Q. When the deposition first began, the designation of your testimony by Westinghouse was read and it included a term, that you were going to testify regarding all substances and including asbestos.

How do you understand that to fit into your testimony, the "all substances" phrase?

A. How do I see that fitting into my testimony?

Q. Yes.

A. Comparing asbestos to all other substances that based upon some level of exposure can be toxic or produce adverse effects, occur naturally or as a result of use in a variety of products. So it would be comparatively, comparing asbestos to all other substances.

MR. CARTER: I think that's all the questions I have.

REDIRECT EXAMINATION

BY MR. PATRICK:

Q. I just need to follow up because I just read the first portion of the statement. Let me just read the statement into the record and just make sure that the doctor agrees with this statement of testimony, and I'm going to read the whole thing again.

It says, "Raymond D. Harbison, M.S., Ph.D, practices pharmacology and toxicology. Dr. Harbison will testify about the evolution of toxicology and pharmacology as it relates to all substances, including asbestos. Dr. Harbison will offer expert testimony concerning all substances and how all substances are toxic at some level. Dr. Harbison will further opine how the field of toxicology has studied ways in which to utilize substances at safe levels.

"Dr. Harbison is not offered as an expert on asbestos or asbestos-containing products, accepted or purported asbestos hazards or the history of medical knowledge of asbestos."

Doctor, does that statement fairly and adequately give us an idea of what you will testify to at trial?

A. Yes, sir, it does.

MR. PATRICK: All right. Those are all the questions I have.

MR. CARTER: I do have one more, if you don't mind: I forgot to ask, have you either prepared any exhibits or any charts or graphs or other visual medium upon which you intend to rely or use at trial?

MR. HENDRICKSON: Let me answer that for him. He will have those. We haven't prepared exactly what we're going to use yet, and as the court has ruled, we have to show those to you all before we use them. But they're just going to be along the lines of the same types of things referred to in his other depositions, aspirin and how many is a safe level and how many will kill you, those kind of things. So it's nothing that hasn't been really covered in his prior testimony.

MR. PATRICK: Let me ask you this: Can you get for me the charts that he used in the prior case in 1993?

MR. HENDRICKSON: I'll be glad to.

MR. PATRICK: Would those be generally the same type of thing?

MR. HENDRICKSON: Generally the same ones. And I'll go even further than that. Once we get ready -- well, we've got to show them to you before we use them with the jury, the judge has ruled that, because the judge doesn't want us to, or your side, to flash something in front of the jury that one side or the other may find objectionable before someone testifies. Okay?

MR. PATRICK: I don't know and I haven't been involved in the West Virginia litigation, but I have been involved in some of the depositions. Is it his order that if there is going to be exhibits like this to be used that we would have the right to have a short deposition prior to his testimony, say the night before?

MR. HENDRICKSON: No, that has not been his ruling.

MR. PATRICK: Obviously I can't ask him about it now because he doesn't have them ready.

MR. HENDRICKSON: Right. Let's just say this. If there is something there that takes you all by surprise that you need to ask

questions about, then before he testifies I'll be glad to put him up for that.

MR. PATRICK: All right. Thank you.

MR. HENDRICKSON: Why don't we let him read it.

(Witness excused.)

(Whereupon, at 3:00 p.m. the taking of the deposition was concluded.)

CERTIFICATE OF OATH

STATE OF FLORIDA

COUNTY OF ALACHUA

I, Pamela A. Chorlog, a Notary Public of the State of Florida at Large, being duly authorized by Statute to administer oaths (Ch. 29, F.S., and Fla.R.Jud.Admin. 2.070), certify that the witness, RAYMOND D. HARBISON, M.S., PH.D., was first duly sworn by me to testify the whole truth.

Witness my hand and seal at Gainesville, Florida, this 4th day of April, 1994.

Pamela A. Chorlog

C.M., R.P.R., AND NOTARY PUBLIC
STATE OF FLORIDA AT LARGE
MY COMMISSION EXPIRES 2/16/94

PAMELA A. CHORLOG
MY COMMISSION # CC 343362
EXPIRES: February 16, 1998
Bonded Thru Notary Public Underwriters

CERTIFICATE WITH ACKNOWLEDGEMENT

I, Pamela A. Chorlog, C.M., Registered Professional Reporter, certify that I was authorized to and did stenographically report the foregoing deposition; and that the transcript is a true record of the testimony given by the witness.

I further certify that I am neither attorney or counsel for any of the parties, nor a relative or employee of any attorney or counsel connected herewith, nor financially interested in the event of this case.

I further certify that the original of this deposition was delivered to Mr. Patrick and was true and correct at the time of delivery.

Pamela A. Chorlog

PAMELA A. CHORLOG, CM, RPR

STATE OF FLORIDA
COUNTY OF ALACHUA

The foregoing certificate was acknowledged before me this 4th day of April, 1994, by Pamela A. Chorlog who is personally known to me.

Bernice Oosterhoudt

BERNICE OOSTERHOUDT, Notary Public, State of Florida
My Commission Expires 2/1/97

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Legal Aspects of Medical Practice

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DEPARTMENTS

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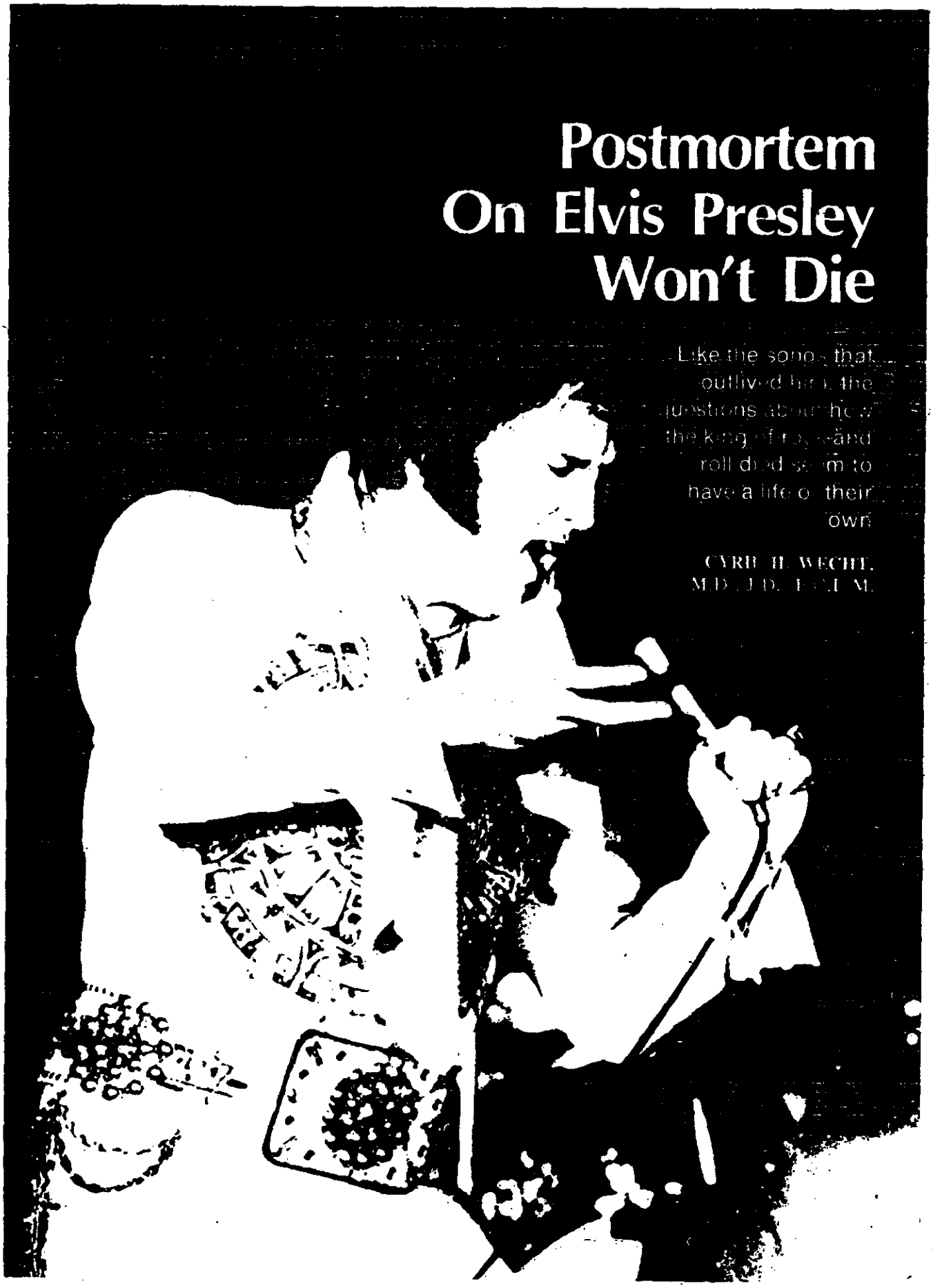
Postmortem On Elvis Presley Won't Die

Like the song that outlived him, the questions about how the king of rock and roll died seem to have a life of their own

CYRIL H. WECHT, M.D., J.D., F.C.L.M.



United Press International Photo

ELVIS PRESLEY is dead, but the questions that remain about how he died have uncorked a royal medicolegal controversy that seems made to order for the late singer's reputation as the king of rock and roll.

Two years ago, Memphis medical examiner Dr. Jerry T. Francisco told a press conference that Presley died of heart disease. After he took part in a private autopsy that had been requested by Presley's personal physician, Dr. Francisco said the death was due to "cardiac arrhythmia of undetermined cause." Two months later he concluded his investigation by describing the death as "HCVD associated with ASHD," hypertensive cardiovascular disease associated with atherosclerotic heart disease.

But today, two years later, the questions about Elvis Presley's death won't go away. Like the songs that outlived him, the doubts about the Presley diagnosis are replayed again and again.

The controversy has never faded far from the public eye. But recently it was drawn into sharper focus by a couple of new developments—each casting new doubt on the validity of Dr. Francisco's conclusion. One concerned a hearing that was scheduled to begin last month by the Tennessee Board of Medical Examiners. The board was looking into charges that Presley's physician, Dr. George Nichopoulos indiscriminately prescribed drugs for Presley.

The toxicology report shows that traces of nine drugs were discovered in Presley's body. They included codeine, morphine, methaqualone, diazepam metabolite, ethinamate, ethchlorvynol, pentobarbital, phenobarbital, and butabarbital.

The second event that renewed the controversy was a documentary aired earlier this year by ABC-TV that raised questions after the network did an extensive investigation about whether there was a coverup surrounding the circumstances of Presley's death. Prior to the program, I was contacted by ABC to find out if I would be willing to look through materials they had obtained to advise them of the cause and manner of death.

I cannot go so far as to say it was a coverup, as I did with the Kennedy assassination, because that indicates that you have knowledge of a deliberate attempt to hide things. I have no knowledge of such an attempt. However, ABC's characterization of the diagnosis as a coverup is not illogical or unfair. And I don't think its reporters can be accused of cheap journalism because they obtained expert medical testimony before arriving at their conclusions.

When you examine the toxicology report and see that nine central nervous system depressant drugs were found and when you see the levels that were discovered in the body, you have to conclude it was a drug death. If you take these nine drugs consider their effects on the central nervous and the depressive effects on cardiovascular spiratory functions—which is how they kill—there be no question.

Dr. Matthew J. Ellenhorn, who specializes in cal pharmacology and medical toxicology, told the drugs are "additive in their nature and contribute to what were probably the central nervous system depressant effects leading to the death of this individual." He added: "The prescribing of such a together is not within the scope of accepted medical standards and would cause harm in provoking a serious central nervous system depression if not death."

I don't think you can call Presley's death a typical classical multidrug overdose, however. That's because you don't have many cases—if any—involving nine different drugs, as was the case here. The classic overdose is when someone combines tranquilizers and alcohol or combines barbiturates, tranquilizers, and alcohol. But no traces of alcohol were found in Presley's system.

> **Sudden death in such circumstances would universally be considered a medical examiner's case**

Those who support the theory that Presley had built up a tolerance to these drugs are only speculating. They have no basis for saying that. And even if we assume there was some tolerance, with these nine drugs involved, you have an overwhelming quantity. You get a cumulative effect. It produces the pharmacological phenomenon known as synergism, which means you get more of a geometric than a simple arithmetic effect when you add one drug to another. One compounds the effect of the next. In this case, it's like adding two plus two plus two. Only you don't come up with six. You might come up with nine or 12 or even 15.

From the start, the case was not handled well by the medical examiner's office. Dr. Francisco claims he did not take charge of the case because an autopsy can only be ordered by the county district attorney. The DA can order an autopsy only if he suspects homicide or foul play, but the medical examiner can recommend one. Why didn't he?

Look at the facts. Here you have a 42-year-old man—and let's forget for the moment that he's Elvis Presley. A man is suddenly found dead and you have no reason to suspect any prior life-threatening cir-

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cumstances. That kind of situation would universally be a medical examiner's or a coroner's case.

But what happened? An autopsy was requested by Presley's physician, and it was performed at Baptist Memorial Hospital. I think Dr. Francisco's explanation of his role in the autopsy and why it was not

United Press International Photo

conducted at his office leaves much to be desired and raises questions about whether he acted properly in this situation. I believe the autopsy should have been performed at the medical examiner's office and that he could have recommended that to the DA. Instead, Dr. Francisco claims he did not want to transport Presley's body across the street from the hospital to his office because a crowd was gathering outside. He tells us that the private autopsy was done under the auspices of the hospital and that he was only there as a consultant.

His inconsistencies here are unbelievable. On the one hand, he says it was not a case under the jurisdiction of the medical examiner's office. But immediately after the autopsy was performed, he held a news conference to give the cause of death. If it were not his case, why did he raise the point about his fearing the gathering crowd and the difficulty in transporting the body to his office? It's inconsistent.

There are other problems—not only with conclusions drawn from the autopsy—but with the procedures employed. For example, the sequence of events following the autopsy—the haste with which Dr. Francisco declared that Presley had died of heart disease—casts doubt on that diagnosis. The microscopic slides from the autopsy would have taken 24 or 48 hours to get back for study; similarly, the toxicology results that would indicate whether drugs were involved could not have been received. Yet, Dr. Francisco immediately held a news conference giving the cause of death as cardiac arrhythmia.

Cardiac arrhythmia is something a pathologist cannot see when he is doing an autopsy. There are only two ways he can make that diagnosis: in a live person with a stethoscope held to the chest and with an ECG. When you examine someone after death, nothing will tell you he had ventricular fibrillation when he died. For Dr. Francisco to be giving such a diagnosis when he had no information about his condition immediately prior to death and without the benefit of microscopic sections and toxicologic analyses is incredible.

Dr. Francisco advanced the argument that the size of Presley's heart, reportedly twice what it should have been, pointed to heart disease as a probable cause and that the arteries were 60% clogged. His heart supposedly weighed 550 gm. That is not twice normal size. A 400 gm heart for someone Presley's size would have been only slightly larger than the upper limits of the normal range, so it was nowhere near twice normal size. Moreover, an informed source has told me that Dr. Francisco based his finding that the arteries were 60% clogged in only a focal area of one coronary artery. That is not highly unusual to discover in a 42-year-old man. It is not an exciting finding in the absence of fresh hemorrhage into an atheromatous plaque or a recent thrombotic occlusion.

In the wake of the controversy surrounding the diagnosis, Dr. Francisco has refused to make the autopsy report public, arguing that because it was done privately, its findings are confidential. There may have been some justification to that initially—but not anymore. Formal legal proceedings are now underway against one of the doctors (Nichopoulos). The veil of confidentiality should be lifted.

It is unfortunate that Dr. Francisco has stuck to his contention that the findings of the autopsy must remain confidential. Perhaps he is embarrassed by the charges that he should have requested that the DA order an autopsy. This is one issue that continues to bob to the surface.

Dr. Francisco says the reason for the autopsy was that it was requested by Presley's physician. The doctor requested it because Presley's father reportedly mentioned that he was concerned about a threat to poison his son. When a man dies unexpectedly and suddenly after a threat has reportedly been made against his life, doesn't that raise the suspicion of foul play? ■

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1 (BACK75)
UI - 76016065
AU - Harbison RD ; Braude MC
TI - Overview of the perinatal narcotic addiction conference and future research goals in developmental pharmacology of abused drugs.
SO - Addict Dis 1975;2(1-2):1-5

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UI - 76016088
AU - Evans MA ; Stevens MW ; Mantilla-Plata B ; Harbison RD
TI - Drugs of abuse: teratogenic and mutagenic considerations.
SO - Addict Dis 1975;2(1-2):45-61

3 (MEDLINE)
UI - 92410995
AU - Boyd VL ; Kvamme P ; Harbison R ; Kadowitz PJ ; McNamara DB
TI - Actions of SQ 29,548 on contractile responses and arachidonic acid metabolism of intrapulmonary arteries, in vitro.
SO - Agents Actions 1992 Mar;35(3-4):280-8

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UI - 81130754
AU - Sumaya CV ; Harbison RW ; Britton HA
TI - Pneumococcal vaccine failures. Two case reports and review.
SO - Am J Dis Child 1981 Feb;135(2):155-8

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UI - 78163332
AU - Wilson JT ; Kasantikul V ; Harbison R ; Martin D
TI - Death in an adolescent following an overdose of acetaminophen and phenobarbital.
RF - REVIEW ARTICLE: 78 REFS.
SO - Am J Dis Child 1978 May;132(5):466-73

6 (MEDLINE)
UI - 92391653
AU - Roberts SM ; Munson JW ; James RC ; Harbison RD
TI - An assay for cocaethylene and other cocaine metabolites in liver using high-performance liquid chromatography.
SO - Anal Biochem 1992 May 1;202(2):256-61

7 (BACK80)
UI - 80196432
AU - Wood M ; Berman ML ; Harbison RD ; Hoyle P ; Phythyon JM ; Wood AJ
TI - Halothane-induced hepatic necrosis in triiodothyronine-pretreated rats.
SO - Anesthesiology 1980 Jun;52(6):470-6

Most articles about drugs + their effects - is that his specialty? Out of 111 articles - none asbestos related - none pneumoconosis GWI

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AU - Harbison RD ; Spratt JL
TI - Disappearance of plasma bilirubin fractions in the rat after phenobarbital.
SO - Arch Int Pharmacodyn Ther 1968 Mar;172(1):32-6

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UI - 89165777
AU - Harbison RG
TI - 'Intestinal surgery in gynaecological oncology' [letter]
SO - Aust N Z J Obstet Gynaecol 1988 Aug;28(3):242

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UI - 68003765
AU - Harbison RD ; Boerth RC ; Spratt JL
TI - Quantitative determination of free and conjugated bilirubin by diazo coupling and a liquid-extraction and column-chromatographic technique.
SO - Biochem J 1967 Sep;104(3):46C-47C

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UI - 92281565
AU - Roberts SM ; Roth L ; Harbison RD ; James RC
TI - Cocaethylene hepatotoxicity in mice.
SO - Biochem Pharmacol 1992 May 8;43(9):1989-95

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UI - 84079990
AU - Smith AC ; Berman ML ; James RC ; Harbison RD
TI - Characterization of hyperthyroidism enhancement of halothane-induced hepatotoxicity.
SO - Biochem Pharmacol 1983 Dec 1;32(23):3531-9

13 (BACK80)
UI - 82256659
AU - James RC ; Harbison RD
TI - Hepatic glutathione and hepatotoxicity: effects of cytochrome P-450 complexing compounds SKF 525-A, L-alpha acetylmethadol (LAAM), norLAAM, and piperonyl butoxide.
SO - Biochem Pharmacol 1982 May 15;31(10):1829-35

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UI - 81207361
AU - Fant M ; Speeg KV Jr ; Harper S ; Harbison RD
TI - Cholinergic elements in a human choriocarcinoma cell line.
SO - Biochem Pharmacol 1981 May 1;30(9):967-70

15 (BACK80)
UI - 81184058
AU - Smith AC ; Freeman RW ; Harbison RD
TI - Ethanol enhancement of cocaine-induced hepatotoxicity.
SO - Biochem Pharmacol 1981 Mar 1;30(5):453-8

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UI - 81281924
AU - Goodman DR ; Harbison RD
TI - Characterization of enzymatic acetylcholine synthesis by mouse brain, rat sperm, and purified carnitine acetyltransferase.
SO - Biochem Pharmacol 1981 Jun 15;30(12):1521-8

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UI - 81232322
AU - Freeman RW ; Harbison RD
TI - Hepatic periportal necrosis induced by chronic administration of cocaine.
SO - Biochem Pharmacol 1981 Apr 1;30(7):777-83

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UI - 76252826
AU - Bishop MR ; Sastry BV ; Schmidt DE ; Harbison RD
TI - Occurrence of choline acetyltransferase and acetylcholine and other quaternary ammonium compounds in mammalian spermatozoa.
SO - Biochem Pharmacol 1976 Jul 15;25(14):1617-22

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UI - 76231698
AU - Rama Sastry BV ; Olubadewo J ; Harbison RD ; Schmidt DE
TI - Human placental cholinergic system. Occurrence, distribution and variation with gestational age of acetylcholine in human placenta.
SO - Biochem Pharmacol 1976 Feb 15;25(4):425-31

20 (BACK80)
UI - 84205834
AU - Goodman DR ; Adatsi FK ; Harbison RD
TI - Evidence for the extreme overestimation of choline acetyltransferase in human sperm, human seminal plasma and rat heart: a case of mistaking carnitine acetyltransferase for choline acetyltransferase.
SO - Chem Biol Interact 1984 Apr;49(1-2):39-53

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AU - Harbison RD
TI - Acetaminophen as an aspirin substitute: is it safer?
SO - Chem Depend 1980;4(1-2):71-84

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TI - Acetaminophen as an aspirin substitute: is it safer?
SO - Chem Depend 1980;4(1-2):71-84

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UI - 85229280
AU - Harbison RW ; de Lemos RA ; Boldt DH
TI - Neonatal pneumonia.
SO - Compr Ther 1985 May;11(5):33-43

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UI - 85026878
AU - Teaf CM ; Freeman RW ; Harbison RD
TI - Cocaine-induced hepatotoxicity: lipid peroxidation as a possible mechanism.
SO - Drug Chem Toxicol 1984;7(4):383-96

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UI - 84207591
AU - James RC ; Freeman RW ; Harbison RD
TI - L-alpha-acetylmethadol-induced tissue alterations in mice.
SO - Drug Chem Toxicol 1984;7(1):91-112

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AU - Roberts SM ; Harbison RD ; James RC
TI - Inhibition by ethanol of the metabolism of cocaine to benzoylecgonine and ecgonine methyl ester in mouse and human liver.
SO - Drug Metab Dispos Biol Fate Chem 1993 May-Jun;21(3):537-41

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UI - 92201092
AU - Roberts SM ; Harbison RD ; James RC
TI - Human microsomal N-oxidative metabolism of cocaine.
SO - Drug Metab Dispos Biol Fate Chem 1991 Nov-Dec;19(6):1046-51

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UI - 81236161
AU - Freeman RW ; Uetrecht JP ; Woosley RL ; Oates JA ; Harbison RD
TI - Covalent binding of procainamide in vitro and in vivo to hepatic protein in mice.
SO - Drug Metab Dispos Biol Fate Chem 1981 May-Jun;9(3):188-92

29 (BACK66)
UI - 70241107
AU - Eling TE ; Harbison RD ; Becker BA ; Fouts JR
TI - Kinetic changes in microsomal drug metabolism with age and diphenylhydantoin treatment.
SO - Eur J Pharmacol 1970 Jul 1;11(1):101-8

30 (BACK80)
UI - 82165745
AU - Goodman DR ; James RC ; Harbison RD
TI - Placental toxicology.
RF - REVIEW ARTICLE: 68 REFS.
SO - Food Chem Toxicol 1982 Feb;20(1):123-8

31 (BACK89)
UI - 89276751
AU - Harbison RD ; Marino DJ ; Conaway CC ; Rubin LF ; Gandy J
TI - Chronic morpholine exposure of rats.
SO - Fundam Appl Toxicol 1989 Apr;12(3):491-507

32 (BACK80)
UI - 84209525
AU - Smith AC ; James RC ; Berman ML ; Harbison RD
TI - Paradoxical effects of perturbation of intracellular levels of glutathione on halothane-induced hepatotoxicity in hyperthyroid rats.
SO - Fundam Appl Toxicol 1984 Apr;4(2 Pt 1):221-30

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UI - 84029666
AU - James RC ; Roberts SM ; Harbison RD
TI - The perturbation of hepatic glutathione by alpha 2-adrenergic agonists.
SO - Fundam Appl Toxicol 1983 Jul-Aug;3(4):303-8

34 (BACK80) .
UI - 83288235
AU - Jernigan JD ; Pounds JG ; Harbison RD
TI - Potentiation of chlorinated hydrocarbon toxicity by 2,5-hexanedione in primary cultures of adult rat hepatocytes.
SO - Fundam Appl Toxicol 1983 Jan-Feb;3(1):22-6

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UI - 92234474
AU - Roth L ; Harbison RD ; James RC ; Tobin T ; Roberts SM
TI - Cocaine hepatotoxicity: influence of hepatic enzyme inducing and inhibiting agents on the site of necrosis.
SO - Hepatology 1992 May;15(5):934-40

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UI - 77029788
AU - Mantilla-Plata B ; Harbison RD
TI - Influence of alteration of tetrahydrocannabinol metabolism on tetrahydrocannabinol-induced teratogenesis. pp. 733-42.
SO - In: Braude MC, Szara S, ed. Pharmacology of marihuana. Vol 2. New York, Raven Press, 1976. WM 276 P536 1974. ;:

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AU - Evans MA ; Dwivedi C ; Harbison RD
TI - Enhacement of cocaine-induced lethality by phenobarbital. pp; 253-67.
SO - In: Ellinwood EH Jr., Kilbey MM, ed. Cocaine and other stimulants. New York, Plenum Press, 1977. W3 AD215 v. 21 1975. ;:

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AU - Mantilla-Plata B ; Harbison RD
TI - Alteration of delta9-tetrahydrocannabinol-induced prenatal toxicity by phenobarbital and SKF-525A. pp. 469-80.
SO - In: Nahas GG, et al., ed. Marihuana: chemistry, biochemistry, and cellular effects. New York, Springer, 1976. QV 109 S253m 1975. ;:

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UI - 74125032
AU - Wilson BJ ; Harbison RD
TI - Rubratoxins.
RF - REVIEW ARTICLE: 15 REFS.
SO - J Am Vet Med Assoc 1973 Dec 1;163(11):1274-6

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UI - 86195680
AU - Ignarro LJ ; Wood KS ; Harbison RG ; Kadowitz PJ
TI - Atriopeptin II relaxes and elevates cGMP in bovine pulmonary artery but not vein.
SO - J Appl Physiol 1986 Apr;60(4):1128-33

41 (BACK75)
UI - 79194192
AU - Fant ME ; Harbison RD ; Harrison RW
TI - Glucocorticoid uptake into human placental membrane vesicles.
SO - J Biol Chem 1979 Jul 25;254(14):6218-21

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UI - 91353301
AU - Shoaf AR ; Shaikh AU ; Harbison RD ; Hinojosa O
TI - Extraction and analysis of superoxide free radicals (.O2-) from whole mammalian liver.
SO - J Biolumin Chemilumin 1991 Apr-Jun;6(2):87-96

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UI - 81143323
AU - Koshakji RP ; Bush MT ; Harbison RD
TI - Metabolism and distribution of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) in pregnant mice.
SO - J Environ Sci Health [C] 1979;13(4):315-34

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UI - 93163938
AU - James RC ; Busch H ; Tamburro CH ; Roberts SM ; Schell JD ; Harbison RD
TI - Polychlorinated biphenyl exposure and human disease.
RF - REVIEW ARTICLE: 58 REFS.
SO - J Occup Med 1993 Feb;35(2):136-48

45 (BACK75)
UI - 78028563
AU - Evans MA ; Harbison RD
TI - GLC microanalyses of phenacetin and acetaminophen plasma levels.
SO - J Pharm Sci 1977 Nov;66(11):1628-9

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AU - Agrawal AK ; Parmar SS ; Dwivedi C ; Harbison RD
TI - Synthesis of 5-substituted 2-oxazolidinethiones and their antagonism to uterotropic effect of diethylstilbestrol.
SO - J Pharm Sci 1977 Jun;66(6):887-9

47 (BACK75)
UI - 77167803
AU - Evans MA ; Harbison RD
TI - Micromethod for determination of meperidine in plasma.
SO - J Pharm Sci 1977 Apr;66(4):599-600

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UI - 75171833
AU - Gupta AK ; Dwivedi C ; Gupta TK ; Parmar SS ; Harbison RD
TI - Synthesis of 2-(N-arylcarboxamide)-3-substituted ethoxyindoles and their monoamine oxidase inhibitory and anticonvulsant activities.
SO - J Pharm Sci 1975 Jun;64(6):1001-5

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UI - 75213633
AU - Ali B ; Kumar R ; Parmar SS ; Dwivedi C ; Harbison RD
TI - Antihemolytic and anticonvulsant activities of 1-(2,4-dichloro/2,4,5-trichlorophenoxyacetyl)-4-alkyl/arylthiosemicarbazides and their inhibition of NAD-dependent oxidations and monoamine oxidase.
SO - J Pharm Sci 1975 Aug;64(8):1329-33

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UI - 74308010
AU - Dwivedi C ; Harbison RD ; Ali B ; Parmar SS
TI - *Synthesis of substituted* anilino-(3-methoxy-4-(4-arylthiosemicarbazidocarbonylmethyleneoxy))benzyl-idenes: correlation between anticonvulsant activity and monoamine oxidase inhibitory and antihemolytic properties.
SO - J Pharm Sci 1974 Jul;63(7):1124-8

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AU - Parmar SS ; Pandey BR ; Dwivedi C ; Harbison RD
TI - Anticonvulsant activity and monoamine oxidase inhibitory properties of 1,3,5-trisubstituted pyrazolines.
SO - J Pharm Sci 1974 Jul;63(7):1152-5

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AU - Harbison RD ; Becker BA
TI - Barbiturate mortality in hypothyroid and hyperthyroid rats.
SO - J Pharm Sci 1969 Feb;58(2):183-5

53 (BACK85)
UI - 87283387
AU - James RC ; Schiefer MA ; Roberts SM ; Harbison RD
TI - Antagonism of cocaine-induced hepatotoxicity by the alpha adrenergic antagonists phentolamine and yohimbine.
SO - J Pharmacol Exp Ther 1987 Aug;242(2):726-32

54 (BACK85)
UI - 86227141
AU - Ignarro LJ ; Harbison RG ; Wood KS ; Kadowitz PJ
TI - Activation of purified soluble guanylate cyclase by endothelium-derived relaxing factor from intrapulmonary artery and vein: stimulation by acetylcholine, bradykinin and arachidonic acid.
SO - J Pharmacol Exp Ther 1986 Jun;237(3):893-900

55 (BACK85)
UI - 86088700
AU - Ignarro LJ ; Harbison RG ; Wood KS ; Kadowitz PJ
TI - Dissimilarities between methylene blue and cyanide on relaxation and cyclic GMP formation in endothelium-intact intrapulmonary artery caused by nitrogen oxide-containing vasodilators and acetylcholine.
SO - J Pharmacol Exp Ther 1986 Jan;236(1):30-6

56 (BACK85)
UI - 85237014
AU - Ignarro LJ ; Harbison RG ; Wood KS ; Wolin MS ; McNamara DB ; Hyman AL ; Kadowitz PJ
TI - Differences in responsiveness of intrapulmonary artery and vein to arachidonic acid: mechanism of arterial relaxation involves cyclic guanosine 3':5'-monophosphate and cyclic adenosine 3':5'-monophosphate.
SO - J Pharmacol Exp Ther 1985 Jun;233(3):560-9

57 (BACK80)
UI - 82216404
AU - James RC ; Goodman DR ; Harbison RD
TI - Hepatic glutathione and hepatotoxicity: changes induced by selected narcotics.
SO - J Pharmacol Exp Ther 1982 Jun;221(3):708-14

58 (BACK80)
UI - 82145271
AU - Wells PG ; Kupfer A ; Lawson JA ; Harbison RD
TI - Relation of in vivo drug metabolism to stereoselective fetal hydantoin toxicology in mouse: evaluation of mephenytoin and its metabolite, nirvanol.
SO - J Pharmacol Exp Ther 1982 Apr;221(1):228-34

59 (BACK80)
UI - 81242139
AU - Freeman RW ; Harbison RD
TI - The role of benzoylmethylecgonine in cocaine-induced hepatotoxicity.
SO - J Pharmacol Exp Ther 1981 Aug;218(2):558-67

60 (BACK80)
UI - 81071782
AU - Olson RD ; MacDonald JS ; vanBoxtel CJ ; Boerth RC ; Harbison RD ; Slonim AE ; Freeman RW ; Oates JA
TI - Regulatory role of glutathione and soluble sulfhydryl groups in the toxicity of adriamycin.
SO - J Pharmacol Exp Ther 1980 Nov;215(2):450-4

61 (BACK75)
UI - 77230052
AU - Harbison RD ; Mantilla-Plata B ; Lubin DJ
TI - Alteration of delta 9-tetrahydrocannabinol-induced teratogenicity by stimulation and inhibition of its metabolism.
SO - J Pharmacol Exp Ther 1977 Aug;202(2):455-65

62 (BACK66)
UI - 72127356
AU - Harbison RD ; Mantilla-Plata B
TI - Prenatal toxicity, maternal distribution and placental transfer of tetrahydrocannabinol.
SO - J Pharmacol Exp Ther 1972 Feb;180(2):446-53

63 (BACK66)
UI - 71037308
AU - Harbison RD ; Becker BA
TI - Effect of phenobarbital and SKF 525A pretreatment on diphenylhydantoin teratogenicity in mice.
SO - J Pharmacol Exp Ther 1970 Nov;175(2):283-8

64 (BACK66)
UI - 70083343
AU - Eling TE ; Harbison RD ; Becker BA ; Fouts JR
TI - Diphenylhydantoin effect on neonatal and adult rat hepatic drug metabolism.
SO - J Pharmacol Exp Ther 1970 Jan;171(1):127-34

65 (BACK89)
UI - 90134018
AU - Gandy J ; Millner GC ; Bates HK ; Casciano DA ; Harbison RD
TI - Effects of selected chemicals on the glutathione status in the male reproductive system of rats.
SO - J Toxicol Environ Health 1990;29(1):45-57

66 (BACK80)
UI - 83010431
AU - Jernigan JD ; Harbison RD
TI - Role of biotransformation in the potentiation of halocarbon hepatotoxicity by 2,5-hexanedione.
SO - J Toxicol Environ Health 1982 May-Jun;9(5-6):761-81

67 (BACK75)
UI - 77145913
AU - Gerber JG ; MacDonald JS ; Harbison RD ; Villeneuve JP ; Wood AJ ; Nies AS
TI - Effect of N-acetylcysteine on hepatic covalent binding of paracetamol (acetaminophen) [letter].
SO - Lancet 1977 Mar 19;1(8012):657-8

68 (BACK85)
UI - 87227924
AU - Roberts SM ; James RC ; Harbison RD ; Grund VR
TI - Histamine and hepatic glutathione in the mouse.
SO - Life Sci 1987 May 25;40(21):2103-10

69 (BACK66)
UI - 74044015
AU - Harbison RG
TI - Amniotic fluid embolism.
SO - Med J Aust 1973 Oct 6;2(14):687-8

70 (BACK66)
UI - 71200551
AU - Harbison RG, Williams RW
TI - Doctors' fees.
SO - Med J Aust 1971 May 1;1(18):985

71 (BACK66)
UI - 69296599
AU - Harbison RF
TI - Patient transportation.
SO - Med J Aust 1969 Aug 9;2(6):302-8

72 (BACK85)
UI - 86014131
AU - Teaf CM ; Harbison RD ; Bishop JB
TI - Germ-cell mutagenesis and GSH depression in reproductive tissue of the F-344 rat induced by ethyl methanesulfonate.
SO - Mutat Res 1985 Oct;144(2):93-8

73 (BACK80)
UI - 83272102
AU - Ali SF ; Cranmer JM ; Goad PT ; Slikker W Jr ; Harbison RD ; Cranmer MF
TI - Trimethyltin induced changes of neurotransmitter levels and brain receptor binding in the mouse.
SO - Neurotoxicology 1983 Spring;4(1):29-36

74 (BACK85)
UI - 88143853
AU - Goetz DW ; Hall SE ; Harbison RW ; Reid MJ
TI - Pediatric acquired immunodeficiency syndrome with negative human immunodeficiency virus antibody response by enzyme-linked immunosorbent assay and Western blot.
SO - Pediatrics 1988 Mar;81(3):356-9

75 (BACK80)
UI - 82221267
AU - Corman LI ; Foshee WS ; Kotchmar GS ; Harbison RW
TI - Simplified urinary microscopy to detect significant bacteriuria.
SO - Pediatrics 1982 Jul;70(1):133-5

76 (BACK66)
UI - 70009061
AU - Harbison RD ; Klaassen CD ; Becker BA
TI - Hemodynamics of the isolated perfused liver of hypothyroid and hyperthyroid rats.
SO - Proc Soc Exp Biol Med 1969 Oct;132(1):96-9

77 (BACK75)
UI - 77079560
AU - Evans MA ; Harbison RD ; Brown DJ ; Forney RB
TI - Stimulant actions of delta9-tetrahydrocannabinol in mice.
SO - Psychopharmacology (Berl) 1976 Nov 24;50(3):245-50

78 (BACK75)
UI - 75218762
AU - Ali B ; Parmar SS ; Dwivedi C ; Harbison RD
TI - Selective inhibition of nicotinamide adenine dinucleotide dependent oxidations by substituted carbamides.
SO - Res Commun Chem Pathol Pharmacol 1975 May;11(1):163-6

79 (BACK66)
UI - 75066772
AU - Koshakji RP ; Sastry BV ; Harbison RD
TI - Studies on the levels and nature of cholinesterase in human and mouse placenta.
SO - Res Commun Chem Pathol Pharmacol 1974 Sep;9(1):181-4

80 (BACK66)
UI - 74129426
AU - Jones MM ; Harbison RD
TI - Phthalyltetrathioacetic acid (PTTA) a new mercury chelating agent.
SO - Res Commun Chem Pathol Pharmacol 1974 Feb;7(2):389-98

81 (BACK66)
UI - 74024916
AU - Koshakji RP ; Cole J ; Harbison RD
TI - Influence of injection volume on parathion toxicity and plasma and brain cholinesterase inhibition.
SO - Res Commun Chem Pathol Pharmacol 1973 Sep;6(2):677-87

82 (BACK66)
UI - 73168742
AU - Koshakji RP ; Wilson BJ ; Harbison RD
TI - Effect of rubratoxin B on prenatal growth and development in mice.
SO - Res Commun Chem Pathol Pharmacol 1973 May;5(3):584-92

83 (BACK75)
UI - 75103360
AU - Perry LJ ; Harbison RM ; Lumb RH
TI - The use of Herr four-and-a-half clearing fluid for the rapid microscopic examination of thick sections of normal and neoplastic tissues.
SO - Stain Technol 1975 Jan;50(1):47-50

84 (MEDLINE)
UI - 93069004
AU - Gandy J ; Bates HK ; Conder LA ; Harbison RD
TI - Effects of reproductive tract glutathione enhancement and depletion on ethyl methanesulfonate-induced dominant lethal mutations in Sprague-Dawley rats.
SO - Teratogenesis Carcinog Mutagen 1992;12(2):61-70

85 (BACK89)
UI - 91262924
AU - Teaf CM ; Bishop JB ; Harbison RD
TI - Potentiation of ethyl methanesulfonate-induced germ cell mutagenesis and depression of glutathione in male reproductive tissues by 1,2-dibromoethane.
SO - Teratogenesis Carcinog Mutagen 1990;10(6):427-38

86 (BACK85)
UI - 88127593
AU - Teaf CM ; Bishop JB ; Harbison RD
TI - Depression of glutathione in male reproductive tissues and potentiation of EMS-induced germ cell mutagenesis by L-buthionine sulfoximine.
SO - Teratogenesis Carcinog Mutagen 1987;7(6):497-513

87 (BACK85)
UI - 87070502
AU - Shoaf AR ; Jarmer S ; Harbison RD
TI - Heavy metal inhibition of carnitine acetyltransferase activity in human placental syncytiotrophoblast: possible site of action of HgCl2, CH3HgCl, and CdCl2.
SO - Teratogenesis Carcinog Mutagen 1986;6(5):351-60

88 (BACK85)
UI - 86290205
AU - Jarmer S ; Shoaf AR ; Harbison RD
TI - Comparative enzymatic acetylation of carnitine and choline by human placenta syncytiotrophoblast membrane vesicles.
SO - Teratogenesis Carcinog Mutagen 1985;5(6):445-61

89 (BACK80)
UI - 84073811
AU - Freeman RW ; Harbison RD
TI - Analysis of maternal alpha-fetoprotein: a comparison of three radioimmunoassays.
SO - Teratogenesis Carcinog Mutagen 1983;3(5):407-20

90 (BACK80)
UI - 83172260
AU - Goodman DR ; Fant ME ; Harbison RD
TI - Perturbation of alpha-aminoisobutyric acid transport in human placental membranes: direct effects by HgCl2, CH3HgCl, and CdCl2.
SO - Teratogenesis Carcinog Mutagen 1983;3(1):89-100

91 (BACK80)
UI - 82153691
AU - Fant ME ; Harbison RD
TI - Syncytiotrophoblast membrane vesicles: a model for examining the human placental cholinergic system.
SO - Teratology 1981 Oct;24(2):187-99

92 (BACK66)
UI - 74176005
AU - Stevens MW ; Harbison RD
TI - Placental transfer of diphenylhydantoin: effects of species, gestational age, and route of administration.
SO - Teratology 1974 Jun;9(3):317-26

93 (BACK66)
UI - 75141260
AU - Harbison RD ; Becker BA
TI - Comparative embryotoxicity of diphenyldantoin and some of its metabolites in mice.
SO - Teratology 1974 Dec;10(3):237-41

94 (BACK66)
UI - 70075844
AU - Harbison RD ; Becker BA
TI - Relation of dosage and time of administration of diphenylhydantoin to its teratogenic effect in mice.
SO - Teratology 1969 Nov;2(4):305-11

95 (MEDLINE)
UI - 93181887
AU - James RC ; Harbison RD ; Roberts SM
TI - Phenylpropanolamine potentiation of acetaminophen-induced hepatotoxicity: evidence for a glutathione-dependent mechanism.
SO - Toxicol Appl Pharmacol 1993 Feb;118(2):159-68

96 (MEDLINE)
UI - 92067122
AU - Roberts SM ; Harbison RD ; Seng JE ; James RC
TI - Potentiation of carbon tetrachloride hepatotoxicity by phenylpropanolamine.
SO - Toxicol Appl Pharmacol 1991 Nov;111(2):175-88

97 (BACK89)
UI - 89388801
AU - Clevenger MA ; Roberts SM ; Lattin DL ; Harbison RD ; James RC
TI - The pharmacokinetics of 2,2',5,5'-tetrachlorobiphenyl and 3,3',4,4'-tetrachlorobiphenyl and its relationship to toxicity.
SO - Toxicol Appl Pharmacol 1989 Sep 1;100(2):315-27

98 (BACK89)
UI - 89267668
AU - Skoulis NP ; James RC ; Harbison RD ; Roberts SM
TI - Depression of hepatic glutathione by opioid analgesic drugs in mice.
SO - Toxicol Appl Pharmacol 1989 Jun 1;99(1):139-47

99 (BACK89)
UI - 89162498
AU - Kerger BD ; Roberts SM ; Harbison RD ; James RC
TI - Antagonism of bromobenzene-induced hepatotoxicity by the alpha-adrenoreceptor blocking agents phentolamine and idazoxan: role of hypothermia.
SO - Toxicol Appl Pharmacol 1989 Feb;97(2):360-9

100 (BACK85)
UI - 88322340
AU - Kerger BD ; Gandy J ; Bucci TJ ; Roberts SM ; Harbison RD ; James RC
TI - Antagonism of bromobenzene-induced hepatotoxicity by the alpha-adrenergic blocking agents, phentolamine and idazoxan.
SO - Toxicol Appl Pharmacol 1988 Aug;95(1):12-23

101 (BACK85)
UI - 88322346
AU - Kerger BD ; Roberts SM ; Hinson JA ; Gandy J ; Harbison RD ; James RC
TI - Antagonism of bromobenzene-induced hepatotoxicity by phentolamine: evidence for a metabolism-independent intervention.
SO - Toxicol Appl Pharmacol 1988 Aug;95(1):24-31

102 (BACK80)
UI - 84173120
AU - Koshakji RP ; Harbison RD ; Bush MT
TI - Studies on the metabolic fate of [14C]2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in the mouse.
SO - Toxicol Appl Pharmacol 1984 Mar 30;73(1):69-77

103 (BACK80)
UI - 81034285
AU - Wells PG ; Boerth RC ; Oates JA ; Harbison RD
TI - Toxicologic enhancement by a combination of drugs which deplete hepatic glutathione: acetaminophen and doxorubicin (adriamycin).
SO - Toxicol Appl Pharmacol 1980 Jun 30;54(2):197-209

104 (BACK80)
UI - 80236844
AU - Freeman RW ; MacDonald JS ; Olson RD ; Boerth RC ; Oates JA ; Harbison RD
TI - Effect of sulfhydryl-containing compounds on the antitumor effects of adriamycin.
SO - Toxicol Appl Pharmacol 1980 Jun 15;54(1):168-75

105 (BACK75)
UI - 80037154
AU - Freeman RW ; Woosley RL ; Oates JA ; Harbison RD
TI - Evidence for the biotransformation of procainamide to a reactive metabolite.
SO - Toxicol Appl Pharmacol 1979 Aug;50(1):9-16

106 (BACK75)
UI - 79077733
AU - Evans MA ; Harbison RD
TI - Cocaine-induced hepatotoxicity in mice.
SO - Toxicol Appl Pharmacol 1978 Sep;45(3):739-54

107 (BACK75)
UI - 77128589
AU - Evans MA ; Harbison RD
TI - Prenatal toxicity of rubratoxin B and its hydrogenated analog.
SO - Toxicol Appl Pharmacol 1977 Jan;39(1):13-22

108 (BACK75)
UI - 77151749
AU - MacDonald JS ; Harbison RD
TI - Methyl mercury-induced encephalopathy in mice.
SO - Toxicol Appl Pharmacol 1977 Feb;39(2):195-205

109 (BACK75)
UI - 78097387
AU - Harbison RD ; Jones MM ; MacDonald JS ; Pratt TH ; Coates RL
TI - Synthesis and pharmacological study of a polymer which selectively binds mercury.
SO - Toxicol Appl Pharmacol 1977 Dec;42(3):445-54

110 (BACK75)
UI - 76178769
AU - Harbison RD ; Dwivedi C ; Evans MA
TI - A proposed mechanism for trimethylphosphate-induced sterility.
SO - Toxicol Appl Pharmacol 1976 Mar;35(3):481-90

111 (BACK75)
UI - 76105340
AU - Mantilla-Plata B ; Harbison RD
TI - Distribution studies of (14C)delta-9-tetrahydrocannabinol in mice: effect of vehicle, route of administration, and duration of treatment.
SO - Toxicol Appl Pharmacol 1975 Nov;34(2):292-300

112 (BACK75)
UI - 75219857
AU - Harbison RD
TI - Comparative toxicity of some selected pesticides in neonatal and adult rats.
SO - Toxicol Appl Pharmacol 1975 May;32(2):443-6

113 (BACK75)
UI - 75199349
AU - Dwivedi C ; Harbison RD
TI - Anticonvulsant activities of delta-8 and delta-9 tetrahydrocannabinol and uridine.
SO - Toxicol Appl Pharmacol 1975 Mar;31(3):452-8

114 (BACK75)
UI - 75219862
AU - Harbison RD
TI - Parathion-induced toxicity and phenobarbital-induced protection against parathion during prenatal development.
SO - Toxicol Appl Pharmacol 1975 Jun;32(3):482-93

115 (BACK75)
UI - 76034372
AU - Mantilla-Plata B ; Clewe GL ; Harbison RD
TI - Delta9-Tetrahydrocannabinol-induced changes in prenatal growth and development of mice.
SO - Toxicol Appl Pharmacol 1975 Aug;33(2):333-40

116 (BACK66)
UI - 74301093
AU - Mantilla-Plata B ; Harbison RD
TI - Effects of phenobarbital and SKF 525A pretreatment, sex, liver injury, and vehicle on delta9-tetrahydrocannabinol toxicity.
SO - Toxicol Appl Pharmacol 1974 Jan;27(1):123-30

117 (BACK66)
UI - 72243732
AU - Harbison RD ; Becker BA
TI - Diphenylhydantoin teratogenicity in rats.
SO - Toxicol Appl Pharmacol 1972 Jun;22(2):193-200

118 (BACK66)
UI - 72141687
AU - Harbison RD ; Becker BA
TI - Effects of phenobarbital or SKF 525A pretreatment on diphenylhydantoin disposition in pregnant mice.
SO - Toxicol Appl Pharmacol 1971 Dec;20(4):573-81

119 (MEDLINE)
UI - 91205453
AU - Simmons HF ; James RC ; Harbison RD ; Patel DG ; Roberts SM
TI - Examination of the role of catecholamines in hepatic glutathione suppression by cold-restraint in mice.
SO - Toxicology 1991 Mar 25;67(1):29-40

120 (MEDLINE)
UI - 92055760
AU - Harbison RD ; James RC ; Roberts SM
TI - Hepatic glutathione suppression by the alpha-adrenoreceptor stimulating agents phenylephrine and clonidine.
SO - Toxicology 1991;69(3):279-90

121 (BACK89)
UI - 90194189
AU - Simmons HF ; James RC ; Harbison RD ; Roberts SM
TI - Depression of glutathione by cold-restraint in mice.
SO - Toxicology 1990 Mar 30;61(1):59-71

122 (BACK89)
UI - 89332717
AU - Skoulis NP ; James RC ; Harbison RD ; Roberts SM
TI - Perturbation of glutathione by a central action of morphine.
SO - Toxicology 1989 Aug;57(3):287-302

123 (BACK85)
UI - 88265123
AU - Smith AC ; Roberts SM ; Berman LM ; Harbison RD ; James RC
TI - Effects of piperonyl butoxide on halothane hepatotoxicity and metabolism in the hyperthyroid rat.
SO - Toxicology 1988 Jun;50(1):95-105

124 (BACK80)
UI - 84148705
AU - Liston TE ; Harbison R
TI - Sulfisoxazole chemoprophylaxis and recurrent otitis media.
SO - West J Med 1984 Jan;140(1):47-9

125 (BACK85)
UI - 89045913
AU - Smith AC ; Roberts SM ; James RC ; Berman LM ; Harbison RD
TI - Comparison of covalent binding from halothane metabolism in hepatic microsomes from phenobarbital-induced and hyperthyroid rats.
SO - Xenobiotica 1988 Aug;18(8):991-1001

Not by harbison, but I thought you might be interested - if he really did the autopsy, he should be mentioned in the article.

1
UI - 83087695
AU - Wecht CH
TI - Postmortem on Elvis Presley won't die.
SO - Leg Aspects Med Pract 1979 Dec;7(12):13-5

ABATE (34) HAS CONDUCTED NO AN ANIMAL INHALATION STUDIES HIMSELF (*)

Garrett (39) — NO ANIMAL STUDIES W/

Garrett (21) SILICA OR ASBESTOS

GARRETT Testified in PCB cases for (6) defense - 30 times

Testified about 20 times in dep + trial for O-I

(20) NO ARTICLES DEALING WITH ASB-RELATED DISEASE

NO TEXTS

NO CHAPTERS

(*) NOT EXPERT IN MINERALOGY IN IND. HYGIENE

NO dust counting.

(P.24) (*) NO COURSES IN IND. HYG. (OVER)

C.I.H

- Ind. Hygienist -
- Dust counting -
- Member ACGIH -

- NO ARTICLES ON ASBESTOS -

⊛ Testify for MERRILL DOW Chemical
Hill vs. Merrill Dow

⊛ Testify for operator of a chemical waste dump

Village of Wilsonville v. SCA Services

⊛ Testify for WITCO CHEMICAL & MONSANTO CHEMICAL

Pulsonic v. Witco Chem.

⊛ AMORELLO v. MONSANTO CHEMICAL

⊛ Testify for rubber industry
Knight v. Armstrong Rubber

⊛ Testify for VELSICOL + CHLORDANE Mfgrs -

CONDE v. VELSICOL

⊛ TESTIFY AGAINST WOMEN IN BREAST IMPLANT CASES -
DOW CORNING -

Castlemen page 322 – 327

Table 2. Asbestos Disease in Insulation Workers and Other Asbestos Product Users Medical Literature: Cases of Disease Reported Before 1964

Year	Authors	Reference	Occupations	Diseases
1932	Russell	*Proceedings of Conference Concerning Effects of Dusts Upon the Respiratory System* (held by the Industrial Commission of Wisconsin; p.180), Democrat Press	insulator	asbestosis
1933	Ellman	*J. Indust. Hyg 15:* 165-183	insulator	asbestosis
1934	Ellman	*Brit. J. Radiol.* 7:281-295	insulator	asbestosis
1934	Wood & Gloyne	*Lancet 2:* 1383-1385	boiler riveter	asbestosis
1935	Jacobson	*Acta Med, Scand. 78:* 482-488	welder	"early acute asbestosis"
1936	Ellman	*Seventh International Congress of Occupational Accidents and Illnesses*, Jean Vremans, Brussels, pp. 408-423	insulator	asbestosis
1939	Arnold, Beal and Cookson	*Brit. J. Tuberc. 33:* 45-48	asbestos handler in a chemical plant	asbestosis
1940	Wolff	*Nord. med. 5:* 535-541	3 asbestos handlers in a chemical plant	asbestosis
1940	Kuhn	*Arch. Gewerbepath. Gewerbehyg. 10:* 133-150	shipyard insulator	asbestosis

E+28

Pltf's EXHIBIT
DATE:
WITNESS:
RENEE C. ROBERTS

1941	Schrumpf	*Nord. Med. 9:* 704-706	asbestos handler in a chemical plant	asbestosis
1942	Holleb & Angrist	*Amer. J. Path. 18:* 123-131	2 insulators	asbestosis & lung cancer in both
1942	Williams	*Med. Bul. Vet. Admin.*, Vol. 18, pp. 250-253	aluminum plant worker wearing asbestos apron and gloves	asbestosis
1947	Kennaway & Kennaway	*Brit J. Cancer 1:* 260-297	insulator	asbestosis & lung cancer
1947	Mallory, Castleman, and Parris	*New Eng. J. Med 236:*407-412	insulator	asbestosis & mesothelioma
1949	Franchini & Canepa	*Med. Lavoro 40:* 161-172	insulator	asbestosis
1950	Frost	*Ugeskr. Laeger. 112:* 1284-1289	shipyard insulator	asbestosis
1951	Stoll, Bass & Angrist	*Arch. Internal Med. 88:* 831-834	plumber/insulator	asbestosis & lung cancer
1953	van Luyt	*Int'l. Conf. of Experts on Pneumoconiosis,* Sydney, Feb.-Mar. 1950, Record of Proceedings, pp. 169-171	"some" insulators	asbestosis
1953	Weiss	*Medizinische 3:* 93-94	shipyard insulator	asbestosis & pleural mesothelioma
1955	Sander	*Arch. Industr. Health 12:* 208-211	plumber's helper sawing pipecoverings	asbestosis
1956	Hempe	*Ned. Tifdschr. Geneesk. 100:* 2965-2966	insulator	asbestosis

1956	Molfino & Zannini	*Folia Med. 39:* 525-539	6 shipyard insulators	asbestosis
1957	Marks, et al.	*Am. J. Med.*, Vol. 22:51-73	pipefitter	asbestosis
1958	Pendergrass	*Am. J. Roent. 80:* 1-41	insulator	asbestosis
1958	Pendergrass	*The Pneumoconiosis Problem* Charles C. Thomas, Springfield, pp. 97-100	insulator	asbestosis
1958	Van der Schoot	*Ned. Tijdsch. Geneesk. 102:* 1125-1126	3 insulators	asbestosis & mesothelioma

TABLE 5
Publications on Mesothelioma and Asbestosis 1933-1960

Author(s), Year	Cases Reported	Ref. No.	Reference	Comment
Gloyne, S.R. (1933)	1	1	"The Morbid Anatomy and Histology of Asbestosis," *Tubercle 14*: 550-558.	"no evidence at the moment" that pleural cancer was related to asbestosis
Wood, W.B., and Gloyne, S.R. (1934)	1	2	"Pulmonary Asbestosis: A Review of One Hundred Cases," *Lancet 2:* 1383-1385.	same as above
Wedler, H.W. (1943)	2	31	"Lung Cancer in Asbestosis Patients," *Deut. Arch. Klin. Med. 191*: 189-209.	asbestos considered to cause of pleural mesothelioma

EXHIBIT #7
DATE
WITNESS
RENEE C. ROBERTS, M.P.

P. 106-108
Ex 29

Wedler, H.W. (1943) Wedler, H.W. (1944) Welz, A. and Wedler, H.W. (1944)	2	31 34 36	"Lung Cancer in Asbestosis Patients," *Deut. Arch. Klin. Med. 191*: 189-209. "Asbestose und Lugenkrebs," *Bull. Hyg. 19:* 362 Abstracts from *Bull. Hyg.* reprinted in *Abstreacts of the Literature of Industrial Hygiene,* supplement to *J. Indust. Hyg. Tox. 26:*8, 183.	same as above; abstracted in U.K. and U.S
Mallory, T.B., Castleman, B., and Parris, E.E. (1947)	1	74	"Case Records of the Massachusetts General Hospital," *New Engl. J. Med. 236:* 407-412.	pleural mesothelioma not attributed to the man's asbestos exposure
(1949)	?	84	*Annual Report of The Chief Inspector of Factories for the Year 1947* London: H.M. Stationery Ofc., pp. 79-81.	cancers of the lung and pleura counted together in demonstrating excess incidence among asbestotics
Wyers, H. (1949)	1	90	"Asbestosis," *Postgrad. Med. J. 25:* 631-638.	"endothelioma of the pleura" counted among pulmonary tumors in asbestos workers

Doig, E.T. (1949)	--	100	"Other Lung Diseases Due to Dust," *Postgrad. Med. J. 25:* 639-649.	review article mentions cancers of lung and pleura in asbestos workers
Cartier, P. (1955)	2	123	"Some Clinical Observations of Asbestosis in Mine and Mill Workers," *Arch Indust. Health 11:* 204-207.	degree of asbestosis in miners dying with pleural mesothelioma assessed as "minimal" and "none"
Smith (1952)	2	120	"Abstract of Discussion." *Arch. Indust. Hyg. Occup. Med. 5:* 262-263.	pathological material from asbestosis victims in England
Weiss, A. (1953)	1	130	"Cancer of the Pleura with Lung Asbestosis in Vivo Morphologically Ascertained," *Medizinische 3:* 93-94.	asbestos considered the cause of pleural tumor in insulation worker
Kraussler, J. and Seyss, R. (1954)	--	131	"Zur Fruhdiagnose mittels Vergrosserungsaufnahmen," *Z. Unfallmed. Berufskrankh. 47:* 59-64.	cited Weiss' case

Leicher, F. (1954)	1	139	"Primary Epithelial Tumor of the Peritoneum in Asbestosis," *Arch. Gewerbepath. Gerwebehyg. 13:* 382-391.	asbestos considered the cause of peritoneal tumor in asbestos factory worker: report abstracted in *Bulletin of Hygiene*
Leicher, F. (1955)		140	Abstract of preceding article. *Bull. Hyg. 30:* 324.	
Leicher, F. (1959)		141	Preceding abstract reprinted in *Pneumoconiosis Abstracts Vo. 3* Pitman Medical Publ. Co. London, 179.	
Bonser, G.M., Faulds, J.S., and Stewart, M.J. (1955)	6	142	"Occupational Cancer of the Urinary Bladder in Dyestuffs Operatives and of the Lung in Asbestos Textile Workers and Iron-Ore Miners," *Amer. J. Clin. Path. 25:* 126-134.	2 pleural and 4 peritoneal cancers among 72 asbestosis victims seen at autopsy
Doll, R. (1955)	1	125	"Mortality from Lung Cancer in Asbestos Workers," *Brit. J. Indust. Med. 12:* 81-86.	"endothelioma of the pleura" counted among pulmonary cancer cases

Smith, Cartier

A. M. A.
Archives of Industrial Hygiene and Occupational Medicine

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PROCEEDINGS OF THE CANCER PREVENTION COMMITTEE

THIS committee is devoted to the study of environmental factors in cancer. The proceedings published herewith are records or abstracts of lectures and discussions bearing on this subject. The views expressed are those of the individual authors and do not necessarily represent the views of the committee as a whole. The membership is composed of individuals from universities, industries, insurance companies, and public health services interested in environmental, particularly occupational, tumor problems. The committee is incorporated as a nonprofit organization in the State of New York. Meetings are held at the Department of Industrial Medicine, New York University Post-Graduate Medical School. Communications should be addressed to the Secretary, William E. Smith, M.D., 477 First Ave., New York 16.

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July, 1950, a conference was held at Oxford dealing with cancer in relation to sociological and environmental factors other than occupation. Proceedings of this conference have been published.[51]

ABSTRACT OF DISCUSSION

DR. PAUL CARTIER, L'Annonciation, Que., Canada: I have been asked to describe our experience at the Thetford Industrial Clinic in Quebec, where we see men engaged in the asbestos-mining industry in Canada. Table 3 presents data on eight cases of primary cancer of the lung which we have detected among 4,000 asbestos workers between 1940 and 1950.

In addition to those listed in the table, there was a case in which there was quite a definite clinical and radiological history of cancer of the lung but for which, unfortunately, no autopsy was performed. Also there were three other cases with a strong suspicion in favor of a cancer of the lung but for which no sufficient data are available.

On analyzing these data it seems obvious that many points would need discussion before anyone will be able to establish a causal relationship between these pathological findings and the asbestos factor.

Is it not more logical to think that the same causal factor will produce the same type of tumor, and, therefore, why do we observe such a variety of malignant tumors?

TABLE 3.—*Cases of Carcinoma of the Lungs Detected Among 4,000 Asbestos Workers 1940-1950*

Patient	Years of Exposure	Years in Industry	Age at Death	Degree of Asbestosis	Type of Tumor
J. A.	20	34	48	Minimal	Bronchogenic carcinoma
A. A.	26	35	57	Minimal	Bronchogenic carcinoma
A. L.	21	23	51	Advanced	Reticulosarcoma with pulmonary metastasis
L. G.	11	17	54	None	Bronchogenic carcinoma
L. L.	16	26	46	Minimal	Pleural mesothelioma*
E. G.	2	14	44	None	Bronchogenic carcinoma
C. B.	None significant	27	59	....	Bronchogenic carcinoma (thoracotomy)
W. C.	None	26	65	None	Pleural mesothelioma*

* "Standard Nomenclature of Diseases and Operations" published by the American Medical Association (New York, The Blakiston Company, 1933) suggests the term "mesothelial sarcoma."

If for a moment we may assume that the asbestos fibers might produce a bronchogenic carcinoma in asbestotic employees with significant exposure, then only two cases of the series would serve as evidence because the cases of mesothelioma or lymphosarcoma and cases without exposure or without asbestosis do not have the conditions required.

As a last remark, it would appear necessary that any statistics on occupational cancer of the lung, to be conclusive, should indicate the precise variety of lung cancer.

MR. EDWARD A. LEW, New York: The difference in the frequency of lung cancer in persons with asbestotic lungs as compared with the large group of silicotics provided by the Pneumoconiosis Board seems impressive. However, one might wonder whether the two groups had been examined for cancer with equal care. I am impressed by the fact that when different groups of lungs were studied by the same observer, Dr. Gloyne, the differences in frequency of lung cancer were much less. If we restrict our attention solely to Dr. Gloyne's series, I am not sure that the differences are statistically significant.

DR. A. J. LANZA, New York: I think it is important to distinguish between cases of clinically recognizable and fatal lung cancer, on one hand, and cases in which the diagnosis of lung cancer has been made as more or less of an incidental finding at autopsy, on the other. The English claim for an association between lung cancer and asbestosis has been based, it seems to me, on data obtained from lungs that a pathologist has searched with a microscope. I wonder how many

51. Conference on Geographical Pathology and Demography of Cancer, J. Nat. Cancer Inst. 11:627-662, 1950.

cases of lung cancer would be found if lungs from any sort of population were subjected to the same minute scrutiny?

DR. SMITH: In regard to Dr. Cartier's query as to the significance of different types of lung cancer, it may be pertinent to note that a diversity of tumors was found in the lungs of chromate workers reviewed by Dr. Anna Baetjer. Experimentally, the polycyclic hydrocarbons evoke a great variety of tumor types. If asbestos is carcinogenic, I would not expect to see it produce only one type of tumor. I am interested in your observation of two cases of pleural mesothelioma. This is a rather rare tumor. In examining pathological material sent us from England by Dr. Hinson, we found among six cancerous asbestotic lungs two presenting alveolar cell carcinoma. These tumors are often diagnosed as "pleural mesothelioma." I should very much like to exchange slides with you.

In reply to Mr. Lew and Dr. Lanza, it was my understanding that the English cases of silicosis and their cases of asbestosis were handled in the same general fashion through the Pneumoconiosis Board and that the lung cancer rate found in silicotics could therefore serve as a reasonably satisfactory control for the lung cancer rate in cases of asbestosis. In Dr. Gloyne's series of 81 cases of lung cancer in the several pneumoconiosis groups and in the control group, the tumor was evident in the gross in all except 6. Some of his material was destroyed during the bombing of London, but his recollection was that all of the cancers in asbestotics were recognizable in the gross.

DR. JOHN L. POOL, New York: Have experimental studies thrown any light on the question of lung cancer in relation to asbestosis?

DR. SMITH: In 1941, Nordmann and Sorge in Germany claimed to have produced cancer in the lungs of mice by exposing them repeatedly to asbestos dust. Their statement that cancer had resulted in 20% of their exposed animals is occasionally quoted. Actually, their "20%" figure referred only to two mice, one of which had a type of tumor that occurs commonly as a spontaneous growth in mice. The other they considered to be a squamous cell carcinoma, but their photograph does not impress me as that of epidermoid carcinoma. It seems rather to show merely an area of squamous cell metaplasia.

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TABLE 5
Publications on Mesothelioma and Asbestosis 1933-1960

Author(s), Year	Cases Reported	Ref. No.	Reference	Comment
Gloyne, S.R. (1933)	1	1	"The Morbid Anatomy and Histology of Asbestosis," *Tubercle 14*: 550-558.	"no evidence at the moment" that pleural cancer was related to asbestosis
Wood, W.B., and Gloyne, S.R. (1934)	1	2	"Pulmonary Asbestosis: A Review of One Hundred Cases," *Lancet 2:* 1383-1385.	same as above
Wedler, H.W. (1943)	2	31	"Lung Cancer in Asbestosis Patients," *Deut. Arch. Klin. Med. 191*: 189-209.	asbestos considered to cause of pleural mesothelioma

Ex 29

Wedler, H.W. (1943)	2	31	"Lung Cancer in Asbestosis Patients," *Deut. Arch. Klin. Med. 191:* 189-209.	same as above; abstracted in U.K. and U.S
Wedler, H.W. (1944)		34	"Asbestose und Lugenkrebs," *Bull. Hyg. 19:* 362	
Welz, A. and Wedler, H.W. (1944)		36	Abstracts from *Bull. Hyg.* reprinted in *Abstracts of the Literature of Industrial Hygiene,* supplement to *J. Indust. Hyg. Tox. 26:*8, 183.	
Mallory, T.B., Castleman, B., and Parris, E.E. (1947)	1	74	"Case Records of the Massachusetts General Hospital," *New Engl. J. Med. 236:* 407-412.	pleural mesothelioma not attributed to the man's asbestos exposure
(1948) 7	?	84	*Annual Report of The Chief Inspector of Factories for the Year 1947* London: H.M. Stationery Ofc., pp. 79-81.	cancers of the lung and pleura counted together in demonstrating excess incidence among asbestotics
Wyers, H. (1949)	1	90	"Asbestosis," *Postgrad. Med. J. 25:* 631-638.	"endothelioma of the pleura" counted among pulmonary tumors in asbestos workers

Doig, E.T. (1949)	--	100	"Other Lung Diseases Due to Dust," *Postgrad. Med. J. 25:* 639-649.	review article mentions cancers of lung and pleura in asbestos workers
Cartier, P. (1955)	2	123	"Some Clinical Observations of Asbestosis in Mine and Mill Workers," *Arch Indust. Health 11:* 204-207.	degree of asbestosis in miners dying with pleural mesothelioma assessed as "minimal" and "none"
Smith (1952)	2	120	"Abstract of Discussion." *Arch. Indust. Hyg. Occup. Med. 5:* 262-263.	pathological material from asbestosis victims in England
Weiss, A. (1953)	1	130	"Cancer of the Pleura with Lung Asbestosis in Vivo Morphologically Ascertained," *Medizinische 3:* 93-94.	asbestos considered the cause of pleural tumor in insulation worker
Kraussier, J. and Seyss, R. (1954)	--	131	"Zur Fruhdiagnose mittels Vergrosserungsaufnahmen," *Z. Unfallmed. Berufskrankh. 47:* 59-64.	cited Weiss' case

Leicher, F. (1954)	1	139	"Primary Epithelial Tumor of the Peritoneum in Asbestosis," *Arch. Gewerbepath. Gerwebehyg. 13:* 382-391.	asbestos considered the cause of peritoneal tumor in asbestos factory worker: report abstracted in *Bulletin of Hygiene*
Leicher, F. (1955)		140	Abstract of preceding article. *Bull. Hyg. 30:* 324.	
Leicher, F. (1959)		141	Preceding abstract reprinted in *Pneumoconiosis Abstracts Vo.* 3 Pitman Medical Publ. Co. London, 179.	
Bonser, G.M., Faulds, J.S., and Stewart, M.J. (1955)	6	142	"Occupational Cancer of the Urinary Bladder in Dyestuffs Operatives and of the Lung in Asbestos Textile Workers and Iron-Ore Miners," *Amer. J. Clin. Path. 25:* 126-134.	2 pleural and 4 peritoneal cancers among 72 asbestosis victims seen at autopsy
Doll, R. (1955)	1	125	"Mortality from Lung Cancer in Asbestos Workers," *Brit. J. Indust. Med. 12:* 81-86.	"endothelioma of the pleura" counted among pulmonary cancer cases

Table 2. Asbestos Disease in Insulation Workers and Other Asbestos Product Users Medical Literature: Cases of Disease Reported Before 1964

Year	Authors	Reference	Occupations	Diseases
1932	Russell	*Proceedings of Conference Concerning Effects of Dusts Upon the Respiratory System* (held by the Industrial Commission of Wisconsin; p.180), Democrat Press	insulator	asbestosis
1933	Elilman	*J. Indust. Hyg 15*: 165-183	insulator	asbestosis
1934	Elilman	*Brit. J. Radiol.* 7:281-295	insulator	asbestosis
1934	Wood & Gloyne	*Lancet 2:* 1383-1385	boiler riveter	asbestosis
1935	Jacobson	*Acta Med. Scand. 78:* 482-488	welder	"early acute asbestosis"
1936	Elilman	*Seventh International Congress of Occupational Accidents and Illnesses*, Jean Vromans, Brussels, pp. 408-423	insulator	asbestosis
1939	Arnold, Seal and Cookson	*Brit. J. Tuberc. 33:* 45-48	asbestos handler in a chemical plant	asbestosis
1940	Wolff	*Nord. med. 5:* 535-541	3 asbestos handlers in a chemical plant	asbestosis
1940	Kuhn	*Arch. Gewerbepath. Gewerbehyg. 10:* 133-150	shipyard insulator	asbestosis

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1941	Schrumpf	*Nord. Med. 9:* 704-706	asbestos handler in a chemical plant	asbestosis
1942	Holleb & Angrist	*Amer. J. Path. 18:* 123-131	2 insulators	asbestosis & lung cancer in both
1942	Williams	*Med. Bul. Vet. Admin.*, Vol. 18, pp. 250-253	aluminum plant worker wearing asbestos apron and gloves	asbestosis
1947	Kennaway & Kennaway	*Brit J. Cancer 1:* 260-297	insulator	asbestosis & lung cancer
1947	Mallory, Castleman, and Parris	*New Eng. J. Med 236:*407-412	insulator	asbestosis & mesothelioma
1949	Franchini & Canepa	*Med. Lavoro 40:* 161-172	insulator	asbestosis
1950	Frost	*Ugeskr. Laegar. 112:* 1284-1289	shipyard insulator	asbestosis
1951	Stoll, Bass & Angrist	*Arch. Internal Med. 88:* 831-834	plumber/insulator	asbestosis & lung cancer
1953	van Luyt	*Int'l. Conf. of Experts on Pneumoconiosis,* Sydney, Feb.-Mar. 1950, Record of Proceedings, pp. 169-171	"some" insulators	asbestosis
1953	Weiss	*Medizinische 3:* 93-94	shipyard insulator	asbestosis & pleural mesothelioma
1955	Sander	*Arch. Industr. Health 12:* 208-211	plumber's helper sawing pipecoverings	asbestosis
1956	Hempe	*Ned. Tifdschr. Geneesk. 100:* 2965-2966	insulator	asbestosis

1956	Molfino & Zannini	*Folia Med. 39:* 525-539	6 shipyard insulators	asbestosis
1957	Marks, et al.	*Am. J. Med.*, Vol. 22:51-73	pipefitter	asbestosis
1958	Pendergrass	*Am. J. Roent. 80:* 1-41	insulator	asbestosis
1958	Pendergrass	*The Pneumoconiosis Problem* Charles C. Thomas, Springfield, pp. 97-100	insulator	asbestosis
1958	Van der Schoot	*Ned. Tijdsch. Geneesk. 102:* 1125-1126	3 insulators	asbestosis & mesothelioma

MEMORANDUM

TO: **Shep Hoffman**

FROM: **Sheri Ingram (Ness, Motley)**

RE: **Raymond D. Harbison**

DATE: **June 17, 1994**

The following is a compilation of deposition summaries, testimony summary and my editorial notes regarding Raymond Harbison. Please give me a call if you have any questions.

1. Deposition of Raymond Harbison taken 03/10/94 for this Angeletti trial.

Page 11 - Has never testified on behalf of Plaintiffs in any asbestos related matter. Has testified on behalf of Owens-Illinois for 3 or 4 years although does not admit to testifying "on behalf of"

Page 20 - Has never written any chapter for text or any articles about asbestos-related diseases. Has previewed articles dealing with asbestos-related diseases. Has never conducted any laboratory studies dealing with asbestos or asbestos-related diseases.

Page 21 - Not an expert in epidemiology, not an expert in mineralogy, not an expert in cardiology, not an expert in pulmonology.

Page 22 - Does have some knowledge of cardiology and pulmonology because he uses in the field of toxicology, but he is not an expert. Is an expert in oncology; teaches oncology and pathology and has for about 10 years.

Page 23 - Has expertise in occupational medicine; has been involved in the managing and development process in the development of programs in this area for about 20 years. Considers himself an expert in pathology because he teaches it currently at the University of Florida, College of Medicine. His formal training consists of two courses taken at the University of Iowa and he has no post-graduate degrees in pathology.

Page 24 - He claims some expertise in industrial hygiene because he uses it in the area of toxicology but he has taken no courses. He uses industrial hygiene in the evaluation of exposure and to assess risk. Industrial hygiene is the science which is used to evaluate risk. He is not an expert in dust measurements.

Page 25 - Has conducted a search of the scientific or medical literature on asbestos-related diseases for the past 20 years because it is part of his teaching responsibilities. He teaches the effects of asbestos and it's exposure and has for about 20 years.

Page 26 - He claims to have been teaching the hazards of asbestos in the early '70s. He would not cite any specific text that he has used, he simply refers to the scientific literature.

When asked what toxicology text he considered to be valuable, he cited Ellen Horn's textbook of toxicology and Goldfrank's textbook of toxicology.

Page 30 & 31 - He has published chapters in Dr. Doull's text but he may not agree with all of his opinions. When asked what he did not agree with he said that he was not familiar with Doull's opinions.

Page 31 - He has had courses or training regarding asbestos-related diseases from the late '60s up until the present. He cites those courses as continuing education and I guess the training comes from his membership in professional societies and the attendance of some classes. He was not more specific.

Page 32 - 1961 was his first experience with animal studies. He would not agree that the protocol of animal inhalation studies was known in 1940.

There was some confusion about what Raymond Harbison will testify about in this upcoming matter because the disclosure statement was inaccurate.

Page 36 - When I began asking him different state-of-the-art questions regarding his review of the scientific and medical literature, he was advised by the OI attorneys that he would not be testifying on anything regarding state-of-the-art. His testimony is limited to simply his review of the Saranac Lake documents and his evaluation of that testing procedure.

He does not claim expertise in conducting animal studies. He does have knowledge of the history of animal studies, however.

Page 37 - He did not conduct any dust inhalation studies while at Drake; he did not conduct any dust inhalation studies while at Iowa; he did not conduct any dust inhalation studies during his doctoral training. He did conduct dust inhalation studies while at Tolane and says that he is presently doing so.

Page 37 - *** He has never conducted any dust inhalation studies dealing with asbestos.

Page 42 - He will not testify about mesothelioma or anything dealing with the latency periods.

Page 43 - Again, he states that he will only testify regarding his review of the Saranac Lake documents. He will not testify about the two plaintiff's specific case at all.

Page 44 - He again states that he will only testify about the Saranac Lake documents and his evaluation of that testing. He has only review the Hazard deposition and the exhibits thereto.

Page 45 - *** His opinion is that the conclusions provided by the Saranac were consistent with the animal testing data, that at some levels of exposure to the dust there was a finding after some period of time, in that the changes were consistent with asbestosis.

Page 47 - When asked what he thought the purpose of the Kaylo study was, he answered that Owens-Illinois had the testing conducted on the Kaylo product because of OI's concerns about silica and whether the exposure to silica would cause silicosis. **(He contradicts himself here to a certain extent compared to the direct examination in the West Virginia trial 1993 where he stated that the concerns of OI were regarding tuberculosis. On page 46 of the direct examination he says that the purpose of the Kaylo study was to determine whether or not there was a hazard that would be associated with the use of this product)**

Page 48 - In my deposition of him in this matter he said that there was no concern on the part of OI about asbestosis when they asked Saranac to conduct the studies on Kaylo.

Page 54 - Does not have an opinion when scientific methodology existed to conduct animal inhalation studies. Saranac had a good reputation for inhalation studies in the '30s and '40s.

Page 54 - He does not know if Dr. Gardner was considered an authority on asbestos-related diseases in the '30s and '40s.

Page 56 - He agreed that to known the latency period of a disease would influence the testing protocol. He agreed that it would be helpful but that it was no necessary to known whether a disease was progressive or not.

Page 58 - He does not know if Gardner or any of the other scientists at Saranac had an understanding of the pathogenesis of asbestosis in the '40s and '50s.

Page 59 - He evaluated the dust studies on Kaylo based on scientific knowledge at that time.

Page 60 - He will not render an opinion about what was known in the 1940s about levels of exposure to asbestos and what levels would cause asbestosis. He then goes on to state that it is not necessary to known what levels of exposure cause asbestosis. **(I think you've got some inconsistency here that you can definitely work with.)**

Page 61 - ** He evaluated levels of exposure to Kaylo that resulted in changes or effects to lab animals and used the information to evaluate the adequacy of the studies and the amounts it took to produce those specific effects.

When asked if there was anything which appeared in the Saranac documents or in the deposition of Mr. Hazard which indicated that there was a safe exposure to Kaylo dust which contained asbestos he stated that the threshold limit values were the levels that were not associated with adverse effects and those levels were the levels that OI used for protecting individuals exposed to the Kaylo material. (How does he know that when they only tested 100 million particles per cubic foot of air?)

Page 62 - He says that the Kaylo products were safe because they complied with the TLV from 1942-1958.

Page 64 - He is familiar with the Selikoff studies. **(Check that because I'm pretty sure that in Charles Patrick's deposition he's never her of Selikoff)**.

Page 69-76 - Goes through the disclosure statement and I point out certain statements that he does not understand what they mean. Specifically, on Page 75. ** On page 76 & 77 the only two data sets or data evaluation that he has completed is out of number one the levels of exposure of 5 million particles per cubic foot of air and the Saranac testing, which was the 100 million particles. He has not look at what levels would cause various diseases or effects. **(Therefore, his only knowledge is derived from those two data sets and he shouldn't be competent to testify regarding anything in between and/or below it.)**

Page 78 - The 26b statement in this matter said that he would testify about the difference between the significance of toxicology in both the regulatory process and the scientific process.

Page 79 - He does not plan to testify about that but on Page 81 he does say with regard to regulatory process, there is not a requirement for an animal test to be extrapolatable to humans but that it is assumed that it can be. Also on 81 the disclosure statement also says he will discuss the development of knowledge as to the risk of asbestos-related disorders.

Page 82 - He has not been asked to do that.

Summary of the testimony of Harbison from Defense witness for OI 03/12/93 West Virginia.

Obtained his Ph.D.s in pharmacology and toxicology from the University of Iowa 1969 **he is not an M.D.**

Page 6 - Serves as an advisor to; National Institute of Environmental Health Sciences, he serves on the committee; Science Advisory Board of the EPA where he responds to chemical spills and provides technical assistance to those teams;

Page 7 - Serves as advisor to NIOSH reviewing studies that become the basis for their regulations to monitor or control chemicals in the workplace; serves as Consultant to the Department of Justice and Dept. of Agriculture; teaches second year med students pathology and teaches toxicology to the Pharmacology program at the University of Florida, College of Medicine.

Page 8 - He has done research which has been funded by NIH for about 25 years, the cause by which chemicals produce adverse effects on living systems.

Page 9 - Also to make judgments of how potential adverse effects that chemicals have on humans. He also has a private practice in toxicology.

Elvis - He participated in the autopsy of Elvis which is his claim to fame.

Page 10 - When he was at Vandebuilt Medical Center he was involved in the autopsy of Elvis in ultimately trying to determine his cause of death which was drug overdose. He seems to be very proud of that fact.

Page 12 - Toxicology has only been a recognized science since the early '60s.

Page 13 & 14 - He gives a long speech on the explanation of risk assessment but seems to get himself into a little bit of trouble here. Maybe there is something you can use. Basically, he says that there are two things that as a scientist you need to know for risk assessment. Number one you must know what can this chemical do, or the toxicity, in other words what are it's potential harmful effects and number two the exposure. He goes on to explain toxicity by stating that once I know what the toxicity is, I can't change the toxicity. It is what it is. If that chemical causes cancer, then I can't change that. **(This statement indicates that some substances are toxic, while some are not. As you will see, his contention is that everything is toxic at a certain level. So I would ask him at this point that if his contention was that everything, including water is toxic at a certain level therefore, you could find toxicity by exposure, then I don't quite understand his statement regarding the two tiered definition of risk assessment.)**

Page 18 & 19 - They are a long explanation of TLV. He states that TLV is calculated or determined by taking that concentration that does not produce an effect and applying a margin of safety to that concentration to lower it even further.... that is a value.... which we can recommend with confidence to that worker. **(So I would ask him that if a manufacturer complied with the TLV, then his contention would be that there actions would be prudent and safe. If Harbison states yes to this answer then go to page 90 which is the OI May 25, 1951 report, that the maintenance of the working environment is a condition which conforms to regulations promulgated by local health agencies or which is in accord with accepted standards of good practice should <u>not</u> be considered as a complete protection for a worker from acquiring a disease as a result of his occupation.**

(Obviously after he goes through the discussion about water and salt and shampoo and asbestos are all alike in that at a certain level they can all be harmful to you. You could obviously go into the fact that we don't have TLVs for water or shampoo, or the requirement of warning labels.)

Page 29 - He was asked about the lack of warnings for all these other products and he stated that if we put warnings on everything it would loss it's effect on the consumer, therefore we only label those materials which have a greater propensity or greater likelihood for producing harm. **(So you would agree that asbestos has a greater propensity for producing harm than all of these substances you have told the jury could be toxic. So that is a statement that I would read to him and ask him if he agreed with it, since he made it. Then obviously asbestos containing products are required to have warning labels, are they not. You could go into asking him since when the warnings were required and what governmental body first authorized it. He will tell you that OSHA authorized the warnings but I don't know that he knows the year. Also, you can insert at this point page 16**

of Charles Patrick's deposition which was taken March 29, 1994 for Kanau County, West Virginia wherein he squirmed around a bit and made himself look silly when Charles read the statement "that OSHA is aware of no instance in which exposure to a toxic substance has more clearly demonstrated detrimental health effects on humans than has asbestos exposure" Harbison was then asked if he agreed or disagreed with this statement and he said that he would have to look at the context in which the statement was made, whether that was in reference to an overexposure or in reference to an affective dose. He then had to agree that for asbestos there are clearly demonstrated toxic effects that can occur as a result of some levels of exposure to asbestos this is on page 16 & 17 of this depo.)

Page 36 - Regarding asbestos and any effects it may have he has only testified for the defendant OI.

Page 39 - He's been asked to review the methodology of the Saranac studies for it's appropriateness and whether it is usable for some intended purpose. He has not reviewed the medical literature. **(In Patrick's deposition he said that he had reviewed the medical literature so I'm not sure what he intends to testify about in this trial.)**

Page 40 - They now go through the Saranac documents and exhibits to Hazard deposition and the first Saranac letter is dated 02/12/43 which is a letter from Bowes to Dr. Gardner stating that OI is concerned with the new product Kaylo in that it contains silica materials which is a derivative of sand and are questioning whether there could be any lung problems related to this product.

Page 41 - The second letter is dated 03/12/43 it is a letter from Saranac to OI which states the fact....that you were starting with a mixture of quartz and asbestos....suggest a first class hazard.

Page 45 - Harbison's interpretation is that Saranac cannot tell them about this new form until it is tested.

Page 46 - The purpose of the Kaylo study was to determine whether or not there was a hazard that would be associated with the use of this product.

Page 47 - He stated that this study used guinea pigs, rats and rabbits and not white mice because they are not as good to use because they are highly vulnerable to developing carcinomas. They have very high spontaneous occurrence of some forms of cancer, particularly lung cancer.

Page 48 - They started with rabbits because of the size. They crushed the Kaylo and injected it with a syringe into the rabbits. They all died of pneumonia because of the way the product was given to the rabbits so the experiments were abandoned. Then, they exposed the rats and guinea pigs by air and although it was supposed to be for 8 hours a day for a five day week the attorney points out on direct examination that indeed the rats and guinea pigs were not removed from the cages so therefore they were in the dust 24 hours a day.

Page 49 & 50 - The particle concentration was 115 million per cubic foot of air. The TLV at the time was 5 million.

Page 50 - He goes on about the reputation of the Saranac lab being the best in the country.

Page 52 - Yet they conducted a test to simply answer the question, at any level of exposure, can an effect be produced? **(Don't need to be an industrial hygienist or a toxicologist to know that, even in that year.)**

Page 54 - Witness is talking about how this type of testing is not common. He explains that it is very difficult to do this type of testing even today. It is technically very difficult, it is extremely expensive, and most substances would not be tested because of those difficulties. I think the work that was done by Saranac Lake was certainly pioneering work in that time, in the '40s. And even today it would be very difficult to do those kinds of studies.

Page 55 - Response of Saranac Laboratories back to OI "at the present time there is nothing to suggest that reaction comparable to asbestosis or silicosis will ultimately develop". It was a fifteen month study. Saranac was asked by OI to continue the study to see if a longer period of exposure might result in adverse effects.

Page 57 - The next document is a letter from Dr. Vorwald to Hazard 10/30/47 the study comprised of 18 months testing on the rats and 30 months with the guinea pigs concerns. The concerns were whether tuberculosis would develop with longer exposure; no fibrosis present.

Page 60 - Many of the particles were too big to stay suspended in the air. The conclusion with the guinea pigs was that Kaylo alone fails to produce significant pulmonary damage when inhaled into the lungs. There was no tissue reaction.

Page 65 - 11/16/48 continue to study because of tuberculosis concerns.

Page 66 - *** When all the animals showed early lesions and also true fibrosis of the type characteristic of a response of asbestos at the end of 36 months.

Page 67 - Was OI surprised at the results and the answer was no I don't think that is surprising. I think that they were probably expecting a fibrosis to occur and to find an asbestosis. **(In 1948-there were no surprises, i.e. knew asbestos could cause asbestosis. Also this contradicts his earlier contention that they did not mention that one of the goals or the purposes behind the study was to determine whether asbestosis could occur from Kaylo dust. His contention was at one point silicosis and then tuberculosis.)**

Page 70 - The witness knows that OI kept dust levels at their plants that made Kaylo at 5 million particles per cubic foot of air. He also goes on to talk about how the X-ray program showed no changes.

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Page 71 - Conclusion asbestos is one of many dusts that are hazardous if at a certain level of exposure over a long time. His opinion is that the data transmitted to OI did not indicate that at the current use level of 5 million particles per cubic foot that there was a hazard or risk associated with the use of Kaylo.

Page 72 - OI kept pushing Saranac to publish the results.

Page 73 - OI had no input into the preparation of the report.

Page 74 - 1955 the effect of inhaled commercial hydrocalcium silicate dust or animal tissue by Schepers printed in the American Medical Association Archives of Industrial Health.

Page 75 - No reporting of any cancer in the study. He could not find any cancerous changes therefore OI knew Kaylo did not cause cancer. There was also no mention of mesothelioma. **(The attorney leads the witness by stating that Saranac did not tell OI to stop making Kaylo nor did it tell them to remove the asbestos from Kaylo nor did it tell them to warn. I think it would be worth pointing out that a prudent manufacturer should not seek to escape it's responsibilities by blaming a lab that simply did it's testing, not to mention that it was not Saranac's job to enforce warnings.**

Cross-Examination begins which I did not think was very strong.

Page 81 - Witness does not recall Kaylo as being toxic until the attorney points out that a January 1952 Saranac document

Page 84 - which refers to the propensities of Kaylo as being toxic. Witness states that all materials are toxic and that the dose determines whether they are or not.

Page 90 - The attorney reads OI May 25, 1951 report, Kaylo Division plant, Industrial Hygiene survey by Saranac "the maintenance of the working environment in a condition which conforms to regulations promulgated by local health agencies or which is in accord with accepted standards of good practice should not be considered as a complete protection for a worker from acquiring a disease as a result of his occupation." **(This was referred to earlier if the witness stated that compliance with the TLV was prudent behavior exercised by a manufacturer at the time. Witness says that what Saranac is saying is that compliance alone with TLV and not looking at the workers themselves would not be prudent or sufficient.... need to maintain medical program too. This is his interpretation of the document which talks about protection from acquiring a disease.**

Page 95 - Witness knew in 1943 when the studies began that it was known that asbestos could cause asbestosis. **(Again, he contradicts himself to a certain extent.)** The witness goes on to state that the Saranac documents do not tell us about cancer.

Page 97 - Witness does not know anything about the industrial health digest materials that OI had been receiving over the years.

Page 99 - The witness would not agree with Dr. Gardner's 1943 letter to Bowes "that the fact that you were starting with a mixture of quartz and asbestos would certainly suggest that you have all the ingredients for a first class hazard." The witness agreed that it turned out to be a hazard at some levels of exposure. Witness feels all substances at some level of exposure can produce harm.

Page 105 - Witness doesn't know what safety measures OI formulated for the end users. Witness doesn't know about the approval of respirators.

Page 107 & 108 - Conclusion of the 1948 report was that Kaylo, because of it's content of an appreciable amount of fibrous chrysotile is capable of producing asbestosis and should be handled as a hazardous industrial dust.

Redirect on 109 - Witness explains the differences with the TLV. Witness says the test showed at high concentrations of 100 million particles per cubic foot of air you could product asbestosis. But TLV, that level which or below which no adverse effects would occur was 5 million. Therefore there was so large a safety margin between the two and that the standard practice of the 5 million particles was appropriate and also OI went further and used the advise of Saranac to continue to look at those employees by medical examination of those individuals continuously.

Page 110 - Reports only talk about asbestosis. Kaylo would not be considered toxic in substance for causing lung cancer or mesothelioma. **(How in the world does this man have the knowledge or foundation to make this sort of statement.)**

Page 111 - It is correct that OI did not warn if the dust levels were above 5 million particles per cubic foot of air.

There is also a deposition taken by Charles Patrick, a partner with our firm, in the West Virginia Kanau County case on 03/29/94. Harbison was being offered on behalf of Westinghouse. I have read that deposition but have not made very many notes. For the most part, Harbison goes into the same type of contentions that asbestos can be toxic just as all other substances depending on the level of exposure. He also talks about the TLV quite a bit and states that levels below the present .2 fibers have no increased risk of asbestosis, lung cancer or mesothelioma. Charles again has made it clear that this man is not designated as an expert in asbestos or state-of-the-art. Charles thought that most of his testimony was not very relevant and he would make a motion to exclude on the basis of comparative fault. He feels that his testimony is not even relevant in this type of case and that it only tends to confuse the jury. As far as his review of the Saranac documents is concerned, Charles stressed the fact that this man is neither a medical historian nor was he being offered as an expert in asbestos. Apparently he obtained his expertise in asbestos about 3 or 4 years ago when OI first hired him to review these documents. Charles also felt that while the purpose of this man's testimony was to demistify TLVs, he obviously knows very little about the intent of the TLV.

Again although he is not listed as an expert in asbestos, he does believe that he is an expert nonetheless. However, he does not know the name of Vagner, and he knows very little about the Selikoff studies.

I have had a med-line done on him as well as a West Law search. I have enclosed the Med-Line articles and the West Law material. Primarily all of his testimony has been dealing with chemical exposure cases. Most all of the articles that he has written or jointly written have dealt with drugs in one form or another. His big claim to fame is the fact that he participated in the autopsy of Elvis Presley. However, I have enclosed an article about the problem surrounding that autopsy and the fact that there was some cover-up. No where in the article is the name Raymond D. Harbison mentioned at all.

Charles had also mentioned that because he testified on behalf of OI regarding the Kaylo documents, Charles had asked him whether or not he had met or read transcripts of prior OI experts. Charles asked him if he had ever read transcripts of Harry Demopoulous. The witness claimed never to have talked or to have known anything about Harry Demopoulous. The witness the amended his statement to say that he had seen Harry Demopoulous' name in a credit in a movie. He is obviously lying about this which indicates that he is lying about other matters as well. I am not familiar with Harry Demopoulous or many of the OI experts so it is difficult for me to comment any further on this matter.

Good luck. I'll be glad to help you any way I can.